

2022

Environmental Effects on Health: Ignorance and Undone Science

Don Want

Follow this and additional works at: <https://ro.uow.edu.au/theses1>

University of Wollongong

Copyright Warning

You may print or download ONE copy of this document for the purpose of your own research or study. The University does not authorise you to copy, communicate or otherwise make available electronically to any other person any copyright material contained on this site.

You are reminded of the following: This work is copyright. Apart from any use permitted under the Copyright Act 1968, no part of this work may be reproduced by any process, nor may any other exclusive right be exercised, without the permission of the author. Copyright owners are entitled to take legal action against persons who infringe their copyright. A reproduction of material that is protected by copyright may be a copyright infringement. A court may impose penalties and award damages in relation to offences and infringements relating to copyright material.

Higher penalties may apply, and higher damages may be awarded, for offences and infringements involving the conversion of material into digital or electronic form.

Unless otherwise indicated, the views expressed in this thesis are those of the author and do not necessarily represent the views of the University of Wollongong.

Research Online is the open access institutional repository for the University of Wollongong. For further information contact the UOW Library: research-pubs@uow.edu.au



UNIVERSITY OF WOLLONGONG AUSTRALIA

**Environmental Effects on Health:
Ignorance and Undone Science**

A thesis submitted as fulfilment of the requirement for the award of the degree

**Doctor of Philosophy
from
University of Wollongong**

by

Don Want, BE (Hons)

**School of Humanities and Social Inquiry,
Faculty of the Arts, Social Sciences and Humanities**

2022

Certification

I, Don Want, declare that this thesis, submitted for the award of Doctor of Philosophy in the School of Humanities and Social Inquiry, University of Wollongong, is wholly my own work unless referenced or acknowledged. The document has not been submitted for qualifications in any other academic institution.

Don Want

30 Jan 2022.

TABLE OF CONTENTS

Certification	2
TABLE OF CONTENTS	3
ABSTRACT	4
ACKNOWLEDGEMENTS.....	6
ABBREVIATIONS	7
Chapter 1	10
Introduction	10
Chapter 2	35
Is Health Affected by Environmental Exposures?.....	35
Chapter 3	177
Multiple Chemical Sensitivity (MCS).....	177
<u>APPENDIX 1: THE MCS MECHANISM HYPOTHESIS BY PALL AND ITS OVERLAPS.....</u>	<u>307</u>
<u>APPENDIX 2: SOME TECHNICAL DETAILS ON CIRS.....</u>	<u>312</u>
Chapter 4	320
Ignorance Concerning Environmental Effects on Health	320
Chapter 5	450
Manipulating Knowledge.....	450
<u>APPENDIX 1: GENERAL ANALYSIS OF A PUBLISHED STUDY CONSIDERING THE CHEMICALLY SENSITIVE PORTION OF THE POPULATION</u>	<u>507</u>
Chapter 6	513
Conclusion.....	513

ABSTRACT

Considerable research has been completed showing that environmental exposures can have significant effects on people's health, especially in terms of autoimmune conditions, cancers, and neurological and psychological conditions. Health effects are possible at exposure levels far below those generally considered safe by orthodox health authorities. A prime example is multiple chemical sensitivity (MCS), where sufferers themselves have made clear, short-term associations between health effects and low-level environmental exposures. The condition of MCS is not clearly definable and significantly overlaps with other, largely unrecognised health conditions including fibromyalgia (FMS), chronic fatigue syndrome (CFS), electro hypersensitivity syndrome (EHS) and chronic inflammatory response syndrome (CIRS). The orthodox medical diagnostic process is implicated in the production of ignorance on such health conditions.

Despite the large amount of research showing health effects from low level environmental exposures, there remains much "undone science" in the field - research that could be done but isn't. The reasons for undone science and the consequent societal ignorance are generally due to society's ingrained desire for technological improvements. Industry, responsible for technological developments the use of chemical products or radiation devices, is not interested in possible health effects, so expensive scientific research into them is left undone. When subsequent research or firsthand experiences of health effects start to be realised there is ample evidence that the industries responsible for environmental exposures then become active in generating ignorance. Due to close ties with industry, medical and health systems become complicit in industry's strategy, and knowledge is manipulated by the industry funding of scientific studies, which then influences the conclusions of the research. The support of industry products by institutions, including regulatory agencies, due to conflicts of interest also contributes to knowledge manipulation. Common industry strategies of generating ignorance also include using doubt, blame, power, industry skills, astroturfing, smear campaigns, media manipulation and fact checking services.

Future generations of children who inherit contaminants from their conception will be most affected by the gross neglect of their effect on health. The carry-through of health effects and their magnification in subsequent generations is a tragedy in the making.

ACKNOWLEDGEMENTS

I would like to thank my supervisors, Andrew Whelan and Brian Martin for their generosity with advice and guidance. Brian's weekly skype sessions, comments, support and proof reading were particularly outstanding to me and I am very grateful for.

I had started out in life quite ignorant of environmental effects on health. Then when my wife and I had children we experienced health problems with them. It was my wife Ann, together with our paediatrician that started suspecting environmental links. I learnt first-hand the value of a mother's knowledge and intuition regarding her children. In seeing their health being affected from environmental exposures it became a hard and long journey. It was a lonely battle with neither Ann's family or mine believing us, not to mention a medical system that would actively dismiss us. I am thankful to Ann who made me see and fight the subsequent battles for our children's health and 'the system'.

My driving reasons in this research and qualification have been my grandchildren. They have been born into a world that has vastly changed and become more hazardous to health than in the past, with a medical and health system under the influence of big pharma. Also they now need to deal with a government that discriminates, and does not respect privacy to an individual's health choices. The grandchildren have benefitted from our awareness through our five sons and our intelligent and objective daughter-in-laws. I dedicate this thesis to my extended family and hope it assists in their understanding of our sometimes ignorant orthodox health system and toward their thoughts and actions for their health without relying on "the system".

ABBREVIATIONS

AAAI American Academy of Allergy & Immunology
AAAS American Association for the Advancement of Science
ACC American Chemical Council
ACCME Accreditation Council for Continuing Medical Education
ACSH American Council of Science and Health
AEC US Atomic Energy Commission
ADHD Attention deficit hyperactivity disorder
ADTP 4-amino-5,6-dimethylthieno[2,3-d]pyrimidin-2(1H)-one
AHA American Historical Association
AHPRA Australian Health Practitioner Regulation Agency
ALF Atlantic Legal Foundation
ALS Amyotrophic lateral sclerosis
AMA American Medical Association
ANSES Agency for Food Environmental and Occupational Health and Safety
APL Acute Promyelocytic Leukemia
APVMA Australian Pesticides and Veterinary Medicines Authority
ASD Autism spectrum disorder
ASIA Autoimmune syndrome induced by adjuvants
ASIC Australian Securities and Exchange Commission
ATP Adenosine triphosphate
BBB Blood Brain Barrier
BMJ British Medical Journal
BPA Bisphenol A
BPF Bisphenol F
BPS Bisphenol S
CAD Consequential amazing development
CAS Chemical Abstracts Service
CCD Colony collapse disorder
CDC U.S. Centers for Disease Control and Prevention
CFS Chronic Fatigue Syndrome
CHD Children's Health Defense
CI Chemical Intolerance
CIRS Chronic Inflammatory Response Syndrome
CKDu chronic kidney disease of unknown etiology
CMA Chemical Manufacturer's Association
CME Continuing medical education
CMI Chronic Multisystem Illness
COI Conflicts of Interest
COVID SARS-CoV-2 (2019-nCoV) coronavirus
CROs Contract Research Organisations
CS Central Sensitivity
CTIA Cellular Telecommunications and Internet Association
DALY Disability adjusted life years
DDE p,p'-dichlorodiphenyldichloroethylene
DDT Dichlorodiphenyltrichloroethane
DES Diethylstilbestrol
DHPC Direct health professional communications
DOJ US Department of Justice

DTaP Diphtheria, tetanus, pertussis (vaccine)
EBM Evidenced-Based Medicine
EDCs Endocrine disrupting chemicals
EE2 17 α - Ethinyl Estradiol
EHS Electromagnetic hypersensitivity syndrome
EMF Electromagnetic Fields
EPA Environment Protection Agency (US)
ESRI Environmental Sensitivities Research Institute
FCC US Federal Communications Commission
FDA Food and Drug Administration (United States)
FM Fibromyalgia
FMS Fibromyalgia Syndrome
FOIA Freedom of Information Act
GBHs Glyphosate based herbicides
GLP Good laboratory practice
GMO Genetically Modified Organisms
GWS Gulf War Syndrome
HIV Human immunodeficiency viruses
HPV Human papillomavirus
IARC International Agency for Research on Cancer (a WHO agency)
IBS Irritable Bowel Syndrome
ICD International Classification of Diseases
ICNIRP International Commission on Non-Ionizing Radiation Protection
ICRP International Commission for Radiological Protection
IEI Idiopathic Environmental Intolerance
ILO International Labour Organisation
IPCS International Program on Chemical Safety
IPPNW International Physicians for the Prevention of Nuclear War
J&J Johnson & Johnson
LHC Lymphohematopoietic cancer
LPS Lipopolysaccharides
MRI Magnetic resonance imaging
MCS Multiple chemical sensitivity
MeHg Methyl mercury
MCS Multiple chemical sensitivity
MCRS Medical Claims Review Service
ME Myalgic Encephalomyelitis
MSA Mutualite sociale Agricole
MSH Melanocyte Stimulating Hormone
NASEM US National Academies of Sciences, Engineering and Medicine
NCPH National Council on Public History (US)
NGO Non-government organization
NHANES National Health and Nutrition Examination Survey
NHL Non-Hodgkin lymphoma
NIH National Institutes of Health (US)
NIOSH US National Institute for Occupational Safety and Health
NMDA N-Methyl-D-aspartate
NSAIDs Nonsteroidal anti-inflammatory drugs
O&G Oil and gas
OP Organophosphate
OPIDN Organophosphorous ester-induced delayed neurotoxicity

OPICN Organophosphorous ester-induced chronic neurotoxicity
OPPT US EPA Office of Pollution Prevention and Toxics
ONOO- Peroxynitrite
OTA Mycotoxin Ochratoxin A
OthV1 Otarine herpesvirus-1
PCB Para-dichlorobenzene
PFAS Per-fluoroalkyl substances
PFAS Poly-fluoroalkyl substances
PFOA Perfluorooctanoic acid
PI Pharmaceutical industry's
PP Precautionary principle
PPA Phenylpropanolamine
PR Public relations
PTSD Post-Traumatic Stress Disorder
PVC Polyvinyl chloride
RTI Research Triangle Institute
SAVN Stop the Australian Vaccination Network
SCCS European Commission Scientific Committee on Consumer Safety
SIDS Sudden infant death syndrome
SLAPP Strategic Lawsuits Against Public Participation
SLS Sodium laurel or lauryl sulfate
TGA: Australian Therapeutic Drug Authority
TILT Toxicant Induced Loss of Tolerance
UNEP United Nations Environment Programme
US CDC: US Centre for Disease Control and Prevention
USRTK U.S. Right to Know
VOCs Volatile organic chemicals
WGA Wheat germ agglutinin
WHO World Health Organisation
WTR Wireless Technology Research project

Chapter 1

Introduction

BACKGROUND

We were a typical young Australian family. There was no reason to question our modern-day lifestyle and the society in which we lived. When my wife and I encountered many health problems in two of our five young sons, we relied on, and believed in, our medical system to assist us in getting them well.

Early on when we faced these health problems, our paediatrician asked us if we had a water purifier. Within a matter of weeks of installing one, we noticed definite improvements in our boys' health, such as our third son no longer needing asthma medication. But later we encountered severe health problems facing him and another one of our boys. The two children were admitted to the Children's Hospital many times to try to get to the bottom of the situation.

Following the boys' temporary improvements after the water purifier was installed, Ann, my wife, heard through the media about blood testing for pesticides and heavy metals in the US. Since we had not pieced together the puzzle of our sons' health problems and the Children's Hospital was not coming up with anything, we decided to get a sample from one of our sons tested. I had been sceptical of pesticides and heavy metals causing health problems: I had a farming background and pesticides were generally regarded as safe, otherwise why would they be used? Our approach was more in the spirit of leaving no stone unturned.

To our surprise, the blood test results came back indicating significant levels of pesticides, solvents and heavy metals. Receiving these results just before one of our son's was admitted to the Children's Hospital in Sydney, we asked staff whether such residues could have anything to do with their problems. We were amazed with the hospital's reaction: the head toxicologist visited my wife, and simply told her she should not push such a topic any further. Ann was

staying in at the hospital with our son, and after that, the staff's treatment seemed to change. This eventually culminated in our paediatrician informing Ann that he had received a report from the hospital doctors saying that she was fictitiously reporting health problems for our sons¹.

It was amazing to find that we were being accused of inventing our two sons' poor health. After a few days of trying to come to terms with it, basically "in shock", we decided to take matters into our own hands after our medical system had let us down so badly. Following the distinct health improvements after installing a water purifier, and in light of the blood test results, we basically decided to research the chemicals found in our son who had the blood test. Basically, where did the chemicals come from and could they explain his symptoms?

While we went on this path, our two sons continued to have severe health problems at different times. One son, at 4 years of age, consistently had bloodied stools. This was very concerning. After a colonoscopy in the Children's hospital, the main diagnosis was that he was self-mutilating. This was unbelievable to us: how could a 4-year-old child be self-mutilating? We were astounded by the attitude of the hospital doctors. Was this more along the lines of their last report that Ann was making all this up? It was interesting that the three bloodied stool samples taken while our son was at the hospital had simply been lost. They had been noted by the registrar in his medical records but the samples mysteriously disappeared, and pathology had no records of them either.

This was our last-ever visit to the Children's Hospital. We knew we were on our own, that we could not return and keep receiving such accusations and handling.

We had started with full faith in the medical system, with no reason to question it. We had simply wanted our sons' health looked after. How could we be accused of making up their health problems? The drive to the Children's Hospital (from Coffs Harbour to Sydney) took us about 8 hours in those days. It was no fun for any family member. Then there was the painful

¹ Years later, we discovered this was how the hospital initiated the diagnosis of Munchausen by Proxy which we found from talking with others who had seen it first-hand. It was used by the medical system to basically silence parents, usually mothers, who questioned doctors on their diagnoses or treatment. It could potentially destroy families with Community Services then able to remove the children and the parents left to mount legal cases to clear their name and get the children back.

situation for us as parents, in seeing our sons suffer from their symptoms. How could we have “enjoyed” what we’d been through by making up our children’s problems?

A long hard journey of discoveries about our modern environment started. We had concluded that the medical system could not help us. We were lucky to have a supportive paediatrician, however. A few years down the track we discovered we were not alone in our problems. There were many other parents with similar stories and treatment by our medical “authorities”.

As we modified our family’s environment and converted to certified organic food, improvements occurred. We achieved good health in all our sons. The two boys who had been hospitalised remained chemically sensitive and always had to be conscious of exposures when away from home.

After finding the boys’ health consistently improved as we made modifications around environmental exposure, we decided to move to an area away from any agricultural pesticide vapour drift. The new house was double brick, and all carpets and insulation were removed, a separate garage, electrical wiring modified, etc. It also involved moving to an area with good air movements, about 1 km from the sea, and away from the agricultural areas of Coffs Harbour, especially the bananas. The boys’ health improved tremendously.

When our sensitive children started school, however, their health again plummeted and Ann became active in the P&C association to ensure a nontoxic school environment. Ann attended various meetings, further afield than the actual school, to make changes in schools’ general chemical usage, such as cleaning products. She eventually talked at conferences and became more prominent. The local media noticed us and our sons’ chemical sensitivities and covered our situation. Following the newspaper articles that came out, we discovered many other families that had a similar story: they contacted us.

We had solved many of our children’s health problems ourselves, simply by implementing environmental modifications. In the process, we discovered we were certainly not alone in this process and we started to gain an education in what was really happening in our society².

² Subsequent chapters cite references for the topics introduced here.

With my engineering/science background and the fact that I'm a working engineer, I am used to trying to investigate and solve problems so that systems will eventually work. If they don't work, we have to keep trying. (That's not to say that engineers are always doing the right thing, or are free from bias.) In dealing with health authorities, whilst they gave an impression of their approaches being science-based, I felt they adhered to their belief systems even when their interventions didn't work. It was a world of paradigms, undone science (areas where there were insufficient scientific studies), professional arrogance, politics and the influence of the huge chemical and pharmaceutical industries. I could see that it was the innocent families who felt the effect of such attitudes and corruption, and today the situation seems worse than it was for us all those years ago.

It must be acknowledged that if there were an emergency situation I would have full faith in orthodox medical practitioners and their system to save me. The problem seems to occur when such professionals feel they are the ultimate authority on all health issues, especially chronic health conditions. They practise what they have been taught and have spent a significant part of their life learning, to the best of their ability. These practitioners usually have full faith in the pharmaceutically based medical system they work in. Whilst their belief is that the medical system is based on a sound scientific foundation, it is clear - when anyone takes the time to sit back, to see the big picture and study a full range of scientific viewpoints - that our medical system is quite the opposite of "scientific" in many areas. The average practitioner, however, is always too busy and they may be influenced by the big pharma approach or peer pressure to never deviate from their paradigm. It also must be realised that the doctors are caught within a rigid system that demands standardised diagnostic approaches and treatment protocols. To date the Australian Health Practitioner Regulation Agency (AHPRA), has been ruthless on practitioners who deviate from established protocols. We have experience with doctors privately admitting their predicament. It also remains clear however that many doctors are rigidly within such paradigms and actively stand by and defend them.

A similar situation exists with regulatory authorities. Neoliberal approaches influence governments to cut costs, serve industry, support self-regulation and demand cost recovery. This results in regulatory capture where government departments see industry as their clients and lose sight of the fact that their main function is to protect the silent public. In Australia I have continually seen consultative committees with one or no members of the public, and

where government representatives are outnumbered by industry representatives: this has now become the accepted committee composition. If concerned members of the public start objecting, they are dismissed one by one for being too radical or uneducated. The closeness between regulatory departments and industry also has revolving doors in respect to employment.

The whole situation was very frustrating when we saw our children being affected and we tried to ask simple questions, with our original full faith in “the system”. To be treated so dreadfully, as if we were the problem came quite hard to us. It seemed to us that the scientists and medical staff within such systems are simply ruled by the system itself. I’m sure that most practitioners have never thought to question the system they are part of.

Our hard-slog learning process, where our kids had been the guinea pigs, had taken us a long way toward realising how paradigmatic our medical system and society had become. In addition to the medical doctors and nurses we tried to deal with, it could also be seen in our friends and extended family. Because we started to get consumed by what we were finding and how we were modifying our family environment - real life struggles - it was hard to talk to our friends about the weather when they had full faith in the orthodox medical system. As a result, we dropped-out of our local social circles. The extended family/friends thought we had become hippies and regarded our problems as just restricted to our family. Through our hard-won experiences, we saw that many of the health problems occurring in our extended family and friends could be explained by environmental factors. Although we tried talking about such situations, it was clear that we were now outside the paradigm. It was difficult, but we gradually learnt to say nothing about what we thought on significant matters, just to avoid social isolation.

Now that the children have grown up and are leading regular lives, although remaining continually-aware of environmental influences on their health, it has come time to systematically investigate the science and causes of our seemingly out-of-touch medical system. Hence this thesis.

THE ISSUES

Modern economies are dependent on commodification. A high standard of living is achieved through commodities such as houses, properties, cars, highways, salaries, luxury items, schools, energy, etc. There is, however, a consequence of these commodities that are considered important. In this paradigm, aspects that cannot be measured or sold such as, clean water, air and safe food are considered to have no value. The environment consequently becomes filled with substances produced in extracting, making, using, burning or discarding the many marvellous products on which society now depends. It is pertinent to find many observations noting that when a country develops an industrial economy, an epidemiological shift from infectious to degenerative diseases occurs. Commodification and industrialisation contribute to the sacrifice of a healthy environment. Generally, as seen in this thesis, the increase in chronic diseases can be linked to the lack of a healthy environment. Instead of being a health issue, pollution is regarded as something that affects our “external” environment only, not public health. “As a society we need to regain a focus on pollution as a threat to human health,” stated the UK chief medical officer (Davies 2017p4).³

Many examples in this thesis show that the government will almost always side with industry viewpoints and statements in matters around chemical poisoning and its regulation. This is generally substantiated by seeing these problems as the price of economic progress. Furthermore, that each issue considered on its own is insignificant. These government attitudes and its inaction on these issues contribute to the increasing burden of health problems and premature deaths in our society. The increase in healthcare costs in turn undermines economic growth and demands tax increases to pay for it. The medical system’s ignorance of the mounting evidence of environmental influences on health does not help the situation: they remain firmly in the grip of the pharmaceutical industry’s approach, sometimes using quite toxic chemicals to simply treat the symptoms.

Certain comments in a recent United Nations report are pertinent: “Urgent action needed to tackle chemical pollution as global production is set to double by 2030.” The report goes on:

³ References are given at the end of each chapter. The citation system used in this thesis will list the surname of the first author only, then the year published, and where the text is quoted it will include the page number as in the above citation (p4).

The benefits of action to minimize adverse impacts have been estimated in the high tens of billions of United States dollars annually. The World Health Organization estimated the burden of disease from selected chemicals at 1.6 million lives in 2016 (this is likely to be an underestimate). (UNEP 2019pvi)

Yet industrialised products, such as those produced by the chemical industry, continue to increase in volume worldwide.

The size of the global chemical industry exceeded United States dollars 5 trillion in 2017. It is projected to double by 2030. Consumption and production are rapidly increasing in emerging economies. (UNEP 2019p3).

The traditional attitude to chemical applications, spills and accidents is that they are diluted, absorbed and broken down by the soil, atmosphere and oceans. But the earth's environment is not a limitless sink for such pollution. Our finite planet has now gone past saturation point and background pollutant levels are now detectable and increasing. The most well-known pollutants are the increasing concentrations of greenhouse gases, however they are only the tip of the iceberg. More than 250 billion tonnes of fossil fuels, carbon, topsoil, hazardous wastes, minerals, material and mineral wastes enter the environment each year. There is now no part of the earth where toxic chemical residues are not found. There are polychlorinated biphenyls (PCBs) in animals 10,000 metres deep in ocean sediment; the fresh snow at the peak of Mt Everest is now too polluted to drink; organochlorine pesticides have been detected in the Himalayan glaciers; and various toxic residues are found from remote Pacific atolls to the Arctic and the Antarctic.

As a result of environmental pollution, toxic chemicals are routinely found in birds, fish, whales, squid, etc. The animals and plants we eat also have these background toxin levels, biomagnified in the animal groups higher up the food chain. But there are many variants of environmental pollution, not only in molecular form. There are increasing amounts of non-ionising radiation, from wi-fi through to the new 5G and 6G systems, and further increases from the intended grid of 10,000 plus satellites: this is happening virtually in one generation, when human evolution of millions of years has never encountered such exposures before. Background levels of ionising radiation have also been increasing; this is produced even by coal-fired power stations, without considering the various nuclear disasters at nuclear facilities and their long-lived radioactive

wastes. Due to the breakdown of plastics and particulates from internal combustion engines and other sources, the resultant nanoparticles are inhaled and consumed by humans. Such nanoparticles are small enough to enter human blood streams and become deposited in various organs.

There are, as well, a variety of toxins which have been intentionally added to our food. The agricultural paradigm of increasing yields without any concern for food quality (in respect to vitamins and minerals) can see a variety of chemical residues and genetically modified (GM) products present in food. Further, the increased consumption of processed foods sees the extensive use of chemical additives such as food glues, emulsifiers, preservatives, colourings, flavourings and bulking agents.

In the home, most products are synthetic, e.g., paint, carpets, furniture, laminates, particle board products, window blinds, non-stick and aluminium cookware, etc. Even the bedding we sleep in is synthetic: mattresses (especially fire resistant treated), pillows, etc. Our clothing now has a high synthetic content as well as coatings for non-creasing or water resistant properties. Cleaning products, such as detergents, anti-bacterial wipes and deodorisers, can be quite toxic. Such products are rarely stable, with plasticisers, solvents and other breakdown products entering the air we breathe, the food we eat, and our skin that it comes into contact with. The public's ignorance on this subject is consistently illustrated in the use of direct chemical exposures for pleasure, such as smoking, spraying houses with pesticides, use of toxic cosmetics, continuous use of wi-fi, anti-bacterial cleaners, etc. Synthetic chemicals are part of our daily lives. They protect, adorn, solve problems, kill pests, improve efficiency, save lives and provide convenience. It is almost unthinkable to be without such chemicals, which are a part of "who we are" in many more ways than most of the population suspects.

Environmental chemical exposures are ubiquitous, usually invisible and have no sudden clear health effects that would prompt health-effect connections being made. The public is largely unaware of the situation (which some regard as humanity's greatest effect on itself), let-alone its worsening status (Cribb 2021). There have been well-publicised but isolated catastrophes such as the cases of Minamata, Love Canal, Seveso, Bhopal, Fukushima and the Indian school-lunch tragedy. These, however, have not been understood as indicators of the general situation but have been regarded as isolated, distant, unfortunate co-incidences.

The 2018 report of the Lancet Commission on Pollution and Health identified chemical pollution as having a significant effect on human health.

Pollution is the largest environmental cause of disease and premature death in the world today. Diseases caused by pollution were responsible for an estimated 9 million premature deaths in 2015—16% of all deaths worldwide—three times more deaths than from AIDS, tuberculosis, and malaria combined and 15 times more than from all wars and other forms of violence. In the most severely affected countries, pollution-related disease is responsible for more than one death in four (Landrigan 2018p462).

Some countries now do sample population testing for industrial “chemicals of concern”. In the US about 300 to 400 chemicals are tested for and are routinely detected. This includes dioxins and furans in breast milk, phthalates in urine, and heavy metals in human blood. Previously banned flame retardants have been found in the umbilical-cord blood of newborns. This shows one pathway of many for the transfer of such toxic substances to new generation. It is typical of bio-accumulative and persistent substances. These test results show that the modern society population now carries lifelong chemical burdens starting from when they were a foetus. Studies in mice on just one of these chemical residues, glyphosate, show that after one generation is exposed then detrimental health effects continue and increase in subsequent generations even with no further exposure after the first generation (Kubsad 2019, Milesi 2021).

In a recent report on endocrine disrupting chemicals, the World Health Organization (WHO) and United Nations Environment Programme (UNEP) warned that reproductive and other hormonal disorders are on the rise globally, that human-made substances are increasingly implicated as the cause according to laboratory studies, and that the scale of the problem is probably underestimated (WHO 2013). Falling sperm counts in males, reduced fertility in females, genital deformities and changes in male/female gender are now all linked to endocrine-disrupting chemicals.

The regulation of industrial chemical products has achieved the banning of about 19 of the approximately 144,000 industrial chemicals in a handful of countries. Clearly, the regulatory system is ineffective. While pesticides and pharmaceutical drugs have some premarket testing requirements whose shortcomings are highlighted in chapter 4, most industrial chemicals,

about 80-90% of all chemicals, do not have any premarket testing (Cranor 2014, Guth 2007, Claxton 2010). Furthermore, while some chemical products are either banned or quietly withdrawn in light of negative health effects, the replacement chemicals may be similarly toxic. For example, the diethylstilbestrol (DES) hormone was replaced by new synthetic hormones, the “bisphenol A (BPA) free” products are being replaced by bisphenol S (BPS) products with initial evidence indicating these may be as bad, if not worse for human health (Gu 2019).

Most of these chemicals have unknown individual effects, let alone their combined effects. Yet each year at least 1000 new chemicals are released onto world markets. This represents an uncontrolled, unmonitored, unregulated and unconscionable mass release of possible toxins.

There are many examples in recent events where thousands of people’s lives have been detrimentally affected due to the medical system’s inaction on toxicant exposures (Whaley 2013). In most of these cases, strong political/ industrial action suppressed proper scientific debate. Among the many examples, were tobacco smoke, asbestos, lead dust, benzene, polychlorinated biphenyls, chlorofluorocarbons, phthalates, organochlorines, and dioxins. For each substance, warning observations were ignored decades before its emergence as a devastating public health issue (Bijlsma 2016, Harremoës 2001).

With the now-common situation of low-level chemical exposures in everyday life, there are no sudden health effects. Instead, there are latency times between exposures and effects. This is well established: for example, in the case of brain cancer latency times are a statistical distribution. Typical brain-cancer latency times are considerably longer than 10 years. An Israeli study of children (mean age 7.1 years, range < 1–15 years) whose heads were X-rayed to cure tinea capitis were found to have increased risk of brain cancer. Of the children, 26% were diagnosed < 20 years, totalling 45% up to 30 years later and the remaining 55% were diagnosed between 30 and 40 years after their exposure (Sadetzki 2005). In a study of Hiroshima and Nagasaki A-bomb survivors, gliomas (a type of tumour that occurs in the brain and spinal cord) occurred 14 to 51 years after exposure (Preston 2002).

While very rarely seen in the past, cancers are now appearing in animals. One example has been documented in the relatively long-lived sea lions (20-30 years) on American’s west coast. Longer lifetimes magnify bioaccumulation of chemicals and cancer has been documented in 25% of California sea lions (Gulland 2020). The sea lions are mostly suffering from urogenital

carcinoma previously associated with organochlorines such as polychlorinated biphenyls (PCBs) and dichlorodiphenyltrichloroethanes (DDTs). Dumping of persistent organic pollutants occurred off the California coast prior to their ban (Kivenson 2019). In the sea lions, cancer risk was found to be 43.6 times higher in those infected with otariine herpesvirus-1 (OtHV1). Synergism between the pollutants and OtHV1 infection seems to be driving up the sea lions' cancer rates. This reflects serious implications for humans but is an area of undone science: simply not studied.

This thesis will make connections with environmental exposures and the rise of chronic diseases in the last 50 to 80 years with many occurring in epidemic proportions (such as allergies and autoimmune diseases). Diabetes, cardiovascular disease and cancer are attributed to genetic causes by orthodox medicine, yet their rates have exploded over the last half century - which is not possible if the cause is genetic. Since changes to the human genome occur over much longer periods (thousands of years), genetic susceptibility can only contribute to this phenomenon through epigenetics. This has been reflected in thousands of studies, some of which will be referred to in this thesis, e.g., Vojdani (2020).

We have recently seen extreme regulatory responses enacted by government health authorities, to ensure public health when facing one particular corona virus. The virus is invisible, like most environmental exposures and must also be tested for using involved technical methods. Approximately 20% of the general population was susceptible to COVID-19, which had a relatively low incidence and death rate compared to many other health conditions, such as combinations of influenza with a heart condition, etc. Yet for environmental toxic exposures affecting the whole population, there is no concern about pesticides, antibiotics and many additives in the food we eat. Most of these have not undergone any form of safety testing. Food has consequently become a form of environmental exposure. Consumption of organic food has been shown to significantly reduce body pesticide levels (Fagan 2020, Hyland 2019). This raises notions of social responsibility for the future health of bodies and populations. The lack of governmental stewardship of the food environment (Landecker 2011) contrasts markedly with COVID-19 responses.

The WHO and UNEP have estimated that one in 12 people die from environmental toxins and about 86 million are maimed each year.

“An estimated 12.6 million people died as a result of living or working in an unhealthy environment in 2012 – nearly one in four of total global deaths, according to new estimates from WHO. Environmental risk factors, such as air, water and soil pollution, chemical exposures, climate change, and ultraviolet radiation, contribute to more than 100 diseases and injuries” (WHO 2016). The portion of the population affected is greater than for COVID-19, HIV, malaria or car crashes. One in five cancers worldwide are attributable to exposure to environmental risks (Gatto 2021). Some scientists have warned of a 'silent pandemic' of childhood brain damage caused by the global release of neurotoxins through human activity. Where is the government action to reduce exposures for these pandemics/health conditions affecting a greater percentage of the population?

Over the past 40 years, there has been a significant increase in a variety of endocrine-associated diseases including, infertility, premature puberty, attention deficit hyperactivity disorder (ADHD), obesity and diabetes, and endocrine cancers such as prostate, ovarian and breast cancer. It is biologically plausible that endocrine disrupting chemicals (EDCs) are playing a significant role in these and other diseases (Schug 2011). Many substances cause endocrine disruption, including pharmaceuticals, dioxin and dioxin-like compounds, polychlorinated biphenyls, DDT and other pesticides, and plasticisers such as bisphenol A. EDCs can be found in many everyday products including plastic bottles, metal food cans, detergents, flame retardants, food, toys, cosmetics, and pesticides (Skinner 2011, Whitelaw 2008).

In 1996, the US Congress passed the *Food Quality Protection Act* (which amended the *Federal Insecticide, Fungicide and Rodenticide Act*) and an amendment to the *Safe Drinking Water Act*. Both amendments directed the US Environment Protection Agency (EPA) to establish a testing and screening program on endocrine disrupting chemicals (Vogel 2004). Twenty-six years later this has hardly been started.

With less than 1 percent of chemicals on the market today ever having had safety studies done, children effectively become “guinea pigs in an uncontrolled experiment” (Lautenberg 2010). While there are no specific safety tests done for children on the thousands of chemical products available, there are also no studies defining safe levels of exposure for children:

the impact of various exposures, whether individual, simultaneous, sequential, or cumulative over a lifetime, may not be simply additive. Instead, combinations of

exposures may have synergistic effects that intensify or otherwise alter their impact compared with the effect of each contaminant alone. In addition, we now recognize that critical periods of time exist across the life span (e.g., prenatal and early life, puberty) when individuals are particularly susceptible to damage from environmental contaminants. Moreover, a person's genetic make-up can significantly affect his or her susceptibility to the harmful effects of an environmental agent, and it also is becoming clear that some exposures can have effects across multiple generations. (Reuben 2010p2)

Environmental toxins/industrial chemicals are carried by parents even before they decide to have a child. Women's eggs and men's sperm, the very genetic sources of children, may have already been affected or be in contact with such toxins. National biomonitoring studies such as those done by the Centers for Disease Control and Prevention (CDC) in the United States show clearly that everyone now carries significant levels of xenochemicals (chemicals foreign to the body). It is no surprise that these chemicals can be transferred into the next generation: it is now well-known that umbilical cord blood contains many such chemicals (Cao 2018, Leung 2018). As well, "it is clearly evident that there really is no placental barrier per se: the vast majority of chemicals given [to] the pregnant animal (or woman) reach the foetus in significant concentrations soon after administration" (Schardein 2000). Yet it is generally not realised that the next generation starts out with such chemical burdens at a time when the foetus cannot handle such a burden. Children may now start accumulating persistent xenochemicals from near levels that may have taken their parents decades to bioaccumulate. "Each time we look for the latest chemical of concern in infant cord blood, we find it" (EWG 2009a,b). This is similar to researching xenochemicals in breast milk: in breastfeeding the mother's body burden of industrial chemicals is transferred to the child (Heinzow 2009). In both instances this may mean that the foetus/newborn is exposed to higher doses than adults relative to body weight (Grandjean 2008). For example, methylmercury was found to be five times more concentrated in one foetal brain than in its mother's blood (Honda 2006). It has been estimated that a nursing child's daily PCB dose may be up to 100 times the mother's blood levels (Grandjean 2008). Although the original persistent chemicals (such as the organochlorines) are still found, there are now more new chemicals appearing: the supposedly non-persistent ones (Croes 2012, Luzardo 2013, Weldon 2011).

There are now books which highlight the fact that:

In western countries, sperm counts and men's testosterone levels have declined dramatically over the last four decades ... also, increasing numbers of girls are experiencing early puberty, and grown women are losing good quality eggs at younger ages than expected: they are also suffering more miscarriages. It's no longer business as usual when it comes to human reproduction. (Swan 2020p1).

As seen from the above, there already seems to be significant documented awareness of environmental effects on health, so why then is it being ignored by health and medical authorities?

In short, poisoning by human-made chemicals has become the biggest preventable healthcare issue of our time - and - is due to grow bigger still (Cribb 2014p158).

THE CENTRAL THESIS

The central thesis of this research is that:

Environmental exposures can have significant effects on people's health but industry, via undone science and the generation of ignorance, suppresses much potential knowledge about these effects. The orthodox medical system and government contribute to, or do little to oppose, this process.

Environmental effects on health represent a broad area of study. It is not in the scope of this research to define or analyse all environmental influences, due to the immensity of the task. The focus will be on the area of chemical and radiation exposures from industrial and commercial products. Examples of environmental exposures will be used to show effects on human health which have been studied without industry funding or influence. These show that environmental exposures are an important consideration in the causation of human health problems.

There is usually limited knowledge about disease etiology in respect to original causal mechanisms. For example, leukaemia can start when bone marrow begins to show certain characteristics. The usual approach to etiology starts at this point and is acceptable as an "explanation" in orthodox medical circles. In this thesis, however, I am more interested in what had caused or triggered the bone marrow to commence showing those unusual characteristics. As will be seen, the modern living environment subjects everyone to millions of non-natural, manufactured chemicals (xenochemicals). Although many of these exposures are at what the medical orthodoxy regards as low levels, they can still have impacts. Continual exposures and bioaccumulation over long periods of time can magnify these health effects. This presents a complication for scientific studies in that there can be no such thing as a control group experiencing zero environmental risk, or a lab test that can study the myriad of real-life exposures and stressors.

This research will not attempt to address detailed conceptual and measurement difficulties in defining diseases, but rather to highlight paradigmatic resistance to health conditions having clear environmental causes from within the medical and health system. It is frequently found

that there is a gap between what public health officials say about some health conditions and what is found in scientific journals, for example, in the case of autism (Rogers 2019).

This thesis will illustrate many health effects of low-level xenochemical exposure from the environment. Most chronic diseases that are increasing year after year in developed countries can be explained as having causal links with various xenochemicals. The complicit medical system, influenced by the manufactured ignorance produced by the pharmaceutical/chemical industry, simply becomes a tool to promote public ignorance of these issues. Yet even those who are aware of the situation will mostly choose to selectively ignore it.

Although some environmental exposures are clearly chosen by the individual, such as diet, alcohol, smoking, recreational drugs, etc., these will not be considered in this thesis. A clear focus will be on environmental exposures that the public, in general, is either ignorant of or has no choice as to whether they want to be exposed.

In presenting material in support of the general thesis stated above, I will not be undertaking specific analyses of events or occasions where disjunctive views come into contact, or how critical views in support of multiple chemical sensitivity (MCS) sufferers were overruled by establishment views. This has been well-covered by past literature. I will also not provide a detailed description of how medical paradigms have changed over time. The orthodox medical system has had the same paradigmatic approach to chemical sensitivities for the last century, as promoted by industry. It is this paradigm only which is explored in this thesis.

If I analysed one or two central showpiece examples in detail, that would be valuable but it would not cover the big picture that I aim to address. I do not aim to analyse the mechanics of specific examples as to how industry creates ignorance about whether and how a chemical X produces health effects Y and Z. Rather, I present a range of examples showing extensive areas of ignorance and put that in the context of the methods used to maintain or create ignorance. Rather than relying on extrapolation from a small number of detailed studies, I endeavour to illustrate the extensive evidence suggesting widespread suppression of knowledge about environmental impacts on health.

OVERVIEW OF CHAPTERS

Chapters 2 and 3 aim to provide detailed case material in support of my central thesis. They provide the empirical background for the commentary on ignorance and manipulating knowledge that appears in chapters 4 and 5. This way of organising the material avoids a very long discussion of theory before case material is presented. Because the subject is broad, this approach avoids very long analysis and discussion chapters. A different order of chapters could be possible, for example starting with theory then giving the case studies, and the chapters can be read in a different order if desired.

It has long been acknowledged that environmental factors were a causal factor for diseases. The definition of “environmental” used to define these factors was a very tight, conservative one so was likely underestimated. Even so, more than two decades ago estimates of 25% to 33% of the worldwide burden of disease was so-attributed (Smith 1999).

Scientific studies can be financed or sponsored, directly, indirectly or influentially by large companies responsible for the myriad manufactured or synthetic substances now ubiquitous in our world. The usual approach is for a company to sponsor or influence studies where there is good potential for positive results for their product. This could be a pharmaceutical company looking to obtain study results that show no health side effects from their drug or vaccine, or the same for a pesticide-manufacturing or telecommunications company considering their product’s use in society. This aspect will be discussed in more detail in chapter 4, but the financial influence is recognised throughout this thesis and the various companies are referred to summarily as “industry”.

Industry can also confuse the public by referring to substances as “environmental factors” as if those substances were naturally occurring. A patient with a chemically caused disease who is told it is due to “environmental factors” may think the cause was natural rather than being preventable or avoidable. So the term itself generates a great deal of public ignorance on the subject.

My own background has provided hard-learned lessons in seeing environmental effects on the health of my family members over the last three decades, especially those who were most sensitive. These experiences prompted my approach to firstly search for the published research

on environmental effects. This is done in the chapter 2, which looks at the available research on the effects on human health of low concentrations of xenochemicals in the environment. It considers whether common health problems found in the population can be attributed to such chemical exposures. A comprehensive analysis or presentation of evidence is not needed to illustrate these aspects. Numerous and varied scientific studies and some major health conditions will be briefly considered. From the examples referenced and the opinions expressed by various authorities, it will be shown that there is considerable evidence showing links between environmental exposures and human health conditions, especially chronic diseases. Using published studies chapter 2 will show that there are important areas that have been ignored by the orthodox medical system. This will be by referencing studies of low concentrations of environmental toxin exposures on animals and humans which have been shown to be linked with various human health conditions. The diversity and quantity of studies cited illustrates that the effects are broad and not limited to a few health areas. This research shows that there are a great many studies of environmental effects demonstrating that it's a serious issue that deserves more attention.

However, some studies on environmental exposures find negligible health effects from particular industrial-product exposures. These studies are typically sponsored directly or indirectly by industry. Since it is popular in medical science to utilise meta-analyses and evidence-based-medicine approaches, when all relevant studies are considered together the environmental effects on health become less clear. This enables industry to continue with its activity and products due to the generated uncertainty.

Chapter 2 will point out how the orthodox medical and health system is unable to explain the causes of most chronic health conditions. The system is rather pre-occupied with quickly deriving a diagnosis, then treating the symptoms. Possible environmental causes are rarely pursued. In enquiring why this situation exists, this research found that many of the methods of creating ignorance apply to environmental exposures. These methods are then pursued to explain the current situation.

The word "orthodox" will be used frequently through this thesis and is referring to government health authorities and the medical system from AHPRA through to the doctors who rigidly work within such regulated paradigms. It also refers to medical doctor associations which work

unquestioning with these authorities. This term is also used in referring to doctors who are under the influence of the pharmaceutical industry for treating the symptoms of disease rather than to attention the causes.

In chapter 3 one illness, multiple chemical sensitivity (MCS), unrecognised by the orthodox medical system is considered. Sufferers from this illness commonly make connections between environmental exposures and health effects. By avoiding known environmental exposures they can avoid health problems. These exposures may be at low levels which have no noticeable health effects on other people. The sensitised nature to environmental exposures for this portion of the population makes this condition very relevant to this thesis. The non-recognition of this condition by the orthodox medical system becomes important to understand and is pursued in this chapter.

Chapter 3 illustrates how many environmentally triggered health conditions have significant overlaps: these health conditions are not clearly delineated. A significant percentage of MCS sufferers can also exhibit symptoms typical of fibromyalgia syndrome (FMS), chronic fatigue syndrome (CFS), chronic inflammatory response syndrome (CIRS), central sensitisation, electromagnetic hypersensitivity syndrome (EHS), and schizophrenia. Yet most studies investigate just one of these conditions, resulting in siloed research situations. This seems to be typical of environmental effects due to their immensity and diversity. This siloed situation was also seen in studies concerning declining bee populations: some studies considered pesticides as causes (referred to in chapter 4) and others considered only electromagnetic radiation.

Rather than becoming the canary in the coal mine for environmental effects on health, MCS, which affects 13% to 27% of the population (discussed in chapter 4), is generally dismissed by the orthodoxy in line with the many industry-sponsored studies that label the condition as psychological. This enables the status quo to be maintained by industry, without affecting financial bottom lines. It will be shown in chapter 3 how the medical profession avoids research into environmental causes of MCS by blaming sufferers for their condition. This is usually done by attributing MCS symptoms to a psychological problem or one's genetic make-up. Other similar examples will be discussed, such as diabetes and obesity, to further illustrate the attribution of blame to the patient.

In addition, the psychological conditions of depression and ADHD will be compared with MCS. Depression, a diagnosis commonly applied to MCS sufferers, itself has strong environmental triggers. ADHD has been the subject of fewer scientific studies than MCS, yet ADHD is readily accepted by the orthodoxy and MCS is not.

Chapter 3 also shows that the medical system's popular basis for establishing diagnostic guidelines, evidence-based medicine (EBM), has stifled the ability of doctors to apply their own observations and experience to cases. Furthermore, it will be discussed how EBM is so readily corruptible by industry.

The paradigmatic aspects preventing the recognition of a range of emergent diseases in general will also be considered through the example of MCS. The orthodox medical system has moved from the belief systems of centuries-past to a supposed scientific basis which uses strong positivist approaches. Yet these positivist approaches become a hindrance to change because of new illnesses' complexity and tendency to overlap with other emergent diseases (such as MCS). An example illustrating how outspoken representatives of the orthodox medical system dismiss chemical sensitivities is demonstrated by a case related to aircraft cabin fumes. Some expert comments from Dr Loblay are outlined to illustrate this without detailing the mechanics of the case. Dr Loblay at the time was the Director of the Allergy Unit at the Royal Prince Alfred Hospital and Senior Lecturer in Immunology, Department of Medicine, University of Sydney.

Given so much evidence, so many published studies and such urgency around environmental effects on health, why aren't orthodox doctors and the health and regulatory systems recognising and acting on this issue? To explain the situation, it is useful to turn to agnotology, the study of ignorance. This is begun in chapter 4 where the various forms of ignorance are first introduced. Through coverage of undone science and ignorance, this chapter will illustrate that the current situation can be explained by the active production of ignorance by industry and government.

In ignorance studies, undone science is sometimes treated as known unknowns, although in researching areas of undone science, nescience (knowledge we discover as a surprise) can sometimes result (Gross 2010). It will be seen in this thesis that undone science is generally desired to be researched by public interest groups and industry is not prepared to fund such

research. This would typically be where a public interest group wants studies to be done on particular health effects from environmental exposure situations to a certain chemical product.

Doubt, alongside the manufacture of ignorance, is commonly used to downplay published studies that run counter to industry's interests. The credibility of detrimental product study results is undermined through government committees, think tanks, and all types of media – including scientific journals. Even for environmental exposure crises, there are common ways that industry avoids responsibility. The main approach is to blame the victims. In the case of lead, blame ranged from the children's bad behaviour through to the parents' lack of discipline, lack of moral values and even their being single parents (Warren 2000). Asbestos mine workers with lung problems were blamed for smoking or having an unhealthy lifestyle (Schneider 2004). After health warning labels were put on cigarette packets, blame was put on smokers for ignoring the warnings (Michaels 2008). Crises can last for decades before any action is taken, commonly via legal channels.

Chapter 5 on the manipulation of knowledge, follows on from the chapter on the deliberate creation of ignorance. It considers how knowledge can be manipulated and distorted by industry and government entities. The importance of generating ignorance and manipulating knowledge is obviously in industry's financial interests, but it is also important for governments. Chapter 5 considers this aspect and sets out the reasons why, along with corruption, governments are so complicit in this situation.

The thesis concludes that there are ample studies and first-hand cases, such as MCS, which show the many ways that environmental exposures can affect health. There could however be more science done to achieve a clearer understanding. This undone science has been both generated and utilised by industry in its active production of ignorance, due to its sole focus on financial interests to generate the sales of industrial products which are the source of environmental exposures. Industry's influence on the orthodox medical and health care systems and government agencies allows the current situation to continue.

REFERENCES

Bijlsma N, Cohen M, Environmental chemical assessment in clinical practice: unveiling the elephant in the room, *Int J Env Res & Pub Hth*, 2 Feb 2016.

Cao W, Liu X, Liu X, Zhou Y, Zhang X, Tian H, Wang J, Feng S, Wu Y, Bhatti P, Wen S, Perfluoroalkyl substances in umbilical cord serum and gestational and postnatal growth in a Chinese birth cohort, *Env int*, 2018, 116, 197-205.

Claxton L, Umbuzeiro G, DeMarini D, The salmonella mutagenicity assay: the stethoscope of genetic toxicology for the 21st century, *Env Health Persp*, 2010, 118, 1515–22.

Cranor C, Chapter 9, Reckless laws, contaminated people: science reveals legal shortcomings in public health protections, In: *Powerless Science?*, Science and Politics in a Toxic World, Ed. Jas B, Berghahn Books, 2014, 195-214

Cribb J, *Poisoned planet: how constant exposure to man-made chemicals is putting your life at risk*, Allen & Unwin, Sydney, 2014.

Cribb J, *Earth detox: how and why we must clean up our planet*, Cambridge University Press, UK, 2021.

Croes K, Colles A, Koppen G, Govarts E, Bruckers L, Van de Mieroop E, Nelen V, Covaci A, Dirtu AC, Thomsen C, Haug L, Persistent organic pollutants (POPs) in human milk: a biomonitoring study in rural areas of Flanders (Belgium), *Chemosphere*, 2012, 89, 8, 988-94.

Davies S, Annual Report of the Chief Medical Officer 2017, Health impacts of all pollution – what do we know? Chapter 1, Department of Health and Social Care, UK
https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/690846/CMO_Annual_Report_2017_Health_Impacts_of_All_Pollution_what_do_we_know.pdf

EWG: Environmental Working Group, Toxic chemicals found in minority cord blood, Press Release, 2 Dec 2009a <https://www.ewg.org/news/news-releases/2009/12/02/toxic-chemicals-found-minority-cord-blood> accessed 6 Mar 2019.

EWG: Environmental Working Group. 2009b. Pollution in People: Cord Blood Contaminants in Minority Newborns, https://static.ewg.org/reports/2009/minority_cord_blood/2009-Minority-Cord-Blood-Report.pdf?_ga=2.119449289.1788180067.1544876684-1840960985.1544876684 accessed 15 Dec 2018.

Fagan J, Bohlen L, Patton S, Klein K, Organic diet intervention significantly reduces urinary glyphosate levels in US children and adults, *Env research*, 2020, 189, 109898.

Frickel S, Gibbon S, Howard J, Kempner J, Ottinger G, Hess D, Undone Science: Social movement challenges to dominant scientific practice, *Science Technology and Human Values* 2010, 35, 4, 444–73.

Gatto N, Environmental carcinogens and cancer risk, *Cancers (Basel)*, 2021, 13, 4, 622..

Grandjean P, Bellinger D, Bergman A, Cordier S, Davey-Smith G, Eskenazi B, Gee D, Gray K, Hanson M, Hazel V, Heindel J, Heinzow B, Hertz-Picciotto I, et al., The faroes statement: human health effects of developmental exposure to chemicals in our environment, *Basic and Clinical Pharma & Tox*, 2008, 102, 73–75.

Gross M, *Ignorance and Surprise: Science, Society, and Ecological Design*, Cambridge, MA, MIT Press, 2010.

Gu J, Zhang J, Chen Y, Wang H, Guo M, Wang L, Wang Z, Wu S, Shi L, Gu A, Ji G, Neurobehavioral effects of bisphenol S exposure in early life stages of zebrafish larvae (*Danio rerio*), *Chemosphere*, 2019, 217, 629-35.

Gulland F, Hall A, Ylitalo G, Colegrove K, Norris T, Duignan P, Halaska B, Acevedo Whitehouse K, Lowenstine L, Deming A, Rowles T, Persistent contaminants and herpesvirus othv1 are positively associated with cancer in wild california sea lions (*zalophus californianus*), *Frontiers in Marine Sc*, 2020, 7, 1093.

Guth J, Denison R, Sass J, Require Comprehensive Safety Data for All Chemicals, *New Solutions*, 2007, 17, 233–58.

Harremoës P, Gee D, MacGarvin M, Stirling A, Keys J, Wynne B, Vaz S, Late lessons from early warnings: the precautionary principle 1896–2000, Office for Official Publications of the European Communities: Luxembourg City, Luxembourg, 2001.

Heinzow B, Endocrine disruptors in human breast milk and the health-related issues of breastfeeding. In: *Endocrine-Disrupting Chemicals in Food*, Ed. Shaw I, 322–55. Cambridge: Woodhead Publishing, 2009.

Honda S, Hylander L, Sakamoto M, Recent advances in evaluation of health effects on mercury with special reference to methylmercury—a minireview, *Env Health Prev Med*, 2006, 11, 171–76.

Hyland C, Bradman A, Gerona R, Patton S, Zakharevich I, Gunier R, Klein K, Organic diet intervention significantly reduces urinary pesticide levels in US children and adults, *Env research*, 2019, 171, 568-75.

Kempner J, Perlis C, Merz J, Forbidden Knowledge, *Science*, 2005, 307, 854.

Kivenson V, Lemkau K, Pizarro O, Yoerger D, Kaiser C, Nelson R, Carmichael C, Paul B, Reddy C, Valentine D, Ocean dumping of containerized DDT waste was a sloppy process, *Env science & tech*, 2019, 53, 6, 2971-80.

Kubsad D, Nilsson E, King S, Sadler-Riggelman I, Beck D, Skinner M, Assessment of glyphosate induced epigenetic transgenerational inheritance of pathologies and sperm epimutations: generational toxicology, *Sci Rep*, 2019, 9, 6372

Landecker H, Food as exposure: Nutritional epigenetics and the new metabolism, *Bio Societies*, 2011, 6, 2, 167-94.

Landrigan P, Fuller R, Acosta N, Adeyi O, Arnold R, Baldé A, Bertollini R, Bose-O'Reilly S, Boufford J, Breyse P, Chiles T, The Lancet Commission on pollution and health, *Lancet*, 2018, 391, 10119, 462-512.

Lautenberg F, Hearing of the senate subcommittee on superfund, toxics and environmental health, In: *Everyday chemicals may be harming kids, panel told*, CNN, 26 Oct 2010 [Everyday chemicals may be harming kids, panel told - CNN.com](https://www.cnn.com/2010/10/26/health/chemicals/index.html) accessed 1 Jun 21.

Leung Y, Ouyang B, Niu L, Xie C, Ying J, Medvedovic M, Chen A, Weihe P, Valvi D, Grandjean P, Ho S, Identification of sex-specific DNA methylation changes driven by specific chemicals in cord blood in a Faroese birth cohort, *Epigenetics*, 2018, 13, 3, 290-300.

Luzardo O, Ruiz-Suárez N, Almeida-González M, Henríquez-Hernández L, Zumbado M, Boada L, Multi-residue method for the determination of 57 persistent organic pollutants in human milk and colostrum using a QuEChERS-based extraction procedure, *Analytical and bioanalytical chemistry*, 2013, 405, 29, 9523-36.

Michaels D, *Manufactured uncertainty: Contested science and the protection of the public's health an environment*, In: *Agnotology, The making and unmaking of ignorance*, Stanford University Press, 2008, Chapter 4, 90-107.

Milesi M, Lorenz V, Durando M, Rossetti M, Varayoud J, Glyphosate herbicide: reproductive outcomes and multigenerational effects, *Frontiers in endocrinology*, 2021, 12, 672532.

<https://doi.org/10.3389/fendo.2021.672532>

Preston D, Ron E, Yonehara S, Kobuke T, Fujii H, Kishikawa M, Tokunaga M, Tokuoka S, Mabuchi K, Tumors of the nervous system and pituitary gland associated with atomic bomb radiation exposure, *J. Natl. Cancer Inst*, 2002, 94, 1555–63.

Reuben S, Reducing environmental cancer risk: what we can do now, 2008–2009 Annual Report for The President’s Cancer Panel April 2010 [Reducing Environmental Cancer Risk: What We Can Do Now \(nih.gov\)](#) accessed 1 Jun 21.

Rogers T, The Political Economy of Autism, PhD Thesis, Department of Political Economy, School of Social and Political Sciences, Faculty of Arts and Social Sciences, University of Sydney, 2019.

Sadetzki S, Chetrit A, Freedman L, Stovall M, Modan B, Novikov I, Long-term follow-up for brain tumor development after childhood exposure to ionizing radiation for Tinea capitis, *Radiat Res*, 2005, 163, 424–432.

Schardein J, Chemically Induced Birth Defects, Third Edition Revised and Expanded. New York, Marcel Dekker, 2000.

Schneider A, McCumber D, An air that kills: How the asbestos poisoning of Libby Montana, uncovered a national scandal, New York, NY, G. P. Putnam’s Sons, 2004.

Schug T, Janesick A, Blumberg B, Heindel J, Endocrine disrupting chemicals and disease susceptibility, *J steroid biochem molec biology*, 2011, 127, 3-5, 204-15.

Skinner M, Manikkam M, Guerrero-Bosagna C, Epigenetic transgenerational actions of endocrine disruptors, *Reproductive toxicology*, 2011, 31, 3, 337-43.

Smith K, Corvalán C, Kjellstrom T, How much global ill health is attributable to environmental factors?, *Epidemiology-Baltimore*, 1999, 10, 5, 573-84.

Swan S, Count down: how our modern world is threatening sperm counts, altering male and female reproductive development, and imperiling the future of the human race, Scribner NY, 2020.

UNEP: United Nations Environment Programme, UN report: Global chemicals outlook II, from legacies to innovative solutions, Implementing the 2030 agenda for sustainable development, 2019

<https://wedocs.unep.org/bitstream/handle/20.500.11822/28113/GCOII.pdf?sequence=1&isAllowed=y> and https://www.unep.org/news-and-stories/press-release/un-report-urgent-action-needed-tackle-chemical-pollution-global?_ga=2.7467698.183529524.1552589407-2141506034.1552589407 accessed 1 Jun 21.

Vogel J, Tunnel vision: The regulation of endocrine disruptors, *Policy Sciences*, 2004, 37, 3, 277-303.

Vojdani A, Vojdani E, Vojdani C, Immune system under fire: the rise of food immune reaction and autoimmunity. integrative and functional medical nutrition therapy, *Humana Cham*, 2020, 843-62.

Warren C, Brush with death: A social history of lead poisoning. Baltimore, MD, Johns Hopkins University Press, 2000.

Weldon R, Barr D, Trujillo C, Bradman A, Holland N, Eskenazi B, A pilot study of pesticides and PCBs in the breast milk of women residing in urban and agricultural communities of California, *J Env Monitoring*, 2011, 13, 11, 3136-44.

Whaley P, Systematic review and the future of evidence in chemicals policy (report); policy from science project, Lancaster University, Lancaster, UK, 2013.

Whitelaw N, Whitelaw E, Transgenerational epigenetic inheritance in health and disease, Current opinion in genetics & development, 2008, 18, 3, 273-9.

WHO, UNEP, State of the science of endocrine disrupting chemicals – 2012, An assessment of the state of the science of endocrine disruptors prepared by a group of experts for the United Nations Environment Programme (UNEP) and World Health Organization (WHO), 2013
<https://www.who.int/ceh/publications/endocrine/en/> accessed 16 Mar 2019.

WHO: World Health Organization, An estimated 12.6 million deaths each year are attributable to unhealthy environments, News release, 15 Mar 2016, Geneva <https://www.who.int/news/item/15-03-2016-an-estimated-12-6-million-deaths-each-year-are-attributable-to-unhealthy-environments> accessed 18 Aug 21.

Chapter 2

Is Health Affected by Environmental Exposures?

Contents

Chapter 2.....	35
Is Health Affected by Environmental Exposures?.....	35
INTRODUCTION.....	36
INDIRECT ENVIRONMENTAL EXPOSURES	47
EXAMPLES OF XENOCEMICAL EFFECTS OF WHICH THE PUBLIC REMAINS LARGELY IGNORANT	53
Textiles	53
Food ingredients	54
Plastics and Food Packaging	55
Air pollution	56
Food	61
WHAT CAUSES CHRONIC DISEASE?.....	66
Autoimmune Diseases	72
Cancer	85
INDIRECT ENVIRONMENTAL HEALTH EFFECTS	96
HOW DO DOCTORS RECOGNISE ENVIRONMENTAL EFFECTS?	103
BIOMONITORING	108
CUMULATIVE EFFECTS	113
LIFESTYLE CHANGES	119
GENERATIONAL TOXICITY	120
CHILDHOOD EXPOSURES.....	125
PRENATAL	126
MULTIGENERATIONAL TOXICITY.....	131
TOXIN EFFECTS ON CHILDHOOD INTELLIGENCE	133
ENDOCRINE DISRUPTING CHEMICALS (EDCS).....	134
CONCLUSION.....	137
COMING CHAPTERS: WHERE THIS THESIS IS HEADING.	140
REFERENCES.....	142

INTRODUCTION

This chapter will discuss broad aspects of environmental exposures that affect human health. Environmental exposures generally occur when toxins are encountered in current everyday life. “Natural” toxins will not be considered, only manufactured chemicals and radiation that are foreign to human bodies and potentially toxic to human health. This fits with the thesis as it illustrates that environmental exposures can affect human health and should be considered in the treatment of health-compromised individuals. Subsequent chapters will consider why the medical orthodoxy does not show such consideration in health treatment.

The science that already exists on the effects of low concentrations of manufactured chemicals in our environment will be used in this chapter. Chapter 2 will consider whether common health problems found in the general population can be attributed to situations of environmental exposure. Comprehensive coverage is not needed to illustrate the intended point, although a great variety of scientific studies and a few major health conditions will be briefly addressed. In so doing, the randomness of the studies and the extent of possible effects will show that environmental exposure is an important consideration. They indicate significant areas of undone science, since the majority of research and/or authoritative pronouncements on health conditions, in general, do not consider environmental causation. Those studies done in this area commonly have their findings ignored or dismissed, resulting in general ignorance in this area. Nevertheless, there has been a reasonable amount of research as will be shown.

In demonstrating evidence to connect environmental exposures and health effects, many chronic health conditions are also linked in. The varied examples will illustrate the extent that previously undone science has contributed to allowing such environmental exposures and the consequential public ignorance on the topic. They also illustrate the extent of the problem in seemingly innocent areas of the living environment. It will finally be seen that the real tragedy of the situation is how children and future generations are being affected.

From the examples referenced and the opinions expressed by various authorities in this chapter, it will be seen that low-level environmental exposures can affect public health.

There is usually limited knowledge about disease etiology in respect to causal mechanisms, for example, leukaemia can begin when bone marrow starts to show certain characteristics. The

orthodox etiological analysis begins here and this is accepted as an “explanation” or “cause” in orthodox medical circles. But this thesis seeks to highlight earlier aspects that could have triggered the bone marrow to start presenting unusual characteristics. As will be seen in this and subsequent chapters, the modern living environment now subjects everyone to thousands of non-natural, manufactured chemicals (henceforth termed “xenochemicals”). Although many of these exposures are at low levels, the sheer number of exposures to the hundreds of thousands of these chemicals, the constant rate of such exposures, and their bioaccumulation, together over long periods of time can affect human health. This clearly presents a complication for scientific studies in finding a control group facing zero environmental risk.

This thesis will also not attempt to address detailed conceptual and measurement difficulties in defining diseases, but rather will highlight paradigmatic examples of resistance to the idea of diseases having clear environmental causes, as seen in the medical and health systems.

Although some environmental exposures are clearly influenced by individual decisions, such as diet, stress, exercise, alcohol, smoking, recreational drugs, etc., and have an effect on health, these will not be considered in this thesis. It is simply acknowledged that these exposures are commonly affected by individual choice and are associated with individuals’ social situation.

There are many hypotheses that if a sufficiently long time horizon is used, all disease is environmental, even those related to genetic factors (Smith 1999). More than two decades ago it was starting to be acknowledged that environmental factors were causing 25-33% of the worldwide burden of disease, although the definition of “environmental” used to determine these factors was a very tight, conservative one (Smith 1999). Unfortunately, industry, which is responsible for the myriad of manufactured or synthetic substances now ubiquitous in the world, frequently confuses the public by referring to such substances as “environmental factors” as-if those substances were naturally occurring. Even a patient being told that a chemically caused disease is due to “environmental factors” may think the cause was natural, rather than being preventable or avoidable. Hence the term itself is also responsible for generating a great deal of public ignorance on the subject.

Through the decades, well-researched articles have been assessing the science that clearly attributes major health conditions to low-level chemical-mixture effects. One example is the paper by Carpenter et al. (2002) which links development disorders, neuro-behavioural

abnormalities, sexual disruption, neurodegeneration, cancers and heart disease to low-level chemical mixtures.

Environmental exposures that can affect health may come from a broad array of exposures:

- Air pollution: the absorption of many types of toxins via the lungs, olfactory and trigeminal nerve pathways, or dermally through the skin.
 - Outside air pollution, from industries, vehicle emissions, agricultural sprays, etc., in molecular to particulate forms.
 - Indoor air pollution from use of pesticides, deodorants, sanitisers, detergents, cleaning products, etc. through to outgassing by new products such as painting, flooring materials, plastics, furniture, new cars, etc.
- Water pollutants: toxins present in drinking water, shower water, bath/swimming pool water, etc.
- Food pollutants and types of food
 - Pesticide residues from agricultural use
 - Genetically modified food
 - Processed food with its preservatives, emulsifiers, flavourings, glues, colourings, bulking agents, etc.
 - Microwave-heated food (in respect to reduction of nutrient levels and other alterations due to microwave radiation).
 - Cooking tools such as aluminium pots and trays, non-stick frypans, etc.
- Fragrances and cosmetics: from their extensive use of many synthetic chemicals
- Electromagnetic fields (EMF): including wi-fi, bluetooth devices, mobile phones and their transmission towers, power transmission lines, household electricals from the power boxes (e.g. smart meters) through to electrical circuitry and devices (e.g. electric blankets, heaters, TVs), radio transmissions, satellite communication, etc.
- Ionising radiation: air-borne from coal-fired power stations, nuclear facilities, use of X-rays, medical diagnostic and security machines, and from background environmental levels increased by mining activities.
- Consumer chemical products: sunscreens, drink products, tattoos, nail polish, anti-sweat products, hair dyes, hair shampoos, etc.

- Clothing and fabric materials: their synthetic nature results in dermal absorption, outgassing and nano-particles.
- Medical treatments: drugs, magnetic resonance imaging (MRI), X-rays
- Diet in general in respect to nutritional value (apart from pollutants).
- Self-imposed influences such as stress, lack of exercise, smoking, drug use, etc.

As can be seen from this list, many consumer products are involved. These include products and items which are taken for granted in society and are regarded as necessary or normal. Most people view them as a product of technological advancement, but most such advancements are not assessed as to whether they could have any health effects. Each product on its own is regarded as posing negligible environmental risk, with never a thought to the combined exposure effects long term, or on young children. Since no immediate or obvious health effects can be quickly noted, the product's use is simply accepted for the particular advantage that its technological development offers.

Technological developments have shown amazing creativity and manipulation of matter in their adaption into commercial products. Historically, most have seemingly benefitted and advanced society's standard of living. Unfortunately much of the developed technology has unexpected secondary effects on human health and environment, with substantial public ignorance around the extent of its effects. As prominent animal bioethicist, Lisa Moses, states, "Humans have a very long history of messing around in nature with all kinds of unintended consequences ... It's really hubris [sic] of us to assume that we know what we're doing and that we can predict what kinds of bad things can happen" (Rana 2019).

A few examples among many will show where technological developments have had unexpected consequences:

- Heavy metals and other xenochemicals, with half-lives between decades and centuries, became present in streams and lakes, the ambient air, at the earth's poles and in the depths of the oceans, e.g., Dichlorodiphenyltrichloroethane (DDT), mercury, thorium, glyphosate, lead, Para-dichlorobenzene (PCB), fluorinated and brominated chemicals, etc. These have accumulated in animals, fish and humans.
- Mercury and aluminium were detected (decades ago) in human brains.

- Lead from the industrial plants, paints and car exhausts has affected children's intelligence
- The use of DDT was initially successful in reducing insects, but destroyed the birdlife that fed on those insects. When the insects developed resistance to DDT there were then no birds to keep the insects in-check, producing an endless market for insecticides: capitalism benefitted from the destruction/manipulation of nature. This was repeated with other pesticides.
- Toxic chemical residues began showing up and concentrating in fetuses and infants (Rea 2017).
- Plastics affected endocrine systems unseen, and visually filled the oceans. They eventually degenerate to nanoparticles that have been found in fish and so, in humans as well.
- Endocrine disrupting chemicals affected fertility and health in future generations, e.g., per- and poly-fluoroalkyl substances (PFAS), that make products water-oil-grease-stain-resistant and take thousands of years to degrade. Accumulating in humans, they have become associated with diverse effects such as increased incidence of perimenopause and menopause symptoms starting in women as young as 20 years of age (Taylor 2014).
- Accumulating in humans, prenatal exposure to phthalates (from plastics) has lowered children's IQ (Factor-Litvak 2014, Lee 2018). Phthalates have been associated with attention deficit hyperactivity disorder (ADHD) (Kim 2009) and other neurological problems.
- Communication demands have resulted in most areas of the earth being bombarded with electromagnetic radiation with suppressed studies since the 1950s showing the effects of EMF on most living organisms
- In the time since wi-fi and mobile phones existed, sperm counts have dropped by more than half alongside decreasing fertility rates, with many studies confirming such effects are possible (Agarwal 2009, Aitken 2005, Behari 2006, Dasdag 1999, De Luliis 2009, Eroglu 2006, Fejes 2005, Kumar 2012, Levine 2017, Otitoloju 2010, Yan 2007, Wdowiak 2007).
- Monsanto for decades claimed that glyphosate had no negative effect on human health despite accumulating empirical evidence to the contrary. The regulatory authorities

took no action in the face of the mounting evidence. There are now many successful lawsuits that have proven severe and fatal health effects.

- Demand for energy has seen coal burning inject thousands of tonnes of mercury, dioxins, nanoparticles and radioactive particles into the atmosphere every year, with thousands of tonnes of toxic fly-ash stored in holding dams. Nuclear power stations produce tens of thousands of tonnes of radioactive waste per year with half-lives of up to 15.7 million years (as for Iodine-129).
- Extensive use of antibiotics in livestock to enable concentrated but less healthy, animal rearing conditions has contributed to the human toll from drug-resistant microbes. 80% of antibiotics produced in the US are used in agriculture. Such continual use has evolved resistant bacteria and these, plus the antibiotics, are carried through to the human population through the meat produced, including vegetables fertilised with the manure of animals fed antibiotics.
- Fungicides used throughout agricultural cropping have enabled fungi to develop into new “superbugs”, such as candida auris (Chowdhary 2013, Rhodes 2019). The superbugs are now affecting immune-compromised people and being detected in hospitals. The normal antifungal medications applied to humans had been the same, or only slightly different to, the fungicides used in agriculture.
- Creation of nanoparticles for toothpastes, sun-block lotion, cosmetics, vaccines, paint, etc. through to the degradation of plastics, clothes, burning of fossil fuels, metal processing and even fires. These nanoparticles can pass through the human skin, blood brain barrier, maternal-foetal barrier, lung lining, digestive lining, cell walls and other biological barriers designed to protect human life. They can be dermally absorbed, breathed-in or enter the food chain through fish and livestock. They are accumulating in the environment as well as in humans.
- Chlorine was added to drinking water to kill bacteria, but reacts with natural substances in the water to create toxins that have been implicated in miscarriages, stillbirths and birth defects.
- Coated paper or styrofoam coffee cups release high levels of toxins when exposed to heat.

- “Because of linked-epitope suppression, all children who were primed by DTaP [diphtheria, tetanus, and whooping cough (pertussis)] vaccines will be more susceptible to pertussis throughout their lifetimes, and there is no easy way to decrease this increased lifetime susceptibility” (Cherry 2019p334).

The above examples are but a few of hundreds that could be listed. There are also other indirect effects in considering waste from products we take for granted. In considering e-waste for example, toxins are released into the environment from rubbish tips or waste centres: epoxy resins, fibreglass, PCBs, perfluorooctanoic acid (PFOA) and other flame retardants, polyvinyl chloride (PVC) and various plastics, lead, tin, copper, gold, silicon, beryllium, carbon, iron, aluminium, cadmium, mercury, thallium, americium, antimony, arsenic, barium, bismuth, boron, cobalt, chromium, europium, gallium, germanium, indium, lithium, manganese, mercury, nickel, niobium, palladium, platinum, rhodium, ruthenium, selenium, silver, tantalum, terbium, thorium, titanium, vanadium, yttrium, etc. (Cribb 2014).

New words and concepts such as biopersistence, bio-accumulation, endocrine disruption, mutagenesis, epigenetics, etc. have been employed to describe some of the effects which have gradually become apparent through people, flora and fauna being affected. Seemingly with each advance came an invisible, delayed cost to humans and the environment. The saying that “there’s no such thing as a free lunch” seems very appropriate. Human progress seems to have come at a cost to the environment we live in. It continues to grow at an increasing rate since the contamination of persistent chemicals, radioactive waste, greenhouse gases, etc. is breaking down at much slower rates than that contamination is being produced. The effects of such progress are potentially having effects on every person and living thing on the one finite planet that we live in. The situation is escalating at such a rate that the next generation will experience many more severe problems than the last.

Of the approximately 85000 chemicals that are deliberately manufactured for public consumer chemical products, about 3000 are produced in amounts greater than 454 tonnes per year. For half of these there is no basic toxicity information. For 80% of them there was no Information on their effect on developing children. This is unfortunate since studies have shown that there are higher concentrations of these chemicals in children than adults (Landrigan 2006) and the data gaps are well documented (Allanou 1999, Applegate 2006, Birnbaum 2003, Guth 2005,

NRC 2007, USEPA 1998). Many liken the situation to “conducting a vast toxicological experiment in our society, in which our children and our children's children are the experimental subjects” (Crumpler 2014p230).

The total number of manufactured xenochemicals is unknown. There are many chemicals manufactured which are not for specific chemical products as referenced in the last paragraph. The largest database on chemicals, the Chemical Abstracts Service (CAS) Registry, lists more than 100 million chemicals (CAS 2015) with more added each week (Obodovskiy 2015). Commercial chemical production had risen from 1 million tons in 1930 to 400 million tons in 2001 (Walters 2006). Yet with so many xenochemicals in the environment, the small percentage of these chemicals that have been studied are each then evaluated on their own, in isolation. Large population biomonitoring studies show the widespread chemical exposures from the “womb to the tomb” and many chemical human body burdens are at levels individually that are known to cause health effects (Bijlsma 2016).

But the biomonitoring studies are generalised and averaged over the population studied and there is a shortage of meaningful studies, e.g., the accumulation of phthalates (endocrine-disrupting chemicals found in plastics) beginning from conception or birth. There is now so much plastic in the environment that it is estimated that the average person eats about 5 grams of plastic per week or the amount found in one credit card; this reaches about 20kg over a lifetime (WWF 2019). There are isolated studies that illustrate that 36.5% of total exposure to phthalates can occur via indoor dust, especially for toddlers (Wang 2013). A study of 144 adults found that 90% had at least eight different plasticisers in their urine (Karrer 2020). There is significant evidence of plastics disrupting hormones, including the feminisation of males, miscarriages, infertility and low levels of vitamin D. During the ongoing COVID-19 situation 129 billion face masks are being used globally every month (3 million / minute) with most being disposable face masks made from plastic microfibres (Prata 2020).

In 2012, the World Health Organization (WHO) estimated that manufactured chemicals contributed to 4.9 million deaths and disabilities (Bijlsma 2016, UNEP 2012). However this excluded deaths attributed to cancer, heart disease, obesity, diabetes, stroke, lung disease, genetic disorders, mental disorders, etc. many of which have links with environmental xenochemical exposures, as shown in numerous studies to be presented in this thesis.

Some tell-tale signs that cancers, for example, can be environmentally related are:

- One study that showed that for every 100,000 people, there were 296 cases of cancer in developed regions, while there were only 115 (less than half as many) in less developed ones (Bray 2018).
- “Cancer is a leading cause of death for children and adolescents around the world and approximately 300,000 children aged 0 to 19 years old are diagnosed with cancer each year” (WHO 2018); “the recorded incidence tends to increase with time” (Steliarova-Foucher 2017p719).
- “Positive associations were observed between childhood leukemia and residential pesticide exposures” (Turner 2010p33).
- One Nordic country study reported, “The last 10 years of melanoma incidence trends, increasing at an exponential rate, suggest that responsible authorities now need to consider possible influences also from other radiation sources in addition to UV radiation from the sun” (Hallberg 2016p103).
- Cervical cancer rates increased by 54% in British 25- to 29-year-olds (Cancer Research UK 2020, Rees 2020). The shortcomings of one vaccine’s trials (Gardasil) have been highlighted by the US Food and Drug Administration (FDA) Vaccine and Related Biological Products Advisory Committee, as the administration of the vaccine actually increased (by 44.6%) the risk of cervical cancer among women with a current infection, or those previously exposed to, human papillomavirus (HPV) (VRBPAC 2006). This was consistent with another study of Alabama girls which found the highest cervical cancer rates were in the state’s most highly vaccinated counties (USA 2019).

More-specific xenochemical examples that show evidence of a causal link with cancer will be covered later in this chapter. There are, however, a myriad of less lethal conditions such as developmental disabilities, which have also been increasing markedly in the last few decades, such as autism spectrum disorder (ASD) and ADHD. These are frequently attributed to improved or altered diagnosis and marketisation of medicine, yet studies specifically looking into this cannot attribute the increases to this explanation alone (Hansen 2015, Neggers 2014). Developmental disabilities are common, and presented in about one in six children in the United States in 2006–2008 (Boyle 2011).

The increase in diseases of children has been noticed by older doctors over the years (Goldberg 2019p3). Even high blood pressure in children has been noticed and related to environmental exposures (Suarez-Lopez 2019).

Allergic/asthmatic diseases have steadily increased globally: about 30–40% of the world's population are now affected by one or more such conditions (Pawankar 2013).

In 2017--18, 47.3% of Australians had at least one chronic condition. This increased from the 2007--08 rate of 42.2% (ABS 2018).

The following sections will consider specific health problems linked with environmental exposures. At the same time, the extent of environmental exposures will be highlighted, illustrating that many products in widespread use contain and emit toxic chemicals. The common absence of immediate effects on human health from exposure to such chemicals hides their toxic nature medium-to-long term. Industry's dismissal of the exposures as being insignificant further influences the public not to investigate further. Furthermore, the generalised ignorance of the sheer number (in the millions) of chemicals we are exposed to is a problem: a single jigsaw piece may be insignificant on its own but when combined with thousands of other pieces can present an alarming picture.

The chapter starts by considering various indirect environmental exposures, such as those from the food we eat, plastics, and various consumer goods. Some examples of significant xenochemical exposures that the public is mostly unaware-of are covered: particularly textiles, and food ingredients, residues and packaging. Air pollution will also be touched on; although the public knows this exists, they usually lack knowledge on pollution's potential to cause direct effects.

The strong connections between environmental exposures and chronic disease will then be discussed. Two chronic diseases will be considered: autoimmune disease and cancer.

While indirect environmental exposures are covered early in chapter 2, the further indirect health effects of environmental chemicals when they enter the body will be mentioned in a later section. This will highlight the complexities involved in attempting to study environmental exposure effects, which is followed by comments on how orthodox doctors recognise (or do not recognise) environmental health effects.

At the end of this chapter, the consequential effects of failing to pay attention to environmental exposures are mentioned. The discovery of xenochemical residues through broadscale sample testing of national populations is covered through consideration of biomonitoring surveys, with special mention of the United States. As those surveys find a myriad of xenochemicals present in each person tested, I then consider the effects of cumulative exposures, to multiple xenochemicals at the same time. This highlights how few studies have been done on these possible effects: the immense area of undone science in studying real-life situations of exposure. Mention is made of informed, and sometimes simple, lifestyle changes that can have a restorative effect on the body and protect against chronic diseases in a short lifestyle change section.

As was seen in the acknowledgements, the author's grandchildren were a central reason for doing this thesis. However I initially underestimated the seriousness of the situation. Through the thesis research, the real tragedy of the situation is clearly found to be the detrimental consequences for future generations. As a result, the chapter finishes by outlining some of these consequences in sections on generational toxicity.

From this coverage, Chapter 2 will illustrate that there are many links between environmental exposures and human health effects. This will then act as the basis for considering, in subsequent chapters, why this area is overlooked by society and not considered by the medical system or government regulators.

INDIRECT ENVIRONMENTAL EXPOSURES

This section highlights evidence that many xenochemicals in the environment have an indirect effect on human health. Direct exposures to such chemicals are only the tip of the iceberg with public ignorance being substantial in this area

There are a multitude of indirect environmental exposures. One of the most prominent is via the food we eat. The drive by agriculture for increasing yields from the land has seen hybridization for size and growth rates, the use of pesticides, synthetic fertilisers, and GMOs. Farmland is no longer rested, being fed with an artificial chemical cocktail to enable the continual tilling, producing serious mineral depletion. Pesticides such as Roundup have, at least initially, been effective against pests, resulting in farmers becoming complacent. So rather than crop rotation, a traditional practice to reduce pests and replenish the soil, the same crops have been planted year after year, relying on pesticides to keep pests at bay. Systemic insecticides, promoted as far less toxic to the environment than pesticides of the past, further add to this phenomenon. But they have been suspected of causing long term colony collapse disorder decimating the bees, essential for crop pollination.

The agricultural pesticides “resulted in widespread contamination of agricultural soils, freshwater resources, wetlands, non-target vegetation and estuarine and coastal marine systems”, meaning that many organisms in those habitats were “repeatedly and chronically exposed to effective concentrations of these insecticides” (Van der Sluijs 2015p148). We now not only have food from such farming practices with chemical residues but also the contaminated marine food sources from the run-off of such agricultural land.

The drive to increase yields in food production has seen yields on onions and nectarines up about 200%; celery and garlic, 250%; broccoli, beets and cantaloupe, 300%; tomatoes and almonds, 500%; strawberries, 550%, and similar increases in other crops (Schatzker 2016). The result is a “dilution effect” recognising that water and carbohydrate content has risen, with protein, vitamins and minerals substantially reduced in most of these crops, e.g., reductions in calcium, magnesium, copper reduced by 80% between mid to end 1900s (Mayer 1997). Researchers studying the drop in nutrient levels in food hypothesise it is due to lack of soil microbial diversity due to pesticides, GM plants, soil erosion, seed quality decline, and air and

water pollution. This results not only in contaminated food, but also low quality in respect to nutrient and mineral content for optimal health.

There are many examples where people have considered their environmental exposures, especially in the food they eat. After modification of such environmental influences an improvement in their chronic condition can be noticed. One example is the healing of Grave's disease (an autoimmune disorder that causes hyperthyroidism, or overactive thyroid). This has been done through lifestyle changes including an ancestral diet, oral health practices, exercise, avoidance of environmental toxins (purified water, organic food, natural cleaning/personal hygiene products), and supplements (Brogan 2019). There are thousands of testimonials like this regarding many other health conditions.

Indirect environmental exposures are diverse. Even the use of a seemingly innocent products like sunscreens, exposing the users to toxic ingredients such as oxybenzone, an endocrine disruptor, found in an estimated 70 percent of sunscreens. A significant percentage of these chemicals can reduce male fertility by affecting calcium signalling in sperm, in part by exerting a progesterone-like effect (Rehfeld 2018). Further effects are discussed in chapter 4 using sunscreens as an example of public ignorance.

Organisations such as the Lancet Commission on pollution and health have noted that anthropogenic chemical releases into the environment from industrial, agricultural and consumer waste sources presents major threats to global public health. This is more so in developing countries where chemical products are made, utilised, and disposed of with little regulation. While the emphasis in the past in these countries has been on water quality, sanitation and hygiene, the resultant environmental xenochemicals have been recently suspected as the reason for various health problems such as the lack of significant reduction of diarrhoea, or improvement in child growths. It has been hypothesised that control needs to be for both pathogens and chemicals (Kearns 2019). This is due to the ability of synthetic chemicals to suppress or dysregulate human immune function. Immunotoxicity has been shown in many chemical families, such as the halogenated organics, plasticisers (phthalates and bisphenol-A, etc), many pesticides (atrazine, chlordane, etc.), flame retardants, and per-fluoroalkyl and poly-fluoroalkyl substances (PFAS).

In developed countries, the Environmental Burden of Disease Project studied nine environmental stressors (benzene, dioxins including furans and dioxin-like PCBs, second-hand smoke, formaldehyde, lead, noise, ozone, particulate matter and radon) in respect to the effects on health in six countries (Belgium, Finland, France, Germany, Italy, and the Netherlands). It was found that these nine environmental stressors were estimated to cause 3 to 7% of the disease burden in those countries (Hanninen 2014).

Even the xenochemicals that are used to deliberately affect human health, in the form of medicines and pharmaceutical products, can find themselves in the general environment. 17 α -Ethinyl Estradiol (EE2) is a strongly estrogenic component of oral contraceptives for women. EE2 has stronger activity than Bisphenol A (BPA), a well known toxic chemical. It is equal to BPA for affecting estrogen receptors associated with cell membranes (Watson 2011). As 16-68% of EE2 medications can be excreted (Johnson 2004) it has been found in aquatic environments downstream of wastewater treatment plants. Both EE2 and BPA can be present in water in sufficient quantities to affect development, osmoregulation, and reproduction in aquatic organisms (Bhandari 2015b, Brown 2009a). One study found a 30% decrease in fertilization rates of fish two generations after exposure and a 20% reduction after three generations. This shows that EDCs may not affect the life of the exposed fish, but may detrimentally affect future generations (Bhandari 2015b). Aside from effects on the fish, biomagnification into humans which consume them will occur.

Microplastics have been increasingly reported in air samples, food, tap water and bottled water (EFSA 2016; Gasperi 2018; Lusher 2017; Van Cauwenberghe 2014; Wright 2017a; Yang 2015a, Kosuth 2018; Mason 2018; Mintenig 2019, Schymanski 2018). These as well as a variety of products such as baby feeding bottles during infant formula preparation (Li 2020). Molluscs, such as oysters, scallops, and mussels, which effectively filter seawater, are registering the highest microplastic levels in fish (Danopoulos 2020). The potential for microplastics to affect human health is concerning (Wright 2017a, Koelmans 2019). While microplastic exposure via ingestion or inhalation is possible, their health effects on humans is undone science. A small number of animal studies indicate that microplastics accumulate and cause an immune response (Deng 2017; Gasperi 2018). Microplastic additives as well as adsorbed toxins can leach out causing chemical toxicity (Diepens 2018; SAPEA 2019). Microbial pathogens may also be present in biofilms growing on the microplastics (GESAMP 2016).

Most members of the general public do not know that many consumer goods can result in exposure to quite toxic substances. One example is that of skin moisturisers which can contain (Zenn 2012):

- Mineral Oil, Paraffin, and Petrolatum – Petroleum products that coat the skin. Slows cellular development and a suspected cause of cancer. Disrupt hormonal activity.
- Parabens - Approximately 13,200 cosmetic and skin care products contain these. These have been associated with cancer. They have hormone-disrupting qualities and mimic estrogen.
- Phenol carbolic acid – Linked to circulatory collapse, paralysis, convulsions, coma and respiratory failure.
- Propylene glycol – Also used as a carrier in fragrance oils. Causes dermatitis, kidney or liver abnormalities and can inhibit skin cell growth or cause skin irritation.
- Acrylamide – present in many face and hand creams: Linked to mammary tumours in lab research.
- Sodium laurel or lauryl sulfate (SLS), also known as sodium laureth sulfate – It is used in car washes, engine degreasers, garage floor cleaners, yet is also in over 90% of personal care products! It breaks down the skin's moisture barrier and penetrates the skin allowing other chemicals to also penetrate. SLS converts in the body to a "nitrosamine," a potent class of carcinogen, when combined with other chemicals. Hair loss can also be caused. SLS is commonly disguised with labelling "comes from coconut" or "coconut-derived."
- Toluene – A known poison, is harmful if inhaled or absorbed through the skin. It is derived from petroleum or coal tar. It is found in most synthetic fragrances also. Chronic exposure is linked to anaemia, lowered blood cell count, liver or kidney damage and can affect a developing foetus. Butylated hydroxytoluene contains toluene. Labelling names may include benzoic and benzyl.
- Dioxane– Many personal care products contain this. It can be found in compounds known as PEG, Polysorbates, Laureth and ethoxylated alcohols. The compounds are usually contaminated with the highly volatile 1,4-dioxane, easily absorbed by the body. It has been known since 1965 to be carcinogenic. It particularly affects the nasal

passages and liver. It is a synthetic derivative of coconut; labels may say "comes from coconut."

It is no surprise that tests of adolescent girls find levels of many of these chemicals from cosmetics and body care products. One test featured exposure data for parabens in teens indicating that young women are widely exposed, with 2 parabens, methylparaben and propylparaben, detected in all girls tested (EWG 2008). Fragrances are another mine-field: "In 1989 the US National Institute of Occupational Safety and Health evaluated 2,983 fragrance chemicals for health effects. They identified 884 of them as toxic substances." (NCEHS 1990). Furthermore, the U.S. Centers for Disease Control and Prevention (CDC) measured for 13 different phthalate metabolites in urine, finding such in the general population indicating widespread exposure. Women had higher levels of urinary metabolites than men, due to more use of soaps, body washes, shampoos, hair dyes and other personal care products (CDC 2020a).

One of the greatest concerns of the effect of some of the xenochemicals in the environment is their potential for human endocrine system disruption, touched on before. Endocrine Disrupting Chemicals (EDCs) can be found in such substances as pesticides, electronics, personal care products, cosmetics and additives or contaminants in food. Established health problems from EDCs include "the development of non-descended testes in young males, breast cancer in women, prostate cancer in men, developmental effects on the nervous system in children, attention deficit /hyperactivity in children and thyroid cancer" (WHO 2013b). The European Environment Agency went further stating that "the link between some diseases and EDCs is now accepted. For example, exposure to oestrogen or to oestrogenic EDCs is an accepted risk factor for breast cancer, endometriosis, fibroids and polycystic ovarian syndrome in women....There is a trend towards the earlier onset of puberty in girls, which may be influenced by EDCs" (EEA 2016). It is however pertinent that all authorities agree that many other disease risks are expected and that more research is urgently required, indicating that such research was never performed on these many products before being used in consumer products by industry.

While a few EDCs have been banned in some countries, the amount of EDCs now accumulating in the global system continues to grow. While the warnings about this situation have been growing louder over the last generation, industry and governments are not acting to attention

the situation. Among the prominent people to have highlighted this decades ago, is Theo Colborn, author of the book, *Our Stolen Future* and founder of The Endocrine Disruptor Exchange (TEDX). The UN Environmental Programme and WHO produced a joint report (WHO 2013a) stating that:

... endocrine systems are very similar across vertebrate species and [that] endocrine effects manifest themselves independently of species. The effects are endocrine system related and not necessarily species dependent. Effects shown in wildlife or experimental animals may also occur in humans if they are exposed to EDCs at a vulnerable time and at concentrations leading to alterations of endocrine regulation. Of special concern are effects on early development of both humans and wildlife, as these effects are often irreversible and may not become evident until later in life.

It has been illustrated in this section that direct exposures to environmental pollutants are only part of the concern. The indirect exposures may in fact be significant. In illustrating this the section drew firstly on how agricultural xenochemical use has both contaminated our food but also decreased the mineral and vitamin content and affected bees responsible for the pollination of crops. Sunscreens were highlighted for their effects on the endocrine system which in turn can affect male fertility. Immunotoxicity has been shown in many chemical families, leaving the body's defence system impaired to maintain health. Medicinal product indirect effects on marine life and the subsequent biomagnification back to humans was also discussed. Plastic product breakdown into microplastics were highlighted for their effects on human immune systems with their adsorbent nature for toxins and pathogens. Cosmetics were mentioned for their toxic contents and production of human toxin loads, especially in women. Lastly, concerns were expressed how EDCs are suspected of causing problems for future generations from their exposures to the current generation.

Indirect effects of a toxic exposure have been recently found to be able to be multiplied through exogenous exosomes from cell to cell and mouse to mouse (Cho 2018). This is a similar mechanism as cancer metastasis and the promotion of angiogenesis (Costa-Silva 2015, Peinado 2012). This means that the toxic exposure of one cell can affect the cells around it without the surrounding cells having direct exposures to the toxin.

EXAMPLES OF XENO-CHEMICAL EFFECTS OF WHICH THE PUBLIC REMAINS LARGELY IGNORANT

This section highlights some examples on which there is much public ignorance. The public in general has little awareness of the extent of environmental exposures even from seemingly innocent common commodities and products. Only three examples will be touched-on: textiles, food ingredients and plastics.

Textiles

There is some awareness of the amount of environmental plastic particulate pollution that comes from the washing of synthetic clothing. However the prolonged, mostly direct contact exposure of the various synthetic chemicals present in clothing can become significant. Possible health effects from such are generally not normally thought of by the general public. A pertinent example of direct health effects involves Delta Airline staff after new uniforms were issued. The uniforms had been introduced in mid-2018. The employees shortly after reported health concerns, including skin rashes, headaches, fatigue and chemical sensitivities.

One attendant reported (Elk 2019):

I noticed right away after I put the uniforms on that I had shortness of breath and I have been a runner my whole life. I don't smoke or anything like that, so when I couldn't get up the stairs without being extremely winded, I kn[e]w there was some sort of problem.

Sara Nelson, president of the Association of Flight Attendants-CWA, commented on the clothing concerns (Griffith 2020):

This issue is real. It affects different people in different ways, and the reactions can vary in severity with symptoms such as rashes, headaches, hair loss and breathing problems when wearing the uniform to becoming so sensitized to the chemicals that it's impossible to even be in the same space without getting extremely sick.

The uniforms were high stretch, wrinkle and stain-resistant, waterproof, anti-static and deodorizing. The chemicals used to achieve such characteristics were attributed to causing the

various health problems. It was suspected that formaldehyde and teflon, for example, had been used in the uniforms to make them stain resistant and durable.

Another example can be found in considering baby nappies and tampons. These have been found to contain high phthalate and volatile organic chemicals (VOCs). “As sanitary pads and diapers are in direct contact with external genitalia for an extended period, there is a probability that a considerable amount of VOCs or phthalates could be absorbed into the reproductive system” (Park 2019p114).

Food ingredients

One could write books on the aspects of food production and ingredients. Following the theme of this chapter, simple examples are employed here only. The public are generally ignorant of the degree foods are made with synthetic chemicals. Many would expect that lemonade has a lemon content, or strawberry syrup has been derived from real strawberries, or ‘chocolate’ in cheap confectionaries was made from real chocolate. Such is the accomplishment of chemistry experts that can now make all of these to fool the taste buds to believing such contents are real. There are examples where unhealthy food is promoted as healthy, such as gummy fruit snacks which are usually corn syrup or concentrated fruit juice, contain mostly sugar, and rather than real fruit, they are loaded with artificial flavours and dyes (Huff Post 2012).

We also have unprocessed food affected by the modern farming methods. Apples for example, can now be found with pesticide residues on the skin and shigatoxin present inside. This is because the ultimate composition of a fruit’s microbiota is colonised by the microbial community present at pollination (Schmitt 2014). *Escherichia coli* and *Shigella* bacteria are present in conventionally grown apples: both produce the shigatoxin (Zuo 2013). Organic apples do not have this and have a far greater diversity of microbes compared to conventional apples. They contain higher amounts of bacteria that enhance flavour. Organic apples were found to contain lactobacilli bacteria that break down sugars in healthy digestion, enhance immune function and mental health (Koutsos 2015).

The butter flavourant, diacetyl, is used on popcorn and in fermented beverages such as beer, and chardonnay wines with a buttery taste. This has been linked to respiratory and other

problems in workers at microwave popcorn stations and food-flavouring factories. Diacetyl enhances toxic effects of beta-amyloids and inhibits a protective protein called glyoxalase I on nerve cells. Other lab experiments showed that diacetyl penetrates the protective blood-brain barrier (More 2012).

Plastics and Food Packaging

It is a common belief that when a product is released on the market that it must be safe. Unfortunately this is far from the reality of the situation. Thirty three scientists recently cited 1200 peer-reviewed studies in a consensus statement to plead with lawmakers "to take swift action to reduce exposure" to plastics in food packaging (EHN 2020). They highlighted "... reducing exposure to hazardous food contact chemicals contributes to the prevention of associated chronic diseases in the human population" (Muncke 2020). One of this article's 33 authors, P Myers, added (EHN 2020):

... hazardous chemicals can transfer from food contact materials into food, and some are known endocrine disrupting chemicals, or 'EDCs.' EDCs are associated with chronic diseases such as diabetes, obesity, cancer and neurological disorders like ADHD.

And concluded:

The authors say while there is a great amount of information for some of the most well-studied food contact chemicals, such as bisphenol A (BPA) and phthalates, many of the 12,000 reported food contact chemicals lack data on their hazardous properties or level of human exposure. This suggests that the human population is exposed to unknown and untested chemicals migrating from food wrappings, with unknown health implications.

It was raised that there were approximately 6000 chemicals used 10 years ago in food packaging, with this number increasing to 12000 in recent times as referenced above (Gross 2020).

The Environmental Defense Fund has highlighted misconceptions that the US FDA ensure safety of plastics in contact with food (Neltner 2019):

- Manufacturers claim that anything in contact with food must be reviewed. But their use of the "Generally Recognized as Safe" (GRAS) rule (meant to exempt common ingredients such as vinegar and baking soda), has been used to pass various chemicals such as the PFAS chemicals. Yet in the case of PFAS chemicals there are concerns on multiple types of cancers such as breast, liver, ovarian, testicular, prostate and kidney, as well as Non-Hodgkin lymphoma (Vieira 2013).
- The FDA does not require toxicology studies for chemicals in contact with foods. The manufacturer simply needs to provide what chemical, toxicological and environmental data it has. This can be quite brief and very lacking.
- The Environmental Defense Fund reviewed some applications accepted by the FDA. While the amount of information varied, the toxicity data was very poor.
- The agency has no duty to reassess their approved food contact chemicals. There is no subsequent process to monitor or re-evaluate an allowed chemical once evidence of harm, or health effects are found.

The above examples illustrate the use of toxic chemicals in everyday products, with little or no testing, regulation, monitoring, or review.

Air pollution

Air pollution is a common form of environmental exposure that everyone seemingly is aware of but have little general knowledge of the extent of its toxicity or the possible resultant health problems. This section contains examples illustrating some of the health effects that have been associated with this form of environmental exposure.

We have only one atmosphere on a finite planet. The orthodox attitude however seems to be that the air is so voluminous that any release will dilute very fast and/or gradually break-down. Many air pollutants have been dismissed by industry and government departments by classifying them as non-persistent due to having short half-lives or being metabolised within the human body in hours to days. However little thought is given to the fact

that an estimated 92% of the population breathes them in all the time (WHO 2016). It should be noted that while most air pollution originates from combustion engines and industrial plants, there are significant amounts of nitrogen oxides off gassed from agricultural synthetic fertilisers.

A study of a chosen group on children living in New Mexico city found they exhibited neurodegenerative characteristics typical of Alzheimer's and Parkinson's diseases. The researchers were so alarmed at their findings they concluded that "studies into the association between air pollution exposures and the development of neuroinflammation and neurodegeneration in children are of pressing importance for public health" (Calderón-Garcidueñas 2013p1). Later it was found that people living in areas of lower air pollution have lower risks of developing dementia including Alzheimer's disease (Wang 2021, Letellier 2021, Park 2021).

Air pollution has been linked to about 7 million, nearly one in eight, world-wide deaths in 2012 (WHO 2014). This makes it one of the world's most significant health risks. It suggests that outdoor pollution from industry, coal-burning, traffic fumes, and indoor pollution from wood and coal stoves, kills more people than smoking, diabetes and road deaths combined. Air pollutants can cause or aggravate existing lung and heart disease and many other health conditions (Hime 2015, SOE 2016).

Many health problems have been linked with air pollution such as, chronic respiratory conditions pulmonary disease, and cardio-respiratory deaths. The International Agency for Research on Cancer (IARC) has classified outdoor air pollution chemicals and particulate matter as carcinogenic to humans, from evidence of carcinogenicity in humans and experimental animals and strong mechanistic studies.

Numerous studies from China, especially genetic biomarker studies in exposed populations, support that the polluted air in China is genotoxic and carcinogenic to humans. The evaluation by IARC indicates both the need for further research into the cancer risks associated with exposure to air pollution in China and the urgent need to act to reduce exposure to the population (Loomis 2014p189).

More than 3000 premature and preventable deaths per year in Australia have been attributed to air pollution (IHME 2016, Begg 2007). Motor vehicles (especially diesel-powered), coal-fired

power stations and industry are the main sources of the toxic air pollutants of sulphur dioxide and nitrogen dioxide in Australia (DEE 2005a,b).

People who live or work near major roadways or sources of high air pollutants, and children attending childcare centres and schools in these areas, are at higher risk of harm. Of people living within 500 metres of a major road, the children have a greater risk of developing asthma, and adults have an increased risk of lung and heart-related illnesses (HEI 2010). Young children living near a major road are twice as likely to score lower on communications skills tests than those living further away. One study (Hoyer 2004) highlighted that all vehicle emissions contain mercury and that diesel trucks exhaust much higher levels than cars. It was found that children living within 300m from a freeway at birth had almost twice the risk of autism compared to those living further away (Volk 2011). Other studies have found similar indications from exposures during pregnancy and during the first year of a child's life (Volk 2013). Also children born to women exposed during pregnancy experiencing the higher levels of traffic-related pollutants, fine particulates and ozone, had an increased likelihood of developmental delays in infancy, early childhood and adolescence (Qihong 2015, NIH 2019, Newbury 2019). One study has found that air pollution from road traffic in London adversely affects foetal growth (Smith 2017).

Other generalised air pollution effects include:

- Short-term (within days, hours or minutes) exposure to air pollution is associated with acute asthma attacks, increased exacerbations of allergies, rhinosinusitis, chronic obstructive airways diseases, heart attacks and sudden death (Bowatte 2015, Gasana 2012, Di 2017, Dennekamp 2010, Zhao 2017).
- Medium-term (weeks to months) of exposure is associated with low birthweight in pregnancy (Pedersen 2013).
- Chronic long-term exposure to air pollution is associated with poor lung development in children, cancer, cardiovascular and chronic respiratory diseases, lower cognitive ability, and increased brain atrophy in adults (HEI 2010, Beelen 2013, Gehring 2013, Hamra 2014, Chen 2008, Pope 2002, Hong-Bae 2018, Bowatte 2017, Qian 2017, Lepeule 2012, Wang 2019, Younan 2020).

- Maternal exposure to air pollution from traffic was found to induce DNA damage in newborns indicating “...an increased risk for adverse health effects later in life” (Pedersen 2009p1012)

Some Australian studies demonstrated that there was no safe level of air pollutants well below the threshold standards that government departments regard as safe (Barnett 2014). The WHO air particulate matter quality guidelines state, “...research has not identified thresholds below which adverse effects do not occur...” (WHO 2006a p7). Some studies simply point to previous estimates of the health effects due to air pollution as being significantly underestimated (Lelieveld 2019).

One type of the many air pollutants is the toxic heavy metals, such as aluminium, mercury and lead. In a blood sampling procedure performed by the German toxicologist, Dr Stobel on a cross section of people, aluminium was found 94 times that of any other heavy metal pollutant (Klinghardt 2018). The average concentrations in the population was noticed to be increasing as years passed. On investigating the aluminium exposure sources, the two main sources found were vaccines and airplane exhausts.

Mercury pollutant sources can be from such products as vaccines, tooth fillings and various industries. One prominent industrial source in Australia is the coal fired power stations. The National Pollutant Inventory 2017/8 showed that “mercury output from Loy Yang B power station alone more than doubled to 831kg in 2016-2017, an increase of 123% over five years. The brown coal burning power station produced more than 640 times the airborne mercury pollution of Eraring power station near Newcastle, New South Wales” (Wahlquist 2018).

There are various independent studies finding health effect relationships with proximity to industrial sites, In the case of oil and gas (O&G) facilities, for example, one study found “air pollutant concentrations increased with proximity to an O&G facility, as did health risks” (McKenzie 2018p4514). They studied neurological, haematological, and developmental health effects. They found that cancer risks for people living within 152m of an O&G plant were above the US Environmental Protection Agency's allowable rate, and so people living near O&G facilities are exposed to unacceptable levels.

In studying magnetite pollution nanoparticles in the human brain, it was found that the link between air pollution and dementia was "particularly strong" (Maher 2016). A distinctive spherical type of magnetite was found "strikingly similar" to those formed from burnt fuel such as that from power stations, vehicle engines, open fires, and from frictional heating like in vehicle brakes, and poorly sealed stoves within homes. Particles of nickel, platinum and cobalt were also found which do not occur naturally in the brain. Shocking even the researchers, was that for every crystal-shaped endogenous or "natural" particle, there were a hundred pollutant particles. There were millions of magnetite particles for every gram of brain matter. A specialist in Alzheimer's, and one of the study's authors, Prof David Allsop, commented "This finding opens up a whole new avenue for research into a possible environmental risk factor for a range of different brain diseases" (LU 2016). The best particulate face masks have a rating of P100, blocking particles 0.3 microns (300 nm) or larger. The magnetite particles were <150nm in this study.

Until recently, research has mostly associated air pollution exposure and pregnancy with premature birth, infant mortality, low birth weight, and childhood respiratory problems. There had been a lack of solid evidence that inhaled particles could move from the lung into the blood stream. In a study looking at placental macrophages (Liu 2018), it was found that inhaled pollution particles can pass from the lungs into the blood stream as well as being able to flow to the placenta. Dr Liu commented:

We do not know whether the particles we found could also move across into the foetus, but our evidence suggests that this is indeed possible. We also know that the particles do not need to get into the baby's body to have an adverse effect, because if they have an effect on the placenta, this will have a direct impact on the foetus (Packham 2018).

Examples of silo research can be found in studying various air pollutants. Looking at end point effects, some synergism is deemed possible. For example, various air pollutants have been linked to kidney function problems (Bragg-Gresham 2018). Other studies may follow a particular chemical group, such as the PFAS chemicals and their links with kidney disease (Monaco 2018). Others can look at particulates causing rapid decline in renal function (Xu 2018). This is a common situation found in studying environmental effects: each is studied on

its own in isolation disregarding possible cumulative/synergistic effects. This common situation will be seen many times throughout this thesis.

As seen from the above, air pollution can have many effects on health, and can be a significant environmental exposure.

Food

We are what we eat: Although this acknowledged generality is utilised more to prompt the eating of healthy foods, it has a very worrying meaning when considering the contamination of foods with environmental toxins. While food is generally considered as a modifiable input to one's health, the general state of public ignorance about what food can contain is unfortunate. This section will describe examples in respect to food toxins from indirect sources, or those deliberately added, or its 'accidental' contamination. The highly toxic heavy metal, aluminium, will be used as just one example of a toxin present in foods. Although there is much undone science on the effects of all the toxins found in food, it will be referenced how even for the seemingly insignificant effects of food abundance or nutritional content, significant effects are produced on future generations.

It was seen in a previous section that food can be contaminated indirectly, via packaging. This can include glues, coatings, dyes, paperboard, paper, plastic, and other polymers, or from manufacturing equipment. However direct food additives are a further minefield given the variety and quantities of xenochemical products that can be present in food. These include food glues, emulsifiers, preservatives, colourings, flavourings and bulking agents.

Aluminium, used as an example in the previous air pollution section, can also be found as both a direct and indirect additive in various foods. It is added to various foods as a firming agent, carrier, colouring agent, anticaking agent, buffer, neutralizing agent, dough strengthener, emulsifying agent, stabilizer, thickener, leavening agent, curing agent and texturizer. These additives are used, for example, in milk, processed cheese, yogurt, preserves, jams and jellies, baking soda, sugars, cereals, flours, grains and powdered or crystalline deserts (Pennington 1988). Most food additives have been allowed through a "generally regarded as safe" (GRAS) approach by government regulatory authorities mentioned before.

Some common direct uses of aluminium compounds which generate human exposures are:

- the addition to self-rising flour as an acidifying, anti-caking and leavening agent
- adding to processed cheese, cheese food and cheese spread as emulsifying agents for a soft texture, ease of melting and desirable slicing properties (Ellinger 1972, Lione 1983). The compounds achieve a smooth, uniform film around each fat droplet which prevents separation and bleeding of the fat thereby achieving these characteristics.
- Addition to salt as an anti-caking agent
- Leaching into food from aluminium foil and cans
- Leaching into food from aluminium cookware
- Baking powder and baking soda (aluminium-free versions are available)
- Antacids and buffered medications

Aluminium and some of its compounds are used as indirect food additives. These are substances that can contact food and enter food products during growing, processing or packaging. They include adhesives, coatings, paper, paperboard components, polymers, adjuvants and production aids.

As a side note, aluminium exposure to humans is extensive, and continues in other sources such as

- Composite dental fillings and ceramic/porcelain crowns.
- Toothpaste.
- Algae growth toxins for municipal water, lakes, and reservoirs.
- Aluminium-containing antiperspirants and other cosmetics (easily absorbed through the skin).
- Industrial pollution, car exhaust, tobacco smoke, and fireworks.
- Preservatives and/or adjuvants in most vaccines.

In the US, the Federal Food, Drug, and Cosmetic Act currently accepts a GRAS designation as sufficient to ensure the safety of food additives. There is an absence of protection against conflict of interest. Furthermore, the FDA has inadequate authority to obtain data on chemicals added to food or review their safety. Studies on the effects of food additives on infants and children are generally missing; yet it is known that infants and children are more sensitive to

chemical exposures. It is clear that improvements to the regulatory system are needed in respect to food additives, including the replacement of the GRAS process, establishing a scientific foundation of the FDA and FSANZ assessment programs, retesting all existing additives, and labelling of direct additives with little or no toxicity data (Trasande 2018).

In only 370 of over 10,000 labelled as GRAS chemicals, the FDA Select Committee on GRAS Substances, carried out a post-review as to the safety of those substances and simply assigned one of 5 conclusions from any data on such substances that was generally published (USFDA 2020a). There were 5 standard conclusions they used with the best being:

There is no evidence in the available information on [substance] that demonstrates, or suggests reasonable grounds to suspect, a hazard to the public when they are used at levels that are now current or might reasonably be expected in the future.

The worst conclusion was:

In view of the almost complete lack of biological studies, the Select Committee has insufficient data upon which to evaluate the safety of [substance] as a [intended use].

Although the worst conclusion was used for 4% of them, the best conclusion is based-on no news is good news: if there are no studies done, then there is no hazard evidence so it must be OK. No comprehensive testing had been performed, let alone necessarily on humans (USFDA 2020b). Furthermore, as highlighted throughout this thesis, where some testing is done, each chemical is considered on its own, there is no evaluation of the possibility of synergistic toxicity from the various combinations that can be present.

There is evidence that even dietary patterns can be correlated with various diseases, for example, it has been noticed that when Japanese women change to westernised dietary patterns, an increase in the risk of breast cancer occurs (Shin 2016). Even dietary groups such as the 'no-calorie' sugar alternatives like aspartame and splenda, cause hypoglycemia and/or elevated levels of insulin. These can further stimulate appetite and molecular processes associated with fat storage and have carcinogenic and neurotoxic effects: as covered in a later chapter.

Aside from the intentionally added chemicals to food there are many examples where accidental contamination was eventually discovered. Some examples which illustrate the diverse nature of contamination:

- The Tyson company had to recall approximately 5450 tonnes of frozen, ready-to-eat chicken strip products due to metal fragment contamination. These fragments were highly toxic, with lead and other toxic heavy metals (Fottrell 2019).
- Testing surveys of leading brands of baby foods and formulas found arsenic, lead, acrylamide, cadmium, and BPA present (CLP 2020).
- A survey found, in 90% of samples of strawberries, spinach and kale, two or more pesticide residues. The most contaminated in order were strawberries, spinach, kale, nectarines, apples, grapes, peaches, cherries, pears, tomatoes, celery and potatoes (EWG 2020).
- Meats have been found to contain drugs banned for use in food animals. These included “Ketamine, a hallucinogenic party drug and experimental antidepressant; Phenylbutazone, an anti-inflammatory deemed too risky for human use; Chloramphenicol, a powerful antibiotic linked to potentially deadly anemia” (Peachman 2018).

An important theme that will be highlighted at the end of this chapter, is how environmental exposure in pre-conception, in-utero or early infancy affect the subsequent development of animals and humans. However there can be simple food nutritional effects without superposing xenochemical effects. Studies have been conducted on mice which adjusted their diets to be deficient in methyl group-donor content within the first 60 days of life (Waterland 2006). The effect was to lower their methylation process which persisted for the rest of their lives, even if their diet was changed to be nutritionally sufficient after 60 days. This illustrates how gene expression can be affected by diet.

These results taken as a whole suggest that early nutritional environments, whether they are in utero or in early infancy, can act to ‘set’ the range of possibility for gene expression for the life of the organism and perhaps that of its descendants....This is a model in which food enters the body and in a sense never leaves it, because food transforms the organism's being as much as the organism transforms it. It is a model for

how social things (food, in particular) enter the body, are digested, and in shaping metabolism, become part of the body-in-time, not by building bones and tissues, but by leaving an imprint on a dynamic bodily process (Landecker 2011p176).

Even food availability has generational effects. Significant findings in epidemiology show that disease incidence correlates with food availability. This has been shown in descendants of people experiencing a famine or an abundance of food: You are what your grandparents ate (Kaati 2002, Pembrey 2002, 2006). An example of this was seen from the Dutch famine of 1944. Those that were in-utero during that time had higher incidences of schizophrenia and diabetes.

It has been suggested that so-called 'racial' disparities in health start as socioeconomic differences or events and become embedded biologically through epigenetic mechanisms: stress and poor nutrition disproportionately affect some people's gene regulatory mechanisms according to the historically and culturally shaped striations of society (Kuzawa 2009)... What may look like genetic 'racial' differences between groups of people in something like diabetes incidence is recast as the physiological sign of a population that has recently undergone 'severe cultural and economic disruptions and nutritional stress' (forced relocation, indentured labour, poverty), followed by rapid transitions to Western diets and sedentary lifestyles, political violence that shapes present and future metabolisms (Benyshek 2007p14) This is a very specific form of naturalization of social change that recasts social suffering as molecularly heritable, the past borne forward into the future via a metabolic interface that modulates 'predictive signaling' to subsequent generations about the world they will be born into (Kuzawa, 2005) (Landecker 2011p194).

As can be seen from the above, subtle epigenetic mechanisms can affect the future generations. It will be seen through this thesis that there are many xenochemical exposures that produce significant epigenetic effects.

This section has simply illustrated that while food is generally considered as a modifiable input to one's health, the general state of public ignorance about what food can contain is unfortunate. Some examples have been touched on in respect to food toxins from indirect sources, those deliberately added, and 'accidental' contamination. The highly toxic heavy

metal, aluminium, was used as just one example of a toxin present throughout foods. Although there is much undone science on the effects of all the toxins found in food, examples showed that even for the seemingly insignificant effects of food abundance or nutritional content, there is evidence that significant effects are produced on future generations.

WHAT CAUSES CHRONIC DISEASE?

After referring to examples of environmental exposures, we will now consider their more generalised links with chronic diseases. Once again, this is a large topic with much undone science present, however as will be seen, there is already substantial evidence of such links. This section begins the process of more directly linking environmental exposures with health conditions.

The US Centers for Disease Control and Prevention (CDC) defines chronic diseases as “conditions that last 1 year or more and require ongoing medical attention or limit activities of daily living or both”. They mention also that 4 in 5 older adults will have at least one chronic condition, such as heart disorders, osteoporosis, arthritis, etc, and 50% will battle at least two.

The orthodox view of chronic disease is commonly one of risk factors (WHO 2006b). They are modifiable and the same in men and women: unhealthy diet; physical inactivity; tobacco and alcohol use and infectious agents. These risks then produce symptoms of raised glucose levels, raised blood pressure, abnormal blood lipids, overweight and obesity. Sometimes orthodox explanations of chronic diseases are only through the symptoms, such as overweight and obesity (AIHW 2017). In Australia 70% of cardiovascular disease mortality is linked to the combined effects of high blood pressure, high cholesterol and physical inactivity (Begg 2007). Modifiable risk factors, along with non-modifiable risk factors of age and heredity, are used to explain the increasing incidences of stroke, heart disease, chronic respiratory diseases and some cancers.

The incidence of chronic diseases has significantly increased over the past five decades, some of which are: autism, Alzheimer’s, Chronic Obstructive Pulmonary Disease, diabetes, sleep apnea, ADHD, celiac disease, chronic fatigue syndrome, asthma, depression, multiple sclerosis, bipolar disease in youth, hypothyroidism, lupus, inflammatory bowel disease, fibromyalgia and

osteoarthritis. The cause of, or a pathogen for, each of these diseases has not been established. So why would the orthodox risk factors for such diseases be increasing? As the general human genome has not significantly altered in this time, environmental exposures are highly suspect (Rappaport 2010, Bijlsma 2016). Consider for example, obesity: in the U.S., the prevalence of obesity was 42.4% in 2017-2018, up from 30.5% in 1999-2000 (Hales 2020). It has been suggested that if America doesn't change its eating habits, over half the nation will be obese within 10 years (Ward 2019). In looking for explanations, environmental exposure links with obesity are present in hundreds of scientific articles.

Orthodox views on the causes of chronic diseases puts blame on the sufferers in their lifestyle choices. Subsequent drug treatment for these conditions commonly worsens the individual's situation, especially long term. Take for example, the use of decongestant medications like pseudoephedrine and nonsteroidal anti-inflammatory drugs (NSAIDs) like ibuprofen. These drugs can damage the heart, especially with risk factors for cardiovascular disease. NSAIDs and decongestants increase blood pressure, which is generally regarded as producing increased risk for stroke and heart attack. People who take NSAIDs, to manage a respiratory infection, had a three times higher risk of heart attack compared to other times when they weren't taking NSAIDs (Wen 2017). Yet many people find themselves taking multiple medications. The top ten prescription drugs are all for diseases that ecologically aware physicians recognise as having an environmental aetiology (Crumpler 2014).

The orthodox view does not explain incidences of cancer in many health-conscious individuals in the middle class of developed countries, or what causes the low birth weights of babies where the mothers had reasonable diets, or the increasing incidence of autoimmune diseases in developed countries rather than the undeveloped ones.

The term metabolic syndrome is increasingly being linked with heart disease, diabetes, and stroke: high blood pressure, blood sugar, body fat/weight, and cholesterol. It has been observed that people with three or more abnormally high biological risk factors are more likely to experience these health conditions compared to those with one or two. So metabolic syndrome is becoming recognised as a legitimate, diagnosable, and treatable disease (Hatch 2014). But why do these biological risk factors occur: what causes the risk 'factors' themselves?

Mercury is a prime example of a toxicant disrupting metabolic pathways. Whilst being able to disrupt a variety of important enzymes, it can disrupt the methylation process in cells. Methylation is required for over 150 important metabolic processes, including DNA repair, therefore mercury not only disrupts this generation, but others as well.

A common preoccupation of orthodox disease research is the paradigmatic viewpoint that germs are the human body's enemy combatants and are the root cause of most detrimental human conditions. This is the cornerstone for the substantiation of the development of drugs and vaccines. Yet with the realisation of the role of the microbiome and virome in health, especially the immune system, with its gastro-intestinal links, one can have an ecologically-based view of the inseparability of the body and its environs. This is further reinforced when one considers the essential human building blocks of mitochondria, human DNA itself, and cellular exosomes exhibit many viral features.

Common environmental xenochemical exposures such as to chlorine, heavy metals, pesticides, antibiotics, etc. are not only toxic to human cellular structures but also to the human biome. The human biome consists of the thousands of microscopic organisms which reside in the skin, vagina, sinuses, and most significantly, the gastrointestinal tract. Most of these organisms are essential to good health of the body they inhabit. They play an essential part in many processes such as release of neurotransmitters, proper digestion, nutrient production, immune system modulation, detoxification, etc. Furthermore, environmental xenochemicals activate the immune system.

The indirect effects of environmental exposures, when the immune system becomes dysregulated, or hypersensitive from environmental toxins, are associated with profound metabolic effects. There are many examples of induced hypersensitivity, such as linking prenatal maternal exposures to IgE related allergy incidences in children (Peters 2013). Hypersensitive states have been also found in subsequent generations following industrial chemical plant disasters such as the Bhopal tragedy (Mishra 2009).

Centuries ago Hippocrates put forward that health and wellbeing are profoundly affected by the individual's diet, lifestyle and environment (Kleisiaris 2014). It was mentioned in the introduction that in 2012, the World Health Organization estimated that 4.9 million deaths and

86 million disability adjusted life years (DALY)⁴ could be due to environmental chemicals (Bijlsma 2016, UNEP 2012). Furthermore about one-quarter of the global disease burden, and greater than one-third of the burden among children under the age of five, have been attributed to modifiable environmental factors (Bijlsma 2016, WHO 2006c).

The extent of enquiry needed to define the root causes of health problems caused by environmental exposures is well-beyond an orthodox 15 to 20 minute doctor visit, or pathological testing. If for example, the immune system is dysregulated due to persistent environmental influences, the cause of the chronic disease can be missed (Winans 2011, Vos 1989).

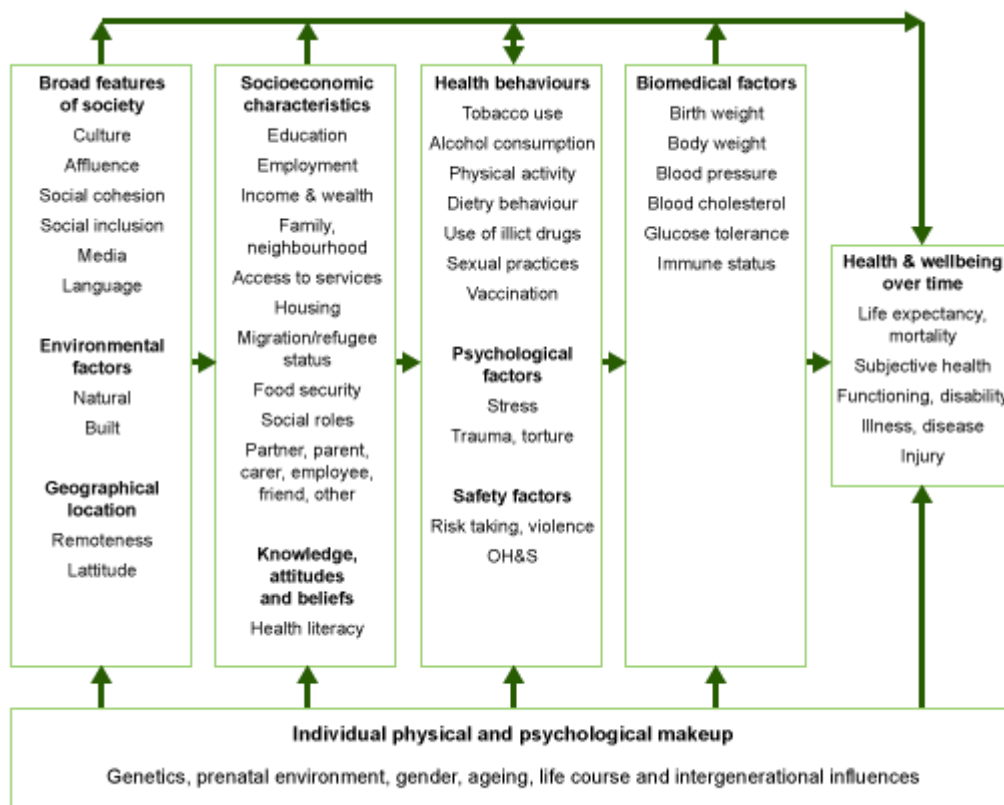
Recent studies on epigenetics and new terms such as “exposome” (total exposures over a lifetime) are increasingly being associated with chronic disease (Paoloni-Giacobino 2011). These represent a threat to current paradigms for chronic disease aetiology as it is gradually recognised that infectious and chronic diseases are primarily encountered in developing countries (Laborde 2015, Haggerty 1975, PAHO 2017) and chronic diseases are most commonly encountered in developed countries (WHO 2020). This has led to generalisations that “all of the chronic, debilitating illnesses that are in epidemic proportions now are all directly related to the build-up of environmental toxicants [in] our body” (Crinnion 2016, 00.08.00).

In the US, more than 50 years ago, diabetes incidence was less than one percent. Current projections are that one-third of the population will get diabetes in their lifetime (Pizzorno 2020). In searching for reasons for this increasing incidence a common orthodoxy will attribute to increased sugar consumption, however that peaked at least a decade before the diabetes epidemic started. In considering obesity however, it is found that obese women, have a 20 times increased risk for diabetes. From one practitioner’s experience in treating obese people, 10% with the lowest environmental toxin body load, do not have increased risk for diabetes. Whereas for the top 20% of obese people having the highest body loads: 60% of these already have diabetes (Pizzorno 2016, 2020). The trend appeared to correspond with blood levels of chlorinated pesticides from residues in food. The resultant hypothesis was that because these toxins can occupy cellular insulin receptor sites, the cells are blanketed to the effects of insulin.

⁴ One DALY represents the loss of the equivalent of one year of full health. DALYs for a disease or health condition are the sum of the years of life lost due to premature mortality and the years lived with a disability due to prevalent cases of the disease or health condition in a population (WHO definition).

This produces insulin insensitivity metabolic syndrome. As a result the pancreas starts ramping up insulin production, but cannot keep up such long term production and gradually burns out (sometimes after 20 to 30 years). As a result, diabetes eventually develops. The real tragedy is that babies are now being born with such pesticide levels and there are rising childhood obesity levels.

It is acknowledged that there are many definitions for ‘environmental’ influences on chronic disease. From below diagram, many researchers would classify most of the included factors as ‘environmental’. In this thesis however, there will be a focus on environmental exposures from industrial and commercial product origins



Source: (AIHW 2016). *Risk factors contributing to chronic disease.*

From the above, there are two listed major influences that will be later shown to be environmentally related: “Individual physical and psychological makeup” and “Biomedical factors”. Endocrine disrupting chemicals, can cause the ‘intergenerational influences’ for example, and will be touched-on in the next section. Specific risks can also accumulate over

time and if not modified, age becomes a marker for chronic diseases. Chronic diseases and poverty/class and race are circularly interlinked (Mohai 1992, White 1992, Hird 1993, Zimmerman 1993, GAO 1995) also suggesting a relationship with environmental hazards (Brown 1995). Poverty can be caused by chronic diseases, but the poor usually have greater exposure to environmental risks and limited health services. It is acknowledged that psychosocial stress can contribute to this.

The health impact of xenochemical exposures is evident in some areas of paediatric medicine where chronic diseases rather than infectious diseases, are the most common (Genuis 2010). Paediatrics was the first medical discipline to regard chemical exposures as an important issue. The American Academy of Paediatrics established an environmental health committee in 1958, publishing its first edition of Paediatric Environmental Health for Clinicians in 1999 (Etzel 2011).

As a primary causative determinant of chronic disease, toxicant exposures induce metabolic disruption in myriad ways, which consequently result in varied clinical manifestations, which are then categorized by health providers into innumerable diagnoses. Chemical disruption of human metabolism has become an etiological determinant of much illness throughout the lifecycle, from neurodevelopmental abnormalities in-utero to dementia in the elderly (Genuis 2017p477).

...metals and chemicals in our environment have now become the primary drivers of chronic disease throughout the world, but particularly the industrialized worlds (Pizzorno 2020).

Most people follow a diet that “includes not only excess sugar and refined carbohydrates but also industrially processed foods, chemical additives, GMO foods, and hybridized foods. This combination of such diets and “chronic exposures to pollutants, toxins, medications, and inflammatory foods” contributes “to the incidences of autoimmunity, autism, neurodegenerative diseases, and other chronic inflammatory conditions that result from heavily industrialized, immune-reactive environment” (Bilbo 2018p242, Furman 2019).

Genetic factors have generally been acknowledged as only playing a minor role in chronic disease (Rosenfeld 2015, Sutcliffe 2008). But there is evidence linking toxicant exposure and bioaccumulation with induced hypersensitive states such as various allergies through to multi-

morbidity states such a Multiple Chemical Sensitivity (MCS). MCS will be used in subsequent sections as an example of an emergent disease, strongly environmentally related, in respect to how the medical and legal systems treat such a disease.

There is extensive evidence that environmental exposures before birth and in early childhood affect health in adulthood. Low birth weight, for example, has been linked with increased rates of high heart disease, blood pressure, stroke and diabetes. Later in this chapter, it will be seen that various environmental exposures are linked to low birth weight causation.

It will later be shown in this thesis, that each environmental influence on human health can be magnified by other environmental influences. At most times, people are exposed to a myriad of environmental exposures, food additives/contaminants, air fresheners and electromagnetic fields (wi-fi, mobile phone towers, etc). Even the condition of one's gut microbiome will affect the toxicity of many pollutants (Claus 2016).

This section has illustrated how the orthodox views of the causes of chronic disease are at a pathological level, for example, high blood pressure. This view then forms a paradigmatic limit since there is no further enquiry as to what has caused these pathological conditions/symptoms to originally develop. The next two sections will consider two major chronic health conditions. In considering environmental exposures it will be shown that despite much undone science in the area, there are studies that have found clear links with these chronic conditions.

Autoimmune Diseases

Autoimmune diseases include more than 80 diseases such as rheumatoid arthritis, multiple sclerosis, lupus, celiac disease, diabetes, thyroid disease, ulcerative colitis, Crohn's disease, inflammatory bowel disease, Guillain-barre syndrome, inflammatory demyelinating polyneuropathy, psoriasis, myasthenia gravis, vasculitis, Hashimoto's thyroiditis, dementia and Alzheimer's. A whole spectrum of disorders are seen often as very separate things, but autoimmunity has common roots in that it is the body attacking itself, and there must be a reason for this.

This section firstly considers the orthodox explanations for the causes of autoimmune disease. As genetic susceptibility and improved diagnosis are universal explanations from orthodoxy, these are firstly dismissed through a number of studies. Similarly the links between clear infections (bacteria, fungi, viruses and parasites) and autoimmune diseases do not seem to have significance in their own right, but could be a contribution. The new food plant and animal varieties, genetic modifications, chemical ingredients, flavours, and preservatives are shown to have causal effects. Their links with leaky gut seems to be a well-recognised precursor to an autoimmune condition as recognised by the functional medicine practitioners. It is to such an extent that orthodox research is now studying leaky gut. Gluten, wheat germ agglutinin, and glyphosate are particularly considered in subsections of this chapter, all tied in with leaky gut issues. Other xenochemicals such as BPA, heavy metals, as well as vaccines are mentioned for their links with autoimmunity. Finally there is a consideration of the shortcomings of the orthodox treatment of the autoimmune diseases.

Autoimmune diseases have substantially increased in the last 25 years. Estimates of the prevalence of autoimmune diseases vary from one in 15 people to one in three being affected. At the turn of the 20th Century, it was about 1 in 10,000 individuals (Brady 2017). In the US, “autoimmune diseases are among the top 10 causes of death in female children and women in all age groups up to 64 years of age. The US National Institutes of Health estimate autoimmune diseases cost an estimated \$100 billion a year in medical care” (BRI 2020).

Though there are many aspects of the pathological mechanisms that remain unknown, the small amount of knowledge and orthodox risk factors in respect to autoimmune diseases indicate they have a common origin (Anaya 2012a,b). The occurrence of more than one autoimmune disease in an individual is well documented and termed polyautoimmunity (Anaya 2012a, Rojas-Villarraga 2012). One autoimmune problem is usually followed by another in sufferers.

The orthodox explanations of autoimmune diseases vary as follows:

The exact cause of autoimmune disorders is unknown. One theory is that some microorganisms (such as bacteria or viruses) or drugs may trigger changes that confuse the immune system. This may happen more often in people who have genes that make them more prone to autoimmune disorders (NIH 2020b).

Autoimmune disease occurs when, instead of attacking bacteria, viruses or other sources of infection, the immune system attacks healthy organs and tissues. It's not known why this happens, although autoimmune conditions most often affect people with a genetic predisposition. An environmental factor such as an infection, stress, medication, diet or even ultraviolet radiation then triggers the symptoms of the autoimmune disease (DoH 2020).

The causes of autoimmune diseases are unknown. In many cases there appears to be some inherited tendency. Other factors such as infections and some drugs may also play a role in triggering autoimmune diseases (ASCIA 2020).

In respect to the role of genetic susceptibility, there are studies finding that genetics can account for only a minority of autoimmunity cases (Rosenfeld 2015, Sutcliffe 2008). Disease discordance exists in monozygotic twins (Bogdanos 2012). Due to such clear findings in this regard, environmental factors in autoimmunity including xenochemicals, infections, and dietary components are considered here reflecting the remaining orthodox explanations.

In 2006, a WHO report attributed the “induction, development, and progression of autoimmune diseases” to “..intrinsic factors (e.g. genetics, hormones, age) and environmental factors (e.g. infections, diet, drugs, environmental chemicals)” (WHO 2006d p1). The report dismissed the attribution of the rise in incidence to better diagnosis. It called on doctors, public health authorities, and government agencies to be aware of increased autoimmune disease incidence from exposure to physical and chemical agents.

The immune response to clear infections is significantly different to a dysregulated response as in autoimmune and immune-mediated diseases (Vojdani 2014, Kuchroo 2012, Bayry 2007). There is however some evidence of some synergism with xenobiotics and xenochemicals. The activation of toll-like receptors by xenobiotics can predispose individuals to toxicant induced inflammatory cytokine production, progressing into autoimmune diseases (Selmi 2012). Otherwise further links between clear infections (bacteria, fungi, viruses and parasites) and autoimmune diseases do not seem to exist as being of any significance.

People's diet today are mostly quite different from three or four decades ago. There are new food sources, new food plant and animal varieties, genetic modifications, chemical ingredients,

flavours, and preservatives. The increase in the incidence of autoimmune diseases such as diabetes and multiple sclerosis (MS) in developed countries over the same period has led to postulations that diet is an environmental risk factor. The association between gluten ingestion and gluten sensitivity is established and accepted (Miller 2012). High dietary sodium level is associated with high blood pressure, cardiovascular health concerns (Brown 2009b), and immune system effects (Rose 1988). Low levels of vitamin D have been associated with MS, systemic lupus erythematosus, rheumatoid arthritis and other autoimmune disorders (Miller 2012). Lactose intolerance and other milk-related disorders are also present. The overlap with leaky gut as a contributing mechanism in these intolerances and autoimmune precursors will be subsequently covered.

The induction of autoimmune conditions by toxicants has been covered by Pollard et al. (2010) in their paper, "Toxicology of autoimmune diseases." An example used was a meta-analysis (Barragan-Martinez 2012) which linked systemic sclerosis, primary systemic vasculitis, and MS with exposure to organic solvents.

Modification of DNA methylation is also a mechanism where environmental triggers induce changes in gene expression. Trigger examples include environmental pollutants, cigarette smoke and alcohol consumption (Baccarelli 2009, Bigazzi 1997).

In the US alone, over about 20 years there has been a doubling of chemicals produced and imported, with the current rate being about 33 million tonnes per year (O'Brien 2017). With these xenochemicals now being found to be accumulating in the environment, and humans, it is not surprising that the immune systems have become increasingly active in general. It has been suggested that the "autoimmune disease epidemic is really just an adaptation to a new environment" (O'Brien 2017ep5).

The digestive system includes a large portion of the immune system. Some attribute two thirds of the immune system being intricately linked with the digestive system (Lipski 2017). The intestinal lining primarily consists of one cell thickness, separating food from the blood supply. The space between these cells, called enterocytes, relates to intestinal permeability. Many environmental influences have been found to directly or indirectly affect the permeability. Once there is leakage between these cells, substances from inside the intestine can enter the blood stream, where they should not be. The immune system then sees these foreign

substances in the blood and organs. It then starts to mount an immune response typically leading to inflammation. Xenochemicals can be part of this leakage. Many of these, such as mercury and lead, can attach themselves to proteins in human tissue. The immune system then starts attacking such tissue and produces an autoimmune response. The consistency in the relationship sees functional medicine practitioners in observing autoimmune symptoms, then assuming that there is an intestinal permeability problem (Malterre 2017, Fitzgerald 2017). A general functional medicine understanding of autoimmune disease appears to be that it has three foundations: environmental stressors, gene expression mechanisms, and leaky gut.

In a screening process for students for clinical trials on use of probiotics for reducing permeability, one research group found that a single challenge meal (McDonald's breakfast), induced leaky gut in 65% of the students. "They are seemingly healthy, but every single time they eat food, especially the bad food and things like gluten, you're getting this huge toxic response in your body that you don't feel. You feel fine, but it's damaging tissue. It's messing up your immune system. It's confusing the immune response, and every single day of doing that over the next 10 to 15 years, sets you up for every kind of autoimmune disease in your mid 30's, 40's, and 50's" (Krishnan 2017p16).

Once a leaky gut has occurred, different foods leaking through into the bloodstream can trigger a variety of inflammatory responses (Hyman 2017). Indeed, "leaky gut" is attributed to a variety of problems including Crohn's disease, ulcerative colitis, inflammatory bowel disease, fatty liver disease and type 2 diabetes. Some researchers link leaky gut more generally to autoimmune, infective, metabolic, and tumoral diseases (Fasano 2020). Western medicine has been slow to accept leaky gut as a medical syndrome, although this seems to be changing. Many studies are now focussed on this aspect. One recent finding (Raftery 2015) conducted with Crohn's disease patients shows that a hormone (vitamin D) can substantially improve leaky gut. Vitamin D is already receiving kudos from researchers for its potential to prevent various cancers, heart disease and serious respiratory conditions such as pneumonia.

A similar end result to a leaky gut can occur with vaccines, which are injected directly into the bloodstream avoiding the normal bodily barriers to foreign substances. The damage can be magnified in babies and infants whose immune system is not fully developed. Take for example, the human flu vaccine antigen. This is produced using insect and chicken egg material.

The vaccine virus particles will therefore contain foreign proteins causing unpredictable immunological human responses: different than would be expected from influenza viral particles on their own. One possible outcome is that the foreign proteins could produce antigens that cross-react with self-structures resulting in autoimmunity. Present safety testing does not test for such cross reactions. The potential inter-related induced problems are undone science. It was only recently found that "influenza" is very dependent upon a host for its transmissibility and immunogenicity. This follows the findings of the first ever study to ascertain what is the composition of an influenza virus (Hutchinson 2014). Considering the decades of vaccine use and promotion, it is surprising that the full definition the proteins contained and their origin was not previously achieved. Billions of dollars annually have been invested into flu vaccines to fight a viral enemy whose basic building blocks were not known until a few years ago.

Comorbidity

Although we are primarily concerned with what can cause, or initiate autoimmunity from an environmental viewpoint, it is pertinent to consider, as mentioned previously, that it is common for a sufferer to have more than one autoimmune disease. It is also possible to see multiple symptoms across different organs depending upon the person's genetic characteristics. There is much medical literature that people who have diabetes have a high prevalence of other autoimmune diseases (Krzewska 2016, Alves 2016). One popular theory concerns the fact that in diabetes there is excessive sugar present in the blood than what cells absorb for fuel (Bland 2017a). The excess sugar then starts reacting to various bodily tissue proteins producing glycation on the surface of cells. The glycosylation end products, AgE proteins, are then seen as foreigners by the immune system. The immune system then attacks the cells coated with such.

Gluten and Wheat Germ Agglutinin (WGA)

These substances are covered here since they are essentially substances that the human digestive system cannot deal with due to not having the organs to digest them nor the required enzymes. They become environmental exposures due to their clear presence in today's

environment. Although most xenochemical exposures are not realised by the public, gluten has finally risen in prominence in the last decade due to many people seeing short to medium term health effects from its withdrawal from their individual diets. But, as will be seen in this section, there is also the WGA which has had little research on its health effects, and from the little knowledge available, is potentially another concern in general in wheat based diets.

The human digestive system is quite different to ruminants, so is not suited to grasses, or the seeds of grasses, or field grass grains. These have been introduced into the diet, be it hundreds of years ago, an insignificant time in respect to evolutionary spans of millions of years, and increased in concentrations since, with hybridisation. Although these have mostly been studied on their own, these in-combination with the thousands of other environmental effects on health must be considered in respect to potential potentiation or synergism which simply have not been studied.

It has been found that the presence of gluten on the intestinal wall (epithelium) stimulates the body to produce a substance called zonulin (Fasano 2011). Zonulin causes the epithelium cells to contract to produce gaps rather than tight junctions between them. This is a bodily process normally utilised to quickly flush an intestinal area, washing the foreign substance away. This was first found as an automatic reaction inside the intestines to the presence of cholera protein complex and explained the amount of liquid present in the resultant diarrhoea. While there is much undone science on this subject, it seems that in certain people with an epigenetic predisposition, the zonulin production continues. This results in the permeability between the epithelial cells which separate the intestinal contents from the surrounding blood vessels. This results in many foreign substances entering the bloodstream initiating an immune response to clean them up. Increased permeability is seen from radiation, chemotherapy and other toxins from environmental exposure.

Many autoimmune diseases have been found to involve alterations in intestinal permeability. Some include type 1 diabetes, multiple sclerosis, and rheumatoid arthritis. The permeability is also involved in cancer development, infections, and allergies (Fasano 2011).

It is generally accepted that it is the interplay between environmental factors and specific susceptibility genes that underlies the aberrant immune response responsible for the onset of these diseases. Less than 10% of those with increased genetic

susceptibility progress to clinical disease, suggesting a strong environmental trigger in the pre-disease state (75, 152). Environmental factors are also likely affecting the outcome of the process and the rate of progression to disease in those who develop pathological outcomes (Fasano 2011p160).

Wheat has been hybridised for yield and aside from being now able to grow 8 to 10 times the tonnage from the same area of soil than the original wheat varieties, it has also been bred for pest and mould resistance. This has been achieved by increasing wheat germ, gluten and phytate content. Phytates bind (take-out) zinc, potassium, calcium, magnesium and iron, all critical for many processes in the body.

In functional medicine, many practitioners seem to regard gluten as being indigestible in a human being. “some people can react to this very strongly, some mildly and some are not feeling anything at all. But it doesn’t mean, that if you don’t feel it, it doesn’t mean it’s not harming you” (De Carvalho 2017). There is a growing body of evidence suggesting that autoimmunity may result from molecular mimicry between gliadins (a component of gluten) or transglutaminase enzyme tissue and various tissue antigens, including nervous system proteins (Kaufman 1992, Sadeharju 2001, Calcinaro 2005, Davies 2008). The gliadins can also amplify opioid effects on the brain. Depending on the tendencies of the host, these can then magnify problems, for example, cause auditory hallucinations in schizophrenics, behavioural outbursts and reduced attention spans in ASD people, trigger mania in bipolar illness, etc. (Davis 2020). More on gluten can be found in a later chapter on an ignorance topic.

Traditional antibody testing registers proteins that cross react with human tissues, but there is a potential elephant in the corner that is missed. Wheat germ agglutinin (WGA) binds to human tissue and by binding to tissue it causes autoimmunity against that tissue, and because it doesn't belong there the immune system, in trying to get rid of it, ends up destroying tissue in the process. There is no genetic susceptibility or immune system antibody status required for WGA to do its damage. This could explain why chronic inflammatory and degenerative conditions are common in wheat-consuming societies when gluten allergies or intolerances are not apparent.

There are pathogenic similarities between WGA and certain viruses. They are of similar size and both enter cells through endocytosis. The influenza virus and WGA do this via the sialic acid

coatings on the cell membranes. WGA's ability to affect cells through these same pathways has been noticed in competitive cell binding studies (Weis 1988, Finlay 1987). Once viruses enter cells, their incorporation into the genetic material for reproduction starts to blur the line between self and non-self in the virally transformed cells. To clear the infection, the immune system must attack its own virally transformed cell. As WGA antibodies have been shown to cross react with other proteins (Tchernychev 1996), even if the phenotype of cells is not changed into "other," the cross-reactivity of the WGA antibodies with normal cells can produce autoimmunity.

Plants do not have the cell-mediated immunity of some insects or an antibody driven secondary immune system like humans. They have a simple system for their immune defence. Grass family seeds, e.g., wheat, rice, rye, spelt, use high levels of defensive lectins. Unfortunately, lectins are resistant to a wide range of pH and temperatures, so normal cooking, fermentation, sprouting and digestion processes are generally ineffective in modifying them. WGA lectin has the same disulfide bonds that are present in vulcanised rubber and human hair, so are strong and durable. Since it is such a powerful insecticide, biotech companies have used it to create genetically modified WGA-enhanced plants. The human intestines allow the passage of molecules up to 1000 kilodaltons through its walls: WGA is only 36 kilodaltons in size so can easily pass through.

Glucosamine is frequently used for reducing pain and inflammation. We have no dietary deficiency of shells of crab, lobster or shrimps made into such a powder. However, WGA will bind to such substances thereby reducing the load on the human cells and relieve symptoms thereby giving glucosamine its usefulness. NSAIDS are also frequently prescribed. The affected individual however, may be better served by removing the WGA sources from their diet: attention the cause rather than treating the symptoms.

Even in celiac disease, WGA seems to play a role totally independent from gluten. Significantly higher levels of IgG and IgA antibodies against WGA have been found in patients with celiac disease. These antibodies do not react with gluten antigens (Sollid 1986, Fälth-Magnusson 1995).

As WGA can readily pass into the bloodstream, it can flow to the brain where it can also easily pass the blood brain barrier (Broadwell 1988) pulling bound substances into the brain with it

(Damak 2008). This ability is utilised in researching ways to deliver drugs into the brain. WGA in the brain can cause extensive damage leading to neurodegenerative disorders, affecting myelin sheaths (around nerves) and inhibiting nerve growth factor (Dolapchieva 1996, Hashimoto 1989).

WGA can cause weight gain and insulin resistance (Yevdokimova 2001), as well as other endocrine system effects such as attaching itself to sperm and ovary cells. It has affinity for thyroid tissue (Sasano 1989), can cause pancreatic hypertrophy, increased risk of cancer, and the list goes on for a variety of effects. In respect to gastrointestinal effects, relevant in making this area more susceptible to other environmental exposure effects: it affects the epithelial membrane, reduces villi surface area, causes cytoskeleton degradation in intestinal cells, and decreases quantities of heat shock proteins (Ovelgönne 2000).

As can be seen from gluten and WGA's varied effects, their actions can aid the passage of many other environmental xenochemicals across the intestinal wall and in even crossing of the blood-brain barrier. Their induction of immune system reactions and over-reactions adds to the immune system load in trying to handle the millions of xenochemicals that the human body is exposed to. Their direct connections with autoimmune conditions highlight another concerning environmental exposure in the food system.

Glyphosate as a Pesticide Example

Following on from the above section on wheat, a herbicide, glyphosate, has been used extensively in cropping, as well as a desiccant commonly applied to harvested grains. As a result, many agricultural foods have glyphosate residues present. This transfers to the food chain resulting also in human glyphosate residue levels. It is also extensively found throughout the environment in general. It has been chosen simply to comment on one of many pesticides and its health effects, particularly here for autoimmunity.

Glyphosate's story in becoming a pesticide for food crops is typical of many in that it was originally used in a different industry. In glyphosate's case, it was a chelating agent in pipes, boilers etc. to clean the inside of metal surfaces. After being repurposed by Monsanto as a herbicide, the company promoted it as a safe chemical since it only affected plants by

interfering with their shikimate pathways, which humans did not have. However, its antibiotic properties causing this pathway interference affected the human microbiome where all such bacteria had this pathway. But not only that, its chelating properties bound minerals in soil and plants making them unavailable. It also obliterated the soil microbiome which affected plant nutrient content, and stopped a source of microbes that we need to colonize the gut.

The antibiotic and nutrient sequestering properties of glyphosate affect the human gut in similar ways, thereby indirectly suppressing the immune system.

Generally, once consumed, glyphosate can cause many problems such as suppression of enzymes in the liver used for detoxification, and detrimental effects on metabolic transport mechanisms underlying important biochemical pathways. Its link with many autoimmune diseases is hypothesised to be via molecular mimicry (Samsel 2017, Samsel 2013).

Another source of glyphosate exposure to humans is through vaccines (Samsel 2017). The live measles virus, for example, can produce glyphosate-containing haemagglutinin, which then can cause an autoimmune attack on myelin protein, typical of autism.

While many studies have focused just on glyphosate, the normal product mix, Roundup, of which glyphosate is a minor component, appears to be 1000-fold more toxic than glyphosate alone (Seralini 2017). “There was a fraud in the declaration of the active ingredient. There are hidden poisons in Roundup which are, in fact, some petroleum distillate residues, burned, oxidized a lot, and that will irritate any kind of membrane in the cells, like the gut membrane, but also the kidney at the epithelium, and that will allow the penetration of the pollutant inside the body” (Seralini 2017,00:10:00).

This section has simply used one pesticide example and considered some of the evidence linking it to health effects. As has been seen, this particular pesticide has many health effects.

Other Environmental Xenochemical Health Effect Examples

There are millions of toxic xenochemicals in the environment that one could consider in respect to autoimmunity effects as well as a myriad of other health conditions. Here however let's

close this section by mentioning some other diverse examples to show the diversity of causes focusing on autoimmunity.

Bisphenol A (BPA) is a chemical used to make hard plastic items, like re-useable water bottles, food containers, baby bottles, pitchers, tableware and other storage containers, eyeglass lenses, CDs, DVDs, computers, appliances, sports safety equipment and many other products. It is used in epoxy resins in food can inside coatings, bottle tops and water supply pipes. Small amounts of BPA are released into the contacting food and beverages especially through storage and heating. Infants can have considerable exposure to BPA since infant formula usually utilises epoxy-lined cans and the warming of baby bottles increases leaching from the plastic. BPA has been found in human breast milk. It is also present in indoor air and dust, dental sealants and numerous other products.

BPA disrupts the endocrine system and can bind to proteins, thereby creating a trigger for autoimmunity (Kharrazian 2017).

In diverse environmental exposures, such as the workers who worked at ground zero, after the 9/11 destruction of the twin towers, many of the workers developed autoimmune diseases. They were exposed to many different metals, such as aluminium (Shoenfeld 2017, Webber 2016). It has long been known that heavy metals such as lead, cadmium and mercury affect the immune system (Koller 1973, 1980).

Although industry and orthodox medicine would argue that smoking is a personally imposed exposure, it is pertinent to point out its impact on the immune system. It can also induce the immune system to react toward the body's own organs. In smoking, the proteins in the alveoli in the lung change to an extent that they are not recognized anymore as self-tissues, and the immune system attacks them (Shoenfeld 2017). There is a diversity of the xenochemicals present in smokes.

Autoimmune cross-reactivity has been seen to be induced by vaccines. The cross reactivity has been linked with narcolepsy, Guillain-Barré syndrome, multiple sclerosis, demyelinating neuropathies, systemic lupus erythematosus, and postural orthostatic tachycardia syndrome (Segal 2018).

Rather than researching what induces autoimmunity, a recent study found that EMF shielding improved the autoimmune disease conditions of the people studied (Marshall 2017).

As can be seen in considering a sample variety of autoimmunity causal links studied in the literature, the potential of a wide variety of environmental exposures to cause autoimmune diseases is significant.

Shortcomings of Orthodox Treatment on Autoimmune Diseases

It is pertinent here to utilise quotes from practitioners having many years' experience in treating autoimmune diseases. These are from interviews in the Betrayal documentary series aired in 2017, specifically on autoimmune disease. Whilst the statements are reflected in the scientific literature, the frank, and to the point comments were valuable for use. They highlight the orthodox non-recognition that environmental exposures can be causes of the diseases and the orthodox approach of treating symptoms only.

...our body actually doesn't respond to itself. It's responding to something that happens to our body that makes it a non-self. Now, that is a paradigm shifting concept (Bland 2017b, 00:08:30).

Unfortunately, most common therapies are designed to suppress or block the body's own immune system instead of figuring out why it's so pissed off in the first place (Hyman 2017, 00:05:00).

Any intelligent person could analyze this from the perspective that if we only mask symptoms, and in the process of masking symptoms, we create new disease with the drugs we use to treat those symptoms. Autoimmune is notorious for this. Look at the drugs used, the immune-suppressing drugs that are used to treat lupus, and rheumatoid arthritis, and psoriatic arthritis, it cause[s] cancer. It's very clear on the warning label. Very clear, they cause cancer. They shut down the immune system (Osborne 2017, 00:59:30).

...debilitating diseases like rheumatoid arthritis or asthma or multiple sclerosis or eczema or schizophrenia can actually turn around by taking gluten and wheat out of

your diet. It happens every day. Thousands and thousands of people (O'Bryan 2017 00:18:18).

A leaky gut puts you at a serious risk of any of the terrible 80 estimated autoimmune diseases, and the new ones that are being labelled...There is a connection between leaky gut and arthritis... It's not just toxins, but it's also about what you're not getting. Getting vital vitamins is essential for having your body function properly and optimally (O'Bryan 2017,00:18:18, 00:25:30, 00:38:16).

There's been a 25% increase in the last 15 years in the presentation of inflammatory bowel disease. This is absolutely a reflection of the loss of relationship between the environment which lives inside them and the human that's possessing that environment. People have become incapacitated, co-dependent upon strong pain killers or different types of medication, and a result of that always leads to a compression of life choices (Ash 2017, 00:01:30).

The above comments reflect a cross section of practitioners who treat people suffering autoimmune diseases. They have been specifically interviewed due to their various successes in treating autoimmune diseases. Unfortunately, most come from the functional or integrative medicine side rather than from the orthodox medicine side which typically is successful only in treating the symptoms short term before other complications ensue. Orthodox approaches do not consider environmental exposure potentials.

Cancer

There have been billions of dollars spent in trying to find a cure for cancer over many decades. The treatment of cancer is itself is a multi-billion-dollar industry worldwide. High technology treatments such as radiation therapies and patented drugs are the orthodox treatments, despite the presence of thousands of suppressed studies on successful dietary modifications and treatments, such as mushrooms, celery seeds, cannabis oil, etc. (Zhang 2009, Gao 2011). The dominant orthodox cancer treatment of chemotherapy has a 3% success rate in the US (Johnson 2020). It is interesting that the side effect of most chemo drugs is cancer.

Little is spent on researching the causes of cancer with the view of instigating preventative measures or even simple public awareness measures for other than a handful of exposures including smoking and skin cancers. The US National Cancer Advisory Board was highlighted decades ago for its board of 23 members as not having one member that had experience in preventative medicine or epidemiology to which Epstein summed up as “Billions for cures. Barely a cent to prevent” as a title for his paper (Epstein 1984).

This section will consider cancer as another major health condition with potential causes from environmental exposures. It will briefly consider three xenochemical exposures and their cancer links.

The history of explanations provided for the causes of cancer by the orthodoxy has been quite inconsistent (Proctor 1995p32):

- In the 1880s and '90s the consumption of tomatoes was regularly offered as the cause of cancer
- In a ridicule of the above it was suggested that increased sanitation saw people living longer and therefore more chance of succumbing to cancer
- The director of the New York Institute for Malignant Disease in 1908 proposed the theory that the distribution of cancer followed the distribution of Trout (fish)
- In 1915 it was proposed that “excessive nutrition” was linked to cancer.
- The above gradually developed by 1937, to the consumption of canned and preserved foods, white bread, meats, sweets, and other foods connected to constipation, ulcers and obesity.

In looking at the current state of knowledge on the causes, there is a lack of incorporation of the many potential environmental triggers shown in the literature. Below is a typical cross-section of explanations from health authorities of what causes cancers.

Most mutations are spontaneous, with no real reason for the change, however there are certain things that can cause mutations or make cells with mutations more likely to become cancers. These include:

- damage to cell DNA
- radiation, including ultraviolet radiation from the sun

- chemicals in tobacco smoke
- substances produced by the body, called reactive oxygen species
- cells dividing more often
- getting older, which increases the number of times cells have divided
- the female hormone oestrogen which stimulates some cells in the breast or womb to divide
- abnormal cells not dying
- the human papilloma virus can help abnormal cells in certain areas of the body to continue to live and divide (Cancer Institute NSW 2020).

We do not know all of the risks and causes of cancer. However, there are a number of chemical, physical and biological agents that have been shown to trigger the mistakes in the cell blueprint that cause cancer. These are called carcinogens and include tobacco, ultraviolet radiation and asbestos.....Cancer can sometimes develop without any specific causes (Australian Cancer Council 2020).

Gene mutations can occur for several reasons, for instance:

- Gene mutations you're born with. You may be born with a genetic mutation that you inherited from your parents. This type of mutation accounts for a small percentage of cancers.
- Gene mutations that occur after birth. Most gene mutations occur after you're born and aren't inherited. A number of forces can cause gene mutations, such as smoking, radiation, viruses, cancer-causing chemicals (carcinogens), obesity, hormones, chronic inflammation and a lack of exercise (Mayo Clinic 2020).

A sporadic (occurs by chance) cell change or mutation is usually what causes childhood cancer. In adults, the type of cell that becomes cancerous is usually an epithelial cell. Epithelial cells line the body cavity and cover the body surface. Cancer occurs from environmental exposures to these cells over time. Adult cancers are sometimes referred to as acquired for this reason (Standford Health Care 2020).

In referring to genetic explanations, some of the above orthodox viewpoints conflict with much current literature and experience, for example:

- Less than 5% of cancer incidence is genetically related (Johnson 2020).
- Acknowledging the increasing cancer rates in children, a report to President Obama in 2010 commented that

The causes of this increase are not known, but as a meeting presenter emphasized, the changes have been too rapid to be of genetic origin. Nor can these increases be explained by the advent of better diagnostic techniques such as computed tomography (CT) and magnetic resonance imaging (MRI). Increased incidence due to better diagnosis might be expected to cause a one-time spike in rates, but not the steady increases that have occurred in these cancers over a 30-year span (Reuben 2010).

- It has repeatedly been presented by orthodox doctors that cancer is a genetic disease. However, it has been proposed that it is metabolic. With many 'experts' saying it is genetic, are they looking in the wrong area? It has been strongly argued that genetic damage is secondary (Christofferson 2017, Lee 2012).

In the early 1900s cancer was a rare disease but today it is the second largest cause of death in most developed nations. Rachel Carson drew attention to cancer incidence being only 4% in 1900 and 15% in 1958 (now around 20%+) corresponding with the increasing use of agricultural chemicals.

The idea of environmental causes of cancer originated from the 1950's when John Higginson attributed 70 to 80% as being environmentally related, linked to personal habits, pollution and occupational exposures. The WHO in 1964 endorsed this view that "the majority of human cancer is potentially preventable" (WHO 1964p4). Higginson went on to champion this viewpoint (Higginson 1969). John Cairns in 1975 said that "almost all cancers appear to be caused by exposure to factors in the environment" (Cairns 1975p64). An implication of environmental causation is that the cancer policy of just pursuing a cure was the wrong "war" to be fighting. Was the multi-billion-dollar expenditure of the US government on the war on cancer a medical failure?

In a book written to highlight the industrial and political interests in maintaining the status quo of treating cancer once formed, and ignoring the causes, Robert Proctor wrote his book titled

Cancer Wars. A Professor of History of Science, at Stanford University, Proctor highlighted, per his book's subtitle, "how politics shapes what we know and don't know about cancer". He later expanded this theme co-editing a book on agnotology which is an area of study this thesis will utilise.

Cancer is caused by chemicals in the air we breathe, the water we drink, and the food we eat. Cancer is caused by bad habits, bad working conditions, bad government, and bad luck-including the luck of your genetic draw and the culture into which you are born (Proctor 1995p1).

A press release by the International Agency for Research on Cancer, stated: "Global battle against cancer won't be won with treatment alone. Effective prevention measures [are] urgently needed to prevent [a] cancer crisis" (IARC 2014p2).

Cancer is regarded by some to be a final over-presentation of a lack of homeostasis that can have happened 10 to 20 years ago. Chemo is used to treat cancer only when it is in its final stages (Jimenez 2020, Hueper 1942). This presents part of the problem in recognising the actual causes and is perhaps why the orthodox view is slanted toward the disease just happening in that there can be no currently obvious explanation.

In his large book on the subject Hueper, in 1942, attributed the gigantic growth of modern industry as producing many chronic diseases never seen before by exposing us to new synthetic substances in never ending number (Hueper 1942). Years later Hueper wrote that government censorship of the radiation health effects from uranium mining, delaying attention to the situation, had caused the death of countless men which could have been avoided (Hueper 1976).

Samuel Epstein contended that "there is now growing recognition that the majority of human cancers are due to chemical carcinogens in the environment" (Epstein 1974p2435). He also seemed to believe "that the germ-line genetic effects of environmental chemicals could be as serious as the shorter term cancer they caused" (Epstein 1970p817). Epstein had also co-authored papers such as a highly cited review of the carcinogenic nitrosamines produced in the stomach from consuming nitrates/nitrites used in meat and fish as preservatives, still extensively used today (Lijinski 1970). In his book there were many statistics such as in respect

to chemical food additives where it was estimated that about 4kg were consumed per year per person (Epstein 1978).

A recent study identified chemicals that may cause breast cancer through upregulation of estradiol (estrogen) and progesterone. Almost 300 chemicals found in pesticides, consumer products, food additives, and drinking water were identified. About 13% of these were previously identified as “unlikely” carcinogens or reproductive/developmental toxicants, despite showing cancer-causing potential in studies (Cardona 2021).

The warning of increasing cancer rates was only one of Epstein’s points. He also charged that industry deflected, distorted and destroyed evidence of carcinogenicity of its various products. The industry approaches he put forward summed up (Proctor 1995p58) as below:

Blocking regulation of hazardous products and processes:

- Minimising of hazard potential
- Maximising compliance costs
- Exaggeration of product efficacy

Strategies employed:

- Attention diversion
 - Insisting on greater precision in studies
 - Insisting on longer term animal/human studies
 - Public propaganda such as media blitzes
 - Blaming the victims (through genetics or smoking habits etc.)
- Controlling information: usually by withholding it from government departments
- Influencing government policy via lobbying through to writing of regulatory legislation
- Government agency exhaustion by repeatedly challenging assumptions in risk assessment procedures etc.

Perhaps the most serious of the above to human deaths was the withholding of information of which there are other examples within this thesis. One example cited was when European plastic manufacturers in the early 1970s, found that vinyl chloride could cause cancer. They

shared the findings with the US Manufacturing Chemists' Association and they then withheld their publication. It wasn't until the deaths of several workers in such plants died of rare liver cancers many years after, did they finally release such information due to public pressure (Proctor 1995).

An example of industry ability to delay government action was that of aldrin and dieldrin, primarily used on corn, where the US FDA had evidence of their carcinogenicity from 1962, yet the Shell Chemical Co.'s influence saw a ban on agricultural uses in 1975: their use in termite treatment in houses was not banned. A similar thing happened with chlordane and heptachlor: they were finally phased out more than a decade after the US FDA discovered cancer in mice studies. In that time 24 million houses had been treated with these persistent chemicals (Proctor 1995p23).

Epstein likened the fact that industry was the main supplier of cancer information to the government regulatory agencies as like "asking the Mafia to give the police a list of people who should be imprisoned" (Proctor 1995p59).

Of the thousands of xenochemicals in the environment that have carcinogenic links in various studies, three are addressed here: glyphosate, aluminium and PFAS chemicals.

Glyphosate and Cancer

This has been touched on in the previous section on autoimmune disease connections. Until recently, after court cases in the US have been won based on glyphosate use inducing various cancer conditions in exposed workers, the orthodox position had been along the lines of:

"There is no good evidence that people exposed to glyphosate at low levels, for example when using it as a weed killer in their garden, have an increased risk of cancer" (Cancer Research UK 2020).

Yet there were studies that had connected low level exposures to various cancers. One was a study on breast cancer which concluded that two glyphosate containing products "...can cause cellular damage at low doses in a relatively short period of time ...mainly affecting cell cycle and DNA repair" (Thongprakaisang 2013p129). The mounting evidence was such that in 2015, the International Agency for Research on Cancer (IARC) classified glyphosate as a probable human

carcinogen (IARC 2015). Yet in the same year the European Food Safety Authority (EFSA) concluded in a review that “glyphosate is unlikely to pose a carcinogenic hazard to humans.” (EFSA 2015p5). This then prompted an article authored by 92 scientists, without industry affiliations, criticizing the EFSA review. One of the criticisms the EFSA review was that:

...almost no weight is given to studies from the published literature and there is an over-reliance on non-publicly available industry-provided studies using a limited set of assays that define the minimum data necessary for the marketing of a pesticide. The IARC WG evaluation of probably carcinogenic to humans accurately reflects the results of published scientific literature on glyphosate and, on the face of it, unpublished studies to which EFSA refers (Portier 2016p743).

The misinformation produced by Monsanto is discussed further in this thesis’s coverage of ignorance in a later chapter. In the US, Edwin Hardeman took Monsanto to court alleging that it concealed the risks of Roundup Herbicide causing non-Hodgkins lymphoma. He was successful with the case with his attorney stating:

It is clear from Monsanto's actions that it does not care whether Roundup causes cancer, focusing instead on manipulating public opinion and undermining anyone who raises genuine and legitimate concerns about Roundup (Levin 2019).

Later in 2019, a study showed that glyphosate has multi-generational effects. Pregnant rats were exposed to half of the European Food Safety Authority no-observed-adverse-effect-level for glyphosate between the eighth and 14th day of gestation. The offspring had higher rates of birth defects, obesity, and diseases of the kidneys, prostate, testes, ovaries and mammary glands (breasts). A 30% higher rate of prostate disease occurred in the third generation males. The third generation females showed a 40% higher rate of kidney disease. Cancer was not unusual in the first and third generations but was at an increased incidence in the second-generation (Kubsad 2019).

In the discussion on the possible distortion of meta-analyses in chapter 4, two such analyses, published three years apart, are cited. The Monsanto-funded analysis concluded “a causal relationship has not been established between glyphosate exposure and risk of any type of LHC [lymphohematopoietic cancer which includes NHL]” (Chang 2016p402). The later meta analysis

concluded there was “a compelling link between exposures to GBHs [glyphosate based herbicides] and increased risk for NHL [non-Hodgkin lymphoma]” (Zhang 2019p186).

As seen from the above there has been an effort displayed by the manufacturer, and government authorities in some instances, to overlook the independent study results showing links between glyphosate and cancer.

Aluminium and Cancer

Aluminium has been chosen here as an example of a heavy metal that is now widely found in the environment: in the air, food, cooking products, cosmetics, medicines, vaccines, dentistry, etc. (Exley 2013). Aluminium has been recognised as a mutagen for many years but specific research in humans is scarce. The majority of studies focus on effects in cell lines. Aluminium is a pro-oxidant, excitotoxin, inflammagen, and immunogen (Exley 2013). There are studies that have generally associated cancer with aluminium exposures (Andreotti 2012, Gibbs 1985). Mentioned in other thesis chapters are the findings of aluminium levels in Alzheimer's disease.

There has been recent interest in aluminium exposure and breast cancer. The typical orthodox position is:

Because underarm antiperspirants or deodorants are applied near the breast and contain potentially harmful ingredients, several scientists and others have suggested a possible connection between their use and breast cancer (Darbre 2009, McGrath 2003). However, no scientific evidence links the use of these products to the development of breast cancer (NIH 2020d).

In the above orthodox position statement, it is interesting to note that the two papers referred to make clear science-based concerns, firstly about the link between aluminium-based antiperspirant use and breast cyst development (Darbre 2009). The second is a population sample study showing that the “frequency and earlier onset of antiperspirant/deodorant usage with underarm shaving were associated with an earlier age of breast cancer diagnosis”

(McGrath 2003p479). Yet, despite acknowledging these studies, the statement asserts there is “no scientific evidence.”

Aluminium is now commonly found in human breast cells. With the incidence of breast cancer increasing in industrialised countries since the mid 1960’s, the introduction of aluminium-based antiperspirants at the end of the 1950’s seems very suspect considering the time delay from cancer initiation to diagnosis. Furthermore, the majority of the tumours occurring in the upper outer quadrant of the breast (McGrath 2003, Darbre 2016, Mandriota 2017) adds to the association. It is interesting that after finding links (Sappino 2012), especially in studies published in 2016 and 2017 (Mandriota 2016, Linhart 2017), one of the lead authors, Mandriota, authored a ‘more studies needed’ paper one year later (Mandriota 2017).

As seen in this section, aluminium’s links to cancer has seen studies ignored as well as suspect professional intimidation to maintain the orthodox position.

PFAS Chemicals and Cancer

Per- and Poly-Fluoroalkyl Substances (PFAS) are a group of manufactured chemicals that have been used in various products since 1950. PFAS are heat, oil, grease, and water resistant so their uses have been broad: from fire retardants/foam to “grease-resistant paper, fast food containers/wrappers, microwave popcorn bags, pizza boxes, and candy wrappers”, pesticides, non-stick “cookware such as Teflon® coated pots/pans, stain resistant coatings such as Scotchguard® used on carpets, upholstery, and other fabrics, water resistant clothing such as Gore-Tex®, cleaning products, personal care products (shampoo, dental floss) and cosmetics (nail polish, eye makeup), paints, varnishes, and sealants” (ATSDR 2017). As early as the 1950s it was known that the chemicals could leach from packaging into food (Melnick 1953).

Most PFAS chemicals, such as perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA), do not break down in the environment. PFAS being stable chemicals, can travel long distances, seep through soil into groundwater, and be carried through air. They bioaccumulate in animals and humans. There are now levels found in rain, human blood & adipose tissue, breast milk, indoor cats and the Arctic and Antarctic.

PFAS chemicals have been detected in vegetables (Eun 2020), milk and dairy products (Sznajder-Katarzyńska 2019), potatoes, cereal seeds, and fruits (Ghisi 2019). In 2017 the USFDA found a PFAS chemical in a sample of a chocolate cake 250 times higher than federal recommendations yet simply dismissed any public health threat without substantiation (Genualdi 2017). Recently a consensus statement from 33 independent scientists from 11 countries warned that inadequate global regulations of chemicals in food packaging pose a growing risk to human health (Muncke 2020). Few of the approximate 12000 chemicals used in food packaging have been adequately studied to determine what risks they may pose to consumers. PFAS chemicals are dominant in many food packaging items.

Immunotoxicity and carcinogenicity effects of PFAS chemicals were found in studies done more than thirty years ago. This was mostly from blood samples from exposed workers and in the general population and community water supplies around manufacturing plants, yet nothing appeared to be done to restrict further use in the general community.

The association between cancer and PFAS chemicals is now well established. These are some of the chemicals found in drinking water in cities through the U.S., in foods and in people. In 2015, data from the 2007 to 2010 US National Health and Nutrition Examination Survey found 97% to 100% of serum samples taken contained PFAS chemicals (Lewis 2015).

The authors of a recent study (Temkin 2020) undertook an analysis of 26 different PFASs to determine the mechanism these chemicals may use to trigger cancer growth in humans and animals. They used the Key Characteristics of Carcinogens framework to identify hazards. Analyses were done on how the chemicals may change DNA, affect the immune system, alter cellular communication, trigger inflammation and other factors. One of the toxicologists involved in the study commented on the carcinogenic nature: "We found that every single one of them exhibited at least one of the key characteristics" (Lee 2020). PFOA and PFOS chemicals had the greatest number, with each chemical exhibiting up to five characteristics that may trigger cancer.

Replacements for these toxic chemicals are also likely not safe. DuPont introduced GenX as a replacement for PFOA in Teflon in 2009 and it has since tested positive to affect hormone activity (Conley 2019). The EPA has also classified GenX as "having suggestive evidence of carcinogenic potential" (Temkin 2020p1).

This section highlights that despite considerable evidence over decades, of the carcinogenic nature of this group of chemicals, authorities allowed their extensive use to continue. Where they have been replaced, the lacking assessment system has allowed yet another generation of toxins to be extensively used.

In this short coverage of cancer, considerable evidence is available linking cancer to many environmental exposures. Glyphosate, aluminium, and PFAS, have clear potential links to cancer causation. There however remains large areas of undone science on the potential for cancer causation with many exposures such as to nano-materials. From previous experiences with similar substances such as asbestos, there is a need to research exposure by thorough biomonitoring.

We have now considered two large areas of chronic disease, autoimmune diseases and cancer. These diseases have been shown to have many environmental connections in respect to their origins. We will now move on to illustrate more-general health effects from environmental exposures.

INDIRECT ENVIRONMENTAL HEALTH EFFECTS

Xenochemicals can cause a myriad of indirect health effects. These indirect effects are rarely addressed in regulatory style studies looking only at specific end points where it is sought to determine whether chemical 'X' will produce symptoms 'Y' by a simple definable mechanism. This section briefly considers this aspect.

The complexity of human body functions with the multitude of natural (endo) chemicals and chemical reactions is generally acknowledged by scientists. Many mechanisms are still not understood. There are many bodily chemical reactions that occur for normal healthy body functions, e.g., the complex pathway of digesting food, converting it to essential nutrient chemicals, and their distribution and absorption by different parts of the body. There are many complex sequences of chemical reactions that occur with most reactions interacting with many others directly or indirectly. To study the effect of one xenochemical in amongst these millions of reactions that occur in the human body is quite a challenge. If a xenochemical does, or does

not, produce significant detected effects then that does not discount a myriad of other non-obvious effects. Most studies look at only defined end points, so if no effects are observed in those end points, this cannot be interpreted as an absence of any effects. Yet industry sponsored studies, and the government regulatory authorities, frequently assume such and reassure the public on this basis.

There are many examples where risks of contracting certain diseases have been related to environmental xenochemical exposures. One example is the increasing incidence of celiac disease. In a recent study young people who had high blood levels of pesticides related chemicals (DDEs) and polybrominated di-phenyl ethers (PBDEs) (Substances used as flame retardants in electronics, upholstered furniture, and mattresses) had twice the chance of being diagnosed with celiac disease. In females, those with higher-than-normal exposure to perfluoroalkyl substances (PFAs) (used as polymers, surfactants and non-stick cookware) were between five and nine times more likely to end up with celiac disease (Gaylord 2020).

Today, compared to previous centuries, there are unprecedented exposures to xenochemicals from the environment: in the food, water, air, the products applied to the skin, the medical and dental materials used, and even from within the womb. Population biomonitoring confirms bioaccumulation of myriad toxicants (refer to a later section in this chapter).

As a primary causative determinant of chronic disease, toxicant exposures induce metabolic disruption in myriad ways, which consequently result in varied clinical manifestations, which are then categorized by health providers into innumerable diagnoses. Chemical disruption of human metabolism has become an etiological determinant of much illness throughout the lifecycle, from neurodevelopmental abnormalities in-utero to dementia in the elderly (Genius 2017p477).

A low dosage xenochemical entering the human body would rarely have a simple effect on the body's health. It may induce chemical chain reactions producing a cascade of different outcomes. For example, it may induce oxidative stress affecting mitochondrial functions, causing cell malfunction, inducing inflammation in tissues, causing malabsorption in the digestive tract with subsequent nutritional problems with various signs and symptoms. Rarely does xenochemical X produces a clear Y problem only. Another example is where some

xenochemicals can be transformed by the liver into reactive metabolites which then cause further and different adverse effects.

Recently a paper by Genuis & Kyrillos, in 2017, reviewed the available scientific literature as to the evidence for low level chemical exposures causing human biological effects. Since this is directly along the aims of this chapter to illustrate such effects, this paper has been used in the following paragraphs reflecting some of their findings. There are many other effects documented in this and other papers than the ones summarised below. The short coverage shows the diversity of effects which have been studied. Detailed references are provided in the paper and are not repeated in the summary below other than when supplemental comments have been added. The remainder of this section can be skipped if one has an understanding that there have been many studies done by independent researchers where environmental toxins have been found to have direct and indirect effects on human biological functions.

Cytotoxic Mechanisms Affected

Heavy metals and pesticides can affect the endoplasmic reticulum in cells producing chronic problems including atherosclerosis, kidney ailments, diabetes, and tumour formation.

Mitochondrial damage from many xenochemicals relates to pathogenesis of many chronic diseases ranging from autism, neurodegenerative diseases, cardiovascular and metabolic diseases through to cancer.

Oxidative stress or free radical destruction by xenochemicals is regarded as the main mechanism involved in neuronal damage, inflammation, carcinogenesis and other processes. It is also thought to be involved in such diseases as Parkinson's, Alzheimer's, ADHD, ASD, heart failure, atherosclerosis, myocardial infarction, vitiligo, and chronic fatigue syndrome.

The now most prevalent brain disease, Parkinson's Disease, has doubled in incidence over the last 25 years. This has been attributed to environmental exposures (Dorsey 2020).

Peroxynitrates can be formed from common chemical exposures. These interact with DNA, lipids and proteins by oxidative or radical mediated mechanisms. Peroxynitrate is a pathogenic mechanism in conditions such as myocardial infarction, stroke, chronic heart failure, diabetes, chronic inflammatory diseases, circulatory shock, neurodegenerative disorders and cancer (Pacher 2007)

Cell receptors enable the communication with organs, and between physiological responses and cellular actions. There are many chemicals, e.g., PCBs, PBDEs, phthalates, mercury, that can occupy receptor positions and disrupt normal body functions over time.

Epigenetic change is a regular occurrence and is clearly linked with environmental exposures as well as other factors such as age and disease state. Studies show that such changes may be initiators for chronic illnesses and that the changes can be passed on to the next generations. These have been particularly linked with pesticide and heavy metal exposures and various illnesses such as Parkinson's, Alzheimer's, ALS, multiple sclerosis, diabetes, atherosclerosis, and even longevity.

The body's detoxification process is important to prevent bioaccumulation of toxicants that are entering the body all the time in the modern way of life. If this detoxification process is inhibited and cannot keep up with the amount of toxicants entering the system then the toxic overload will cause severe health effects. It has been shown that common environmental pollutants, such as organochlorines, brominated chemicals, PCBs, etc. can inhibit the detoxification pathways. These are chemicals that are commonly found in the modern environment and inhibit detoxification at such concentrations.

Many toxicants have been found to form plaque-like structures or deposits. Alpha-synuclein protein assists in the control of neurotransmitters at the synaptic junctions of nerve cells. Studies show some pesticides and solvents can produce increased levels and abnormal deposition of this protein as seen in Parkinson's disease. Neurotoxic plaques are typical in Alzheimer's disease. Toxins such as lead, mercury, aluminium, cadmium,

arsenic, some pesticides and some metal based nanoparticles are increasingly being implicated in these neurotoxic plaques.

Various xenochemicals such as allylamine and benzo(a)pyrene are being associated with the formation of atherosclerotic plaque linked with cardiovascular diseases such as hypertension, stroke, and coronary arterial disease.

A toxicant can displace normal molecules that may be essential for good health. This occurs in relation to the receptors in the various types of cells. Rather than a nutrient, for example, binding to the cell, via a receptor, the toxicant binds to the cell taking its place. Carbon dioxide poisoning is a prime example of this phenomena. CO₂ displaces oxygen from its binding sites on haemoglobin through its higher binding affinity which is more than 200 times that of oxygen. By interfering with the normal body oxygen unloading system CO₂ becomes a dangerous toxin. The brominated chemicals and cadmium are other examples of this mode of toxification.

Cellular toxicity can occur by many other mechanisms:

- Many toxicants can impair and dysregulate signalling in biochemical pathways
- Impairment of protein degradation by toxicants can affect antigen processing, cell transcription, division and repair, biogenesis of organelles, etc. Such pathway disruption has been associated with illnesses from dementia to cancer.
- The interruption of the transport of essential biochemicals for metabolic functions can be inhibited by various toxins
- The intra cellular degradation system (Autophagy) which is important for a variety of cellular functions is inhibited by various toxins

Pathophysiological Mechanisms Affected

In addition to the deleterious effects that can happen on cells as generally outlined above, metabolic alterations can occur from xenochemicals.

We had mentioned endocrine disruption earlier. There are many toxicant effects in the mimicking of hormones, others may act as hormone blockers, interfere with hormone excretion or transportation, or alter gene expression affecting future generations. The possible effects on human health are almost too numerous to cover. Effects range from sex ratio imbalances (frequently fewer male offspring), increases in fibroids, endometriosis, cryptorchidism, decreased thyroid hormone levels, through to links with cancers, adrenal disorders, bone disorders, and many metabolic disorders. There are also suggested links of EDCs with gender issues, sexual preference, and sexual behaviour.

Inflammation can be caused by toxicants by triggering an immune response through pro-inflammatory cytokines, oxidative stress, or cellular damage.

The dysregulation of the immune system resulting in its suppression, hypersensitivity state or autoimmune state has been studied and linked to many environmental toxins. The immune system suppression has been shown in many studies over the decades and leaves the body open to viruses and bacteria induced illnesses that would otherwise be able to be guarded against. The conventional medical system then treats the symptoms with the root cause missed. If the immune system is hypersensitised, this precipitates in allergic reactions.

There is increasing evidence linking environmental toxins with autoimmune diseases (refer previous section). There are studies that have observed increased autoimmune disease incidence near certain industrial areas as well as workers in various chemical exposure situations. A more-common example is simply the higher risk of smokers to seropositive rheumatoid arthritis. It has been hypothesised that the cells and tissues that retain various toxins present differently to the immune system which activates an autoimmune response. As the incidence of most autoimmune diseases is rising and there is such an extensive link to low level environmental chemical exposures an extended coverage of this disease condition had been made in an earlier section.

Neurotransmitters are vital for involuntary and voluntary actions. Endogenous chemicals are involved for neurons to transmit signals across the synaptic junctions or various cells (glands, muscles etc.). In the many toxicant effects, glutamate levels may

be affected for example, which influences cognitive and motor functions such as learning, memory and behaviour control.

Toxicant exposure can cause dysregulation and degranulation of cells such as mast cells, basophils and eosinophils. Mast cells, for example, are a part of the immune system and contain granules rich in histamine and heparin. While best known for their role in allergy and anaphylaxis they also are active in wound healing and defence against pathogens and are tissue resident. Toxicant triggered release of histamine from these cells can affect gut function, immune system processes, trigger inflammatory responses, affect neuro-transmission and affect numerous physiological roles. Mast cell degranulation can be affected by common low level toxicants such as mercury, phthalates, bisphenol A, pesticides, moulds and mycotoxins.

Nutrient Absorption

Nutrient absorption and utilisation is another area where various toxicants can have effects. Cadmium for example, commonly found in vehicle emissions, interferes with calcium absorption and assimilation and increases its excretion (Sugawara 1977). It is also well recognised that toxins in the body disrupt vitamin D pathways. An example is where lead and cadmium have been shown to reduce vitamin D activity (Moon 1994). Many toxic metals including cadmium, mercury, lead, glyphosate, aluminium and endocrine disrupting chemicals such as Bisphenol A and phthalates, and a medical drug bisphosphonate can affect absorption, and utilisation of various essential vitamins and minerals (Goyer 1995,1997, Samsel 2013, Johns 2017). These highlight further resultant indirect effects.

Along with the known bioaccumulation of toxins in humans worldwide, it is generally acknowledged that the worldwide population has low vitamin D levels (Holick 2008). Let's consider briefly the health consequences of the vitamin D levels being affected. Low vitamin D levels can result in varied problems including:

- Children with vitamin D deficiency are likely to display behaviour problems in adolescence (Robinson 2020, Föcker 2017).

- “Externalizing” problems, such as adolescent aggressive and rule-breaking behaviours, were more common where as a child they were vitamin D deficient (Robinson 2020).
- Low levels of vitamin D were linked to anxiety, depression, schizophrenia and autism. (Weydert 2014, Högberg 2012).
- lower vitamin D status was linked with emotional problems, peer relationship issues and behavioural difficulties in children (Husmann 2017).
- low vitamin D status may make a causative contribution to many cancers, infection, multiple sclerosis, hypertension, and diabetes mellitus (Grant 2005, Liu 2006).

As can be seen for the examples in this section, xenochemicals can cause a myriad of indirect health effects. These indirect effects are rarely addressed in regulatory style studies looking only at specific end points. Furthermore, how do orthodox doctors recognise environmental effects if they are so indirectly varied? In the next section we will consider this situation.

HOW DO DOCTORS RECOGNISE ENVIRONMENTAL EFFECTS?

This section will not take a mainstream approach of being based in orthodoxy and looking at the alternative view. Here a look at orthodox medicine from a functional medicine viewpoint will be utilised.

While much could be written about the limited perspectives of orthodox medical doctors, this section simply highlights their difference from the environmental or functional medicine attitude and approaches. The orthodox approach as defining a pathological abnormality as the ‘cause’ of a health condition is at odds with the functional medicine approach of getting to root causes in respect to what has induced the misbalance in the body that has produced such pathology. This helps explain why the orthodox doctors cannot make environmental exposure connections. This then generally creates much public ignorance of such concepts.

There is little coverage of environmental causation of health effects in orthodox medical schools. Medical and health authorities have an orthodoxy related to classical toxicology, diagnosis of symptoms, awareness of infectious diseases, epidemics and monitoring. In these days of doctor visits being 15 to 20 minute appointments, there is an expectation to define the

symptoms and prescribe a 'suitable' drug. If not, a regime of pathological testing is undertaken for a more precise diagnosis for more targeted symptom treatment.

But there are 'alternative' health treatment approaches in what is generally termed environmental or functional medicine. There, it seems to be a general belief that the human body can generally maintain a healthy state given ideal inputs (food & water), exercise and a clean harmonious environment. If one deviates from this ideal then any disease/illness must then be able to be explained from a specific cause which is counter or foreign to such an ideal. Furthermore, when such a cause is defined, the human body may recover fully or partially depending on the extent of damage.

To illustrate environmental causation, it is suggested that orthodox medicine would not currently be able to diagnose the cause and effective treatment of the following symptom combination:

- "Skin itch/rash/flushing/burning and/or tingling: Many describe a "burning pins and needles" kind of pain, especially in the head and chest area
- Confusion/poor concentration and/or memory loss
- Fatigue and muscle weakness
- Headache
- Chest pain and heart problems".

The above are typical symptoms suffered by a person with electromagnetic hypersensitivity syndrome (EHS) (Hedendahl 2015p210). This is where a person is sensitive to electromagnetic fields (EMF), e.g., from wi-fi, mobile phone towers, power lines, etc. Orthodox medicine could not tell the patient that they must reduce/eliminate their EMF exposures to improve their health but would rather follow a psychological and/or drug treatment solution which would be in their biomedical paradigm of belief. This particular EHS condition is addressed in the MCS Chapter.

Another example is where doctors would mistake chlorpyrifos pesticide exposure for a common flu, which would not only be a misdiagnosis, but also a missed report of a pesticide poisoning case (Nash 2004).

The shortcomings of the biomedical paradigm are illustrated through the literature with typical comments as below:

Chronic diseases and illnesses associated with non-specific symptoms are on the rise. In addition to chronic stress in social and work environments, physical and chemical exposures at home, at work, and during leisure activities are causal or contributing environmental stressors that deserve attention by the general practitioner as well as by all other members of the health care community. It seems necessary now to take “new exposures” like electromagnetic fields (EMF) into account. Physicians are increasingly confronted with health problems from unidentified causes. Studies, empirical observations, and patient reports clearly indicate interactions between EMF exposure and health problems. Individual susceptibility and environmental factors are frequently neglected (Belyaev 2016p365).

Successful treatments outside the current medical paradigm approach are reflected by a highly regarded environmental medicine practitioner, Dr Klinghardt. An example is where he draws attention to the toxicity of mercury to immune system cells enabling the growth of various microbes. This effect was first noticed by Dr Y Omura in 1995. Dr Klinghardt has experience with *Borrelia burgdorferi*, mycoplasma, and herpes viruses such as Epstein-Barr, herpes type 6. As viruses in general cannot move on their own into the nervous system, they tend to adhere to heavy metal toxicants. When the neuron doesn't have enough magnesium, calcium, or zinc and the heavy metals are nearby, the heavy metals can be drawn in and take the viruses with them. This understanding has been derived from his experience in the treatment of patients with, for example, the Epstein-Barr virus. He found that neither the established medical drugs nor alternative treatments for the Epstein-Barr virus were effective. But if he performed a heavy metal detox with agents that pulled mercury, lead, nickel and cadmium then normal anti-viral strategies become effective (Klinghardt 2018).

It is interesting to see that some cause and effect relationships which the environmental medicine practitioners have been working with for decades are gradually receiving increased independent research published in orthodox journals. One example is the relationship between the microbiome in the gastro-intestinal tract and various health conditions such as below.

Dr. Cristina Menni, from the Department of Twin Research and Genetic Epidemiology at King's College London, said: "There is considerable interest in finding ways to increase the diversity of gut microbes for other conditions such as obesity and diabetes. Our findings now suggest that finding dietary interventions to improve the healthy bacteria in the gut could also be used to reduce the risk of heart disease" (UON 2018). (basis for comments: Menni 2018).

This research will eventually fit into the biomedical paradigm once standardised testing and diagnosis criteria are established. It would then be regarded as the cause of the many health conditions recognised, but it would not explain what caused the microbiome imbalance in the first place. Until such protocols are established, which may take more decades, the orthodox medical system will not consider the microbiome connection and continue treating the resultant symptoms. In the meantime, the environmental medicine practitioners forge ahead noting cause and effect first-hand in their patients and pursuing environmental exposure modifications to correct the microbiome imbalance producing health condition improvements.

But what are the root causes of a microbiome imbalance? There are now many studies showing xenochemicals can be responsible. For example,

Recent studies have demonstrated that an imbalance in gut bacterial communities, or "dysbiosis", may be a contributor to the pathophysiology of IBS (Menees 2018p1).

Environmental factors add a substantial level of complexity to the understanding of IBD pathogenesis but also promote the fundamental notion that complex diseases such as IBD require complex therapies that go well beyond the current single-agent treatment approach (Ananthkrishnan 2018p39).

Our results show that oral exposure to PCBs can induce substantial changes in the gut microbiome, which may then influence their systemic toxicity (Choi 2013p725).

... glyphosate's adverse effects on the gut microbiota, in conjunction with its established ability to inhibit the activity of cytochrome P450 enzymes, and its likely impairment of sulfate transport, can remarkably explain a great number of the diseases and conditions that are prevalent in the modern industrialized world (Semsel 2013).

Epidemiological studies demonstrate a closely matched rise in the incidence of more than thirty human diseases with the increased utilization of glyphosate, the active ingredient in Monsanto's RoundUp, and the increased consumption of foreign proteins created by genetically modified plants. More than 140 studies have supported the deleterious effects of glyphosate upon the biome, while studies demonstrating positive effects upon humans consuming GMOs have been widely questioned due to researchers' ties to biotech affiliates (D'Brant 2014).

Obesity is associated with phylum-level changes in the microbiota, reduced bacterial diversity and altered representation of bacterial genes and metabolic pathways (Turnbaugh 2009p480).

As was mentioned in the previous section, the disturbance of different pathways, or sequences of events within the body can have various end point health effects. While one xenochemical or environmental exposure may not be recognised as directly producing a certain end point health problem, it may over time have initiated through disruption, a sequence of events which later result in the problem/s. This of course is extremely difficult to study and with the "evidence based" system set-up for studies of single xenochemicals in animals short to medium term, these links may be missed apart from errors of simply not testing for various end points. Furthermore, if no studies have been done at all on a particular environmental exposure effect, the evidenced-based approach will conclude that there is no evidence for such effects. This may then be the rationale to dismiss such an effect being possible.

In reviewing the history of environmental medicine in the US, one paper concluded:

The development of environmental medicine in the United States should have been one of the greatest achievements of 20th-century medicine. Instead, commercial interests have effectively destroyed its potential in the name of corporate profits. The suffering from diseases ranging from depression to obesity and type 2 diabetes could have been greatly reduced by environmental medicine instead of soaring out of control (Meggs 2017p78).

In an article in *The Lancet*, Dr. Patricia Garcia, affiliate professor of global health at Cayetano Heredia University in Lima-Peru and a former minister of health, states that "Corruption is

embedded in health systems" (Garcia 2019p2119). She points out that dishonesty and fraud in the health care system, including its academic and research communities, is "one of the most important barriers to implementing universal health coverage" (Garcia 2019p2119). Such corruption is rarely addressed.

This section highlighted the orthodox approach of defining a pathological abnormality as the 'cause' of a health condition was at odds with the functional medicine approach of getting to root causes in respect to what has induced the misbalance in the body that has produced such pathology. This helps explain why the orthodox doctors cannot make environmental exposure connections. Commercial interests in maintaining the orthodox paradigms further contributes to ignorance about environmental effects on health.

BIOMONITORING

In this section we highlight that many xenochemicals are now being found in the general population. This situation is known by all government and medical authorities. The biomonitoring studies provide clear evidence of exposure to, and bodily absorption of, many toxins from the environment. Yet there have not been any apparent reactions by government regulatory authorities to such alarming findings. Furthermore, the use of this data with associated studies that could be undertaken has not been pursued/funded.

Various countries around the world carry out biomonitoring of a sample of their population for the presence of xenochemicals. In the US it is part of the National Health and Nutrition Examination Survey (NHANES) (CDC 2015). Australia does not do this. Biomonitoring is the measurement of environmental xenochemicals in the body through the presence of the chemical itself, its metabolite(s), or its reaction product(s) in human blood, urine, breast milk, saliva, or other human tissues (Needham 2007). The tests are for a pre-defined array of chemicals, usually over 300, and determine their concentration in the fluid or tissue that has also been selected. The testing does not check for the other hundreds of thousands of xenochemicals that are in the environment, nor in all the fluids and tissues of the body. The chemicals tested for and found however, range from toxic heavy metals, plastic chemicals,

PFAS groups, and pesticides. The results show that all the humans tested have many of these toxic xenochemicals present in their bodies.

From a study done on a group of breast feeding mothers, all of their breast milk was contaminated with PFAS. Some of the measured levels were 2,000 times higher than that recommended as safe for drinking water. "Detection frequencies of current-use short-chain PFAS have been increasing with a doubling time of 4.1 years" (Zheng 2021p7510).

Biomonitoring studies have highlighted the hidden trespass of industrial toxins into human bodies. In so doing it has shifted focus on occupational exposures, to what the general public is exposed to. This demonstrates how pollution to various degrees can become personal, regardless of income, occupation and location. Since chemical cocktails found through biomonitoring have not been the subject of testing, it becomes a concern to people (Morello-Frosch 2009) and points to regulatory failures.

The use of national biomonitoring studies has shortcomings, such as

- The tests are only a snapshot. People may be more or less exposed at other times.
- There is no historical body burden data for the people tested
- There is no locational data, as to whether those tested live such as near farms, in cities, near industry etc.
- The results are only of tests done for specific chemicals: there is no idea of the full load of the thousands of other xenochemicals in the environment that were not tested for. For example, biomonitoring for environmental toxins carried out in some countries, such as the US, do not usually have any testing for aluminium which is significant in the environment.
- As many chemicals used in industrial products may have never been evaluated, the type of test needed to check its presence in the body may not be known. For example, most effects are determined indirectly through depressed cholinesterase levels since organophosphates rapidly break-down in the body before/after the damage has been done: their concentration level or absence, has little meaning. Other chemicals may do their damage and be quickly eliminated or metabolise into something else. Tests then produce false negative results.
- No health status/history is obtained with such individual testing.

- Where industry has replaced former toxic chemicals with new ones, there are difficulties in knowing how to test for them.
- It is relatively easier to measure residues in blood, urine, breast milk, saliva, breath, and hair compared to determining the concentration in adipose tissue. For example, mercury can be locked up in fat and brain tissue so the biomonitoring results do not provide an accurate representation. The body's tendency to accumulate and store various chemicals can be overlooked. The biomonitoring results may indicate either a continuing environmental absorption of the chemicals found or the results may indicate a gradual leaching from adipose tissue storages.
- The absence of tests that provide whole body burdens or body organ/compartiment burdens (e.g., brain) is the tragedy of the situation which makes the biomonitoring studies of questionable use. There seems to be no established correlations relating blood, urine and body burdens of chemicals in general. Even the determination of essential metals, such as iron levels, can only be guessed at from indirect tests of ferritin levels and serum iron measurements. It seems one can only obtain the real situation upon autopsy testing.

A typical orthodox response to biomonitoring results is that the presence of a xenochemical in the body does not automatically indicate harm (CDC 2007). Another response is that the chemical detection methods have advanced more than knowledge to interpret the potential health consequences, so much more research is needed. Statistical, risk based reassurances by the authorities using biomonitoring equivalent values (Hays 2007) and biomonitoring screening values (Faure 2020) have been attempted but rely on existing regulatory authority allowable exposures of each chemical on its own as well as subjective hazard/risk categories. In a later chapter the shortcomings of these individual chemical allowable exposures and risk assessments will be covered. Other public health responses are along the lines of an avoidance of an answer saying that the biomonitoring results simply provide an expedited approach to prioritize chemical regulation. This however disregards that only a relatively small number of chemicals were tested for, which in itself is subjective.

Biomonitoring studies over the years have highlighted that there is much undone science in being able to understand how they have or would affect human health and the difficulty of

epidemiological studies with so many chemicals present. Such gaps in knowledge complicate regulatory science, ethics and the use of risk assessment approaches.

In one of few studies that was carried out in conjunction with the NHANES population testing (the US national biomonitoring study), a survey on the health condition of the people with significant levels of 2,5-DCP was carried out in relation to the prevalence of cardiovascular disease and cancers. 2,5-DCP is a metabolite of para-dichlorobenzene (PCB) which has been detected in indoor ambient air <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6377840/> - R4 (Dodson 2007, Saijo 2004). PCB is commonly used for synthesis of dyes and resins and is used in moth balls as well as in room and toilet deodorizers. 2,5-DCP is also a by-product of chlorination of drinking and waste water, and degradation/metabolism of 1,4-dichlorobenzene and 2,4,5-T pesticide (NCBI 2020). The authors definitively conclude: "...higher urinary concentrations of 2,5-DCP were associated with greater prevalence of cardiovascular diseases and all cancers combined" (Rooney 2019p8). In the NHANES tests, 81% of participants had this chemical in their urine.

Environmental health and justice social movements have used their own biomonitoring to highlight shortcomings of regulations and policies. They have surveyed vulnerable population groups such as pregnant women and newborns near industrial sites and compared them to a control group away from such proximities. This has assisted at times in attempts to change government policies and regulations (Morello-Frosch 2009). An example of this was when residents of Woburn, Massachusetts gathered data and mapped a childhood leukemia cluster around a contaminated well (Brown 1990). This brought local and federal regulatory attention to the toxic pollution situation.

In using biomonitoring to bring environmental justice aspects to the fore there are examples such as (Ramirez-Andreotta 2016):

- Links between minority status, low socioeconomic status and proximity to toxic sites, such as landfills and industry (USGAO 1983, UCC 1987).
- Environmental inequality through minority status (Bullard 2000, Pastor 2001).
- Lack of privilege, political influence, communication skills (Brown 1995, Morello-Frosch 2002, Agyeman 2010),
- Racial differences (Chakraborty 2014),

- Information disparities (Emmett 2010),
- Class differences in health (Grineski 2007),
- Rural health differences (Hartley 2004).

The Environmental Working Group and Commonweal, in 2005, tested the cord blood of 10 newborns (Burden 2005). Two hundred and eighty-seven chemicals were found to be present of which 180 were carcinogenic in humans or animals, 217 were toxic to the brain and nervous system, and 208 were linked to birth defects or abnormal development in animal tests.

In 2008, an environmental group coalition, the Coming Clean Body Burden Work Group, published a report titled “Is it in Us? Toxic Trespass, Regulatory Failure, and Opportunities for Action” (Curtis 2007). The report began with, “What would it feel like to learn you are contaminated with toxic chemicals that permanently damage laboratory animals? What would it feel like to learn those chemicals come from shampoo, soda cans, baby bottles, and thousands of other products that you and your family use every day?” It published the test results of 35 participants in seven US states for 20 toxic chemicals. All had at least seven of the twenty chemicals in their body. All had bisphenol A (in plastics) and PBDE’s (flame retardants) due to their now ubiquitous presence in the environment.

While the focus is on humans, it is interesting to note that limited biomonitoring done on dogs and cats indicates much higher toxin levels. In a first of its kind study, 20 dogs and 37 cats in a typical veterinary clinic were tested for the presence of 70 industrial chemicals by the Environmental Working Group (EWG 2008b):

Average levels of many chemicals were substantially higher in pets than is typical for people, with 2.4 times higher levels of stain- and grease-proof coatings (perfluorochemicals) in dogs, 23 times more fire retardants (PBDEs) in cats, and more than 5 times the amounts of mercury, compared to average levels in people found in national studies conducted by the Centers for Disease Control and Prevention (CDC) and EWG

This section highlighted that many xenochemicals are now being found in all the general population as well as pets. This situation is known by all government and medical authorities. The biomonitoring studies provide clear evidence of exposure to, and bodily absorption of,

toxins from the environment. Yet there have not been any apparent reactions by government regulatory authorities to such alarming findings. Nor, given the limited knowledge on the consequences of such data, has there been any urgency to conduct additional research.

CUMULATIVE EFFECTS

As seen from the above section on biomonitoring there is now extensive evidence that humans are being exposed to many toxic xenochemicals. Most of them have been inadequately assessed, or not at all, on the extent of health effects that they can induce in various concentrations on their own. There has been even less investigation into whether the various chemicals can potentiate or act synergistically to create various health effects. This section considers the real life situation where, as the biomonitoring results show, we are environmentally exposed to many xenochemicals, something not addressed by the regulatory approach of considering each on its own.

It is generally acknowledged that multiple environmental exposures can modify the toxic effects of each exposure acting alone either antagonistically (less than additive) or synergistically (more than additive) (Carpenter 2002, Kortenkamp 2007, Monosson 2005, Sexton 2007). Since "...traditional risk assessment has not routinely taken account of the potential for combined effects from exposure to diverse environmental factors, like those found in the real world" (Sexton 2011p81), adverse health effects can be classed as (Sexton 2011p81):

- (1) those where exogenous agents interfere with normal development and distort physiologic function, such as neurobehavioral abnormalities and sex steroid hormonal disruption;
- (2) those where exogenous agents cause direct cellular damage, such as neurodegenerative diseases and cancer; and
- (3) those that contribute to illness through a combination of both physiologic disruption and cell damage, for example, in cardiovascular disease (Carpenter 2002).

It is generally known that cumulative effects are possible with environmental exposures:

- exposure to tobacco smoke and asbestos (Erren 1999) or radon (Morrison 1998) substantially increases the risk of lung cancer compared to adding the effects of each acting individually.
- the risk of hepato-cellular carcinoma significantly increases with exposure to both aflatoxin-contaminated food and hepatitis B infection (Kuper 2001),
- exposure to noise and toluene causes higher incidence of hearing loss than from each on their own (Franks 1996),
- exposure to ultraviolet radiation and aromatic hydrocarbons increases toxicity to aquatic life (Oris 1985),
- adults with high perceived stress (Cohen 1999) and children of parents who are stressed (Boyce 1995) have increased susceptibility to viral respiratory infections.
- workers exposed to low levels of formaldehyde plus other particles or chemicals show significant adverse reactions (Alexandersson 1982, Johnson 1985).
- headache, skin, eye, nose and throat irritation in painters with exposure levels below irritation levels of the various individual chemicals they are exposed to (Hansen 1987, Bijlsma 2016).
- “...coexposure to BPA and soy-based phytoestrogens results in additive estrogenic effects, and may contribute to estrogen-linked diseases, including breast cancer” (Katchy 2014p21).
- “polychlorinated biphenyls (PCBs) can interact together with methyl mercury (MeHg), as well as PCB together with polybrominated diphenyl ether (PBDE 99) to exacerbate developmental neurotoxic effects when present during a critical period of [mice] neonatal brain development... Furthermore, PBDE can interact with MeHg causing developmental neurotoxic effects similar to those we previously have observed between PCB 153 + MeHg and PCB 52 + PBDE 99. This is of vital importance, as the levels of PBDEs are increasing in mother's milk and in the environment generally.” (Fischer 2008p275).
- Mixtures can also cause pro and anti-carcinogenic effects (Czarnota 2015)

- Health effects of a pharmaceutical oestrogen and a persistent organochlorine pesticide at levels well below regulatory guidelines (Delfosse 2015, Silva 2002, Soto 1997, Crofton 2008).
- from an investigation by the Center for Biological Diversity in 2016 it was found that 96 pesticide products approved contained synergistic compounds. These had at least 1 patent application that claimed synergy between the ingredients. Many of these products were the most-utilised in the US (Donley 2016).
- In a study based on pesticide residues found in food in France, it was found that the mixture produced more significant genotoxic and receptor effects than for the response to the individual chemicals (Graillet 2012)
- Xenoestrogens can also be more reactive together, for example, when each are at a concentration that would normally produce no effect on their own, they modulated the effect of an estradiol in a yeast estrogen system assay. Estradiol is an estrogen steroid hormone and the most common hormone prescribed by orthodox medicine. (Silva 2002).
- Endocrine disrupting chemical mixtures were associated with autistic behaviours of children (Braun 2014).
- A study found cumulative effects on pubertal development in boys. Dioxin-like mixtures of xenochemicals may delay, while non-dioxin-like PCB mixtures advance, the timing of puberty (Burns 2016).
- Toxic metal synergistic effects were found in a study on children's brain development (Bobb 2015).
- There are >1000 chemicals that are known or suspected as endocrine system disruptors. Such chemicals in low doses can be hypothesised as leading to hormone dependent genital cancers including ovarian and endometrial cancer, through potentiation effects with each other and possibly endogenous estrogens inhibiting enzymes responsible for estrogen metabolism (Dogan 2016)
- The PFAS chemical family effects can be additive, yet each chemical has an acceptable drinking water level independent of the others (Post 2017).
- There are many xenochemicals in consumer products, food, and the environment that can cause irreversible demasculinization and malformations of sex organs in young

males. One study looked at only 4 such common xenochemicals. Many effects were found, including where each of these were at the accepted no observable adverse effect levels (NOAELs) (used by regulatory authorities) of each on their own. It was concluded that “Evaluations that ignore the possibility of combination effects may lead to considerable underestimations of risks associated with exposures to chemicals that disrupt male sexual differentiation” (Christiansen 2009p1839).

- One of the main arguments against mercury being involved in Alzheimer’s disease is that there seems to be no correlation between the level of mercury and the severity of the disease. The shortcomings of this line of argument however can be illustrated simply via two papers that show the synergistic effects of mercury with other metal residues. An early paper (Schubert 1978) tested rats by administering a mercury lethal dose LD1 (which would cause 1 in 100 rats to die) in combination with another metal such as lead in a LD10 concentration (10 in 100 rats would die): this caused a 100% death rate. Any metal, even zinc, iron, cadmium, lowers the level of mercury required to inhibit creatine kinase (a type of protein related to muscle or heart damage) (Haley 2018). Antibiotic use also enhances mercury toxicity in ocular solutions and prevents its excretion (Haley 2005).
- Neurons in cultures can last 24 hours without significant deaths. Thimerosal (mercury form in vaccines) added caused no significant deaths after 6 hours, but 70% neuron deaths after 24 hours. Aluminium added on its own (another culture) caused no significant deaths after 6 hours, and 10% neuron deaths after 24 hours. These same amounts of thimerosal and aluminium put together in another culture caused 50% death rate after 6 hours and 92% after 24 hours clearly showing synergism (Haley 2005). It should be noted that aluminium is also present in many vaccines. In a similar experiment with testosterone, at 3 hours after introduction to the culture it, like the thimerosal on its own, showed no significant cell death. Yet when both the thimerosal and the testosterone were present together, in 3 hours there was 100% neuron deaths.
- A six month exposure study to real life mixture levels of 13 chemicals below individual NOAELs “induced non monotonic sex-dependent biochemical and redox status changes in rats” (Docea 2018p470).

- The long term administration of very low doses (below the regulatory acceptable/tolerable daily intakes) of a cocktail of 13 chemicals in rats led to a dose-dependent stimulation of the nervous system (Tsatsakis 2019).
- There are studies that show endotoxins, lipopolysaccharides (LPS), can interact synergistically with environmental toxins increasing health effects. One neonatal rat study showed that a pesticide and LPS produced motor and neurobehavioral impairments whereas the pesticide on its own did not (Fan 2011). As the LPS caused brain inflammation, the authors hypothesised that perinatal brain inflammation likely enhances adult susceptibility to environmental toxins (Cai 2013). This again indicates long term consequences from natal and early childhood exposures.

The effects of xenochemicals and ionising and non-ionising radiation on the creation of micronuclei in various cells is a significant development in testing for synergism (Heedle 2019). A micronucleus is a small nucleus that forms if a chromosome or its fragment does not become part of the daughter nuclei during cell division. It indicates genotoxic events and chromosomal instability. This effect is hypothesised as resulting from an overload of repair mechanisms. However regulatory systems do not address this possible effect when testing the chemicals it routinely approves. One clear use would be to assess the xenochemical combinations that are being found in the population biomonitoring discussed in the previous section.

But even high priority cumulative effect studies that would have been expected to have been done for the good of the public, are either not done at all, or with gross shortcomings. Some examples:

- drinking water disinfection by-products such as tri-halomethanes, haloacetic acids, and haloacetonitriles (Feron 2002, Simmons 2002),
- diesel exhaust, which includes hundreds of chemicals both as particulates or gases (HEI 1995; USEPA 2002),
- coal-fired power station emissions such as particulates, heavy metals, nitrogen oxides, and sulfur dioxide (Levy 2002; Sram 1996).

In writing about the gap between actual and potential public knowledge, Jeschke et al termed such as 'knowledge in the dark'. They highlighted OECD data that showed only a fraction of investments in research is done with the main intention of increasing public knowledge

(Jeschke 2019). This way ignorance is created concerning effects on public health. This, and the consequence of undone science, will be taken further in subsequent chapters.

Cumulative exposures however have another dimension to them: one of time. It is necessary to determine historical exposures as well when a doctor is endeavouring to assist the patient:

Determining the exposure history for the period of interest often means that we are concerned about exposures that occurred 10 or more years ago. This is a difficult challenge for exposure assessors because relatively scant exposure-related information is available for most real-world mixtures of public health concern. In some cases, past exposure (over months or years) to persistent compounds can be estimated from concentrations of the chemicals, their metabolites, or reaction products in bodily tissues or fluids (e.g., Pb in blood). Other nonpersistent compounds (e.g., benzene), however, may produce adverse effects that occur long after the chemicals are no longer present in the body, although some of these chemicals do produce DNA adducts or protein adducts that can be used as exposure- and/or early-effect bio-markers. In general, accurate assessment of historical exposure to mixture constituents over the past several years, let alone the past several decades, is problematic. It is important to remember that exposures need not have actual temporal overlap in order to create interactions by changing the vulnerability of sensitive receptors (Sexton 2007p827).

Aside from historical cumulative effects affecting the individual, there can be consequential cumulative effects which can affect subsequent generations. An example is with levels of thyroid hormones in pregnant women. Neurological development of their children has been shown to be related to the mother's thyroid hormones (Woodruff 2008). It has been demonstrated that one group of chemicals including dioxins, dibenzofurans and dioxin-like PCBs affect the levels of a particular liver enzyme. The non-dioxin-like PCBs will affect another type of liver enzyme. However both affected enzyme types can lead to decreased thyroid hormone levels. The brominated fire retardant chemical group and perchlorate can also contribute to the same effects. The real concern with this example is that such different classes of xenochemicals can produce "a dose-additive effect on [thyroid hormones] at environmentally-relevant doses ... demonstrating exposures to chemicals acting on different [biological] pathways can have cumulative effects" (Woodruff 2008p1570). "It is appropriate to

presume cumulative effects unless there is evidence to the contrary, and it is important for risk assessments to consider real-life exposure mixtures.”

“We must move beyond treating chemical exposures as isolated incidents and look at their cumulative biological effects on organs and their role in the onset of chronic diseases. The time has come to overhaul chemical risk assessment” (Maffini 2015p496).

This section has illustrated that cumulative effects of many chemical exposures in the environment are not addressed by the regulatory system approach of considering each chemical on its own. Research needs to address real world situations rather than only isolated lab style tests of each on their own.

LIFESTYLE CHANGES

A reoccurring discovery by many people and environmental health practitioners is the improvement of many diseases through lifestyle changes. This has been highlighted in the last few years with the emergence of many documentary series being aired through the internet with large population followings. “The Truth About Cancer” series by Ty Bollinger, “Betrayal: The Autoimmune Epidemic” by Tom O’Bryan, were among the first produced, with a long list of others by different authors essentially highlighting that lifestyle changes can have an enormous effect on health. While this area is well outside the scope of this thesis, it is pertinent to mention it, since many lifestyle changes involve reducing manufactured environmental exposures.

While the orthodox medical approach demands many consistent scientific studies over many decades for a new treatment of diseases, the environmental/functional medicine doctors seem to be seeing the results clearly and repeatedly in their patients. As there are no patentable drugs required for such treatment, it is argued: why spend money on trials when the results can be seen first-hand? There are hundreds of thousands of testimonials that can be found on such treatment successes from the patients.

There are some papers occasionally published by alternative health practitioners illustrating their experiences in treating chronic conditions, such as the following example on a graves

disease (GD) sufferer. Graves disease is an endocrine system disease. It is an autoimmune disorder that causes hyperthyroidism, or overactive thyroid. The thyroid is attacked by the immune system causing more thyroid hormones to be made than the body needs. Common symptoms are anxiety, irritability, tremor of hands or fingers, heat sensitivity, weight loss, enlargement of thyroid gland (goitre), change in menstrual cycles, erectile dysfunction or reduced libido, frequent bowel movements, bulging eyes, fatigue, thick red skin on the shins or tops of feet, irregular heartbeat.

GD is considered by orthodox medicine to be chronic and incurable. However a 34 year old Dutch woman was studied who made the lifestyle changes below, and no longer suffered from the disease after 6 months (Brogan 2019).

- 1) Ancestral type of diet
- 2) Oral health interventions
- 3) Yoga with exercise and meditation
- 4) Avoiding environmental toxins (purified water, organic food, natural cleaning/personal hygiene products)
- 5) Occasional vitamin supplements
- 6) No medications

Lifestyle changes address many of the environmental exposures that are within the individual's control. There are extensive numbers of testimonials from individuals that have experienced various health improvements from such a process. Functional medicine seems to build on these experiences and helps yet more people in similar ways, while orthodox medicine requires many randomised, controlled trials before a consideration is made.

GENERATIONAL TOXICITY

After noting in the previous section that lifestyle changes can have an effect on health, let us now consider a worse situation of not having a say in the toxins that one is born with, or affected from in-utero. There is ample evidence that environmental exposures are having profound effects on subsequent generations. This section starts out looking at major childhood

health problems, many of which are increasing in incidence, and what orthodox medicine states as the causes. There will be subsections considering childhood exposures, prenatal exposures and preconception exposure consequences. A further sub-section will comment government inaction on generational toxicity aspects. Finally reference will be made to a special group of xenochemicals which affect the human endocrine system: endocrine disrupting chemicals (EDCs).

“Internal medicine used to be essentially geriatrics when I started twenty years ago...What’s been happening over the past twenty years is that internal medicine patients are becoming younger and younger. With more and more comorbid conditions” (Goldberg 2019p3).

The above quote is representative of general literature and statistics suggesting children have more chronic diseases and health conditions than previous generations. In looking for chronic disease rates in children there is a general absence of monitored and published data, however, it can generally be ascertained:

- In 2010, more than 8 percent of US children had a health condition that interfered with daily activities: an increase of more than 400 percent in fifty years (Newacheck 1984, Bethell 2008, Van Cleave 2010). In 2017–2018, 43% of Australian children had at least 1 long-term condition, with 20% having 2 or more chronic conditions (ABS 2019a).
- In Australia, one in every nine children has asthma (ABS 2019b). Although the incidence appears to have plateaued in this century, it had undergone increases in the previous century such as airway hyperresponsiveness prevalence doubling between 1982 & 1992 (Peat 1994). A typical orthodox explanation for high rates of allergy and asthma is:
 - “living conditions in much of the world might be too clean and that kids aren't being exposed to germs that train their immune systems to tell the difference between harmless and harmful irritants” (AAAA 2020).
- Between 2000 and 2006 type 1 diabetes was increasing in prevalence 2.8 - 3% per year, making it at the time the fastest growing chronic disease amongst Australian children. In 2017 the incidence in children 0-14YO was 159/100,000. (AIHW 2002, 2005, Catanzariti 2009). Type 2 diabetes prevalence rose 257% from 7/100,000 in 1993–96, to 18/100,000 in 2005–06 among 10–24YO (Sillars 2010). The Orthodox hypotheses for the increasing incidence:

- when the immune system learns not to overreact to allergens, it also learns to tolerate compounds from the body's own tissues and therefore prevents the autoimmune attack that destroys the ability to make insulin (McKenna 2012).
 - The exact cause of type 1 diabetes is unknown. We do know that some people carry genes that make them more likely to develop type 1 diabetes. Diabetes occurs in these people when something triggers the immune system to start destroying the beta cells in the pancreas that produce insulin (NDSS 2020).
- In the UK an increase in emotional disorders (including anxiety and depression) for children aged 5-15, rose from 3.9% in 2004 to 5.8% in 2017 (NHS 2018). This increased the incidence in the UK from one in ten to one in nine children. In Australia one in every seven children aged 4–17 had a mental disorder in the previous 12 months (AIHW 2016). This compares to one in six U.S. children aged 6 -17 having a treatable mental health disorder (Whitney 2019). In the US children aged 6–17 years diagnosed with either anxiety or depression had increased from 5.4% in 2003 to 8.4% in 2011–2012 (Bitsko 2018). Typical orthodox explanations are varied for these high incidences and increases:
 - this trend may be partially due to increased use of electronic communication and digital media, which may have changed modes of social interaction enough to affect mood disorders. [Also] ...research shows that young people are not sleeping as much as they did in previous generations (APA 2019).
 - Decline in young people's sense of personal control over their fate; shift toward extrinsic goals, away from intrinsic goals"; "decline of free play may have caused a decline in sense of control and in intrinsic goals, and a rise in anxiety and depression"; "coercive schooling deprives young people of personal control, directs them toward extrinsic goals, and promotes anxiety and depression (Gray 2010).
 - Every day our frontline services see children and teenagers struggling to understand how they fit into the world. They have to contend with things like intense pressure at school, bullying, problems at home, all while navigating a

complex 24/7 world with constant stimulation from social media (Siddique 2018).

- Between 1982 and 2001, cancers increased by an average of 0.6% per year for children aged 0–14 years (AIHW 2005). The total childhood cancers increased by 68% in Australia between 1983 & 2016 (ACC 2020). Cancer rates in this age group had increased by 11 per cent from 2006 to 2014, with almost half of the cases aged between 0-4 years (ACC 2017). A typical orthodox explanation of the continuing increase:
 - The fact is we just really don't know what is contributing to childhood cancers because we don't know the causes of it. Unlike adult cancers, lifestyle factors are rarely part of the cause of cancer in children, and from a medical perspective the focus for childhood cancers is almost exclusively on improving outcomes rather than prevention (Layt 2020).
 - The Queensland Cancer Council, who was part of this research, suggests that better diagnostics and reporting can account for some of the increases, but says more research needs to be done into what's causing childhood cancers in order to better prevent and treat them (TMS 2020).
 - ...environmental causes of childhood cancer have been difficult to identify, partly because cancer in children is rare, and partly because it is difficult to determine what children might have been exposed to early in their development. In fact, most childhood cancers are not thought of as being caused by environmental exposures (NIH 2020c).
- Prior to the 1960s, autism incidence had been 2 to 4 per 10,000 (Lotter 1966, Rutter 2005, Treffert 1970). In 1966, it was estimated as 1 in 2,500 children (Wright 2017b). In 2018 it was 1 in 70 in Australia (ASPECT 2018), having increased by 25.1% between 2015 and 2018 (ABS 2018). Prevalence was then 1 in 42 for Australian boys. Typical orthodox explanations for the large increase:
 - Awareness and changing criteria probably account for the bulk of the rise in prevalence, but biological factors might also contribute... For example, having older parents, particularly an older father, may boost the risk of autism. Children born prematurely also are at increased risk of autism, and more premature infants survive now than ever before. (Wright 2017b).

- ...the “autism epidemic” in most likelihood does not represent an epidemic at all, rather the understanding of autism has improved. Back in the 60s and 70s we may have labelled them as “odd”, “eccentric” or “minimally brain damaged”. Some may argue that we are “labelling” kids too easily – that these children with what might be considered to be milder forms of autism should not be diagnosed at all

The situation with children is now to the extent that in most developed countries, health and educational services are now required to cope with the increases in childhood chronic diseases and developmental disabilities (Boyle 2011).

There are many studies showing links between environmental exposures and the above health conditions. Whilst it is not intended to portray that environmental exposures are the only cause, there is evidence suggesting that their contribution is significant.

Ignoring the effects of the parent toxin levels on sperm and egg quality for the moment, it is generally recognised that there are critical windows of development both in the foetus and early childhood. Toxins can readily cross the placenta in a very vulnerable period of human development (Schardein 2000). As mentioned in the biomonitoring section, children are born already carrying a body burden of pesticides, cosmetic ingredients, and industrial chemicals. A recent study of ten newborns revealed, 232 industrial chemicals were found in their umbilical chords.

Of the 287 chemicals we detected in umbilical cord blood, we know that 180 cause cancer in humans or animals, 217 are toxic to the brain and nervous system, and 208 cause birth defects or abnormal development in animal tests. The dangers of pre- or post-natal exposure to this complex mixture of carcinogens, developmental toxins and neurotoxins have never been studied (EWG 2005).

From other studies,

Seven of the 10 babies had in their umbilical cord blood, synthetic musks known as Galaxolide and Tonalide, which are toxic to aquatic life and have been shown in preliminary studies to cause hormonal changes. The musk is used in scented soaps,

perfumes and colognes, indicating the infants were contaminated by cosmetics their mothers used (Fimrite 2009).

“The mother’s chemical body burden will be shared with her foetus or neonate, and the child may, in some instances, be exposed to larger doses relative to the body weight” (Grandjean 2008p74). For example, methylmercury was found to be 5 times more concentrated in one foetal brain than the mother’s blood level (Honda 2006). It has been estimated that a nursing child’s daily PCB dose may be up to 100 times the mother’s blood levels (Grandjean 2008). Children can also be more exposed to EMF (Gandhi 2015).

There is now no starting out in life with zero toxin levels. Then in breastfeeding, more of the mother’s body burden of industrial chemicals are transferred to the child (Heinzow 2009).

CHILDHOOD EXPOSURES

Different effects are possible with toxins depending on the stage of development. This is typical of toxicants such as lead, mercury, PCBs, and alcohol in respect to neurological effects at concentrations well-below a usual expected toxic dose concentration. Developing children can be neurologically affected by at least 200 environmental xenochemicals (Grandjean 2006). Some of these have no lowest safe level (Wigle 2005). The metabolization and elimination of xenochemicals is also dependent on the age and development. For example, an adult can absorb 10% of ingested lead, but a toddler absorbs as much as 50%.

Yet childhood exposures are virtually unregulated. For example, in testing for the presence of just 4 toxic heavy metals in 168 samples of baby food, up to 95% contained one or more of lead, arsenic, cadmium and mercury, with 1 in 4 baby foods containing all 4 (Houlihan 2019). Yet considering breastmilk and placental chemical residues there are many that directly affect the foetus and infants (Bijlsma 2016, WHO 2007ab). An example can be seen with studies on a toxic group of persistent environmental chemicals, the PFAS group. The chemical family includes stain and water repellent fabrics, non-stick products (Teflon), cleaning products, fire-fighting foams, polishes, waxes, paints, chrome plating, electronics manufacturing and oil recovery chemicals. These are now found throughout the environment, including breast milk.

Various toxic effects have been studied such as their effect on infant growth (Jin 2020). A memorable quote illustrating the situation was by a prominent researcher in the area: “We have not measured a single breast milk sample in Australia that would pass water quality standards” (Donohoe 1994p61). Despite these risks, it is generally acknowledged that breast feeding has many other advantages for babies. Alternatives such as regular baby foods have been found to have a variety of toxic residues present, such as heavy metals like arsenic, lead, cadmium, and mercury (USHR 2021).

Childhood environmental exposures as general as air pollution, have been shown to have immediate effects on immune cell expression and cardiovascular effects. In the long term exposure during childhood caused increased risk for hypertension, heart disease, immune dysfunction, and other types of chronic disease later in their adult life (Prunicki 2021).

PRENATAL

As many toxicants have a long latency period for adverse effects to occur, foetal toxicant exposure can explain many of the childhood chronic diseases as seen in the following sample of links that illustrate such:

- “Early life exposures may lead to behavioural and developmental problems not appreciated until problems arise in school or later in life” (UCSF 2020).
- “Early exposure to carcinogens may increase the risk of adulthood cancer. For some toxicants such as arsenic, early life exposure is associated with a greater potency resulting in lung and bladder cancer decades later” (UCSF 2020).
- Early life environmental cues shaping immune function later in life have been illustrated by studies such as where they were found to alter T cell function and gene expression (Meyers 2018).
- “...environmental exposures may harm the fetus by impairing the epigenome of the developing organism to modify disease risk later in life” (Tiffon 2018p3425).
- Through the literature, among the many effects of xenochemicals, are their effects on the methylation pathway (Cao 2013). Prenatal exposures and their effect on this pathway were illustrated by a well-known study that showed that when the diet of

pregnant mice included folic acid and other nutrients, methylation changes occurred in the offspring. There was a resultant difference in the genetically identical mice in being born with brown or yellow fur. The yellow mice fed folic acid and nutrients, became obese, but their brown siblings did not (Waterland 2003).



The evidence for heritability for obesity has been considered negligible (Yang 2015b), and there is evidence that environmental factors contribute to obesity etiology (Rohde 2019). It is generally recognised that nutritional diet status and exercise will also have an influence on gene expression.

- “All forms of mercury are toxic, and children, as well as the developing fetus, are particularly sensitive to most, if not all of these forms. As a neurodevelopmental toxicant, mercury poses a specific threat to the developing fetus and to the child in early life” (WHO 2010p7).
- Phthalate metabolite levels in the urine of pregnant mothers were associated with a range of behavioural problems when the children were 4 to 9 years old including aggression, attention, conduct, and depression (Engel 2010). This was echoed in another review of 11 studies which linked prenatal phthalate exposures with later childhood problems of attention, hyperactivity, lower IQ and poorer social communication (Ejaredar 2015).
- An association between prenatal exposure to 52 xenochemicals and children’s autistic behaviours has been previously shown (Braun 2014).

- A study found cumulative effects on pubertal development in boys. Dioxin-like mixtures of xenochemicals may delay, while non-dioxin-like PCB mixtures advance, the timing of puberty (Burns 2016).
- Synergistic effects of toxic metals on children’s brain development has been found (Bobb 2015).
- “...human association studies have provided a foundation for the association of prenatal exposure to particulate matter with early immunosuppression and later allergic disease in the offspring” (Rychlik 2019p178).

A prominent example of prenatal effects involves the xenochemical diethylstilbestrol (DES). This was a pharmaceutical drug taken by women in pregnancy. It was prescribed between 1940 and 1971 to prevent miscarriage, premature labour, and related complications of pregnancy. There was an increased incidence of breast cancer associated with the drug (Titus-Ernstoff 2001, Calle 1996). But the drug also demonstrated the effects of xenochemicals in early periods of foetal and perinatal development where the female child from such mothers developed cancers of the reproductive tract in later life. Effects on their children in general included obesity and an array of cancers in all their siblings which were also demonstrated in animal studies (Franssen 2014, Newbold 2004, McLachlan 2016, Palmer 2006, Al Jishi 2017).

The difficulty in studying health effects of foetal/neonatal exposures is obvious, but the growing awareness initiated studies that showed many early life exposure effects (Braun 2014, Ochoa-Acuña 2009a,b, Giordano 2010, Wolff 2008, Vrijheid 2010). Confidence in the consistency of such studies sees comments such as:

In human studies, it is well established that exposures during embryonic and fetal development periods can influence immune health. Coupled with genetic predisposition, these exposures can alter lifetime chronic and infectious disease trajectory, and, ultimately, life expectancy” Immune systems effects include: “immunosuppression, autoimmunity, inflammation and tissue damage, hypersensitivity, and general immunomodulation (Rychlik 2019p178).

As can be seen from the above, exposures during and after pregnancy are being increasingly linked with subsequent health problems.

The blood brain barrier in humans, which protects neurological tissues from many toxins, does not start to develop until six months after birth. Many detoxifying enzymes are also undeveloped in most young children. So young children usually have fewer defences than adults (Cranor 2014). Interestingly a lone positive has been espoused in that since many enzymes are not present in young children, some toxins cannot be metabolised into more toxic substances. However in considering genetic sensitivity and diversity, the range of adverse effects can be much greater. Consider these examples:

Polycyclic Aromatic Hydrocarbons (PAHs)

These can be formed from the incomplete combustion of organic compounds such as tobacco, coal, oil and gas and are common in the environment. These can cross the placenta and bind to DNA (Perera 1999). In such binding, incorrect repair and other mutations can be induced leading to cancers and various diseases. Such PAH-DNA effects are observed to be more pronounced in foetuses (Miller 2002, Perera 1999). PAH affects can also be seen in smaller head circumference and other adverse effects in the newborns.

Organophosphate (OP) Pesticides

There are many chemicals in the OP family of toxins. One, for example, still in wide use in Australia, is Chlorpyrifos: an insecticide used to control various pests, including ants, termites and mosquitos. Chlorpyrifos is found in many products used inside and outside the home, public spaces and agriculture.

Although it is recognised that vulnerability varies with age, a genetic trait may also render an individual deficient in an enzyme which assists in the metabolization of OPs (Eskenazi 2008). The resultant effects range from neurotoxic to cardiovascular (Ecobichon 2001). One study followed children to 12 years of age, of mothers exposed to organophosphates during pregnancy. The study reported effects such as: shorter pregnancy duration, poorer neonatal reflexes, lower IQ and cognitive functioning, and concentration problems in the children (UC 2019).

Children experience a myriad of exposures. Although only referencing petrochemicals, this quote sums up the situation succinctly.

As a growing number of scientific studies are showing, today's children first encounter petrochemicals in the womb, and then in their mother's milk. Seemingly unaware, parents then clothe their children in garments made from petrochemicals, feed them on food that contains petrochemicals, give them toys made from petrochemicals to play with, bathe them in petrochemicals, put them to bed in them and surround them in their daily lives with homes furnished and decorated and cars made of these substances. Then they wonder why their children are becoming increasingly sickly and suffering more unexplained conditions. So they take them to the doctor, who prescribes medication which is, quite often, made from petrochemicals (Cribb 2014p114).

There are many studies in the literature on a molecular basis, cells in-vitro, rats, mice and other animals showing generational effects of xenochemical and EMF exposure. Yet in everyday life children are exposed without consideration of the relevance of such studies. It is an uncontrolled experiment with children as the subjects.

Effects occur simply due to the early timing of children's exposure to toxins. Most disease processes require one to three critical steps to occur for the disease to commence. If some of these steps occur early in one's life then fewer steps are required later in life. This was seen in the case of DES (referenced above and in a subsequent chapter) and lead exposure (Heindel 2008). This is also seen with cancer: "Cancer is a multistage process and the occurrence of the first stages in childhood increases the chance that the entire process will be completed, and a cancer produced, within an individual's lifetime" (Miller 2002p412). This seems consistent with the finding of high residues in babies and children and the high and rising childhood cancer rates mentioned previously.

An increasing awareness is present in some isolated quarters: "Prenatal exposures have known adverse effects on maternal and neonatal outcomes. Professional societies recommend routine screening for environmental, occupational, and dietary exposures to reduce exposures and their associated sequelae" (Grindler 2018p1).

MULTIGENERATIONAL TOXICITY

In the last section the aspect of prenatal exposure effects on health was covered. The focus was on the effects on the child from the mother's exposures prior to, or during conception. In presenting this an orthodox perception is commonly encountered that everything would otherwise have been reset through the sperm and ova: one starts with a 'clean slate' so to speak, and this clean slate is then contaminated. However there is a long term shaping of human DNA from generations before, that becomes the hidden elephant in the room and shatters such a 'clean slate' possibility.

A woman is born with all her reproductive eggs, a significant part of the genetic make-up comes directly from the grandmother. The DNA is shaped by the generations before. This has provided a dampened reaction to the xenochemicals in the present day environment as the grandparents were at the end of the period where food was grown locally, and fresh, meat animals grass fed, and no significant levels of processed food, much lower pollution levels in general, etc. Epigenetic effects occurring in the present day, by simply the same reasoning, will be transgenerational (Ledowsky 2019).

Transgenerational effects are evidenced by polymorphisms in the mother and father causing health effects in subsequent generations, not only in their immediate children. An example is where environmental exposures cause MTHFR polymorphisms (Ledowsky 2020). It seems that whenever a study is done, for example, with simple nematodes, generational effects are found (Dai 2019). Multi-generational effects of various xenochemical products in widespread use have been found in the relatively few studies that have been published on such, although very few studies on humans. For example,

- PFOA, used in stain-resistant carpets, water-repellent clothes, paper and cardboard packaging, ski wax, fire fighting foams, and created on the break down of other chemicals: it has been found to result in multi-generational reproductive toxicity (Lee 2017).
- Dimethoate, is a broad-spectrum systemic insecticide on fruit, vegetables, lucerne, peanuts, pastures, cotton, and ornamentals. It has been under review by the APVMA for

some time. One study found parental exposure could result in increased sensitivity and decreased fitness in the offspring over generations in aquatic organisms (Guo 2012).

- Mycotoxins, commonly found in cereal food, can also produce multi-generational toxic effects on different organisms (Zhou 2018).
- Another persistent chemical found throughout the environment, glyphosate, a herbicide and antibiotic, has been found to induce the transgenerational inheritance of disease and germline, for example, sperm epimutations. It has been suggested that the generational toxicology of glyphosate must be considered in future generation disease etiology (Deepika 2019).
- Generational epigenetic inheritance is increasingly being acknowledged in various health conditions, especially those of a metabolic nature (Tollefsbol 2019).

Even stress effect from previous generations have been found to have effects on subsequent generations. Descendants of Holocaust survivors have abnormal stress hormone profiles, and low cortisol production (Yehuda 2008). Due to their altered cortisol response and stress reactivity, Holocaust survivor children have an increased risk of post-traumatic stress disorder (PTSD), anxiety, and depression (Aviad-Wilcheck 2013).

“We have toxins in our soaps and lotions to shampoos, air fresheners, and cleaning products to toxic food (herbicides, pesticides) and all the artificial colouring, flavouring, and preservatives. Our bodies are accumulating junk over a lifetime, and then these toxins that we’ve stored away in our bones are being passed down to babies in utero. It’s turning on problematic genes that would otherwise be lying dormant and prompting problems and sensitivities. Our babies are being born with their toxic load already to the brim, and then the slightest trigger is setting off severe health problems... Because of our accumulated toxicity, our children are not given an optimal chance at thriving in life” (Pompa 2019). This section has shown evidence that the polymorphisms created in each generation from such toxic loads are themselves also passed-on to subsequent generations.

The US Centers for Disease Control has reflected the above attributing all illness as being a result of genetic predisposition in combination with environmental factors (OGDP 2000). Other articles attribute 70-90% of all disease resulting from modifiable environmental exposures (Rappaport 2010). This converges in the now common saying that genetics loads the gun, the

environment pulls the trigger. It seems that the genetic 'loading' has potential to be magnified through multigenerational accumulation and the pulling of the trigger affects subsequent generations.

TOXIN EFFECTS ON CHILDHOOD INTELLIGENCE

A hideous affect which governments and industrial corporations appear to actively avoid and downplay, are the affects of environmental toxins on childhood intelligence. There tends to be a focus only on metabolic/pathological effects with little research on the effect on childhood intelligence. This section is important to illustrate this and how broad the effects can be on humans.

Lead, like many other heavy metals and chemicals such as fluoride, has been linked with lower IQ and academic achievement and a range of socio-behavioural problems and delinquency. Cribb (2014) makes a point of highlighting such effects of exposures over many generations raising the concern that human civilisation can become less intelligent. He acknowledges literature which shows that even a decline of 2 to 5 IQ points has been associated with increased "murder rates, violent crimes, juvenile delinquency, reduced school performance and unwanted pregnancies." One study found the greatest effect of lead on IQ occurs in children with blood lead levels below 5µg/dl: this was the past US CDC acceptable level (Markowitz 2013). The US CDC now admits "No safe blood lead level in children has been identified. Even low levels of lead in blood have been shown to affect IQ, ability to pay attention, and academic achievement. And effects of lead exposure cannot be corrected" (CDC 2020).

Highlighting how easy environmental influences can affect a child's intelligence, one study showed that children fed healthy food were found to be almost 3 IQ points higher than those fed mainly processed foods (Landrigan 2006). Even murder rates fell in the US when lead was banned in petrol (Grandjean 2014).

Initial recognition was of obvious, massive damage caused by high-dose exposures...But later studies with more sophisticated tools have shown in every case that the brain injury caused by toxic chemicals is not limited to the obvious conditions. It is now

recognised that there exists an entire spectrum of diminished brain function in persons exposed to toxic chemicals, termed subclinical toxicity. Widespread subclinical neurotoxicity can affect the health, well-being, intelligence and even the security of entire societies (Landrigan 2006).

Landrigan and Forman went-on to point out that a loss of five IQ points per child would increase the mentally ill number in society by two thirds. Also, “Early exposure to toxic chemicals may increase risk of degenerative brain disease in later life,” such as Alzheimer’s and Parkinson’s disease. They warned that the reduced intelligence and lowered productivity would result in economic costs of tens of billions of dollars: “Environmentally attributable disease is very costly to society.”

IQ scores had been rising for most of the 20th century, but are now declining. This fits with a common belief that people with higher IQs would produce higher IQ children and that people with lower IQs would have more children, and so gradually lowering of IQ scores across the population. However, a new Norwegian study challenges such beliefs and suggests that declining IQ scores are not strongly linked with genetics but rather the environment (Bratsberg 2018).

There are few studies done on the environmental links with declining childhood intelligence levels. Most of the studies have been on the effects of just one toxin: lead. The lack of studies on other toxins in this area does not make them innocent or safe. This remains a significant area of undone science.

ENDOCRINE DISRUPTING CHEMICALS (EDCS)

Many xenochemicals, both naturally occurring and manufactured, can mimic or interfere with the body’s hormonal system, known as the endocrine system. They can alter the synthesis, transport, secretion, binding, action, and elimination of the body’s natural hormones responsible for development, behaviour, fertility, brain, immune, sleep, digestion, growth, and maintenance of normal cell metabolism. The endocrine system’s central organs include the testes, ovaries, pancreas, adrenal glands, pituitary glands, hypothalamus and pineal glands.

EDCs are found in common products, such as, most plastic bottles and containers, metal food can linings, detergents, food, toys, cosmetics, flame retardants, and pesticides.

The European Environment Agency (EEA 2012) has said in relation to EDCs:

In recent decades, there has been a significant growth in many human diseases and disorders including breast and prostate cancer, male infertility and diabetes. Many scientists think that this growth is connected to the rising levels of exposure to mixtures of some chemicals in widespread use (EEA 2012).

The above is reflected in a 2012 WHO and United Nations Environment Programme (UNEP) publication suggesting EDCs play a substantial role in global increases in genital malformations in baby boys, adverse pregnancy outcomes, neurobehavioral disorders, endocrine-related cancers, obesity, and type 2 diabetes (Bergman 2015, Aho 2017). The publication had been authored by independent scientists from many countries. However after the report was published. industry backed scientists published criticism of the report (Lamb 2014) citing the following shortcomings:

- 1) “failure to apply a systematic framework for identifying, reviewing and evaluating data,
- 2) adoption of an unduly informal approach to assessing causation from EDCs,
- 3) reliance on disease trends to suggest associations with EDCs,
- 4) disregard for the role of exposure, dose and potency in endocrine disruption” (Bergman 2015p1008).

Many of the original independent scientist authors then analysed the industry rebuttal and concluded:

...we discovered that the conclusion of Lamb et al. (2014) was based on many distortions, inaccuracies, false generalizations, non-scientific argumentation, and erroneous claims (Bergman 2015p1008).

As we have shown, Lamb et al.’s attempt of deconstructing the UNEP/WHO (Bergman 2015) report is not particularly erudite scientifically. It appears that the critique is not intended to be persuasive to the scientific community, but is designed to speak to bureaucrats, politicians and other decisionmakers not intimately familiar with the topic

of endocrine disruption and therefore susceptible to false generalizations of bias and subjectivity (Bergman 2015p1016).

In conjunction with the industry sponsored paper there was evidence of industry active attempts to lobby and influence regulatory authorities (Horel 2015). In later chapters of the thesis, various ways of promoting ignorance employed by industry will be discussed.

More recently, there seems to be a prevailing orthodox view that the issue is acknowledged, but it is too hard to do anything about it:

Endocrine disrupting chemicals cause adverse effects in animals. But limited scientific information exists on potential health problems in humans. Because people are typically exposed to multiple endocrine disruptors at the same time, assessing public health effects is difficult (NIEHS 2020).

In the US the NIH (now NIEHS) had co-convened an independent panel of experts back in 2000, which concluded “...there was credible evidence that very low doses of some hormone-like chemicals can adversely affect bodily functions in test animals” (NIEHS 2020). Yet after 20 years, their entries under “What has the NIEHS Discovered” on their website is simply the independently discovered toxic effects of DES, PFAS, and arsenic as well as a suspicion that lavender and tea tree oils are EDCs.

The laid-back approach without regard for a precautionary approach by regulators in general, is treated in more detail in the chapter on Regulatory Toxicology. The mounting evidence of endocrine disruption however continues through a trickle of studies in this enormous area of undone science. For example, one of the most common human birth defects in Australia, affecting 1 in 115 live males born, is hypospadias: a disorder affecting development of the urethra (Nassar 2007). This incidence rate is rising, having doubled in 40 years. Of the approximately 1500 EDCs currently identified, many negatively affect male reproductive development.

In the Endocrine Society’s second scientific statement on EDCs there appears to be sufficient evidence that EDCs can cause diseases and dysfunctions including neurodevelopmental defects, cognitive defects, infertility and other reproductive disorders, obesity, diabetes, immune dysfunction, asthma and cancers (EDC-2 2015). “Individuals exposed to EDCs in the

womb face elevated risk of disease later in life. Additionally, some EDCs have multigenerational effects through modification of DNA and other heritable mechanisms, thereby placing future generations at higher risk of disease” (End Soc 2015).

It is pertinent to note that Carson (1962) did not push for new regulation (although she did later in Senate testimony). Rather she advocated for a change in worldview: not the killing individual pest species by chemicals, but biological pest management achieving healthy ecosystems. This comment can be extended to advocating an ecosystem free from toxic environmental exposures.

This section on generational toxicity has illustrated large concerns for future generations from various environmental exposures. The disregard of the situation by medical and health authorities and the regulatory departments will clearly lead to more wider future health problems of children and grandchildren. This is the real tragedy of environmental effects on people’s health.

CONCLUSION

Chapter 2 has illustrated that there is considerable evidence showing links between environmental exposures and human health conditions, especially chronic diseases. In contrast to the orthodox medical view, the chapter considered the effects of low concentrations of xenochemicals in the environment rather than higher levels, such as those allowed in the workplace. Using published studies, it indicates the importance of areas that have been relatively neglected scientifically, where research findings have been commonly ignored or dismissed.

One of the aspects highlighted has been how the orthodox medical and health system is unable to explain the causes of most chronic health conditions. The orthodox approach of making a diagnosis and treating symptoms does not consider the large amount of science that links environmental exposures to root causes. This chapter has illustrated the large amount of evidence and opinion arguing that environmental causation is an important area to consider.

It will be seen in subsequent chapters, there are also many industry sponsored studies that conflict with and criticise the many independent studies used here. As is common in orthodox circles, such as government regulatory departments, if one combines unconflicted studies with conflicted studies for an overall conclusion, as in a meta-analysis or evidence-based approach, the conclusions often become unclear and meaningless. As this situation suits industry and allows it to continue its profit-focused activities, significant effort continues to be expended to achieve this neutralisation of evidence-based assessments (Michaels 2008, Rogers 2019). In researching autism causes, for example, Rogers commented, “if I were to exclude all studies with a financial conflict of interest (which would be warranted), then in many cases I would be excluding the majority of the research on a particular environmental trigger” (Rogers 2019p58). This aspect was also elaborated on in Michaels (2008).

Meta analyses themselves are subject to the authors’ filtering the studies to be considered in their analysis. For example, some meta analyses conclude there is “a significant relationship between mercury concentration and autism. These conclude that the concentration of mercury can be listed as a pathogenic cause (disease-causing) for autism” (Jafari 2020p369). Another example of this was used in the section Glyphosate and Cancer, where two meta analyses were published three years apart: the Monsanto-funded analysis concluded “a causal relationship has not been established between glyphosate exposure and risk of any type of LHC [lymphohematopoietic cancer which includes NHL]” (Chang 2016p402). Another meta analysis concluded there was a “a compelling link between exposures to GBHs [glyphosate based herbicides] and increased risk for NHL” (Zhang 2019p186). That section of the chapter discussed the first of many successful court cases against Monsanto in regard to glyphosate products causing NHL, a case which clearly supported the positive association analysis that was not Monsanto-funded.

Like the proverbial frog sitting in water that is being heated, not realising the gravity of its situation, the public generally remains ignorant of the serious situation that continues to build because of the effects (explained above) on coming generations. Much of that ignorance stems from the public’s faith in science, ethics, the health system, the regulatory system and the medical system. The public perception is that science, being the central tenant of all these areas, will protect them from any health issues, since the entities responsible supposedly aim for public protection. Yet across all health areas, rates of most chronic diseases, infertility,

reproductive problems, neurological problems, etc. are increasing. Even the “war on cancer” is being consistently lost, despite decades of intense research funded by the billions of dollars that have been, and continue to be, raised for it. It may be seen from this and subsequent chapters that we do not appear to have a health care system but rather a sick-care system, aiming to treat symptoms and not causes.

To most people, it is normal to expect that creating new technology offers a perceived higher standard of living. This is paradigmatic in society: costs of technology developments are only in dollars, whereas environmental consequences are simply ignored or dealt with as cheaply as possible to stay within any basic environmental regulations. If crop yields can be improved by using a new pesticide, for example, then this is clearly desirable. For industry, with its primary focus being the bottom line in its annual financial reports, these developments and products are then deemed essential for their very existence. Industry gets no financial reward for protecting the health of the public. The largest corporations--the pharmaceutical companies--make more money from poor public health.

With little interest in the “real” cost of technology developments, companies are non-living entities profiting through technology developments. It becomes advantageous for industry that public ignorance is maintained around the “real” costs. It has done this in the last century by a general approach which aims to stall the creation of new regulations for as long as possible. Failure to carry out research in those “real” cost areas is a primary tool in this process. Industry pushes for voluntary regulations and for further research that takes decades on every concern as it is raised, in order to delay any change (Michaels 2008). (These and other tactics will be explored in chapter 4). A state of risk is then created in our society:

It signals a moment when our technoscientific and socio-political mechanisms of control (developed through and in parallel to the very same technologies that have created this situation) prove inadequate, outdated, and useless for moving beyond a constant state of risk (Roberts 2014p255).

The sciences are entirely incapable of reacting adequately to civilizational risks, since they are prominently involved in the origin and growth of those very risks. Instead ... the sciences become the legitimating patrons of a global industrial pollution and

contamination ... as well as the related generalized sickness and death of plants, animals, and people (Beck 1992p59).

Although it would be interesting to pursue risk-society issues per the last quote from Beck, these pertinent issues will only be noted here in this thesis.

COMING CHAPTERS: WHERE THIS THESIS IS HEADING.

Chapter 2 has highlighted that environmental causation of human health conditions can be an important area to consider for the treatment of the conditions. Medical orthodoxy does not in general consider such causes and rather, treats the symptoms. Industry typically sponsors studies which find no link between human health impacts and their products, sometimes countering independent studies that do. Subsequent meta analyses and evidence-based principles then frequently conclude that conclusions are unclear. This enables industry to continue selling its products (Michaels 2008, Rogers 2019).

Although all risk assessments, and most studies, in respect to health are generally based on an “average” healthy adult, there is a broad spectrum across the population. This is especially the case in regard to health effects from various environmental exposures. Chapter 3 looks at the portion of the population that is most sensitive to environmental exposures. This significant part of the population is labelled as having multiple chemical sensitivity (MCS). These people may be an indication of the worsening environmental situation for the general population.

Recognising MCS would undermine the orthodox medical and health systems by alerting the public to the consequences of technological developments by industry. The ignorance purposely generated by industry would also be featured and potentially affect the financial bottom lines of many industries that depend on such offending products. These aspects will be covered in the next chapter.

Chapter 3 will also consider the common strategy by orthodox medicine and industry to dismiss MCS as a psychological condition. It also briefly refers to two common, and increasing,

psychological conditions -- depression and ADHD – which is included in an appendix.

Depression is a common label given to MCS sufferers but paradoxically, many studies have pointed to environmental exposures as setting the root cause for depression. It becomes a contradiction for treating environmental exposures psychologically when the condition itself is highly linked to environmental exposures.

ADHD has also been referred to in chapter 3 as it is a psychological condition that is widely accepted in all areas of society, yet there are far fewer studies substantiating the condition and its diagnosis than there are for MCS. In contrast to ADHD, the orthodox medical and industry system actively downplays and discredits MCS and those who have studied it.

The following chapters consider the reasons why we are in this alarming situation, where studies on the links between environmental exposure and health-problem causation continue to be ignored. They will consider the generation of ignorance and its use in society, with the following two chapters covering ignorance of environmental effects on health and of the manipulation of knowledge.

REFERENCES

- AAAA: The American Academy of Allergy, Asthma and Immunology, Conditions & Treatments, Library, Allergy Library, Increasing rates of allergies and asthma <https://www.aaaai.org/conditions-and-treatments/library/allergy-library/prevalence-of-allergies-and-asthma> accessed 4 May 2020.
- ABS: Australian Bureau of Statistics, 4364.0 - National Health Survey: Summary of Results, 2004-05 MEDIA RELEASE, February 27, 2006, National Health Survey shows mixed results <https://www.abs.gov.au/AUSSTATS/abs@.nsf/mediareleasesbyCatalogue/BAC45D309CF29D20CA257121007A23BA?OpenDocument> accessed 3 May 2020
- ABS: Australian Bureau of Statistics, 4430.0 - Disability, Ageing and Carers, Australia: Summary of Findings, 2018 <https://www.abs.gov.au/AUSSTATS/abs@.nsf/Lookup/4430.0Main+Features102018?OpenDocument> accessed 3 May 2020.
- ABS: Australian Bureau of Statistics, National Health Survey 2017–18, Findings based on Detailed Microdata analysis, 2019a
- ABS: Australian Bureau of Statistics, 4364.0.55.001 - National Health Survey: First Results, 2017-18 Mar 26, 2019b <https://www.abs.gov.au/ausstats/abs@.nsf/Lookup/by%20Subject/4364.0.55.001~2017-18~Main%20Features~Asthma~35> accessed 3 May 20
- ACC: Australian Cancer Council, Australian Childhood Cancer Statistics Online, https://cancerqld.org.au/research/queensland-cancer-statistics/accr/?gclid=Cj0KCCQjwLT1BRD9ARIsAMH3BtXq-8_gWM6Ox6WAOiRE54IPVl_2rmijneWwtPSRNfrNlr1d6JaPH5AaAhFZEALw_wcB accessed 3 May 20
- ACC: Australian Cancer Council, Australia's youngest affected by rising cancer rates 8 Dec, 2017 in [2017, General Media Release, Research.](https://cancerqld.org.au/news/australias-youngest-affected-rising-cancer-rates/) <https://cancerqld.org.au/news/australias-youngest-affected-rising-cancer-rates/> accessed 4 May 20.
- Aho B, Disrupting regulation: understanding industry engagement on endocrine-disrupting chemicals, Science and Public Policy, 2017, 44, 5, 698-06.
- AIHW: Australian Institute of Health and Welfare, Diabetes: Australian facts 2002. AIHW Cat. No. CVD 20. Diabetes Series No. 3, 2002, Canberra.
- AIHW: Australian Institute of Health and Welfare, Selected chronic diseases among Australia's children, Bulletin 29, Aug 2005 <https://www.aihw.gov.au/reports/chronic-disease/selected-chronic-diseases-among-children/contents/table-of-contents> accessed 20 Oct 2020.
- AIHW: Australian Institute of Health and Welfare 2016. Australia's health 2016b. Australia's health series no. 15. Cat. no. AUS 199. Canberra <https://www.aihw.gov.au/getmedia/42e2f292-4ebb-4e8d-944c-32c014ad2796/ah16-5-5-mental-health-australias-young-people-adolescents.pdf.aspx> accessed 20 Oct 2020.
- AIHW: Australian Institute of Health and Welfare, Impact of overweight and obesity as a risk factor for chronic conditions, Publication, 13 Apr 2017 <https://www.aihw.gov.au/reports/burden-of-disease/impact-of-overweight-and-obesity-as-a-risk-factor-for-chronic-conditions/formats> accessed 3 Apr 2020.

AIHW: Australian Institute of Health and Welfare, Web report, Evidence for chronic disease risk factors 19 Apr 2016a <https://www.aihw.gov.au/reports/chronic-disease/evidence-for-chronic-disease-risk-factors/contents/summary> accessed 6 Apr 2020.

Agarwal A, Desai N, Makker K, Varghese A, Mouradi R, et al. Effect of radiofrequency electromagnetic waves (RF-EMF) from cellular phones on human ejaculated semen: an in vitro study, *Fertil Steril*, 2009, 92, 4, 1318–25.

Agyeman J, Bullard R, Evans B, Exploring the nexus: Bringing together sustainability, environmental justice and equity, *Space Polity*, 2010, 6, 77–90.

Alexandersson R, Kolmodin-Hedman B, Hedenstierna G, Exposure to formaldehyde: effects on pulmonary function, *Archives of Environmental Health*, 1982, 37, 5, 279-84.

APA: American Psychological Association, Mental health issues increased significantly in young adults over last decade: Shift may be due in part to rise of digital media, study suggests, *Science News*, 15 Mar 2019 <https://www.sciencedaily.com/releases/2019/03/190315110908.htm> accessed 5 May 2020.

Allanou R, Hansen BG, Van der Bilt Y. Public availability of data on EU high production volume chemicals. European Commission; 1999.

Alves C, Santos L, Toralles M, Association of type 1 diabetes mellitus and autoimmune disorders in Brazilian children and adolescents. *Indian journal of endocrinology and metabolism*, 2016, 20, 3, 381.

Al Jishi T, Sergi C, Current perspective of diethylstilbestrol (DES) exposure in mothers and offspring. *Reproductive Toxicology*, 2017, 1, 71, 71-7.

Ananthakrishnan A, Bernstein C, Iliopoulos D, Macpherson A, Neurath M, Ali R, Vavricka S, Fiocchi C, Environmental triggers in IBD: a review of progress and evidence, *Nature reviews, Gastroenterology & hepatology*, 2018, 15, 1, 39.

Anaya J, Castiblanco J, Rojas-Villarraga A, et al., The multiple autoimmune syndromes. A clue for the autoimmune tautology, *Clin Rev Allergy Immunol*, 2012a, 43, 256–64.

Anaya J, Rojas-Villarraga A, García-Carrasco M, The autoimmune tautology: from polyautoimmunity and familial autoimmunity to the autoimmune genes, *Autoimmune Dis*, 2012b, 1–2.

Andreotti G, Silverman D, Occupational risk factors and pancreatic cancer: A review of recent findings, *Molecular Carcinogenesis*, 2012, 51, 98–08

Aitken R, Bennetts L, Sawyer D, Wiklendt A, King B, Impact of radio frequency electromagnetic radiation on DNA integrity in the male germline, *Int J Androl*, 2005, 28, 3, 171–9.

Aitken R, Koopman P, Lewis S, Seeds of concern, *Nature*, 2004, 432, 7013, 48–52.

Applegate J, Baer K, Strategies for Closing the Data Gap, Centre for Progressive Reform, 2006 http://www.progressivereform.org/articles/Closing_Data_Gaps_602.pdf accessed 27 Nov 2021.

ASCIA: Australasian Society of Clinical Immunology and Allergy, Patients, Autoimmunity, Autoimmune diseases <https://www.allergy.org.au/patients/autoimmunity/autoimmune-diseases> accessed 9 Apr 20.

Ash M, Autoimmune diseases of the gut: the role of food and digestion, *Betrayal: The autoimmune solution they're not telling you*, Ep 4 Transcript, Doco Series, 2017.

ASPECT: Autism Spectrum Australia, Autism prevalence rate up by an estimated 40% to 1 in 70 people, Media Release, 11 July 2018, <https://www.autismspectrum.org.au/news/autism-prevalence-rate-up-by-an-estimated-40-to-1-in-70-people-11-07-2018> accessed 3 May 2020.

ATSDR Agency for Toxic Substances & Disease Registry
https://www.atsdr.cdc.gov/pfc/sources_of_exposure.html accessed 13 Apr 17.

Australian Cancer Council, About cancer, Causes of cancer <https://www.cancer.org.au/about-cancer/causes-of-cancer/> accessed 15 Apr 2020.

Aviad-Wilcheck Y, et al., The effects of the survival characteristics of parent Holocaust survivors on offsprings' anxiety and depression symptoms, *The Israel Journal of Psychiatry and Related Sciences*, 2013, 50, 3, 210-16.

Barnett A, It's safe to say there is no safe level of air pollution, *Australian and New Zealand Journal of Public Health*, 2014, 38, 5, 407-8 <https://onlinelibrary.wiley.com/doi/full/10.1111/1753-6405.12264>

Baccarelli A, Wright R, Bollati V, et al., Rapid DNA methylation changes after exposure to traffic particles, *American Journal of Respiratory and Critical Care Medicine*, 2009, 179, 7, 572–78.

Barragan-Martinez C, Speck-Hernandez C, Montoya-Ortiz G, et al., Organic solvents as risk factor for autoimmune diseases: a systemic review and meta-analysis, *PLOS ONE*, 2012, 7, 12, Article ID e51506.

Bayry J, Sib´eril S, Triebel F, Tough D, Kaveri S, Rescuing CD4+CD25+ regulatory T-cell functions in rheumatoid arthritis by cytokine-targeted monoclonal antibody therapy, *Drug Discovery Today*, 2007, 12, 13-14, 548–52.

Beelen R, Raaschou-Nielsen O, Stafoggia M, Andersen ZJ, et al., Effects of long-term exposure to air pollution on natural-cause mortality: an analysis of 22 European cohorts within the multicentre ESCAPE project, *Lancet*, 2014, 383, 9919, 785-95. <https://www.ncbi.nlm.nih.gov/pubmed/24332274>

Beck U, *Risk society: towards a new modernity*, London, Sage, 1992.

Begg S, Vos T, Barker B, Stevenson C, Stanley L, Lopez A, The burden of disease and injury in Australia 2003, Cat. no. PHE 82, Canberra, 2007, AIHW. https://www.aihw.gov.au/getmedia/8f7bd3d6-9e69-40c1-b7a8-40dca09a13bf/4_2-chronic-disease.pdf.aspx

Behari J, Kesari K, Effects of microwave radiations on reproductive system of male rats, *Embryo Talk*, 2006, 1(Suppl 1), 81–5.

Benyshek D, The developmental origins of obesity and related health disorders-prenatal and perinatal factors, *Collegium Antropologicum*, 2007, 31, 1, 11–17.

Belyaev I, Dean A, Eger H, Hubmann G, Jandrisovits R, Kern M, Kundi M, Moshhammer H, Lercher P, Müller K, Oberfeld G, EUROPAEM EMF Guideline 2016 for the prevention, diagnosis and treatment of EMF-related health problems and illnesses, *Reviews on environmental health*, 2016, 31, 3, 363-97.

Belyaev I, Dean A, Eger H, Hubmann G, Jandrisovits R, Kern M, Kundi M, Moshhammer H, Lercher P, Müller K, Oberfeld G. EUROPAEM EMF Guideline 2016 for the prevention, diagnosis and treatment of EMF-related health problems and illnesses, *Reviews on environmental health*, 2016, 31, 3, 363-97.

Bergman Å, Becher G, Blumberg B, Bjerregaard P, Bornman R, Brandt I, Casey S, Frouin H, Giudice L, Heindel J, Iguchi T, Manufacturing doubt about endocrine disrupter science—A rebuttal of industry-sponsored critical comments on the UNEP/WHO report “State of the Science of Endocrine Disrupting Chemicals 2012”. *Regulatory Toxicology and Pharmacology*, 2015, 73, 3, 1007-17.

Bethell C, Read D, Blumberg S, Newacheck P, What is the prevalence of children with special health care needs? Toward an understanding of variations in findings and methods across three national surveys, *Matern Child Health J*, 2008, 12, 1, 1 – 14.

Bhandari R, et al., Effects of the environmental estrogenic contaminants bisphenol A and 17 α -ethinyl estradiol on sexual development and adult behaviors in aquatic wildlife species, *Gen Comp Endocrinol*, 2015a, 214, 195-219.

Bhandari R, Vom Saal F, Tillitt D, Transgenerational effects from early developmental exposures to bisphenol A or 17 α -ethinylestradiol in medaka, *Oryzias latipes*, *Scientific reports*, 2015b, 5, 9303.

Bigazzi P, Autoimmunity caused by xenobiotics, *Toxicology*, 1997, 119, 1, 1–21.

Bijlsma N, Cohen M, Environmental Chemical Assessment in Clinical Practice: Unveiling the Elephant in the Room, *Int J Env Res & Pub Hth*, 2016, 13, 181.

Bilbo S, Block C, Bolton J, Hanamsagar R, Tran P, Beyond infection-Maternal immune activation by environmental factors, microglial development, and relevance for autism spectrum disorders. *Experimental neurology*, 2018, 299, 241-51.

Birnbaum L, Staskal D, Diliberto J, Health effects of polybrominated dibenzo-p-dioxins (PBDDs) and dibenzofurans (PBDFs), *Env Int*, 2003, 29, 6, 855-60.

Bitsko R, Holbrook J, Ghandour R, Blumberg S, Visser S, Perou R, Walkup J, Epidemiology and impact of health care provider–diagnosed anxiety and depression among US children, *J developmental and behavioral pediatrics*, 2018, 39, 5, 395.

Bland J, Autoimmune diseases of the gut: the role of food and digestion, *Betrayal: The autoimmune disease solution they're not telling you*, Documentary Series, Episode 4 Transcript, 2017a.

Bland J, The autoimmune epidemic: Root causes and solutions, *Betrayal: The autoimmune solution they're not telling you*, Ep 1 Transcript, Doco Series, 2017b.

Bobb J, Valeri L, Claus Henn B, Christiani D, Wright R, Mazumdar M, et al., Bayesian kernel machine regression for estimating the health effects of multi-pollutant mixtures. *Biostatistics*, Oxford, 2015, 16, 3, 493–08.

Bogdanos D, Smyk D, Rigopoulou E, et al., Twin studies in autoimmune disease: genetics, gender and environment, *J of Autoimmunity*, 2012, 38, 2-3, J156–69.

Boyle C, Boulet S, Schieve L, Cohen R, Blumberg S, Yeargin-Allsopp M, Visser S, Kogan M, Trends in the prevalence of developmental disabilities in US children, 1997–2008, *Pediatrics*, 2011, 127, 6, 1034-42.

Bratsberg B, Rogeberg O, Flynn effect and its reversal are both environmentally caused, *Proceedings of the National Academy of Sciences*, 2018, 201718793.

Braun J, Kalkbrenner A, Just A, Yolton K, Calafat A, Sjodin A, et al., Gestational exposure to endocrine-disrupting chemicals and reciprocal social, repetitive, and stereotypic behaviors in 4- and 5-year-old children: the HOME study, *Environmental health perspectives*, 2014, 122, 5, 513–20.

Bray F, Ferlay J, Soerjomataram I, Siegel R, Torre L, Jemal A, Global cancer statistics 2018: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. *CA: a cancer journal for clinicians*, 2018, 68, 6, 394-24.

Boyce W, Chesney M, Alkon A, Tschann J, Adams S, Chesterman B, et al., Psychobiological reactivity to stress and childhood respiratory illnesses: results of two prospective studies, *Psychosom Med*, 1995, 57, 411–22.

BRI: Benaroya Research Institute, Disease Information, Autoimmune disease
<https://www.benaroyaresearch.org/what-is-bri/disease-information/autoimmune-diseases> accessed 13 Feb 2020.

Brady D, *Betrayal: The autoimmune disease solution they're not telling you*, Documentary Series, Episode 1 Transcript, 2017.

Brial F, Le Lay A, Dumas M, et al., Implication of gut microbiota metabolites in cardiovascular and metabolic diseases, *Cell Mol Life Sci*, 2018, 75, 3977.

Brogan K, Marcelino G, Pedro C, Siefert A, Healing of Graves' disease through lifestyle changes: A case report, *Advances*, 2019, 33, 2, 4-11.

Brown K, Schultz I, Nagler J, Lack of a heritable reproductive defect in the offspring of male rainbow trout exposed to the environmental estrogen 17 α -ethynylestradiol, *Aquat Toxicol*, 2009a, 91, 71–4.

Brown J, Tzoulaki I, Candeias V, Elliott P, Salt intakes around the world: implications for public health, *International Journal of Epidemiology*, 2009b, 38, 3, 791–13.

Brown P, Mikkelsen E, *No Safe Place: Toxic Waste, Leukemia, and Community Action*, Berkeley, University of California Press, 1990.

Brown P, Race, class, and environmental health: A review and systematization of the literature, *Environ Res*, 1995, 69, 15–30.

Bowatte G, Erbas B, Lodge CJ, et al., Traffic-related air pollution exposure over a 5-year period is associated with increased risk of asthma and poor lung function in middle age, *Eur Respir J*, 2017, 50, 1602357. <https://www.ncbi.nlm.nih.gov/pubmed/29074540>

Bowatte G, et al., The influence of childhood traffic-related air pollution exposure on asthma, allergy and sensitization: a systematic review and a meta-analysis of birth cohort studies, *Allergy Cochrane Library*, 2015, 245-56. <https://onlinelibrary.wiley.com/doi/full/10.1111/all.12561>

Bragg-Gresham J, Morgenstern H, McClellan W, Saydah S, Pavkov M, Williams D, Powe N, Tuot D, Hsu R, Saran R, Centers for Disease Control and Prevention CKD Surveillance System, County-level air quality and the prevalence of diagnosed chronic kidney disease in the US Medicare population, *PLoS one*, 2018, 13, 7, e0200612.

Broadwell R, Balin B, Salzman M, Transcytotic pathway for blood-borne protein through the blood-brain barrier, *Proceedings of the National Academy of Sciences*, 1988, 85, 2, 632-6.

Bullard R, Johnson S, *Environmental justice: Grassroots activism and its impact on public policy decision making*, *J Soc Issues*, 2000, 56, 555–78.

Burden E, The pollution in newborns. a benchmark investigation of industrial chemicals, pollutants and pesticides in umbilical cord blood, *Env Working Gr*, 2005 Jul 14.

Burns J, Lee M, Williams P, Korrnick S, Sergeev O, Lam T, et al., Associations of peripubertal serum dioxin and polychlorinated biphenyl concentrations with pubertal timing among russian boys. *Env health persp*, 2016, 124, 11, 1801–7.

Cai Z, Fan L, Kaizaki A, Tien L, Ma T, Pang Y, Lin S, Lin R, Simpson K, Neonatal systemic exposure to lipopolysaccharide enhances susceptibility of nigrostriatal dopaminergic neurons to rotenone neurotoxicity in later life, *Developmental neuroscience*, 2013, 35, 2-3, 155-71.

Cairns J, The cancer problem. *Scientific American*, 1975, 233, 5, 64-79.

Calcinaro F, Dionisi S, Marinaro M, et al., Oral probiotic administration induces interleukin-10 production and prevents spontaneous autoimmune diabetes in the non-obese diabetic mouse, *Diabetologia*, 2005, 48, 8, 1565–75.

Calderón-Garcidueñas L, Franco-Lira M, Mora-Tiscareño A, Medina-Cortina H, Torres-Jardón R, Kavanaugh M, Early Alzheimer's and Parkinson's disease pathology in urban children: friend versus foe responses—it is time to face the evidence, *BioMed research int*, 2013, Article ID 161687, 16p.

Calle E, Mervis C, Thun M, Rodriguez C, Wingo P, Heath Jr C, Diethylstilbestrol and risk of fatal breast cancer in a prospective cohort of US women, *Am j of epid*, 1996, 144, 7, 645-52.

Cancer Institute NSW, What causes cancer, Published 11 March 2019.

<https://www.cancer.nsw.gov.au/learn-about-cancer/what-causes-cancer> accessed 15 Apr 20.

Cancer Research UK, About cancer, causes of cancer, cancer myths, can glyphosate cause cancer?

<https://www.cancerresearchuk.org/about-cancer/causes-of-cancer/cancer-controversies/can-pesticides-or-herbicides-cause-cancer> accessed 17 Apr 2020.

Cao J, Zhang H, Du L, Influence of environmental factors on DNA methylation, *Yi chuan Hereditas*. 2013, 35, 7, 839-46.

Cardona B, Rudel R, Application of an in vitro assay to identify chemicals that increase estradiol and progesterone synthesis and are potential breast cancer risk factors, *Env Health Persp*, 2021, 129, 7, 077003.

Carpenter D, Arcaro K, Spink D, Understanding the human health effects of chemical mixtures, *Environ Health Perspect*, 2002, 110(suppl 1), 25–42.

Carson R, *Silent Spring*, Fawcett pub., Greenwich, Conn. 1962.

CAS: CAS REGISTRY—The Gold Standard for Chemical Substance Information. 2015, Available online: <http://www.cas.org/content/chemical-substances> accessed on 24 June 2015.

Catanzariti L, Faulks K, Moon L, Waters A, Flack J, Craig M, Australia's national trends in the incidence of type 1 diabetes in 0-14-year-olds, 2000-2006, *Diabet med*, 2009, 26, 596-601.

CDC: US Centers for Disease Control and Prevention: "[Interpreting the Data](#)". Third national report on human exposure to environmental chemicals, 2007.

CDC: US Centers for Disease Control and Prevention, Fourth national report on human exposure to environmental chemicals, Feb 2015.

CDC: US Centers for Disease Control and Prevention, National Biomonitoring Program, Phthalates Fact Sheet https://www.cdc.gov/biomonitoring/Phthalates_FactSheet.html accessed 12 Mar 2020a.

CDC: US Centers for Disease Control & Prevention, Childhood lead poisoning prevention, blood lead levels in children <https://www.cdc.gov/nceh/lead/prevention/blood-lead-levels.htm> accessed 12 May 2020b.

Chakraborty J, Collins T, Grineski S, Montgomery M, Hernandez M, Comparing Disproportionate Exposure to Acute and Chronic Pollution Risks: A Case Study in Houston, Texas, *Risk Anal.* 2014, 34, 2005–20.

Chang E, Delzell E, Systematic review and meta-analysis of glyphosate exposure and risk of lymphohematopoietic cancers, *J Env Science and Health, Part B*, 2016, 51, 6, 402-34.

Chen H, Goldberg M, Villeneuve P, A systematic review of the relation between long-term exposure to ambient air pollution and chronic diseases, *Rev Environ Health*, 2008, 23, 4, 243-97.
<https://www.ncbi.nlm.nih.gov/pubmed/19235364>

Cherry J, The 112-year odyssey of pertussis and pertussis vaccines—mistakes made and implications for the future, *J Pediatric Infect Dis Soc*, 2019, 8, 4, 334–41.

Cho Y, Seo W, Kim D, Moon P, Kim S, Lee B, Song B, Baek M, Exogenous exosomes from mice with acetaminophen-induced liver injury promote toxicity in the recipient hepatocytes and mice, *Scientific reports*, 2018, 8, 1, 16070.

Choi J, Eum S, Rampersaud E, Daunert S, Abreu M, Toborek M, Exercise attenuates PCB-induced changes in the mouse gut microbiome, *Env health persp*, 2013, 121, 6, 725-30.

Chowdhary A, Sharma C, Duggal S, Agarwal K, Prakash A, Singh P, Jain S, Kathuria S, Randhawa H, Hagen F, Meis J, New clonal strain of *Candida auris*, Delhi, India, *Emerging infectious diseases*, 2013, 19, 10, 1670.

Christiansen S, Scholze M, Dalgaard M, Vinggaard A, Axelstad M, Kortenkamp A, Hass U, Synergistic disruption of external male sex organ development by a mixture of four antiandrogens, *Env Health Persp*, 2009, 117, 12, 1839-46.

Christofferson T, *Tripping over the truth: how the metabolic theory of cancer is overturning one of medicine's most entrenched paradigms*, Chelsea Green Publishing, 2017.

Claus S, Guillou H, Ellero-Simatos S, The gut microbiota: a major player in the toxicity of environmental pollutants? *Npj biofilms and microbiomes*, 2016, 2, 16003.

CLP: Clean Label Project, Baby food & infant formula heavy metals blinded raw data
<https://www.cleanlabelproject.org/baby-food-heavy-metals-raw-data/> accessed 31 Mar 20.

Cohen S, Doyle W, Skoner D, Psychological stress, cytokine production and severity of upper respiratory illness, *Psychosom Med*, 1999, 61, 175–80.

Conley J, Lambricht C, Evans N, Strynar M, McCord J, McIntyre B, Travlos G, Cardon M, Medlock-Kakaley E, Hartig P, Wilson V, Adverse maternal, fetal, and postnatal effects of hexafluoropropylene oxide dimer acid (genx) from oral gestational exposure in sprague-dawley rats, *Env health persp*, 2019, 127, 3, 037008.

Costa-Silva B, et al., Pancreatic cancer exosomes initiate pre-metastatic niche formation in the liver, *Nature cell biology*, 2015, 17, 816–826.

Cranor C, Reckless laws, contaminated people science reveals legal shortcomings in public health protections. In *Powerless Science? Science and politics in a toxic world*, Eds Boudia S, Jas N, Berghahn, New York, 2014, Chap 9, 195-214.

Cribb J, *Poisoned Planet: How constant exposure to man-made chemicals is putting your life at risk*, Allen & Unwin, Sydney, 2014.

- Crinnion W, Environmental toxins: the hidden drivers of disease, Betrayal Series (Documentary series), 2016, The autoimmune disease solution they're not telling you about, Episode 5.
<https://betrayalseries.com/sales-page-10780365>
- Crofton K, Thyroid disrupting chemicals: mechanisms and mixtures, *Int J andrology*, 2008, 31, 2, 209-23.
- Crumpler D, Prostituting Science: The psycholisation of MCS, CFS and EHS for political gain, Inkling Aust, 2014.
- Curtis K, Wilding B, Commonweal S, Is it in Us?: Toxic trespass, regulatory failure, & opportunities for action, 2007, Communities for a Better Environment, Building a Regional Voice for Environmental Justice. <http://www.cbecal.org/pdf/regional-voice-enviro-justice.pdf>. accessed 20 Sep 19.
- Czarnota J, Gennings C, Wheeler D, Assessment of weighted quantile sum regression for modelling chemical mixtures and cancer risk, *Cancer informatics*, 2015, 14, CIN-S17295.
- Dai S, Zhang Y, Miao Y, Liu R, Pu Y, Yin L, Intergenerational reproductive toxicity of chlordecone in male *Caenorhabditis elegans*, *Environmental Science and Pollution Research*, 2019, 26, 11, 11279-87.
- Damak S, Mosinger B, Margolskee R, Transsynaptic transport of wheat germ agglutinin expressed in a subset of type II taste cells of transgenic mice, *BMC neuroscience*, 2008, 9, 1, 96.
- Danopoulos E, Jenner L, Twiddy M, Rotchell J, Microplastic contamination of seafood intended for human consumption: a systematic review and meta-analysis, *Env Health Persp*, 2020, 128, 12, 126002.
- Darbre P, Aluminium and the human breast, *Morphologie*, 2016, 100, 65–74
- Darbre P, Underarm antiperspirants/deodorants and breast cancer, *Breast Cancer Research*, 2009, 11, Supp 3, S5.
- Dasdag S. Whole-body microwave exposure emitted by cellular phones and testicular function of rats, *Urol Res*, 1999, 27, 3, 219–23.
- Davies T, Infection and autoimmune thyroid disease, *J Clinical Endocrinology and Metabolism*, 2008, 93, 3, 674–676.
- Davis W, Autoimmune Secrets, Documentary Series, Ep 1 Transcript, 1.11.08, 2020.
- D'Brant J, GMOs, gut flora, the shikimate pathway and cytochrome dysregulation, *Nutritional Persp: Journal of the Council on Nutrition*, 2014, 37, 1.
- Deepika K, Nilsson E, King S, Ingrid S, Beck D, Skinner M, Assessment of glyphosate induced epigenetic transgenerational inheritance of pathologies and sperm epimutations, *Generational Toxicology, Scientific Reports (Nature Publisher Group)*, 2019, 9, 1.
- DEE: Australian Government Department of the Environment and Energy, Nitrogen dioxide air quality fact sheet, 2005a. <http://www.environment.gov.au/protection/publications/factsheet-nitrogen-dioxide-no2> Accessed 4th Aug 2019.
- DEE: Australian Government Department of the Environment and Energy, Sulfur Dioxide fact sheet 2005b <http://www.environment.gov.au/protection/publications/factsheet-sulfur-dioxide-so2> . Accessed 4th Aug 2019.

De Carvalho G, Betrayal: The autoimmune disease solution they're not telling you, Documentary Series, 2017 <https://www.facebook.com/thedr.com.english/posts/gabriel-de-carvalho-well-i-say-that-gluten-its-indigestible-to-any-human-being-s/2970788199619125/> accessed 27 Jul 21.

Delfosse V, Dendele B, Huet T, Grimaldi M, Boulahtouf A, Gerbal-Chaloin S, Beucher B, Roecklin D, Muller C, Rahmani R, Cavaillès V, Synergistic activation of human pregnane X receptor by binary cocktails of pharmaceutical and environmental compounds, *Nature communications*, 2015, 6, 1, 1-0.

De Iuliis G, Newey R, King B, Aitken R, Mobile phone radiation induces reactive oxygen species production and DNA damage in human spermatozoa in vitro, *PLoS One*, 2009, 4, 7, e6446.

Dennekamp M, Akram M, Abramson MJ, Tonkin A, Sim MR, Fridman M, Erbas B, Outdoor air pollution as a trigger for out-of-hospital cardiac arrests, *Epidemiology*. 2010, 21, 4, 494-500.
<https://www.ncbi.nlm.nih.gov/pubmed/20489649>

Deng Y, Zhang Y, Lemos B, RenTissue H, Accumulation of microplastics in mice and biomarker responses suggest widespread health risks of exposure, *Sci. Rep.*, 2017, 7, Art 46687.

Di Q, Dai L, Wang Y, Zanobetti A, Choirat C, Schwartz J, Dominici F, Association of short-term exposure to air pollution with mortality in older adults, *JAMA*, 2017, 318, 24, 2446-2456.
<https://www.ncbi.nlm.nih.gov/pubmed/29279932>

Diepens N, Koelmans A, Accumulation of plastic debris and associated contaminants in aquatic food webs, *Environ. Sci. Technol.*, 2018, 52, 8510-20.

Docea AO, Gofita E, Goumenou M, Calina D, Rogoveanu O, Varut M, Olaru C, Kerasioti E, Fountoucidou P, Taitzoglou I, Zlatian O, Six months exposure to a real life mixture of 13 chemicals below individual NOAELs induced non monotonic sex-dependent biochemical and redox status changes in rats, *Food and chemical toxicology*, 2018, 115, 470-81.

Dodson R, Houseman E, Levy J, et al., Measured and modeled personal exposures to and risks from volatile organic compounds, *Env Science & Tech*, 2007, 41, 24, 8498–505

Dogan S, Simsek T, Possible relationship between endocrine disrupting chemicals and hormone dependent gynecologic cancers, *Medical hypotheses*, 2016, 92, 84-7.

DoH: Australian government department of health, Health Direct, Overview of autoimmune diseases <https://www.healthdirect.gov.au/autoimmune-diseases> accessed 9 Apr 20.

Dolapchieva S, Distribution of concanavalin A and wheat germ agglutinin binding sites in the rat peripheral nerve fibres revealed by lectin/glycoprotein-gold histochemistry, *The Histochemical Journal*, 1996, 28, 1, 7-12.

Donley N, Toxic Concoctions: How the EPA ignores the dangers of pesticide cocktails, Center for Biological Diversity, July 2016
https://www.biologicaldiversity.org/campaigns/pesticides_reduction/pdfs/Toxic_concoctions.pdf

Donohoe M, Altered Neurological Function Following Chemical Exposure, Toxic Chemicals and Human Consequences, Nullamanna, ACTA Conference, 1994.

Dorsey R, Sherer T, Okun M, Bastiaan R, Bloem B, Ending Parkinson's Disease: A Prescription for Action, Public Affairs, 1st ed, 2020.

Ecobichon D, Toxic Effects of Pesticides, In: Casarett and Doull's Toxicology, ed. Curtis D. Klaassen, 6th ed., 2001, 763–810. New York, Pergamon Press.

EDC-2: Gore A, Chappell V, Fenton S, et al., The endocrine society's second scientific statement on endocrine disrupting chemicals, *Endocr Rev*, 2015, 36, E1-150.

EEA: European Environment Agency, Increase in cancers and fertility problems may be caused by household chemicals and pharmaceuticals, Press Release, 10 May 2012
<https://www.eea.europa.eu/media/newsreleases/increase-in-cancers-and-fertility> accessed 16 Mar 2019.

EEA: European Environment Agency, Increase in cancers and fertility problems may be caused by household chemicals and pharmaceuticals, Press Release, 10 May 2012, Last modified 02 Sep 2016 <https://www.eea.europa.eu/media/newsreleases/increase-in-cancers-and-fertility> accessed 2 Feb 2020.

EFSA: European Food Safety Authority, Pesticides unit, EFSA explains the carcinogenicity assessment of glyphosate, 12 Nov 2015 https://planet4-eu-unit-stateless.storage.googleapis.com/2018/08/5a1c1ca4-5a1c1ca4-4302_glyphosate_complementary.pdf accessed 18 Apr 20

EFSA: European food safety authority - panel on contaminants in the food chain - statement on the presence of microplastics and nanoplastics in food, with particular focus on seafood, *EFSA Journal*, 2016, 14, 6, 4501.

EHN: Environmental Health News, Food packaging can harm human health, 3 Mar 2020
<https://www.ehn.org/food-packaging-dangers-2645361648.html> accessed 8 Mar 2020.

Elk M, 'Chemical burns': Delta flight attendants say new uniforms cause rashes, *The Guardian*, 4 Apr 2019 <https://www.theguardian.com/business/2019/apr/03/delta-flight-attendants-uniforms-rash-claims> accessed 15 Mar 2019.

Ellinger R, *Phosphates as food ingredients*. Ed Weast R, Cleveland, OH, CRC Press, 1972.

Emmett E, Desai C, Community first communication: Reversing information disparities to achieve environmental justice, *Environ. Justice*, 2010, 3, 79–84.

End Soc: Endocrine Society Position Statement, Endocrine-Disrupting Chemicals in the European Union, June 2015.

Engel S, Miodovnik A, Canfield R, Zhu C, Silva M, Calafat A, Wolff M, Prenatal phthalate exposure is associated with childhood behavior and executive functioning, *Env Health Persp*, 2010, 118, 4, 565–571.

Ejaredar M, Nyanza E, Eycke K, Dewey D, Phthalate exposure and children's neurodevelopment: A systematic review, *Environmental Research*, 2015, 142, 51–60.

Epstein S. Environmental determinants of human cancer, *Cancer Research*, 1974, 34, 10, 2425-35.

Epstein S, Control of chemical pollutants, *Nature*, 1970, 228, 5274, 816-9.

Epstein S, *The politics of cancer*, Sierra Club Books, San Francisco, 1978.

Epstein S, Billions for cures, barely a cent to prevent, *Env Action*, 1984, Nov-Dec, 10-11.

Erogul O, Oztas E, Yildirim I, Kir T, Aydur E, et al., Effects of electromagnetic radiation from a cellular phone on human sperm motility:an in vitro study, *Arch Med Res*, 2006, 37, 7, 840–3.

Erren T, Jacobson M, Piekarski C, Synergy between asbestos and smoking on lung cancer risks, *Epidemiology*, 1999, 10, 405–411.

Eskenazi B, Rosas L, Marks A, Bradman A, Harley K, Holland N, Johnson C, Fenster L, Barr D, Pesticide toxicity and the developing brain, *Basic & clinical pharmacology & toxicology*, 2008, 102, 2, 228-36.

Etzel R, Balk S, Pediatric environmental health, *American Academy of Pediatrics*, 2011 Oct 17, Elk Grove Village, IL, USA.

Eun H, Yamazaki E, Taniyasu S, Miecznikowska A, Falandysz J, Yamashita N, Evaluation of perfluoroalkyl substances in field-cultivated vegetables, *Chemosphere*, 2020, 239, 124750.

EWG: Teen girls' body burden of hormone-altering cosmetics chemicals, Environmental Working Group, 2008a <https://www.ewg.org/research/teen-girls-body-burden-hormone-altering-cosmetics-chemicals> accessed 7 Mar 2019.

EWG: Environmental Working Group, Body burden: the pollution in newborns: detailed findings, 14 July 2005 <https://www.ewg.org/research/body-burden-pollution-newborns/detailed-findings> accessed 11 May 20.

EWG: EWG's 2020 Shopper's Guide to Pesticides in Produce, 99 Percent of Non-Organic Raisins Tainted With at Least Two Chemicals, EWG Science Team, 25 Mar 20 <https://www.ewg.org/foodnews/summary.php#summary> accessed 31 Mar 20.

EWG: Environmental Working Group, 2009, Pollution in People: Cord Blood Contaminants in Minority Newborns, https://static.ewg.org/reports/2009/minority_cord_blood/2009-Minority-Cord-Blood-Report.pdf?_ga=2.119449289.1788180067.1544876684-1840960985.1544876684 accessed 15 Dec 2018.

EWG: Environmental Working Group, Polluted Pets, High Levels of Toxic Industrial Chemicals Contaminate Cats And Dogs, April 17, 2008b <https://www.ewg.org/research/polluted-pets#.WsmqeS5ubIU> accessed 30 Apr 20.

Exley C, Human exposure to aluminium. *Environmental Science: Processes & Impacts*, 2013, 15, 10, 1807-16.

Factor-Litvak P, Insel B, Calafat A, Liu X, Perera F, Rauh V, Whyatt R, Persistent associations between maternal prenatal exposure to phthalates on child IQ at age 7 years, *PloS one*, 2014, 9, 12, e114003.

Fälth-Magnusson K, Magnusson K, Elevated levels of serum antibodies to the lectin wheat germ agglutinin in celiac children lend support to the gluten-lectin theory of celiac disease, *Pediatric Allergy and Immunology*, 1995, 6, 2, 98-102.

Fan L, Tien L, Lin R, Simpson K, Rhodes P, Cai Z, Neonatal exposure to lipopolysaccharide enhances vulnerability of nigrostriatal dopaminergic neurons to rotenone neurotoxicity in later life, *Neurobiology of disease*, 2011, 44, 3, 304-16.

Fasano A, Zonulin and its regulation of intestinal barrier function: the biological door to inflammation, autoimmunity, and cancer, *Physiological reviews*, 2011. 91, 1, 151-75.

Fasano A, All disease begins in the (leaky) gut: Role of zonulin-mediated gut permeability in the pathogenesis of some chronic inflammatory diseases, *F1000 Research*, 2020, 9.

Faure S, Noisel N, Werry K, Karthikeyan S, Aylward L, St-Amand A, Evaluation of human biomonitoring data in a health risk based context: An updated analysis of population level data from the Canadian Health Measures Survey, *Int J hygiene and env health*, 2020, 223, 1, 267-80.

Fejes I, Zavacki Z, Szollosi J, Koloszar Daru J, Kovacs L, et al., Is there a relationship between cell phone use and semen quality? *Arch Androl*, 2005, 51, 5, 385–93.

Feron V, Cassee F, Groten J, ve Vliet P, van Zorge J, International issues on human health effects of exposure to chemical mixtures, *Env Health Persp*, 2002, 110, (suppl 6), 893–899.

Fimrite P, Study: Chemicals, Pollutants Found in Newborns. *San Francisco Chronicle*, 3 Dec 2009, http://articles.sfgate.com/2009-12-03/news/17183043_1_campaign-for-safe-cosmetics-chemicals-umbilical accessed 8 May 20.

Finlay D, Newmeyer D, Price T, Forbes D, Inhibition of in vitro nuclear transport by a lectin that binds to nuclear pores, *J cell biology*, 1987, 104, 2, 189-200.

Fischer C, Fredriksson A, Eriksson P, Coexposure of neonatal mice to a flame retardant PBDE 99 (2, 2', 4, 4', 5-pentabromodiphenyl ether) and methyl mercury enhances developmental neurotoxic defects, *Toxicological Sciences*, 2008, 101, 2, 275-85.

Fitzgerald K, Betrayal: The autoimmune disease solution they're not telling you, *Documentary Series*, Episode 1 Transcript, 2017.

Föcker M, Antel J, Ring S, et al., Vitamin D and mental health in children and adolescents, *Eur Child Adolesc Psychiatry*, 2017, 26, 1043–66.

Fottrell Q, This may hard to swallow: Tyson will use X-ray metal detectors to screen its chicken for 'metal fragments', *Market Watch*, May 11, 2019 <https://www.marketwatch.com/story/tyson-recalls-12m-pounds-of-frozen-chicken-strips-over-contaminated-metal-concerns-2019-05-06> accessed 31 Mar 20.

Franks J, Thais M, Ototoxic effects of chemicals alone or in concert with noise: a review of human studies. In: *Scientific Basis of Noise-Induced Hearing Loss*, New York, Thieme, 1996, 437–44.

Franssen D, Ioannou Y, Alvarez-Real A, Gerard A, Mueller J, Heger S, Bourguignon J, Parent A, Pubertal timing after neonatal diethylstilbestrol exposure in female rats: neuroendocrine vs peripheral effects and additive role of prenatal food restriction, *Reproductive toxicology*, 2014, 44, 63-72.

Furman D, Campisi J, Verdin E, Carrera-Bastos P, Targ S, Franceschi C, Ferrucci L, Gilroy D, Fasano A, Miller G, Miller A, Chronic inflammation in the etiology of disease across the life span, *Nature medicine*, 2019, 25, 12, 1822-32.

Gandhi O, Yes the children are more exposed to radiofrequency energy from mobile telephones than adults, *IEEE Access*, 2015, 3, 985-8.

GAO: U.S. Government Accountability Office, *Hazardous and Non-Hazardous Waste: Demographics of People Living Near Waste Facilities*, GAO, Washington, DC, USA, 1995.

Gao L, Feng L, Yao S, Jiao P, Qin S, Zhang W, Zhang Y, Li F, Molecular mechanisms of celery seed extract induced apoptosis via s phase cell cycle arrest in the BGC-823 human stomach cancer cell line, *Asian Pac J Cancer Prev*, 2011, 12, 10, 2601-6.

García P, Corruption in global health: the open secret, *Lancet*, 2019, 394, 10214, 2119-2124.

Gasana J, Dillikar D, Mendy A, Forno E et al., Motor vehicle pollution and asthma in children: a meta-analysis, *Environ Res*, 2012, 117, 36-45. <https://www.ncbi.nlm.nih.gov/pubmed/22683007>

Gasperi J, Wright S, Dris R, Collard F, Mandin C, Guerrouache M, Langlois V, Kelly F, Tassin B, Microplastics in air: are we breathing it in? *Curr. Opin. Environ. Sci. Health*, 2018, 1, 1-5.

Gaylord A, Trasande L, Kannan K, Thomas K, Lee S, Liu M, Levine J, Persistent organic pollutant exposure and celiac disease: A pilot study, *Environmental Research*, 2020 May 11, 109439.

Gehring U, Gruzieva O, Agius RM, Beelen R, et al., Air pollution exposure and lung function in children: the ESCAPE project, *Environ Health Perspect*, 2013, 121, 11-12, 1357-64.
<https://www.ncbi.nlm.nih.gov/pubmed/24076757/>

Genualdi S, deJager L, Begley T, Investigation of Per- and Polyfluoroalkyl Substances (PFAS) in US food products, Center for Food Safety and Applied Nutrition, Food and Drug Administration, 2017
https://www.khlaw.com/Files/39730_SETAC%20Europe%202019.pdf accessed 19 Apr 20.

Genuis S, Kyrillos E, The chemical disruption of human metabolism, *Toxicology mechanisms and methods*, 2017, 27, 7, 477-500.
<https://www.tandfonline.com/doi/abs/10.1080/15376516.2017.1323986>

Genuis S, Evolution in pediatric health care, *Pediatr. Int*, 2010, 52, 640–43.

GESAMP, Sources, fate and effects of microplastics in the marine environment: part two of a global assessment, Eds, Kershaw P, Rochman C, (IMO/FAO/UNESCO-OC/UNIDO/WMO/IAEA/UN/UNEP/UNDP Joint Group of Experts on the Scientific Aspects of Marine Environmental Protection). *Rep. Stud*, 2016, 93, GESAMP, 220.

Ghisi R, Vamerli T, Manzetti S. Accumulation of perfluorinated alkyl substances (PFAS) in agricultural plants: a review, *Environmental research*, 2019, 169, 326-41.

Gibbs G, Mortality of aluminum reduction plant workers, 1950 through 1977, *J. occup. Med.*, 1985, 27, 761-70.

Gibbs V, Autism on the rise? *Autism Spectrum Australia*, 8 May 2018.
https://www.autismspectrum.org.au/blog/autism-on-the-rise?gclid=EAlaIqobChMIneeLocac6QIVVB2PCh1ItQxNEAAYASAAEgKJNfD_BwE Accessed 5 May 20.

Giordano F, Abballe A, De Felip E, Di Domenico A, Ferro F, Grammatico P, Ingelido A, Marra V, Marrocco G, Vallasciani S, Figà-Talamanca I, Maternal exposures to endocrine disrupting chemicals and hypospadias in offspring. *Birth Defects Research Part A, Clinical and Molecular Teratology*, 2010, 88, 4, 241-50.

Goldberg S, Science about Wireless and 5G, *5G Crisis: Awareness and Accountability*, Docu series transcript, 2019.

Graillot V, Takakura N, Hegarat L, Fessard V, Audebert M, Cravedi J, Genotoxicity of pesticide mixtures present in the diet of the French population, *Environmental and molecular mutagenesis*, 2012, 53, 3, 173-84.

Grandjean P, Bellinger D, Bergman A, Cordier S, Davey-Smith G, Eskenazi B, Gee D, Gray K, Hanson M, Hazel V, Heindel J, Heinzow B, Hertz-Picciotto I, et al., The faroes statement: human health effects of developmental exposure to chemicals in our environment, *Basic and Clinical Pharmacology and Toxicology*, 2008, 102, 73–75.

Grandjean P, Landrigan P, Neurobehavioural effects of developmental toxicity, *Lancet neurology*. 2014, 13, 3, 330-8.

Grandjean P, Landrigan P, Developmental Neurotoxicity of Industrial Chemicals. *Lancet* 2006, 368, 2167–78.

Grindler N, Allshouse A, Jungheim E, Powell T, Jansson T, Polotsky A, OBGYN screening for environmental exposures: A call for action, *PloS one*, 2018, 13, 5, e0195375.
<https://journals.plos.org/plosone/article/file?id=10.1371/journal.pone.0195375&type=printable> accessed 11 May 20.

Goyer R, Nutrition and metal toxicity, *Am J Clinical Nutrition*, 1995, 61, 3, supp, 646S–650S.

Goyer R, Toxic and essential metal interactions, *Annual Review of Nutrition*, 1997, 17, 37–50.

Grant W, Holick M, Benefits and requirements of vitamin D for optimal health: a review, *Altern Med Rev*, 2005, 10, 94, 111.

Gray P, The Decline of Play and Rise in Children's Mental Disorders: There's a reason kids are more anxious and depressed than ever, *Psychology Today*, Depression, 26 Jan 2010
<https://www.psychologytoday.com/au/blog/freedom-learn/201001/the-decline-play-and-rise-in-childrens-mental-disorders> accessed 5 May 20.

Griffith J, Delta employees sue Lands' End alleging new uniforms made them sick, *NBC News*, 4 Jan 2020
<https://www.nbcnews.com/news/us-news/delta-employees-sue-lands-end-alleging-new-uniforms-made-them-n1110121> accessed 15 Mar 2020.

Grineski S, Incorporating health outcomes into environmental justice research: The case of children's asthma and air pollution in Phoenix, Arizona, *Environ. Hazard*, 2007, 7, 360–71.

Gross L, Scientists say lax regulation of chemicals in food packaging endangers human health, *Food and Environment Reporting Network* https://thefern.org/ag_insider/scientists-say-lax-regulation-of-chemicals-in-food-packaging-endangers-human-health/amp/ accessed 18 Mar 2020.

Guo R, Ren X, Ren H, Effects of dimethoate on rotifer *Brachionus calyciflorus* using multigeneration toxicity tests. *Journal of Environmental Science and Health, Part B*. 2012, 47, 9, 883-90.

Guth J, Denison R, Saas J, Background Paper for Reform No. 5 of the Louisville Charter for Safer Chemicals: Require Comprehensive Safety Data for All Chemicals 2005 <http://www.louisvillecharter.org/downloads/CharterBkgrdPaper5.pdf> accessed 27 Nov 2021.

Haggerty R, Rothman J, *Child Health and the Community*, New York, John Wiley & Sons, 1975.

Hales C, Carroll M, Fryar C, Ogden C, Prevalence of Obesity and Severe Obesity Among Adults: United States, 2017–2018, *NCHS Data Brief No. 360*, Feb 2020.
<https://www.cdc.gov/nchs/products/databriefs/db360.htm> accessed 8 Apr 2020.

Haley B, Mercury toxicity: Genetic susceptibility and synergistic effects, *Medical Veritas* 2, 2005, 535–542 <http://homeoint.ru/pdfs/haley.pdf> accessed 28 Jan 2018.

Haley B, Effective Mercury detoxification strategies, *Heavy Metals Summit Documentary Series*, 2018, Myers W, Schaffner C (transcript).

Hallberg Ö, Johansson O, Eger H, A melanoma trend forecast from 2002–What happened then? *Electromagnetic biology and medicine*, 2016, 35, 2, 103-5.

Hamra G, Guha N, Cohen A, Laden F, Raaschou-Nielsen O, et al., Outdoor particulate matter exposure and lung cancer: A systematic review and meta-analysis, *Env Health Persp*, 2014, 122, 9, 906-11. <https://www.ncbi.nlm.nih.gov/pubmed/24911630>

- Hanninen O, Knol A, Jantunen M, Lim T, Conrad A, et al., Environmental burden of disease in Europe: assessing nine risk factors in six countries, *Env Health Persp*, 2014, 122, 5, 439–46.
- Hansen S, Schendel D, Parner E, Explaining the Increase in the Prevalence of Autism Spectrum Disorders: The Proportion Attributable to Changes in Reporting Practices, *JAMA Pediatr*, 2015, 169, 1, 56–62.
- Hansen M, Larsen M, Cohr K, Waterborne paints: a review of their chemistry and toxicology and the results of determinations made during their use, *Scand J of work, env & health*, 1987, 473-85.
- Hashimoto S, Hagino A, Wheat germ agglutinin, concanavalin A, and lens culinalis agglutinin block the inhibitory effect of nerve growth factor on cell-free phosphorylation of Nsp100 in PC12h cells, *Cell structure and function*, 1989, 14, 1, 87-93.
- Hartley D, Rural Health Disparities, Population Health, and Rural Culture, *Am. J. Public Health*, 2004, 94, 1675–78.
- Hatch A, Technoscience, Racism, and the Metabolic Syndrome, *Handbook of Science, Technology & Society*, Eds: Kleinman D, Moore K, Routledge, Chap 2, 2014.
- Hays S, Becker R, Leung H, Aylward L, Pyatt D, Biomonitoring equivalents: A screening approach for interpreting biomonitoring results from a public health risk perspective. *Regulatory Toxicology and Pharmacology*, 2007, 47, 1, 96–109.
- Hedendahl L, Carlberg M, Hardell L, Electromagnetic hypersensitivity—an increasing challenge to the medical profession, *Reviews on environmental health*, 2015, 30, 4, 209-15.
- Heedle J, Fenech M, Stopper H, Kirsch-Volders M, Bolognesi C, Nersesyan A, Bonassi S, Hayashi M, Eckl P, Jenkins G, Thybaud V, The Micronucleus Assay in Toxicology, *Royal Society of Chemistry*, 18 Jul 2019.
- HEI: Health Effects Institute Panel on the Health Effects of Traffic-Related Air Pollution, *Traffic-Related Air Pollution: A Critical Review of the Literature on Emissions, Exposure, and Health Effects*, Health Effects Institute, Boston, 2010. Available at www.healtheffects.org.
- HEI: Health Effects Institute 1995, *Diesel Exhaust: A Critical Analysis of Emissions, Exposure, and Health Effects (A Special Report of the Institute's Diesel Working Group)*, Cambridge, MA, Health Effects Institute.
- Heindel J, Animal Models for Probing the Developmental Basis of Disease and Dysfunction Paradigm, *Basic and Clinical Pharmacology and Toxicology*, 2008, 102, 76–81.
- Heinzow A, Birger G, Endocrine Disruptors in Human Breast Milk and the Health-Related Issues of Breastfeeding. In: *Endocrine-Disrupting Chemicals in Food*. Ed. Shaw I, 2009, 322–55.
- Higginson J, Present trends in cancer epidemiology, *Proc Can Cancer Conf*, 1969, 8, 40-75.
- Hime H, et al., Review of the health impacts of emission sources, types and levels of particulate matter air pollution in ambient air in NSW, 2015. NSW Ministry of Health. Eds: Woolcock Institute of Medical Research, Centre for Air Quality and Health Research and Evaluation.
- Hird J, Environmental policy and equity: The case of Superfund, *J. Policy Anal. Manag*, 1993, 12, 323–43.
- Högberg G, Gustafsson S, Hällström T, Gustafsson T, Klawitter B, Petersson M, Depressed adolescents in a case-series were low in vitamin D and depression was ameliorated by vitamin D supplementation, *Acta Paediatrica*, 2012, 101, 7, 779-83.

Holick M, Chen T, Vitamin D deficiency: a worldwide problem with health consequences, *Am J Clinical Nutrition*, 2008, 87, 4, 1080S–1086S.

Holland N, Fucic A, Merlo D, Sram R, Kirsch-Volders M, Micronuclei in neonates and children: effects of environmental, genetic, demographic and disease variables, *Mutagenesis*, 2011, 26, 1, 51-6.

Hong-Bae Kim, Jae-Yong Shim, Byoungjin Park, Yong-Jae Lee, Long-term exposure to air pollutants and cancer mortality: a meta-analysis of cohort studies, *Int J Env Res Public Health*, 2018, 15, 11, 2608. Published online 2018 Nov 21.

Horel S, A toxic affair – how the chemical lobby blocked action on endocrine disrupting chemicals, 2015 <http://corporateeurope.org/food-and-agriculture/2015/05/toxic-affair-how-chemical-lobby-blocked-action-hormone-disrupting> accessed 19 May 20.

Houlihan J, Brody C, What’s in my baby’s food? Healthy babies bright futures, Oct 2019 https://www.healthybabyfood.org/sites/healthybabyfoods.org/files/2019-10/BabyFoodReport_FULLREPORT_ENGLISH_R5b.pdf accessed 11 May 20.

Hoyer M, Baldauf R, Scarbro C, Barres J, Keeler G, Mercury emissions from motor vehicles, Report for the U.S. Environmental Protection Agency, 2004. <http://infohouse.p2ric.org/ref/43/42656.pdf>

Hueper W, Occupational tumors and allied diseases, Springfield Ill., Charles C Thomas, 1942.

Hueper W, Adventures of a physician in occupational cancer: a medical cassandra’s tale. unpublished autobiography, 1976, 290-91.

Huff Post: Huffington Post, What’s in your fruit chew snacks? 31 Aug 2012 https://www.huffpost.com/entry/fruit-chew-snacks-ingredients_n_1304369 accessed 16 Mar 2020.

Husmann C, Frank M, Schmidt B, Jöckel K, Antel J, Reissner V, Libuda L, Hebebrand J, Föcker M, Low 25 (OH)-vitamin D concentrations are associated with emotional and behavioral problems in German children and adolescents, *PloS one*, 2017, 12, 8.

Hutchinson E, Charles P, Hester S, Thomas B, Trudgian D, Martínez-Alonso M, Fodor E, Conserved and host-specific features of influenza virion architecture, *Nature communications*, 2014, 16, 5, 1, 1-1.

Hyman M, Betrayal: The autoimmune disease solution they’re not telling you, Documentary Series, Episode 4 Transcript, 2017

IARC: International Agency for Research on Cancer, Press Release N° 224, Lyon/London, 3 February 2014 https://www.iarc.fr/wp-content/uploads/2018/07/pr224_E.pdf accessed 16 Mar 2019.

IARC (International Agency for Research on Cancer) Working Group, Glyphosate, In: Some organophosphate insecticides and herbicides: diazinon, glyphosate, malathion, parathion, and tetrachlorvinphos, IARC Monogr Prog, 2015, 112, 1–92.

IHME: Institute for Health Metrics and Evaluation. GBD compare data visualization. Seattle, WA: IHME, University of Washington, 2016. Available from: <https://vizhub.healthdata.org/gbd-compare/>

Jafari M, Rahmatian A, Sayehmiri F, Rafiei M, The relationship between the level of copper, lead, mercury and autism disorders: a meta-analysis, *Pediatric Health Med Ther*, 2020, 11, 369-378.

Jeschke J, Lokatis S, Bartram I, Tockner K, Knowledge in the dark: scientific challenges and ways forward, *Facets J*, 26 August 2019, <https://www.facetsjournal.com/doi/full/10.1139/facets-2019-0007> accessed 2 May 20.

Jimenez T, Cancer, Live Longer Feel Better, Doco Series, Feb 2020
<https://www.livelongerfeelbetter.com/cancer-e5/> accessed 9 Feb 2020.

Jin H, Mao L, Xie J, Zhao M, Bai X, Wen J, Shen T, Wu P, Poly-and perfluoroalkyl substance concentrations in human breast milk and their associations with postnatal infant growth, *Science of The Total Environment*, 11 Jan 2020,136417.

Johns L, Ferguson K, Cantonwine D, McElrath T, Mukherjee B, Meeker J, Urinary BPA and phthalate metabolite concentrations and plasma vitamin D levels in pregnant women: a repeated measures analysis, *Env health persp*, 2017, 125, 8, 087026.

Johnson B, Cancer, Live Longer Feel Better, Doco Series, Ep5, Feb 2020
<https://www.livelongerfeelbetter.com/cancer-e5/> accessed 9 feb 2020.

Johnson A, Williams R, A model to estimate influent and effluent concentrations of estradiol, estrone, and ethinylestradiol at sewage treatment works, *Environ. Sci. Technol.*, 2004, 38, 3649–58.

Johnson A, Moira C, MacLean L, Atkins E, Dybuncio A, Cheng F, Enarson D, Respiratory abnormalities among workers in an iron and steel foundry, *Occ Env Med*, 1985, 42, 2, 94-100.

Kaati G, Bygren L, Edvinsson S, Cardiovascular and diabetes mortality determined by nutrition during parents' and grandparents' slow growth period, *European J Human Genetics*, 2002, 10, 682–88.

Karrer C, Andreassen M, von Goetz N, Sonnet F, Sakhi A, Hungerbühler K, Dirven H, Husøy T, The EuroMix human biomonitoring study: Source-to-dose modelling of cumulative and aggregate exposure for the bisphenols BPA, BPS, and BPF and comparison with measured urinary levels, *Environ Int.*, 2020, 136, 105397.

Katchy A, Pinto C, Jonsson P, Nguyen-Vu T, Pandelova M, Riu A, Schramm K, Samarov D, Gustafsson J, Bondesson M, Williams C, Coexposure to phytoestrogens and bisphenol a mimics estrogenic effects in an additive manner, *toxicological sciences*, 2014, 138, 1, 21-35.

Kaufman D, Erlander M, Clare-Salzler M, Atkinson M, Maclaren N, Tobin A, Autoimmunity to two forms of glutamate decarboxylase in insulin-dependent diabetes mellitus, *J Clinical Investigation*, 1992, 89, 1, 283–92.

Kearns J, Moving towards transformational WASH, *Lancet Global Health*, 2019, 7, 11, e1493.

Kern J, Geier D, Homme K, Geier M, Examining the evidence that ethylmercury crosses the blood-brain barrier, *Env Toxicol Pharma.*, 2019, 74, 103312.

Kharrazian D, Environmental toxins: the hidden drivers of disease, *Betrayal: The autoimmune solution they're not telling you*, Ep 5 Transcript, Doco Series, 2017.

Kim B, Cho S, Kim Y, Shin M, Yoo H, Kim J, Yang Y, Kim H, Bhang S, Hong Y, Phthalates exposure and attention-deficit/hyperactivity disorder in school-age children, *Biological psychiatry*, 2009, 15, 66, 10, 958-63.

Kleisariis C, Sfakianakis C, Papathanasiou I, Health care practices in ancient Greece: The Hippocratic ideal, *J Med Ethics Hist Med*, 2014, 7, 6.

Klinghardt D, Biophysics of heavy metal detox, *Heavy metals summit documentary series*, 2018, by Myers W, Schaffner C (transcript).

Koelmans A, Nor N, Hermsen E, Kooi M, Mintenig S, De France J, Microplastics in freshwaters and drinking water: Critical review and assessment of data quality, *Water research*, 28 Feb 2019.

Koller L, Immunotoxicology of heavy metals, *Int. J immunopharmacology*, 1980, 2, 4, 269-79.

Koller L, Immunosuppression produced by lead, cadmium, and mercury, *Am. J. Vet. Res. US*, 1973, 34, 11.

Kortenkamp A, Faust M, Scholze M, et al., Low-level exposure to multiple chemicals: reason for human health concerns, *Env Health Persp*, 2007, 115, (suppl 1), 106–14.

Kosuth M, Mason S, Wattenberg E, Anthropogenic contamination of tap water, beer, and sea salt, *PLoS One*, 2018, 13, 4, e0194970.

Koutsos A, Tuohy K, Lovegrove J, Apples and cardiovascular health--is the gut microbiota a core consideration? *Nutrients*, 2015, 7, 6, 3959–98. <https://doi.org/10.3390/nu7063959>

Krishnan K, Betrayal: The autoimmune disease solution they're not telling you, Documentary Series, Episode 3 Transcript, 2017.

Krzewska A, Ben-Skowronek I, Effect of associated autoimmune diseases on type 1 diabetes mellitus incidence and metabolic control in children and adolescents, *BioMed research international*, Oct 2016.

Kubsad D, Nilsson E, King S, Sadler-Riggelman I, Beck D, Skinner M, Assessment of glyphosate induced epigenetic transgenerational inheritance of pathologies and sperm epimutations: generational toxicology, *Scientific Reports*, 2019, 9, 6372.

Kuchroo V, Ohashi P, Sartor R, Vinuesa C, Dysregulation of immune homeostasis in autoimmune diseases, *Nature Medicine*, 2012, 18, 1, 42–47.

Kumar S, Behari J, Sisodia R, Impact of microwave at x-band in the aetiology of male infertility. *Electromagnetic Electromagn Biol Med*, 2012, 31, 3, 223–32.

Kuper H, Adami H, Trichopoulos D, Infections as a major preventable cause of human cancer, *J Int Med*, 2001, 249, 61–74.

Kuzawa C, Fetal origins of developmental plasticity: Are fetal cues reliable predictors of future nutritional environments? *Am J Human Biology*, 17, 1, 5–21.

Kuzawa C, Sweet E, Epigenetics and the embodiment of race: Developmental origins of US racial disparities in cardiovascular health, *Am J Human Biology*, 2009, 21, 1, 2–15.

Laborde A, Tomasina F, Bianchi F, Bruné M, Buka I, Comba P, Corra L, Cori L, Duffert C, Harari R, Iavarone I, Children's health in Latin America: the influence of environmental exposures, *Env health persp*, 2015, 123, 3, 201-9.

Lamb IV J, Boffetta P, Foster W, Goodman J, Hentz K, Rhomberg L, Staveley J, Swaen G, Van Der Kraak G, Williams A, Critical comments on the WHO-UNEP state of the science of endocrine disrupting chemicals–2012, *Reg Tox Pharmacology*, 2014, 69, 1, 22-40.

Landecker H, Food as exposure: Nutritional epigenetics and the new metabolism, *BioSocieties*, 2011, 6, 2, 167–94.

Landrigan P, Forman J, Chemicals and children's health: the early and delayed consequences of early exposure, paper presented at WHO Forum Budapest 2006.

Layt S, Slowly but steadily, more children are getting cancer, research finds, *Brisbane Times*, 16 Feb 2020, Quote from: Assoc Prof D Youlden <https://www.brisbanetimes.com.au/national/queensland/slowly-but-steadily-more-children-are-getting-cancer-research-finds-20200213-p540n5.html> accessed 4 May 20.

Ledowsky C, Methylation, epigenetics and beyond, FX Omics, Ep 5, Transcript, 2019
<https://www.fxmedicine.com.au/content/methylation-epigenetics-and-beyond-carolyn-ledowsky>
accessed 12 May 20.

Ledowsky C, The Why Infertility Is Rising, Transcript, Genius of Your Genes Summit, hosted by Donna Gates 2020

Lee J, Cancer as a metabolic disease: on the origin, management, and prevention of cancer, 2012, New Jersey, Wiley ISBN: 978-0-470-58492-7.

Lee S, Here Are The Ways That PFAS chemicals might cause cancer, a new study says: PFAS chemicals, which have turned up in drinking water in cities across the US, have been linked to non-Hodgkin's lymphoma and kidney, testicular, prostate, breast, liver, and ovarian cancers, Buzz Feed News, 3 Mar 2020 <https://www.buzzfeednews.com/article/stephaniemlee/pfas-cancer-study> accessed 19 Apr 20.

Lee J, Lee J, Kim K, Shin Y, Kim J, Kim S, Kim H, Kim P, Park K, PFOA-induced metabolism disturbance and multi-generational reproductive toxicity in *Oryzias latipes*, J hazardous materials, 2017, 15, 340, 231-40.

Lee D, Kim M, Lim Y, Lee N, Hong Y, Prenatal and postnatal exposure to di-(2-ethylhexyl) phthalate and neurodevelopmental outcomes: A systematic review and meta-analysis, Env research, 2018, 1, 167, 558-66.

Lelieveld J, Klingmüller K, Pozzer A, Pöschl U, Fnais M, Daiber A, Münzel T, Cardiovascular disease burden from ambient air pollution in Europe reassessed using novel hazard ratio functions, European heart journal, 12 Mar 2019.

Lepeule J, Laden F, Dockery D, Schwartz J, Chronic exposure to fine particles and mortality: an extended follow-up of the Harvard Six Cities study from 1974 to 2009, Env Health Persp, 2012, 120, 7, 965-70.

Letellier N, et al. Association of air quality reduction with incident dementia: effects of natural course and hypothetical air pollutant interventions using g-computation. In 2021 Alzheimer's Association International Conference 2021 Jul 26. ALZ.

Levin S, Monsanto found liable for California man's cancer and ordered to pay \$80m in damages, Agrochemical corporation found responsible for Roundup weedkiller's health risks in 'bellwether' federal trial, Guardian, 29 Mar 2019 <https://www.theguardian.com/business/2019/mar/27/monsanto-trial-verdict-cancer-jury#maincontent> accessed 18 Apr 2020.

Levine H, Jørgensen N, Martino-Andrade A, Mendiola J, Weksler-Derri D, Mindlis I, Pinotti R, Swan S, Temporal trends in sperm count: a systematic review and meta-regression analysis, Human reproduction update, 2017, 23, 6, 646-59.

Levy J, Greco S, Spengler J, The importance of population susceptibility for air pollution risk assessment: a case study of power plants near Washington, DC, Env Health Persp, 2002, 110, 12, 1253-60.

Lijinski W, Epstein S, Nitrosamines as environmental carcinogens, Nature, 1970, 225, 21-23.

Lipski E, Betrayal: The autoimmune disease solution they're not telling you, Documentary Series, Episode 1 Transcript, 2017.

Lewis R, Johns L, Meeker J, Serum biomarkers of exposure to perfluoroalkyl substances in relation to serum testosterone and measures of thyroid function among adults and adolescents from NHANES 2011–2012, Int J env research public health, 2015, 12, 6, 6098-114.

Li D, Shi Y, Yang L, et al., Microplastic release from the degradation of polypropylene feeding bottles during infant formula preparation, *Nat Food*, 2020, 1, 746–54.

Linhart C, Talasz H, Morandi E, Exley C, Lindner H, Taucher S, Egle D, Hubalek M, Concin N, Ulmer H, Use of underarm cosmetic products in relation to risk of breast cancer: a case-control study, *EBioMedicine*, 2017, 21, 79-85.

Lione A, The prophylactic reduction of aluminium intake, *Food Chem Toxicol*, 1983, 21, 1, 103-109.

Liu N, Miyashita L, Mcphail G, Thangaratnam S, Grigg J, Late Breaking Abstract - Do inhaled carbonaceous particles translocate from the lung to the placenta? *European Respiratory Journal*, 2018, 52 (suppl 62) PA360 https://erj.ersjournals.com/content/52/suppl_62/PA360 accessed 25 Mar 2020.

Liu P, Stenger S, Li H, Wenzel L, Tan B, Krutzik S, Ochoa M, Schaubert J, Wu K, Meinken C, Kamen D, Toll-like receptor triggering of a vitamin D-mediated human antimicrobial response, *Science*, 2006, 311, 5768, 1770-3.

Lotter V, Epidemiology of autistic conditions in young children, *Social Psychiatry*, 1966, 1, 3, 124–135.

LU: Lancaster University, Toxic air pollution nanoparticles discovered in the human brain, News and Blogs, 5 Sep 2016 <https://www.lancaster.ac.uk/news/articles/2016/toxic-air-pollution-nanoparticles-discovered-in-the-human-brain/> accessed 24 Mar 2020.

Loomis D, Huang W, Chen G, The International Agency for Research on Cancer (IARC) evaluation of the carcinogenicity of outdoor air pollution: focus on China, *Chin J Cancer*, 2014, 33, 4, 189-96.

Lusher A, Hollman P, Mendoza-Hill J, Microplastics in Fisheries and Aquaculture: Status of Knowledge on Their Occurrence and Implications for Aquatic Organisms and Food Safety, 2017, FAO Fisheries and Aquaculture Technical Paper No. 615. Rome, Italy.

Maffini M, Neltner T, Brain drain: the cost of neglected responsibilities in evaluating cumulative effects of environmental chemicals, *J Epidemiol Community Health*, 2015, 69, 5, 496-9.

Maher B, Ahmed I, Karloukovski V, MacLaren D, Foulds P, Allsop D, Calderon-Garciduenas L, Magnetite pollution nanoparticles in the human brain, *Proceedings of the National Academy of Sciences of the United States of America*, 2016, 113, 39, 10797–801.

Malterre T, Betrayal: The autoimmune disease solution they're not telling you, Documentary Series, Episode 1 Transcript, 2017.

Mandriota S, Tenan M, Ferrari P, Sappino A, Aluminium chloride promotes tumorigenesis and metastasis in normal murine mammary gland epithelial cells, *Int J Cancer*, 2016, 139, 12, 2781-90.

Mandriota S, A case-control study adds a new piece to the aluminium/breast cancer puzzle, *EBioMedicine*, 2017, 22, 22-23.

Markowitz G, Rosner D, Lead wars: The politics of science and the fate of America's children, Berkeley, CA, University of California Press, 2013.

Marshall T, Heil T, Electrosmog and autoimmune disease, *Immunologic research*, 2017, 1, 65, 1, 129-35.

Mason S, Welch V, NeratkoSynthetic J, Polymer Contamination in Bottled Water, University of Fredonia-State, New York, 2018.

Mayer A, Historical changes in the mineral content of fruits and vegetables, *British Food Journal*, 1997, 99, 6, 207–11.

Mayo Clinic, Cancer, Causes, What causes gene mutations? <https://www.mayoclinic.org/diseases-conditions/cancer/symptoms-causes/syc-20370588> accessed 15 Apr 20.

McGrath K, An earlier age of breast cancer diagnosis related to more frequent use of antiperspirants/deodorants and underarm shaving, *European Journal of Cancer*, 2003, 12, 6, 479–85.

McKenna M, Diabetes mystery: why are type 1 cases surging? Researchers are baffled by the worldwide increase in type 1 diabetes, the less common form of the disease, *Scientific American, Health*, 1 Feb 2012 <https://www.scientificamerican.com/article/a-diabetes-cliffhanger/> accessed 4 May 20.

McKenzie L, Blair B, Hughes J, Allshouse W, Blake N, Helmig D, Milmo P, Halliday H, Blake D, Adgate J, Ambient nonmethane hydrocarbon levels along colorado's northern front range: acute and chronic health risks, *Environ Sci Technol*, 2018, 52, 8, 4514-25.

McLachlan J, Environmental signaling: from environmental estrogens to endocrine-disrupting chemicals and beyond, *Andrology*, 2016, 4, 4, 684-94.

Meggs W, History of the Rise and Fall of Environmental Medicine in the United States, *Ecopsychology*, 2017, 9, 2.

Melnick D, Luckmann F, Sorbic acid as a fungistatic agent for foods, Migration of sorbic acid from wrapper into cheese, Thirteenth Annual Meeting of the Institute of Food Technologists, Boston, Massachusetts, June 24, 1953, First published: Jan 1954 <https://doi.org/10.1111/j.1365-2621.1954.tb17420.x> accessed 19 Apr 20.

Menees S, Chey W, The gut microbiome and irritable bowel syndrome, *F1000Research*, 2018, 7.

Menni C, Lin C, Cecelja M, Mangino M, Matey-Hernandez M, Keehn L, Mohny R, Steves C, Spector T, Kuo C, Chowienzyk P, Gut microbial diversity is associated with lower arterial stiffness in women, *European heart journal*, 2018, 39, 25, 2390-7.

Mercola J, Seyfried T, TTAC eastern medicine: journey through asia, Episode 5, Apr 2019 <https://www.bitchute.com/video/XCSQ9Lm0JBqj/> accessed 16 Apr 20.

Meyers J, Winans B, Kelsaw E, Murthy A, Gerber S, Lawrence B, Environmental cues received during development shape dendritic cell responses later in life, *PloS one*, 2018, 13, 11, e0207007.

Michaels D, *Doubt is their product: How industry's assault on science threatens your health*, Oxford, UK, Oxford University Press, 2008.

Miller F, Pollard K, Parks C, et al., Criteria for environmentally associated autoimmune disease, *J Autoimmunity*, 2012, 39, 4, 253–58.

Miller M, Melanie A, Arcus A, Brown J, Morry D, Sandy M, Differences between children and adults: implications for risk assessment at california EPA. *Int J Toxicology*, 2002, 21, 403–18.

Mintenig S, Löder M, Primpke S, Gerdt G, Low numbers of microplastics detected in drinking water from ground water sources, *Sci. Total Environ.*, 2019, 648, 631-35.

Mishra P, Samarth R, Pathak N, Jain S, Banerjee S, Maudar K, Bhopal gas tragedy: review of clinical and experimental findings after 25 years, *Int J occupational medicine and env health*, 2009, 1, 22, 3, 193-202.

Mohai, P.; Bryant, B. Environmental racism: Reviewing the evidence. In *Race and the Incidence of Environmental Hazards: A Time for Discourse*; Westview press: Boulder, CO, USA, 1992; p. 164.

Monaco K, Review: PFAS Exposure Tied to Kidney Disease: Researchers warn environmental exposure 'threat' to kidneys, MedPage Today, 13 Sep 2018.

Monosson E, Chemical mixtures: considering the evolution of toxicology and chemical assessment, *Env Health Persp*, 2005, 113, 4, 383–90.

Montague P, Shifting the Burden of Proof, *Rachel's Environment & Health Weekly*, #491, 1996.

Moon J, The role of vitamin D in toxic metal absorption: a review, *J American College of Nutrition*, 1994, 13, 6, 559–564.

More S, Vartak A, Vince R, The butter flavorant, diacetyl, exacerbates β -amyloid cytotoxicity, *Chemical research in toxicology*, 2012, 25, 10, 2083-91.

Morello-Frosch R, Pastor M, Porras C, Sadd J, Environmental justice and regional inequality in Southern California: Implications for future research, *Env Health Persp*, 2002, 110, 149–54.

Morello-Frosch R, Brody J, Brown P, Altman R, Rudel R, Pérez C, Toxic ignorance and right-to-know in biomonitoring results communication: A survey of scientists and study participants, *Env Health*, 2009, 8, 1, 6.

Morrison H, Villeneuve P, Lubin J, Schaubel D, Radon-progeny exposure and lung cancer risk in a cohort of Newfoundland fluorspar miners, *Radiat Res*, 1998, 150, 58–65.

Muncke J, Andersson A, Backhaus T, et al., Impacts of food contact chemicals on human health: a consensus statement, *Env Health*, 2020, 19, 25.

Nash L, *The Fruits of Ill-Health: Pesticides and Workers' Bodies in Post-World War II California*, *Osiris*, 2004, 19, 203-19.

Nassar N, Bower C, Barker A, Increasing prevalence of hypospadias in Western Australia, 1980-2000, *Archives of disease in childhood*, 2007, 92, 7, 580-4.

NCBI: National Center for Biotechnology Information. PubChem Database. 2,5-Dichlorophenol, CID=66, https://pubchem.ncbi.nlm.nih.gov/compound/2_5-Dichlorophenol accessed 30 Apr 2020.

NCEHS: National Center for Environmental Health Strategies, Indoor Pollutants, health risks and involuntary exposure to fragrances, *The Delicate Balance*, 27 Mar 1990, 5.

NDSS: National Diabetes Services Scheme, Understanding type 1 diabetes fact sheet <https://www.ndss.com.au/about-diabetes/resources/find-a-resource/understanding-type-1-diabetes-fact-sheet/> accessed 5 May 20.

Needham L, Calafat A, Barr D, Uses and Issues of Biomonitoring, *International Journal of Hygiene and Environmental Health* 2007, 210, 3-4, 229-38.

Neggers Y, Increasing prevalence, changes in diagnostic criteria, and nutritional risk factors for autism spectrum disorders, *ISRN nutrition*, 2014, 514026.

Neltner T, Think PFAS in food packaging are safe simply because FDA accepted their use? Think again, Environmental Defense Fund, 13 Nov 2019 <http://blogs.edf.org/health/2019/11/13/think-again-pfas-food-packaging-safety/> accessed 19 Mar 2020.

Newbury J, Arseneault L, Beevers S, et al., Association of Air Pollution Exposure With Psychotic Experiences During Adolescence, *JAMA Psychiatry*, Published online 27 Mar 2019.

Newacheck P, Budetti P, McManus P, Trends in childhood disability, Am J Public Health, 1984, 74, 3, 232 – 6.

Newbold R, Jefferson W, Padilla-Banks E, Haseman J, Developmental exposure to diethylstilbestrol (DES) alters uterine response to estrogens in prepubescent mice: low versus high dose effects, Reproductive toxicology, 2004, 18, 3, 399-406.

NHS, Mental Health of Children and Young People in England, 2017, Official statistics, 2018
<https://digital.nhs.uk/data-and-information/publications/statistical/mental-health-of-children-and-young-people-in-england/2017/2017>

NIH: Eunice Kennedy Shriver National Institute of Child Health and Human Development, 9 Apr 2019, Kids living near major roads at higher risk of developmental delays, ScienceDaily, Retrieved 17 Mar 2020
www.sciencedaily.com/releases/2019/04/190409164002.htm

NIH: US National Institute of Environmental Health Services, Endocrine disruptors,
<https://www.niehs.nih.gov/health/topics/agents/endocrine/index.cfm> accessed 17 May 2020.

NIH, US National Library of Medicine, Medline Plus Medical Encyclopedia, Autoimmune disorders
<https://medlineplus.gov/ency/article/000816.htm> accessed 9 Apr 2020b

NIH, National Cancer Institute, Cancer in Children and Adolescents, What are the possible causes of cancer in children? <https://www.cancer.gov/types/childhood-cancers/child-adolescent-cancers-fact-sheet> accessed 25 May 2020c.

NIH, National Cancer Institute, About Cancer, Cancer Causes and Prevention, Risk Factors, Common Cancer Myths and Misconceptions, Antiperspirants/Deodorants and Breast Cancer, Is there a link between antiperspirants or deodorants and breast cancer? <https://www.cancer.gov/about-cancer/causes-prevention/risk/myths/antiperspirants-fact-sheet> accessed 19 Apr 2020

NRC: National Research Council, Toxicity testing in the 21st century: a vision and a strategy, National Academies Press, 2007.

Null G, Dean C, Feldman M, Rasio D, Smith D, Death by Medicine, Praktikos Books, 2010.

Obodovskiy I, The effect of chemicals on biological structures. In: Fundamentals of Radiation and Chemical Safety, Elsevier, Amsterdam, The Netherlands, 2015, 133–79.

O'Bryan T, Intestinal permeability: the gateway to autoimmunity, Betrayal: The autoimmune solution they're not telling you, Ep 2 Transcript, Doco Series, 2017.

O'Brien T, Betrayal: The autoimmune disease solution they're not telling you, Documentary Series, Episode 1-7 Transcripts, 2017.

Ochoa-Acuña H, Frankenberger J, Hahn L, Carbajo C, Drinking-water herbicide exposure in Indiana and prevalence of small-for-gestational-age and preterm delivery, Env health persp, 2009a, 117, 10, 1619-24.

Ochoa-Acuña H, Carbajo C, Risk of limb birth defects and mother's home proximity to cornfields, Science of the total environment, 2009b, 407, 15, 4447-51.

OGDP: Office of Genomics and Disease Prevention: Centers for Disease Control and Prevention, Department of Health and Human S, Gene-Environment Interaction Fact Sheet, 2000.
<http://www.ashg.org/pdf/cdc%20gene-nvironment%20interaction%20fact%20sheet.pdf> accessed 27 May 2017.

Oris J, Geisy J, The photo-enhanced toxicity of anthracene to juvenile sunfish (*Lepomis* spp.), *Aquatic Toxicol*, 1985, 6, 133–46.

Osborne P, The autoimmune epidemic: Root causes and solutions, *Betrayal: The autoimmune solution they're not telling you*, Ep 1 Transcript, Doco Series, 2017.

Otitolaju A, Obe I, Adewale O, Otubanjo O, Osunkalu V, Preliminary study on the reduction of sperm head abnormalities in mice, *Mus musculus*, exposed to radiofrequency radiations from global system for mobile communication base stations, *Bull Environ Contam Toxicol*, 2010, 84, 1, 51–4.

Ovelgönne J, Koninkx J, Pusztai A, Bardocz S, Kok W, Ewen S, Hendriks H, Van Dijk J, Decreased levels of heat shock proteins in gut epithelial cells after exposure to plant lectins, *Gut*, 2000, 46, 5, 680-8.

Pacher P, Beckman J, Liaudet L, Nitric oxide and peroxynitrite in health and disease, *Physiological reviews*, 2007, 87, 1, 315-424.

Packham A, Air Pollution Can Impact Babies, In: *The Womb, Evidence Shows: Scientists discovered sooty particles in pregnant women's placentas*, Huffington Post, 16 Sept 2018.

PAHO: Pan American Health Organization, 2017 main health problems and challenges, In: *Health in the Americas*. <https://www.paho.org/salud-en-las-americas-2017/> accessed 7 Apr 20.

Palmer J, Wise L, Hatch E, Troisi R, Titus-Ernstoff L, Strohsnitter W, et al., Prenatal diethylstilbestrol exposure and risk of breast cancer. *Cancer Epidemiology, Biomarkers & Prevention*, 2006, 15, 1509–14.

Paoloni-Giacobino A, Post genomic decade-the epigenome and exposome challenges, *Swiss medical weekly*, 2011, 141, 5152.

Park C, Barakat R, Ulanov A, Li Z, Lin P, Chiu K, Zhou S, Perez P, Lee J, Flaws J, Ko C, Sanitary pads and diapers contain higher phthalate contents than those in common commercial plastic products, *Reproductive Toxicology*, 2019, 84, 114-21.

Park C, et al. Associations between long-term air pollution exposure and plasma amyloid beta in very old adults. In *2021 Alzheimer's Association International Conference 2021 Jul 26*. ALZ.

Pastor M, Sadd J, Hipp J, Which came first? Toxic facilities, minority move-in, and environmental justice, *J Urban Affairs*, 2001, 23, 1–21.

Pawankar R, Canonica G, Holgate S, Lockey R, (eds) *WAO White book on Allergy 2011–2012* [Internet]. Milwaukee, WI, US, World Allergy Organization, 2013, 228.

Peachman R, Are banned drugs in your meat? How did they get there? What's known about the risks? And what can be done to keep these drugs off your plate? *Consumer Reports*, 27 Nov 18
<https://www.consumerreports.org/food-safety/are-banned-drugs-in-your-meat/> accessed 1 Apr 20.

Peat J, van den Berg R, Green W, Mellis C, Leeder S, Woolcock A, Changing prevalence of asthma in Australian children, *BMJ*, 1994, 308, 6944, 1591–96.

Pedersen M, Giorgis-Allemand L, Bernard C, Aguilera I, et al., Ambient air pollution and low birthweight: a European cohort study (ESCAPE), *Lancet Respir Med*, 2013, 1, 9, 695-704. <https://www.ncbi.nlm.nih.gov/pubmed/24429273>

Pedersen M, Wichmann J, Autrup H, Dang D, Decordier I, Hvidberg M, Bossi R, Jakobsen J, Loft S, Knudsen L, Increased micronuclei and bulky DNA adducts in cord blood after maternal exposures to traffic-related air pollution, *Environmental research*, 2009, 109, 8, 1012-20.

Peinado H, et al., Melanoma exosomes educate bone marrow progenitor cells toward a pro-metastatic phenotype through MET, *Nature medicine*, 2012, 18, 883–91.

Pembrey M, Time to take epigenetic inheritance seriously, *European Journal of Human Genetics*, 2002, 10, 669–70.

Pembrey M, et al., Sex-specific, male-line transgenerational responses in humans, *European J Human Genetics*, 2006, 14, 2, 159–166.

Pennington J, Aluminium content of foods and diets, *Food Addit Contam*, 1988, 5, 2, 161-232.

Perera F, Frederica P, Jedrychowski W, Rauh V, Whyatt R, Molecular epidemiologic research on the effects of environmental pollutants on the fetus. *Env Health Persp*, 1999, 107, 451–60.

Peters J, Boynton-Jarrett R, Sandel M, Prenatal environmental factors influencing IgE levels, atopy and early asthma, *Current opinion in allergy and clinical immunology*, 2013, 13, 2, 187-92.

Pizzorno J, Ruscio M, Environmental Toxins Have Become the Primary Drivers of Chronic Disease, Podcast Interview, 1 Apr 2020 <https://drruscio.com/environmental-toxins-drivers-of-chronic-disease/>

Pizzorno J, Is the Diabetes Epidemic Primarily Due to Toxins? *Integr Med (Encinitas)*, 2016, 15, 4, 8-17.

Pollard K, Hultman P, Kono D, Toxicology of autoimmune diseases, *Chemical Research in Toxicology*, 2010, 23, 3, 455–466.

Pompa D, What is Generational Toxicity? *Safe Detox Methods*, 10 Sept 2019. https://drpompa.com/uncategorized/generational-toxicity-safe-detox-methods/?utm_source=PublicActiveCampaign&utm_medium=Email&utm_term=Generational-Toxicity-09-11-19%2CSeptember112019&utm_campaign=CytoDetox accessed 10 May 20.

Pope C, Burnett R, Thun M, Calle E, Krewski D, Ito K, Thurston G, Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution, *JAMA*, 2002, 287, 9, 1132-41. <https://www.ncbi.nlm.nih.gov/pubmed/11879110>

Portier C, Armstrong B, Baguley B, et al., Differences in the carcinogenic evaluation of glyphosate between the International Agency for Research on Cancer (IARC) and the European Food Safety Authority (EFSA), *J Epidemiol Community Health*, 2016, 70, 741-45.

Post G, Gleason J, Cooper K, Key scientific issues in developing drinking water guidelines for perfluoroalkyl acids: Contaminants of emerging concern, *PLoS Biology*, 2017, 15, 12, e2002855.

Prata J, Silva A, Walker T, Duarte A, Rocha-Santos T, COVID-19 pandemic repercussions on the use and management of plastics. *Environmental Science & Technology*, 2020, 54, 13, 7760–65.

Proctor R, *Cancer Wars*, Basic Books, 1995.

Prunicki M, Cauwenberghs N, Lee J, et al., Air pollution exposure is linked with methylation of immunoregulatory genes, altered immune cell profiles, and increased blood pressure in children, *Sci Rep* 11, 2021, 4067.

Qian Di M, Wang Y, Zanobetti A, Wang Y, Koutrakis P, Choirat C, Dominici F, Schwartz J, Air Pollution and mortality in the Medicare Population, *N Engl J Med*, 2017, 376, 26, 2513-22.

Qihong Deng, Lu C, Norbäck D, Bornehag C, Zhang Y, Liu W, Yuan H, Sundell J, Early life exposure to ambient air pollution and childhood asthma in China, *Environmental Research*, 2015, 143, 83.

Raftery T, Martineau A, Greiller C, Ghosh S, McNamara D, Bennett K, Meddings J, O'Sullivan M, Effects of vitamin D supplementation on intestinal permeability, cathelicidin and disease markers in Crohn's disease: Results from a randomised double-blind placebo-controlled study, *United European Gastroenterology Journal*, 2015, 3, 3, 294–302.

Ramirez-Andreotta M, Brody J, Lothrop N, Loh M, Beamer P, Brown P, Improving environmental health literacy and justice through environmental exposure results communication, *Int J Env research and public health*, 2016, 13, 7, 690.

Rana P, Craymer L, Big tongues and extra vertebrae: the unintended consequences of animal gene editing, *Wall Street Journal*, 12 Mar 2019 <https://www.wsj.com/articles/deformities-alarm-scientists-racing-to-rewrite-animal-dna-11544808779?mod=e2tw> accessed 12 Mar 2019.

Rappaport S, Smith M, Epidemiology, environment and disease risks, *Science*, 2010, 330, 6003, 460-1.

Rask-Andersen M, Karlsson T, Ek W, Johansson Å, Gene-environment interaction study for BMI reveals interactions between genetic factors and physical activity, alcohol consumption and socioeconomic status, *PLoS genetics*, 2017, 13, 9, e1006977.

Rea W, Could multiple chemical sensitivity be the cause of your illness? Interview by J Lee, 2012. <https://www.johnleemd.com/environmental-illness.html>. accessed 22 Jul 2021.

Rea W, Patel K, Organic chemicals. In: *Reversibility of chronic disease and hypersensitivity*, CRC Press, 2017, 4, 481-596.

Rees C, Brhlikova P, Pollock A, Will HPV vaccination prevent cervical cancer?. *J Royal Society of Medicine*, 2020, 0141076819899308. <https://journals.sagepub.com/doi/10.1177/0141076819899308>

Rehfeld A, Egeberg D, Almstrup K, Petersen J, Dissing S, Skakkebaek N, EDC IMPACT: Chemical UV filters can affect human sperm function in a progesterone-like manner, *Endocrine connections*, 2018, 7, 1, 16-25.

Research UK, Cervical cancer progress falters as screening uptake hits record lows, Press Release 22 Jan 2020 <https://www.cancerresearchuk.org/about-us/cancer-news/press-release/2020-01-22-cervical-cancer-progress-falters-as-screening-uptake-hits-record-lows> accessed 26 Jan 2020.

Reuben S, U.S. Department Of Health And Human Services, National Institutes of Health, National Cancer Institute, The President's Cancer Panel, April 2010 https://deainfo.nci.nih.gov/advisory/pcp/annualreports/pcp08-09rpt/pcp_report_08-09_508.pdf

Rhodes J, Fisher M, Global epidemiology of emerging *Candida auris*, *Current Opinion in Microbiology*, 2019, 52, 84-9.

Roberts J, *Unruly Technologies and Fractured Oversight: Toward a Model for Chemical Control for the Twenty-First Century*, Chap 12, 254-68, In: *Powerless Science? Science and Politics in a Toxic World*, Eds Boudia S, Jas N, Berghahn Books, 2014.

Robinson S, Marín C, Oliveros H, Mora-Plazas M, Lozoff B, Villamor E, Vitamin D Deficiency in Middle Childhood Is Related to Behavior Problems in Adolescence, *J Nutrition*, 2020, 150, 1, 140–48.

Rogers T, *The Political Economy of Autism*, Thesis, Uni Syd, 2019.

Rohde K, Keller M, la Cour Poulsen L, Blüher M, Kovacs P, Böttcher Y, Genetics and epigenetics in obesity, *Metabolism*, 2019, 92, 37-50.

Rojas-Villarraga A, Amaya-Amaya J, Rodriguez-Rodriguez A, Mantilla R, Anaya J, Introducing polyautoimmunity: secondary autoimmune diseases no longer exist, *Autoimmune Dis*, 2012, 1–9.

Rooney M, Lutsey P, Bhatti P, Prizment A, Urinary 2,5-dichlorophenol and 2,4-dichlorophenol concentrations and prevalent disease among adults in the National Health and Nutrition Examination Survey (NHANES), *Occup Environ Med*, 2019, 76, 3, 181-88.

Rose G, Stamler J, Stamler R, et al., Intersalt: an international study of electrolyte excretion and blood pressure. Results for 24 hour urinary sodium and potassium excretion, *BMJ*, 1988, 297, 6644, 319–28.

Rosenfeld C, Microbiome disturbances and autism spectrum disorders, *Drug Metab Dispos*, 2015, 43, 1557–71.

Rutter M, Aetiology of autism: Findings and questions. *Journal of Intellectual Disability Research*, 2005, 49, 4, 231–38.

Rychlik K, Sillé F, Environmental exposures during pregnancy: Mechanistic effects on immunity. *Birth defects research*, 2019, 111, 4, 178-96.

Sadeharju K, Lönrot M, Kimpimäki T, et al., Enterovirus antibody levels during the first two years of life in prediabetic autoantibody-positive children, *Diabetologia*, 2001, 44, 7, 818–23.

Saijo Y, Kishi R, Sata F, et al., Symptoms in relation to chemicals and dampness in newly built dwellings, *Int Arch Occ Env Health*, 2004, 77, 7, 461–70.

Samsel A, Seneff S, Glyphosate pathways to modern diseases VI: Prions, amyloidoses and autoimmune neurological diseases, *J. Biol Phys Chem*, 2017, 17, 8-32.

Samsel A, Seneff S, Glyphosate's suppression of cytochrome P450 enzymes and amino acid biosynthesis by the gut microbiome: pathways to modern diseases, *Entropy*, 2013, 15, 4, 1416-63.

SAPEA, Science Advice for Policy by European Academies - A scientific perspective on microplastics in nature and society, SAPEA, Berlin, 2019.

Sappino A, Buser R, Lesne L, Gimelli S, Béna F, Belin D, Mandriota S, Aluminium chloride promotes anchorage-independent growth in human mammary epithelial cells, *J Appl Toxicol*, 2012, 32, 3, 233-43.

Sasano H, Rojas M, Silverberg S, Analysis of lectin binding in benign and malignant thyroid nodules, *Archives of pathology & laboratory medicine*, 1989, 113, 2, 186-9.

Sawyer M, Arney F, Baghurst P, Clark J, Graetz B, Kosky R, et al., Mental health of young people in Australia, DoHA, Canberra 2000 <https://www.abs.gov.au/AUSSTATS/abs@.nsf/mf/4829.0.55.001/>

Schmitt A, The invisible communities of nectar: How yeast and bacteria alter nectar characteristics, *Duluth Journal of Undergraduate Biology*, [Spring 2014](https://pubs.lib.umn.edu/index.php/djub/article/view/19), Vol 1. <https://pubs.lib.umn.edu/index.php/djub/article/view/19> accessed 16 Mar 2020

Schardein J, Chemically induced birth defects, Third Ed, New York, Marcel Dekker, 2000.

Schubert J, Riley E, Tyler S, Combined effects in toxicology-a rapid systematic testing procedure: Cadmium, mercury, and lead, *J Tox Env Health*, 1978, 4, 5-6, 197.

Schymanski D, Goldbeck H, Humpf P, Furst Analysis of microplastics in water by micro-Raman spectroscopy: release of plastic particles from different packaging into mineral water, *Water Res.*, 2018, 129, Suppl. C, 154-62.

Schatzker M, The dorito effect: the surprising new truth about food and flavor, Simon & Schuster, 2016 ISBN: 9781476724232.

Segal Y, Shoenfeld Y, Vaccine-induced autoimmunity: the role of molecular mimicry and immune crossreaction, *Cell Mol Immunol*, 2018, 15, 6, 586-94.

Selmi C, Mechanisms of environmental influences on human autoimmunity: a National Institute of Environmental Health Sciences expert panel workshop, *J Autoimmunity*, 2012, 39, 4, 272–84.

Samsel A, Seneff S, Glyphosate's suppression of cytochrome P450 enzymes and amino acid biosynthesis by the gut microbiome: pathways to modern diseases, *Entropy*, 2013, 15, 4, 1416-63.

Seralini G, Environmental toxins: the hidden drivers of disease, *Betrayal: The autoimmune solution they're not telling you*, Ep 5 Transcript, Doco Series, 2017.

Sexton K, Linder S, Cumulative risk assessment for combined health effects from chemical and nonchemical stressors, *Am J Public Health*, 2011, 101, Suppl 1, S81-8.

Sexton K, Hattis D, Assessing cumulative health risks from exposure to environmental mixtures - three fundamental questions, *Env Health Persp*, 2007, 115, 5, 825-32.

Shin S, Saito E, Inoue M, Sawada N, Ishihara J, Takachi R, Nanri A, Shimazu T, Yamaji T, Iwasaki M, Sasazuki S, Dietary pattern and breast cancer risk in Japanese women: the Japan Public Health Center-based Prospective Study (JPHC Study), *British Journal of Nutrition*, 2016, 115, 10, 1769-79.

Shoenfeld Y, Environmental toxins: the hidden drivers of disease, *Betrayal: The autoimmune solution they're not telling you*, Ep 5 Transcript, Doco Series, 2017.

Siddique H, Mental health disorders on rise among children, *The Guardian*, 22 Nov 2018 <https://www.theguardian.com/society/2018/nov/22/mental-health-disorders-on-rise-among-children-nhs-figures> accessed 5 May 20.

Sillars B, Davis W, Kamber N, Davis T, The epidemiology and characteristics of type 2 diabetes in urban, community-based young people, *Internal Medicine Journal*, 2010, 40, 850–4.

Silva E, Rajapakse N, Kortenkamp A, Something from “nothing”– eight weak estrogenic chemicals combined at concentrations below NOECs produce significant mixture effects, *Environmental science & technology*, 2002, 36, 8, 1751-6.

Simmons J, Richardson S, Speth T, Miltner R, Rice G, Schenck K, et al., Development of a research strategy for integrated technology-based toxicological and chemical evaluation of complex mixtures of drinking water disinfection byproducts, *Env Health Persp*, 2002, 110, (suppl 6), 1013–24.

Smith K, Corvalán C, Kjellström T, How much global ill health is attributable to environmental factors? *Epidemiology*, 1999, 573-84.

Smith R, Fecht D, Gulliver J, Beevers S, Dajnak D, Blangiardo M, Ghosh R, Hansell A, Kelly F, Anderson H, Toledano M, Impact of London's road traffic air and noise pollution on birth weight: retrospective population based cohort study, *BMJ*, 2017, 359.

SOE: State of the Environment: Health impacts of air pollution — Australia State of the Environment Report. Australian institute of Health and Welfare. Australian burden of disease study: impact and causes of illness and death in Australia 2011. AIHW, Canberra, 2016.

<https://soe.environment.gov.au/theme/ambient-air-quality/topic/2016/health-impacts-air-pollution>

Sollid L, Kolberg J, Scott H, Ek J, Fausa O, Brandtzaeg P, Antibodies to wheat germ agglutinin in coeliac disease, *Clinical and experimental immunology*, 1986, 63, 1, 95-100.

Soto A, Fernandez M, Luizzi M, Oles Karasko A, Sonnenschein C, Developing a marker of exposure to xenoestrogen mixtures in human serum, *Env health persp*, 1997, 105, (suppl 3), 647-54.

Sram R, Beneg I, Binkova B, Dejmeek J, Horstman D, Kotesovec F, et al., Teplice program—the impact of air pollution on human health, *Environ Health Persp*, 1996, 104, (suppl 4), 699–714.

Stanford Health Care, What causes cancer? <https://stanfordhealthcare.org/medical-conditions/cancer/cancer/cancer-causes.html> accessed 15 Apr 20.

Steliarova-Foucher E, Colombet M, Ries LAG, et al. International incidence of childhood cancer, 2001-10: a population-based registry study. *Lancet Oncol*. 2017;18(6):719-731.

Suarez-Lopez J, Amchich F, Murillo J, Denenberg J, Blood pressure after a heightened pesticide spray period among children living in agricultural communities in Ecuador, *Environmental research*, 2019, 175, 335-42.

Sugawara N, Inhibitory effect of cadmium on calcium absorption from the rat duodenum, *Archives of environmental contamination and toxicology*, 1977, 5, 1, 167-75.

Sutcliffe J, Genetics: Insights into the pathogenesis of autism. *Science* 2008, 321, 208–9.

Sznajder-Katarzyńska K, Surma M, Wiczkowski W, Cieślik E, The perfluoroalkyl substance (PFAS) contamination level in milk and milk products in Poland, *International dairy journal*, 2019, 96, 73-84.

Taylor M, Lead poisoning of Port Pirie children: a long history of looking the other way, *The Conversation*, 19 July 2012, <http://theconversation.com/lead-poisoning-of-port-pirie-children-a-long-history-of-looking-the-other-way-8296> accessed 14 Mar 2019.

Taylor K, Hoffman K, Thayer K, Daniels J, Polyfluoroalkyl Chemicals and Menopause among Women 20–65 Years of Age (NHANES), *Env Health Persp*, 2014, 122, 2, 145–50.

Tchernychev B, Wilchek M, Natural human antibodies to dietary lectins. *FEBS letters*. 1996, 397, 2-3, 139-42.

Temkin A, Hocevar B, Andrews D, Naidenko O, Kamendulis L, Application of the key characteristics of carcinogens to per and polyfluoroalkyl substances, *Int J Env research and public health*, 2020, 17, 5, 1668.

Thongprakaisang S, Thiantanawat A, Rangkadilok N, Suriyo T, Satayavivad J, Glyphosate induces human breast cancer cells growth via estrogen receptors, *Food and Chemical Toxicology*, 2013, 59, 129-36.

Tiffon C, The impact of nutrition and environmental epigenetics on human health and disease, *Int J molecular sciences*, 2018, 19, 11, 3425.

Titus-Ernstoff L, Hatch E, Hoover R, Palmer J, Greenberg E, Ricker W, Kaufman R, Noller K, Herbst A, Colton T, Hartge P, Long-term cancer risk in women given diethylstilbestrol (DES) during pregnancy, *British journal of cancer*, 2001, 84, 1, 126-33.

Tollefsbol T, Generational epigenetic inheritance. In: *Transgenerational Epigenetics 2019 Jan 1 (1-10)*, Academic Press.

TMS: The Morning Show, Childhood cancer rates in Australia expected to increase by 7 per cent in the next 20 years, Channel 7, 17 Feb 20 <https://7news.com.au/the-morning-show/childhood-cancer-rates-in-australia-expected-to-increase-by-7-per-cent-in-the-next-20-years-c-701056> accessed 5 May 20.

Trasande L, Shaffer R, Sathyanarayana S, Food additives and child health, Policy Statement, Council On Environmental Health, American Academy of Pediatrics, Pediatrics, July 2018.

Treffert D, Epidemiology of infantile autism, Archives of General Psychiatry, 1970, 22, 5, 431–38.

Tsatsakis A, Docea A, Calina D, Buga A, Zlatian O, Gutnikov S, Kostoff R, Aschner M, Hormetic Neurobehavioral effects of low dose toxic chemical mixtures in real-life risk simulation (RLRS) in rats, Food and chemical toxicology, 2019, 125, 141-9.

Turnbaugh P, Hamady M, Yatsunencko T, Cantarel B, Duncan A, Ley R, Sogin M, Jones W, Roe B, Affourtit J, Egholm M, Henrissat B, Heath A, Knight R, Gordon J, A core gut microbiome in obese and lean twins, Nature, 2009, 457, 7228, 480-4.

Turner M, Wigle D, Krewski D, Residential pesticides and childhood leukemia: a systematic review and meta-analysis, Env health persp, 2010, 118, 1, 33-41.

UC: University of California, Berkley, School of Public Health, The CHAMACOS Cohort Study 2019, <http://www.healthresearchforaction.org/sph/chamacos-cohort-study> accessed 8 May 20.

UCC: United Church of Christ. Toxic wastes and race in the united states: A national report on the racial and socio-economic characteristics of communities surrounding hazardous waste sites. United Church of Christ, Commission for Racial Justice, New York, 1987.

UCSF: University of California, San Francisco, Pediatric environment health toolkit, key concepts in pediatric environmental health, unique vulnerability of children <https://peht.ucsf.edu/search.php?pane=concepts> accessed 7 May 20.

UNEP: United Nations Environment Programme, Global chemicals outlook. towards sound management of chemicals, UNEP, Geneva, Switzerland, 2012.

UNEP/WHO: United Nations Environment Programme, World Health Organization, Eds: Bergman A, Heindel J, S. Jobling, Kidd K, Zoeller R, State of the Science of Endocrine Disrupting Chemicals (2013) <http://www.who.int/ceh/publications/endocrine/en/index.html> accessed 19 May 20.

UON: University of Nottingham, New link between gut microbiome and artery hardening discovered, Medicalxpress, May 10, 2018 <https://medicalxpress.com/news/2018-05-link-gut-microbiome-artery-hardening.html> accessed 26 Apr 20.

USA: University of South Alabama HPV-related cancer rates affect vaccine uptake in Alabama, USA Health study says, Alabama Newsletter, March 24, 2019 https://www.alabamane.wscenter.com/2019/03/24/hpv-related-cancer-rates-affect-vaccine-uptake-in-alabama-usa-health-study-says/?fbclid=IwAR0HuKIOu6K-hZM4MbmgnS1Qs_Xo-f_btMjNHjPbuerWfzKngn-jhNvSU4 accessed 3 Mar 2020.

USEPA 2002. Health Assessment Document for Diesel Engine Exhaust. EPA/600/8-90/057F. Washington, DC:U.S. Environmental Protection Agency, National Center for Environmental Assessment, Office of Research and Development.

USEPA, Chemical hazard data availability study: what do we really know about the safety of high production volume chemicals?. US Environmental Protection Agency, Office of Chemical Safety and Pollution Prevention, 1998.

USFDA: US Food & Drug Authority, FDA Home, GRAS Substances (SCOGS) Database, Food Ingredient & Packaging Inventories, SCOGS (Select Committee on GRAS Substances) <https://www.accessdata.fda.gov/scripts/fdcc/?set=SCOGS> accessed 30 Mar 2020a

USFDA: US Food & Drug Authority, Food, Food Ingredients & Packaging, Generally Recognized as Safe (GRAS), GRAS Substances (SCOGS) Database <https://www.fda.gov/food/generally-recognized-safe-gras/gras-substances-scogs-database> accessed 30 Mar 2020b.

USGAO: US General Accounting Office, Siting of hazardous waste landfills and their correlation with racial and economic status of surrounding communities. US General Accounting Office; Washington, DC, 1983.

USHR: Baby foods are tainted with dangerous levels of arsenic, lead, cadmium, and mercury, Staff report subcommittee on economic and consumer policy committee on oversight and reform U.S. House of Representatives, 4 Feb 2021, oversight.house.gov [2-3-21 ECP Baby Food Staff Report.pdf \(wsj.net\)](#) accessed 1 Aug 21.

USNCHS: U.S. National Center for Health Statistics. National Vital Statistics Report, vol. 51, no. 5, 14 Mar 2003.

Van Cauwenberghe L, Janssen C, Microplastics in bivalves cultured for human consumption, Environ. Pollut., 2014, 193, 65-70.

Van Cleave J, Gortmaker S, Perrin J, Dynamics of obesity and chronic health conditions among children and youth, JAMA, 2010, 303, 7, 623 – 30.

Van der Sluijs J Amaral-Rogers V, Belzunces L Van Lexmond M, Bonmatin J, Chagnon M, Downs C, Furlan L, Gibbons D, Giorio C, Girolami V, Conclusions of the Worldwide Integrated Assessment on the risks of neonicotinoids and fipronil to biodiversity and ecosystem functioning, 2015, 148-54

Vieira V, Hoffman K, Shin H, Weinberg J, Webster T, Fletcher T, Perfluorooctanoic acid exposure and cancer outcomes in a contaminated community: a geographic analysis, Env health persp, 2013, 121, 3, 318-23.

Vojdani A, A potential link between environmental triggers and autoimmunity, Autoimmune diseases. 2014, 1.

Volk H, Hertz-Picciotto I, Delwiche L, Lurmann F, & McConnell R, Residential proximity to freeways and autism in the CHARGE study, Env Health Persp, 2011, 119, 6, 873.

Volk H, Lurmann F, Penfold B, Hertz-Picciotto I, McConnell R, Traffic-related air pollution, particulate matter, and autism, JAMA Psychiatry, 2013, 70, 1, 71–7.

Vos J, Van Loveren H, Wester P, Vethaak D, Toxic effects of environmental chemicals on the immune system, Trends in pharmacological sciences, 1989, 10, 7, 289-92.

VRBPAC, US FDA Vaccine and Related Biological Products Advisory Committee, Gardasil™ HPV Quadrivalent Vaccine May 18, 2006 Meeting, <https://web.archive.org/web/20170223195942/https://www.fda.gov/ohrms/dockets/ac/06/briefing/2006-4222B3.pdf> accessed 3 Mar 2020.

Vrijheid M, Martinez D, Fornis J, Guxens M, Julvez J, Ferrer M, Sunyer J, Brief Report: Prenatal exposure to cell phone use and neurodevelopment at 14 months, Epidemiology, 2010, Mar, 259-62.

Wahlquist C, Coal-fired power stations caused surge in airborne mercury pollution, study finds, The Guardian, 4 Apr 2018 https://www.theguardian.com/australia-news/2018/apr/03/coal-fired-power-stations-caused-surge-in-airborne-mercury-pollution-study-finds?CMP=share_btn_fb accessed 24 Mar 2020.

Walters D, Grodzki K, Beyond limits? Dealing with chemical risks at work in Europe; Elsevier: Philadelphia, PA, USA, 2006.

Wang W, Wu F, Huang M, Kang Y, Cheung K, Wong M, Size fraction effect on phthalate esters accumulation, bioaccessibility and in vitro cytotoxicity of indoor/outdoor dust, and risk assessment of human exposure, J hazardous materials, 2013, 261, 753-62.

Wang M, et al., Association between long-term exposure to ambient air pollution and change in quantitatively assessed emphysema and lung function, *JAMA*, 2019, 322, 6, 546-56.

Wang X, Younan D, Petkus AJ, Beavers DP, Espeland MA, Millstein J, Chui HC, Gatz M, Chen JC. Heterogeneous Associations of Air Quality Improvement with Domain-Specific Cognitive Function in Older Women. In 2021 Alzheimer's Association International Conference 2021 Jul 26. ALZ.

Ward Z, Bleich S, Craddock A, Barrett J, Giles C, Flax C, Long M, Gortmaker S, Projected US state-level prevalence of adult obesity and severe obesity. *NEJM*, 2019, 381, 25, 2440-50.

Waterland R, Assessing the effects of high methionine intake on DNA methylation. *J Nutrition*, 2006, 136, 6, 1706S.

Waterland R, Jirtle R, Transposable elements: targets for early nutritional effects on epigenetic gene regulation, *Molecular and Cellular Biology*, 2003, 23, 15, 5293-300.

Watson C, Jeng Y, Guptarak J, Endocrine disruption via estrogen receptors that participate in nongenomic signaling pathways, *J. Steroid Biochem Mol Bio*, 2011, 127, 44-50.

Wdowiak A, Wdowiak L, Wiktor H, Evaluation of the effect of using mobile phones on male fertility, *Ann Agric Environ Med*, 2007, 14, 1, 169-72.

Webber M, Moir W, Crowson C, Cohen H, Zeig-Owens R, Hall C, Berman J, Qayyum B, Jaber N, Matteson E, Liu Y, Post-September 11, 2001, incidence of systemic autoimmune diseases in world trade center-exposed firefighters and emergency medical service workers, In *Mayo Clinic Proceedings*, 2016, 91, 1, 23-32, Elsevier.

Weis W, Brown J, Cusack S, Paulson J, Skehel J, Wiley D, Structure of the influenza virus haemagglutinin complexed with its receptor, sialic acid, *Nature*, 1988, 333, 6172, 426-31.

Wen Y, Hsiao F, Chan K, Lin Z, Shen L, Fang C, Acute respiratory infection and use of nonsteroidal anti-inflammatory drugs on risk of acute myocardial infarction: a nationwide case-crossover study, *J Infectious Diseases*, 2017, 215, 4, 503-9.

Weydert J, Vitamin D in children's health, *Children*, 2014, 1, 2, 208-26.

White H, Hazardous waste incineration and minority communities. In: *Race and the incidence of environmental hazards: a time for discourse*; Westview press, Boulder, CO, USA, 1992, 126-39.

Whitney D, Peterson M, US national and state-level prevalence of mental health disorders and disparities of mental health care use in children, *JAMA Pediatr*, 2019, 173, 4, 389-91.

WHO: World Health Organization, Expert Committee on the Prevention of Cancer (1964). Prevention of cancer: report of a WHO Expert Committee [meeting held in Geneva from 19 to 25 November 1963]. <https://apps.who.int/iris/handle/10665/40584> accessed 16 Apr 20.

WHO: World Health Organization, Occupational and Environmental Health Team, WHO Air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide: global update 2005: summary of risk assessment, WHO, 2006a. <https://apps.who.int/iris/handle/10665/69477>

WHO: World Health Organization, Chronic diseases and their common risk factors, 2006b https://www.who.int/chp/chronic_disease_report/media/Factsheet1.pdf accessed 2 Apr 20.

WHO: World Health Organization, Preventing disease through healthy environments, towards an estimate of the environmental burden of disease, WHO, Geneva, Switzerland, 2006c.

WHO: World Health Organization, Environmental Health Criteria 236, and methods for assessing autoimmunity associated with chemicals, WHO, Geneva, 2006d.

WHO: World Health Organization, Exposure to mercury: A major public health concern. Geneva, Switzerland, WHO, 2007a (<http://www.who.int/phe/news/Mercury-flyer.pdf> accessed 16 May 2020).

WHO: World Health Organization, Fourth WHO-Coordinated Survey of Human Milk for Persistent Organic Pollutants in Cooperation with UNEP; Guidelines for Developing a National Protocol; WHO: Geneva, Switzerland, 2007b.

WHO: World Health Organization, Children's Exposure to Mercury Compounds, WHO Press, 2010. https://apps.who.int/iris/bitstream/handle/10665/44445/9789241500456_eng.pdf;jsessionid=25B407270AFBF7A4471AB90AF493BBF0?sequence=1 accessed 10 Aug 21.

WHO, UNEP, State of the science of endocrine disrupting chemicals – 2012, An assessment of the state of the science of endocrine disruptors prepared by a group of experts for the United Nations Environment Programme (UNEP) and World Health Organization (WHO), 2013a <https://www.who.int/ceh/publications/endocrine/en/> accessed 16 Mar 2019.

WHO: World Health Organization, Media Centre, Effects of human exposure to hormone-disrupting chemicals examined in landmark UN report, 19 Feb 2013b https://www.who.int/mediacentre/news/releases/2013/hormone_disrupting_20130219/en/ accessed 12 Mar 2020.

WHO: World Health Organization, Burden of disease from the joint effects of Household and Ambient Air Pollution for 2012, WHO, published 2014. https://www.who.int/phe/health_topics/outdoorair/databases/FINAL_HAP_AAP_BoD_24March2014.pdf?ua=1 accessed 24 Mar 2020.

WHO: World Health Organization, News release, WHO releases country estimates on air pollution exposure and health impact, WHO, GENEVA, 27 Sep 2016 <https://www.who.int/news-room/detail/27-09-2016-who-releases-country-estimates-on-air-pollution-exposure-and-health-impact> accessed 23 Mar 2020.

WHO: World Health Organization, Newsroom/Fact sheets/Detail/Cancer in Children, WHO, 2018 <https://www.who.int/news-room/fact-sheets/detail/cancer-in-children> accessed 25 Jan 20.

WHO: World Health Organization, Programmes and projects, Nutrition, Nutrition health topics, Section 2 Background, Sect 2.1 The global burden of chronic disease, WHO https://www.who.int/nutrition/topics/2_background/en/ accessed 7 Apr 20

Wigle D, Lanphear B, Human Health Risks from Low-Level Environmental Exposures. PLoS Medicine, 2005, 2, 1232–34.

Winans B, Humble M, Lawrence B, Environmental toxicants and the developing immune system: a missing link in the global battle against infectious disease? Reproductive toxicology, 2011, 31, 3, 327-36.

Wolff M, Engel S, Berkowitz G, Ye X, Silva M, Zhu C, Wetmur J, Calafat A, Prenatal phenol and phthalate exposures and birth outcomes, Env health persp, 2008, 116, 8, 1092-7.

Woodruff T, Zeise L, Axelrad D, Guyton K, Janssen S, Miller M, Miller G, Schwartz J, Alexeeff G, Anderson H, Birnbaum L, Bois F, Cogliano V, Crofton K, Euling S, Foster P, Germolec D, Gray E, Hattis D, Kyle A, Luebke R, Luster M, Portier C, Rice D, Solomon G, Vandenberg J, Zoeller R, Meeting report: moving upstream-evaluating adverse upstream end points for improved risk assessment and decision-making, Env Health Persp, 2008, 116, 11, 1568-75.

- Wright S, Kelly F, Plastic and human health: a micro issue? *Env Sci Technol*, 2017a, 51, 12, 6634-47.
- Wright J, Spectrum, Behavior & society, the real reasons autism rates are up in the u.s., a hard look at whether the rise comes from more awareness, better diagnosis—or something else, *Scientific American*, 3 Mar 2017b <https://www.scientificamerican.com/article/the-real-reasons-autism-rates-are-up-in-the-u-s/> accessed 3 May 2020.
- WWF, Assessing plastic ingestion from nature to people, 2019 https://d2ouvy59p0dg6k.cloudfront.net/downloads/plastic_ingestion_web_spreads.pdf accessed 14 Jan 2020
- Xu X, Nie S, Ding H, Hou F, Environmental pollution and kidney diseases, *Nature Reviews Nephrology*, 2018, 14, 5, 313.
- Yan J, Agresti M, Bruce T, Yan Y, Granlund A, et al., Effects of cellular phone emissions on sperm motility in rats, *Fertil Steril*, 2007, 88, 4, 957–64.
- Yang D, Shi H, Li L, Li J, Jabeen K, Kollandhasamy P, Microplastic pollution in table salts from China, *Env Sci Technol*, 2015a, 49, 22, 13622-27.
- Yang J, Bakshi A, Zhu Z, Hemani G, Vinkhuyzen A, Lee S, Robinson M, Perry J, Nolte I, van Vliet-Ostaptchouk J, Snieder H, Genetic variance estimation with imputed variants finds negligible missing heritability for human height and body mass index, *Nature genetics*, 2015b, 47, 10, 1114.
- Yevdokimova N, Yefimov A, Effects of wheat germ agglutinin and concanavalin A on the accumulation of glycosaminoglycans in pericellular matrix of human dermal fibroblasts. A comparison with insulin, *Acta Biochimica Polonica*, 2001, 48, 2, 563-72.
- Yokel R, Aluminum in food—the nature and contribution of food additives, In *Food Additive*, Intech, 22 Feb 2012, 203-28.
- Younan D, Petkus A, Widaman K, Wang X, Casanova R, Espeland M, Gatz M, Henderson V, Manson J, Rapp S, Sachs B, Particulate matter and episodic memory decline mediated by early neuroanatomic biomarkers of Alzheimer’s disease, *Brain*, 2020, 143, 1, 289-302.
- Zenn J, *The self-health revolution*, Eds: Simon, Schuster, Health & Fitness, 27 Nov 2012, 178 pages.
- Zhang M, Huang J, Xie X, Holman C, Dietary intakes of mushrooms and green tea combine to reduce the risk of breast cancer in Chinese women, *Int J cancer*, 2009, 124, 6, 1404-8.
- Zhang L, Rana I, Shaffer RM, Taioli E, Sheppard L, Exposure to glyphosate-based herbicides and risk for non-Hodgkin lymphoma: a meta-analysis and supporting evidence, *Mutation research/reviews in Mutation research*, 2019, 781, 186-06.
- Zhao R, Chen S, Wang W, Huang J, Wang K, Liu L, Wei S, The impact of short-term exposure to air pollutants on the onset of out-of-hospital cardiac arrest: A systematic review and meta-analysis, *Int J Cardiol*, 2017, 226, 110-117.
- Zheng G, Schreder E, Dempsey J, Uding N, Chu V, Andres G, Sathyanarayana S, Salamova A, per- and polyfluoroalkyl substances (PFAS) in breast milk: concerning trends for current-use PFAS, *Env Sc & Tech*, 2021, 55, 11, 7510-20.
- Zhou H, Tang L, Xue K, Qian H, Sun X, Williams P, Wang J, Trans-/multi-generational effects of deoxynivalenol on *Caenorhabditis elegans*, *Chemosphere*, 2018, 201, 41-9.
- Zimmerman R, Social equity and environmental risk, *Risk Anal*, 1993, 13, 649–66.

Zuo G, Xu Z, Hao B, Shigella strains are not clones of Escherichia coli but sister species in the genus Escherichia, Genomics, proteomics & bioinformatics, 2013, 11, 1, 61–65.

Chapter 3

Multiple Chemical Sensitivity (MCS)

Contents

Chapter 3.....	177
Multiple Chemical Sensitivity (MCS).....	177
INTRODUCTION.....	179
DIAGNOSIS CRITERIA.....	185
EPIDEMIOLOGY.....	189
IMPLICATIONS FOR ORTHODOX MEDICAL PRACTITIONERS.....	197
Adaptation.....	199
Switching Phenomena.....	200
Spreading Phenomena.....	201
POSSIBLE MECHANISMS FOR MCS.....	202
SILO RESEARCH AND OVERLAPPING CONDITIONS.....	205
Central Sensitisation.....	206
Schizophrenia.....	206
Chronic Inflammatory Response Syndrome (CIRS).....	207
Electromagnetic Hypersensitivity Syndrome (EHS).....	209
Conclusion of Silo Research Section.....	220
DISEASE CAUSALITY PARADIGM.....	220
DISEASE AND ILLNESS.....	224
OBSERVATION, PROOF AND PATIENT RESPECT.....	226
EVIDENCE BASED MEDICINE.....	228
RECOGNISING THE KUHNIAN CRISIS.....	229
ATTEMPTS FOR MCS TO BE RECOGNISED AS A DISABILITY.....	232
BIAS IN THE RECOGNITION OF MCS AND THE INFLUENCING ROLE OF INDUSTRY.....	233
THE APPLICABILITY AND COMPARISON OF PSYCHOLOGY AND PSYCHIATRY TO MCS.....	241
LEGAL CASES.....	242
The Daubert Ruling.....	244
Consequences of the Daubert Ruling.....	244
The Australian Workers Compensation Case and Senate Hearing on Aircraft Cabin Fumes.....	247
ENVIRONMENTAL EFFECTS ON HEALTH SUPPRESSED BY THE NEOLIBERAL APPROACH.....	254

Neoliberalism	255
Normalisation.....	256
Insurance and Workers Compensation.....	258
Doctor Normalisation.....	261
Conclusion on Neoliberalism Effects	262
THE CONSEQUENCES IF MCS HAD RECOGNITION	264
Consequences of the Mechanism to Allergists.....	264
Consequences for Toxicological Assessments	264
Consequences for Epidemiological Studies	266
Consequences for Clinicopathological Studies	266
Consequences for Medical Specialisation.....	266
Consequences for Psychiatry	267
Consequences for the Workplace	267
Consequences for the Legal System	267
Consequences in Everyday Life.....	267
Consequence for National Health Expenditures.....	268
INDUSTRY ACTION EXAMPLE: HEALTH EFFECTS FROM EMFS	268
CONCLUSION.....	273
REFERENCES.....	278
APPENDIX 1: THE MCS MECHANISM HYPOTHESIS BY PALL AND ITS OVERLAPS	307
REFERENCES FOR APPENDIX 1	309
APPENDIX 2: SOME TECHNICAL DETAILS ON CIRS.....	312
REFERENCES FOR APPENDIX 2	316

INTRODUCTION

This chapter considers multiple chemical sensitivity (MCS), an illness unrecognised by the orthodox medical system⁵. Many MCS sufferers have established clear links between their illness and a range of environmental exposures, and realise that by avoiding such exposures they can achieve improved health. For sufferers to establish cause and effect, it is a matter of personal observation, consistency and experience. MCS is a key illness used in this thesis to illustrate environmental effects on human health. Since the orthodox medical and health systems do not recognise this condition, it then becomes important to understand why this is the case. It is also pertinent that this illness is epigenetic in its origination, since its onset is commonly related to either repeated low-level environmental exposures or a single, severe environmental exposure.

There are several possible ways to consider MCS: the standard view, that it is not a real disease and that suffering is psychosomatic; a neutral view, that examines both sides of the debate about the illness reality without passing judgement; and the challenger view that MCS is an environmentally induced condition. Although the first two approaches are preferred by many writing about this condition, the final approach will be used here due to the focus of this thesis on the environmental impacts on health. So, the chapter is written from the perspective that MCS is an environmentally-induced condition without assuming that this is the only way the topic can be approached.

The incidence of many diseases has significantly increased over the past five decades, including autism, Alzheimer's disease, chronic obstructive pulmonary disease, diabetes, sleep apnea, attention deficit hyperactivity disorder (ADHD), celiac disease, chronic fatigue syndrome (CFS), asthma, depression, multiple sclerosis, bipolar disease in youth, hypothyroidism, lupus, irritable bowel disease (IBD), fibromyalgia syndrome (FMS) and osteoarthritis. The cause of, or a pathogen for, each of these diseases has not been established. As the general human genome

⁵ Portions of this chapter have been drawn from my article "Multiple Chemical Sensitivity: The Paradigm Breaker for Doctors", *Townsend Letter*, Jan 2018

has not significantly altered in this period, environmental exposures are highly suspect (Bijlsma 2016, Rappaport 2010, Reichard 2015).

Centuries ago Hippocrates put forward that health and wellbeing are profoundly affected by an individual's diet, lifestyle and environment (Kleisiaris 2014). It was pointed out in chapter 2 that in 2011, the World Health Organization estimated that annually 4.9 million deaths and 86 million disability adjusted life years (DALY)⁶ could be due to environmental chemicals (Bijlsma 2016, UNEP 2012). Furthermore about one-quarter of the global disease burden, and greater than one-third of the disease burden among children under the age of five, have been attributed to modifiable environmental factors (Bijlsma 2016, WHO 2006).

Recent studies on epigenetics and new terms such as the "exposome" (or total exposures over a lifetime) are increasingly being associated with chronic disease (Paoloni-Giacobino 2011, Lopez 2014). These represent a threat to current paradigms for chronic disease etiology as it gradually becomes recognised that infectious diseases are primarily only encountered in developing countries (Laborde 2015).

While environmental exposures are likely involved in many of the chronic diseases mentioned before, there is a direct cause-and-effect relationship between a health condition termed multiple chemical sensitivity (MCS) and environmental chemical exposures.

MCS (or toxicant induced loss of tolerance (TILT) or idiopathic environmental intolerance (IEI)) was conceptualised by Dr Theron Randolph in 1954. The sufferer experiences sensitised reactions to low-level exposures of some natural and most synthetic chemicals, e.g., solvents, volatile organic compounds (VOCs), perfumes, deodorisers, plastics, synthetic fabric, petrol, diesel, smoke, pesticides, food additives, paint, new carpet, cleaning products, etc.: "chemicals" in general (Randolph 1954).

The symptoms of MCS vary from one individual to another, although they are usually consistent/repeatable short term with each individual responding to the same triggers. The symptoms overlap with (and some are co-morbidities of) closely related health conditions such as electromagnetic-hypersensitivity syndrome (EHS), CFS, FMS, chronic inflammatory response

⁶ One DALY represents the loss of the equivalent of one year of full health. DALYs for a disease or health condition are the sum of the years of life lost to due to premature mortality and the years lived with a disability due to prevalent cases of the disease or health condition in a population (WHO definition).

syndrome (CIRS), central sensitivity (CS), Gulf War syndrome (GWS), post-traumatic stress disorder (PTSD), myalgic encephalomyelitis (ME), the Vietnam veteran health problems, lupus and irritable bowel syndrome (IBS). One or multiple organs or body systems can be affected. Symptoms such as extreme fatigue/lethargy, wheezing, burning/stinging eyes, breathlessness, nausea, headache/migraine, vertigo/dizziness, poor memory, poor concentration, anxiety, depression, confused thought processes, sleeping problems, runny nose (rhinitis), sensitivity to light and noise, sore throat, cough, sinus problems, skin rashes, digestive problems, muscle pain, and joint pain can be experienced. An unfortunate feature of MCS is that the sensitisation and reactions can progressively get worse with repeated exposures. Conversely, symptoms can abate if exposures to such incitants are removed or avoided.

MCS sufferers react to very low concentration levels of the inciting chemicals. They may experience headaches and fatigue symptoms due to the presence of deodoriser or perfume at levels not able to be smelt (UoT 2019). It is pertinent that one does not need to be recognised as having MCS to react this way: “Exposure to perfumes and other scented products can trigger serious health reactions in individuals with asthma, allergies, migraines, or chemical sensitivities” (UoT 2019).

Depending upon how sensitive an individual is, triggering concentrations may be orders of magnitude lower than the normally accepted exposure levels derived from classical toxicology approaches. It is this aspect that creates the widespread disbelief about the very existence of this condition. The multiple symptoms also result in disbelief by orthodox medical practitioners: their training frequently leads them to regard individuals complaining of multiple organ problems and accompanying neurological symptoms as having a psychological problem. To date, no unique signs, pathology, laboratory test consistencies, or consistencies in illness progression have been clearly established for diagnostic purposes. The general trend by the orthodox medical system is to give sufferers a diagnosis of somatoform disorder or other psychological conditions. The mainstream medical system operates without recognition of the condition of central sensitivity (CS). As there is no training in environmental factors initiating health conditions, most doctors do not recognise their significance as seen in earlier chapters.

A typical orthodox statement about MCS is:

Idiopathic environmental intolerance (IEI), formerly called multiple chemical sensitivity, is a subjective illness marked by recurrent, nonspecific symptoms attributed to low levels of chemical, biologic, or physical agents. These symptoms occur in the absence of consistent objective diagnostic physical findings or laboratory tests that define an illness. Many experiments and observational studies consistently identify psychopathology in patients with IEI and implicate behavioural or psychiatric causes for this illness. This indicates that the underlying illness in many cases of IEI is actually a psychiatric disorder, such as a somatoform, depressive, or anxiety disorder (Black 2019).

The incidence of people with chemical sensitivities in the population is significant, ranging between 2% and 27% of the population. The number of surveys completed to establish this range of incidence is significant, with most results falling between 13% and 27% of the population (Andersson 2008, Ashford 1991, Azuma 2015, Bell 1993, Berg 2008, Caress 2003,2004, Donnay 1999a, Hausteiner 2005, Jeong 2014, Johansson 2005, Katerndahl 2012, Kreutzer 1999, McCampbell 2001, Meggs 1996, Palmquist 2014, Park 2006, Steinemann 2018a,b). A rate of national prevalence found recently in the US/Australia was 12.8/6.4% medically diagnosed with MCS, 25.9/18.9% self-reported chemical sensitivity, and 27.5/19.9% either or both (Steinemann 2018a,b).

Assuming that MCS is environmentally induced, this significant percentage of the population pays a high price for an industrialised and technological culture. As exposure to xenochemicals and electromagnetic fields (EMFs) is now normal in most public venues, this population is robbed of a sense of “place” and experiences impacts on their personal relationships. MCS sufferers are marginalised by orthodox medical practitioners and relegated to mental health classifications due to paradigms that do not consider the possibility that health conditions may be caused by today’s lifestyle (Gibson 2016).

When observing a sufferer of MCS and its many overlapping conditions, they give no outward signs of their illness. This also becomes a disadvantage for them as “The extent to which the diseased body is visible—its ability to be seen and attract attention medically, legally, socially, and politically—is central to the way in which it is understood, treated, funded, and experienced” (Phillips 2017p214).

MCS becomes an ideal example of a health condition which is caused and worsened by environmental exposures, particularly synthetic chemicals, but also EMF effects, mould exposures, etc. Recognition of this condition would then cause problems for the industries that produce the large array of synthetic chemicals and EMF devices used in the modern environment. These industries have much to gain by opposing and undermining such recognition. The paradigmatic nature of the orthodox medical system, in not considering the relationships between environmental exposure and health problems is further fostered by industry efforts.

This chapter considers key aspects of the illness of MCS. It will illustrate that for individual sufferers, there is consistency in simple cause and effect. But, as MCS does not affect a specific organ, and is not a definable disease that can be treated by drugs to improve symptoms, modern medicine is at a loss to deal with it and generally blame's the individual's own psychology for their problems.

First, the diagnostic criteria for the illness will be considered, highlighting the complex nature of environmental health effects. The difference between industry-backed studies that conclude there is an absence of clear diagnostic criteria, and the latest studies that find there are clear criteria, will be covered. It will, however, ultimately be concluded that a diagnosis is not essential to assist or treat such sufferers. The need for epidemiological to understand environmental exposure effects and produce meaningful results will also be considered. This will highlight how industry-backed studies can be used to manufacture misinformation and ignorance.

The lack of education and experience that orthodox doctors show in pursuing the area of environmental effects will be highlighted. The chapter will consider the characteristics established by functional medicine practitioners, including phenomena such as adaptation, switching and spreading. This will illustrate the paradigmatic differences between the functional medicine approaches and orthodox medicine which simply does not pursue any longitudinal studies or even consider people suffering from environmental effects.

Some possible disease mechanisms will be covered for the condition MCS, which will be shown to overlap with those for many other unexplained illnesses. This then leads to considering the overlaps of MCS and shows comorbidities with many other conditions, especially central

sensitivity, schizophrenia, CIRS, and EHS. These health conditions generally have been studied as quite self-contained illnesses, while their overlaps with other illnesses are usually not considered. This creates siloed research areas yet the overlaps between the conditions are significant.

Some of the orthodox medical system's paradigmatic approaches will be discussed to highlight its resistance to recognising MCS. This discussion will cover the disease causality paradigm; the difference between illnesses and disease; proof, observation, evidence based medicine; and patient respect. It will consider the recognition of a Kuhnian-like crisis and the inconvenience of recognising MCS as a disability, and thus why industry actively influences the medical and health systems.

This chapter will also examine the situation around explaining MCS as purely a psychological condition, due to the absence of pathological indicators. The section highlights the absence of objective diagnosis criteria in psychology. While depression is commonly diagnosed in MCS sufferers, there is much evidence that depression itself is induced by various environmental exposures. Other psychological conditions, such as ADHD, lack clear diagnostic criteria and can affect a similar percentage of the population as MCS, yet are more accepted by orthodoxy.

Due to MCS's lack of recognition, its sufferers frequently resort to the legal system for compensation since many sufferers can no longer work. Here again, sufferers experience significant push-back due to industry influence. There is evidence, as discussed, that industry have influenced jurisprudence around evidence in the US, which culminated in the Daubert ruling. The consequences of this ruling are also examined in relation to cases attributing health effects to environmental exposures. An Australian workers' compensation case and senate hearing on aircraft cabin fumes are then used to demonstrate the frustrating situation that sufferers, in this case aircraft crews, can find themselves in. An example is also sketched out of how an industry shill, Dr Loblay, hijacked the legal case and hearings by generating manufactured and selective ignorance. Dr Loblay at the time was the Director of the Allergy Unit at the Royal Prince Alfred Hospital and Senior Lecturer in Immunology, Department of Medicine, University of Sydney.

Consideration of how the governments' neoliberal approach and normalisation characteristics hinder the recognition of an MCS condition is also presented.

Finally, the chapter presents a stark consideration of the contrasting situations to various societal and health science approaches if MCS, and the effects of environmental exposures in general, were recognised.

The chapter will therefore have several major sections. The diagnosis criteria and resultant diagnostic difficulties will be discussed first, reflecting the orthodox starting point for treating any health condition. The epidemiological aspects and problems with this illness will be addressed, followed by the difficulties encountered by orthodox medical practitioners in recognising MCS. The absence of a clear mechanism for relating environmental exposures to the varied pathology is considered, referring to popular hypotheses, and the varied symptomatology and overlaps with other environmentally related illnesses are compared by considering the siloed research into four conditions: central sensitivity (CS), schizophrenia, chronic inflammatory response syndrome (CIRS) and electromagnetic hypersensitivity (EHS). The orthodox positivist approach is shown to become a hindrance to change because of its difficulties in recognising complex and overlapping emergent diseases. Due to the absence of organic pathology expected by orthodox medicine, the classification of MCS as an illness rather than a disease is then covered. This leads to considering observation, proof and patient acceptance and the shortcomings of evidence-based medical approaches. The chapter then moves into aspects of bias in recognising MCS, under the influence of industry which has an interest in defending its products against claims that they cause health effects. The chapter then discusses the general suppression of environmental effects in the context of neoliberalism, using examples of MCS through aspects such as patient and doctor normalisation and resistance by the insurance industry sector. The chapter finishes with a short hypothetical section covering consequences if MCS were recognised, highlighting significant potential societal changes.

DIAGNOSIS CRITERIA

In this section it is shown that despite the general medical and health industry view of no clearly definable diagnostic criteria for MCS, there are now emerging objective criteria for MCS diagnosis, acknowledging its overlaps with other illnesses.

The orthodox view on MCS is reflected by the following quotes, firstly from the South Australian Health Department:

There are no universally accepted diagnostic criteria for MCS/IEI. There are no laboratory tests and no clinical guideline documents. MCS/IEI is not recognised as a medical condition in Australia and most countries.

Individuals with MCS/IEI symptoms who seek help from their GPs are frequently referred to various specialists. If following clinical investigations there is still no explanation for the symptoms, these patients are usually provided with explanation, reassurance, and guidance on trigger avoidance measures (SAH 2019).

Also, from Johns Hopkins Medicine:

At this time, it is a controversial issue as to whether it is a clinical diagnosis or not. Many in the medical community lean towards these symptoms being physical manifestations of psychiatric illness rather than a primary medical illness. While others in the medical community along with organizations agree that multiple chemical sensitivity is a negative physical reaction to certain chemicals. There is debate as to whether multiple chemical sensitivity should be classified and diagnosed as an illness (JHM 2019).

In respect to immunological aspects, the basic orthodox approach to an MCS sufferer being sensitive to low level exposures would suggest a type of allergy however there are no normal pathological immunological abnormalities such as allergic disorders, autoimmune diseases, or immunodeficiencies. This then sees orthodoxy move into psychological diagnoses since the subjective criteria may see the MCS sufferer readily fit to any of the conditions, or combinations of depression, anxiety, somatic symptoms disorder, panic disorder, absorption personality trait, technophobia, or chemophobia.

The difficulty in clearly defining a consistent set of symptoms, let-alone pathology on MCS led to international consensus meetings of 89 and 34 experts respectively in 1989 and 1999 (MCS 1999, Bartha 1999p148) concluding on diagnosis criteria as:

- “The symptoms are reproducible with [repeated chemical] exposure;
- The condition is chronic;

- Low levels of exposure [lower than previously or commonly tolerated] result in manifestations of the syndrome;
- The symptoms improve or resolve when the inciting agents are removed;
- Responses occur to multiple chemically unrelated substances;
- Symptoms involve multiple organ systems”

There is considerable overlap with many other conditions as mentioned before. In an effort to have clearly defined and separate diagnosable conditions for CFS and FM there has been much conjecture and debate in the medical literature (Buchwald 1994, Donnay 1999a, Janson 2000, Aaron 2001,2003, Schafer 2002, Ciccone 2003). In attempts to standardise approaches suggestions have been made to exclude MCS diagnosis if CFS or FM precede the overlapping symptoms, or classify CFS or FM developed in the course of MCS, as co-morbidity (Lacour 2005).

A common frustration of doctors is that the diagnosis of these conditions relies on patients’ self-reports. In a situation relying on patient self-reports it becomes an insurance industry nightmare for MCS to be formally recognised without diagnostic criteria independent from personal opinions.

MCS, and perhaps to a lesser extent fibromyalgia and chronic fatigue syndrome, have been referred to as “Illnesses you have to fight to get” (Dumit 2006p577). This is because unless the sufferer has a medical validation, legitimacy is not conferred. Societal participation and acceptance problems occur due to the lack of recognition of emerging disabilities in general (Fox 2004).

An MCS sufferer may experience unemployment through lack of a suitable work environment or even harassment in an existing workplace (Gibson 2007). Other experiences are the lack of medical care, social isolation (Gibson 2010), and even homelessness (Gibson 1996). Personal distress becomes a common outcome.

Although there is a lack of pathologically based diagnostic criteria, this is not new and has happened with many emergent diseases in the past where there were no clearly visible illness symptoms and no causal explanation, and so were dismissed as either not real or as psychological. Asthma was once thought to be psychological.

In the many studies in the psychogenic origins of MCS there is frequently a disregard for environmental exposure origins. However, there is also no pathology for psychogenic diagnoses. As a result, “studies investigating psychogenic hypotheses of MCS are methodologically problematic and their conclusions questionable” (Davidoff 1994p316).

The diagnostic problems have been the reason for MCS being termed a ‘syndrome’.

A syndrome is defined as a recognizable complex of symptoms and physical or biochemical findings for which a direct cause is not understood. With a syndrome, the components coexist more frequently than would be expected by chance alone. When causal mechanisms are identified, the syndrome becomes a disease (Last 1993).

The National Library of Medicine’s online medical dictionary defines a syndrome as “a group of signs and symptoms that occur together and characterize a particular abnormality.”

But relatively recently, a significant clinical and biologic study was conducted over some years on 727 self-reported MCS and/or EHS cases. It was found that they “can be objectively characterized and routinely diagnosed by commercially available simple tests” (Belpomme 2015p251). A summary of their findings on this cohort is as follows:

Our data strongly suggest that EHS and MCS can be objectively characterized and routinely diagnosed by commercially available simple tests. Both disorders appear to involve inflammation-related hyper-histaminemia, oxidative stress, autoimmune response, capsulothalamic hypoperfusion and BBB opening, and a deficit in melatonin metabolic availability; suggesting a risk of chronic neurodegenerative disease. Finally the common co-occurrence of EHS and MCS strongly suggests a common pathological mechanism.

In a subsequent study, mainly on EHS, up to 80% of the self-reported cases had one to three peripheral blood detectable oxidative stress biomarkers. As this is typical of many chronic pathological disorders, such as cardiovascular diseases, diabetes, cancer, neurodegenerative diseases, “EHS and MCS should clearly be added to the next version of the WHO international classification of diseases (ICD) on the basis on their clinical and pathological description” (Belpomme 2015p265). Irigaray et al. (2018b) recently confirmed that ultrasonic cerebral

tomosphygmography is the best imaging technique in the diagnosis of EHS and to monitor patients being treated for EHS or MCS.

There are generally a number of stages of MCS recognised. There have been many attempts over the decades to formulate the framework for MCS. One relatively recent example was presented in the Italian parliament as a draft law (Rossi 2018p138) which did not gain sufficient support to be enacted, although later care protocols were partly instigated. This framework recognised four stages, beginning with a tolerance condition reflecting adaptation, a sensitization from chronic exposure to low doses and/or after an acute exposure, chronic inflammation in different tissues, organs and systems, and finally a deterioration where the chronic inflammation produces damage to tissues and organs.

As seen from this section, while the orthodox medical system and industry sponsored studies maintain there are no consistent diagnostic criteria, some researchers believe that significant progress has been made in consistent diagnostic criteria, especially as seen from the Belpomme et al paper discussed above.

EPIDEMIOLOGY

This section highlights the complex nature of MCS and its overlaps with many other diseases/illnesses. This is done by considering a cross-section of studies. Although many recent studies of MCS are along the psychological side, there have been a diversity of studies and findings over the last few decades. The spectrum of studies done on the condition also illustrate how inadequate the simple regulatory studies on one chemical at a time in a sterile lab, on healthy test animals, can be far-removed from realities of real life xenochemical exposure effects.

Due to the perceived lack of diagnostic criteria, testable pathology, and industry sponsorship, most recent studies on MCS are swayed toward psychogenic criteria. Some examples of orthodox study conclusions are:

This study demonstrates that polyallergy/multiple chemical sensitivity may serve as a crude yet meaningful indicator of comorbid psychopathology (Jimenez 2019p298).

Ensuring appropriate physical activity and regular lifestyle rather than improving physical environment lead to improvement of Chemical Intolerance (CI). Stable psychosomatic state is essential for preventing CI (Azuma 2019p1).

The results disagree with one of the main etiological hypotheses of MCS, which is based on the existence of hypersensitive people, who present a response after prolonged expositions to very low concentrations during a long period of time (Pérez-Crespo 2018p473).

The present study findings supported an association between MCS and mental illness. The causal mechanism supporting this association remains unclear (Johnson 2017p40).

As clear links with organ pathology or dysfunction have not been established so far, we review critical evidence about alternative causal mechanisms as a platform for a novel unifying model of these conditions. There is consistent evidence that expectancy and placebo mechanisms are critically involved. Using recent predictive coding models of brain functioning, we describe a comprehensive new model to explain how symptoms come about and become linked to specific environmental cues. This new model integrates phenomenally different pathologies, suggests testable new hypotheses, and specifies implications for treatment (Van den Bergh 2017p551).

This article will evoke several examples (Multiple Chemical Sensibility, Mercury Exposure, Electromagnetic Hypersensitivity, and Sick Building Syndrome) and will examine to what extent environmental concerns might contribute to nosographical innovations. This article will also consider their similarities in terms of psychopathology in addition to their underlying psychodynamic mechanisms, such as the fear of serious illness (nosophobia) or the fear of being poisoned (toxicophobia) (Litinetskaia 2017p193).

Our results provide evidence that IEI patients can be distinguished on the phenomenological level from patients with major depression or schizophrenia, and that

distinct domains of psychological and somatic symptoms are particularly problematic in specific diagnostic groups (Weiss 2017p187).

As seen from the above, the tendency for orthodoxy is to attribute MCS solely or mostly to psychological processes, but it is worthwhile considering the similarities to allergies. In considering studies on allergic reactions the distinction between a biological and neurological reaction becomes a little blurred. Brain-triggered allergy attacks were documented as far back as 1886, when a person with asthma viewed an artificial rose and suffered an allergic attack (Mackenzie 1886). Later, it was shown that immune system responses can also be conditioned (Pavlov 1928). This means, that if a person associates a dust mite allergy with a particular odour, then the same smell will trigger an immune reaction even though no dust mite is present. In the 1970s, the interaction between behaviour, the brain, and the immune system became an established field of research called psychoneuroimmunology. Since then, researchers have found that placebo responses in patients with allergies are among the strongest observed in clinical studies, and strong psychological factors are apparent in people with a vast array of allergic disorders. These studies do not establish allergies as a purely psychological state since biological processes are involved even in the cases when no physical trigger is present. It does suggest the immune system responds to certain cues and environments. As with allergies, MCS sufferer reactions can be very debilitating. As with allergies, this phenomenon should be viewed as the body's protective reaction to a perceived threat based on prior experience, in initiating immune system reactions. Many studies on MCS totally miss this phenomenon in their conduct and use placebo reactions to assert a psychological response (Bornschein 2008a,b).

There have been thousands of studies published on purely psychogenic explanations for MCS. In one interesting review, papers published in two databases (PsychLit: American Psychological Association and Medline: National Library of Medicine) meeting the following criteria were selected for analysis:

- original data were gathered on a human sample by the authors,
- the psychogenic origins of MCS syndrome were addressed implicitly or explicitly,
- the findings were published during or after 1980

Only ten articles met the criteria. Of these, five had sample selection problems, seven had measurement problems, and three study design problems were in all except one article. The review concluded: "Current studies investigating psychogenic hypotheses of MCS syndrome are methodologically problematic and their conclusions questionable. Studies of psychiatric profiles observed in MCS syndrome need to be designed to differentiate between competing psychogenic and biogenic hypotheses" (Davidoff 1994). Psychological explanations remain opinion-based due to there being no measurable signs.

Despite the studies referenced in the above diagnostic section showing that there are tests that can be done with good correlations, the general health system remains in denial of MCS as a legitimate health condition. Industry funded studies help support this view. While there have been many studies published on MCS, most have shortcomings, mainly due to the paradigmatic viewpoints of the orthodox medical system. The typical study problems and shortcomings are shown below, mostly drawn from Rossi, who assessed many studies (Rossi 2018):

- There is the problem of what individuals to include. MCS is not a neat, delineated disease and overlaps with many other syndromes such as CFS, FM, GWS, EHS, Lupus, IBS, PTSD. Many of the studies excluded participants if they indicated any such condition. Yet such individuals may have 'general MCS' symptoms more than the accepted 'other' condition/s: no-one can distinguish.
- Most researchers with an orthodox infectious disease epidemiological approach have found it implausible that a single cause can initiate different diseases and preclude applicable cohorts (Michaels 2008).
- Some studies exclude current/ex-military personnel. Yet the large number of ex-military personnel who show MCS traits is quite significant, e.g., Vietnam vets and agent orange, GWS vets, anthrax vaccine vets, PTSD, etc.
- Individuals sensitised from dental amalgams are sometimes excluded: yet an MCS sufferer can typically undergo a sensitising event from an amalgam.
- Excluding women due to 'cyclical hormonal changes at childbearing age'. Yet it is well known that the clear majority of MCS sufferers are women (2:1 women to men incidence). There are some studies however, that are quite the opposite in excluding men.

- Individuals with diseases having symptoms overlapping with those of MCS were also excluded from many studies. These diseases include cancer, diabetes, neurological and psychiatric diseases, renal and hepatic diseases, hypothyroidism, olfactory dysfunction, pulmonary and cardiovascular diseases, endocrine diseases, hyperlipidaemia, anosmia and allergic rhinitis hypertension.
- General neurological and immunological problems that can be typical of MCS and diseases of the upper respiratory tract have been also excluded yet there are strong links between MCS and genetic predisposition.
- While many usual exclusion criteria were adopted by some studies, such as smoking, alcohol, drug abuse, some studies also excluded women who were pregnant or breast feeding.

When the Italian Dept of Environment and Health reviewed the epidemiology state of the art on MCS, it had discarded consideration of studies with the above overlaps so that only people with 'simply MCS' were considered. In doing so they found 27 studies based on chemical provocation on the subjects (Rossi 2018). In these studies many authors hypothesised that MCS was psychosomatic due to stress being found as a significant risk factor. Other authors however concluded a neurogenic inflammatory origin and limbic system stimuli hyper-reactivity. This was associated with prefrontal and frontal cortex hypo-activity in MCS sufferers compared to controls.

In imaging studies there has been a large variation in results, possibly due to the many variations of selection criteria for the study groups, exposure substances used, symptom severity, and variation in the imaging techniques. Some studies (Alessandrini 2016, Chiaravalloti 2015, Orriols 2009) illustrated brain dysfunction after stimulus exposures. Others (Azuma 2013, Hillert 2007) however conclude that MCS subjects show an emotional, "top down", response which produce the abnormal patterns. These contrast to Belpomme's research on diagnostic criteria covered previously.

A toxicological basis of neuronal stimulation has been the basis of various papers (Kimata 2004, Saito 2005, Östberg 2003) however there are many more that attribute a psychological response, or at least an anxiety risk factor for MCS development (Haumann 2003, Zucco 2008, Papo 2006, Fiedler 2000,2008, Lee 2003, Andersson 2009, Bornschein 2008a,b)

There have been many studies where varying results see no definitive conclusions reached (Andersson 2016, Dantoft 2015, Ojima 2002, Caccappolo 2000, Alobid 2014, Joffres 2005, Georgellis 2003, Van Thriel 2008).

Observational and longitudinal epidemiological studies show more women than men had MCS (usually 2 to 1), mentioned before, but their socio-economic and cultural level is medium to high (Caress 2004, Hojo 2008). This is at odds with most other studies showing lower incidence of health problems in such population classes.

There are hundreds of other studies, looking into the various possible differences between MCS sufferers and controls. A summarised cross-section, rather than an exhaustive list is below:

- Psychological considerations
 - Association with levels of anxiety, depression, and psychotic disorders (Andersson 2008, Skovbjerg 2015, Eek 2010, Katerndahl 2012, Cui 2015, Österberg 2007, Eis 2008, Bailer 2008a, Black 2001, Hausteiner 2006, Skovbjerg 2012, Bornschein 2007, Park 2007). Yet some studies found no anxiety and depression relationships (Gundersen 2015)
 - Anxiety, associated with CS as well as allergy and asthma (Bloch 2007).
 - panic has a non-unique relationship with MCS, suggesting “that chemical sensitivity is a member of a larger family of diseases that have a common relationship with anxiety and panic” (Bloch 2007p142)
 - mental health indicators improving after treatment for chemical sensitivities: pointing to psychological problems being secondary to the illness (Bertschler 195)
 - Psychological questionnaires doubted due to possible overlaps with states of chronic disease contributing to anxiety and frustration (Davidoff 2000)
- Neurological links
 - Neurotoxic exposure causing central nervous system effects producing symptoms similar to panic attacks (Hojo 2008)
 - neurological changes during and after chemical exposure of MCS sufferers compared to controls (Orriols 2009)

- from clear evidence of neurological abnormalities, that the “psychiatric presentation by many of these patients may well have a neurological basis.” (Heuser 1992p117)
- blood–brain barrier opening and temporal lobe decreased blood flow (Belpomme 2015) and other correlations referred to in the above diagnosis section.
- The olfactory kindling hypothesis (Bell 1992) with evidence from animal studies (Rogers 1999, Rossi 1996)
- Inflammatory links
 - upper airway inflammation, showing mixed results (Dantoft 2015, Meggs 1995a).
 - elevated histamine levels at time of exposures, as well as after (Belpomme 2015, Elberling 2015).
 - Studies on whether an inflammatory condition is induced without concurrent infections. These are diverse and complex in their inter-relationships studying interleukins, alpha factor of tumorous necrosis (Dantoft 2014), cytokine levels, and polymorphisms in the detoxification of xenobiotics (Cui 2013, Caccamo 2013, DeLuca 2010, Fujimori 2012)
- Immunological links
 - immunological factors (Meggs 1992), including proinflammatory indicators (Dantoft 2014)
 - Studies showing that MCS people are poor acetylators and/or have a homozygous GSTM1 and / or GSTT1 deletion (Schnakenberg 2007)
 - Glutathione and cytokine pattern alterations in MCS (DeLuca 2010)
 - Frequencies of Cyp isoforms altered in MCS (DeLuca 2010, Caccamo 2013)
 - A higher frequency of various haplotypes of the Cyp 450 family in MCS people (Caccamo 2013)
 - Low levels of oxidised/reduced glutathione and Coenzyme Q10 and higher lymphocytes damage with MCS (Gugliandolo 2016). This infers a decrease in detoxifying enzymes leading to increased oxidative stress (increase in free radicals and peroxy nitrite).
 - vitamin and mineral deficiencies (Galland 1987)

- Genetic links
 - The ill-defined nature of genetic susceptibility and epigenetic origins (Rossi 2018)
 - enzymatic and genetic alleles deficiencies (McKeown-Eyssen 2004)
- Other disease overlaps
 - The prevalence of MCS in other diseases, for example, asthma, allergies, atopic dermatitis, neurological, gynaecological, cardiopulmonary diseases, etc. (Gundersen 2015, Trabacchi 2009, Jeong 2014, Dantoft 2014, Baldwin 1998, Caress 2009).
 - The suggestion of MCS being the initial stages of other diseases such as autoimmunity, heart disease, respiratory, neuropsychiatric, etc. (De Luca 2011, Lacour 2005)
- Workplace chemical exposure studies showing occurrence of MCS/MCS-like symptoms (Davidoff 1998, Bornschein 2008b)
- Positive correlation between measured exposures and reports of symptoms (Saito 2005, Mizukoshi 2015)

With the difficulties and expense in conducting controlled studies of MCS, case studies in the workplace would seem to present an opportunity to study actual cases (yet rarely happens) rather than the many ad hoc experiments that have been done as seen from the above examples. This seems to be suggested in the literature such as through the Martini 2013 paper which calls for an occupational health surveillance protocol to be developed. Yet there is little evidence this has been pursued.

Silo research is common in respect to chemical sensitivities. One example is the studies done on fragrances. Fragranced consumer products, such as air fresheners, deodorisers, perfumes, laundry venting and cleaning supplies, have been linked with health and societal impacts. Averaged over the United States, Australia, United Kingdom, and Sweden, 32.2% of the population report fragrance sensitivity (Steinemann 2019a). Health problems from mild headaches to disabling sicknesses have been reported by 17.4% of the population. With 26% of the population in these same countries having asthma, 57.8% of those report adverse health effects, including respiratory problems (37.7%), asthma attacks (25.0%), and migraine

headaches (22.6%), from fragranced product exposure. Some major areas of silo research with significant MCS overlaps is discussed in a following section.

This section has highlighted the complex nature of MCS and its overlaps with many other diseases/illnesses and the resultant difficulties of effectively studying it. This was illustrated by considering a cross-section of studies. Although many recent studies of MCS are along the psychological side, there have been a diversity of studies and findings over the last few decades. The spectrum of studies done on the condition also show how the simple regulatory studies on one chemical at a time in a sterile lab, on healthy test animals, with one or two end points tested for, can be far removed from realities of xenochemical exposure effects.

IMPLICATIONS FOR ORTHODOX MEDICAL PRACTITIONERS

In the vein of the approach discussed in the chapter introduction, in covering the condition of MCS as environmentally induced, the orthodox medical system is ignorant, ill-equipped, untrained, and not organised to handle chemical sensitivity health conditions. With negative industry backed studies and criticism of independent positive studies on this subject, the orthodox doctors are caught in a rigid system which is not capable of dealing with environmental conditions such as MCS.

Some phenomena that have been noticed over the decades by the functional medicine practitioners in trying to assist affected patients, are touched on in this section. As initiated in last chapter, these illustrate that orthodox medicine's diagnostic approaches considering symptoms only, can be very inadequate for patients suffering from environmental exposures.

In general, MCS has been dismissed by orthodox doctors as a psychological problem (Ashford 1991, McCampbell 2001, Pall 2000a,b, Phillips 2015). If MCS were accepted as having a physiological basis, this would constitute a radical change in direction for the orthodox medical system. At the most basic level, a physiological mechanism would recognise the modern environment as causing health problems. In the past medical authorities have actively criticised any practitioner considering such a mechanism. Environmental causation of MCS also challenges the one-size-fits-all mentality of a medical system influenced by the pharmaceutical industry where diagnosis, drug administration and vaccination are done on the basis that

everyone is similar. Drug research and treatment of patients are based-on an 'average' human, never making any allowance for a sensitive subset of the population. When a doctor starts to question such an approach they are criticised by their peers. It is pertinent to note that it has been observed generally that "If people who question the standard view...or the view backed by powerful groups regularly come under attack, then it is plausible that suppression is involved" (Martin 2012p221).

Consider a common symptom of MCS, with its significant overlaps with CFS: fatigue. It is interesting to note orthodox medicine's approach: "Treatment of all types of fatigue should include a structured plan for regular physical activity that consists of stretching and aerobic exercise, such as walking. Caffeine and modafinil may be useful for episodic situations requiring alertness. Short naps are proven performance enhancers. Selective serotonin reuptake inhibitors, such as fluoxetine, paroxetine, or sertraline, may improve energy in patients with depression. Patients with chronic fatigue may respond to cognitive behaviour therapy" (Rosenthal 2008p1173). Orthodox medicine lacks clear and proven approaches for the treatment of the common condition of fatigue.

The 'blame the patient' approach by many doctors contributes to the societal slowness to recognise chronic chemical poisoning. The encouragement for patients to blame themselves through their imagination, mental state, or their genes is at odds with a logical approach to at least investigate the person's living environment and the toxins involved. The same dismissive approach had been experienced by tens of thousands of ex-servicemen from Vietnam, The Gulf and Afghanistan. Their depressions, cancers, and deformed or affected children were dismissed from the association of chemical exposures during their service. The relative few doctors that assisted these veterans gradually saw some legal recognition after decades of work, even though the officials have never acknowledged the link, suggesting that some figures in authority have accepted that it wasn't just in their imagination. This however did not carry through to the civilian cases.

Rather than doctors establishing a diagnosis based on a short list of symptoms in a brief visit, environmental causation of MCS suggests they should carefully listen to the patient's history and observations on cause and effect. This would be far different than a typical 15 to 20 minute visit ending in the doctor handing out a prescription for drugs based on symptoms.

Industry has long been opposed to the recognition of MCS (McC Campbell 2001, CMA 1990). A recognition of this illness on a pathological basis would acknowledge that many industrial products can affect human health in low doses. Industry backed studies and scientists dismissing the growing body of research on MCS would be expected to be welcomed and even supported by many orthodox practitioners so that they do not have to change their beliefs or practices.

Although orthodox medical doctors do not recognise MCS the functional medicine/environmental medicine practitioners have been actively trying to work with sufferers for decades and it is pertinent to consider some of their observations on MCS developed over this period. This contrasts to the orthodox medical system that has not worked with the sufferers due to the paradigmatic walls present and requiring pathological diagnosis criteria.

The approaches used by functional medicine doctors are consequently quite foreign to orthodox doctors. It is generally known that many doctors will not even diagnose conditions for which they do not have any training or background, and sometimes simply because they cannot bring themselves to recognise a condition questioned by health departments or in media reports. Many Chronic Fatigue Syndrome (CFS), Fibromyalgia Syndrome (FMS) and Lyme Disease sufferers can recount situations going from one doctor to another until they find one who will recognise and diagnose their condition. This is even more so for MCS (personal testimonies, Donohoe 2008p50).

In considering the experience gained by the functional medicine practitioners over decades of studying and assisting sufferers affected by the environment, the following are some of the phenomena established for the condition. These are quite foreign to orthodox medical training.

Adaptation

In endeavouring to address an MCS potential condition in patients, the condition recognised as 'adaptation' needs to be adequately dealt with. Adaptation, or acclimatisation, has been recognised in workers who become accustomed to exposures in their workplace (Ashford 1991). Such workers can undergo withdrawal symptoms over the weekend and perhaps start peaking in symptom intensity on the Monday morning, which gradually disappears as exposures begin again. Workers in the dynamite industry of the past, for example, would adapt

to nitro-glycerine exposure and place some under their hatbands to avoid withdrawal, or breathe in their clothes over the weekend. The adaptation phenomenon appears to be the body's first response to chemical exposures until it seems to reach a limit, varying in individuals, from which it can no longer tolerate the exposures and descends into an MCS condition. This seems to be linked-in with the increasingly recognised cell danger response which is a metabolic response that protects cells and hosts from harm triggered from threats that exceed the cellular capacity for homeostasis (Naviaux 2020). To determine whether a patient is exhibiting a degree of adaptation, minor symptoms such as irritability, moodiness, drowsiness, fatigue, migraines, runny nose, etc. would need to be noted as to whether they relate to an underlying condition. The doctor who makes a quick diagnosis from such symptoms, such as cold/flu, would be missing something more systemic and serious.

The best way to unmask adaptation is by the use of an Environmental Isolation Unit (Donohoe 2008p71-3). The unit protects the patient from all environmental exposures, including most foods, for about 5 days. Then different food groups and exposures are measurably re-introduced. Since all adaptations are unmasked, such a unit can define a patient's sensitivities. Australian health authorities effectively closed down most such units 20 to 30 years ago after making the situation intolerable for the doctors (mostly functional medicine doctors) to keep operating such clinics (Donohoe 2008p29).

Switching Phenomena

A switching phenomenon was observed as far back as the 1700s by Savage (Johnson 1992). Switching is terminology for the changing of one end-organ response for another. Savage observed that when mental patients were at their worst, their asthma or sinusitis abated, however when they were better mentally they had an incidence of asthma or sinusitis problems.

After thousands of environmental unit controlled challenges switching had been frequently observed (Johnson 1992), for example, transient brain dysfunction, followed by arthralgia, followed by diarrhoea, followed by arrhythmia. The switching phenomena can therefore make treatment seem effective, but if the pollutant cause remains then another organ may be switched to. An example would be a cortisone treatment of sinusitis, and later arthralgia, then arthritis may develop, or one's colitis clears to then develop cystitis.

Doctors would need to be aware of seemingly unrelated events. Examples where this is commonly overlooked are statements to the effect that a child will 'grow' out of a health problem, for example, switch phenomenon may be recurrent ear infections, replaced by recurrent bed wetting, then replaced by asthma. A further example would be where an adult has his house treated with pesticide then finds they develop persistent headaches afterward, then later finds they need to visit a rheumatologist due to symptoms of arthritis.

Spreading Phenomena

The common explanation for the increasing sensitivities is referred to as the spreading phenomena. This relates to a secondary response of the body to pollutants. Spreading has been defined as the phenomena involving new excitants when the body has developed increased sensitivity to increasing numbers of biological inhalants, toxic chemicals and foods at decreasingly smaller doses. At such times the body's overload threshold changes to such an extent that minute exposures may trigger a response. So a person may have this condition initially triggered by a pesticide and then find that a myriad of different exposures may trigger the condition.

The spreading may be due to failure of detoxification mechanisms: oxidation, reduction, degradation, and conjugation. This could possibly occur due to toxic overloading or depletion of enzymes or coenzyme nutrients such as zinc, magnesium, B vitamins, amino or fatty acids. The result can be an increasing inability to detoxify. Another explanation is the damage to the bodily barriers: blood-brain, or peripheral membranes of the skin, lung, nasal mucosa, gastrointestinal, genitourinary systems for substances to penetrate compared to before thereby increasing the risk of harm. Immune systems may become so damaged that they start triggering to other toxic substances, then non-toxic (e.g., food) substances. There is evidence that antigen recognition sites are affected by pollution overload (Desforges 2016, Franceschi 1999, Ostan 2008, Candore 2006, Köllner 2002, Yuan 2018).

The body's hormonal system can also be adversely affected through progressive damage or even simply by xenochemicals mimicking hormones.

It is typical of MCS sufferers to have one or more isolated organs involved in sensitivities, perhaps from days to years, only for other organs to gradually become involved as resistance mechanisms gradually decline (Rea 1992).

Doctors are Limited by a Rigid System

While not having the training on environmental effects on health, orthodox doctors are caught in a system that imposes rigid diagnosis criteria for established diseases. The system requires the doctor to diagnose a patient's sickness into a standardised category. If there are no appropriate categories then it becomes appealing to utilise the psychological one which is broad and subjective. Furthermore, if a doctor were to actively investigate a patient by running a variety of tests needed to define environmental effects then they would risk the medical board (AHPRA in Australia), accusing them of overservicing and if investigated, would be reprimanded for not utilising standard diagnostic protocols.

As has been gathered from this section, the typical 15-20 minute patient consultation is not sufficient to adequately assist those that have environmentally induced health problems. This promotes treating symptoms rather than getting to the causes of their illness.

Accepting MCS as an environmentally induced illness, the above section has highlighted how the orthodox medical system is ignorant, ill-equipped, untrained and not organised to handle chemical sensitivity health conditions. With industry backed negative studies and criticism of independent positive studies on this subject, the orthodox medical system has ridden on the manufactured and selective ignorance created. Some phenomena that have been noticed over the decades by the functional medicine doctors in trying to assist affected patients, has been referenced in this section to illustrate that orthodox medicine's diagnostic approaches considering symptoms only, can be inadequate for patients suffering from environmental exposures.

POSSIBLE MECHANISMS FOR MCS

This section refers to some possible MCS mechanisms. It is not intended to be detailed, but rather to illustrate the complexity of possible mechanisms and the subsequent difficulties in establishing a mechanism. This is important to illustrate since orthodox medicine argues that the absence of a clear mechanism is the main reason for not recognising the condition. Again, the overlaps with many other diseases will be seen. The mechanisms are far from being straight-forward, so it is not a case of finding a simple singular pathway for one particular chemical exposure direct to an affected organ. For MCS, many direct and indirect pathways can be involved. As illustrated in the previous section, these pathways can alter/spread as chronic exposures extend. Since all humans are being exposed to a variety of xenochemicals and electromagnetic fields (EMFs), it is not a simple task to study the effect of one exposure on its own when there is synergism of the exposure effects, and all the various pathways can multiply/enhance. This section covers some of the popular models for this complex problem.

The ideal situation for orthodox medicine is to have a simple, definable, step by step biological mechanism. It is not expected that a mechanism be explained from an external environmental exposure cause. It is normal to start at morphological features which are somehow disturbed, which lead to the disease being studied. Examples of this is where an accepted mechanism 'starts' with a genetic defect, yet the consideration of how such defects have initiated is simply not considered. Cancer can be explained to initiate from many environmental causes, yet orthodox mechanism explanations begin after the cells have changed to an initial cancerous state, in many cases recognising 4 stages of development, yet not considering what pushes the cells to each stage. This is typical of orthodox mechanistic models. Yet, in the case of MCS, orthodox medicine insists that it must have a clear mechanism to link it to any of the millions of environmental exposures that a sufferer is exposed to. The vast number of studies that would be required to precisely establish a mechanism for the many combinations of environmental exposures to a genetically variable population will prevent clear mechanisms ever being established for a spectrum as broad as MCS.

To propose biological mechanisms to explain MCS is in-itself, a display of 'normalisation' in a medical system with such 'positivist' views. To some experienced doctors, it is apparent that susceptible individuals are suffering neurotoxic injuries, so to focus on underlying hypersensitivity mechanisms is missing the point in addressing the real problem (Donohoe 2008): As there are clearly people affected, it is rather to take care of them than ignore or

dismiss them due to there being no established mechanism. Nevertheless, it can be worthwhile to examine a popular hypothesised mechanism from recent studies since in doing so, the complexity and huge overlaps with many other health conditions become clear.

There have been many hypotheses put forward to explain MCS. These include neurogenic inflammation (Meggs 1993,1995b, Bascom 1997), sensitization of limbic neurons (Bell 1992, 1995, 2001), classical conditioning (Siegel 1997; Van den Bergh 2001), and oxidative stress involving nitric oxide/peroxynitrate (Pall 2003). “A common denominator of the models seems to be the assumption of an acquired, persistent hyper-reactivity most often developed during a period characterized by high strain on the bodily systems” (Andersson 2016p80).

The hypotheses overlap with observed effects of environmental xenochemical exposures present in many studies. One of the proposed mechanisms by Martin Pall is outlined in Appendix 1 to this chapter.

To put the mechanism concern in perspective, it should be realised that “the mechanism of action of most toxicants is unknown” (Welshons 2003p995). We simply do not know mechanisms involved with many known human carcinogens: dioxins and arsenic for example. So logically, for an illness caused by environmental toxicants, it is not surprising that the MCS mechanism/s are not established. Endpoints, such as tumours or liver toxicity in cancer, are measured without understanding the pathways leading to the pathology (Hanahan 2000). Pathophysiology is incompletely understood in Alzheimer’s disease, amyotrophic lateral sclerosis (ALS), Parkinson’s disease, Huntington’s disease, and other motoneuron diseases (MND). It is therefore a convenient argument that an MCS mechanism is not established, for an otherwise inconvenient public health condition for industries producing toxic products.

We have evolved through the last few hundred years with bodily systems, such as the immune system, protecting against viruses and bacteria, and surviving plagues. The human body has never been subjected to the myriad of toxins and electromagnetic fields as we are experiencing today. The irony of the situation is that those that have the best alert systems to environmental stressors are going to become the sickest.

The above section referred to some possible MCS mechanisms. The mechanism complexity and the overlaps with many other diseases has been demonstrated. It illustrates the difficulty and

large number of studies required to establish the mechanisms for MCS demanded by orthodox medicine. The value of insisting on a mechanism becomes an excuse and stalling tactic for orthodox medicine not to recognise MCS.

SILO RESEARCH AND OVERLAPPING CONDITIONS

In the previous sections covering epidemiology and mechanisms, overlaps with many other health conditions have been seen. MCS does not exhibit unique pathways and organ effects. In reading the scientific studies on environmental effects, one could be forgiven for thinking that the health effects can simply involve a myriad of pathways/mechanisms and one's present and past exposure history, and genetic make-up, will determine the main health condition that will be experienced, such as MCS, CFS, CIRS, EHS, etc. So to illustrate this situation this section will consider briefly some of the MCS overlapping conditions. In doing so, the similarities and overlaps can be seen indicating that MCS does not present a unique set of symptoms: environmental exposures result in a broad range of overlapping health conditions. MCS is rather part of the spectrum of possible effects.

There is a general lack of funding into mechanistic, diagnostic and treatment protocols for MCS. However in some of the overlapping conditions there has been research and progress to a greater extent. Yet usually the overlapping conditions are considered on their own. Many of the findings, however, may be applied to MCS. Dependent on the researcher's funding and previous experiences there are many studies done on common overlapping conditions such as electromagnetic-hypersensitivity syndrome (EHS), chronic fatigue syndrome (CFS), fibromyalgia syndrome (FMS), chronic inflammatory response syndrome (CIRS), central sensitisation (CS), gulf war syndrome (GWS), myalgic encephalomyelitis (ME), the Vietnam veteran health problems, Lupus, and irritable bowel syndrome (IBS). Occasionally some researchers become aware that there is another condition that greatly overlaps such as the overlap between MCS and EHS (Belpomme 2015). It has been shown that people with one functional somatic syndrome likely suffer from another (Aaron 2001). An example is where 51% of patients with CFS and 49% of patients with FMS have irritable bowel syndrome (Whitehead 2002). Yet if that study example had considered MCS as well, then that would likely have shown significant

overlaps. These studies can at times foster further studies to look for common predisposing risk factors (Moss-Morris 2006, Hamilton 2009).

Although there is a broad spectrum of environmental effects on population health, it is a normal trait by scientists to try to isolate studies and exclude any perceived confounding situations. This can see the deliberate exclusion of people in the study that suffer also from MCS, producing extensive silo research areas. Some examples overlapping with MCS are the research areas of the central sensitisation (CS) condition, the chronic inflammatory response syndrome (CIRS), and even the schizophrenia condition expanded on below, along with EHS. Significant portions of MCS sufferers have one or more of these conditions.

Central Sensitisation

Central Sensitivity (CS) or Chronic Multisystem Illness (CMI) refers to a heightened sensitivity condition through the central nervous system. This frequently precipitates chronic pain. It has an initial wind-up period in respect to pain intensity over time. Peripheral, repeated nociceptive input establishes improved neuronal pathways for such pain transmission producing functional changes in the brain (Yunus 2007, Wallace 2005, Seifert 2011). Even after the initial source of pain may have healed, the same pain pathways may remain active with pain still being felt by the sufferer (ICP 2016). A source of chronic inflammation, e.g., arthritis, can act as a source of nociception. Neuroendocrine-immune pathology can also develop CS (Desmeules 2003).

Many doctors fail to recognise a CS condition in patients: it needs a different approach than would otherwise be taken from a non-detailed (non-history-style) diagnosis approach (Yunus 2008). It is a challenging idea that localised pain is not necessarily an indicator of a local physiological problem

So there is an increasingly recognised CS condition which loosely fits sensitisation phenomena typical of MCS. Recognition of MCS and overlapping conditions as being a CS is gradually occurring (Yunus 2008).

Schizophrenia

The high levels of C4a expression from C4 genes produced by biotoxins in the following CIRS section overlaps with the breaking research into the causes of schizophrenia. In 2016 links between C4 gene expression of C4a and schizophrenia represented one of the first consistent biological markers of a psychological condition (Sekar 2016). Yet the schizophrenia research

had not found the C4 connections by studying environmental exposures and had just made this historical pathological finding. This overlap of two silo research areas is another example of how environmental exposures can result in neurological conditions.

Chronic Inflammatory Response Syndrome (CIRS).

This syndrome has had much attention in recent decades with significant diagnosis protocols developed. It represents a prime example of silo research due to its overlaps into many health conditions including MCS. This section illustrates effects from the thousands of biotoxins connected with mould. It is not comprehensive, but reveals that there have been many studies showing myriad effects. Many of the effects mimic or amplify manufactured xenochemical exposures, and as will be seen in the following section on EHS, mould growth is magnified in the presence of electromagnetic fields, such as wi-fi.

The overlaps with MCS are large. The following coverage addresses effects in CIRS which are in such overlaps. It is striking that these effects are more defined by more research on this condition than for MCS.

The orthodox view has been summarised by Tuuminen (2017p2):

The official rhetoric of denial of the mold-related illness can be summarized into three main points: (1) asthma is the only clear disease that can be associated with moisture-damaged buildings; (2) there is not sufficient evidence that dampness and mold overgrowth are associated with adverse health conditions; and (3) the mechanisms causing dampness-related illness are still unknown.

However, it will be seen below, this view clashes with many studies done on this condition.

The orthodox views can lead to the use of antibiotics and steroids. These can enhance fungal growth in many parts of the body including gastrointestinal, nasal, and sinus tissue (Hope 2013). This is another example where a lack of knowledge can send a sufferer down a complex path of further health problems.

Dampness and mould in buildings are common. Occurrence is between 18% to 50% of all buildings (Gunnbjörnsdóttir 2006; Mudarri 2007). The likelihood of a significant portion of the population experiencing exposures is therefore significant. In the past the Massachusetts

Department of Public Health attributed 50% of all illness to indoor air pollution (CoM 1989p15). It is unlikely such a statistic has declined since then.

Water damaged buildings can be due to inadequate building design, inappropriate ventilation, faulty construction of crawl spaces, fake stucco cladding without adequate caulking, flat roofs, damp basements, not fixing water leaks, etc. Such conditions can start hosting fungi, bacteria, mycobacteria, and actinomycetes.

People affected by water damaged buildings are diagnosed by functional medicine as having contracted mould illness. The illness rarely conforms to allergy diagnoses. It is an internal inflammation caused by an abnormally functioning immune system. The term “mould illness” is commonly regarded as a subcategory of a biotoxin illness called Chronic Inflammatory Response Syndrome (CIRS). CIRS can be defined as:

An acute and chronic, systemic inflammatory response syndrome acquired following exposure to the interior environment of a water-damaged building with resident toxigenic organisms, including, but not limited to fungi, bacteria, actinomycetes and mycobacteria as well as inflammagens such as endotoxins, beta glucans, hemolysins, proteinases, mannans and possibly spirocyclic drimanes; as well as volatile organic compounds (Vetter 2018p24).

More details on CIRS can be found in Appendix 2.

In conclusion to this section on CIRS:

In a classic silo style situation, CIRS and MCS have been treated by the medical and scientific community as being separate illnesses. There is usually an acknowledgement that people with CIRS also develop MCS characteristics and that people with MCS usually have mould sensitivity. In reading the literature studies, mechanism and diagnosis characteristics, one can be tempted to say they are one and the same, yet no clear consensus on such can be found. An interesting study on a nine member family who moved into a house which was water damaged saw most family members develop “a plethora of mucosal irritation, neurological, skin, allergic, and other symptoms, with all family members ultimately developing a multiple chemical syndrome”

(Tuuminen 2017). There are other more generalised papers that show development of new onset chemical sensitivity after mould exposures (Rea 2004).

It is a large area of undone science in studying whether the tests and treatments for CIRS work on MCS sufferers who have developed the condition through exposures to manufactured xenochemicals. As possible environmental exposures overlap it will be a difficult task to perform effective epidemiological studies on humans.

Electromagnetic Hypersensitivity Syndrome (EHS)

This section on EHS is more extensive than the previous sections on silo research and conditions. This is because there is such a significant overlap of this condition with MCS: most MCS sufferers experience various degrees of EHS (and vice versa). It illustrates clear cause/effect studies and experiences from environmental electromagnetic field (EMF) exposures. However, unlike MCS sufferers who can usually, to some degree, reduce their environmental exposures to toxins for example, the EMF exposures are more widespread and invasive.

A brief coverage of the overlaps between the EHS and MCS conditions will firstly be made in respect to diagnosis and mechanisms. This will be followed by a short section illustrating that the terminology using 'hypersensitivity' is rather misleading due to implying that only a part of the population is affected and should rather be referring to the more-sensitive part of our population who can be affected by EMFs. This moves into mentioning the industry tactics to downplay this and other effects from EMFs. The following section discusses the industry viewpoint in its attempts to downplay and manufacture ignorance on any findings. This is followed by a section highlighting how the orthodox medicine s

ystem usually misses connections of symptoms to EMF exposures. The EHS section finally finishes in mentioning the large number of studies which show EMF effects on children, reproduction and the immune system.

EMF sensitivity was first described by Rea (1991). Later, in a report on the condition to the European Commission, the term electromagnetic-hypersensitivity syndrome (EHS) was coined (Bergqvist 1997). Other key studies followed (Santini 2002, 2003).

EMFs are constantly encountered in society today. They can be generated by power lines, mobile phones, mobile phone towers, weather radar, home wiring, electrical appliances, home computers, tablets, wi-fi, bluetooth devices, TV and radio transmitters, smart meters, baby monitors, security and anti-theft devices, airport security walk-throughs, automated highway toll systems, fluorescent lights, hair dryers, electric blankets, clock radios, home electrical wiring, etc. EMFs are invisible and not readily sensed, so they usually cannot be avoided. One can elect to buy organic produce, drink filtered water, stay away from cigarette smokers or pesticide applicators, yet EMFs can be ubiquitous and constant in the modern environment.

EHS seems to have a growing recognition by disability administrators and case workers, health authorities, politicians, and courts of law. Although being a chronic multisystem illness, the underlying cause is clearly the environment. EHS symptoms may at first occur occasionally, and over time increase in severity and frequency. They include sleep disturbances; chronic fatigue; chronic pain; poor short-term memory; difficulty concentrating (“foggy brain”); anxiety and depression; skin problems like prickling, burning sensations and rashes; muscle ache and pain; concentration difficulties; irritability; nausea; flu-like symptoms; dizziness; loss of appetite; movement difficulties; visual problems; tinnitus; frequent night-time urination including bedwetting among children; heart palpitation; reproductive problems (including infertility); difficulty regulating blood sugar levels; nose bleeds; asthma; cold extremities; cancer; and various other symptoms. Sensitive people develop these symptoms at exposure levels not affecting the general population. Some are so affected that they must cease work and change their lifestyle, with others experiencing mild symptoms and trying to avoid the fields.

It has been estimated that the population percentage of EHS self-reporting persons is 5% in Switzerland (Rösli 2010), 5% in Ireland, 9% in Sweden, 9% in Germany, 11% in England, and 13% in Taiwan (Hallberg 2006, Belpomme 2015, Tseng 2011). One Austrian study found that the prevalence of EHS had increased by 75% between 1994 and 2008 (Schröttner 2008). It is expected that the EHS incidence will grow as more EMF is introduced, especially the 5G+

bands. In the US only 6% of adults owned a cell phone in 1993, this had increased to 95% by 2018 (Hertsgaard 2018).

Due to the growing awareness of EHS, in 2004 the WHO described it as a phenomenon where health effects are experienced when using or being close to devices emitting magnetic, electric, or electromagnetic fields ... EHS can be experienced and can debilitate the affected persons (Hansson 2006). They later renamed the syndrome as “Idiopathic environmental intolerance (IEI) attributed to electromagnetic fields” influenced by industry representation on its panels reflecting the problem on the individual rather than the products causing such. The WHO however will not recognise EHS as a diagnosis (WHO 2005).

In January 2019, the French Court of Cergy-Pontoise (CP 2019) ruled that EHS can be disease experienced in the workplace at radiation exposure levels considered safe by the government.

EHS can be regarded as being present in the population on the sensitive side of the distribution curve. It is now becoming well recognised that everyone can be affected by EMF to various degrees. The Council of Europe adopted a report to “Take all reasonable measures to reduce exposure to electromagnetic fields, especially to radiofrequencies from mobile phones, and particularly the exposure of children and young people who seem to be most at risk from head tumours”, or “Pay particular attention to ‘electrosensitive’ people who suffer from a syndrome of intolerance to electromagnetic fields and introduce special measures to protect them, including the creation of wave-free areas not covered by the wireless network” (COE 2011).

The Bioinitiative Report in 2012 and 2016 provided evidence of EMF effects on “gene and protein expression, DNA, immune function, neurology and behaviour, blood-brain barrier, brain tumors and acoustic neuromas, childhood leukemia, melatonin, Alzheimer’s disease, breast cancer, fertility and reproduction, fetal and neonatal disorders, autism” (Belyaev 2016p366). In later studies on animals, radiation from mobile phones was found to reduce the number of eggs in the ovaries (Shahin 2017, Türedi 2016), and increased risk of miscarriage in pregnant women in magnetic fields similar to that from mobile phones (Li 2017). Autoimmune diseases improved when EMFs were shielded (Marshall 2017).

The articles linking health effects from typical EMF exposures continue to grow in numbers. A meta analysis recently linked mobile phone usage to brain tumour incidence based on 46 case-

controlled studies (Choi 2020). The Mayo Clinic and the National Cancer Institute cite studies showing a link between heavy cell phone use and an increased incidence of gliomas and salivary gland cancers. Yet there are industry sponsored studies showing reduced tumour risk.

There remains much undone science in the health effects from EMFs, such as how 5G could possibly have a role in promoting viruses, such as coronaviruses, in human cells (Fioranelli 2020).

It is not new that health effects can occur from constant EMF exposure. Solar flares occur in 11 year cycles in respect to their intensity. The intensity from high to low parts of the solar flare cycle is equivalent to varying from 7 to 1 chest X-rays in a 6 hour flight (O'Brien 2019). This is usually accepted as an explanation as to why pilots have one of the highest incidences of lymphomas of any profession. Flight attendants have a high incidence of reproductive disorders, miscarriages, and infertility. The introduction of millimetre wave airport full body scanners in the last decades adds to exposures.

It is interesting that most insurance companies will not now provide public liability insurance for the mobile phone companies for ill effects from non-ionising radiation.

EHS Overlaps with MCS: Diagnosis and Mechanisms

There is a significant overlap between EHS and MCS to such an extent that most researchers on these conditions regard them as two etiopathogenic conditions of a single pathological disorder. The study findings by Belpomme et al. (2015) reflected this.

Belpomme et.al.'s findings are reinforced by subsequent studies such as where approximately 80% of EHS self-reporting patients had one, two or three detectable oxidative stress biomarkers in their blood. As for Alzheimer's disease, cancer or other pathological conditions, these people show an objective pathological disorder (Irigaray 2018a). Another study found that eliminating EMF exposure produces symptom changes in 90% of autoimmune disease patients irrespective whether they had EHS (Marshall 2017). Other studies have found EHS sufferers have a profound increase in facial mast cells (Johansson 1995, 2001).

The reproduction of clinical symptoms in blind and double blind studies has always been a problem in MCS and EHS sufferers. This has reinforced that MCS and EHS are psychiatric

illnesses rather than pathological ones. However the complexity of these conditions with varying individual reactions does not readily lend itself to a simple scientific approach of one exposure and not taking into account the current status of the tested individual and their varying background of environmental influences which produced their condition. Most sensitive people do not know the chemical or EMF concentration levels to which they are exposed when they have an immediate or latent effect. It is a stab in the dark in any study to hit upon the combination that affects an individual, let alone a group of sensitive people. Further study difficulties were also mentioned in the MCS epidemiology section.

Simultaneous low frequency magnetic fields and toxicant exposure has been found to increase the harms caused by the toxicants (Juutilainen 2006). Simultaneous exposures have been shown to release proinflammatory cytokines (De Luca 2010b, Costa 2010, Burns-Naas 2001). Other combined effects have been evidenced where mobile phone EMF accelerates mercury vapour release from amalgam teeth fillings (Mortazav 2008). Two studies have shown that prenatal (Choi 2017) or postnatal (Byun 2017) exposure to mobile phone results in elevated neurobehavioral effects in children with high lead levels compared to children with elevated lead alone.

As with MCS, EHS has a diverse range of symptoms occurring singly or simultaneously in different organs in different sufferers. There is clearly no single mechanism involved. At the intra/inter cellular level there are possible interactions via free radical formation or oxidative or nitrosative stress (Friedmann 2007, Simko 2007, Pall 2007, Bedard 2007, Consales 2012, Pacher 2007, Desai 2009, Straub 2010, Gye 2012, Yakymenko 2015). These can have pathways involving reactive oxygen species, peroxidation activation, DNA oxidative damage, antioxidant enzyme activity, voltage-gated calcium channels, or peroxy-nitrate formation. Detoxifying enzymes can also differ in EHS sufferers (De Luca 2014). Other studies identify ATP effects, myelin integrity effects, etc.

The European Academy for Environmental Medicine (EUROPAEM) EMF Guideline 2016 (Belyaev 2016p374) recommended “classifying EHS as part of CMI” (chronic multisystem illness) (Pall 2007, Warnke 2013). So here again there is recognition of the many overlapping illnesses.

Electromagnetic-Hypersensitive or Simply Electromagnetic-Sensitive?

In looking for studies showing health effects from EMFs many times below allowable exposure levels, one finds thousands of studies. The allowable levels presently used in society have been based only on thermal effects: the intensity required to raise human skin temperature by one degree centigrade. Thousands of studies documenting health effects 50-plus years ago were listed by the US Navy (Glaser 1971).

EHS, like MCS, can gradually develop from chronic exposures or after a brief, high intensity exposure (Carpenter 2015). In studies observing ELF/ RF effects between EHS and control subjects, similar cellular effects have been observed in both groups. But “It is likely that compensatory reactions at a more complex level of biological organization such as reactions of tissues, organs, and organ systems are less efficient in persons with EHS, thereby providing a stronger connection of the EMF cellular response with symptoms of hypersensitivity” (Belyaev 2016p369).

Pall (2018p1) has summarised many studies which support his eight clear health effects on humans (not just those with EHS).

EMFs:

- 1) “Attack our nervous systems including our brains leading to widespread neurological/neuropsychiatric effects and possibly many other effects. This nervous system attack is of great concern.
- 2) Attack our endocrine (that is hormonal) systems. In this context, the main things that make us functionally different from single celled creatures are our nervous system and our endocrine systems – even a simple planaria worm needs both of these. Thus the consequences of the disruption of these two regulatory systems is immense, such that it is a travesty to ignore these findings.
- 3) Produce oxidative stress and free radical damage, which have central roles in essentially all chronic diseases.
- 4) Attack the DNA of our cells, producing single strand and double strand breaks in cellular DNA and oxidized bases in our cellular DNA. These in turn produce cancer and also mutations in germ line cells which produce mutations in future generations.

- 5) Produce elevated levels of apoptosis (programmed cell death), events especially important in causing both neurodegenerative diseases and infertility.
- 6) Lower male and female fertility, lower sex hormones, lower libido and increased levels of spontaneous abortion and, as already stated, attack the DNA in sperm cells.
- 7) Produce excessive intracellular calcium [Ca²⁺] and excessive calcium signalling.
- 8) Attack the cells of our bodies to cause cancer. Such attacks are thought to act via 15 different mechanisms during cancer causation.

There is also a substantial literature showing that EMFs also cause other effects including life threatening cardiac effects. In addition substantial evidence suggests EMF causation of very early onset dementias, including Alzheimer's, digital and other types of dementias; and there is evidence that EMF exposures in utero and shortly after birth can cause ADHD and autism".

Pall then elaborates on a mechanism of activation of voltage-gated calcium channels as to how the variety of effects can be initiated by EMFs.

The International Agency for Research on Chemicals (IARC) classified radiofrequency EMFs as possibly carcinogenic to humans (Baan 2011) and warns that EMF exposure is dangerous. It has been argued that based on the same criteria used by IARC in light of more-recent studies, the EMF radiation should now be reclassified as a Class 1 "Definite Carcinogen" (Miller 2018, Hardell 2013,2017, Kundi 2012). This is also evidenced by various studies such as on breast cancer incidence from carrying mobile phones near the breasts (West 2013).

An area of study presently being countered through industry backed studies, is the effects of EMF on the blood-brain barrier permeability. Many studies have found permeability in the presence of EMF (Huang 2016, Tang 2015) while others find no effects (Masuda 2015). A traditional aspect countered by industry-backed research were studies, as far back as 1979, finding health effects from power transmission lines (Wertheimer 1979, Li 1998, Mizoue 2004, Beale 1997, Lowenthal 2007, Draper 2005).

A prominent practitioner treating sufferers with multisystem illness, Dr D Klinghardt, comments: "the human bio field uses frequencies in the ranges from ten to thirty gigahertz. That has been measured and established by clever Russian scientists fifteen years ago, twenty years ago. The one that rolled out already in London is twenty-eight gigahertz" (Klinghardt

2019p10). Most wi-fi seems to be within this range, such as in Australia and America. This would indicate clear possibilities of affecting the human biofield through interference with such.

With his large practices in both America and Europe, Klinghardt (2019p7) has observed, and mentioned in an interview:

When a parent is exposed to constant bombardment with electromagnetic radiation... they have increased food allergies, they have increased brain fog, and a variety of symptoms that they are not relating to the electromagnetic exposure. If these parents have children, [the parent negative responses] will be quadruple[d] or increased more in the offspring. And the offspring will be extremely electrosensitive. And if these children, and I have some in the third genera[tion], [then] have children, their children will be autistic. So, there is a cascade of worsening from generation to generation. And we understand now that the Wi-Fi amongst many of its other blessings, is that [it] damages the DNA. And that is what causes problems [i]nto the next generation. And it goes up exponentially.

Industry representatives typically claim that there is insufficient science to establish any health effects from EMF exposure. This is considered in more detail in chapter four covering examples of ignorance. A fitting comment has recently been made by Dr S Goldberg (2019p4).

The misconception is that we don't have enough science to make a decision as far as whether or not we need to warn the public, and take action to lower exposures. And this is completely false. We have such clear evidence that microwave radiation, that it is what I would call a broad spectrum pathogen. So it causes all sorts of different diseases. And a multisite carcinogen. So it causes cancer in many different parts of the body. We understand a whole bunch of basic mechanisms about why this would be. We understand that. So it's completely false from the scientific perspective when you hear, "Well, we need more research." "It's still a debate." There isn't anything to debate. That's I think the biggest misperception out there.

In light of the industry assertions that there are not enough studies to make any conclusions, then it could be regarded as an uncontrolled experiment on the population which contravenes many human rights, even the Nuremberg Code.

Orthodox Medicine Viewpoint

In the early 1900s, factory workers began to show up at doctors' offices with symptoms that looked like the common cold, the flu, and bronchitis. The doctors simply tended to the symptoms. Most knew about asbestos and heard the early research in connection to its early mortality aspects, but didn't think one invisible toxin could be the root cause of all these mystery illnesses. The number of workers totalled thousands over the years, but never got the medical care they needed.

As an analogy, if we replace asbestos with EMFs, the 'mystery symptoms' are extreme fatigue, insomnia, brain fog, depression, anxiety, and any of the myriad of other effects mentioned before, that often don't go away with diet, exercise, and supplements, let-alone drugs. As for MCS, a comprehensive medical history covering all symptoms and their occurrences of EMF exposures, is important in making the diagnosis. The sufferer's best treatment is to reduce or remove themselves from EMF exposures. The total body burden from other environmental exposures should also, of course, be addressed (Belyaev 2016). This is far from being able to be achieved in an average 15 to 20 minute orthodox doctor's appointment.

Children and Reproduction

One survey in 2014 found that 75% of 4-year old children possess a cell phone (Kabali 2015). But children are at heightened risk, as their head absorbs more radiation compared to adults:

When electrical properties are considered, a child's head absorption can be over two times greater, and absorption of the skull's bone marrow can be ten times greater than adults (Gandi 2012p34).

The Gandi et al. paper highlights the shortcomings of the present allowable radiation levels established from adults.

This is a particular concern in children, given the rapid expansion of use of wireless technologies, the greater susceptibility of the developing nervous system, the hyperconductivity of their brain tissue, the greater penetration of radiofrequency

radiation relative to head size and their potential for a longer lifetime exposure” (Belpomme 2018p643).

Stem cells are more affected by EMFs than any other cell type illustrating the foetal susceptibility to EMFs (Burgio 2015). It is accepted in society to have ultrasounds checking foetal development during pregnancy, yet these are not just sound waves, they emit significant EMFs. This has long been known (Liebeskind 1981,1982) and there have been calls to end such practice (Berlatsky 2015).

There are many isolated studies on EMF effects with different end points. Two examples are:

- Lower sperm counts and inactive or less mobile sperm (DEODC 2020, Adams 2014, Avendaño 2012, Gorpichenko 2014). Sperm counts around the world declined by 50% to 60% between 1973 and 2011 (Levine 2017). Of course, as illustrated in other chapters, there are also many other possible environmental co-contributors.
- prenatal exposure to power-frequency fields can almost triple a pregnant woman’s risk of miscarriage (Li 2017). Other studies also show a link between magnetic field exposure and a heightened risk of miscarriage (Li 2002, Lindbohm 1992, Juutilainen 1993, Wang 2013, Mahmoudabadi 2015, Zhou 2017).

Various reports have occurred over the years where mobile phone towers have been positioned near schools and health effects have been noticed from between months and years. One example was in California where 4 students and 3 teachers contracted cancer after the installation of a nearby tower (CBS 2019)

From an experiential background, treating hundreds of patients, D Klinghardt has noticed a synergistic effect with vaccines. He sees that the more vaccines a child has received, the more electro-sensitive their children will be (Klinghardt 2019).

Immune System Effects

There is plenty of evidence that EMFs depress the immune system. The BioInitiative Report in 2012 had 129 references (Grigoriev 2012) used to substantiate such a claim. Furthermore, the EMF detrimental effect on mineral absorption and effectiveness, such as for zinc, has also been shown. Zinc is a potent mineral the body uses to fight viruses, such as COVID 19.

General Health Effects

The general health effects of EMFs on the general public and environment can be best summarised by Martin Pall's summary (Pall 2020): neurodegenerative and chronic diseases, infertility, cancers, autism, hormonal dysfunction, DNA damage, still-births, blindness, kidney failure, insect holocaust, conflagrations of forests.

In Conclusion to this Section on EHS

The overlap between MCS and EHS is so pronounced that studies such as the Belpomme et.al study in 2015, automatically consider both illnesses. EMF exposures simply add to the environmental exposure load on MCS sufferers. EMF exposures, like chemical exposures, are mostly invisible and the effects are on molecular and cellular levels.

Most industry sponsored scientific studies on health effects of various technologies are performed on an average population: not a varied sensitivity test population. The test subjects are usually vetted to ensure they are 'healthy' or 'average'. On the other hand, most studies on EHS begin with sensitive test subjects. While EHS sufferers react more quickly to lower exposure levels than the general public, this condition occurs after their body's defences reach a point where an 'average' person suddenly became a 'sensitive' person. This is a similar story to MCS.

As in other environmental exposures mentioned in the last chapter, the effects of EMF on the majority of the population, will likely be latent and more pronounced in subsequent generations. DNA damage is increasing being found to occur from radiation outside of the ionizing spectrum (Aitken 2005, Akdag 2016, 2018, Al-Serori2018). One of the main points seen in this section however, is that the 'hypersensitivity' word in its title is deceiving. People suffering from EHS have developed this syndrome from their environmental exposure to EMFs. It is indirectly related to their genetic susceptibility to such. This section has illustrated that EMFs will have varying health effects on the public in general, whether in the current generation or subsequent ones. The EHS sufferers are simply those in the current generation that have/are being affected.

Conclusion of Silo Research Section

In conclusion to this silo research section, environmental exposures do not exhibit unique pathways and organ effects. In considering only a sample of overlapping conditions, similarities and overlaps can be seen showing that MCS does not present a unique set of symptoms: environmental exposures result in a broad range of overlapping health conditions with varying labels.

DISEASE CAUSALITY PARADIGM

MCS will now be considered through the lens of medical paradigms. This section discusses the paradigmatic aspects which prevent recognition of emergent diseases in general: MCS in particular. The orthodox medical system has tried to move from centuries-past belief systems, to a perceived scientific basis with strong positivist approaches. Yet these positivist approaches become a hinderance to change due to the resultant difficulties in recognising complex and overlapping emergent diseases such as MCS.

Prior to 1854, doctors believed in Miasma Theory. This theory was based on the belief that disease was spontaneously generated by 'bad air'. A theory sceptic, Dr John Snow, observed that a cholera outbreak in London appeared to be occurring around a particular water well. He was subject to much ridicule by the medical fraternity for such a hypothesis. However once the well was shut down the outbreaks subsided (Snow 1857, Hempel 2007). This heralded the Kuhnian crisis (the start of the end) of the Miasma Paradigm: some paradigms take years/decades/generations to merge into the next paradigm.

Today, as in the past, most of the public do not see any reason, let alone time, to question the authority of government health departments and orthodox medical practitioners. Their faith in science is from being taught in school that it is a simple, respectable, logical process. It starts with observation, through hypothesising, experimenting, evaluation, then starting such a process over until a consistent set of conclusions can be made. The general public's belief in the medical system is that sound science is applied as is its foundation, any evidence to the contrary is an exception to the rule. Even public media have a tendency to suppress negative news due to this standard image of science (Buckman 2017, Lupton 2012, Martin 1996).

The orthodox medical system has historically tried to adopt a strict interpretation of the scientific approach to illustrate its professional respectability. It has always valued facts over hypotheses and theories that have not been adequately tested. It has a clear preference for being able to measure and clearly define. Medical science is therefore said to have as its basis a positivist viewpoint: “A philosophical system recognizing only that which can be scientifically verified or which is capable of logical or mathematical proof” or “The theory that laws and their operation derive validity from the fact of having been enacted by authority or of deriving logically from existing decisions, rather than from any moral considerations” (Oxford Dictionaries 2016).

With this viewpoint, a paradigm has developed for health conditions that if clear measurements cannot be made on a particular health condition, then it does not exist (Romanucci-Ross 1991, Pall 2007, Winters 2003). It is an immense barrier for the recognition of emergent conditions such as MCS. There has been condemnation of this viewpoint by prominent social scientists due to discounting knowledges about health conditions below the required level of cognition or scientificity (Foucault 1973,1976, Lupton 2012, Romanucci-Ross 2007). It is however acknowledged that overly reductionist critics of positivism sometimes overlook positivist contributions that have led to the debunking of former scientific myths: there have been formal scientific awards made for new discovered innovations which have led to paradigm shifts, such as Dr Snow’s example above.

Adding to the maintenance of the positivist paradigm is the insurance and legal system. These systems pressure the medical system to quantify their evidence of health conditions. This then responds to and reinforces a cultural trust in numbers.

To therefore argue for the existence of a unique health condition that does not fit normal diagnosis criteria and affects a small percentage of the population, becomes quite a difficult task. Furthermore when an argument is presented, ‘positivist scepticism’ is experienced where the political sectors of medical science, law, and the insurance industry will not accept the validity of a novel medical explanation without an acceptable level of proof. This is regarded as neutral, measured, rational and scientific. The possibility that a theory is valid with some uncertainty is often deemed biased, emotional, and unscientific (Pall 2007). Treating uncertain conditions that could be psychosomatic as though they are caused by environmental exposures

is medically, politically, and economically difficult. It underscores that while sceptical decision-makers wait for an undeniable crisis, for a critical mass of sufferers, for a moment of truth, for precedent, a public health crisis can steadily grow.

As far back as the 18th century biomedicine started to localise disease to particular body organs and disease became a visible and material entity (Van Diest 2006). With the advent of medical technology diseases became measurable. The philosophy of Rene Descartes (1596-1650) initiated a clear distinction between the body and the mind and saw medical science become a measurable study of the body.

In MCS, there is no localisation to one organ, clear objective measurements, or consistent cause/symptoms. This puts it outside the above medical, insurance and legal system paradigm. But there are many other disbelieved health conditions in the same situation, e.g., back pain, where patient reported suffering is inconsistent with measurable pathology (Niemeyer 1991).

To strengthen the appearance of a scientific approach the medical system openly promotes a peer review system. The process is inherently conservative as covered in the ignorance chapter. Reviewers are not inclined to support unorthodox research or high risk hypotheses. Thus to achieve publication, researchers tend to stay main-stream on low risk projects (Laudel 2006). This situation historically helps to protect existing paradigms in medicine to such an extent that medicine has sometimes been referred to as a 'closed system of knowledge' (Romanucci Ross 1991)

At its core, science is a study of phenomena in the environment, and it is the formulation of hypotheses and theories based-on a continual re-appraisal process. This continual process starts with observation. It is then quite stark that an emergent condition in up to 27% of the population does not trigger a classic scientific investigation but is simply criticised as not being real, by a 'science based' medical system.

The medical establishment wishes to maintain an image of its search for truth without being affected by power politics. However there is much evidence that power politics greatly influences medical science (Boffey 1975, Dickson 1984, Primack 1974, Martin 1996a,b). There have been well-researched books written on how the science in drug testing has been distorted by the pharmaceutical industry and its influence on the approval processes of the government

authorities (Davis 2013, Abraham 1995, Gotzsche 2014). Furthermore, the influence of the pharmaceutical industry on medical practitioners and their associations is well known (Dukes 2014, Goldacre 2012, Angell 2004, Kassirer 2004, Gotzsche 2014). Packard et al. suggest that new illness "emergence" and acceptance involves both "an epidemiological phenomenon and a social process." They note that both those arenas involve politics, usually related to the class of people affected with the illness (Packard et al. 2004p2). It would be costly for industry to have MCS recognised, where chemical products and EMFs are acknowledged to affect human health. There has been clear evidence of industry influence to restrict debate, confound the science and the condition's recognition (CMA 1990, McCampbell 2001). A denial of such influences is common by the medical establishment.

The paradigmatic aspects of the medical fraternity of course extend through to the public. Although people wearing fragrances and other personal care chemicals obviously have pleasure in applying them, they in general come with great risk to the individual due to their sometimes extremely toxic nature. The risk extends to those in the immediate locality and draws a direct comparison to a smoker in the past. But while there are many examples where industry has contaminated our living environment, frequently with government departments being aware, the personal care industry with its personal care products is supported by an ignorant public coming at a great risk to their personal health and that of their children. Julian Cribb (2014p59) appropriately commented "Breast cancer comes at a high price to pay for great hair".

In writing about the "huge medical iceberg of accumulating silence, reticence, and indifference toward environmental toxins", one of Sellers' (2018) examples included a mother's experience in Texas. She had taken her three year old daughter to a local hospital for help after her child's legs went limp, so she kept falling, and was generally weak. The hospital doctors came up with a diagnosis of Guillain-Barré syndrome caused by an 'idiopathic' immunological attack on the nerve linings. What had been completely overlooked was that the family lived beside a lead smelter.

It can be said that this section has shown that in general, the orthodox medical system truly "cannot see the forest for the trees". Their preoccupation for a positivist approach looking for clear proof and detailed mechanisms results in missing the environmental effects along with large areas of undone science on health effects.

DISEASE AND ILLNESS

In this section another distinction by the orthodox medical system which is used to dismiss MCS is considered: the difference between a disease and an illness. The reliance by the orthodox medical system on organic or structured pathology, excluding brain and spinal cord diagnosis criteria is a convenient approach for the exclusion of MCS as a 'real' disease. Furthermore, the process required to classify a disease as legitimate subject sufferers to a normalisation process. As MCS is quite different to orthodox diseases the sufferers cannot fit the normalisation process where the problem is perceived to be with the individual rather than the environment the sufferers are exposed to.

The MCS condition is commonly dismissed by the medical fraternity by arguing that there is an absence of 'organic' pathology. With such dismissal, a default 'illness' label is then assigned since it cannot be regarded as a 'disease'.

Central Sensitivity Syndrome (CSS), of which MCS is part, does have "abnormal conditions" and "functional changes" (Yunus 2008). Brain imaging techniques and pain study neurophysiological testing enable neuroendocrine-immune systems in CSS be included in the pathology definition due to their objective and reproducible nature (Yunus 2007, Gracely 2002). Yet the term 'organic' pathology is used in the context of diseases with structured pathology, as if the brain and spinal cord are not human organs.

There seem to be only a few voices in the literature that argue that it doesn't matter whether one has a disease or an illness when it comes to patient care (Yunus 2008, Drossman 2006, Lipkin 1969). The difference between disease and illness results in a two-tier practice of medicine today (Drossman 2006, Jennings 1986, Van Houdenhove 2003): those with structural pathology are 'real' patients, and those without, such as the CSS/MCS patients, become the second-class patients (Yunus 2008). Yet nowhere in the Hippocratic oaths taken by Doctors is the requirement that service and respect must be only to those with structured pathology (Phillips 2015).

A disease-illness dualism, or an illness implied by a diagnosis, can also create quite a negative attitude in many doctors (McWhinney 1997, Yunus 2004). This attitude can initiate an

attribution of the patient symptoms to the patient themselves and commence taking the patient down the psychosomatic spiral pathway (Ford 1997, Nettleton 2006, Bohr 1996, Ehrlich 2003, Hadler 2003, McWhinney 1997). In the case of children with MCS characteristics, it is usually the mother that witnesses the reactions of her child and in trying to find sense in the attitude of the orthodox doctors pushes them for some rationality rather than a psychosomatic write-off. This can then see the orthodox doctors, ignorant of the MCS condition in the extreme, try to quiet an insistent parent by use of the Munchausen By Proxy Syndrome label. This has been seen to happen to mothers who have been too persistent in searching for answers and question/complain about the seeming second-class treatment of their children (Hayward-Brown 1999). In the author's experience, this has seen children removed from the parents and treated conventionally with a worsening of their general health condition. In the best outcome situation, when the parent realises the Munchausen By Proxy Syndrome label is about to be applied to them, and they sense some environmental links, they leave the doctor/hospital, never to return and treat the children outside the orthodox system.

It is interesting to note that psychological factors seem rarely attentioned in patients with a diagnosed disease (structured pathology) (Yunus 2008). Yet when a psychological diagnosis is labelled, all physical symptoms are then 'all in the head' (Kroenke 2006).

It has been suggested that the needed new paradigm is that illnesses are diseases as well (Yunus 2008). Disease should not be a reductionist concept since chronic disease in medical textbooks incorporates both psychological and functional aspects (Yunus 2008, Powers 2005, Longo 2000).

The whole concept of disease and illness is a relative one when considering MCS. It relates to classification of people fitting the normalised concept of health and being able to fit into society today. But is the MCS condition highlighting an unhealthy environment rather than unhealthy people? Are we misguided in expecting that those people must fit into an unhealthy environment? When MCS sufferers are withdrawn from offending environmental exposures, they seem to be healthier (Donohoe 2008p23): this has been a consistent finding and experience. As orthodox medicine attentions only symptoms, a standing back to look at the 'forest' rather than each 'tree' (the cause rather than the symptoms) is not in their reductionist-type paradigm.

This section has highlighted the difficulties in fitting effects from environmental exposures to conform with normal disease diagnosis and behavioural characteristics.

OBSERVATION, PROOF AND PATIENT RESPECT

This section highlights that the orthodox medical system and health authority demand for proof that the MCS condition is real, is an unnecessary requirement.

The initial basic scientific approach to 'observe' is not usually taken to its fullest extent by doctors and health professionals. Observation to orthodox doctors is usually what can be gathered within a 15 to 20 minute appointment to also provide adequate time to write appropriate prescriptions/referrals etc. If a doctor would spend adequate time to make more detailed observations to link environmental exposures as possible triggers to the health problems seen, they would be criticised by the system. Doctors are simply expected to fit standardised diagnosis criteria to symptoms/pathology, so in the case of MCS, without any standardised diagnosis criteria, it can only be concluded to be a patient belief and treated psychologically. This then becomes circular reasoning in that a psychological diagnosis is also not provable.

Through recent history there have been situations where observations at the clinical level resulted in significant improvements in health. Another to Dr Snow's cholera example mentioned previous (Disease Causality Paradigm section) is Sir Percival Pott who stopped a scrotal cancer 'epidemic' in chimney sweeps by requesting them to attention their genital hygiene (Edward 2011). Observation by doctors today however is restricted by a system requiring a fit to a defined set of diagnosis criteria. As there are no orthodox medicine MCS diagnosis criteria there is no slot to fit such sufferers. There are only a few that will refuse to put MCS sufferers in the too hard slot, so most sufferers are referred to a psychologist.

Without a clear test for MCS, the doctor is left with the patient's claims of health problems. Pain, for example, is not observable and defies language, but the characteristics can be diagnosed by experienced practitioners. It is a common human trait however, that when hearing a description of one's pain without obvious reasons, that doubt emerges (Scarry 1988). This presents the challenge to a doctor who has no experience with central sensitisation

conditions. In a clinical situation, to assist, the doctor must respect the patient's observations on exposures and effects. It is usually the CS patient in these conditions that has applied a scientific approach to their condition. For example, when they encounter a certain environmental exposure a particular health condition/symptom results. If such knowledge is not respected by the doctor then valuable observations are lost and a scientific approach is compromised.

Dr Mark Donohoe is one of Australia's most experienced and best known medical practitioners in the fields of Nutritional and Environmental Medicine. He has extensive experience with the MCS and related conditions. The following paragraphs draw on some of Dr Donohoe's comments earlier in his career.

The regulatory system allows the use of chemicals in the environment based-on isolated animal (usually on mice and rats) mortality tests. These are far-removed from the myriad of exposures and health effects which are possible in today's environment. The primary care health professionals should be the net for observation and "post-marketing surveillance" of health effects (Donohoe 2008p63). Yet when those few doctors try to assist affected people, especially in the context of legal proceedings, they are dismissed and discredited for such observations and environmental links found. Evidence of specialists and those of academic position dictating right/wrong, good/bad, science and anti-science in maintaining existing paradigms is common without having applied a basic scientific approach themselves directly with such patients. In many cases no data or successful experience in treating the MCS condition is required to substantiate such criticism (Donohoe 2008p25). Dr Loblay's dismissal of the health effects reported by the aircraft crews due to aircraft cabin fumes, without even examining or talking to any of the crew, is a prime example of this and is expanded on in a following section.

In orthodox circles the level of exposure of most xenochemicals that affect MCS sufferers is regarded as insignificant. MCS sufferers can react to concentration levels orders of magnitude below workplace recognised 'safe' levels and below recognised odour concentration levels that one can smell. But in the last decade there has been extensive research into non monotonic dose responses, especially on endocrine disrupting chemicals (Lagarde 2015). This is where more-severe effects can increase with lower doses of such chemicals. This research undermines

the very basis of classical toxicological and risk assessment of 'safe' exposure levels of chemicals. Yet governments, industry, and legal system 'experts' cling to the old school theories and paradigms where there were no health effects below a certain dose/exposure. This is now far from the case with many recently tested chemicals (Lagarde 2015, Gore 2015).

Doctors in psychiatry have quite a unique position. Their diagnosis is subjective, requires no proof, cannot be disproven, and does not need to fit factual observations. They can attest to the implausibility of the MCS condition without having other medical qualifications, or extensively observed and studied the condition in the affected person or their family. Unfortunately, this path can be self-reinforcing due to the subsequently prescribed psychotropic medications making MCS sufferers worse in their neurological and general condition (Donohoe 2008p28). The very act of prescribing xenochemicals, in the form of psychological drugs, to an MCS sufferer, indicates a paradoxical disbelief of the condition.

This section has highlighted that the orthodox medical system and health authority demand for proof that the MCS condition is real is in itself unnecessary. The basic scientific approach to fully observe is not used by doctors and health professionals. Doctors are simply expected to fit standardised diagnosis criteria to symptoms, so in the case of MCS, without any standardised criteria, it can only be 'in one's head', and treated psychologically. This then becomes a double standard in that a psychological diagnosis is also not provable. Respect for what the patient is saying as to reactions from environmental exposures is usually not present in orthodox medicine.

EVIDENCE BASED MEDICINE

This section highlights that the medical system's basis for establishing diagnosis guidelines, evidence based medicine (EBM), has stifled the ability of doctors to apply their own observation and experience. Furthermore, EBM is readily corruptible by industry.

The latest trend in medicine for standardising diagnoses is to base it on EBM. In the search for the best available evidence, randomised control trials have become the benchmark, producing statistical results on large samples where results are compared to control groups. Statistical

evidence is now part of doctor training rather than reliance of observations in clinical practice (Ecks 2008). Rather than rely on intuition and personal experience it is now becoming expected the doctor must reason based on the results of clinical research (Phillips 2015, Willis 2003). Furthermore doctors are directed to use clinical practice guidelines: consensus documents presented as best-practice standards. In the case of MCS, it is seldom that such guidelines are released either due to the condition simply not being recognised or through lack of consensus.

EBM is not without its shortcomings. Consider for example studies focussing on the olfactory system (sense of smell) as a mechanism for MCS (Phillips 2015): One school of thought concludes from such trials that the MCS sufferer smells chemicals before experiencing symptoms (inferring a psychosomatic condition) (Van Diest 2006, Winters 2003), yet the other school of thought infers an organic reaction between the olfactory system and the brain (Bell 1992, Pall 2007, Ashford 1991). So EBM has an interpretive dimension. Furthermore as less research occurs on an emerging condition, EBM favours established conditions (Ecks 2008). If more research has been carried out concluding MCS's psychosomatic attribution then the summarised findings on the EBM database, or a meta-analysis, will favour that conclusion. It is pertinent to note that most scientists seem to adapt or modify their research to suit their funding condition (Laudel 2006) so the chemical industry can easily influence EBM. This is discussed further in the chapter on ignorance where undone science is discussed. Furthermore on MCS it has been observed that "the only diversity of opinion comes from the authors with industry affiliations" (Bero 1998p1141)

While EBM at first appears quite a logical tool, it is open to manipulation by industry. It has been adopted as a basis of approach by the medical profession to standardise treatments of established health conditions. Yet the emergent conditions and underfunded areas of medical research can be overlooked in applying this general approach.

This section has highlighted how EBM has stifled the ability of doctors to apply their own observation and experience. As EBM is corruptible by industry it is concerning due to its use by the medical system for the basis of establishing diagnosis guidelines.

RECOGNISING THE KUHNIAN CRISIS

The previous sections have illustrated that the orthodox medical system has paradigmatic belief systems that treat environmental health effects producing conditions such as MCS as anomalies. This section discusses the overall situation from a philosophical viewpoint, showing that there is no fundamental belief system crisis occurring due to the orthodox system's ability to slot, or dismiss such sufferers as psychosomatic.

The historian of science Thomas Kuhn proposed that a paradigm crisis can occur when many observations/occurrences conflict with the current paradigm. In some cases, a 'scientific revolution' can lead to the end of the current paradigm.

To most doctors there is no Kuhnian crisis in relation to MCS and related conditions. There is no need to question their training or knowledge. MCS can be attributed to odour aversion or other subjective psychiatric conditions.

The medical profession "fosters belief in the superior effectiveness" of their treatments (Phillips 2010p1031; see also, Kroll-Smith 1997, Niemeyer 1991). This is combined with medical students being taught 'differential diagnosis' where the patient's signs, symptoms and any raw data are used to work out all that could possibly be affecting the patient: they then are required to isolate the probable 'cause' of the condition (Clarke 2003, Pall 2007). This effectively develops a collective defence mechanism for the perils of uncertainties they confront. This becomes an ideal situation for maintaining a professional paradigm as this then enables the doctors to avoid dealing with uncertainties. By fitting MCS into an existing framework that they work in, no uncertainty occurs to them regarding the paradigm they work in, hence psychosomatic diagnoses are preferred, rather than acknowledging a problem that biomedical science may not have a solution to. So to these professionals there is no Kuhnian crisis: no need to question their training based in paradigm fundamentals. Having established their careers in a set of fundamentals, they have a personal and collective interest in protecting their paradigm.

The extent to which a doctor applies an orthodox line of reasoning to an emergent condition will determine whether they become a sceptic of MCS diagnoses or a sympathiser.

The sceptic's positivist requirements will demand clear pathology for mainstream conformance as well as their own standard of proof. As in all scientific controversies, it is common for experts

to claim their approach is more scientific: more logical, more objective and closer to pure fact. This “affords an impregnable position from which to snipe at the enemy” (Barnes 2009p166). These doctors will not focus on their profession’s lack of knowledge on low level chemical exposures, but rather focus on clear-cut areas of advancement, e.g., neural imaging advancements, successful drugs, etc.

The sceptical doctors also reference ‘weight of evidence’ and ‘scientific consensus’ to circularly reinforce their paradigm. So an emergent health condition is pushed aside until this scientific precipice is considered to be reached. As already commented, there are many acknowledged health conditions, documented for decades in medical journals, such as bad backs, which do not have clear pathology. Added to this, are the pharmaceutical and chemical industries actively trying to confuse the science on MCS recognition, just as the tobacco industry attempted to confound recognition of the health effects from smoking elaborated in the chapter on ignorance.

Many studies on doctor and patient communication have been performed (Deledda 2013) and prominent in these are the importance of listening to the needs and preferences of the patient, and avoiding unjustified psychological explanation. However with the absence of training for doctors in the effects of low level chemical exposures, and the MCS condition not yet fully recognised, the affected patients commonly experience dissatisfaction interfacing with doctors (Crumpler 2014chap6). In all too many cases, a parent has seen cause and effect situations with common everyday chemical exposure, e.g., with deodorisers, pesticide use, etc., and the doctor seemingly dismisses the importance of such reports. Some doctors may even accuse the parents of making fictitious claims as mentioned before: Munchausen Syndrome by Proxy (Onconurse 2016, Haywood-Brown 1999). The use of convenient psychological diagnoses such as this appears to have been used in an apparent retaliatory approach by some doctors when mothers of young patients have questioned diagnoses or made a complaint (Haywood-Brown 1999). There are many variations and degrees of such behaviour by sceptical doctors that tend to be more vocal/questioning. Nowhere in the Hippocratic oath is the requirement that service and respect must be only to those with structured pathology.

The sympathetic doctor is more ready to rely on experiential knowledge. This may come from their experience with patients through observation, consistencies and logical reasoning. The

patient's story and experience with their health condition are respected. The doctor doesn't try to fit a subjective psychosomatic condition to the patient, instead remaining open to different possible understandings of the problem. If they are questioned about the MCS mechanism they are likely to reply along the lines of "medicine doesn't know" illustrating their own acceptance of questioning of the current paradigm. In respect to doubts on the MCS condition, it would be one of doubting the medical system's understanding of health effects from low level exposures and the level of proof required for an emerging condition rather than one of doubting the patient (Phillips 2015).

Each side thinks the other is not being scientific or logical. Scientific reasoning by each side comes to different conclusions. The sceptics remain undeterred. The sympathisers become disillusioned with their profession's attitudes and lack of understanding. We are therefore far away from a Kuhnian crisis when considering the situation with MCS.

ATTEMPTS FOR MCS TO BE RECOGNISED AS A DISABILITY

It is interesting to consider the economic costs of recognition of the MCS condition. The widespread acknowledgement and accommodation of the condition would be revolutionary due to the multitude of everyday products that trigger its adverse effects. The economic cost would be very significant which explains why the chemical and communications industries actively try to downplay and confound the science on the condition. It would be far more economical to simply recognise the condition as a disability, although this then presents compensation aspects. But to treat and compensate a condition that 'may be' psychosomatic is politically and medically troublesome. The *Medical Journal of Australia* published a letter which called on doctors to resist calls from MCS and CFS patients for disability pensions (MJA 1991). One survey of doctor attitudes found that half of them indicated that they were unlikely to accept a patient applying for worker's compensation on the basis of having MCS (Gibson 2011). So while the sceptical decision-makers wait for the moment of undeniable fact, a public health crisis steadily grows.

BIAS IN THE RECOGNITION OF MCS AND THE INFLUENCING ROLE OF INDUSTRY

This section highlights that the various difficulties previously covered in recognising MCS are not simply due to the orthodox medical system's lack of knowledge. Ignorance is actively manufactured by industry in a deliberate attempt to ensure MCS is not recognised.

Industry backed front groups are actively trying to hinder recognition, confuse, and create ignorance on MCS. This section uses some examples to illustrate this as well as show how the 'science' can be distorted on the subject.

If low level exposure to chemicals used in commercial, farming, household products, foods, drugs, vaccines, etc. have a recognised effect on a significant percentage of the population then this has major ramifications commercially to billion dollar industries that manufacture such products. Worse still, if the mostly non-sensitive portion of the population recognise the sensitive people as being an indicator of toxicities, more of the population may want to avoid such potential exposures.

The Chemical Manufacturers Association's Environmental Illness Briefing Paper claimed in 1990 "The primary impact on society would be the huge cost associated with legitimisation of environmental illness" (CMA 1990). This Association (now the American Chemistry Council (ACC)), decided to act to prevent the recognition of MCS due to potential lost profits and liability concerns if MCS became recognised (McC Campbell 2001). This Association then proceeded at the same meeting, to plan how they would influence society's views on MCS.

Vakas (2007) investigated the controversy over the possibility of fumes causing health problems in crews of the BAe 146 aircraft. The study highlighted the contradiction of societies that require a healthy workforce to generate capital yet in the process generates toxic chemicals which damage health (Kroll 1993p24-25). The cabin fume example will be utilised later in this chapter in observing an outspoken critic of MCS recognition.

Vakas outlines the Chemical Manufacturer's Association (CMA), now the American Chemistry Council, and its efforts to undermine any legitimacy of MCS as an illness or disease such as in its briefing papers (CMA 1990). The organisation advocated:

- monitoring of the situation
- the identification of medical personnel who can speak as experts,
- guidance material for reporters
- establishment of organisations, such as the Environmental Sensitivities Research Institute (ESRI), Quackwatch, etc., to confound the science and public understanding
- formation of a coalition with various other industry sectors
- influencing international bodies such as the International Program on Chemical Safety (IPCS)

It is pertinent that at the IPCS workshop in 1996 covering MCS, dominated by industry affiliated representatives defined it by a new name, of Idiopathic Environmental Intolerances (IEI). This term utilising 'idiopathic', places the illness outside models seeking a chemical or environmental causation. This shifted the focus on to the individual for cause and treatment. The pronouncement by an international organisation, particularly one internationally recognised, imparts legitimacy via a perceived objectivity.

The IPCS was established in 1980 by the WHO and is sponsored by WHO, International Labour Organisation (ILO) and the United Nations Environment Programme (UNEP). The association with these bodies contributes to significant credibility.

Eighty one scientists, including the IPCS workshop chair, Dr Howard Kipen, were signatories to a published letter "criticising the IPCS and this workshop in particular for its chemical industry bias" (Donnay 1998).

Concerns about industry dominance compromising the integrity of the IPCS were raised as early as 1993 (Abrams 1996). The US National Institute for Occupational Safety and Health (NIOSH) decided to cease all collaboration with the IPCS until its process became more "objective". The responses from the IPCS asserted objectivity and a thorough review of scientific data but used generalisations to avoid addressing the many criticisms raised. The IPCS's terms such as 'sound science' that was utilised, conveyed a perception of detached evaluation but did not adequately address the politics involved in research direction, evaluation and conclusions.

Following criticism of the MCS workshop the IPCS issued disclaimers, one of which was:

...the document does not represent the decisions or stated policy of UNEP, ILO, or WHO; that it does not constitute a formal publication; and that it should not be reviewed, abstracted, or quoted without written permission (Vakas 2007p194).

Yet a subsequent industry sponsored and dominated conference (a quite separate one) summary paper was placed as an anonymous paper at the end of the conference papers outlining the 'consensus' achieved. As well as being very negative toward MCS, it also termed MCS as idiopathic environmental intolerance syndrome. The conference's publications, as well as this anonymous paper, appeared in the journal *Regulatory Toxicology and Pharmacology*. This was a supposedly peer-reviewed journal. From here on, industry sponsored articles on MCS referenced what appeared as the IPCS 'consensus' and implied WHO, ILO and UNEP endorsement.

The American Academy of Allergy and Immunology (AAAAI) heralded IEI as the new name for MCS without acknowledging the resulting controversy and implied WHO endorsement. The Australian equivalent, the Australian Society of Clinical Immunology and Allergy uses the IEI term and centres on psychological explanations. It references industry backed papers and even the Quackwatch website (ASCIA 2017).

There are many industry backed experts posing as independent in corporate front groups such as the American Council on Science and Health (ACSH) (Beder 2000p28). As can typically happen with 'experts' in such organisations, generalised unsubstantiated conclusions are frequently published in favour of industry, e.g., Gilbert Ross, while a medical doctor with the ACSH, published that arsenic in pressure treated wood poses "no risk to human health" and PCBs in fish "are not a cause of any health risk, including cancer" (Hogan 2005). Dr Ross had quite a colourful history in medical fraud and was once described by a judge as 'an untrustworthy individual'. He later became Medical Director/Executive Director of the ACSH (Hogan 2005).

Another of the many organisations formed with industry backing in the US was the National Council Against Health Fraud. The main aim of this organisation was to take legal action against non-orthodox medical views or those at odds with industry positions and to generally intimidate those expressing the alternative views. The legal grounds pursued are for nuisance, conspiracy, defamation, invasion of privacy and interference with business or economic

expectancy. Used since the 1970's these sorts of actions have become known as Strategic Lawsuits Against Public Participation (SLAPP). The main instigator of such approaches in the US, Dr Barrett, also has associations with the American Medical Association to discredit the chiropractic profession (Barrett 2002).

The winning of SLAPPs is not important, and in fact 77% of the cases are won by those being sued. Their real success is to scare people off by threat of a legal action. Those involved in a SLAPP pay in time and money and removes their attention from the original controversy. Furthermore, debate is then drawn to the legal arena and away from the public (Beder 2000p64-66).

The ACSH scientific adviser, Dr R Gots, president of the International Centre for Toxicology and Medicine in Rockville, Maryland, founded and was director of the Medical Claims Review Services (MCRS). This company specialised in providing paper reviews of insurance claim cases for insurance companies, especially State Farm Insurance, the largest insurance company in the US. It reviewed patient claims and medical reports, without any contact with the patient, and provided explanations of causation (Larson 2000). In John Larson's NBC Dateline report, serious problems were found with such reports, including doctors not authoring, or having their signatures forged. They were being used to deny medical claims. The investigation illustrated the extent that industry-paid advocates are prepared to go and are analogous to the amicus briefs mentioned below.

One of the interviewees in Larson's investigation was J Mathus, a former superintendent for State Farm who said "...it is decisively and deliberately orchestrated" and "there's only one motivation for using a peer review. And that's to increase profits by reducing costs" (Larson 2000).

In a court case by an insurance claimant previously denied adequate compensation, the judge referred to MCRS as a "completely bogus operation" and that the insurance company knew the reports "were not objective, but slanted to favour the denial or reduction of claims" (Larson 2000)

In a subsequent interview with Dr Gots, he claimed his company was "above standards in industry by far" and that other such companies "don't even have doctors. They have all nurses

or clerical people to do the reviews” (Larson 2000). There seemed no concern for those injured and the wider public interest. His focus was on his individual revenue from the corporations he worked for. This is a prime example of using the language of science and corporate financial reward to shift blame onto the sufferers.

The American Chemical Council (ACC) seeks to influence physicians, medical associations and government departments. Physicians paid by the industry become vocal in promoting

that people with MCS are ‘hypochondriacs’ ‘hysterical,’ ‘neurotic,’ suffer from some other psychiatric disorder, belong to a ‘cult,’ or just complain too much. Most of these physicians are high-paid expert witnesses although their financial ties are usually not disclosed in their journal articles, interviews, or speaking engagements. Therefore, many people, including those in the health care profession, are often led to believe that these physicians’ opinions reflect an honest appraisal of MCS rather than the chemical industry’s agenda (McC Campbell 2001p1).

With billions of dollars at stake, the ACC has plenty to spend on having many of such physicians throughout most countries.

With the top ten prescription drugs being for diseases that ecologically aware physicians recognise as having an environmental aetiology (Crumpler 2014), direct influence on physicians is also advantageous to the pharmaceutical industry.

Physicians have regular contact with the pharmaceutical industry and its sales representatives, who spend a large sum of money each year promoting to them by way of gifts, free meals, travel subsidies, sponsored teachings, and symposia....The present extent of physician-industry interactions appears to affect prescribing and professional behaviour and should be further addressed... (Wazana 2000p373).

At a medical conference of the American College of Allergy and Immunology in 1990, Sandoz (now Novartis) was sponsoring a workshop that was to attribute MCS to mental illness (McC Campbell 2001, ACAI 1990). Sandoz was a large manufacturer of both pesticides and pharmaceuticals, including anti-depressants, sedative medications and anti-psychotics (McC Campbell 2001).

Sandoz stood to benefit both from pesticides being exonerated as the cause of MCS and from people with MCS being treated with psychiatric drugs. As it turned out, people with MCS – outraged by the workshop – risked their health to protest the event and were able to shut it down” (McC Campbell 2001p4; see also Hileman 1991).

This was a learning curve for the chemical industry that would make such conference influences much less prominent in the future.

Industry-backed physicians and representatives on research funding committees have influenced the blocking of research into MCS (McC Campbell 2001). An editorial in the *New England Journal of Medicine* stated: (Angell 2000p1516):

The ties between clinical researchers and industry include not only grant support, but also a host of other financial arrangements. Researchers serve as consultants to companies whose products they are studying, join advisory boards and speakers’ bureaus, enter into patent and royalty arrangements, agree to be the listed authors of articles ghost written by interested companies, promote drugs and devices at company-sponsored symposiums, and allow themselves to be supplied with expensive gifts and trips to luxurious settings (Angell 2000p1516).

With research funding tied-up by the chemical/pharmaceutical industry it is little wonder why there is a large deficit in impartial research into MCS. There is much discussion in the literature about research being influenced, such as:

Analysis of more than 1100 clinical studies by researchers at Yale Uni revealed that when academics had backing from industry, 80% reached pro-industry conclusions (Crumpler 2014p148).

There is “... a statistically significant association between industry sponsorship and pro-industry conclusions...odds ratio, 3.60” (Bekelman 2003p454). An odds ratio is a measure of association between an outcome and an exposure. It represents “the odds that an outcome will occur given a particular exposure, compared to the odds of the outcome occurring in the absence of that exposure” (Szumilas 2015p227).

In respect to fraud in published papers:

The authors concluded that their study provided firm evidence to prove their 'deliberate fraud' hypothesis. Papers retracted because of data fabrication or falsification represent a calculated effort to deceive. Such behaviour is neither 'naive, reckless nor inadvertent'; the perpetrators did so on purpose (Van der Wall 2012p49).

Dr. Marcia Angell, a physician and former Editor in Chief of the *New England Journal of Medicine* (NEJM):

It is simply no longer possible to believe much of the clinical research that is published, or to rely on the judgment of trusted physicians or authoritative medical guidelines. I take no pleasure in this conclusion, which I reached slowly and reluctantly over my two decades as an editor of the *New England Journal of Medicine* (Angell 2009p9).

A further influence on Journals is the amount of advertising as well as the huge return from reprint purchases from the Big Pharma companies. Furthermore, as pharmaceutical companies typically orchestrate a large number of ghost-written secondary publications that cite published trial reports, the journal's Impact Factor can also be enhanced. Such influence is to an extent that articles are sometimes refused to be published if counter to Big Pharma interests. The *British Medical Journal's* former editor, Richard Smith, wrote a paper titled "Medical Journals are an extension of the marketing arm of pharmaceutical companies" (Smith 2005p364). Another author wrote, "The practice of buying editorials reflects the growing influence of the pharmaceutical industry on medical care" (Brennan 1994p673).

With the most-prominent medical journals being influenced so much by the chemical/ pharmaceutical industry it is little wonder that genuine research papers which are counter to the generated belief system rarely can get past either the editors or the peer-review panels of such journals. Unfortunately when a researcher publicly recognises physiological aspects of MCS there are many cases through the years where they find themselves with research resources pulled from their reach, career opportunities taken away, their funding/employment terminated or made such that they resign (Phillips 2015, Crumpler 2014) . Even when an independent research company makes an inconvenient finding for industry in relation to toxicity of products, such as when Anderson Laboratories found that lab mice were dying from exposures to new carpets (Anderson 1995), one multinational chemical company hired public relations companies to "erode the credibility of the Anderson Study" (Duehring 1994p45)

In the cases of physicians who have spoken up or supported MCS sufferers there has been the threat of deregistration, e.g., the conviction of Dr J Krop by the Ontario College of Physicians and Surgeons after he diagnosed MCS in a patient (Ferrie 1999)

Another alarming trend is the number of Big Pharma initiated/indirectly-owned/funded journals and organisations. Some of these have innocent names such as Environmental Sensitivities Research Institute (ESRI) (no longer functioning under this name): ESRI was founded “to serve the needs of industries affected by MCS litigation” (McC Campbell 2001, ESRI 2000). Its main work is to disseminate anti-MCS literature, hold anti-MCS conferences, to intervene in government and legal affairs, and to impede MCS progress (McC Campbell 2001). There are many other seemingly independent organisations that are funded by, and speak with the same voice as, big pharma. Some such organisations are under the innocent guise of supporting ‘science’ in society yet commonly exhibit an absence of science in their public attacks on researchers and sufferer support organisations.

It is a long path for the recognition of MCS as a legitimate health condition, let alone publication of the little research done and the mechanism established. This has been due to a concerted effort by industry to prevent MCS physiological research so that there is little substantive research that can be found simply because little has been done. Such an ‘undone science’ situation is exploited frequently by industry representatives to argue that MCS existence is “controversial”. It is to such an extent that journalists covering the area mostly feel obliged to present “both sides”.

As seen above, there are many medical professional paradigms blocking recognition of MCS. These professional paradigms complement the above ‘undone science’ situation. Medical professionals have established their careers on orthodox paradigms and have a personal and collective interest in protecting their paradigm.

As has been seen from the above examples, industry influence preventing the recognition of MCS is beyond being suspected. Industry backed front groups are actively trying to hinder recognition, confuse, and create ignorance on MCS. This section has therefore illustrated that the orthodox medical system’s ignorance on environmental effects and MCS in particular, is added-to with industry manufactured ignorance.

THE APPLICABILITY AND COMPARISON OF PSYCHOLOGY AND PSYCHIATRY TO MCS

With psychosomatic diagnoses being the way orthodox medicine is dismissing or treating MCS, it is worthwhile to consider the logic and lack of scientific approach in taking this line.

A memorable example illustrates the situation:

“Myra reported to her doctor that she frequently collapsed while idling the car at traffic lights: she was promptly referred to a psychiatrist. The solution was of course obvious: a red light connotes sex” (Crumpler 2014p241).

This example should rightly be regarded as ridiculous and extreme. Yet frequently in orthodox medicine, it is decided that a patient with symptoms characteristic of MCS has a psychological problem.

In trying to more seriously consider such sufferers who attribute a myriad of health problems to very low chemical exposures that seemingly do not affect most people, there are a variety of studies that have been done by the orthodox researchers. The Modern Health Worries Scale studies concerns about tainted food, environmental pollution, radiation, and toxic interventions. In studying MCS sufferers Bailer (2008b) found higher worry scores than controls. It was then concluded that chemical sensitivity was an attribution style of belief. An alternative conclusion would be that MCS sufferers are rightfully worried about potential sources of their conditions.

The orthodox doctors attribute the condition as psychological due to the absence of signs of pathology. But there are no such signs for psychogenic diagnoses. In the many studies in the psychogenic origins of MCS there is frequently a disregard for environmental exposure origins. As a result, “studies investigating psychogenic hypotheses of MCS are methodologically problematic and their conclusions questionable” (Davidoff 1994p317).

Since MCS, and many of the overlapping conditions such as EHS, are normally treated psychologically by the orthodox medical approach, a closer look at the situation with diagnosis and treatment of psychological conditions is warranted. Depression is an example of a common

psychological condition attributed to MCS sufferers. There is evidence of a lack of science behind depression diagnosis and a strong relationship with environmental exposures. Another well-recognised psychological diagnosis is Attention Deficit Hyperactivity Disorder (ADHD) with a similar percentage of the population affected as MCS. This condition is substantiated by fewer scientific studies than for MCS yet MCS is not recognised, while ADHD is widely accepted.

LEGAL CASES

This section is included since it adds indirectly to the non-recognition of the MCS condition due to the general absence of legal substantiation. The section will highlight once again, the influence of industry. In a legal sense, the sufferer of MCS must clearly prove direct causation from their identifiable exposure, through established and 'accepted' scientific studies. As seen from the previous sections this becomes almost impossible for a 'lay person'. It will also be seen that the use of 'expert' witnesses does not provide the plaintiff a clear advantage. A legal pursuit for justice is usually not an avenue for an MCS sufferer.

It is pertinent in the light of so much evidence referenced on the MCS condition's existence as well as the many references throughout this thesis on health effects from low level xenochemical exposures, to question why the legal system hasn't assisted in MCS recognition and its debilitating consequences. The legal system has seen many cases of plaintiffs with MCS trying to obtain recognition and compensation for their condition. Many more cases never make the court room due to legal costs and advice of little chance of success. So here too, the individual sufferers, just like the low dose chemical exposures, are dismissed one by one in isolation. If one considers the enormity of the problem with the numbers that have suffered directly and indirectly, a quite different picture would be seen.

In looking into this area one finds that industry groups have again been organised in influencing the judicial system, especially in the US. An analysis by Edmond and Mercer (2004p231) had made conclusions:

It is our contention that recent federal jurisprudence, including Supreme Court jurisprudence, seems to have been shaped, and simultaneously reinforced, by a range

of values which appear closely aligned to the perspectives and concerns promoted by politically conservative corporate-sponsored proponents of tort and evidence reform. ...corporate-sponsored polemicists have been more influential, as sources of authority, about the nature of science and expertise, than specialist philosophers and social scientists.

Two industry-sponsored organisations, the Atlantic Legal Foundation (ALF) and the Manhattan Institute were described by Edmond and Mercer as being very dominant. Both these organisations have openly claimed to have influenced evidence jurisprudence in the US.

The ALF has been very active in the use of an amicus curiae brief (a professional person or organization that is not a party to a particular litigation but is permitted by the court to advise it in respect to some matter of law that directly affects the case in question (Merriam Webster Dictionary)) to attack the credibility of scientific claims (Galbato 1998p274, Edmond 2004).

The Manhattan Institute has promoted its senior fellow, Peter Huber, who has popularised the concept of 'junk science' in his articles and books. They hosted numerous judicial forums and dinners around the US for both state and federal court judges to present their point of view. The term 'junk science' and his books 'Galileo's Revenge: Keeping junk science out of the courtroom' and 'Judging Science' have been widely cited by judges and the media. Claims were presented such as that paranoia based on ignorance is due to concerns with technological risk and uncertainties of new technologies. Such simplistic views seem to have been the reason for its seemingly popular uptake.

Huber takes aim at many contested illnesses. One of his common strategies is to use history for justification. In the case of MCS, this includes Hippocrates' use of the term 'hysteria' for a range of ill-defined medical complaints and his 1881 article on neurasthenia being a nervous condition with multiple symptoms (Huber 1991p107-108).

The US Community Rights Council expressed its concern about these sponsored judicial education programs due to influencing "corporate friendly" views of public health, risk, and environmental litigation (Edmond 2004, Kendall 2000, Miltenberg 2001).

The Daubert Ruling

The use of the ALF's amicus curiae briefs as effective tools in court cases has been enabled by the US Supreme Court Ruling in the 1993 case of Daubert vs Merrill Dow Pharmaceuticals. This ruling was for determining whether the intended evidence is 'scientific knowledge' and would assist to establish facts for the jury. The four requirements have become generally known as the 'Daubert criteria' as presented by Edmond and Mercer (2004p234):

"[1] Ordinarily, a key question to be answered in determining whether a theory of technique is scientific knowledge that will assist the trier of fact will be whether it can be (and has been) tested."

"[2] Another pertinent consideration is whether the theory or technique has been subjected to peer review and publication. Publication (which is but one element of peer review) is not a sine qua non of admissibility; it does not necessarily correlate with reliability."

"[3] Additionally, in the case of a particular scientific technique, the court ordinarily should consider the known or potential rate of error."

"[4] Finally, "general acceptance" can yet have a bearing on the inquiry." (Daubert 1993 at 593-4)"

After the Daubert Ruling, evidence that the courts had become more determined to exclude plaintiffs' evidence occurred in the Joiner, Kumho and Weisgram rulings (Edmond 2002b p399). Such judicial gatekeeping had been described as a win for defendants (Cranor 2001p6, Gottesman 1998p766). Many commentators have described the Daubert ruling triumphing over junk science.

Consequences of the Daubert Ruling

This is best illustrated through examples of its effects, combined with the industry's judicial influence mentioned before. The below examples highlight a simplistic, ignorant view of science disregarding the political and sociological issues involved in science covered by many articles and books over the decades and in this thesis.

Electromagnetic fields

Newman v Motorola Inc (2002,2003) was a case where the claimant was attributing his brain tumour behind his right ear to being caused by his mobile phone usage. The judge excluded Newman's expert testimony. A weight of evidence basis was applied where the clear majority of published epidemiological studies found no links with brain cancer. No awareness that these studies were industry funded/influenced was evident. Animal studies were also discounted as not being able to be applied conclusively to humans. Although the judge acknowledged that the expert had relevant qualifications his evidence was ruled as not satisfying the Daubert standards. The expert was Lennard Hardell, Prof of Oncology at the University Hospital Orebro, Sweden and had published many studies on cancer epidemiology.

The above judge also suggested that Hardell's work did not have proven replication due to other studies referenced did not show increased risk. However, the interpretation and meaning of experimental replication has been shown to be controversial and negotiable by past sociologists (Collins 1985). The judge also surprisingly passed comment that two of his recent papers had not been accepted by the more prestigious journals.

Benzene Exposure

The Parker v Mobil Oil (2006) case involved a petrol station attendant who had been exposed to petrol fumes and developed cancer attributed to such fumes. The judge passed comment that the lack of any confirmatory disease causation studies by the petrol supply or petrol station companies was indicative that there was no disease causation. Furthermore, that the plaintiffs did not have any 'epidemiological-statistical evidence' and failed to rule out other exposures or lifestyle factors (no differential diagnosis). The judge also noted that cancer was common in the population and that "all of us are exposed to substances known to be cancer risk factors, without becoming ill and that multiple cancers cannot be attributed to one causal agent". This contradicted the panel present in the multiple DuPont C8 cases (Mordock 2016), the scientific literature (Vlaanderen 2011, Smith 2007), and subjectively discounted workplace risks adding to a cumulative risk (Rodericks 1992).

In another case, Milward v. Acuity Specialty Products Group, Inc. (2011) where Milward sought redress for his Acute Promyelocytic Leukemia (APL), Billauer (2017p11) illustrated how a differential diagnosis was ruled out by the judge:

After the expert witness had eliminated the possibility of being caused by obesity or smoking, she then 'ruled-out' idiopathic causes (diagnoses without a known cause), and as the benzene exposure was the only significant cause remaining she concluded that it was the culprit. This was rejected by the judge due to no scientifically reliable method being provided to accept benzene as the cause. By using the US Torts (2010) regulations: {The underlying premise [of differential etiology] is that each of the known causes is independently responsible for some proportion of the disease in each population. [However,] When the causes of a disease are largely unknown . . . differential etiology is of little assistance (Billauer 2017p11note 61)}, the judge claimed the reasoning was circular: idiopathic APL was ruled out by 'ruling-in' benzene without scientific justification. Even if smoking and obesity could be eliminated as causes, the judge said that differential diagnosis provided little help since there is a high percentage of APL diagnoses without identifiable causes.

In some interesting statistics by Perlin (2017p4):

In sixty-seven cases of challenged government expertise, the prosecution prevailed in sixty-one of these. Out of fifty-four complaints by criminal defendants that their expertise was improperly excluded, the defendant lost in forty-four of these. Contrarily, in civil cases, ninety percent of Daubert appeals were by the defendants, who prevailed two-thirds of the time.

After a survey of about 400 pre- and post-Daubert US federal cases (1980-1999) involving expert evidence it was found that the Daubert general acceptance became a barrier to admission of cases (Dixon 2001). It was also found that the judges added further admissibility criteria including consideration of theories, methods and the experts' qualifications (Bernstein 1996,2001, Dixon 2001). This is further extended by the Edmond and Mercer (2004p244) prediction that the Daubert aspects of falsification, error rates and peer review may become "simplified and legally tractable folk epistemologies" producing importance of testing and mainstream science based on "legal rather than formal philosophical authority" (Edmond 2002a).

Despite applying strict requirements to evidence in toxic tort cases the lack of requirements applied to forensic expert witnesses (not reviewed or tested) presents quite an inconsistency

(Nordberg 2003). A further inconsistency is that the US fifth circuit courts recognise 'environmental illness' as a disability under the Social Security Act. 'When causation is not in controversy, scientific validity under Daubert will not necessarily block the admissibility of the MCS testimony' (Galbato 1998p295)

This section has highlighted once again, the influence of industry, on the legal system. In a legal sense, the sufferer of MCS must clearly prove direct causation from their identifiable exposure, through established and 'accepted' scientific studies. As seen from the previous sections this becomes almost impossible. This is primarily due to the amount of industry backed studies that confound the science. The use of 'expert' witnesses also does not provide the plaintiff a clear advantage. A legal pursuit for justice is usually not an avenue for an MCS sufferer. In the next section, a prime example will be considered to illustrate the situation sufferers face.

The Australian Workers Compensation Case and Senate Hearing on Aircraft Cabin Fumes

It is pertinent to consider an example where health effects from low-level chemical exposure has been argued in a worker compensation case in Australia. It shows how people with health effects from low-level chemical exposures are handled and dismissed by 'authorities' in the situation with aircraft cabin fumes. It highlights how passengers and crew suffering health effects from cabin fumes have been dismissed through personal attacks on plaintiffs and their expert witnesses, 'weight of evidence' arguments, orchestrated consensus statements, and industry skills. It shows the difficulties faced in recognising varied health effects from low-level exposures well below workplace acceptable levels and how industry avoids the use of scientific enquiry and study of the actual situation. It also represents an example where an MCS condition is initiated in previously healthy people.

From about 1955, civilian aircraft started to bleed unfiltered air from the engine compressors into the cabin. The cabin air consists of a 50/50 mix of recirculated cabin air and bleed air. From the various reports from crews it was soon realised that engine oil, hydraulic and de-icing fluids could leak into the compressor air. The synthetic engine oils include a wide variety of triaryl phosphates, organophosphates (usually tricresyl phosphate), amine antioxidants plus 'proprietary ingredients' which are unknown. Hydraulic fluids can be tributyl and triphenyl phosphates and de-icing fluids are ethylene and propyl glycols (Michaelis 2017, de Boer 2015). Through the high temperatures in the engines these with the other ingredients can be pyrolysed into many toxic substances, although many are very toxic before this.

There have been many cases internationally concerning health effects from aircraft cabin fumes. Many cabin crew have been unable to continue flying due to the health effects which include MCS initiation. Strategies in the courts have been similar to those in the US as mentioned previous where the causation is shifted from the industry interests to the individual.

A typical case was that of flight attendant on BAe146 aircraft, Alysia Chew (1995). One of the experts put forward by the defence was an organic chemist, Dr Crank. A chemist, not a doctor/scientist experienced in studying health effects from chemical exposures. He noted that the Mobil Jet Oil II that had leaked into the cabin contained tri-ortho-cresyl-phosphate (TOCP). It was argued with unqualified certainty that the concentration of this ingredient was very low and that there was no toxicity due to the 60% fresh air circulation rate. The judge accepted Crank's opinion and dismissed Chew's expert witness, Dr Winder, since he did not consider the amount and extent of the exposure and had 'predetermined that there had been harmful exposure'. Crank's unqualified safety of exposure was not in question. Later in a government committee in 2000, van Netten pointed out that the exact composition of the jet oil was unknown by Mobil (SRRACRC 2000p37).

Another defence expert witness, Dr J Lee, thoracic physician, stated that he did not recognise MCS as a medical illness. Another expert, Dr P Carroll specialist physician, also added "where non clinicians support the claim they do so in contradiction to all published literature by scientific organisations" (Chew 1995p9). This ignores the extensive scientific literature since the 1980s where many clinicians have debated the symptoms and causation of MCS. It implies that these doctors do not recognise even the physical condition and the use of 'proper' science would simply make the problem disappear. This would deny the sufferers of both a diagnosis and their experience of all their health problems.

A consensus statement from the Ansett Odour Committee was submitted to the court. It outlined general dismissal that any measured contaminants could cause any threats to health, the levels were below occupational health standards, and the standard smoke removal procedures provided a large margin of safety (Chew 1995p8-9). This committee had been dominated by industry and failed to mention that employee and union members of this committee refused to sign the statement.

In Australia, the Director of the Allergy Unit at the Royal Prince Alfred Hospital and Senior Lecturer in Immunology, Department of Medicine, University of Sydney, Dr R Loblay, serves a gatekeeper role for industry interests. "Through his various professional positions he is able to stymie and marginalise the association of illness, disease and disability from chemical exposure" (Vakas 2007p196). When he appeared he suggested that an "individual's belief about the nature of an odour (e.g., that it is potentially harmful), based on what they are told by an authority figure, can modify their subsequent degree of sensitivity" (Chew 1995p9). He dismissed the MCS label by saying "she has a tendency to food intolerances" and her illness was a "sensory phenomenon, rather than a disease process." It was further added that "clinical ecologists have a vested interest in promoting its continued use" (Chew 1995p10). In also alluding to the name change to IEI per the IPCS workshop he falls into the familiar approach against the plaintiffs: discredit the professionals, suggest a strong psychological component, and establish institutional legitimacy for such views.

In a later forum, Loblay admitted he had no experience with the sufferers that had been made ill on the BAe 146 aircraft (Australia Senate 2000a p101). The lack of knowledge and experience had not stopped his preparedness to testify against them on industry's behalf.

In his appearance at the Senate Inquiry, Loblay again attempted to discredit other professionals. In reference to Dr M Donohoe, perhaps the leading doctor in Australia dealing with MCS sufferers, he said that he "is not a mainstream medical practitioner....his views are not considered to have much scientific validity within the mainstream specialty of medicine. This is common knowledge" (Australia Senate 2000a p101).

Loblay also criticised Dr C Winder and others who had argued about the potential increased toxicity of chemicals at altitude due to the decreased oxygen levels. He generally dismissed such concerns as "arm waving pseudoscience" (Australian Senate 2000a p102). The following day Susan Brookes from the Flight Attendants Association of Australia pointed out that their training manual noted that "alcohol, nicotine and some other drugs" are influenced by lower oxygen availability. She proposed that the possibility of toxicity of other chemicals such as those from the jet oil being affected by oxygen levels appears to be prudent rather than pseudoscience (Australian Senate 2000b p160)

Loblay also criticised Dr R Teo and claimed that his evoked response testing examining altered brain functioning should be “taken with a grain of salt” due to methodological problems. Dr Teo, appearing after Loblay, argued the assertions were incorrect and he was actually ‘lying’. Dr Teo’s experience in the area dated back 20 years, with a PhD, 46 publications and numerous conference presentations on the subject. Loblay had been a co-author of one of his papers in 1993.

Loblay attacked Dr J Ford personally and professionally and had lodged a complaint with Ford’s professional body (Australia Senate 2000b p103). Dr Ford had undertaken genetic testing on 5 cabin crew and had found 3 of them had significant exposure to chemical toxins (Australia Senate 2000b p73-77).

As another target Loblay argued that ‘acute hyperventilation’ can explain the crews’ reported symptoms of “dizziness, inability to think clearly, pins and needles in the arms and legs and around the mouth, blurring of vision and tunnel vision.” Also, hysteria and acute anxiety could better explain the symptom of tunnel vision (Australia Senate 2000b p103). Brendan Treston of the Flight Attendants Association of Australia pointed out that his organisation had over 700 fume reports going back to the 1990s and that hyperventilation does not explain the attendants vomiting blood, oesophagus ulcerations, or becoming stupefied over a considerable time period (Australia Senate 2000c p160)

A “spreading belief system” was the reason Loblay put forward in explaining why the aircraft crew had greater symptoms than the passengers: this being put-forward after claiming that others had no scientific basis (Australia Senate 2000b p103-104). Perhaps this had something to do with the fact that the cabin crew spent a greater time on fume prone aircraft.

The Senate Inquiry report mentioned the underreporting of cabin fume incidents due to fear of reprisal and job security. In a survey of 106 pilots, 93 B757 pilots experienced smoke and fume events so the total number was estimated to be over 1660 yet only 61 were logged as air safety reports (Cox 2002, Michaelis 2003). There had been internal memos circulated to the senior pilots, engineers and attendants to discourage fume incident reports to the authorities (Australian Senate 2000c p230-231). There were also reports of union survey forms being removed from pigeon holes by company representatives (Devine 2000).

Three surveys (Cox 2002, Michaelis 2003, Wright 1999) found aircrews had long and short term health problems. These clearly illustrated the true situation and highlighted the industry denial and Loblay's estranged nature with a scientific approach to this problem in not actually examining any of the sufferers.

As summarised by Vakas (2007), the final Senate Inquiry report observed that Loblay had argued that there were no health effects from such fume exposures. It did note that he did not supply a written submission and his evidence mostly attacked "the personal and professional integrity and status of other witnesses" (SRRATRC 2000p29). It did not note that Loblay had not examined or tested any of the crew members. Rather, Loblay's numerous claims were cited, including those of the crew's 'belief system' in that the problem was more a psychological one (SRRATRC 2000p31-33). With the report not citing Loblay's personal attacks and the contrary replies in some cases, this tends to raise Loblay's standing when reading. This therefore contributed to the report's failure to acknowledge long term health effects and finding danger in flight safety.

It is interesting that the chair of the Senate Enquiry, John Woodley, later published an article (Woodley 2005). In the article he referenced Loblay's evidence as 'dubious in the extreme'. He also noted Loblay's scandalous attacks on other witnesses and the attribution of 'mass hysteria' to explain the crew's health problems. It was also noted that Loblay did not reveal to the committee that he had been witness for insurance companies on many court cases for aircrew compensation.

Loblay's Inconsistencies and Attribution to Individuals

The above section outlined Loblay's poor conduct in the aircraft fumes issue. In making generalised statements about sufferers' health without examining or testing them as he did, it is interesting to note that he had been one of two convenors of a working group to develop CFS clinical practice guidelines in 2002 with funding from the Commonwealth Government (WGRACP 2002). These guidelines implicated chemical exposures and referenced MCS, with others, as possible overlapping/related conditions. It recommended "...a systematic review of the patient's self-reported functional capacity and an assessment of whether this was accurate". Also that "...conclusions about the role of infection, chemical exposure or the emotional demands of the workplace should be appropriately tentative unless the clinical

evidence is clear-cut and compelling” (WGRACP 2002p546-547). Such approaches had not been followed by Loblay in the aircraft fumes cases or the Senate hearing, but rather he slandered the opinion of other doctors that had examined and assessed the sufferers. It also keeps being overlooked that the fumes that the sufferers were exposed to has never been conclusively defined both in chemical types and concentrations especially to a time base.

Loblay actively appears in many forums to critique research linking chemical exposure to CFS or MCS. Many of his comments echo those of the chemical industry or its lobby groups. He has appeared in editorials in journals (Loblay 1995), radio, and made submissions to parliament (Vakas 2007p229). His thrust is on a psychological basis of MCS. In so doing his use of the name IEI rather than MCS also infers it is an individual’s problem. He has used one of his preferences of ‘Intolerance to smells and fumes’ (ISF), again individually oriented. In focusing on the individual attention is removed from environments where the health problems arise, such as the workplace.

Current Situation with Cabin Air Quality

The continual denial of a problem by the airlines and aircraft authorities has prevented official recognition of the problem. The threat of job loss and even legal action against aircrew that report such problems has also helped its suppression. It is now referred to as Aerotoxic Syndrome, Aerotoxicity and various other terms.

The reports and case studies continue to increase. However the number of toxic components that are present in the fumes, the diffuse neurological and other symptoms documented, and the lack of a causal mechanism leave room for much criticism from the industry backed scientists, regarded as ‘debate’ in the public arena (Wolkoff 2016, Harrison 2015, COT 2013). As there is no accepted medical investigation protocol for such incidents, consistent data is difficult to obtain.

Except for the Boeing 787, all current aircraft still use a bleed air system for cabin ventilation. While it has been argued that oil leakage rarely occurs, there is a growing recognition that small amounts of oil continuously leak past engine seals, especially during engine power changes (Michaelis 2016). It is also recognised that cases are underreported (Michaelis 2010, ICAO 2015).

Since the affected crew/passengers can be individually treated by their physician, without diagnosis criteria for the syndrome, a diagnosis and treatment is simply established based on the symptoms exhibited by that individual (Michaelis 2017). The fact that it should be epidemiologically recognised in a population subgroup is not possible under normal physician treatment situations. It is generally recognised that chronic exposure to OPs for example, produce non-specific and diffuse symptoms. In treating one case at a time will not necessarily alert physicians to the syndrome reality. This relates to the concept of aggregated ignorance which will be introduced and expanded-on in the ignorance chapter, where one looks at each tree rather than notice the forest.

The seemingly different vulnerability between the aircrew and the passengers can start to be explained by Terry's studies of chronic low dose exposures with some higher dose episodes (Terry 2012). In an in-vitro study with neuroblast cells, low level chronic exposure made them more-susceptible to neurotoxic damage when they encountered a higher dose exposure than cells that did not have any such pre-exposure (Axelrad 2003). It has also already been established that some of the population have low levels of livers enzymes such as paraoxonases which detoxify OPs in the liver. This was evidenced in farmers who used OPs in sheep dips: those with low paraoxonase were likely to suffer from 'dippers flu' (Cherry 2002).

The diverse constituents of the cabin toxins bring up the growing evidence of the combined action of all such toxins (Carvalho 2014, IGHRC 2009) as covered in other parts of this thesis.

In the study into Aerotoxic Syndrome by Michaelis et al (2017) together with other studies, the Bradford Hill causation criteria were met in 8 of the 9 criteria: the exception was a dose-response relationship. A cause and effect relationship was therefore established. Causation can also be established by other studies (Ramsden 2012).

The typical industry reaction to such studies is simply to refer to the set exposure standards for workplaces which are far above the levels found to be reactive in the above cases and studies. It is interesting to note however that such exposure standards admit that such levels are to protect most (not all) of those exposed. It seems that it has always been recognised that some people will react to concentrations below those occupational standards.

In-Conclusion to Australian Workers Compensation Case and Senate Hearing on Aircraft Cabin Fumes

This section has shown how people with health effects from low level chemical exposures are handled and dismissed by the 'authorities' by considering the situation with aircraft cabin fumes. It highlights how passengers and crew suffering health effects from cabin fumes have been dismissed through personal attacks and on their expert witnesses, 'weight of evidence' arguments, orchestrated consensus statements, and industry shills. It shows the difficulties faced in recognising varied health effects from low level exposures well below workplace acceptable levels and how industry avoids the use of scientific enquiry and study of the actual situation. It also represented an example where an MCS condition was initiated in previously healthy people.

ENVIRONMENTAL EFFECTS ON HEALTH SUPPRESSED BY THE NEOLIBERAL APPROACH

There is a litany of examples through recent history where thousands of people's lives were detrimentally affected due to the political and medical system's inaction on toxicant exposures (Whaley 2013). In most cases there was strong political and/or industrial action that suppressed proper scientific debate. Some, of many examples, were tobacco smoke, organochlorines, dioxins, asbestos, lead dust, benzene, polychlorinated biphenols, chlorofluorocarbons, and phthalates. Warning observations were ignored decades before their emergence as devastating public health issues (Bijlsma 2016, Harremoës 2001).

The political/industry influence also carried through to the legal redress side for the victims even in the face of clear evidence. An example of this is the Bhopal Union Carbide incident in India in 1984 where a gaseous methyl isocyanate leak from a factory killed between 2500 and 10000 people within the first few days (Shrivastava 1987). Having not even counted the victims, the Supreme Court of India ruled that Union Carbide pay only US\$470 million and be granted immunity from criminal prosecution (Das 2000). As stated in a 2006 government affidavit, the leak caused 558,125 injuries which includes 38,478 temporary partial injuries and about 3,900 severely and permanently disabling injuries (Dubey 2010). The abandoned chemicals are still

leaking and pollute groundwater with consequential health effects (Broughton 2005, Shaini 2008, Chander 2001, Ram 2012).

The acceptance of new illnesses involving toxicants is also typically confounded by political/industry influences. The acceptance therefore involves more than an epidemiological acknowledgement and a social process (Brickman 2006). This section will discuss how neoliberalism has contributed to this situation.

Neoliberalism

The neoliberal governance features a reduced economic regulation with the corporate sector obtaining more freedom and authority. For 'increased efficiency', public services such as health become more privatised and managed as profit making centres. The quality of health becomes measured through 'efficiency dividends' and 'competition' (Phillips 2015, Gadiel 2012). This extends through to the government regulatory departments where their classification as profit making centres eventually can unofficially reclassify the regulated industry as clients.

Regulatory Capture has been documented in many countries, especially the US and UK (Davis 2013, Dukes 2014, Abraham 1995, Gotzsche 2014). This phenomenon is well-established, with Bernstein's book as far back as 1955 (Bernstein 1955) establishing the theory. It occurs where a regulatory authority was initially established to safeguard a significant portion of the population acting quite stringently at first. Over time as the originators tire or retire, the authority comes to see the viewpoint of the industry it regulates (Abraham 1995, Judge Loevinger 1964). This may be to such an extent to 'revolving doors' in the employment of managers and key personnel from the Authority to the industry and vice versa. This cycle can end if a significant scandal triggers the commencement of a new cycle for the generally politically inactive public interest.

The flawed and corrupt approval processes for pharmaceutical drugs have been highlighted in recent years (Davis 2013, Dukes 2014, Abraham 1995, Gotzsche 2014). The neoliberal government approaches since the 1980s have led to corporate bias and regulatory capture with many drugs, vaccines and chemical products coming onto the market whether or not they were in the patients' or publics' health interest. The typical 21st century environment is now a multitude of chemical exposures, aside from pharmaceutical drugs, which have not been adequately assessed individually, let alone on a total load basis, especially for sensitive,

genetically pre-disposed, people. MCS is quite inconvenient to industry, as well as politically, if such a condition becomes recognised since the limited toxicological tests that actually have been done also become inadequate due to never considering such a significant portion of the population that are sensitive.

The effects of neoliberal governance are also experienced on another part of the MCS health cycle: after the MCS condition is contracted by a sufferer. It extends through to the insurance industry, particularly in Workers' Compensation, where there must be more-competition through lower premiums. This is usually done by lowering claim rates. So a new and varied health condition from common environmental exposures needs to be strongly resisted. This is evidenced by numerous legal cases where MCS sufferers have unsuccessfully sought compensation in workplace exposure situations. MCS sufferers are then denied the concept of a 'sick role' (Parsons 1951) legally and thus denied medical support, exemption from various work duties, and even recognition by friends and family.

MCS sufferers are actually seeking a redefinition of the sick role: they do not require their exemption from the workplace: they require the workplace (and public places) to be environmentally inert (Kroll-Smith 1997).

Normalisation

The concept of 'governmentality' refers to the mechanism where power is internalised by individuals in the community (Foucault 1991). As the State imposed its power through institutions such as prison, medical hospitals, and mental institutions, categories established in this process shaped how individuals in society should judge and conduct themselves. So rather than a top-down power imposition, a form of capillary power has developed in society: to be a good citizen, one needed to be compliant, law abiding and healthy. The behaviour of others is policed by society so that they too become normalised. Thus a 'normalisation' process to be part of present-day society.

The orthodox medical perception of illness is that it has uniform characteristics independent of the person (Brunton 2014, Ashcroft 2016). The characteristics need to have established pathology. So normalisation has become central for determining health status. This is further helped by an overemphasis on the use of technology for the pathology which provides the elevation of the associated diagnosis beyond challenge (Lock 2010). If a health issue cannot be

suitably slotted into such a definable illness then these issues are transformed into problems of the individual and solved accordingly (Treichler 1998). When an individual starts reacting to everyday chemical exposures it is also little wonder why the MCS condition is seldom respected by society in general.

It is interesting that society also shows willingness to accept imposed normalisation situations in respect to the growing environmental influences. For example, puberty is now accepted in girls as young as 7 years old. The public may be concerned, but does not perceive the general situation as a problem to react to but rather a new norm (Crumpler 2014p196). MCS, which is a prime example of environmental effects on health, is just the individual's 'problem' with no perceived indication of consequences for the rest of the population: no 'canary in the coal mine' realisation is made.

In the workplace the maintenance of the neoliberal trend with the moral importance of returning to work (Niemeyer 1991) and a moral behaviourism in 'getting better' showing loyalty to group's standards (Clarke 2003, Tishelman 1998), places perceived responsibility on the MCS sufferer. Of course with life-long sensitivities in the MCS condition and 'getting better' being only a relative term between exposures, the sufferer is perceived quite negatively in society. If the sufferer cannot be diagnosed/labelled as legitimately ill by an appropriate authority, then blame cannot be absolved for their condition. Thus as mentioned previously, they are denied a 'sick role' (Clarke 2003, Dumit 2006) if there is no clear recognition.

A trend attributed to neoliberalism in which the above situations fall is in relation to the erosion of the welfare state (Wacquant 2009). The net result can be seen in the wind-back and imposed requirements for the receipt of social security. In the American and Australian context it can be seen as the imposition of behavioural obligations for receipt of public assistance. To the MCS sufferer, such obligations present further hurdles due to contributing to a 'normalisation' process.

For most MCS sufferers, the first thought is frequently for legal recognition to obtain justice, recognition and problem resolution. This however demonstrates an acceptance by society of power as a commodity as handed down by the political/legal elite (Smart 1989). If the political/legal elite are already influenced by corporations and positivist medical representatives it is little wonder that this legal avenue has not been fruitful.

In turning to the legal system the MCS sufferers must also conform to social norms. Their initiating exposure must be documented and verified by recognised medical practitioners. A structural pathology must be tested for. The claimant's actions must be consistent with normal sickness behaviour concepts: especially-so with insurance claims where the claimant is 'observed' by surveillance activities after making a claim. The Workers' Compensation scheme also requires standardised assessments (discussed below). Experts representing the claimants must present their disciplinary knowledge within accepted medical paradigms/theories since unorthodox theories are discounted. Thus normalising forces define the boundaries and possibilities of scientific debate even before a case is presented to the bench. Finally once in court, the combination of medico-scientific positivism, insurance policies and legal conservatism has been shown to result in negative outcomes for most MCS claimants as mentioned previously (Phillips 2015).

As industry has an interest in MCS not being recognised, their common tactic is to confound the science. In the literature, for most conclusions about chemically related health risks, a dissenting view can be found. Any literature review then concludes "more research is needed" (Bijlsma 2016). In a courtroom situation this denies the MCS sufferer published literature support to their case.

Insurance and Workers Compensation

In Australia, most states have an equivalent of a workers' compensation and rehabilitation system. One of the first steps a claimant must take is report to his workplace's preferred doctor who must localise the affected area and recommend any tests available to measure and quantify the injury. In some states, the affected area must be circled on a picture of a person of ambiguous sex on a standardised medical certificate. Furthermore the doctor is required to codify the injury/disease from schedules of disability which often have predefined percentages of compensation directly linked there to. MCS and other central sensitisation conditions are absent from such schedules. It is left up to the doctor, who may receive many referrals from the employer concerned, to select an appropriate disability for each case handled. Such doctors commonly have no training in low level chemical exposure effects, let-alone, central sensitisation conditions. In the absence of common pathology, presence of multiple organ effects combined with psychological resultant effects, one wonders how ever justice could be afforded to an MCS victim.

If the claimant is not satisfied with the above compensation system, the employer may be sued under common law. However to qualify for common law access, the claimant must receive a percentage disability assessment from a qualified assessor who works to set guidelines for such evaluation. The guidelines are typically from the American Medical Association. In Western Australia (WA), for example, the permanent whole of person impairment percentage threshold is at least 25%. As MCS is essentially a chronic condition, it is interesting to note for example, in Queensland, only 2 of the 296 (as of October 2016) trained and approved assessors were approved to assess chronic pain. In New South Wales (as of October 2016) there was one approved doctor who worked in the area of Occupational and Environmental Medicine, but approved only to assess upper and lower extremities and the spine.

The Australian state governments introduced legislative changes over recent years to reduce the number of workers gaining access to common law which they deemed as an expensive process. In the period 1999-2005 this was relatively successful for WA in reducing the number of workers meeting the threshold from 864 to 56 per annum (Phillips 2015). These legislative changes have therefore been a radical improvement for the insurance companies and improved the cost-efficiency for the compensation system (WorkCover 2007). This is a further example of a neoliberal governance effect which advantages the corporations with diminished rights for the affected, commonly lower-class, worker.

The standardised diagnoses of this system provide no category for a central sensitisation condition. An individual seeking compensation for their illness or disability must reflect the appropriate sick role normalisation. The possible insurance industry surveillance of the claimant in day-to-day activities places the claimant in a panopticon situation mentioned before. A claimant for a back condition preventing them from walking is therefore not expected to routinely walk the dog or go for an exercise run. Insurance industry monitoring can be from phone calls, social activities and actions at home. The claimant must demonstrate consistent injury behaviour in their sick role. For the MCS sufferer this is difficult since if they are diligent and practise avoidance of chemical exposures, they can be relatively 'healthy'. Even when they are suffering there can be little outwardly unusual. In their own normalisation process they aim, and wish, to lead normal lives in the community. A mild-MCS female dancer may love attending dancing classes for a short time each week, realising that the deodorants, muscle heat relaxants, etc. will have a personal effect, such as a headache and fatigue for the rest of

the day, but yet otherwise thoroughly enjoy the activity and social engagement. Yet surveillance will show that the claimant had gone out to a social event. Even shopping in a supermarket is dangerous for MCS sufferers due to the myriad of off-gassing chemicals, let alone the areas where the cleaning and pesticide products are. Such an activity could lay an MCS sufferer down for half or full day afterward due to migraines, gastro-intestinal pains, etc. Yet for surveillance, going to the supermarket appears normal and does not fit the sick role expected. The MCS sufferer's determination and ability despite their condition, to lead as normal a life as possible is not rewarded and becomes negative for their legal case (Phillips 2015).

The power of governmentality is such that it can cause affected workers to be self-aware to normalise their injury in medical-legal situations. They need to ensure that they adhere to the injury's formal description of symptoms and repress aspects that do not conform. In so doing they reinforce the power of the law to define their injury.

Quite often industry defence in legal cases relies on arguing along the lines of acceptable chemical exposure standards in the workplace. In Australia there is WorkSafe's Workplace Exposure Standards for Airborne Contaminants (April 2013) which are based on US guidelines. It is interesting to find statements in its guidelines which recognise individual variability, decades delayed health effects, synergism, potentiation, and sensitising characteristics of some chemical exposures. However it essentially lists acceptable levels of exposure to individual chemicals in workplace situations. Since legislation recognises this standard, it prescribes what society can accept in the workplace. Corporations can meet legal requirements by simply applying these chemical concentration limits. There is no moral duty or incentive to do better than what the law requires (Phillips 2015)

In respect to the adequacy of such standards, it has been pointed-out (Donohoe 2008) that the current Australian Time Weighted Average (TWA) exposure to dieldrin, for example, is 250 micrograms/m³ whereas the saturated vapour concentration is 64 micrograms/m³. This effectively means that even above a closed vat of dieldrin the air concentration would not be above 64 micrograms/m³. As there have been cases of deaths from vapour exposure then the current Australian standards allow 4 times such lethal levels in the workplace. Perhaps these levels enable the survival of rats on which such exposure levels have mostly been based. The

shortcomings of such toxicological approaches done decades ago are well-known today with the regulatory capture of the American, thus Australian authorities, quite evident in looking at these standards. The chemical industry has also introduced untested chemicals into commercial products by exploiting loopholes in regulation and enforcement (Reuben 2010, Sass 2011), highlighted throughout this thesis.

There are many other cases showing governance being influenced by corporate interests on costs rather than workers' health and future well-being. One of these was in the mid 1990's where the NSW WorkCover Authority started to notice an influx of claims under a "psychological stress" classification. Some of the claimants from work environments like painting, cleaning, agriculture, and indoor offices had gone to a clinic that recognised effects from low level chemical exposure. The clinic's tests indicated neurological damage as distinct from anxiety and depression. Their problems were confusion, emotional lability, short term memory loss, delayed reaction times and learning difficulties (Donohoe 2008). However after this rise in 'stress disorders' the state government legislated to cut out compensation for such conditions simply to save money.

Doctor Normalisation

In Australia, in 1993, the four foremost doctors who took seriously the complaints of their MCS and CFS patients, guided them to understand their illness, and helped to improve their condition, were brought before committees of the government's Health Insurance Commission (HIC). The HIC had seized and copied the clinical notes of all their patients with MCS and CFS. During the course of multiple inquisitions of each doctor, they were accused of conducting unnecessary tests on their patients rather than referring them to psychiatrists. The fact that such testing showed abnormalities was disregarded. They were then served with demands for the repayment of all government money connected with the consultations and tests as well as HIC 'administration fees'. It appears that none of the HIC committee members had ever seen a person with MCS and simply did not recognise MCS or CFS as clinical entities. Such actions effectively either bankrupted or ended the careers of two of these doctors.

One of the above doctors drew an apt conclusion: "In the face of a growing understanding that there is a problem to be addressed, medicine's way of dealing with it is not 'how do we address the problem?' but, 'how do we get rid of the people who are addressing the problem?'" (Donohoe 1994).

But such HIC actions are simply the culmination of the power of governmentality in the suppression of MCS recognition. It is the capillary power of governmentality that produces even more casualties of caring professionals (Phillips 2015p130-8). There are examples such as an Australian expert in genetic science, after developing a technique for detecting toxins, thus a physiological basis of MCS, then having all funding for her laboratory stopped. There are also examples where the industry funded scientists and doctors have defamed researchers by floods of bogus letters to journals, and other orchestrated public campaigns (Crumpler 2014p149). It is interesting to know that Australian government research contracts all now have censorship clauses present (Kypri 2015). But commonly the most damaging influence is the hostile gaze of the doctors' professional community and the subtle disempowering treatment of anyone who challenges the prevailing medical paradigm. Decisions by accrediting bodies, social gatherings and courtroom hearings are all situations where existing paradigms are reinforced. Aside from the financial and reputational aspects in supporting unorthodox theories, it is the desire to conform that also influences a doctor in dealing with a MCS sufferer.

Conclusion on Neoliberalism Effects

The neoliberal trend in governance over the last few decades has seen more corporate influence and reliance in the health and legal areas. Regulatory Capture and legal reforms in the Workers' Compensation area have seen the gradual erosion of public rights for the chemically affected.

The governmentality and normalisation of societal structure through various forms of capillary power have standardised what is 'expected' in sicknesses and medical treatment. The legal system has in-turn been affected by government legislation, medical positivism and insurance industry attitudes.

An emergent condition, such as MCS, which is triggered from everyday products in the modern environment, not producing pathological results for diagnosis of a 'normal' disease is quite outside societal normalisation expectations. Of course, the recognition of a reaction to the very products that many prominent industries manufacture can fuel quite a resistance by the industries that in-turn influence governments and society aided by a neoliberal approach. Such a health condition would be commercially inconvenient for both the chemical and insurance industries. To avoid medical and legal recognition, it must be resisted, even debunked it seems.

It has been generally recognised in social medicine that neoliberalisation erodes the general public's rights, particularly the working classes. This part of society also happens to have the least influence and ability to be able to avoid or modify environmental exposures.

Environmental effects on health, not only MCS, can transfer into the next generation as evidenced by studies into epigenetics, non-monotonic dose responses and endocrine disruptors (Lagarde 2015, Gore 2015). The neoliberal approach has suppressed and destroyed the lives of many MCS sufferers in today's society, but it is also affecting future generations. The broad evidence of the resultant environmental connections with many of today's most common diseases is significant as has been shown in chapter 2.

THE CONSEQUENCES IF MCS HAD RECOGNITION

It has previously been pointed out that there would be quite large consequences for industry and insurance companies in compensation cases if MCS was to be recognised as a legitimate illness. Below are some examples of this. The examples are intended to be brief rather than detailed simply for illustration of the consequential societal changes.

Consequences of the Mechanism to Allergists

Originally health problems triggered by environmental exposures were all regarded generally as 'allergies'. In 1925, 'allergy' was redefined in the context of antibodies and antigens. In the period after, it is interesting to note that even allergy shots were still regarded as "witchcraft" and "voodoo medicine" by most orthodox medical practitioners (Miller 2001). When the IgE mechanism was discovered in 1967, the allergists finally had a scientific basis and subsequent recognition by orthodox medicine. This resulted in quite a separation between allergists and clinical ecologists, who were dealing with MCS sufferers without the precondition for a recognised mechanism.

The allergists confounded the case for MCS recognition over the years. As allergists had a 'scientific' immunologic basis to their diagnoses they had quite often contributed to the write-off of MCS sufferers under psychological banners due to not fitting their allergy definitions/paradigm, especially so when being 'experts' in disability claims.

There have been quite a number of allergists recently who have recognised the need to deal with the unexplained hypersensitivity aspect of allergic reactions. In peanut allergy, for example, the threshold dose for exposure reactions varies tremendously: as little as 100 µg of peanut protein provokes reactions in some people (Hourihane 1997). In many allergic reactions, even with cow's milk allergy, and many other food allergies, there are both IgE-mediated and non-IgE-mediated mechanisms. The sometimes poor treatment of MCS sufferers who have non-IgE-mediated conditions over the years has illustrated the importance to the allergists, of staying inside their accepted paradigm.

Consequences for Toxicological Assessments

Toxicological chemical assessment, including all food chemicals and drugs, is based on the principle that there is a level of exposure (dose) above-which is a toxic reaction and below which there is no detected adverse reaction. Frequently there is a second assumption that once

a maximum reaction occurs there are no further effects in altering the dose. To determine safe dosage animal studies have been used extensively. Unfortunately such assessments are concerned with only short term major effects, mainly specific adverse events or specific end points such as skin/eye irritation, cancer, or liver toxicity. LD50 values (Lethal Dose that kills 50% of the animals exposed) and NOELs (No Observable Effect Levels) are the cornerstones of such testing. The animals of course cannot advise their state of depression, anxiety, nausea, headache, fatigue etc., nor it seems, are the authorities concerned with such.

It is orthodoxy to assume that one size fits all: Mr Joe Average is the healthy adult male whose responses are used to derive safe dosages. There is no compensation for the immune-compromised, the detoxification-compromised, infants' developing immune/neurological systems, etc. A crude concentration per kg of body mass is frequently applied to compensate for young children. The whole medical drug-administration system employs such an approach. This had already conflicted with the paediatric acknowledgement of the uniqueness of a developing child: it is not just a small adult.

Orthodox toxicological approaches test each toxicant in-vivo or in-vitro on its own in a sterile lab environment, far-removed from real-life exposures to a myriad of chemicals, viruses and bacteria. It has been argued that the 'safe' dosage values should be much lower in the external environment. The applicability of rodent in-vivo testing for human adverse health effects has traditionally been disputed by industry when their product comes into question, however in other situations, depending on the animal studied, such testing may underestimate risk for humans (Holmes 2010, Council 2007).

The latest preference for in-vitro tests to assess a chemical's toxicity falls far short of simulating the complex inter-connectedness of human bodily systems involved in the MCS mechanism. Even before such mechanisms were proposed it was claimed "we are conducting a vast toxicological experiment in society, in which our children and our children's children are the experimental subjects". "Little information on possible toxic potential is available for the 80,000 chemicals registered today with the EPA, only 43% have received even minimal toxicological assessment, and a mere 23% have been tested to determine whether they have the potential to cause developmental damage" (Weiss 2000p373).

Consequences for Epidemiological Studies

Epidemiological studies are most predictive when everyone reacts to each chemical exposure with the same symptoms. If however the affected people react with a variety of symptoms ranging from headaches, to muscle spasms, to poor concentration, etc., there may not be any single symptom that has a statistical significance over controls. In MCS studies, even the identification of a suitable control group presents a major problem.

As most epidemiological studies are based on clearly defined and testable end points, most studies are not helpful for understanding or treating MCS.

Consequences for Clinicopathological Studies

Clinicopathological studies rely on presence of clinical signs (e.g., tachycardia or decreased reflexes), laboratory measurements or tissue pathology. Meaningful results on a sample population require consistency of such findings after a specific chemical exposure. In the case of MCS, there are no consistent clinicopathological test results. The variety of organs affected further complicates what tests should be employed. Such tests however depend on end-organ damage: If such testing is relied on then subjective complaints from sufferers may miss the early effects in the condition's development.

Consequences for Medical Specialisation

The trend in the orthodox medical system has been toward increased medical specialisation. But the human body is an integrated unit and, as the MCS mechanism shows, many bodily systems are simultaneously affected. These aspects are best summed up by two quotes:

Polysymptomatic illness requires a holistic approach, which is at odds with the reductionism of the current paradigm.Doctors are taught to regard polysymptomatic illness as psychiatric illness; that the more symptoms a patient reports, the less valid any is (Crumpler 2014p163-4).

The time has come to give to the study of the responses that the living organism makes to its environment the same dignity and support which is being given at present to the study of the component parts of the organism.... Exclusive emphasis on the reductionist approach will otherwise lead biology and medicine into blind alleys (Dubos 1964p13).

Consequences for Psychiatry

Psychiatric diagnoses of MCS sufferers have sometimes resulted in broken families, bankruptcy, committal to mental institutions and suicides. The subjective psychiatric diagnoses that have been used on most MCS sufferers to date are an indication of little substantiation in solid research. Whilst it has a place in society, its overuse may be a problem.

If MCS was accepted as having a physiological basis, psychiatrists would need to rethink their whole approach to this condition.

Consequences for the Workplace

Current safe-exposure levels, based-on the inadequate toxicological testing described above, would no longer be valid for the 2 to 27% of the population which has varying sensitivity to chemical exposures at orders of magnitude lower than the usual 'safe' levels for workplaces.

Consequences for the Legal System

There have been thousands of legal cases where the MCS sufferers have attempted to obtain compensation for chemical exposures affecting their lives so extremely. The traditional opposing case is to write them off psychologically (Phillips 2015, EHLM 2016). There are a number of 'expert' psychologists who feature in many such cases with well-polished paradigmatic opinions and criticisms against any physiological basis for MCS. A recognised physiological basis would open-up large compensation costs for industry, and the chemical/pharmaceutical companies.

Consequences in Everyday Life

In any average home or work environment there are thousands of synthetic chemical types that can be present in the ambient air. Carpets alone, for example, can account for in-excess of 160 chemicals ranging from common solvents through to carcinogenic ones, including formaldehyde. But virtually all synthetic materials out-gas to varying degrees (HHI 2016, Stanford 2016), e.g., all plastics, particle board, paint, detergents, cleaning products, perfumes, deodorants, etc. let alone those toxic ones deliberately used such as pesticides for cockroaches, spiders, termites etc. which depend on an outgassing for effect, or the latest trends of constant indoor deodorisation using chemicals to mimic natural odours, or the flurry of antibacterial chemical spray and wipes.

There are also the myriad of other chemical exposures other than the above predominantly dermal and inhalational exposures. Water supplies are routinely dosed in poisonous chemicals,

food contains chemical additives for taste, colour, emulsification, appearance, bulking, preservation, etc. let alone the residues from farming such as artificial hormones, pesticides, preservatives, fumigation, etc.

To reduce/eliminate the above exposures requires a paradigm shift of the population in general due to these being considered as normal product to achieve a modern standard of living.

Consequence for National Health Expenditures

A financial and economic assessment for the orthodox system to consider is putting more emphasis on addressing the causes of disease, rather than sending their patients down a path of continued treatment protocols for the symptoms. This potentially represents an area of significant saving. Reflecting the increased disease incidence in the population the total expenditure on health in Australia over the last 18 years, after adjusting for inflation, increased from \$91 billion to \$185 billion, 10% of Gross Domestic Product (2018), with an average growth rate of 4.3% per year (AIHW 2020). In the US these figures have increased from US\$1.4 trillion to US\$3.6 trillion in the last 18 years, 17.7% of Gross Domestic Product (2018), with a present average growth rate of 4.6% per year (CDC 2019). One would have thought that actively investigating the environmental causes of diseases rather than just treating the symptoms would have also made economic sense. Addressing this issue is outside the scope of this thesis.

INDUSTRY ACTION EXAMPLE: HEALTH EFFECTS FROM EMFs

Through this chapter we have considered general actions by industry to counter recognition of MCS. A self-contained supporting example illustrating the overall argument in this chapter is considering the situation with the consequences of EMF exposures. Such aspects were touched on in the previous EHS section. This section simply considers such health effects from an industry action viewpoint.

The industry attitude can be illustrated by the situation in the early 1990s where a growing public awareness of health effects from cell phone radiation was occurring after publicised court cases attributing cancer incidence to cell phone use. In 1995 the communications body Cellular Telecommunications and Internet Association (CTIA) commissioned George Carlo to direct the industry-financed Wireless Technology Research project (WTR). Its eventual budget

of US\$28.5 million made it a prominent investigation of cell-phone safety. In 1999, Carlo advised the industry body that the WTR's research had found: "The risk of rare neuro-epithelial tumors on the outside of the brain was more than doubled...in cell phone users"; there was an apparent "correlation between brain tumors occurring on the right side of the head and the use of the phone on the right side of the head"; and "the ability of radiation from a phone's antenna to cause functional genetic damage [was] definitely positive...." (Hertsgaard 2018). He urged to give consumers "the information they need to make an informed judgment about how much of this unknown risk they wish to assume," especially since some of the industry spokespersons had "repeatedly and falsely claimed that wireless phones are safe for all consumers including children." The industry then took measures to silence Carlo and remove him from further work on the project. Carlo said that "they would do what they had to do to protect their industry, but they were not of a mind to protect consumers or public health."

Carlo's situation is equivalent to the memo to company executives that Exxon's manager of environmental-affairs programs, sent explaining that burning oil, gas, and coal could raise global temperatures by 3 degrees Centigrade by 2100 (Glaser 1982).

Another study, at about a US\$30 million cost, over a 10 year period from 1999, was done under the US National Toxicology Program. Toxicology studies were done on rats and mice for potential health effects, including cancer risk, from exposure to a range of frequencies from about 700 – 2700MHz. The studies found that high exposure to cell phone frequencies was linked with:

- Tumours in the hearts of male rats (malignant schwannomas).
- Evidence of brain tumours in male rats (malignant gliomas).
- Tumours in male rat adrenal glands (benign, malignant, or complex combined pheochromocytoma).

The studies had been approved in their design by government scientists, including the FDA, and the experiment set-ups built by industry experts. However, Dr. J. Shuren, Director of the FDA's Center for Devices and Radiological Health, dismissed the results as not being relevant to humans (Shuren 2018). But animal testing is used to develop drugs, to evaluate chemicals, to evaluate pesticides. Yet although these same tests with the same protocols have been standard for 40 years, in this particular case, he personally didn't think they were relevant.

The 'revolving door' between industry and regulatory departments is illustrated by the quote:

Many members who are commissioners of the [US Federal Communications Commission] FCC now, formerly worked directly for Verizon, for the Cell Phone Telecommunication Industry Association and others. And they regularly, for the past two decades had moved back and forth between the industry that they regulate and the agency. So that at this point, the FCC sees itself as an industry enabler, not at all as a protector of public health and safety.

[The] report by Norman Alster for the Safra Center for Ethics at Harvard University said exactly that; the FCC is a captured agency. It has not been able to operate independently for years because of this close tie that exists between the industry and the FCC (Davis 2019p4,5, Alster 2015).

Although the FCC has repeatedly said that EMF is safe, they have no health department in their organisation.

With allowable radiation intensities being based on thermal effects only, health effects have been found "orders of magnitude below the safety guidelines, like 1,000 times lower or 100 times lower" (Pall 2019p2). But it was found that many phone brands subjected the user above even the allowable limits (the US federal limit of 1.6 W/kg). Nearly half of the phones failed in testing at 5mm. At 2mm, representing a phone kept in your clothing, all but one failed (Roe 2019). This is a clear example of deceit by industry and lack of regulation by government departments.

Articles by some authors on the effects of EMF seek to simply downplay the issue: "There's no evidence 5G is going to harm our health, so let's stop worrying about it" and blame a campaign of misinformation on its effects (Loughran 2019p1). These statements by industry connected authors seem to be trying to differentiate 5G from other radiation bands and ignore the "at least 14000" (Pall 2019p15) studies showing effects of EMF in many bands, below allowable levels. This statement makes use of undone science on the specific 5G band in that it simply has not been tested for health effects. Although there is an absence of health effect studies, the military has long known that crowd control is possible (Active Denial Systems) using 5G, 95-

96GHz millimetre waves (USDOD 2019). When people are exposed to this radiation they feel like their body is on fire.

US government spoke-persons have often referred to the new 5G technology development as a way to help America be on the leading edge of the 5G wireless 'race' for economic supremacy in an effort to use national pride to steam-roll any issues of concern. "It's a worldwide race. It's a security challenge. It's a geopolitical battle between the United States and China" (Rosenworcel 2019).

The Swiss telecommunication company, Swisscom Ag, gained a patent in 2004 for an invention to reduce electrosmog in wireless local networks. As part of its reasons substantiating the need for such an invention it admitted non-thermal health effects from EMFs (Swisscom 2003) with statements such as:

The influence of electrosmog on the human body is a known problem.

When, for example, human blood cells are irradiated with electromagnetic fields, clear damage to hereditary material has been demonstrated and there have been indications of an increased cancer risk.

Thus it has been possible to show that mobile radio radiation can cause damage to genetic material, in particular in human white blood cells, whereby both the DNA itself is damaged and the number of chromosomes changed. This mutation can consequently lead to increased cancer risk. In particular, it could also be shown that this destruction is not dependent upon temperature increases, i.e. is non-thermal.

While there have been some national guidelines generated for EMF exposure in relation to specific devices, the exposure to wireless local area networks (WLAN) can be considerable. As new applications continue to expand, the desire for quicker transmission rates sees higher energy bands being used. This is so for both the private and public applications. So the WLAN exposures are continually increasing and also represent exposures in relation to where people spend considerable amounts of time: place of work, internet, network games, etc. The radiation impact on people is steadily growing in this area.

Yet Swisscom rolled out 5G which is a much more powerful and potentially more damaging technology. They have rolled out 5G in full knowledge that it will be seriously detrimental to humans.

A group of investigative journalists from the European Union, called Investigate Europe, suggests the existence of an “ICNIRP cartel” (IE 2019). The International Commission on Non-ionizing Radiation Protection (ICNIRP) is a non-government organization (NGO) which has produced EMF exposure guidelines generally adopted by most countries. It consists of a panel of about 14 self-selected members and advisors. Many of these panel members are also on the WHO committees. The organisation simply dismisses studies showing harmful effects from chronic exposure to non-thermal levels of EMF. There are also several dozen EMF scientists in various countries that actively defend the ICNIRP exposure guidelines. But more than 240 EMF scientists from 42 nations who have published research on EMF effects in over 2,000 papers, signed the International EMF Scientist Appeal. The Appeal requests the WHO, the United Nations and all member nations to adopt lower EMF exposure guidelines to enable sub-thermal levels to be addressed and to inform the public on EMF exposure risks.

The insurance industry does not insure cell phone towers: the industry must self-insure. This perhaps reflects the many studies showing health effects from the towers (Dode 2011, Meo 2019, Subhan 2018, Eger 2006, 2008, Bortkiewicz 2012, Santini 2003, Gómez-Perretta 2013, Khurana 2010, Navarro 2003, Hutter 2006, Abdel-Rassoul 2007). In two past technology developments: the nuclear industry and the pharmaceutical industry for vaccines, the US government gave industry immunity from legal action on the resultant health effects. The situation where it could also be argued along similar lines, that communications are essential in society, has similar potential.

The Children’s Health Defense organisation (CHD) filed a lawsuit against the US Federal Communications Commission (FCC) on 2 Feb 2020. The lawsuit argued that the FCC’s guidelines were based on the assumption that non-ionizing radiation at non-thermal levels (levels that do not change temperature in tissue) cannot have biological and adverse health effects, ignore human biology and are based on an obsolete, false and disproven scientific assumption promoted by physicists and engineers. Thousands of studies were cited as well as “hundreds of testimonials by people who have become sick from radiation within the FCC-allowed levels. The

petitioners also argued that the FCC ignored evidence of mechanisms of harm; their guidelines do not apply to actual use of cell phones and disregard the biological effects of crucial elements of this technology such as pulsation, modulation and specific frequencies, nor do they address the current reality of constant and cumulative effects of exposure to many sources of radiation over many years” (CHD 2021). The court’s final ruling in August 2021, was that the FCC did not consider the many health effects possible and “...the FCC completely failed to acknowledge, let alone respond to, comments concerning the impact of RF radiation on the environment...The record contains substantive evidence of potential environmental harms.” The case highlighted how government departments can abandon their statutory obligations to protect the public in favour of industry interests.

With the WHO unable to form committees without being filled by industry backed representation, and government departments turning a blind eye to the research coming out, the public is left generally ignorant of the situation. It becomes similar to the tobacco industry strategy for countering anti-tobacco advocates on which a Brown & Williamson executive wrote, “Doubt is our product, since it is the best means of establishing a controversy...at the public level” (B&W 1969p4). More will be discussed about this approach in the ignorance chapter of this thesis.

CONCLUSION

This chapter has considered the environmentally induced health condition of multiple chemical sensitivity (MCS). It is a key example that shows how mainstream researchers and practitioners resist recognising environmental health effects.

Many sufferers of this illness identify clear links with environmental exposures and learn through experience that by avoiding such exposures they can achieve improved health. It is not simply an academic argument that requires many studies to establish cause and effect: it is simply one of observation, consistency and the experiences of patients themselves.

The dominant view of MCS is that it is psychosomatic. Part of that view is the orthodox medical system’s expectation of a clear diagnosis with distinct pathology and consistent symptoms: this is not possible with MCS. The epigenetic nature of MCS also makes it a more complex illness to

understand, both in terms of its origins and to achieve clear study outcomes. As a consequence, there is widespread dismissal of supporting research and of MCS patients themselves. An industry effort to suppress the recognition of MCS, as a health condition that shows sensitivity to many industrial products, further adds to the orthodox medical and health systems' resistance to recognising the condition.

The complexity of the condition of MCS produces difficulties in establishing a disease mechanism demanded by orthodox medicine. The mechanisms are far from being straightforward and there appears to be no singular pathways linking one particular chemical exposure directly to one affected organ, as per the orthodox expectation. Many direct and indirect pathways can be involved and alter or spread as chronic exposures are prolonged. Since all humans are being exposed to a variety of xenochemicals and EMFs, it is not a simple task to study the effect of one exposure on its own, when exposure synergism and the myriad of endogenous biochemical pathways can multiply and enhance the exposure effects. Despite the immensity of the problem, which is not acknowledged and perhaps not realised by orthodox medicine, the popular mechanistic model hypotheses have been touched on.

With negative industry-backed studies and criticism of positive independent studies of MCS, the orthodox medical system rides on the manufactured and selective ignorance created by industry. The characteristics of MCS have been documented over the decades by clinical ecologists and functional medicine practitioners in trying to assist affected patients. An example illustrated the diagnostic approaches of orthodox medicine that consider symptoms only, and how this can be inadequate for patients suffering from environmental exposures.

Aside from the many possible pathways and mechanisms after an environmental exposure, an individual's present and past exposure history and genetic make-up will determine the health condition they will experience, such as MCS, CFS, CIRS, EHS or another chronic disease. Studies into just one of these conditions in isolation simply become examples of siloed research. In considering studies on overlapping diseases and illnesses, this chapter showed that MCS does not present a unique set of symptoms: environmental exposures result in a broad range of overlapping health conditions. When the many other chronic diseases with environmental exposure links are considered (see Chapter 2), classifying the varied and broadscale effects into defined disease diagnoses becomes a difficult task.

The paradigmatic aspects that prevent the recognition of emergent diseases per se were considered, with MCS the prime example. The orthodox medical system has tried to move from its belief systems of centuries past, to a supposed scientific basis with strong positivist approaches. Yet these positivist approaches become a hindrance to change due to the complexity and overlapping nature of emergent diseases such as MCS.

The difference between a disease and an illness was also presented as another hindrance to acceptance by the orthodox medical system. The system's reliance on organic or structured pathology, excluding brain and spinal cord diagnosis criteria, was shown to be a convenient approach for excluding MCS as a "real" disease. The process required to classify a disease as "legitimate" involves a normalisation process, but MCS is quite different to conventional diseases and its sufferers cannot be "normalised". The problem is therefore perceived to be with the individual rather than the environment that sufferers are exposed to.

The orthodox medical system and health authorities require proof that MCS is real, according to their own criteria. This chapter showed that the initial basic scientific step of observing patients is usually missed by doctors and health professionals. Yet when observations are performed and MCS is treated as environmentally induced doctors can be criticised by the system. Doctors are simply expected to fit standardised diagnosis criteria to existing health symptoms. MCS, without any standardised criteria, can then only be treated psychologically. This then reflects a double standard in that psychological diagnoses usually have no clear diagnostic signs either. The orthodox treatment of these conditions usually involves the prescription of low level xenochemicals, in the form of medications, flying in the face of the medical system's professed disbelief that low-levels of xenochemicals can cause health effects.

The medical system's widespread basis for establishing diagnosis guidelines, evidence-based medicine (EBM), has stifled the ability of doctors to apply their own observation and experience. Furthermore, EBM is readily corruptible by industry as has been discussed.

The paradigmatic belief systems of the orthodox medical system present barriers to recognising environmental health effects. This was discussed in relation to Kuhn's crisis in belief systems. It was shown that no fundamental belief system crisis is underway due to the orthodox system's ability to treat, or dismiss, suffering as psychosomatic. This hands over patients to psychologists

with the original diagnosing doctor removed from further involvement. This effectively removes the doctor's problem and avoids a belief crisis.

This chapter showed that the orthodox medical system's ignorance of environmental effects is actively fostered by industry, in a deliberate attempt to ensure that MCS will not be recognised as a legitimate health condition. Industry has also influenced the legal system. Legally, the MCS sufferer must clearly prove direct causation from an identifiable exposure, through established and "accepted" scientific studies. As seen in this chapter, this becomes almost impossible. The use of "expert" witnesses was also shown to not provide the plaintiff with an advantage because of industry influence on the legal system. The chapter concluded that the legal pursuit of justice is generally not an avenue for an MCS sufferer.

The neoliberal trend in governance over the last few decades has seen greater corporate influence on the health and legal sectors, which have also become more reliant on those corporate bodies. Regulatory capture and legal reforms in workers' compensation have seen governments gradually erode the rights of the chemically affected to obtain compensation. The governmentality and normalisation of societal structure through various forms of capillary power has standardised what is "expected" in sicknesses and medical treatment, as demonstrated in this chapter. The legal system has, in turn, been affected.

An emergent condition such as MCS, triggered by everyday products in the environment, and not exhibiting "normal" pathology that can be diagnosed as a "normal" disease, has been shown to be quite outside societal normalisation expectations. This is the same for workers exposed to various workplace chemicals in respect to compensation. At the very least, this chapter showed that MCS illustrates the inadequacies of the workplace exposure standards that the industry, government and legal system allow. It was shown that recognising a reaction to the very products that prominent industries manufacture opens a Pandora's box and fuels quite a resistance by industry, that in-turn, aided by a neoliberal approach, influences governments and society. Such a health condition is commercially inconvenient for the chemical, communication and insurance industries. To avoid medical and legal recognition, it must be resisted, even debunked it seems, to create the impression that MCS is a "contentious" issue in medical science. The neoliberal approach has been a tool used by industry to achieve a societal position and attitude against recognising such health conditions.

A consistent finding in social medicine shows that neoliberalisation erodes the rights of the general public, particularly the working classes. The part of society who is most affected also happens to have the least influence and ability to avoid or modify environmental exposures. Apart from the major consequences for industry and insurance companies (in compensation cases, were MCS recognised as a legitimate illness), other significant changes would be required in societal approaches, as this chapter discussed.

It is a long path toward recognising MCS as a legitimate health condition, and more independent research needs to be done on the mechanisms involved and its treatment. This has resulted from a concerted effort by industry to keep MCS as a physiological condition so there is little substantive research that can be found simply because little has been done. Such a situation of “undone science” is frequently exploited by industry representatives, who argue that the existence of MCS is “controversial” or “contentious”. This occurs to such an extent that journalists covering the topic mostly feel obliged to present “both sides”.

Medical paradigms complement the situation of “undone science”. Medical professionals have established careers built on orthodox paradigms and have a personal and collective interest in protecting them. The sceptical response to MCS is typical of how many emergent conditions are treated by the medical system. Even the central concepts of “individual sensitisation” and “environmental triggering” are not in most doctors’ vocabulary. If a portion of the population, estimated at up to 27%, react to environmental triggers and this highlights environmental effects on health, this should attract significant interest. Especially so given the medical system has no explanation for so many other unexplained overlapping health conditions whose prevalence has been increasing in the last few decades.

The recognition of MCS has parallels to the issues around lead, asbestos and tobacco, where corporations attempted to slow down recognition, regulation and compensation through targeted medical research, denying problems, shaping debate, running public relations campaigns, shaping medical opinion, enacting political influence and stymieing legislation and compensation: prime examples of the application of corporate power (Lax 1998p729).

The general public dismisses the myriad of constant xenochemical exposures (plastic bottles, coffee mugs and lids, benzene fumes, EMFs, etc.). They do so feeling that they can tolerate such exposures without clear short-term health effects. These environmentally sensitive

victims are blamed for their problem and are metaphorically 'cast out'. However, for those who are sensitive, the MCS sufferers, every exposure can be devastating. Recent history offers many examples, e.g., the plague, HIV, allergy, etc. This has also been the case with asthma, for example, connections with environmental exposures happening to the whole population fail to be recognised. More recently childhood asthma has substantially increased, over the last four decades reaching rates of one in three, but we continue to think that it's a problem for this growing minority only (Donohoe 2008). But the links with effects from environmental influences are becoming extensive. One only has to consider the myriad of allergic reactions to food contents, air pollutants, etc., yet this is never linked to a logic that considers that environmental influences could also be an initiator for many other unexplained diseases whose rate of incidence is increasing.

MCS may be a key, not for recognising the condition itself to acknowledge the experience of a group of genuinely unhealthy people, but that the "normal" environment itself is unhealthy and MCS is simply a condition, a canary, that reveals it. In these days of growing recognition of epigenetics and non-monotonic dose responses to endocrine disrupting chemicals which can affect future generations, MCS and environmental effects in general should be seriously researched as a matter of utmost urgency.

REFERENCES

Aaron L, Buchwald D, A review of the evidence for overlap among unexplained clinical conditions, *Ann Intern Med*, 2001,134, 868-81.

Aaron L, Buchwald D, Chronic diffuse musculoskeletal pain, fibromyalgia and co-morbid unexplained clinical conditions, *Best Pract Res Clin Rheumatol*, 2003, 17, 563-74.

Abdel-Rassoul G, El-Fateh O, Salem M, Michael A, Farahat F, El-Batanouny M, Salem E, Neurobehavioral effects among inhabitants around mobile phone base stations, *Neurotoxicology*, 2007, 28, 2, 434-40.

Abraham J. Science, politics and the pharmaceutical industry: Controversy and bias in drug regulation, UCL Press, 1995.

Abrams H, Anderson H, Anderson J, Ashford N, Baker D, Beaumont P, Bergeisen G, Bingham E, Castleman B, Catlin M, Letter to World Health Organization, International Labour Office, United Nations Environment Program, and International Program on Chemical Safety, *Archives of Env Health*, 1996, 51, 4, 338-40.

ACAI: American College of Allergy & Immunology, Environmental illness, multiple chemical sensitivities, diagnostic and treatment strategies, 9 Nov 1990, San Francisco, CA, brochure.

Adams J, Galloway T, Mondal D, Esteves S, Mathews F, Effect of mobile telephones on sperm quality: a systematic review and meta-analysis, *Env Int*, 2014, 70, 106-12.

AIHW: Australian Institute of Health and Welfare, Health Expenditure, Snapshot, 23 Jul 20
<https://www.aihw.gov.au/reports/australias-health/health-expenditure> accessed 26 Dec 2021.

Aitken R, Bennetts L, Sawyer D, Wiklendt A, King B, Impact of radio frequency electromagnetic radiation on DNA integrity in the male germline, *Inter J Androl*, 2005, 28, 171-79

Akdag M, Dasdag S, Canturk F, Karabulut D, Caner Y, Adalier N, Does prolonged radiofrequency radiation emitted from Wi-Fi devices induce in various tissues of rats? *J Chem Neuroanat*, 2016, 75(Pt B), 116-22.

Akdag M, Dasdag S, Canturk F, Akdag M, Exposure to non-ionizing electromagnetic fields emitted from mobile phones induced DNA damage in human ear canal hair follicle cells, *Electromagn Biol Med*, 2018, 37, 2, 66-75

Alessandrini M, Micarelli A, Chiaravalloti A, et al. Involvement of subcortical brain structures during olfactory stimulation in multiple chemical sensitivity, *Brain Topography*, 2016, 29, 2, 243-52.

Alobid I, Nogue S, Izquierdo-Dominguez A, et al. Multiple chemical sensitivity worsens quality of life and cognitive and sensorial features of sense of smell, *Eur Arch Otorhinolaryngol*, 2014, 271, 12, 3203-8.

Al-Serori H, Ferk F, Kundi M, Bileck A, Gerner C, Mišik M, Nersesyan A, Waldherr M, Murbach M, Lah T, Herold-Mende C, Collins A, Knasmüller S, Mobile phone specific electromagnetic fields induce transient DNA damage and nucleotide excision repair in serum-deprived human glioblastoma cells, *PLoS One*, 2018, 134, e0193677.

Alster N, Captured Agency: How the federal communication commission is dominated by the industries it presumably regulates, Edmond J Safra Center for Ethics, Harvard University, 2015
https://ethics.harvard.edu/files/center-for-ethics/files/capturedagency_alster.pdf accessed 25 Sep 2019.

Anderson R, Toxic Emissions from Carpets, *J Nutr and Env Medicine*, 1995, 5, 375-86.

Andersson L, Johansson A, Millqvist E, Nordin S, Bende M, Prevalence and risk factors for chemical sensitivity and sensory hyperreactivity in teenagers, *Int J Hyg Env Health*, 2008, 211, 690-97.

Andersson L, Bende M, Millqvist E, Nordin S, Attention bias and sensitization in chemical sensitivity. *J Psychosom Res*, 2009, 66, 5, 407-16.

Andersson L, Claeson A, Dantoft T, Skovbjerg S, Lind N, Nordin S, Chemosensory perception, symptoms and autonomic responses during chemical exposure in multiple chemical sensitivity, *Int Arch Occ and Env Health*, 2016, 89, 1, 79-88.

Angell M, Is academic medicine for sale? *NEJM* 2000, 342, 20, 1516-18.

Angell M, The truth about drug companies- how they deceive us and what to do about it, Random House Trade Paperbacks, NY 2004.

Angell M, Drug companies & doctors: A story of corruption, *The New York Review of Books*, 2009, 56, 1, 8-12.

Ashford N, Miller C, Chemical Exposures: Low Levels High Stakes, NY, Van Nostrand Reinhold, 1991.

ASCIA: Australian Society of Clinical Immunology and Allergy website
<https://www.allergy.org.au/health-professionals/papers/unorthodox-techniques-for-diagnosis-and->

[treatment?highlight=WyJpZGlvcGF0aGljiwiZW52aXJvbm1lbnRhbClsludG9sZXJhbmNlliwiaW50b2xlcmlFuY2UnliwiaWRpb3BhdGhpYyBlbnZpcm9ubWVudGFsliwiaWRpb3BhdGhpYyBlbnZpcm9ubWVudGFsIGludG9sZXJhbmNlliwiaW52aXJvbm1lbnRhbCBpbmRvbGVyYW5jZSld](#) accessed 26 Dec 2017.

Ashcroft R, Van Katwyk T, An examination of the biomedical paradigm: A view of social work, *Social Work in Public Health*, 2016, 31, 3, 140-52.

Australian Senate, 2000a, Official Committee Hansard: Rural & Regional Affairs and Transport Committee, 1 May.

Australian Senate, 2000b, Official Committee Hansard: Rural & Regional Affairs and Transport Committee, 1 Feb.

Australian Senate, 2000c, Official Committee Hansard: Rural & Regional Affairs and Transport Committee, 2 Feb.

Avendaño C, Mata A, Sanchez Sarmiento C, Doncel G, Use of laptop computers connected to internet through Wi-Fi decreases human sperm motility and increases sperm DNA fragmentation, *Fertil Steril*. 2012, 97, 1, 39-45.e2.

Axelrad J, Howard C, McLean W, The effects of acute pesticide exposure on neuroblastoma cells chronically exposed to diazinon, *Toxicology*, 2003, 185, 67–78.

Azuma K, Uchiyama I, Takano H, et al. Changes in cerebral blood flow during olfactory stimulation in patients with multiple chemical sensitivity. A multichannel near infrared spectroscopic study, *Plos one*, 2013, 8, 11.

[Azuma K](#), [Uchiyama I](#), [Katoh T](#), [Ogata H](#), [Arashidani K](#), [Kunugita N](#), Prevalence and Characteristics of Chemical Intolerance: A Japanese Population-Based Study, *Arch Environ & Occ Health*, 2015, 70, 6, 341-353.

Azuma K, Uchiyama I, Kunugita N, Factors affecting self-reported chemical intolerance: A five-year follow-up study in Japan, *J Psychosomatic Research*, 2019, 118, 1-8.

B&W: Smoking and health proposal, 1969, Brown & Williamson Records, Minnesota Documents, Tobacco Industry Influence in Public Policy. <https://www.industrydocuments.ucsf.edu/docs/psdw0147>

Baan R, Grosse Y, Lauby-Secretan B, El Ghissassi F, Bouvard V, Benbrahim-Tallaa L, Guha N, Islami F, Galichet L, Straif K, Carcinogenicity of radiofrequency electromagnetic fields, *Lancet Oncol*, 2011, 12, 624–26.

Bailer J, Witthöft M, Rist F, Psychological predictors of short- and medium term outcome in individuals with idiopathic environmental intolerance (IEI) and individuals with somatoform disorders, *J Toxicol Env Health, part A*, 2008a, 71, 11-12, 766-75.

Bailer J, Witthoft M, Rist F, Modern health worries and idiopathic environmental intolerance, *J Psychosom Res*, 2008b, 65, 425–33.

Baldwin C, Bell I, Increased cardiopulmonary disease risk in a community –based sample with chemical odor intolerance. Implications for women’s health and health care utilization, *Arch env health*, 1998, 53, 3, 347-53.

Barnes B, *Scientific Knowledge and Sociological Theory*, Abingdon, Routledge, 2009.

Barrett S vs Sherrell D and another, 2002, Civil No. 99-813-HO, US District Court.

- Bartha L, Baumzweiger W, Buscher D, Callender T, Dahl A, Davidoff A, Donnay A, Edelson S, Elson B, Elliott E, Multiple chemical sensitivity: A 1999 consensus, *Arch Env Health*, 1999, 54,3,147-49.
- Bascom R, Meggs W, Frampton M, et al, Neurogenic inflammation: with additional discussion of central and perceptual integration of nonneurogenic inflammation, *Env Health Persp*, 1997, 105, Suppl 2, 531-37.
- Beale I, Pearce N, Conroy D, Henning M, Murrell K, Psychological effects of chronic exposure to 50 Hz magnetic fields in humans living near extra-high-voltage transmission lines, *Bioelectromagnetics: Journal of the Bioelectromagnetics Society, The society for physical regulation in biology and medicine, The European Bioelectromagnetics Association*, 1997, 18, 8, 584-94.
- Beck E, Souhami R, Hanna M, Holdright D, *Tutorials in Differential Diagnosis*, 4th Ed, Edinburgh: Churchill Livingstone, 2003.
- Bedard K, Krause K, The NOX Family of ROS-Generating NADPH oxidases: physiology and pathophysiology, *Physiol Rev*, 2007, 87, 1, 245-313.
- Beder S, *Global Spin; The corporate assault on environmentalism*, Melbourne, Scribe, 2000.
- Bekelman J, Li Y, Gross C, Scope and impact of financial conflicts of interest in biomedical research: a systematic review, *JAMA*, 2003, 289, 4, 454-65.
- Bell I, Schwartz G, An olfactory-limbic model of multiple chemical sensitivity syndrome: possible relationship to kindling and affective spectrum disorders, *Biological Psychiatry*, 1992, 32, 218-42.
- Bell I, Schwartz G, Peterson J, Amend D, Self reported illness from chemical odors in young adults without clinical syndromes or occupational exposures, *Arch Environ Health*, 1993, 48, 6-13.
- Bell I, Hardin E, Baldwin C, Schwartz G, Increased limbic system symptomatology and sensitizability of young adults with chemical and noise sensitivities, *Environ Res*, 1995, 70, 84-97.
- Bell I, Baldwin C, Schwartz G, Sensitization studies in chemically intolerant individuals: implications for individual difference research, *Ann N Y Acad Sci*, 2001, 933, 38-47.
- Belpomme D, Campagnac C, Irigaray P, Reliable disease biomarkers characterizing and identifying electrohypersensitivity and multiple chemical sensitivity as two etiopathogenic aspects of a unique pathological disorder, *Rev Environ Health*, 2015, 30, 4, 251-71.
- Belpomme D, Hardell L, Belyaev I, Burgio E, Carpenter D, Thermal and non-thermal health effects of low intensity non-ionizing radiation: An international perspective, *Env pollution*, 2018, 242, 643-58.
- Belyaev I, Dean A, Eger H, et al. EUROPAEM EMF Guideline 2016 for the prevention, diagnosis and treatment of EMF-related health problems and illnesses, *Reviews on Env Health*, 2016, 31, 3, 363-97.
- Berg N, Linnegarg A, Dirksen A, Elberling J, Prevalence of self-reported symptoms and consequences related to inhalation of airborne chemicals in a Danish general population, *Int Arch Occup Environ Health*, 2008, 81, 7, 881-87.
- Bergqvist U, Vogel E, Eds. Possible health implications of subjective symptoms and electromagnetic fields. A report prepared by a European group of experts for the European Commission, DGV. Arbete och Hälsa, 1997, 19. Swedish National Institute for Working Life, Stockholm, Sweden.
<https://gupea.ub.gu.se/handle/2077/4156> accessed 22 May 2017.
- Berlatsky N, The most common childbirth practice in America is unnecessary and dangerous, *New Republic*, Aug 2015.

- Bernstein D, 2001, Frye, Frye, Again: The Past Present, and Future of the General Acceptance Test. Law and Economics Research Paper Series, Paper No. 01–07. Arlington, Va.: George Mason University School of Law. Available at http://papers.ssrn.com/paper.taf?abstract_id=262034.
- Bernstein M, Regulating Business by Independent Commission, Princeton, NJ, Princeton University Press, 1955.
- Bernstein D, Junk Science in the United States and the Commonwealth, Yale Journal of International Law, 1996, 21, 123–82.
- Bero L, Barnes D, Industry Affiliations and Scientific Conclusions, letter, JAMA 1998; 280, 13, 1141.
- Bertschler J, Butler J, Lawlis G, Rea W, Johnson A, Psychological components of environmental illness: factor analysis of changes during treatment, Clin Ecol, 1985, III, 2, 85–94.
- Bijlsma N, Cohen M, Environmental chemical assessment in clinical practice: unveiling the elephant in the room, Int J Env Res & Pub Hth, 2016, 13, 181.
- Billauer B, The causal conundrum: examining the medical-legal disconnect in toxic tort cases from a cultural perspective or how the law swallowed the epidemiologist and grew long legs and a tail, Posted: 1 Aug 2017 University of Haifa - Faculty of Law; Institute of World Politics; Foundation for Law and Science Centers, Inc. https://papers.ssrn.com/sol3/papers.cfm?abstract_id=3011402# accessed 30 dec 17.
- Black D, Okiishi C, Schlosser S, The Iowa follow up of chemically sensitive persons, Ann NY Acad Sci, 2001, 933, 48-56.
- Black D, Temple S, Idiopathic environmental intolerance (multiple chemical sensitivity), Up To Date <https://www.uptodate.com/contents/idiopathic-environmental-intolerance-multiple-chemical-sensitivity> accessed 24 June 2019.
- Bloch R, Meggs W, Comorbidity patterns of self-reported chemical sensitivity, allergy, and other medical illnesses with anxiety and depression, J Nutr Environ Med, 2007, 16, 2, 136–148.
- Boffey P, The Brain Bank of America: An Inquiry into the Politics of Science, McGraw-Hill, New York, 1975.
- Bohr T, Problems with myofascial pain syndrome and fibromyalgia syndrome, Neurology, 1996, 45, 593-7.
- Bornschein S, Hausteiner C, Römmelt H, Nowak D, Förstl H, Zilker T, Double -blind placebo-controlled provocation study in patients with subjective Multiple Chemical Sensitivity and matched control subjects, Clinical Toxicology, 2008a, 46, 443-49.
- Bornschein S, Hausteiner C, Drzezga A, et al., Neuropsychological and positron emission tomography correlates in idiopathic environmental intolerances, Scand. J Work environ health, 2007, 33, 6, 447-53.
- Bornschein S, Hausteiner C, Pohl C, et al., Pest controllers: a high-risk group for multiple chemical sensitivity (MCS), Clinical toxicology, 2008b, 46, 193-200.
- Bortkiewicz A, Gadzicka E, Szykowska A, Politański P, Mamrot P, Szymczak W, Zmyślony M, Subjective complaints of people living near mobile phone base stations in Poland, Int J occup med env health, 2012, 25, 1, 31-40.
- Brennan T, Buying editorials, N Engl J Med, 1994, 331, 673-75.

Brickman E, Book Review: Emerging illnesses and society: negotiating the public health agenda, *Med Hist*, 2006, 50, 4, 549–50.

Broughton E, The Bhopal disaster and its aftermath: a review, *Env Health*, 2005, 4, 6, 6.

Brunton D, *Health and wellness in the 19th century*, Oxford, England, Greenwood, 2014.

Buchwald D, Garrity D, Comparison of patients with chronic fatigue syndrome, fibromyalgia, and multiple chemical sensitivities, *Arch Intern Med*, 1994, 154, 2049-53.

Buckman R. 2. Why Breaking Bad News Is Difficult. In *How To Break Bad News* 2017 May 22 (pp. 15-39). University of Toronto Press.

Burgio E, 5th Paris Appeal Congress in Belgium, 2015.

<https://www.youtube.com/watch?v=iPQSotTJORw> at 8.40 accessed 22 Sep 2019.

Burns-Naas L, Meade B, Munson A, Toxic responses of the immune system. In: Klaassen C, Ed. *Casarett and Doull's toxicology: the basic of poisons*, 6th ed, NY, McGraw Hill, 2001, 419–70.

Byun Y, Ha M, Kwon H, Hong Y, Leem J, Salong J, et al., Mobile phone use, blood lead levels, and attention deficit hyperactivity symptoms in children: a longitudinal study, *PLoS One*, 2017, 8, e59742.

Caccamo D, Cesareo E, Mariani S, et al., Xenobiotic sensor and metabolism related gene variants in environmental sensitivity related illnesses: a survey on the Italian population, *Oxidative medicine and cellular longevity*, Vol 2013, Article ID 831969.

Caccappolo E, Kipen H, Kelly- McNeil K, et al., Odor perception : multiple chemical sensitivities, chronic fatigue , and asthma, *Joem*, 2000, 42, n6, 629-38.

Carpenter D, The microwave syndrome or electro-hypersensitivity: historical background, *Rev Env Health*, 2015, 30, 4, 217–22.

Candore G, Balistreri C, Listi F, Grimaldi M, Vasto S, Colonna-Romano G, et al., Immunogenetics, gender, and longevity, *Ann N Y Acad Sci*, 2006, 1089, 516–37.

Caress M, Steinemann A, A Review of a Two-Phase Population Study of Multiple Chemical Sensitivities, *Env Health Persp*, 2003, 11, 12.

Caress S, Steinemann A, A national population study of the prevalence of multiple chemical sensitivity, *Arch Intern Med*, 2004, 59, 300-5.

Caress S, Steinemann A, Asthma and chemical hypersensitivity: prevalence, etiology, and age of onset, *Tox and Ind Health*, 2009, 25, 1, 71-8.

Carvalho R, Arukwe A, Ait-Aissa S, Bado-Nilles A, Balzamo S, Baun A, et al., Mixtures of chemical pollutants at European legislation safety concentrations: how safe are they? *Toxicol Sci*, 2014, 141, 1, 218–33.

CBS News, After several childhood cancer cases at one school, parents question radiation from cell tower, April 4, 2019, <https://www.cbsnews.com/news/cell-tower-shut-down-some-california-parents-link-to-several-cases-of-childhood-cancer/> accessed 6 Oct 2019.

CDC: Centers for Disease Control and Prevention, National Center for Health Statistics, Health Expenditures 2019 <https://www.cdc.gov/nchs/fastats/health-expenditures.htm> accessed 26 Dec 2021.

Chander J, Water contamination: a legacy of the union carbide disaster in Bhopal, India, *Int J Occup Environ Health*, 2001, 7, 1, 72-3.

CHD: Children's Health Defense: CHD v. FCC: 5G & Wireless Radiation Guidelines Lawsuit <https://childrenshealthdefense.org/seeking-justice/legal/chd-v-federal-communication-commission-fcc/#stage8> accessed 29 Sep 21.

Cherry M, Mackness N, Durrington P, Povey A, Dippnall M, Smith T, et al., Paraoxonase (PON1) polymorphisms in farmers attributing ill health to sheep dip, *Lancet*, 2002, 359, 763-4.

Cheruvu P, Kishnani K, Lewis B, Piscitelli S, Ramaswamy S, Weinhoff G, Asare E, inventors; Axovant Sciences GmbH, assignee. Compositions and methods of treating a neurodegenerative disease. United States patent application US 15/677,837, 2018 Feb 15.

Chew A: *Alysia Chew v East West Airlines Ltd and Ansett Australia Ltd*, Matter no.19652 of 1995, New South Wales Compensation Court.

Chiaravalloti A, Pagani M, Micarelli A, et al., Cortical activity during olfactory stimulation in multiple chemical sensitivity: a 18F-FDG PET/CT study, *Eur J Nucl Med Mol Imag*, 2015, 42, 733-40.

Choi K, Ha M, Ha E, Park H, Kim Y, Hong Y, et al., Neurodevelopment for the first three years following prenatal mobile phone use, radio frequency radiation and lead exposure, *Environ Res*, 2017, 156, 810e817.

Choi Y, Moskowitz J, Myung S, Lee Y, Hong Y, Cellular phone use and risk of tumors: Systematic review and meta-analysis, *Int j env research and public health*, 2020, 17, 21, 8079.

Ciccione D, Natelson B, Comorbid illness in women with chronic fatigue syndrome: a test of the single syndrome hypothesis, *Psychosom Med*, 2003, 65, 268-75.

Clarke J, James S, *The Radicalised Self: The impact on the self of the contested nature of the diagnosis of chronic fatigue syndrome*, *Social Science & Medicine*, 2003, 57, 8, 1387-95.

CMA: Chemical Manufacturers Association, Environmental illness briefing paper, *The Reactor*, 1990, 4, 1-13.

COE: Council of Europe – Parliamentary Assembly. The potential dangers of electromagnetic fields and their effect on the environment. Resolution, Doc. 1815, Text adopted by the Standing Committee, acting on behalf of the Assembly, on 27 May 2011 <https://assembly.coe.int/nw/xml/XRef/Xref-XML2HTML-en.asp?fileid=17994> accessed 1 Oct 2019.

Collins H, *Changing Order: Replication and Induction in Scientific Practice*, Chicago, Univ. of Chicago Press, 1985.

CoM: Commonwealth of Massachusetts, Special Legislative Committee on Indoor Air Pollution, *Indoor Air Pollution in Massachusetts*, April 1989.

Consales C, Merla C, Marino C, Benassi B, Electromagnetic fields, oxidative stress, and neurodegeneration, *Int J Cell Biol*, 2012, 683897.

Costa A, Branca V, Minoia C, Pigatto P, Guzzi G, Heavy metals exposure and electromagnetic hypersensitivity, *Sci Total Environ*, 2010, 408, 20, 4919-20.

COT position paper on cabin air. London: Committee of Toxicity; 2013 <https://cot.food.gov.uk/sites/default/files/cot/cotpospapcabin.pdf> accessed 15 Jan 2018.

Council N, Toxicity testing in the 21st century: A vision and a strategy, National Academy Press, Washington, DC, 2007.

Cox L, Michaelis S, A survey of health symptoms in BAe 146 aircrew, *J Occ Health & Safety – Aust & NZ*, 2002, 1, 6, 305-12.

CP: Tribunal Administratif De Cergy-Pontoise N° 1608265 Audience du 20 décembre 2018 Lecture du 17 janvier 2019 <https://wearetheevidence.org/wp-content/uploads/2019/04/French-Decision-Jan-2019.pdf>

Cranor C, Eastmond D, Scientific Ignorance and Reliable Patterns of Evidence in Toxic Tort Causation: Is There a Need for Liability Reform? *Law & Contemporary Problems*, 2001, 64, 5–48.

Cribb J, *Poisoned Planet: How constant exposure to man-made chemicals is putting your life at risk*, Allen & Unwin, Sydney, 2014.

Crumpler D, *Prostituting Science: The Psychologisation of MCS, CFS and EHS for political gain*, Maryborough, VIC; Inkling Australia, 2014.

Crumpler (2014p148): *New Scientist*, 1 Feb 2003.

Crumpler D, MCS and the molecules of memory: a point to ponder, *Sensitivity Matters*, 2002, 34, 17-22.

Cui X, Lu X, Hiura M, Oda M, Miyazaki W, Katoh T, Evaluation of genetic polymorphisms in patients with multiple chemical sensitivity, *PLoS one*, 2013, 8, 8, e73708.

Cui X, Lu X, Hisada A, Fujiwara Y, Katoh T, The correlation between mental health and multiple chemical sensitivity: a survey in Japanese workers, *Env health prev med*, 2015, 20, 123-29.

Cullen M, The worker with multiple chemical sensitivities: an overview. *Occ medicine: State of the Art Reviews*, 1987, 2, 4, 655-61.

Dantoft T, Elberling J, Brix S, Szecsi P, Vesterhauge S, Skovbjerg S, An elevated pro-inflammatory cytokine profile in multiple chemical sensitivity, *Psychoneuroendocrinology*, 2014, 40, 140-150.

Dantoft T, Skovbjerg S, Andersson L, et al., Inflammatory mediator profiling of n – butanol exposed upper airways in individuals with multiple chemical sensitivity, *Plos One*, 2015, 1-15.

Das V, *Suffering, Legitimacy and Healing*. In Kroll-Smith S, Brown P, Gunter V, Eds, *Illness and the Environment: A reader in contested medicine*, NY, New York University Press, 2000, 270-86.

Daubert v. Merrill Dow, Pharmaceuticals, 509 U.S. 579, 586-591, 1993.

Davidoff A, Keyl P, Meggs W, Development of multiple chemical sensitivities in laborers after acute gasoline fume exposure in an underground tunneling operation, *Arch env health*, 1998, 53, 3, 183-89.

Davidoff A, Fogarty L, Keyl P, Psychiatric inferences from data on psychologic/psychiatric symptoms in multiple chemical sensitivities syndrome, *Arch Env Health*, 2000, 55, 3, 165-75.

Davidoff A, Fogarty L, Psychogenic origins of multiple chemical sensitivities syndrome: a critical review of the research literature, *Arch Env Health*, 1994, 49, 5, 316-25.

Davis C, Abraham J, *Unhealthy pharmaceutical regulation: Innovation, politics and promissory science*, Springer, 2013.

de Boer J, Antelo A, van der Veen I, Brandsma S, Lammertse N, Tricresyl phosphate and the aerotoxic syndrome of flight crew members – current gaps in knowledge, *Chemosphere*, 2015, 119, S58–61.

Deledda G, Moretti F, Rimondini M, Zimmermann C, How patients want their doctor to communicate. A literature review on primary care patients' perspective, *Patient Ed Tr*, 2013, 90, 297-306.

De Luca C, Raskovic D, Pacifico V, Chung Sheun Thai J, Korkina L, The search for reliable biomarkers of disease in multiple chemical sensitivity and other environmental intolerances, *Int J Env Res Public Health*, 2011, 8, 2770-97.

De Luca C, Scordo M, Cesareo E, et al., Biological definition of multiple chemical sensitivity from redox state and cytokine profiling and not from polymorphisms of xenobiotic-metabolizing enzymes, *Tox App pharma*, 2010a, 238, 285-92.

De Luca C, Scordo G, Cesareo E, Raskovic D, Genovesi G, et al., Idiopathic environmental intolerances (IEI): from molecular epidemiology to molecular medicine, *Indian J Exp Biol*, 2010b, 48, 7, 625–35.

De Luca C, Thai J, Raskovic D, Cesareo E, Caccamo D, et al., Metabolic and genetic screening of electromagnetic hypersensitive subjects as a feasible tool for diagnostics and intervention, *Mediat Inflamm*, 2014, 924184.

DEODC: Division of Environmental and Occupational Disease Control, California Department of Public Health, How to Reduce Exposure to Radiofrequency Energy from Cell Phones <https://htv-prod-media.s3.amazonaws.com/files/cell-phone-guidance-1513219841.pdf> accessed 7 Oct 2020.

Desai N, Kesari K, Agarwal A, Pathophysiology of cell phone radiation: oxidative stress and carcinogenesis with focus on male reproductive system, *Reprod Biol Endocrinol*, 2009, 7, 114.

Desforges J, Sonne C, Levin M, Siebert U, De Guise S, Dietz R, Immunotoxic effects of environmental pollutants in marine mammals, *Env int*, 2016, 86, 126-39.

Desmeules J, Cedraschi C, Rapiti E, Baumgartner E, Finckh A, Cohen P, et al., Neurophysiologic evidence for a central sensitization in patients with fibromyalgia, *Arthritis Rheum*, 2003; 48, 1420-9.

Devine R, Letter to Laurie Cox Australian Federation of Air Pilots – dated 4 Feb 2000, Senate Rural and RRegional Affairs and Transport References Committee Inquiry into Air Safety – BAe 146 cabin air quality, Vol 3, p6.

Dhir A, Kulkarni S, Nitric oxide and major depression, *Nitric Oxide*, 2011, 24, 125–31.

Dickson D, *The New Politics of Science*, Pantheon, New York, 1984.

Dixon L, Gill B, Changes in the standard for admitting expert evidence in federal civil cases since the daubert decision, Santa Monica, Calif, Rand Institute for Civil Justice, 2001. Available at <http://www.rand.org/publications/MR/MR1439/>. (Reproduced in (2002) *Psychology, Public Policy & Law* 8: 251–308).

Dode A, Leão M, de Tejo F, Gomes A, Dode D, Dode M, Moreira C, Condessa V, Albinatti C, Caiaffa W, Mortality by neoplasia and cellular telephone base stations in the Belo Horizonte municipality, Minas Gerais state, Brazil, *Science of the Total Environment*, 2011, 409, 19, 3649-65.

Donnay A, Ziem G, Press Release, 9 September 1998: Deceptive draft report on multiple chemical sensitivity released for public comment by US Federal Interagency Workgroup on MCS: workgroup provides only limited information on 8 federal agencies and fails to correct numerous errors & omissions reported in prior review. MCS Referral & Resources

- Donnay A, On the Recognition of Multiple Chemical Sensitivity in Medical Literature and Government Policy, *Int'l J Toxicology*, 1999b, 18, 383.
- Donnay A, Ziem G, Prevalence and overlap of chronic fatigue syndrome and fibromyalgia syndrome with multiple chemical sensitivity syndrome, *J Chronic Fatigue Syndr*, 1999a, 5, 71-80.
- Donohoe M, Killing Us Softly, Internet Creative Commons release 2008
<http://web.mac.com/doctormark/DoctorMark/KUS.html>
- Donohoe M, Altered Neurological Function Following Chemical Exposure, Toxic Chemicals and Human Consequences, Nullamanna, ACTA Conference, 1994.
- Draper G, Vincent T, Kroll M, Swanson J, Childhood cancer in relation to distance from high voltage power lines in England and Wales: a case-control study, *Bmj*, 2005, 330, 7503, 1290.
- Drossman D, Functional versus organic: an inappropriate dichotomy for clinical care, *Am J Gastroenterol*, 2006, 101, 1172-5.
- Dubey A, Bhopal Gas Tragedy: 92% injuries termed "minor", *First 14 News*. Archived from the original on 21 June 2010. Retrieved 26 June 2010.
- Dubos R, *Environmental biology*, *Bioscience*, 1964, 11-4.
- Duehring C, Carpet Update: Industry strategizing memorandum comes to light, *Informed Consent* 1994, 1, 5, 44-48.
- Dukes G, Braithwaite J, Moloney J, *Pharmaceuticals, corporate crime and public health*, Edward Elgar 2014.
- Dumit J, Illnesses you have to fight to get: facts as forces in uncertain, emergent illnesses, *Soc Sci Med*, 2006, 62, 3, 577-90.
- Ecks S, Three Propositions for an Evidenced-Based Medical Anthropology, *J Royal Anthropology Institute*, 2008, 577-92.
- Edmond G, Mercer D, Conjectures and Exhumations: Citations of History and Philosophy and Sociology of Science in US Federal Courts, *Law and Literature*, 2002a, 14, 309-66.
- Edmond G, Legal Engineering: contested representations of law, science (and non-science) and society, *Social Studies of Science*, 2002b, 32, 371-412.
- Edmond G, Mercer D, Daubert and the exclusionary ethos: the convergence of corporate and judicial attitudes towards the admissibility of expert evidence in tort litigation, *Law & Policy*, 2004, 26, 2, 231-57.
- Edward J, Sir Percival Pott, *The Bulletin of the Royal College of Surgeons of England*, 2011, 93, 2, 66-67.
- Eek F, Karlson B, Österberg K, Östergren P, Factors associated with prospective development of environmental annoyance, *J Psychosom res*, 2010, 69, 9-15.
- Eger H, Jahn M, Specific health symptoms and cell phone radiation in Selbitz (Bavaria, Germany)—Evidence of a dose-response relationship, *Cell*, 2008 Dec, 3.
- Eger H, Hagen K, Lucas B, Vogel P, Voit H, The influence of being physically near to a cell phone transmission mast on the incidence of cancer, *Occ Env Med*, 2006, 63, 5.

EHLM: Electronic Handbook of Legal Medicine, 20 Apr 16:
<http://www.medlit.info/member/personalnews/vol7iss1/multichem.html>

Ehrlich G, Pain is real; fibromyalgia isn't, *J Rheumatol*, 2003, 30, 1666-7.

Eis D, Helm D, Mühlinghaus T, et al., The German Multicentre study on multiple chemical sensitivity (MCS), *Int J Hyg Env Health*, 2008, 211, 658-81.

Elberling J, Skov P, Mosbech H, Holst H, Dirksen A, Johansen J, Increased release of histamine in patients with respiratory symptoms, *Clin Exp Allergy*, 2007, 37, 11, 1676–1680.

ESRI: Environmental Sensitivities Research Institute, <http://www.esri.org> (10/11/00).

Fan L, Tien L, Lin R, Simpson K, Rhodes P, Cai Z, Neonatal exposure to lipopolysaccharide enhances vulnerability of nigrostriatal dopaminergic neurons to rotenone neurotoxicity in later life, *Neurobiology of Disease*, 2011, 44, 3, 304–16.

Ferrie H, The Kafkaesque Conviction of Dr J Krop, *Consumers Health of Canada*, Jan 1999; *Fraser Forum*, Mar 1999; *Vitality Magazine*, Fall 1999.

Fiedler N, Kelly –Mc Neil K, Ohman-Strickland P, Zhamg J, Ottenweller J, Kipen H, Negative affect and chemical intolerance as risk factors for building-related symptoms: a controlled exposure study, *Psych Med*, 2008, 70, 254–62.

Fiedler N, Kelly –Mc Neil K, Mohr S, Lehrer P, et al., Controlled human exposure to methyl tertiary butyl ether in gasoline : symptoms, psychophysiologic and neurobehavioural responses of self reported sensitive persons, *Env health persp*, 2000, 108, 8, 753-63.

Fioranelli M, Sepehri A, Roccia M, Jafferany M, Olisova O, Lomonosov K, Lotti T, 5G Technology and induction of coronavirus in skin cells, *J Biological Regulators & Homeostatic Agents*, 2020, 34, 4, 3-10

Ford C, Somatization and fashionable diagnoses: illness as a way of life, *Scand J Work Env Health*, 1997, 23(suppl 3), 7-16.

Foucault M, *Ceci N'est Pas Une Pipe Deux Lettres Et Quatre Dessins de René Magritte*, 1973, Fata Morgana.

Foucault M, *Governmentality*, trans. R Braidotti and revised by C Gordon, In: Burchell G, Gordon C, Miller P (eds), *The Foucault Effect: Studies in Governmentality*, Chicago, Uni of Chicago Press 1991, 87-104.

Foucault M, *Discipline & Punish: The Birth of the Prison*, New York, Random House, 1977.

Foucault M, *La volonté de savoir*, Paris, Gallimard 1, 1976, 1976-84.

Fox M, Kim K, Understanding emerging disabilities, *Disabil Soc*, 2004, 19, 4, 323–37.

Fox R, *Medical Uncertainty Revisited*, In: Albrecht G, Fitzpatrick R, Scrimshaw S (eds) *The Handbook of Social Studies in Health and Medicine*, London 2000, Sage Publications: 409-25.

Foucault M, *Governmentality*, trans. R Braidotti and revised by C Gordon, In: Burchell G, Gordon C, Miller P, (eds) *The Foucault Effect: Studies in Governmentality*, Chicago, Uni of Chicago Press 1991, 87-104.

Foucault M, *Birth of the Clinic*, London, Tavistock, 1973.

- Franceschi C, Valensin S, Fagnoni F, Barbi C, Bonafè M, Biomarkers of immunosenescence within an evolutionary perspective: the challenge of heterogeneity and the role of antigenic load, *Experimental gerontology*, 1999, 34, 8, 911-21.
- Friedmann J, Kraus S, Hauptmann Y, Schiff Y, Seger R, Mechanism of short-term ERK activation by electromagnetic fields at mobile phone frequencies, *Biochem J*, 2007, 405, 3, 559–68.
- Fujimori S, Hiura M, Cui X, Lu X, Katoh T, Factors in genetic susceptibility in a chemical sensitive population using QEESI, *Environ Health Prev Med*, 2012, 17, 357-63.
- Gadiel D, Sammut J, How the NSW Government Should Govern Health: Strategies for micro-economic reform, *Papers in Health and Aging*, 2012. Available online at <http://www.cis.org.au/app/uploads/2015/07/pm128.pdf>? accessed 16 Oct 2016.
- Galbato S, Multiple chemical sensitivity: does Daubert v Merrell Dow Pharmaceuticals Inc warrant another look at clinical ecology?, *Syracuse Law Review*, 1998, 48, 1, 261-98.
- Galland L, Biochemical abnormalities in patients with multiple chemical sensitivities, *Occup Med*, 1987, 2, 4, 713–20.
- Gandhi O, Morgan L, de Salles A, Han Y, Herberman R, Davis D, Exposure limits: the underestimation of absorbed cell phone radiation, especially in children, *Electromagn Biol Med*, 2012, 31, 1, 34-51.
- Gandjour A, Lauterbach K, Inductive Reasoning in Medicine: Lessons from Carl Gustav Hempel's 'inductive-statistical' model, *J Evaluation in Clinical Practice*, 2003, 9, 2, 161-69.
- Georgellis A, Lindelöf B, Lundin A, Arnetz B, Hillert L, Multiple chemical sensitivity in male painters; a controlled provocation study, *Int J.Hyg Env Health*, 2003, 206, 531-38.
- Gibson P, Cheavens J, Warren M, Multiple chemical sensitivity/environmental illness and life disruption, *Women Therapy*, 1996, 19, 63–79.
- Gibson P, Lindberg A, Work accommodation for people with multiple chemical sensitivity, *Disabil Soc*, 2007, 22, 7, 717–732.
- Gibson P, Of the world but not in it: barriers to community access and education for persons with environmental sensitivities, *Health Care Women Int*, 2010, 31, 1, 3–16.
- Gibson P, Lindberg A, Physicians' perceptions and practices regarding patient reports of multiple chemical sensitivity, *ISRN Nurs*, 2011, 838930.
- Gibson P, The hidden marginalization of persons with environmental sensitivities, *Ecopsychology*, 2016, 8, 2, 131-7.
- Glaser M, Exxon Internal Memo: CO2 Greenhouse Effect, 12 Nov 1982
<https://insideclimatenews.org/sites/default/files/documents/1982%20Exxon%20Primer%20on%20CO2%20Greenhouse%20Effect.pdf> accessed 18 Sep 2019.
- Glaser Z, Naval medical research institute, bibliography of reported biological phenomena ('effects') and clinical manifestations attributed to microwave and radio-frequency radiation, Research report, mf12.524.015-0004b, Bibliography of reported biological phenomena (•effects') and clinical manifestations attributed to microwave and radio-frequency radiation, Naval Medical Research Institute, National Naval Medical Centre, 4 October 1971
https://archive.org/stream/NavyRadiowaveBrief/Navy_Radiowave_Brief_djvu.txt accessed 25 Sep 2019

Goldacre B, *Bad Pharma – How Drug Companies Mislead Doctors and Harm Patients*, Faber & Faber Inc NY, 2012.

Goldberg S, *Science about Wireless and 5G, 5G Crisis: Awareness and accountability*, 2019, Documentary series interview transcript.

Gómez-Perretta C, Navarro E, Segura J, Portolés M, Subjective symptoms related to GSM radiation from mobile phone base stations: a cross-sectional study, *BMJ open*, 2013, 3, 12, e003836.

Gore A, Chappell V, Fenton S, Flaws J, Nadal A, Prins G, Toppari J, Zoeller R, Executive Summary to EDC-2: The Endocrine Society's Second Scientific Statement on Endocrine-Disrupting Chemicals, *Endocrine Reviews*, 2015, 36, 6, 593-602.

Gorpinchenko I, Nikitin O, Banyra O, Shulyak A, The influence of direct mobile phone radiation on sperm quality, *Central European journal of urology*, 2014, 67, 1, 65.

Gottesman M, From Barefoot to Daubert to Joiner: Triple Play or Double Error? *Arizona Law Review*, 1998, 40, 753–80.

Gotzsche P, *Deadly Medicines and Organised Crime – How Big Pharma has corrupted health care*, Radcliffe Publishing, 2014.

Gracely R, Petzke F, Wolf J, Clauw D, Functional magnetic resonance imaging evidence of augmented pain processing in fibromyalgia, *Arthritis Rheum*, 2002, 46, 1333-43.

Grigoriev Y, *BioInitiative 2012, A Rationale for biologically-based exposure standards for low-intensity electromagnetic radiation*, Section 8, Evidence for Effects on the Immune System Supplement 2012, Immune System and EMF RF <https://bioinitiative.org/table-of-contents/> accessed 10 Oct 2020.

Gugliandolo A, Gangemi C, Calabrò C, et al., Assessment of glutathione peroxidase-1 polymorphisms, oxidative stress and DNA damage in sensitivity related illnesses, *Life sciences*, 2016, 145, 27-33.

Gundersen H, Harris A, Bråtveit M, Moen B, Odor-related chronic somatic symptoms are associated with self reported asthma and hay fever: the Hordaland health study, *Iran J Allergy Asthma Immunol*, February 2015, 14, 1, 19-27.

Gunnbjörnsdóttir M, Franklin K, Norbäck D, Björnsson E, Gislason D, Lindberg E, Svanes C, Omenaas E, Norrman E, Jøgi R, Jensen EJ, Dahlman-Höglund A, Janson C, Prevalence and incidence of respiratory symptoms in relation to indoor dampness: the RHINE study, *RHINE Study Group, Thorax*, 2006, 61, 3, 221-5.

Gye M, Park C, Effect of electromagnetic field exposure on the reproductive system, *Clin Exp Reprod Med*, 2012, 39, 1, 1–9.

Hadler N, Fibromyalgia” and the medicalization of misery, *J Rheumatol*, 2003, 30, 1668-70.

Haley R, Billecke S, La Du B, Association of low PON1 type Q (type A) arylesterase activity with neurologic symptom complexes in Gulf War veterans, *Toxicol Appl Pharmacol*, 1999, 157, 227-33.

Hallberg O, Oberfeld G, Letter to the editor: will we all become electrosensitive? *Electromagn Biol Med*, 2006, 25, 3, 189–91.

Hamilton W, Gallagher A, Thomas J, White P, Risk markers for both chronic fatigue and irritable bowel syndromes: a prospective case–control study in primary care, *Psychol Med*, 2009, 39, 1913-21.

Hanahan D, Weinberg R, The Hallmarks of Cancer, *Cell*, 2000, 100, 57–70.

Hansson K, Repacholi M, Van Deventer E, Ravazzani P, (eds). In: Proceedings, International Workshop on EMF Hypersensitivity, Prague, Czech Republic, October 2006, 25–27. 2004, Milan: World Health Organization. Working group report, 15–26. Available at: http://www.who.int/peh-emf/publications/reports/EHS_Proceedings_June2006.pdf.

Hardell L, World Health Organization, radio frequency, radiation, and health, a hard nut to crack, *Int J Oncol*, 2017, 51, 405-13.

Hardell L, Carlberg M, Using the Hill viewpoints from 1965 for evaluating strengths of evidence of the risk for brain tumors associated with use of mobile and cordless phones, *Rev Env Health*, 2013, 28, 97–106.

Harremoës P, Gee D, MacGarvin M, Stirling A, Keys J, Wynne B, Vaz S, Late Lessons from Early Warnings: The Precautionary Principle 1896–2000, Office for Official Publications of the European Communities: Luxembourg City, Luxembourg, 2001.

Harrison V, Mackenzie Ross S. An emerging concern: toxic fumes in airplane cabins, *Cortex*, 2015, 74, 297–302.

Hausteiner C, Bornschein S, Hansen J, Zilker T, Förstl H, Self-reported chemical sensitivity in Germany: a population-based survey, *Int J Hyg Env Health*, 2005, 208, 4, 271–278.

Haumann K, Kiesswetter E, van Thriel C, Blaszkewicz M, Golka K, Seeber A, Breathing and heart rate during experimental solvent exposure of young adults with self reported multiple chemical sensitivity (sMCS), *Neurotoxicology*, 2003, 24, 179-86.

Hausteiner C, Mergeay A, Bornschein S, Zilker T, Förstl H, New aspects of psychiatric morbidity in idiopathic environmental intolerances, *J Occup Env Med*, 2006; 48, 76-82.

Hayward-Brown H, False and Highly Questionable Allegations of Munchausen Syndrome by Proxy, 7th Australasian Child Abuse and Neglect Conference, Perth, Australia, Oct 1999.

Hempel S, The strange case of the Broad Street pump: John Snow and the mystery of cholera, Berkeley, CA, University of California Press, 2007.

Hertsgaard M, Dowie M, How Big Wireless made us think that cell phones are safe: A special investigation, *The Nation*, 23 Apr 2018. <https://www.thenation.com/article/how-big-wireless-made-us-think-that-cell-phones-are-safe-a-special-investigation/> accessed 18 Sep 2019.

Heuser G, Wojdani A, Heuser S, Diagnostic markers of multiple chemical sensitivity. Multiple Chemical Sensitivities: Addendum to Biologic Markers in Immunotoxicology, Washington, DC: National Research Council, 1992, 117–38.

HFW: Homeopathy for Women. MTHFR. Available: <http://www.homeopathyforwomen.org/MTHFR.htm>. Accessed 21 Aug 2016.

HHi: <http://www.healthyhouseinstitute.com/a-870-Outgassing>: 20 Apr 16.

Hileman B, Multiple chemical sensitivity, *Chem Eng News*, 1991, 69, 29, 26-42.

Hillert L, Musabasic V, Berglund H, Ciumas C, Savic I, Odor processing in multiple chemical sensitivity, *Human Brain Mapping*, 2007, 28, 172-82.

Hogan W, Paging Dr. Ross: A doctor who defends corporations from “inconvenient” science has a secret of his own, *Mother Jones*, November 2005 <http://www.motherjones.com/politics/2005/11/paging-dr-ross/> accessed 26 Dec 2017.

Hojo S, Ishikawa S, Kumano H, Mijata M, Sakabe K, Clinical characteristics of physician-diagnosed patients with multiple chemical sensitivity, *Japan Int J Hyg, Env Health*, 2008, 211, 682–89.

Holmes A, Creton S, Chapman K, Working in partnership to advance the 3Rs in toxicity testing, *Toxicology*, 2010, 267, 14–19.

Hope J, A Review of the mechanism of injury and treatment approaches for illness resulting from exposure to water-damaged buildings, *Mold and Mycotoxins, The Scientific World Journal*, 2013, Article ID 767482, 20 pages.

Hourihane J, Kilburn S, Nordlee J, Hefle S, Taylor S, Warner J, An evaluation of the sensitivity of subjects with peanut allergy to very low doses of peanut protein: A randomized, double-blind, placebo-controlled food challenge study, *J Allergy Clinical Immun*, 1997, 100, 5, 596–600.

Huang Y, Zhang B, Xie S, Yang B, Xu Q, Tan J, Superparamagnetic Iron oxide nanoparticles modified with tween 80 pass through the intact blood–brain barrier in rats under magnetic field, *ACS applied materials & interfaces*, 2016, 8, 18, 11336-41.

Huber P, *Galileo’s Revenge: junk science in the courtroom*, New York Basic Books, 1991.

Hutter H, Moshammer H, Wallner P, Kundi M, Subjective symptoms, sleeping problems, and cognitive performance in subjects living near mobile phone base stations, *Occ Env med*, 2006, 63, 5, 307-13.

ICAO CIR 344-AN/202, Guidelines on education, training and reporting related to fume events, Montreal, International Civil Aviation Organization, 2015.

ICP: Institute for Chronic Pain. Central Sensitisation. Available at: <http://www.instituteforchronicpain.org/understanding-chronic-pain/what-is-chronic-pain/central-sensitization>. Accessed 4 Sep 2016.

IE: Investigate Europe, The ICNIRP Cartel: Who’s Who in the EMF Research World, <https://www.kumu.io/Investigate-Europe/whos-who> accessed 21 Sep 2019.

IGHRC: Interdepartmental Group on Health Risks from Chemicals, Chemical mixtures: a framework for assessing risks to human health, Cranfield, Institute of Environment and Health, Cranfield University, 2009.

Irigaray P, Caccamo D, Belpomme D, Oxidative stress in electrohypersensitivity self-reporting patients: Results of a prospective in vivo investigation with comprehensive molecular analysis, *Int J molecular med*, 2018a, 42, 4, 1885-98.

Irigaray P, Lebar P, Belpomme D, How ultrasonic cerebral tomosphygmography can contribute to the diagnosis of electro-hypersensitivity, *JBR J Clin Diag Res*, 2018b.

Janson L, Taylor R, Kennedy C, Chronic fatigue syndrome, fibromyalgia, and multiple chemical sensitivities in a community-based sample of persons with chronic fatigue syndrome-like symptoms, *Psychosom Med*, 2000, 62, 655-63.

Jennings D, The confusion between disease and illness in clinical medicine, *CMAJ*, 1986, 135, 865-70.

Jeong I, Kim I, Park H, Roh J, Park J, Lee J, Allergic disease and multiple chemical sensitivity in Korean adults, *Allergy Asthma Immunol res*, 2014, 6, 5, 409-14.

JHM: Johns Hopkins Medicine: Health Home: Conditions and Diseases: Multiple Chemical Sensitivity <https://www.hopkinsmedicine.org/health/conditions-and-diseases/multiple-chemical-sensitivity> accessed 19 Nov 2019.

Ji R, Kohno T, Moore K, Woolf C, Central sensitisation and LTP: do pain and memory share similar mechanisms? *Trends in Neuroscience*, 2003, 28, 12, 696-705.

Jimenez X, Shirvani N, Hogue O, Karafa M, Tesar G, Polyallergy (multiple chemical sensitivity) is associated with excessive healthcare utilization, greater psychotropic use, and greater mental health/functional somatic syndrome disorder diagnoses: a large cohort retrospective study, *Psychosomatics*, 2019, 60, 3, 298-310.

Joffres M, Sampalli T, Fox R, Physiologic and symptomatic responses to low-level substances in individuals with and without chemical sensitivities: a randomised controlled blinded pilot booth study, *Env Health Persp*, 2005, 113, 9, 1178-83.

Johansson A, Brämerson A, Millqvist E, Nordin S, Bende M, Prevalence and risk factors for self-reported odour intolerance: the Skövde population-based study, *Int Arch Occup Environ Health*, 2005, 78, 7, 559–64.

Johansson O, Gangi S, Liang Y, Yoshimura K, Jing C, et al., Cutaneous mast cells are altered in normal healthy volunteers sitting in front of ordinary TVs/PCs – results from open-field provocation experiments, *J Cutan Pathol*, 2001, 28, 10, 513–9.

Johansson O, Liu P, “Electrosensitivity”, “electrosensitization” and “screen dermatitis”: preliminary observations from on-going studies in the human skin. In: Simunic D, (Ed) *Proceedings of the COST 244: Biomedical Effects of Electromagnetic Fields – Workshop on Electromagnetic Hypersensitivity*, Brussels/Graz: EU/EC (DG XIII) 1995, 52–57.

Johnson W, Ross G, Butler J, Fenyve E, Griffiths B, Laseter J, Considerations for the diagnosis of chemical sensitivity. Multiple chemical sensitivities: Addendum to biologic markers in immunotoxicology, National Research Council, Division on Earth and Life Studies, Commission on Life Sciences, 1992, 172-73.

Johnson D, Colman I, The association between multiple chemical sensitivity and mental illness: Evidence from a nationally representative sample of Canadians, *J psychosomatic research*, 2017, 99, 40-4.

Judge L Loevinger cited in Harris R, *The Real Voice*, 1964, p145; New York: Macmillan.

Juutilainen J, Kumlin T, Naarala J, Do extremely low frequency magnetic fields enhance the effects of environmental carcinogens? A meta-analysis of experimental studies, *Int J radiation biology*, 2006, 82, 1, 1-2.

Juutilainen J, Matilainen P, Saarikoski S, Läärä E, Suonio S, Early pregnancy loss and exposure to 50-Hz magnetic fields, *Bioelectromagnetics*, 1993, 14, 3, 229-36.

Kabali H, Irigoyen M, Nunez-Davis R, Budacki J, Mohanty S, Leister K, Bonner R, Exposure and use of mobile media devices by young children, *Pediatrics*. 2015, 136, 6, 1044-50.

Kassirer J, *On the take – how medicine’s complicity with big business can endanger your health*, Oxford University Press, 2004.

Katerndahl D, Bell I, Palmer R, Miller G, Chemical intolerance in primary care settings: prevalence, comorbidity, and outcomes, *Ann Fam Med*, 2012, 10, 357-65.

Katz J, *The silent world of doctor and patient*, New York, Free Press, 1984.

Kendall D, Nothing for free: how private judicial seminars are undermining environmental protections and breaking the public's trust, Washington D.C., Community Rights Counsel, 2000
<http://www.communityrights.org>

Khurana V, Hardell L, Everaert J, Bortkiewicz A, Carlberg M, et al., Epidemiological evidence for a health risk from mobile phone base stations, *Int J Occup Env Health*, 2010, 16, 3, 263–7.

Kimata H, Effect of exposure to volatile organic compounds on plasma levels of neuropeptides, nerve growth factor and histamine in patients with self reported multiple chemical sensitivity, *Int J Hyg Env Health*, 2004, 207, 159-63.

Kleisariis C, Sfakianakis C, Papathanasiou I, Health care practices in ancient Greece: The Hippocratic ideal, *J Med Ethics Hist Med*, 2014, 7, 6.

Klinghardt D, Best Protection from EMFs and 5G, 5G Crisis: Awareness and accountability, 2019, Documentary series interview transcript.

Köllner B, Wasserrab B, Kotterba G, Fischer U, Evaluation of immune functions of rainbow trout (*Oncorhynchus mykiss*)—how can environmental influences be detected? *Toxicology letters*, 2002, 131, 1-2, 83-95.

Kreutzer R, Neutra R, Lashuay N, Prevalence of people reporting sensitivities to chemicals in a population-based survey, *Am J Epidemiol*, 1999, 150, 1, 1-12.

Kroenke K, Physical symptom disorder: a simpler diagnostic category for somatization-spectrum conditions, *J Psychosom Res*, 2006, 60, 335-9.

Kroll-Smith S, Ladd A, Environmental illness and biomedicine; anomalies, exemplars, and the politics of the body, *Sociological Spectrum*, 1993, 13, 7-13.

Kroll-Smith S, Floyd H, Bodies in protest: environmental illness and the struggle over medical knowledge, New York University Press, 1997.

Kundi M, Evidence for childhood cancers (Leukemia). In: Sage C, Carpenter D, (eds) *The BioInitiative Report 2012, A Rationale for a Biologically-based Public Exposure Standard for Electromagnetic Fields (ELF and RF)*, 2012, <http://www.bioinitiative.org/>.

Kypri K, Governments shouldn't be able to censor research results they don't like, *The Conversation*, July 20, 2015.

Laborde A, Tomasina F, Bianchi F, Brune M, Buka I, Comba P, Corra L, Cori L, Duffert C, Harari R, Children's health in latin america: the influence of environmental exposures, *Env Health Persp*, 2015, 123, 201-09.

Lacour M, Zunder T, Schmidtke K, Vaith P, Scheidt C, Multiple chemical sensitivity syndrome: suggestions for an extension of the US. MCS case definition, *Int J Hyg Env Health*, 2005, 208, 141-51.

Lagarde F, Beausoleil C, Belcher S, Belzunces L, Emond C, Guerbet M, Rousselle C, Non-monotonic dose-response relationships and endocrine disruptors: a qualitative method of assessment, *Env Health*, 2015, 14, 13.

Larson J, The Paper Chase, Dateline NBC, June 2000: video copy at <http://www.badfaithinsurance.org/#craneP05> accessed 6 Jan 2018.

Last J, Dictionary of epidemiology, *CMAJ: Canadian Medical Association Journal*, 1993, 149, 4, 400.

- Laudel G, *The Art of Getting Funded: How Scientists Adapt to their Funding Conditions*, *Science and Public Policy*, 2006, 33, 7, 489-504.
- Lax M, Multiple chemical sensitivities: the social construction of an illness, *Int J Health Services*, 1998, 288, 4, 725-45.
- Lee Y, Pai M, Chen J, Guo Y, Central neurological abnormalities and multiple chemical sensitivity caused by chronic toluene exposure, *Occ Med*, 2003, 53, 7, 479-82.
- Levine H, Jørgensen N, Martino-Andrade A, Mendiola J, Weksler-Derri D, Mindlis I, Pinotti R, Swan S, Temporal trends in sperm count: a systematic review and meta-regression analysis, *Human Reproduction Update*, 2017, 23, 6, 646–59.
- Li C, Lee W, Lin R, Risk of leukemia in children living near high-voltage transmission lines, *J occ env med*, 1998, 40, 2, 144-7.
- Li D, Chen H, Ferber J, et al., Exposure to magnetic field non-ionizing radiation and the risk of miscarriage: a prospective cohort study, *Sci Rep*, 2017, 7, 17541.
- Li D, Odouli R, Wi S, Janevic T, Golditch I, Bracken T, Senior R, Rankin R, Iriye R, A population-based prospective cohort study of personal exposure to magnetic fields during pregnancy and the risk of miscarriage, *Epidemiology*, 2002, 13, 1, 9-20.
- Liebeskind D, Bases R, Koenigsberg M, Koss L, Raventos C, Morphological changes in the surface characteristics of cultured cells after exposure to diagnostic ultrasound, *Radiology*, 1981, 138, 2, 419-23.
- Liebeskind D, Padawer J, Wolley R, Bases R, Diagnostic ultrasound time-lapse and transmission electron microscopic studies of cells insonated in vitro, *British journal of cancer, Supplement*, 1982, 5, 176.
- Lindbohm M, Hietanen M, Kyyronen P, Sallmen M, Nandelstadh A, Taslomem H, Pekkarinen M, Ylikoski M, Hemminiki K, Magnetic fields of video display terminals and spontaneous abortion, *Am J Epidem*, 1992, 136, 9, 1041-51.
- Lipkin M, Functional or organic? A pointless question, *Ann Intern Med*, 1969, 71, 1013-7.
- Litinetskaia M, Guelfi J, The environment and new psychiatric pathologies, *L'information psychiatrique*, 2017, 93, 3, 193-8.
- Loblay R, Editorial – Chronic fatigue syndrome: What’s in a name? *Med J Aust*, 1995, 163, 285-86
- Lock M, Nguyen V, *An anthropology of biomedicine*, John Wiley & Sons, 2018 Mar 20.
- Longo D, Approach to the patient with cancer. In: Kasper D, Fauci A, Longo D, Braunwald E, Hauser S, (eds). *Harrison’s principles of internal medicine*, New York, McGraw Hill, 2000, 435-41.
- Lopez A, Williams T, Levin A, Tonelli M, Singh J, Burney P, Rehm J, Volkow N, Koob G, Ferri C, Remembering the forgotten non-communicable diseases, *BMC Med*, 2014, 12, 1, 1-9.
- Loria-Kohen V, Marcos-Pasero H, de la Iglesia R, Aguilar-Aguilar E, Espinosa-Salinas I, Herranz J, de Molina AR, Reglero G, Multiple chemical sensitivity: genotypic characterization, nutritional status and quality of life in 52 patients, *Medicina Clínica (English Edition)*, 2017, 149, 4, 141-6.
- Loughran S, There’s no evidence 5G is going to harm our health, so let’s stop worrying about it, *The Conversation*, August 2, 2019.

- Lowenthal R, Tuck D, Bray I, Residential exposure to electric power transmission lines and risk of lymphoproliferative and myeloproliferative disorders: a case-control study. *Internal medicine journal*, 2007, 37, 9, 614-9.
- Ludlam S, To the Millions Missing with ME/CFS, something remarkable is happening, *The Guardian*, 12 May 2018 https://www.theguardian.com/commentisfree/2018/may/12/to-the-millionsmissing-with-mecfs-something-remarkable-is-happening?CMP=share_btn_fb accessed 17 Jul 2019.
- Lupton D, *Medicine as Culture. Illness, Disease and the Body in Western Societies*, London, Sage, 2012.
- Lynch B, MTHFR and Vaccines/Immunizations – Contraindicated? Available: MTHFR.net, <http://mthfr.net/forums/topic/mthfr-and-vaccinesimmunizations-contraindicated/>. Accessed 7 Mar 2012.
- Mackenzie J, The production of the so-called “rose cold” by means of an artificial rose, *Am J Med Sci*, 1886, 181, 45–56.
- Mahmoudabadi F, Ziaei S, Firoozabadi M, Kazemnejad A, Use of mobile phone during pregnancy and the risk of spontaneous abortion. *Journal of Environmental Health Science and Engineering*, 2015, 13, 1, 34.
- Marcovitch H, Editors, Publishers, Impact Factors, and Reprint Income, *PLoS Med*, 2010, 7, 10, e1000355. Published online 2010 Oct 26.
- Marshall T, Heil T, Electrosmog and autoimmune disease, *Immunologic research*, 2017, 65, 1, 129-35.
- Martin B, Saint Martin F, Mobbing and suppression: footprints of their relationships, *Social Medicine*, 2012, 6, 4, 217-26.
- Martin B, *Philosophy and Social Action*, 1996a, 22, 3, 33-55.
- Martin B, Critics of pesticides: whistleblowing or suppression of dissent? *Philosophy and Social Action*, 1996b, 22, 3, 33-55.
- Martini A, Iavicoli S, Corso L, Multiple chemical sensitivity and the workplace: current position and need for an occupational health surveillance protocol, *Oxidative medicine and cellular longevity*, May 2013. <http://dx.doi.org/10.1155/2013/351457>.
- Masuda H, Hirota S, Ushiyama A, Hirata A, Arima T, Kawai H, Wake K, Watanabe S, Taki M, Nagai A, Ohkubo C, No dynamic changes in blood-brain barrier permeability occur in developing rats during local cortex exposure to microwaves, *In Vivo*, 2015, 29, 3, 351-7.
- McCampbell A, Multiple chemical sensitivity under siege, *Townsend Letter for Doctors and Patients*, Jan 2001.
- McKeown-Eyssen G, Baines C, Cole D, Riley N, Tyndale R, Marshall L, Jazmaji V, Case-control study of genotypes in multiple chemical sensitivity: CYP2D6, NAT1, NAT2, PON1, PON2 and MTHFR, *Int J Epidemiol*, 2004, 33, 971-78.
- MCS: Multiple chemical sensitivity: A 1999 consensus. *Arch env health*, 1999, 54, 3, 147-49.
- McWhinney I, Epstein R, Freeman T, Rethinking somatization, *Ann Int Med*, 1997, 26, 747-50.
- Meggs W, Immunological mechanisms of disease and the multiple chemical sensitivity syndrome. *Multiple Chemical Sensitivities: Addendum to Biologic Markers in Immunotoxicology*, Washington, DC: National Research Council, National Academy Press, 1992, 155–68.

Meggs W, Neurogenic inflammation and sensitivity to environmental chemicals, *Env Health Persp*, 1993, 101, 3, 234-238.

Meggs W, Multiple chemical sensitivities: chemical sensitivity as a symptom of airway inflammation, *J Toxicol Clin Toxicol*, 1995a, 33, 2, 107–110.

Meggs W, Neurogenic switching: a hypothesis for a mechanism for shifting the site of inflammation in allergy and chemical sensitivity, *Env Health Persp*, 1995b, 103, 1, 54-56.

Meggs W, Dunn K, Bloch R, Goodman P, Davidoff A, Prevalence and nature of allergy and chemical sensitivity in a general population, *Arch Env Health*, 1996, 51, 4, 275–82.

Meo S, Almahmoud M, Alsultan Q, Alotaibi N, Alnajashi I, Hajjar W, Mobile Phone Base Station Tower Settings Adjacent to School Buildings: Impact on Students' Cognitive Health, *Am J men's health*, 2019, 13, 1, 1557988318816914.

Michaelis S, A survey of health symptoms in BALPA Boeing 757 pilots, *J Occ Health & Safety - Aust & NZ*, 2003, 19, 3, 253-61.

Michaelis S, Burdon J, Howard C, Aerotoxic Syndrome: a new occupational disease?, *Public Health Panorama*, 2017, 3, 2, 198-211.

Michaelis S, Implementation of the requirements for the provision of clean air in crew and passenger compartments using the aircraft bleed air system [MSc thesis], Cranfield, Cranfield University, 2016 http://www.susanmichaelis.com/pdf/2016_Susan%20Michaelis_MSc%20Cranfield-Clean%20air%20requirements%20using%20bleed%20air%20system.pdf accessed 17 Jan 2018.

Michaelis S, Health and flight safety implications from exposure to contaminated air in aircraft [PhD thesis], Sydney, University of New South Wales, 2010 ([http:// handle.unsw.edu.au/1959.4/50342](http://handle.unsw.edu.au/1959.4/50342) accessed 25 May 2017).

Michaels D, *Doubt is their product: How industry's assault on science threatens your health*, Oxford, UK, Oxford University Press, 2008.

Miller C, Toxicant-induced loss of tolerance, *Addiction*, 2001, 96, 1, 115-37.

Miller A, Morgan L, Udasin I, Davis D, Cancer epidemiology update, following the 2011 IARC evaluation of radiofrequency electromagnetic fields (Monograph 102), *Env research*, 2018, 167, 673-83.

Miltenberg N, Out of the fire and into the Frying pan or Back to the Future, *Trial* 37: 18, 2001 http://expertpages.com/news/out_of_the_fire_into_Fryeing.htm. accessed 6 Sep 2020.

Milward v. Acuity Specialty Products Group, Inc. et al., 664 F. Supp. 2d at 148, (D. Mass. 2011) rev'd 639 F.3d 11 (1st Cir. 2011) U.S. App. LEXIS 5727; 31 I.E.R. Cas. (BNA) 1812; CCH Prod. Liab. Rep. at18,600.

Mizoue T, Onoe Y, Moritake H, Okamura J, Sokejima S, Nitta H, Residential proximity to high-voltage power lines and risk of childhood hematological malignancies, *J epidemiology*, 2004, 14, 4, 118-23.

Mizukoshi A, Kumagai K, Yamamoto N, et al., In-situ real-time monitoring of volatile organic compound exposure and heart rate variability for patients with multiple chemical sensitivity, *Int J Env Res Public Health*, 2015, 12, 10, 12446–65.

MJA, A Point of View, *Med J Aust*, 1991, 293.

- Mordock J, Jury orders DuPont to pay \$2M in C-8 case, *The News Journal*, Dec. 21, 2016 <http://www.delawareonline.com/story/money/2016/12/21/jury-orders-dupont-pay-2m-c8-case/95710838/> accessed 2 Jan 18.
- Mortazavi S, Daiee E, Yazdi A, Khiabani K, Kavousi A, et al., Mercury release from dental amalgam restorations after magnetic resonance imaging and following mobile phone use, *Pak J Biol Sci*, 2008, 11, 8, 1142–6.
- Moss-Morris R, Spence M, To “lump” or to “split” the functional somatic syndromes: can infectious and emotional risk factors differentiate between the onset of chronic fatigue syndrome and irritable bowel syndrome? *Psychosom Med*, 2006, 68, 463-69.
- Mudarri D, Fisk W, Public health and economic impact of dampness and mold, *Indoor Air*, 2007, 17, 3, 226-35.
- Müller K, Schnakenberg E, Die Bedeutung der Glukuronidierung bei umweltmedizinischen Erkrankungen am Beispiel der UDP-Glukuronosyltransferase 1A1, *Umwelt Medizin Gesellschaft* 2008, 21, 4, 295-300.
- Navarro E, Segura J, Portolés M, Gómez-Perretta de Mateo C, The microwave syndrome: a preliminary study in Spain, *Electromagnetic biology and medicine*, 2003, 22, 2-3, 161-9.
- Naviaux R, Perspective: Cell danger response Biology—The new science that connects environmental health with mitochondria and the rising tide of chronic illness, *Mitochondrion*, 2020, 51, 40-5.
- Nettleton S, “I just want permission to be ill”: towards a sociology of medically unexplained symptoms, *Soc Sci Med*, 2006, 62, 1167-78.
- Newman v Motorola, Inc., 218 F Supp 2d 769 (D Md 2002).
- Newman v Motorola, Inc., No. 02–2424 Court of Appeals (4th Cir 2003).
- Niemeyer L, Social Labelling, Stereotyping, and Observer Bias in Workers Compensation: The Impact of Provider-Patient Interaction on Outcome, *J Occ Rehab*, 1991, 1, 4, 251-69.
- Nordberg P, Daubert on the Web, 2003 http://www.daubertontheweb.com/leader_board.htm accessed 3 Jan 2018.
- O’Brien T, Wireless Radiation in Autoimmunity, 5G crisis awareness and accountability, *Documentary Series Transcript*, 2019.
- Ojima M, Tonori H, Sato T, et al., Odor perception in patients with multiple chemical sensitivity, *Tohoku J Exp Med*, 2002, 198, 163-73.
- Onconurse, Solving Problems With Your Doctor: Factsheet. Available: http://www.oreilly.com/onconurse/factsheets/solving_doctor.pdf. Accessed 21 Aug 2016.
- Orriols R, Costa R, Cuberas G, Jacas C, Castell J, Sunyer J, Brain dysfunction in multiple chemical sensitivity, *J neurol sc*, 2009, 287, 72-78.
- Ostan R, Bucci L, Capri M, Salvioli S, Scurti M, Pini E, et al., Immunosenescence and immunogenetics of human longevity, *Neuroimmunomodulation*, 2008, 15, 224–40.
- Östberg K, Ørbaek P, Karlson B, Akesson B, Bergendorf U, Annoyance and performance during the experimental chemical challenge of subjects with multiple chemical sensitivity, *Scand J work env health*, 2003, 29, 1, 40-50.

Österberg K, Persson R, Karlson B, Eek C, Ørbaek P, Personality, mental distress, and subjective health complaints among persons with environmental annoyance, *Human & Experimental Toxicology*, 2007, 26, 3, 231-41.

Pacher P, Beckman J, Liaudet L, Nitric oxide and peroxynitrite in health and disease, *Physiol Rev* 2007, 87, 1, 315–424.

Packard R, Brown P, Berkelman R, Frumkin H, Introduction: Emerging Illness as Social Process. *Emerging Illnesses and Society: Negotiating the Public Health*, 2004, 1-35.

Pall M, Explaining “Unexplained Illnesses”: disease paradigm for chronic fatigue syndrome, multiple chemical sensitivity, fibromyalgia, post-traumatic stress disorder, Gulf War Syndrome, and others, New York, NY (US), London (GB), Harrington Park Press/Haworth Press, 2007.

Pall M, Elevated sustained peroxynitrite levels as the cause of chronic fatigue syndrome, *Medical Hypotheses*, 2000b, 54, 115-25.

Pall M, Elevated peroxynitrite as the cause of chronic fatigue syndrome. Other inducers and mechanisms of symptom generation, *J Chronic Fatigue Syndrome*, 2000a, 7, 4, 139-45.

Pall M, 5G: Great risk for EU, U.S. and International Health! Compelling Evidence for Eight Distinct Types of Great Harm Caused by Electromagnetic Field (EMF) Exposures and the Mechanism that Causes Them https://drive.google.com/file/d/1B_jjTWBYdbg-owmOCacWMcubtCzb0qZv/view accessed 10 Oct 2020.

Pall M, Multiple Chemical Sensitivity: Toxicological and Sensitivity Mechanisms, 2012: Available: http://bobnorson.com/pubs/Pall_english.pdf. Accessed 5 Nov 2015

Pall M, 5G crisis awareness and accountability, *Documentary Series Transcript*, 2019.

Pall M, 5G: Great risk for EU, U.S. and International Health! Compelling Evidence for Eight Distinct Types of Great Harm Caused by Electromagnetic Field (EMF) Exposures, A compilation done for Pall’s submission to EU authorities, May 17, 2018 <https://peaceinspace.blogspot.com/files/5g-emf-hazards--dr-martin-l.-pall--eu-emf2018-6-11us3.pdf> [accessed 4 june 2020](#).

Palmquist E, Claeson A, Neely G, Stenberg B, Nordin S, Overlap in prevalence between various types of environmental intolerance, *Int J hyg Environ health*, 2014, 217, 4-5, 427-34.

Paoloni-Giacobino A, Post genomic decade - The epigenome and exposome challenges, *Swiss Med. Wkly*, 2011, 141, w13321.

Papo D, Eberlein –König B, Berresheim HW, et al., Chemosensory function and psychological profile in patients with multiple chemical sensitivity: comparison with odor –sensitive and asymptomatic controls, *J psychosom res*, 2006, 60, 199-209.

Park J, Knudson S, Statistics Canada, Health Division, How Healthy are Canadians? *Health Reports*, Feb 2006, supra note 9, at 44.

Park J, Knudson S, Medically unexplained physical symptoms, *Health reports*, 2007, 18, 1, 43-47.

Parker v Mobil Oil, 7 N.Y.3d 450, 2006.

Parsons T, *The Social System*, Chicago, IL: Free Press 1951.

Pavlov I, *Lectures on conditioned reflexes: Twenty-five years of objective study of the higher nervous activity (behaviour) of animals*, (W Gantt, Trans). Liverwright Publishing Corporation, 1928.

Pérez-Crespo J, Lobato-Cañón R, Solanes-Puchol Á, Multiple Chemical Sensitivity in Chemical Laboratory Workers, *Safety and health at work*, 2018, 9, 4, 473-8.

Perlin M, I've Got My Mind Made Up!: How Judicial Teleology in Cases Involving Biologically Based Evidence Violates Therapeutic Jurisprudence (March 9, 2017). NYLS Legal Studies Research Paper No. 2930061. <http://dx.doi.org/10.2139/ssrn.2930061>

Phillips T, Debating the legitimacy of a contested environmental illness: a case study of multiple chemical sensitivities (MCS), *Sociology of health & illness*, 2010, 32, 7, 1026-40.

Phillips T, *Law, Environmental Illness and Medical Uncertainty*, Abingdon, Routledge, 2015.

Phillips T, Rees T, (In)Visibility online: the benefits of online patient forums for people with a hidden illness: The case of multiple chemical sensitivity (MCS), *Medical Anthropology Quarterly*, 2017, 32, 2, 214–32.

Picard E, Carvalho F, Agosti F, Bourinet E, Ardid D, Eschalier A, Daulhac L, Mallet C, Inhibition of Cav3. 2 calcium channels: A new target for colonic hypersensitivity associated with low-grade inflammation, *British J Pharmacology*, 2019, 176, 7, 950-63.

Powers A, Diabetes mellitus. In: Kasper D, Fauci A, Longo D, Braunwald E, Hauser S, eds. *Harrison's principles of internal medicine*, New York: McGraw Hill, 2005, 2152-80.

Primack J, von Hippel F, *Advice and Dissent: Scientists in the Political Arena*, Basic Books, New York, 1974.

Ram V, A Deterministic and probabilistic analyses of the carbon tetrachloride contaminant plume in groundwater at the former union carbide india limited factory in Bhopal, Madhya Pradesh, India" (Thesis). Rollins School of Public Health, 2012.

Ramsden J, Contaminated aircraft cabin air: aspects of causation and acceptable risk, *J Biol Phys Chem*, 2012, 12, 2, 56–68.

Randolph T, Allergic type reactions to industrial solvents and liquid fuels, allergic type reactions to mosquito abatement fogs and mists, allergic type reactions to motor exhaust, allergic type reactions to indoor utility gas and oil fumes, allergic type reactions to chemical additives of foods and drugs, *J Lab Clin Med*, 1954, 44, 6, 910-14.

Rappaport S, Smith M, Epidemiology: Environment and disease risks, *Science*, 2010, 330, 6003, 460-1.

Rea W, Didriksen N, Simon T, Pan Y, Fenyves E, Griffiths B, Effects of toxic exposure to molds and mycotoxins in building-related illnesses, *Arch Env Health*, 2004, 58, 7, 399–405.

Rea W, Johnson A, Ross G, Butler J, Fenyves E, Griffiths B, Laseter J, Considerations for the diagnosis of chemical sensitivity, multiple chemical sensitivities (workshop), National Research Council, Division on Earth and Life Studies, Commission on Life Sciences, National Academies Press, Washington 1992, 169-192 <https://www.nap.edu/read/1988/chapter/18> accessed 3 Apr 2018.

Reichard A, Gulley S, Rasch E, Chan L, Diagnosis isn't enough: Understanding the connections between high health care utilization, chronic conditions and disabilities among U.S. working age adults, *Disabil Health J*, 2015, 8, 535–46.

Reif D, et al., Genetic basis for adverse events after smallpox vaccination, *J Infect Dis*, 2008, 198, 16–22.

Reuben S, Reducing environmental cancer risk, what we can do now; 2008–2009 Annual Report. US Department of Health and Human Services, National Institutes of Health, National Cancer Institute: Rockville, MD, USA, 2010.

Roe S, We tested popular cell phones for radiofrequency radiation. Now the FCC is investigating, Chicago Tribune, 21 Aug 2019 <https://www.chicagotribune.com/investigations/ct-cell-phone-radiation-testing-20190821-72ggu4nzd5kyuhteieih4da-story.html> accessed 21 Sep 2019.

Rocha O, Ansari K, Doohan F, Effects of trichothecene mycotoxins on eukaryotic cells: a review, Food Additives and Contaminants, 2005, 22, 4, 369-78.

Rodericks J, Calculated risks, the toxicity and human health risks of chemicals in our environment, Cambridge University Press, 1992, 57.

Rogers W, Miller C, Bunegin L, A rat model of neurobehavioral sensitization to toluene, Toxicol Ind Health, 1999, 15, 356–69.

Romanucci-Ross L, Moerman E, Tancredi L, Modern medicine. social structure and ritual in biomedicine, The Anthropology of Medicine, From Culture to Method, New York: Bergin & Garvey 1991, 368

Romanucci-Ross L, & Tancredi L, When law and medicine meet: A cultural view, New York, 2007, NY: Springer Science and Business Media.

Romanucci-Ross L, Moerman D, The Extraneous Factor in Western Medicine. In: Romanucci-Ross L, Moerman D, Tancredi L (eds), The Anthropology of Medicine: From Culture to Method, New York 1991, Bergin & Garvey, 351-69.

Röösli M, Mohler E, Frei P, Sense and sensibility in the context of radiofrequency electromagnetic field exposure, C R physique, 2010, 11, 576–84.

Rosenthal T, Majeroni B, Pretorius R, Malik K, Fatigue: an overview, American family physician, 2008, 78, 10, 1173-80.

Rosenworcel J, Choosing the Wrong Lane in the Race to 5G, Wired, Business, 10 June 2019. <https://www.wired.com/story/choosing-the-wrong-lane-in-the-race-to-5g/> accessed 22 Sep 2019.

Rossi J, Sensitization induced by kindling and kindling-related phenomena as a model for multiple chemical sensitivity, Toxicology, 1996, 111, 87–100.

Rossi S, Pitidis A, Multiple chemical sensitivity: review of the state of the art in epidemiology, diagnosis, and future perspectives, J Occ Env Med, 2018, 60, 2, 138-46.

SAH: South Australian Health, Government of South Australia, Multiple Chemical Sensitivity (MCS) / Idiopathic Environmental Intolerance (IEI) <https://www.sahealth.sa.gov.au/wps/wcm/connect/public+content/sa+health+internet/health+topics/health+conditions+prevention+and+treatment/chemicals+and+contaminants/multiple+chemical+sensitivity+idiopathic+environmental+intolerance> accessed 18 Oct 2019.

Saito M, Kumano H, Yoshiuchi K, et al.. Symptom profile of multiple chemical sensitivity in actual life, Psychosom Med, 2005, 67, 2, 318–25.

Sass, J. The Delay Game: How the Chemical Industry Ducks Regulation of the Most Toxic Substances; Natural Resources Defense Council: New York, NY, USA, 2011.

Santini R, Seigne M, Bonhomme-Faivre L, Bouffet S, Defrasme E, et al., Symptoms experienced by users of digital cellular phones: a study of a French engineering school, *Electromagn Biol Med*, 2002, 21, 1, 81–8.

Santini R, Santini P, Le Ruz P, Danze J, Seigne M, Survey study of people living in the vicinity of cellular phone base, *Electromagn Biol Med*, 2003, 22, 1, 41–9.

Scarry E, *The Body in Pain: The Making and Unmaking of the World*. Oxford University Press, 1988.

Schafer M, On the history of the concept neurasthenia and its modern variants chronic-fatigue-syndrome, fibromyalgia and multiple chemical sensitivities, *Fortschr, Neurol Psychiatr*, 2002, 70, 570-82.

Schnakenberg E, Fabig K, Stanulla M, Strobl N, Lustig M, Fabig N, Schloot W, A cross-sectional study of self-reported chemical-related sensitivity is associated with gene variants of drug-metabolizing enzymes, *Env Health*, 2007, 6, 1, 6-16.

Schröttner J, Leitgeb N, Sensitivity to electricity–temporal changes in Austria, *BMC Public Health*, 2008, 8, 1, 310.

Schur P, *Chronic Fatigue Syndrome (and Chronic Fatigue)*, *Neurorheumatology*, Springer, Cham, 2019, 233-42.

Seifert F, Maihöfner C, Functional and structural imaging of pain-induced neuroplasticity, *Curr Opin Anaesthesiol*, 2011, 24, 515–23.

Sekar A, Biala A, de Rivera H, Davis A, Hammond T, Kamitaki N, Tooley K, Presumey J, Baum M, Van Doren V, Genovese G, Rose S, Handsaker R, Schizophrenia Working Group of the Psychiatric Genomics Consortium, Daly M, Carroll M, Stevens B, McCarroll S, Schizophrenia risk from complex variation of complement component 4, *Nature*, Jan 27, 2016.

Sellers C, To Place or Not to Place: Toward an Environmental History of Modern Medicine, *Bulletin of the History of Medicine*, 2018, 92, 1, 1-45.

Shahin S, Singh S, Chaturvedi C, Mobile phone (1800 MHz) radiation impairs female reproduction in mice, *Mus musculus*, through stress induced inhibition of ovarian and uterine activity, *Reprod Toxicol [Internet]*, 2017, 73, 41–60.

Shuren J, FDA Statement: Statement from Jeffrey Shuren, M.D., J.D., Director of the FDA’s Center for Devices and Radiological Health on the National Toxicology Program’s report on radiofrequency energy exposure, November 01, 2018 <https://www.fda.gov/news-events/press-announcements/statement-jeffrey-shuren-md-jd-director-fdas-center-devices-and-radiological-health-national> accessed 22 Sep 2019.

Shaini K, 30 September 2008, No takers for Bhopal toxic waste, BBC. Archived from the original on 3 December 2009. Retrieved 1 January 2010.

Shrivastava P, *Bhopal Anatomy of a Crisis*, Cambridge, Ballinger, 1987.

Siegel S, Kreutzer R, Pavlovian conditioning and multiple chemical sensitivity, *Env Health Persp*, 1997, 105, (Suppl 2), 521.

Simko M, Cell type specific redox status is responsible for diverse electromagnetic field effects, *Curr Med Chem*, 2007, 14, 10, 1141–52.

Skovbjerg S, Christensen K, Ebstrup J, Linneberg A, Zachariae R, Elberling J, Negative affect is associated with development and persistence of chemical intolerance: a prospective population-based study, *J psychosomatic research*, 2015, 78, 509-14.

Skovbjerg S, Rasmussen A, Zachariae R, Schmidt L, Lund R, Elberling J, The association between Idiopathic environmental intolerance and psychological distress, and the influence of social support and recent major life events, *Env Health Prev Med*, 2012, 17, 2-9.

Skovbjerg S, Andersson L, et al., Inflammatory mediator profiling of n-butanol exposed upper airways in individuals with multiple chemical sensitivity, *PLoS One*, 2015, 10, 11.

Smart C, *Feminism and the Power of Law*, London, Routledge, 1989.

Smith M, et al., Benzene exposure and risk of non-Hodgkin lymphoma. *Cancer Epidemiol Biomarkers Prev*, 2007, 16, 3, 385-91.

Smith R, Medical Journals are an extension of the marketing arm of pharmaceutical companies, *PLoS Med*, 2005, 2, e138.

Snow J, On the origin of the recent outbreak of cholera at West Ham, *BMJ*, 1857, 1, 934–35.

SRRATRC 2000: Air Safety and Cabin Air Quality in the BAe 146 Aircraft, Parliament of the Commonwealth of Australia.

Standford, <http://web.stanford.edu/group/cpima/education/EDKIT/other%20lessons/Outgassing.pdf>: 20 Apr 16.

Stedman's medical dictionary: pathology, Philadelphia, Williams & Wilkins, 1995, 1312.

Steinemann A, National Prevalence and Effects of Multiple Chemical Sensitivities, *J Occ Env Med*, 2018a, 60, 3, 152–56.

Steinemann A, Prevalence and effects of multiple chemical sensitivities in Australia, *Preventive Medicine Reports*, 2018b, 10, 191-94.

Steinemann A, International prevalence of fragrance sensitivity, *Air Quality, Atmosphere & Health*, 2019a, 1-7.

Steinemann A, International prevalence of chemical sensitivity, co-prevalences with asthma and autism, and effects from fragranced consumer products, *Air Quality, Atmosphere & Health*, 2019b,12, 5, 519-27.

Straub R, Cutolo M, Buttgerit F, Pongratz G, Energy regulation and neuroendocrine-immune control in chronic inflammatory diseases, *J Intern Med*, 2010, 267, 6, 543–60.

Subhan F, Khan A, Ahmed S, Malik M, Bakshah S, Tahir S, Mobile antenna's and its impact on human health, *J Med Imaging and Health Informatics*, 2018, 8, 6, 1266-73.

Swisscom: Patent WO2004075583A1 Reduction of electrosmog in wireless local networks filed by Swisscom Ag 2003 <https://patents.google.com/patent/WO2004075583A1/en>

Szumilas M, Explaining odds ratios. *J Can Acad Child Adolesc Psychiatry*. 2010, 19, 3, 227-9. Erratum in: *J Can Acad Child Adolesc Psychiatry*, 2015, 24, 1, 58.

Tang J, Zhang Y, Yang L, Chen Q, Tan L, Zuo S, Feng H, Chen Z, Zhu G, Exposure to 900 MHz electromagnetic fields activates the mcp-1/ERK pathway and causes blood-brain barrier damage and cognitive impairment in rats, *Brain research*, 2015, 1601, 92-101.

- Terry A, Functional consequences of repeated organophosphate exposure: potential non-cholinergic mechanisms, *Pharmacol Ther*, 2012, 134, 3, 355–65.
- Tishelman C, Sachs L, The Diagnostic Process and the Boundaries of Normal, *Qualitative Health Research*, 1998, 8, 1, 48-60.
- Trabacchi V, Riccò M, Pasquarella C, Signorelli C. Sindrome da sensibilità chimica multipla: studio di una popolazione di giovani adulti, *Ig Sanità Pubbl*, 2009, 65, 363-76.
- Treichler P, Cartwright L, Penley C, eds. *The visible woman: Imaging technologies, gender, and science*, NYU Press, 1998.
- Tseng M, Prevalence and psychiatric comorbidity of self-reported electromagnetic field sensitivity in Taiwan: A population-based study, *J Formosan Med Assoc*, 2011, 110.
- Türedi S, Hancı H, Çolakoğlu S, Kaya H, Odacı E, Disruption of the ovarian follicle reservoir of prepubertal rats following prenatal exposure to a continuous 900-MHz electromagnetic field, *Int j radiation biology*, 2016, 92, 6, 329-37.
- Tuuminen T, Rinne K, Severe sequelae to mold-related illness as demonstrated in two finnish cohorts, *Frontiers in immunology*, 2017, 8, 382.
- UNEP: United Nations Environment Programme. *Global Chemicals Outlook. Towards Sound Management of Chemicals*, Geneva, Switzerland, 2012.
- UoT: University of Toronto, Environmental health and safety, Occupational Hygiene & Safety, Guidelines on the Use of Perfumes and Scented Products, What is the issue <https://ehs.utoronto.ca/our-services/occupational-hygiene-safety/guidelines-on-the-use-of-perfumes-and-scented-products/> accessed 7 Mar 2019.
- USDOD: US Department of Defense, Active Denial Technology, <https://jnlwp.defense.gov/Future-Non-Lethal-Weapons/Active-Denial-Technology/> Accessed 22 Sep 2019.
- US Torts: RESTATEMENT (THIRD) OF TORTS: Phys. & Emot. Harm § 28, cmt. c (4), 2010.
- Vakas N, Interests and the shaping of an occupational health and safety controversy: the BAe 146 case, PhD thesis, School of Social Sciences, Media and Communication, University of Wollongong, 2007, Chap 7, 175-241. <http://ro.uow.edu.au/theses/76>
- Van den Bergh O, Devriese S, Winters W, et al., Acquiring symptoms in response to odors: a learning perspective on multiple chemical sensitivity, *Ann N Y Acad Sci*, 2001, 933, 278–90.
- Van den Bergh O, Brown R, Petersen S, Witthöft M, Idiopathic environmental intolerance: a comprehensive model, *Clinical Psychological Science*, 2017, 5, 3, 551-67.
- Van der Wall E, Fraud and Freud: is there an association in scientific misconduct? *Neth Heart J*, 2012, 20, 2, 49–50. Published online 2012 Jan 5.
- Van Diest I, De Peuter S, Piedfort K, Bresseleers J, Devriese S, Van de Woestijne K, Van den Bergh O, Acquired Lightheadedness in response to odors after Hyperventilation, *Psychosomatic Medicine*, 2006, 68, 340-47.
- Van Houdenhove B, Fibromyalgia: a challenge for modern medicine, *Clin Rheumatol*, 2003, 22, 1-5.
- Van Thriel C, Kieswetter E, Schäper M, Blaszkewicz J, Kleinbeck S, Odor annoyance of environmental chemicals: sensory and cognitive influences, *J Toxicol Env Health*, 2008, part A, 71, 11-12, 776-85.

- Vetter P, Rossi L, Edwards C, Mold illness: surviving and thriving: a recovery manual for patients and families impacted by CIRS, BookBaby, 2018 Jan 30.
- Vlaanderen J, et al., Occupational benzene exposure and the risk of lymphoma subtypes: a metaanalysis of cohort studies incorporating three study quality dimensions, *Env Health Persp*, 2011, 119, 2, 159–16.
- Wacquant L, Punishing the poor: the neoliberal government of social insecurity, Durham, Duke University Press, 2009.
- Wallace D, Clauw D, eds. Fibromyalgia and other central pain syndromes, Lippincott Williams & Wilkins, 2005.
- Wang Q, Cao Z, Qu Y, Peng X, Guo S, Chen L, Residential exposure to 50 hz magnetic fields and the association with miscarriage risk: A 2-year prospective cohort study, *Plos One*, 2013, 8, 12, e82113.
- Warnke U, Hensinger P, Steigende, “Burn-out“-Inzidenz durch technisch erzeugte magnetische und elektromagnetische Felder des Mobil- und Kommunikationsfunks, *umwelt-medizingesellschaft* 2013, 26, 1, 31–8.
- Wazana A, Physicians and the pharmaceutical industry, is a gift ever just a gift? *JAMA*, 2000, 283, 3, 373-80.
- Weiss B, Landigan P, The developing brain and the environment, *Env Health Persp*, 2000, 373-4.
- Weiss E, Singewald E, Baldus C, Hofer E, Marksteiner J, Nasrouei S, Ruepp B, Kapfhammer HP, Fitz W, Mai C, Bauer A, Differences in psychological and somatic symptom cluster score profiles between subjects with idiopathic environmental intolerance, major depression and schizophrenia, *Psychiatry research*, 2017, 249, 187-94.
- Welshons W, Thayer K, Judy B, Taylor J, Curran E, vom Saal F, Large Effects from Small Exposures. I. Mechanisms for Endocrine-Disrupting Chemicals with Estrogenic Activity, *Env Health Persp*, 2003, 111, 994–1006.
- Wertheimer N, Leeper E, Electrical wiring configurations and childhood cancer, *Am j epidemiology*, 1979, 109, 3, 273-84.
- West J, Kapoor N, Liao S, Chen J, Bailey L, et al., Multifocal breast cancer in young women with prolonged contact between their breasts and their cellular phones, *Case Rep Med*, 2013, 354682.
- WGRACP, Working Group of the Royal Australasian College of Physicians Chronic fatigue syndrome, Clinical practice guidelines—2002, *Med J Aust*, 2002, 176, Suppl, S23-56.
- Whaley P, Systematic Review and the Future of Evidence in Chemicals Policy (Report); Policy from Science Project, Lancaster University, Lancaster, UK, 2013.
- Whitehead W, Palsson O, Jones K, Systematic review of the comorbidity of irritable bowel syndrome with other disorders: what are the causes and implications? *Gastroenterology*, 2002, 122, 1140-56.
- WHO: World Health Organization. Electromagnetic fields and public health: Electromagnetic hypersensitivity, Backgrounder, December 2005 <https://www.who.int/peh-emf/publications/facts/fs296/en/> accessed 1 Oct 2019.
- WHO: World Health Organization. Preventing Disease through Healthy Environments. Towards an Estimate of the Environmental Burden of Disease, Geneva, Switzerland, 2006.

- Willis E, White K, The Challenge of Evidence Based Medicine, In: Tovey P, Adams J, Easthope (eds) The mainstreaming of complementary and alternative medicine, London, Routledge, 2003, 49-63.
- Winters W, Devriese S, Van Diest I, Nemery B, Veulemans H, Eelen P, Van de Woestijne K, Van den Bergh O, Devriese S, Media warnings about environmental pollution facilitate the acquisition of symptoms in response to chemical substances, *Psychomatic Medicine*, 2003, 65, 332-38.
- Wolkoff P, Crump D, Harrison P, Pollutant exposures and health symptoms in aircrew and office workers: Is there a link? *Environ Int*, 2016, 87, 74–84.
- Woodley J, The politics of aircraft health and safety, *J Occ Health & Safety – Aust & NZ*, 2005, 21, 5, 401-07.
- Woolf C, Evidence for a central component of post-injury pain hypersensitivity, *Nature*, 1983, 306, 686-88.
- WorkCover Western Australia, 2007, Scheme Development Branch, Report on Common Law Proceedings in Western Australia. Available online at: www.workcover.wa.gov.au/NR/rdonlyres/3B32BD59-4440-4BA8-854A-608F369479E6/0/Common_Law_Proc_WA_0406.pdf accessed 10 August 2009.
- Wright J, Clarke D, Cabin crew syndrome: a case study – acute and chronic symptoms, ASHRAE Aviation Subcommittee, 1999.
- Yakymenko I, Tsybulin O, Sidorik E, Henshel D, Kyrylenko O, et al., Oxidative mechanisms of biological activity of low intensity radiofrequency radiation, *Electromagn Biol Med*, 2015, 19, 1–16.
- Yuan J, Liu Y, Wang J, Zhao Y, Li K, Jing Y, Zhang X, Liu Q, Geng X, Li G, Wang F, Long-term persistent organic pollutants exposure induced telomere dysfunction and senescence-associated secretary phenotype, *J Gerontology: Series A*, 2018 Jan 19.
- Yunus M, Fibromyalgia and overlapping disorders: the unifying concept of central sensitivity syndromes, *Semin Arthritis Rheum*, 2007, 36, 339–56.
- Yunus M, Central sensitivity syndromes: a new paradigm and group nosology for fibromyalgia and overlapping conditions, and the related issue of disease versus illness, *Semin Arthritis Rheum*, 2008, 37, 339-52.
- Yunus M, Suffering, science and sabotage, *J Musculoskele Pain*, 2004, 12, 3-18.
- Zhou L, Zhang H, Lan Y, et al., Epidemiological investigation of risk factors of the pregnant women with early spontaneous abortion in Beijing, *Chin J Integr Med*, 2017, 23, 345–49.
- Zomkowski A, Engel D, Gabilan N, Rodrigues A, Involvement of NMDA receptors and l-arginine-nitric oxide-cyclic guanosine monophosphate pathway in the antidepressant-like effects of escitalopram in the forced swimming test, *European Neuropsychopharmacology*, 2010, 20, 11, 793–801.
- Zucco G, Militello C, Doty R, Discriminating between organic and psychological determinants of multiple chemical sensitivity: a case study, *Neurocase*, 2008, 14, 6, 485-93.

APPENDIX 1: THE MCS MECHANISM HYPOTHESIS BY PALL AND ITS OVERLAPS

The MCS mechanism hypotheses overlap with observed effects of environmental xenochemical exposures present in many studies. To illustrate this, one of these proposed mechanisms, by Pall, is outlined here as well as overlapping research.

As will be seen below, peroxynitrite (ONOO-) is postulated by Martin Pall to be a key player in the MCS exposure mechanism. In 2007, prominent scientists from the US National Institute of Health identified this chemical as a key factor in more than 60 chronic diseases through its chemical disruption, cytotoxic effects and tissue damage (Pacher 2007). These diseases include neurodegenerative disorders, IBS, kidney disease, heart disease, Alzheimer's, vascular disease, inflammatory disease, accelerated aging, hypertension, arthritis, liver disease, cancer, stroke, MS and diabetes.

Pall's pathway proposal seems to have been preceded by observations with genetically modified mice with enhanced NMDA receptors being used in Alzheimer's Disease research (Crumpler 2014). NMDA is a receptor for glutamate which is a primary excitatory neurotransmitter. As NMDA is known to be a key factor in the formation of memory, hence intelligence, the mice were unusually smart. But what the researchers also noticed was that the mice felt pain more acutely than normal. The researchers went on to determine whether this was because the mice remembered the pain. They concluded that they were sensitised to pain.

It is believed by many psychologists that the most basic form of learning is in experiencing pain. Increasing sensitivity with each exposure is normal in such learning processes (Crumpler 2014). This is reinforced with neurons in the spinal cord becoming hypersensitive to sustained pain signals (Woolf 1983), thus lowering the pain threshold. Furthermore, as long-term memory is established by the brain creating permanent new branches and connections between neurons, the same can happen with pain-sensing neurons after severe or prolonged pain. So enhanced pain neural pathways can also be created as typical of the Central Sensitivity condition. The memory-pain duality and the enhanced neural connections have been suggested as a model for MCS (Crumpler 2002, Ji 2003). It was also found, separately, that NMDA receptor antagonists,

such as dextromethorphan and ketamine, administered shortly after exposure can block an MCS reaction (Donohoe 2008p101), adding additional support for such a model.

Dr Martin Pall (2000a,b,2007), argued that an initial chemical exposure (citing 7 chemical groups in particular) creates hypersensitivity in brain neurons which then react by increasing inflammatory cytokines, in turn increasing the levels of nitric oxide (NO), which then reacts with superoxide to form peroxynitrite, which in turn increases the cytokines, etc. The NO stimulates the release of glutamate and aspartate, stimulating NMDA receptors, which then further elevate NO levels causing a dramatic sensitisation of neural pathways. The increased peroxynitrite can also damage cell mitochondria, initiating inflammations (Meggs 1993) and damage DNA leading to later genotoxic effects cultivating an ideal environment biologically, for disease (Rappaport 2010). Increased chemical access to the brain occurs via peroxynitrite breaking down the blood brain barrier. NO and peroxynitrite can interact with the regulatory system governing synthesis of porphyrin biosynthetic enzymes: reduced enzymes can then lead to disruption of porphyrin metabolism, accounting for many of the simultaneous MCS symptoms (Donohoe 2008p107). NO also inhibits cytochrome P450 activity, a detoxification pathway (Donohoe 2008p107). A complex mechanism results for the MCS condition due to the clear possibility of synergism between these reactions.

It is interesting that MCS, CFS, FM, PTSD are often treated via muscular injections of B12 in the form of hydroxocobalamin which is a potent nitric oxide scavenger.

There is much awareness in the chemical and drug industry of xenochemicals affecting NMDA receptors. For example in a recent patent (Cheruvu 2018) a variety of xenochemicals were intended to be used for the treatment of neurodegenerative diseases via their effects on NMDA receptors.

A more recent study on suppressed redox defences incorporating substances referred to above (De Luca 2010a), and another documenting polymorphism of CCK-B in MCS patients (Joffres 2005), further develop the proposed MCS mechanism. Other studies reinforce that many neurological conditions, such as depression, are related to NMDA receptors and NO (Zomkowski 2010, Dhir 2011). Recently research has related elevated nitrosine, a peroxynitrate marker, as a possible indicator for MCS (Belpomme 2015).

Over the same period studies have identified genes that produce substances involved in the metabolism of organic solvents and related compounds, the metabolism of pesticides, and that influence susceptibility (Haley 1999, McKeown-Eyssen 2004, Schnakenberg 2007, Müller 2008, Loria-Kohen 2017). Four of these genes particularly help determine susceptibility (Pall 2012). Pall asserted that there is only one way that all these genes could be involved, namely where a chemical from an exposure would act as a toxicant. Enzymes that influence the metabolism of such toxicants then determine how sensitive the individual is in their chemical sensitivity. For example, one of these enzymes, also a gene, MTHFR, has long been recognised as important for clearing environmental chemicals via the methylation process. Testing for MTHFR deficiency has been used for testing the risk in reacting to adjuvant chemicals in vaccines (Lynch 2012, Reif 2008, HFW 2016), although the simple test is rarely used by doctors prior to vaccine injections.

The research by Marshall-Gradisnik and Staines on cellular ion channels has been claimed as a “key unifying factor underlying the bewildering variety of symptoms” (Ludlam 2018). As virtually every cell in the body has these channels, it becomes a unifying factor for a variety of symptoms. Staines pointed out “We now have a tangible nexus between causality at onset, gene changes, receptor changes and clinical consequences. The implications are major, not only to develop a suitable test and pharmacological treatments, but also to show how interventions such as exercise therapy and behavioural therapies are wrong in science and in physiological terms, potentially harmful” (Ludlam 2018).

An example of the undone science areas in respect to mechanisms, is the relationship between the TRAP1 molecular mechanism and calcium channel blockers (Picard 2019). Some MCS sufferers have found that by taking calcium channel blockers, progression into the inflammatory stage is stopped (Schur 2019), although dependent on exposures, especially if alcohol is involved. It remains to be seen whether calcium blockers are treating the symptoms and exacerbating the underlying cause (sensitisation), similar to antidepressants and other antipsychotics which sensitise the NDMA subunit.

REFERENCES FOR APPENDIX 1

Belpomme D, Campagnac C, Irigaray P, Reliable disease biomarkers characterizing and identifying electrohypersensitivity and multiple chemical sensitivity as two etiopathogenic aspects of a unique pathological disorder, *Rev Environ Health*, 2015, 30, 4, 251–71.

Cheruvu P, Kishnani K, Lewis B, Piscitelli S, Ramaswamy S, Weinhoff G, Asare E, inventors; Axovant Sciences GmbH, assignee. Compositions and methods of treating a neurodegenerative disease. United States patent application US 15/677,837, 2018 Feb 15.

Crumpler D, *Prostituting Science: The Psychologisation of MCS, CFS and EHS for political gain*, Maryborough, VIC; Inkling Australia, 2014.

Crumpler D, MCS and the molecules of memory: a point to ponder, *Sensitivity Matters*, 2002, 34, 17-22.

De Luca C, Scordo M, Cesareo E, et al., Biological definition of multiple chemical sensitivity from redox state and cytokine profiling and not from polymorphisms of xenobiotic-metabolizing enzymes, *Tox App pharma*, 2010a, 238, 285-92.

Dhir A, Kulkarni S, Nitric oxide and major depression, *Nitric Oxide*, 2011, 24, 125–31.

Donohoe M, *Killing Us Softly*, Internet Creative Commons release 2008.

Haley R, Billecke S, La Du B, Association of low PON1 type Q (type A) arylesterase activity with neurologic symptom complexes in Gulf War veterans, *Toxicol Appl Pharmacol*, 1999, 157, 227-33.

HFW: Homeopathy for Women. MTHFR. Available: <http://www.homeopathyforwomen.org/MTHFR.htm>. Accessed 21 Aug 2016.

Ji R, Kohno T, Moore K, Woolf C, Central sensitisation and LTP: do pain and memory share similar mechanisms? *Trends in Neuroscience*, 2003, 28, 12, 696-705.

Joffres M, Sampalli T, Fox R, Physiologic and symptomatic responses to low-level substances in individuals with and without chemical sensitivities: a randomised controlled blinded pilot booth study, *Env Health Persp*, 2005, 113, 9, 1178-83.

Lynch B, MTHFR and Vaccines/Immunizations – Contraindicated? Available: MTHFR.net, <http://mthfr.net/forums/topic/mthfr-and-vaccinesimmunizations-contraindicated/>. Accessed 7 Mar 2012.

McKeown-Eyssen G, Baines C, Cole D, Riley N, Tyndale R, Marshall L, Jazmaji V, Case-control study of genotypes in multiple chemical sensitivity: CYP2D6, NAT1, NAT2, PON1, PON2 and MTHFR, *Int J Epidemiol*, 2004, 33, 971-78.

Meggs W, Neurogenic inflammation and sensitivity to environmental chemicals, *Env Health Persp*, 1993, 101, 3, 234-238.

Müller K, Schnakenberg E, Die Bedeutung der Glukuronidierung bei umweltmedizinischen Erkrankungen am Beispiel der UDP-Glukuronosyltransferase 1A1, *Umwelt Medizin Gesellschaft* 2008, 21, 4, 295-300.

Loria-Kohen V, Marcos-Pasero H, de la Iglesia R, Aguilar-Aguilar E, Espinosa-Salinas I, Herranz J, de Molina AR, Reglero G, Multiple chemical sensitivity: genotypic characterization, nutritional status and quality of life in 52 patients, *Medicina Clínica (English Edition)*, 2017, 149, 4, 141-6.

Ludlam S, To the Millions Missing with ME/CFS, something remarkable is happening, *The Guardian*, 12 May 2018 https://www.theguardian.com/commentisfree/2018/may/12/to-the-millionsmissing-with-mecfs-something-remarkable-is-happening?CMP=share_btn_fb accessed 17 Jul 2019.

Pacher P, Beckman J, Liaudet L, Nitric oxide and peroxynitrite in health and disease, *Physiol Rev* 2007, 87, 1, 315–424.

Pall M, Elevated sustained peroxynitrite levels as the cause of chronic fatigue syndrome, *Medical Hypotheses*, 2000b, 54, 115-25.

Pall M, Elevated peroxynitrite as the cause of chronic fatigue syndrome. Other inducers and mechanisms of symptom generation, *J Chronic Fatigue Syndrome*, 2000a, 7, 4, 139-45.

Pall M, Explaining “Unexplained Illnesses”: disease paradigm for chronic fatigue syndrome, multiple chemical sensitivity, fibromyalgia, post-traumatic stress disorder, Gulf War Syndrome, and others, New York, NY (US), London (GB), Harrington Park Press/Haworth Press, 2007.

Pall M, Multiple Chemical Sensitivity: Toxicological and Sensitivity Mechanisms, 2012: Available: http://bobnorson.com/pubs/Pall_english.pdf. Accessed 5 Nov 2015

Picard E, Carvalho F, Agosti F, Bourinet E, Ardid D, Eschalier A, Daulhac L, Mallet C, Inhibition of Cav3. 2 calcium channels: A new target for colonic hypersensitivity associated with low-grade inflammation, *British J Pharmacology*, 2019, 176, 7, 950-63.

Schnakenberg E, Fabig K, Stanulla M, Strobl N, Lustig M, Fabig N, Schloot W, A cross-sectional study of self-reported chemical-related sensitivity is associated with gene variants of drug-metabolizing enzymes, *Env Health*, 2007, 6, 1, 6-16.

Schur P, Chronic Fatigue Syndrome (and Chronic Fatigue), *Neurorheumatology*, Springer, Cham, 2019, 233-42.

Rappaport S, Smith M, *Epidemiology: Environment and disease risks*, Science, 2010, 330, 6003, 460-1.

Reif D, et al., Genetic basis for adverse events after smallpox vaccination, *J Infect Dis*, 2008, 198, 16–22.

Zomkowski A, Engel D, Gabilan N, Rodrigues A, Involvement of NMDA receptors and l-arginine-nitric oxide-cyclic guanosine monophosphate pathway in the antidepressant-like effects of escitalopram in the forced swimming test, *European Neuropsychopharmacology*, 2010, 20, 11, 793–801.

APPENDIX 2: SOME TECHNICAL DETAILS ON CIRS

In addition to regular mould in buildings, CIRS can also be developed from toxins in a “brown recluse spider bite, from tropical fish contaminated with ciguatera toxin, from Pfiesteria and cyanobacteria (from blue-green algae), and from Borrellia Burgdorferi, the organism responsible for Lyme disease. Water damaged buildings are responsible for 80% of CIRS” (Vetter 2018p24). The frequency of mould illness susceptible sufferers is approximately 25% of the general population (Vetter 2018).

CIRS is a legitimate medical diagnosis with lab tests and physical findings that can substantiate the diagnosis. Symptoms include fatigue, weakness, aches, muscle cramps, unusual pains, difficulty learning new information, confusion, ice pick pains, poor focus and concentration, morning sickness, headache, palpitations, light sensitivity, tearing, cough, shortness of breath, diarrhea, difficulty memory problems, skin sensitivity, mood swings, anxiety, depression, headache, abdominal pain, gastrointestinal problems, appetite swings, sweats (especially at night), poor temperature regulation, excessive thirst, increased urination, static shocks, numbness, tingling, vertigo, metallic taste, tremor, fatigue, angioedema, neurocognitive symptoms, myalgia, arthralgia, joint pain, sinus problems, insomnia, dizziness, red eyes, blurred vision, irritability, tremors, balance disturbance, vasculitis, and autonomic nervous system dysfunction (Vetter 2018, Curtis 2004, Rea 2004). New onset chemical sensitivity can also occur from such exposures (Rea 2004). Thyroid, immune dysfunction and autoimmune conditions are often being found in persons exposed to water-damaged buildings (Gray 2004). Sufferers from mould and mycotoxins can exhibit neurologic disorders such as delirium, dementia, pain syndromes, and movement, balance and coordination disorders (Empting 2009).

The consequences of mould exposure and toxicity, frequently unrecognised, may initiate hormonal imbalances, chronic gastrointestinal issues, brain disrepair and multiple autoimmune conditions. Sufferers can be diagnosed with illnesses such as MCS, multiple sclerosis, fibromyalgia, chronic fatigue syndrome, and depression.

A simple test for the condition is the Visual Contrast Sensitivity Test. It measures one of the neurological vision functions known as ‘contrast’. Toxins affect nerve cell function and so also

impair the ability to detect subtle contrast within 24 to 36 hours after exposure. The test can even be done on-line (Vetter 2018). Visual contrast sensitivity, has been a key factor in determining and tracking the potent effects of toxins that have been introduced to the human body (Hudnell 2001, Shoemaker 2001, Thomas 2010, NYS 2010).

A HLA-DR blood test is also used to detect genetic susceptibility which is consistent with CIRS. A lack of HLA-DR genes mean that the immune system cannot form antigens to deal with the toxins hence they remain in the body and accumulate causing repeated disruption/damage. A person not having such genetic susceptibility can more effectively create antigens to toxins such that when they are re-encountered, such as re-entering a contaminated building, antigens can act on the toxins to clear it out of the body: an acquired immune response by which it is broken down and eliminated via the liver. But as well as the genetic make-up, nature and severity of exposure determines the extent to which an individual will be affected (England 2012). The immune system itself may be affected by the exposure leading to various illnesses mentioned later (Al-Anati 2006, Montagnoli 2006).

More than 95% of CIRS sufferers have a low melanocyte stimulating hormone level which also can be checked. Transforming Growth Factor Beta-1 and an inflammatory marker, C4a, will both increase in the presence of biotoxins and decrease when the exposures are removed. Matrix Metalloproteinase 9, Vascular Endothelial Growth Factor, Vasoactive Intestinal Polypeptide, and Lipase levels are also affected in CIRS (Vetter 2018).

The biotoxins in the body initiate complex biochemical events. They bind to receptors (such as Toll receptors) in most cells. This biotoxin binding causes a stimulation of multiple inflammatory pathways, including production of cytokines. Cytokines (inflammatory proteins in the blood) can also bind to cell receptors resulting in the release of MMP9 into the blood. Cytokines in the brain bind to leptin receptors, inhibiting its normal function and reduce melanocyte stimulating hormone (MSH) production. Symptoms such as unstable temperature, muscle ache, headache, and difficulty concentrating can be caused by elevated cytokines. Increased clot formation and blockage of circulation can result from MMP9's effect of transferring inflammatory elements from the blood to sensitive tissues.

The HLA genotypes (genes related to immunity) mentioned above can develop various immune responses. These can include gluten sensitivity, autoimmune issues and blood clotting

problems. The complement system is chronically activated resulting in high C4a levels. The spiralling inflammation increase affects most bodily systems, including the brain. Cytokine elevated levels, such as with the MMP-9 and TGFB-1, as well as the decreased MSH, can cause “leaky junctions” in both the gut and the blood brain barrier. Neuropathic toxins can therefore enter the brain, causing brain inflammation and dysregulation of the hypothalamus and pituitary.

With 95% of CIRS sufferers having low MSH levels, it is a reason for so many other hormones being affected, the inflammation, and many of the immune system problems. Reduced MSH results in decreased melatonin levels producing non-restorative sleep and causing chronic fatigue. Chronic and debilitating pain is also caused by the endorphin production being suppressed. White blood cells are also affected losing normal cytokine response allowing infections to establish as slowness for recovery. Reduced MSH also allows antibiotic-resistant staph bacteria (MARCoNS) to establish on mucous membranes. This bacteria produces toxins that destroy MSH producing a downward health spiral (Vetter 2018).

Low MSH can result in low levels of antidiuretic hormones, due to lower pituitary production, which can cause thirst, frequent urination, low blood pressure (especially on standing), low blood volume, and electric shocks from static electricity. Sex hormone production is also lowered. The pituitary may up-regulate the production of cortisol and adrenocorticotrophic hormone (ACTH) in early illness stages, later dropping to low ranges (Vetter 2018).

Decreased VEGF (vascular endothelial growth factor) results from high levels of cytokines in capillaries. This attracts white blood cells which leads to restricted blood flow (hypo-perfusion) and low levels of oxygen in the tissues. This leads to fatigue, muscle cramps, reduced exercise tolerance and shortness of breath. This overlaps into Dr Moulden’s hypotheses on effects of toxins in vaccines. Oxidative stress can be a significant mechanism of injury (Bouslimi 2008, Sava 2007, Larsen 2001, Cavin 2009, Zhang 2011, Roberts 2009). Exposure to water-damaged environments can trigger inflammation which then plays a significant role in illness during and after (Islam 2007, Jussila 2002, Cremer 2012).

In respect to neurocognitive effects, one study measured IQ scores in children exposed to indoor mould showed IQ deficits of approximately 10 points (Jedrychowski 2011). Also, a longer exposure tripled the risk for low IQ scores below the 25th percentile. This corresponds with

other studies with low scores on cognitive testing (Gordon 2004, Kilburn 2004, Baldo 2002, Crago 2003). Various mycotoxins are neurotoxic in animal studies including mycotoxin ochratoxin A (OTA), fumonisin, macrocyclic trichothecene and T2 toxin (Doi 2011). Satratoxin H, at levels found in water-damaged buildings, can result in neurological system cell damage. Depression has also been shown to result from damp indoor environments (Shenassa 2007). Neuroinflammation has been demonstrated in children and dogs from outdoor air pollution (Calderón-Garcidueñas 2008) with undone science in respect to indoor air toxins.

There are various neurotoxic effects linked to the indoor pollutants such as neuronal degeneration in the cerebral cortex induced from fumonisin B1 (FB1), brain neuronal cell apoptosis induced by the T2 toxin, striatal dopamine depletion, and its metabolites, from OTA, and neuronal cell apoptosis and inflammation in the olfactory epithelium and bulb from macrocyclic trichothecenes (Doi 2011).

A study of fungal growth at levels occurring from water damaged buildings showed that the trichothecene mycotoxin, satratoxin A, could cause neurological system damage (Karunasena 2010). As proposed by Janette Hope “it is believed that the constant activation of inflammatory and apoptotic pathways in human brain capillary endothelial cells, astrocytes, and neural progenitor cells can amplify the devastation of neurological tissues and lead to neurological system cell damage from indirect events triggered by the presence of trichothecenes” (Hope 2013, Karunasena 2010).

People exposed to mould and mycotoxins in indoor settings have exhibited abnormalities in quantitative EEG (QEEG) studies (Crago 2003) and single-photon emission computed tomography (SPECT) scans (Rea 2004, Ross 1999).

The group of mycotoxins, the Trichothecenes, are very toxic and have been used as biological warfare agents (Zajtchuk 1997). The inhibition of protein synthesis seems to be the main cause of the toxicity (Rocha 2005). Some multiorgan effects include emesis and diarrhea, weight loss, skin toxicity, nervous disorders, hemostatic derangements, decreased reproductive capacity, cardiovascular alterations, immunodepression, and bone marrow damage (Zajtchuk 1997).

It has been established that allergic response to mould is usually not IgE mediated. In one study 33.1% of the asthma sufferers were atopic to environmental antigens and 20% had sensitivity

to mould allergens, showing that mechanisms were involved other than for type 1 allergy (Karvala 2010). One estimate attributes 21% of asthma in the US to mould and dampness exposure (Mudarri 2007). Exposure to mould odours in their home increased the risk of childhood asthma by a factor of 2.4 (Jaakkola 2005), and occupational exposure to mould raised the risk of new-onset asthma 4.6 times (Jaakkola 2006).

In the chapter on ignorance and environmental effects on health, mention is made of the steadily increasing levels of ionising radiation in the general living environment: from medical applications of various scanners, through to heavy metals in the atmosphere. It becomes relevant that there are now many observations and studies (Dadachova 2008) that show how fungal growths are enhanced by such radiation and becomes particularly relevant to people living near nuclear facilities, coal power stations, mining areas, etc.

A significant unresearched area is mycotoxin contamination of the food and feed supply (Dohlman 2003). In very low concentrations, difficult to detect, mycotoxins can profoundly affect the endocrine system and contribute sub-clinically to chronic health conditions. On investigating one of these mycotoxins, zearalenone, it was found in the urine of a 9 to 10 year old sample of girls and related to being shorter in height and less likely to have reached the onset of breast development (Bandera 2011). The researchers were able to find an association between the young girls' urinary levels of the mycotoxin and their intake of commonly contaminated sources such as beef and popcorn. Derivatives of this mycotoxin have also been patented as oral contraceptives. It is intentionally used as a fattening agent for cattle and was banned in Europe after being linked to premature puberty. Isolated studies dating back decades ago showed estrogenic effects in pigs such as uterine enlargement, swelling of the vulva and mammary glands, and pseudopregnancy (Mirocha 1979). Effects from such mycotoxins can be more significant than other toxins such as DDT and BPA in similar concentrations (Akiyama 1997, Kuiper 1998). There are more than 40 other mycotoxins identified by various countries as needing studies on their health effects (van Egmond 2007).

REFERENCES FOR APPENDIX 2

Akiyama H, Toyoda M, Kato M, Igimi S, Kumagai S, The degradation of several mycotoxins by human intestinal microflora cultured by continuous flow culture system, *Mycotoxins*, 1997, 44, 21-27.

Al-Anati L, Petzinger E, Immunotoxic activity of Ochratoxin A, *J Vet Pharm Therapeutics*, 2006, 29, 2, 79–90.

Baldo J, Ahmad L, Ruff R, Neuropsychological performance of patients following mold exposure, *Applied Neuropsychology*, 2002, 9, 4, 193–202.

Bandera E, Chandran U, Buckley B, Lin Y, Isukapalli S, Marshall I, King M, Zarbl H, Urinary mycoestrogens, body size and breast development in New Jersey girls, *Sci Total Environ*, 2011, 409, 24, 5221-7.

Boulimi A, Ouannes Z, Golli E, Bouaziz C, Hassen W, Bacha H, Cytotoxicity and oxidative damage in kidney cells exposed to the mycotoxins Ochratoxin A and citrinin: individual and combined effects, *Toxicology Mechanisms and Methods*, 2008, 18, 4, 341–49.

Calderón-Garcidueñas L, Mora-Tiscareño A, Ontiveros E, et al., Air pollution, cognitive deficits and brain abnormalities: a pilot study with children and dogs, *Brain and Cognition*, 2008, 68, 2, 117–27.

Cavin C, Delatour T, Marin-Kuan M, et al., Ochratoxin A—mediated DNA and protein damage: roles of nitrosative and oxidative stresses, *Toxicological Sciences*, 2009, 110, 1, 84–94.

Crago B, Gray M, Nelson L, Davis M, Arnold L, Thrasher J, Psychological, neuropsychological, and electrocortical effects of mixed mold exposure, *Arch Env Health*, 2003, 58, 8, 452–63.

Cremer B, Soja A, Sauer J, Damm M, Pro-inflammatory effects of ochratoxin A on nasal epithelial cells, *European Archives of Oto-Rhino-Laryngology*, 2012, 269, 4, 1155–61.

Curtis L, Lieberman A, Adverse health effects of indoor molds, *J Nutr Env Med*, 2004, 14, 3, 261–74.

Dadachova E, Casadevall A, Ionizing radiation: how fungi cope, adapt, and exploit with the help of melanin, *Current opinion in microbiology*, 2008, 11, 6, 525–31.

Dohlman E, Mycotoxin Hazards and Regulations: Impacts on Food and Animal Feed Crop Trade, chapter 6, *International Trade and Food Safety: Economic Theory and Case Studies*. J. Buzby (ed.), USDA, Econ Res Serv, AER-828, Nov 2003, 97-108.

Doi K, Uetsuka K, Mechanisms of mycotoxin-induced neurotoxicity through oxidative stress-associated pathways, *Int J Molecular Sciences*, 2011, 12, 8, 5213–37.

Empting K, Neurologic and neuropsychiatric syndrome features of mold and mycotoxin exposure, *Tox Ind Health*, 2009, 25, 9-10, 577–81.

England A, Valdes A, Slater-Jefferies J, et al., Variants in the genes encoding TNF- α , IL-10, and GSTP1 influence the effect of α -tocopherol on inflammatory cell responses in healthy men, *Am J Clinical Nutr*, 2012, 95, 6, 1461–67.

Gordon W, Cantor E, Johanning E, et al., Cognitive impairment associated with toxigenic fungal exposure: a replication and extension of previous findings, *Applied Neuropsychology*, 2004, 11, 2, 65–74.

Gray M, Thrasher J, Crago R, et al., Mixed mold mycotoxicosis: immunological changes in humans following exposure in water-damaged buildings, *Archives of Environmental Health*, 2004, 58, 7, 410–20.

Hudnell H, House D, Schmid J, Koltai D, Stopford W, Wilkins J, Savitz D, Swinker M, Music S, Human visual function in the North Carolina clinical study on possible estuary-associated syndrome, *J Toxicol Env Health A*, 2001, 62, 8, 575-94.

Islam Z, Amuzie C, Harkema J, Pestka J, Neurotoxicity and inflammation in the nasal airways of mice exposed to the macrocyclic trichothecene mycotoxin roridin A: kinetics and potentiation by bacterial lipopolysaccharide coexposure, *Toxicological Sciences*, 2007, 98, 2, 526–41.

- Jaakkola J, Jeromnimon A, Jaakkola M, Interior surface materials and asthma in adults: a population-based incident case-control study, *American Journal of Epidemiology*, 2006, 164, 8, 742–49.
- Jaakkola J, Hwang B, Jaakkola N, Home dampness and molds, parental atopy, and asthma in childhood: a six-year population-based cohort study, *Env Health Persp*, 2005, 113, 3, 357–61.
- Jedrychowski W, Maugeri U, Perera F, et al., Cognitive function of 6-year old children exposed to mold-contaminated homes in early postnatal period. Prospective birth cohort study in Poland, *Physiology & Behavior*, 2011, 104, 5, 989–95.
- Jussila J, Komulainen H, Kosma V, Nevalainen A, Pelkonen J, Hirvonen M, Spores of *Aspergillus versicolor* isolated from indoor air of a moisture-damaged building provoke acute inflammation in mouse lungs, *Inhalation Toxicology*, 2002, 14, 12, 1261–77.
- Karunasena E, Larrañaga M, Simoni J, Douglas D, Straus D, Building-associated neurological damage modeled in human cells: a mechanism of neurotoxic effects by exposure to mycotoxins in the indoor environment, *Mycopathologia*, 2010, 170, 6, 377–90.
- Karvala K, Toskala E, Luukkonen R, Lappalainen S, Uitti J, Nordman H, New-onset adult asthma in relation to damp and moldy workplaces, *Int Arch Occ Env Health*, 2010, 83, 8, 855–65.
- Kilburn K, Indoor mold exposure associated with neurobehavioral and pulmonary impairment: a preliminary report, *Arch Env Health*, 2004, 58, 7, 390–98.
- Kuiper G, Lemmen J, Carlsson B, Corton J, Safe S, van der Saag P, van der Burg B, Gustafsson J, Interaction of estrogenic chemicals and phytoestrogens with estrogen receptor beta, *Endocrinology*, 1998, 139, 10, 4252–63.
- Larsen T, Svendsen A, Smedsgaard J, Biochemical characterization of Ochratoxin A-producing strains of the genus *Penicillium*, *App Env Microbiology*, 2001, 67, 8, 3630–35.
- Mirocha C, Schauerhamer B, Christensen C, Niku-Paavola M, Nummi M, Incidence of zearalenol (*Fusarium* mycotoxin) in animal feed, *Applied env microbiology*, 1979, 38, 4, 749–50.
- Montagnoli C, Fallarino F, Gaziano R, et al., Immunity and tolerance to *Aspergillus* involve functionally distinct regulatory T cells and tryptophan catabolism, *J Immunology*, 2006, 176, 3, 1712–23.
- Mudarri D, Fisk W, Public health and economic impact of dampness and mold, *Indoor Air*, 2007, 17, 3, 226–35.
- NYS: New York State Department of Health Center For Environmental Health Bureau Of Toxic Substance Assessment. Tetrachloroethylene (Perc) Exposure And Visual Contrast Sensitivity (VCS) Test Performance In Adults And Children Residing In Buildings With Or Without A Dry Cleaner, March 2010.
- Rea W, Didriksen N, Simon T, Pan Y, Fenyves E, Griffiths B, Effects of toxic exposure to molds and mycotoxins in building-related illnesses, *Arch Env Health*, 2004, 58, 7, 399–405.
- Roberts R, Laskin D, Smith C, et al., Nitrate and oxidative stress in toxicology and disease, *Toxicological Sciences*, 2009, 112, 1, 4–16.
- Ross G, Rea W, Johnson A, Hickey D, Simon T, Neurotoxicity in single photon emission computed tomography brain scans of patients reporting chemical sensitivities, *Toxicology and Industrial Health*, 1999, 15, 3–4, 415–20.
- Sava V, Velasquez A, Song S, Sanchez-Ramos J, Adult hippocampal neural stem/progenitor cells in vitro are vulnerable to the mycotoxin ochratoxin-A, *Toxicological Sciences*, 2007, 98, 1, 187–97.

Shenassa E, Daskalakis C, Liebhaber A, Braubach M, Brown M, Dampness and mold in the home and depression: an examination of mold-related illness and perceived control of one's home as possible depression pathways, *Am J Public Health*, 2007, 97, 10, 1893–99.

Shoemaker R, Hudnell K, Possible estuary associated syndrome: symptoms, vision, and treatment. *Env Health Persp*, 2001, 109, 539-45.

Thomas G, Clark Burton N, Mueller C, Page E, Comparison of mold exposures, work related symptoms, and visual contrast sensitivity between employees at a severely water-damaged school and employees at a school without significant water damage, Department Of Health And Human Services, Centers for Disease Control and Prevention, Sept 2010.

van Egmond H, Schothorst R, Jonker M, Regulations relating to mycotoxins in food: perspectives in a global and European context, *Anal Bioanal Chem*, 2007, 389, 1, 147-57.

Vetter P, Rossi L, Edwards C, *Mold illness: surviving and thriving: a recovery manual for patients and families impacted by CIRIS*, BookBaby, 2018 Jan 30.

Zajtchuk R, "Medical aspects," in *medical aspects of chemical and biological warfare*, Office of The Surgeon General at TMM Publications, Washington, DC, 1997.

Zhang L, Ye Y, An Y, Tian Y, Wang Y, Tang H, Systems responses of rats to aflatoxin B1 exposure revealed with metabonomic changes in multiple biological matrices, *J Proteome Research*, 2011, 10, 2, 614–23.

Chapter 4

Ignorance Concerning Environmental Effects on Health

Contents

Chapter 4.....	320
Ignorance Concerning Environmental Effects on Health.....	320
INTRODUCTION.....	321
IGNORANCE (NON-KNOWLEDGE) & UNDONE SCIENCE	322
Ionising Radiation.....	333
Sunscreens	341
THE RECOGNITION OF IGNORANCE.....	344
Overconfidence.....	345
Salience	345
Vulnerability.....	345
Information Processing and Storage Psychology.....	348
MANUFACTURED IGNORANCE.....	351
Roundup Herbicide: Glyphosate Active Ingredient	354
Phenylpropanolamine (PPA)	357
Bisphenol A (BPA)	358
Other Examples Summarised.....	359
The Most-Common Manufactured Ignorance Attitudes on Health Issues from Environmental Exposures.....	365
SELECTIVE IGNORANCE (STRATEGIC OR STRUCTURED IGNORANCE).....	365
Rational Selective Ignorance.....	372
AGGREGATED IGNORANCE	380
Consequences of Aggregated Ignorance	382
Localised Environmental Disasters	384
ORGANISATIONAL IGNORANCE	386
Unknown Unknowns.....	387
Known Unknowns	388
Knowable Known Unknowns	389
Ignorance About Existing Knowledge and from Suppressing Knowledge	389

Secrecy	390
CRIMINAL IGNORANCE	393
Pluralistic Ignorance.....	396
Denial Through the Presence of Power	397
Large Organisation Obedience.....	398
State Denial.....	399
Official Acknowledgement.....	399
State-Corporate Collaboration and Corruption	400
EPISTEMOLOGY, IGNORANCE AND TOXICOLOGY.....	401
Honeybee Colony Collapse Disorder (CCD).....	402
Absolute Certainty in Health Research Studies	406
BIOMEDICAL ADVERSE-EVENT UNDER-REPORTING	410
THE NEED FOR EVERGREEN SCIENCE POLICY ILLUSTRATED BY CALCITONIN HORMONE	412
CONCLUSION.....	413
REFERENCES.....	423

INTRODUCTION

Previous chapters have inquired into whether environmental exposures can help explain the myriad of health problems present in modern society, particularly chronic diseases and this was highlighted through the case study of multiple chemical sensitivity (MCS). Compelling evidence demonstrates that environmental exposures can indeed be linked to the many health conditions considered. Exposures in these cases are frequently at a level that is conventionally considered insignificant, and the connection is therefore disregarded and not pursued.

In chapter 2, research into the available science around the environmental effects on health was reviewed to ascertain the current state of knowledge and awareness. This revealed a significant number of studies and broad awareness of the situation, and contrasts with the lack of rigour and science applied by regulatory authorities in general.

In chapter 3, MCS was considered as potentially representing a “canary in the coal mine” given a significant portion of the population reacts to environmental exposures in modern society. While current exposure levels do not affect the majority of the population, up to 28% are

mildly to severely affected. The condition is generally dismissed by the orthodox medical system as a psychological problem. An inquiry into two common psychological conditions which possibly have environmental causes -- depression and ADHD -- can be found in Appendix 1. This inquiry found many links with environmental exposures and that diagnosis of such conditions is very subjective. The inconsistencies in recognising these psychological conditions, as compared with MCS, were highlighted.

The preceding chapters have highlighted that a great deal of ignorance is present in society in regard to environmental effects on health. Unfortunately, this seems to be due to the vested interest of industrial parties that stand to make a great deal of money from the status quo. This chapter therefore looks at the ignorance around this issue, and how the creation and maintenance of that ignorance becomes the reason why the medical and health systems, and the public generally, dismiss environmental effects on human health.

This chapter introduces the concept of “undone science” to explain the large amount of research that is missing on this subject. In considering why this is so significant, various forms of ignorance are discussed, using examples that illustrate industry’s complicit role in undone science generation. Regulatory capture of government departments by industry will also become apparent as the discussion progresses. As generating ignorance involves the manipulation of knowledge and relates to typical aspects of human psychology, these will be discussed as contributing factors in the next chapter.

Through its coverage of undone science and ignorance, this chapter will illustrate that the situation of environmental exposures causing health effects (as shown in other chapters) is due to industry’s use of ignorance. In the following chapter the resultant paradigmatic situation in the orthodox-medical and regulatory systems, prevents the widespread recognition of the environmental effects on health.

IGNORANCE (NON-KNOWLEDGE) & UNDONE SCIENCE

List of Terms Used

Aggregated Ignorance: This term is introduced in this thesis due to its relevance. It is bidirectionally used by industry and government departments depending on the problem. On

one hand results or statistics are aggregated together to dilute problem areas/results with non-problem areas/results. On the other hand, it may become advantageous not to aggregate effects, through similar chemical properties or possible synergism when commenting on exposures to a number of toxins. It is deemed better to consider each toxin on its own in order to progressively dismiss any possible health effects. This is fully covered in a section so-titled later in this chapter.

Conscious Ignorance: This form of ignorance occurs in undone science where certain knowledge areas have not been researched, however, it is acknowledged that such areas have not been adequately researched. It is a term used by Wilholt, described simply by him as “what we know we don’t know” (Wilholt 2019 fig8.1). It is the equivalent of “known unknowns” used by Gross (2010).

Criminal Ignorance: There are many studies mentioned in this thesis on the many problems with industrial products such as pesticides, fragrances, food additives, cosmetics, drugs, etc. The blatant disregard shown by regulatory departments to the number of people who have been adversely affected amounts to criminal ignorance. If an individual exposed another to known toxic substances then the perpetrator would be jailed. But government departments mostly allow industry to continue, without actions taken that they are responsible to take. It amounts to a criminal action (or inaction). Theil (2015) introduced this ignorance classification.

Inadvertent Ignorance: This is where a body of knowledge is overlooked by a person who has no idea that it exists (Friedman 2005).

Intentional Ignorance: As the name suggests, intentional ignorance is intentionally created, as in double-blind randomised controlled trials (Kaptchuk 1998).

Irrational Ignorance: This ignorance occurs when an individual avoids information that may be counter to their beliefs or goals (Taber 2006). There is a section on this type of ignorance later in this chapter.

Knowable Known Unknown: This occurs where something is knowable if sufficient resources, or even motivation, are allocated, rather than remaining simply as a known unknown. This is a form of rational ignorance (Roberts 2015p362).

Known Unknowns: This is another name for conscious ignorance as used by Wilholt (2019). It is where there is knowledge about limits to knowledge: there are aspects that we know we have no knowledge about. There is a section on this type of ignorance later in this chapter.

Manufactured Ignorance: “Ignorance can be made or unmade and science can be complicit in either process” (Proctor 2008p3). From such viewpoints ignorance can be an active production: a manufactured ignorance. In the large section in this chapter on manufactured ignorance various examples will be given, e.g., the tobacco industry’s efforts to keep selling their products while mounting evidence of cancer links grew.

Manufacture of Uncertainty: This is a key component of manufactured ignorance. It is where companies deliberately generate uncertainty as to the correctness of detrimental findings on their product. This will be referred to through the experience of Kleinman and Suryanarayanan (2015p183).

Nescience: This is what cannot be known in advance. M. Gross’s explanation: “In the seventeenth century, nescience was sometimes regarded as what cannot be known in advance and sometimes as God’s knowledge (which people cannot know and must not attempt to know). In some streams of philosophy of the nineteenth century, this led to agnosticism, a doctrine that claims that nothing is actually knowable.... nescience belongs to a fundamentally different epistemic class from nonknowledge, ignorance, or terms such as Kerwin’s known unknowns . No one can refer to their own current nescience because it is not part of their conscious (and thus socially constructed) nonknowledge. At most, people can refer to someone else’s or their own earlier nescience. Hence, a sociological observer can ascribe nescience only in retrospect. Accordingly, I prefer the more neutral notion of nescience , which in its etymology and epistemology already clearly indicates its difference from terms such as unknown ignorance or unknown unknowns” (Gross 2010 p55).

Opaque Ignorance: This type of ignorance is a state of total absence of knowledge and there is no awareness of such ignorance or simply unknown unknowns. It has been recognised for some time and has been termed opaque ignorance by Wilholt (2019).

Organisational Ignorance: This form of ignorance occurs mainly in government and industry organisations. To be effective organisations each must be able to manage the various facets of organisational ignorance, such as unknown unknowns, known unknowns, knowable known unknowns, ignorance about existing knowledge, ignorance from suppressing knowledge and secrecy. This form of ignorance has a major section written on it later in this chapter

Pluralistic Ignorance: This refers to group individual members believing that the group holds certain norms and values, whereas in reality no individual member holds them. Alternatively, individual members believe they hold beliefs different to everyone else, but in fact others do share them. It was first written about by Schanck (1932) and Matza (1964) in delinquency.

Rational Ignorance: Somin (2015p274) makes the following definition:

A person is rationally ignorant whenever he or she has decided not to learn some body of knowledge because the costs of doing so exceed the benefits, based on the decision-maker's own objectives. Finding, studying, and assimilating information is a costly activity, not just in terms of money, but in terms of time and effort as well.

Selective ignorance: (also known as strategic or structured ignorance). This form of ignorance occurs when specific information on a topic is produced but there is a failure to produce other information on the topic. In its simplest form selective ignorance could arise from clear social decisions to fund one area of research and not another. It has been written about by many authors including Elliott (2015) and Hess (2016). A major section on this type of ignorance appears later in this chapter.

Undone Science: This can be classified as known unknowns or conscious ignorance, although in researching areas of undone science, nescience (knowledge we discover as a surprise) can result (Gross 2010). Apart from representing simply an absence of knowledge, undone science can represent a structured absence (Hess 2016) due to social inequality which determines which areas of research are to be funded.

unk unk: Short for unknown unknowns. In many companies dealing with high-risk research and development, such as in aerospace, an awareness has long been realised and sometimes referred to as "unk unks" (Longstaff 2005).

Importance is usually given to epistemology (the study of how we know) however what and why we don't know may be as important. While we know a lot about knowledge, it is frequent that little is known about ignorance. It is not necessarily a bad thing or just a void. To want to know everything all the time is not a typical human trait. Furthermore, it is normal for us to know something that we would rather others did not know. Privacy rights are a type of sanctioned ignorance. Juries, for example, are required to be kept ignorant (producing balance) as some knowledge would produce bias. A virtuous ignorance would exist in relation to dangerous knowledge. Examples would include the societal desire for ignorance on how to make explosives, or where are the most vulnerable parts of a passenger plane or a city's water supply. People regularly decide on when and when-not to know, for example, parents not wanting to know their unborn child's gender, not spoiling the ending of a movie or game result before watching.

Trust enables a tolerance of ignorance. Specialisation is a social ignorance arrangement (Smithson 2008) since it arises from an acknowledgement that there is too much for one person to learn or know everything. The use of intentional ignorance in double-blind randomised controlled trials is the cornerstone of experimental objectivity (Kaptchuk 1998).

Many references to ignorance are negative, for example metaphorically, ignorance is blindness, knowledge is to see. This then leads to knowledge being power and ignorance being helplessness, uncertainty, or impotence. As a legitimating influence, ignorance can be used to justify inaction and status quo maintenance to the extent of even guilt being required to be established beyond reasonable doubt in criminal cases. Proctor & Schiebinger (2008p24) looking at courtroom dynamics put forward that knowledge "is interestingly attached to bias, ignorance to balance."

The production of ignorance and knowledge may go hand in hand with science. In pursuing certain lines of research both knowledge and ignorance result, for example, researching cancer detection and treatment rather than researching its causation and prevention.

Kevin Elliott's (2015) 'selective' ignorance can occur from the collection of specific sorts of information on a product, its benefits for example, rather than others such as harmful side

effects. Selective ignorance can also occur from how information is presented to the public or other entities, for example, the pharmaceutical industry undergoes significant “publication planning” to control the information flow so that certain decision makers receive exactly the information that will ensure best results for their products (Sismondo 2007). These approaches highlight the money, power and social influence involved in issuing and collecting information. Examples of selective ignorance are more closely considered in a following section.

Knowledge can be produced as a result of recognising ignorance in certain areas. Socrates expressed that those who realise how little they know are more wise: a precondition for enlightenment is the recognition of one’s ignorance. If his students had discovered the extent of their ignorance it was a sign of a successful education as opposed to mere training (Ravetz 2015). The selective choice of ignorance is possible in enquiry. To focus on ‘this’ can be a decision to ignore ‘that’. However under the guise of learning knowledge to reduce one’s ignorance, a manipulation of people’s perception is also possible. One example of this was the misinformation about the Gulf War. One study found that people were misinformed on the subject proportionally as to the amount of TV viewing (Jhally 1991). Even radio had been early-criticised as a propaganda vehicle: “spreading ignorance, was often said” (Proctor 2008). This leads to the possibility of ignorance not being a simple omission or gap but rather that it can be an active production. There can be a strategic advantage in knowing when to cultivate ignorance (Anderson 2010, McGoey 2012a)

It could be said that science originates and is required due to the proliferation of ignorance. The desire for knowledge is frequently regarded as the main driver for scientific research and work in general. The perception that science is always an open process is challenged when one considers ignorance. This is due to secrecy playing an important role in science, for example in peer review, the guarding of discoveries until an opportune time, confidential industrial research, etc. It has been said that “Ignorance can be made or unmade and science can be complicit in either process” (Proctor 2008p3). From such viewpoints ignorance can be an active production: a manufactured ignorance. The classic example was the tobacco industry’s approach to the cancer link with their product. The industry has been described as “... the masters of fomenting ignorance to combat knowledge. Health fears are assuaged by reassurances in the form of ‘reasonable doubt’ – a state of mind with both PR and legal value.” (Proctor 2008p11). Other examples of manufactured ignorance are present with striking

similarities in their approaches. Many examples of manufactured ignorance can be found in respect to pharmaceutical drugs. Peter Gøtzsche's book, *Deadly Medicines and Organised Crime – How Big Pharma has Corrupted Healthcare*, is full of well researched examples. While detrimental effects on health from drugs can occur by similar mechanisms to environmental toxins, drug effects are not covered in this thesis. These books illustrate how ignorance, through undone science, can be utilised by large pharmaceutical corporations to make huge profits yet severe health problems and deaths of thousands of people do and can occur.

Often in the news one sees companies accused of malfeasance for defending their products before serious problems were found. Falsified or hidden data is attributed in keeping the public and regulators ignorant of the issue. There are many similar stories of people or other companies being threatened economically, or being influenced with money, physical threats, through to actual harm. These actions sometimes border on being illegal through to criminal. They frequently form the basis of books and films such as Erin Brockovitch (2000) and The Insider (1999).

David Michaels in his book *Doubt is their product* shows this is the case for industry which find their interests threatened. Chris Mooney argues the strategy is employed by conservatives in political battles in science (Mooney 2005).

As observed by David Magnus (2008p251), "This story has become so common that we know it by heart: industry and its politically conservative allies oppose science-based regulation and support the creation of uncertainty to protect their interests".

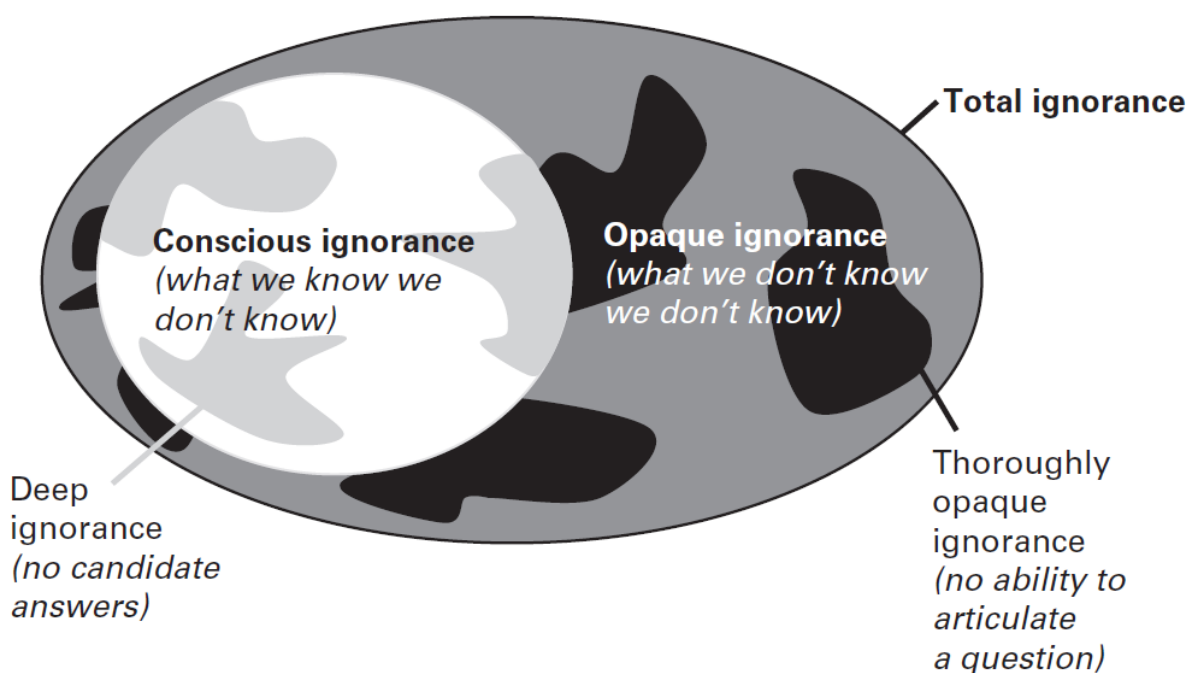
Experience by Daniel Kleinman and Sainath Suryanarayanan (2015p183) shows: "The manufacture of uncertainty is a central strategy of corporations seeking to advance their interests. Companies may bolster uncertainty in an effort to keep a profitable product on the market or to protect themselves from liability in the case of an ostensibly dangerous good. The stories of companies hiding data, falsifying evidence, or selectively using findings to advance their interests are legend".

In the following subsections different types of ignorance applicable to this thesis subject will be covered and illustrated. The coverage will begin with a discussion on undone science and the known unknowns, then progress with how ignorance can be recognised. Only the main forms

of ignorance involved in this issue will be addressed, with examples: manufactured, selective, aggregated, organisational, and criminal ignorance. The examples are brief and refer to areas of ignorance and are not by any means a full coverage of each issue. The ignorance categorisations are general since they are not distinct but significantly overlap.

Undone science results from an unequal power situation linked with absent knowledge involved in conflicts involving reformers, like leaders of social movements, and the political and industrial elites. It is a situation where reformers who advocate for the broader public interest, look to “science” for answers but find a lack of research compared to industry adversaries who have funded research to support their position (Hess 2007). Undone science refers to this systematic production of non-knowledge due to the unequal distribution of power in society. This will be seen in the first example on radiation.

Within agnotology, undone science can be classified as known unknowns or conscious ignorance, although in researching areas of undone science, nescience (knowledge we discover as a surprise) can result (Gross 2010). Some writers term nescience as deep ignorance as seen in the below diagram by Torsten Wilholt. Social movements can view future research areas as positive non-knowledge whereas industry may view them as negative non-knowledge. The distinction is perspectival.



The above figure represents the landscape of ignorance from Torsten Wilholt (2019 fig8.1). Discussion of the opaque area (unknown unknowns) is present in the Organisational Ignorance section further on.

Undone science typically involves a systematic underfunding of certain research areas. Those few scientists that do research in the underfunded areas can experience various industry backed reactions to their work. There is some documentation of the active suppression of these scientists who may find risks and dangers with new technologies or those involving public dispute (Delborne 2008, Martin 1999a,b).

There are however complexities that add to these undone science areas. Although undone science can involve known unknowns this does not mean that it is a matter of simply performing the science to achieve knowledge in these areas. It may not be possible to get the undone science done. This is particularly the case highlighted by this thesis in looking at the lack of scientific studies considering the effects on humans from trillions of combinations of low level xenochemical exposures from modern environment situations. Not only are there combinations with possible potentiation and synergism, but these affect thousands of complex human biochemical reactions occurring in the body. Then there are the added complications of a large percentage of the population already being health-compromised before exposures. Hence such areas may be undone but also undoable (Frickel 2010). The barriers to undoable science are frequently technical and financial but may be potentially overcome in the long run. Recognition of undone science areas should have effects on risk assessments and the application of the precautionary approach until the science has been done. An undoable science situation however should turn a temporary precautionary policy into a permanent one.

If there are social benefits in not pursuing a line of research, social movements can create undone science (Frickel 2010, Kempner 2005). This can be seen in examples of animal rights research.

Reformers and social movements can at times be divided and point to different undone science areas (Frickel 2010). For example, in considering the effects of a particular xenochemical some reformers are concerned about exposure of pregnant mothers and effects on the foetus while industrial workplace reformers may be more concerned about effects of the particular chemical

on adults in the short and medium term. The typical problem is that large civil society organisations have the financial capacity to fund research to get undone science done than the less-funded social movement organisation (Hess 2009).

Conflicts have occurred over decades between social movements and the government and/or industry on the credibility of the basis on which assessment of risk is made on existing or emerging technologies or products. Frequently there is a large amount of undone science involved on risks and potential harm. This can produce movements opposing industry advocating the termination of specific technologies and products (such as toxic chemicals) or whole industries such as nuclear power (King 2007, Weber 2009). Epistemic conflict can occur when scientists find and document risk from existing technologies: their work, however, is rebutted and attacked by industry-funded scientists. When communities affected by toxic exposures find ways to prove the exposure and consequences this can also result in epistemic conflict (Brown 1990, Kroll-Smith 2000, Ottinger 2013).

Broader epistemic conflict results also from how knowledge, or the lack of it, is translated into policy. Commonly, as in the case of GMOs, the government departments release products onto the market simply on the basis that it has not been proven to be unsafe (ignoring the undone science). When social movements demand a precautionary approach and the government does not accept such demands, street protests may call for a moratorium. In many instances a modest regulatory response can occur, such as labelling for GMOs or halting release of some nanomaterials. Industry may make concessions such as decreasing allergenic genes in some GM food. But where there is an absence of social movement mobilisation, regulatory policy usually follows the industry directions (Hess 2015).

When research occurs in the undone science area and identifies risks, in conjunction with criticising such research, the industry may also embark on disinformation campaigns. This is clearly illustrated in the coming section about the tobacco industry tactics. Another example is in climate research where the contrarian scientists, respected in their own fields but who have no standing in the climate science field, had led the criticism and mis-information campaigns for industry. This led to an editorial in the Wall Street Journal by climate scientist Kevin Trenberth (2012), co-signed by 37 other climate scientists. As David Hess (2015p149) summarised:

It compared skeptical statements by a group of prominent scientists to consulting a dentist for a condition that requires heart surgery. Credentialed in their own fields but not in the relevant field, the contra public scientists develop a claim that there is undone science, when in fact there is a relatively high level of consensus among credentialed experts in the research field. Although science is never done, there is enough consensus within the research field to make policy recommendations, such as the need to eliminate airborne emissions that cause acid rain, ozone depletion, and global warming.

In environmental effects on health issues, a frequent situation arises where the industry and governmental departments cite studies that illustrate their claims of little or no risk. At the same time they may even claim there is not enough research to substantiate any modification or regulatory action. So apart from representing simply an absence of knowledge, undone science can represent a structured absence (Hess 2016) due to social inequality which determines which areas of research are to be funded. Any analysis of undone science is perspectival due to the inherent conflicts of both sides of the importance of carrying out the involved research.

Although undone science is frequently viewed as being primarily due to the conflict between industry interests and the concerned public, there are other problems that can affect the production of undone science (Hess 2016).

- The complexity of defining the myriad of effects on the human organism, especially low level exposures over long terms, represents a barrier for achieving it within most political time frames.
- The opposition to animal research by some social movements can create undone science in health effects studies
- Where budgets are limited or claimed to be limited, the funding of certain areas means that other areas are not funded.
- There is usually a spectrum of approaches that can be taken. In the case of cancer, this would vary from the industry view of the research priorities being in areas of curative drugs, through to the influence of various lifestyles and the links between particular types of environmental exposures (from pollution, pesticides, food additives, etc.).

Undone science does draw attention to an epistemic dimension of all social movements. A group with a general focus of corruption and repression, for example, will not have scientific research details as being of high relevance to broad issues due to being a more-debateable aspect rather than say, clear cases of corruption. Yet for many social issues undone science can be a major contributor to one side, usually industry, making use of the resulting ignorance to argue their case.

Ionising Radiation

Health effects from ionising radiation in the environment is a large and complex area of study and is considered in this section in a sample form by looking at some aspects which show its disruptive potential to human health.

The increasing amount of ionising radioactive substances in the environment involves enormous public ignorance and undone science. There have been constant emissions of radioactive substances such as nuclear weapons testing, accidents at nuclear facilities (Three Mile Island, Chernobyl, Fukushima and others) and the authorised constant discharge of radioactive wastes from nuclear and other facilities.

The public perception is such that the well-known disasters are over and finished, yet in the case of Fukushima for instance, the water used to keep cooling the disaster site is now accumulating to more than 1.3 million tonnes in storage, with the power company wanting to dump it in the ocean due to a lack of storage space (McCurry 2019). The power company will lower the radiation levels and discharge through an underwater pipeline going 1km out to sea. The radiation levels will be reduced, by dilution, to the normal discharge allowable levels for nuclear facilities. This is simply the old approach of dilution being the solution. This was rubber stamped by the government in early 2021 (McCurry 2021) with no organised residue monitoring programme of fish or the general population, heavily reliant on seafood, announced as being part of the approval.



Above photo of now more than 1000 tanks at the Fukushima plant from the McCurry article in the Guardian Newspaper, 10 Sep 2019

While it is pertinent to realise both operating and failed nuclear power stations have constant emissions of radioactive material, coal fired power stations are also significant contributors. As well as the dioxins and heavy metals emitted into the atmosphere, frequently overlooked is their contribution to the environmental load of radioactive material. Although uranium and thorium are in trace amounts in coal, upon combustion in the power plants, they are concentrated up to 10 times (Hvistendahl 2007).

The manufactured and selective ignorance by industry and governments for the public on these issues is diverse. An example only quantified in recent years, after decades of occurrence, relates to nuclear facility monitoring. Usually the emissions are monitored by a one-point measurement each week or so. Such monitoring therefore cannot pick-up any variability in the amount of emissions between such measuring times. Although there have been many public concerns about abnormal health problems of communities in surrounding suburbs from nuclear facilities, there is significant ignorance in respect to the magnitude of public exposure to the radioactive particles. In 2011, the International Physicians for the Prevention of Nuclear War (IPPNW) in Germany released a press notice. For the first time it released emissions data measured every half hour from a nuclear power plant over long periods. The data showed that when reactors were refuelled every 12-18 months, there is a sharp spike in radioactive

emissions over a 12 hour period while the reactors were depressurised and opened up. The concentration of radioactive gases was up to 500 times more than usual during these periods producing about two-thirds of the facility's annual emissions. The public has never been warned about such invisible exposures around such plants. Dr Reinhold Thiel, a member of the German IPPNW Board said in the same publication (Fairlie 2014):

Especially at risk are unborn children. When reactors are open and releasing gases, pregnant women can incorporate much higher concentrations of radionuclides than at other times, mainly via respiration. Radioactive isotopes inhaled by the mother can reach the unborn child via blood with the result that the embryo/ fetus is contaminated by radioactive isotopes...This contamination could affect blood-forming cells in the bone marrow resulting later in leukemia. This provides a plausible explanation for the findings of the KiKK study published in 2008 that under-fives living near nuclear power plants are considerably more at risk of cancer, particularly leukemia, than children living further away.

Risk estimates from studies of leukaemia and cancer in people living near nuclear facilities range from no risk to risks of orders of magnitude more than for atomic bomb survivors. The nuclear power industry cannot provide any absolute protection to public health and is reflected by most governments providing the industry a no liability status. But the catastrophic potential for the public remains.

Although there are a myriad of contributors to a global build-up of radioactive materials, there is no doubt they must be accumulating, simply due to the half-lives of most of the contaminants, for example, radium: 1600 years, iodine 129: 15.7 million years, uranium 238: 4.5 billion years, thorium 232: 14 billion years. Yet there is little monitoring done, let alone studies on the effects of the resulting low-level background radiation. The stockpiles of radioactive waste are around 400 000 tonnes worldwide, with no firm plans formulated how to deal with them in the coming centuries. Many of the storage sites supposedly designed for 100-plus years have already leaked into groundwater, for example, the German nuclear waste dump in Asse. This is not to mention the millions of tonnes of radioactive particulates remaining in the biosphere from past testing, catastrophes and the allowed continual contamination from present facilities.

Typical industry downplays take the form of “Radioactivity is a part of our earth - it has existed all along. Naturally occurring radioactive materials are present in its crust, the floors and walls of our homes, schools, or offices and in the food we eat and drink. There are radioactive gases in the air we breathe. Our own bodies - muscles, bones, and tissue - contain naturally occurring radioactive elements. Man has always been exposed to natural radiation arising from the earth as well as from outside the earth” (IAEA 2019). Perhaps this is a self-fulfilling statement by industry.

Further ignorance can be present when talking about half-lives of radioactive substances. The substances do not just stop emitting but break down into other substances which have their own radioactive characteristics and a new set of half-lives. Uranium and thorium break down to radium, which emits almost 3000 times the radiation than uranium and thorium. Radium is present in drinking water in many parts of the US and is regarded as ‘natural’ by health authorities. However, as pointed out by one functional medicine practitioner during a discussion, in respect to human absorption “... the ionizing radiation will actually rip apart DNA, rip apart cells, creating lots of oxidative stress. So with that oxidative stress, your cells can’t function, you’re using up all your oxygen, you’re not making energy, it just destroys everything” (Watts 2019p8). With radium being like lead, fluoride, thallium and many other heavy metals in displacing calcium in bones, it can lodge itself in bone marrow which is intricately linked with the immune system. Furthermore radium can break down to radon gas (76000 years half-life) and so becomes breathable. Radon breaks down into radioactive lead, bismuth and polonium thereby making radon the second most potent cause of lung cancer globally after tobacco, as well as depositing such radioactive metals throughout the environment. Whilst it can be argued by industry that radon is ‘naturally occurring’ large-scale mining operations have increased releases by orders of magnitude into the environment than would otherwise have occurred ‘naturally’.

There are also paradigmatic belief systems concerning health effects of radioactive particles which are outside the scope of this thesis. If one considers two common radioactive particles found now in both the environment and humans, caesium 137 and uranium 238, these particles emit other types of radiation other than gamma: particularly beta particles. These particles can cause ionisation and genetic damage and so can potentially cause significant

damage, especially inside the body, but is a significant area of undone science. There is much debate on photoelectron induction effects from these particles, particularly depleted uranium used in weapons such as those in the gulf war as well as in significant stockpiles from nuclear facilities (Busby 2008, 2009). As uranium 238 has a half-life of 4.5 billion years this potential problem cannot easily go away. Radiation allowable standards are based on gamma radiation, mostly on a heating basis and so have no consideration for these effects which are now being realised but downplayed in the public arena (Pattison 2010). Some predict that the toxicity of these effects is orders of magnitude higher than the current orthodox model is saying.

The ionising radiation environmental contamination has been mostly distributed via the atmosphere due to its small particulate/gaseous nature. The world population has and is being exposed on a global basis rather than only localised situations from points of emission. This creates problems for conducting epidemiological studies on the health effects. The various other toxins and EMFs mentioned in this thesis, which also cause similar problems, make a scientific study of effects almost impossible. It is summed up by the following quote by Dr Sue Wareham (2019p4):

Firstly, health effects such as cancer due to radiation exposure often take decades to develop. Secondly, cancers due to radiation exposure are indistinguishable from any other cancer. Thirdly, radioisotopes can travel great distances. Therefore epidemiological studies investigating the effects of a particular radiation exposure are necessarily very long, they may involve many countries if not continents, and they are extraordinarily complex.

Add to this the fact that cancer is a common disease in any event, and the result is that a small percentage increase in cancer rates due to radiation exposure can readily be overlooked, even when the absolute number of cancers caused by radiation exposure may be very large.

A further source of misleading research results is the mixing, inadvertently or knowingly, of data for populations exposed to quite different levels of radiation, for example after a nuclear accident. The results for heavily exposed populations may then be 'diluted' by results for much less exposed populations and the results overall will appear reassuringly low.

Many governments have touted the economic advantages of using nuclear power, even in global warming arguments, putting health consequences in a clearly subordinate position (Karamoskos 2019).

Radiation is employed by the medical industry in the form of X-rays, CT Scans, mammograms, etc. Doctor awareness of the dangers of radiation exposure have been generally over-ruled by the defensive desire for an immediate and clear diagnosis. Studies are conflicting as to the benefits of the use of radiation. For instance, for CT scans, one study found that “a 45-year-old adult who plans to undergo annual full-body CT examinations up to age 75 (30 examinations) would accrue an overall estimated lifetime attributable risk of cancer mortality of about 1.9%” (Brenner 2004p735). This risk can be compared in Australia with the lifetime chances of dying in a traffic accident of about 0.37% (AG 2019). This is about five times lower than the CT scan risk. The road death situation is generally regarded by many authorities as each death being unacceptable and should be avoidable. A large recent study also found significant CT scan increased risks (Mathews 2013), but there are few studies done longitudinally on health effects from the increasing use of CT scans, especially on infants.

A similar story is found with the use of mammograms for breast screening. Studies show “Low-dose radiation increases breast cancer risk among high-risk women” (Jansen-van der Weide 2010p2547), and a “paradoxical trend of increased mortality with screening was found” (Diniz 2017). Yet industry sponsored studies show the opposite, that breast screening is good for lowering mortality (Saadatmand 2015). While others will admit the problem yet argue there is a net gain: “Annual screening of 100 000 women aged 40 to 74 years was projected to induce 125 breast cancer cases (95% CI, 88 to 178) leading to 16 deaths (CI, 11 to 23), relative to 968 breast cancer deaths averted by early detection from screening” (Miglioretti 2016p205), although the basis of averting the deaths claimed remain arguable.

Reviews of decades of breast cancer screening do not show a significant net gain (Bleyer 2012p1998):

Despite substantial increases in the number of cases of early-stage breast cancer detected, screening mammography has only marginally reduced the rate at which women present with advanced cancer. Although it is not certain which women have been affected, the imbalance suggests that there is substantial overdiagnosis,

accounting for nearly a third of all newly diagnosed breast cancers, and that screening is having, at best, only a small effect on the rate of death from breast cancer.

Many studies since mid-last century show low dose ionising radiation, including X rays, increase the risk of childhood cancer. A more-recent paper concluded "...in the past, a radiographic examination of the abdomen of a pregnant woman produced a proportional increase in risk of about 40%" (Doll 1997p130). In animal foetal studies, "an increased risk of malformation and growth retardation can be observed for two to seven weeks exposure (organogenesis stage), while exposure at later stages (fetogenesis) is mainly associated with brain damage" (De Santis 2007p177).

Although the effects of ionising radiation can be diverse and potentially initiate many disease types, the historic focus has been on cancer. It is categorised as a Class 1 carcinogen by the International Agency for Research in Cancer (IARC) of the World Health Organization (WHO), the highest classification consistent with certainty of its carcinogenicity. There appears to be no threshold below which cancers are not induced (NRC 2006a)

Radiation primarily depresses the immune system. Effects on bone marrow, for example, are well established (Senn 1970, Shadad 2013), affecting immune system precursor cell production. As for any immune-compromised host, the resident flora normally kept in check by the immune system, may then become an invading organism. One of these is the fungus candida albicans (*C. albicans*), which colonizes the skin and is commonly found in infections (Netea 2008, Bassetti 2015, Odds 2007, Brown 2012). Susceptible patients are those with complications, or undergoing invasive procedures or with head and neck cancers undergoing radiation therapy (Bassetti 2015, Redding 1999, Grotz 2003). It is also pertinent to note that growth rates of many fungi flourish in the presence of radiation (Dadachova 2008). If immune response is impaired then the infection may spread beyond the skin, producing more lethal forms of disease. *C. albicans*, on spreading in the blood can infect the kidneys, possibly resulting in renal failure (Spellberg 2005). However focus for potential infections after irradiation has been on models with bacteria species (Elliott 2004a, Cronkite 1955) so there is much undone science.

Industry efforts to downplay the detrimental effects of ionising radiation culminated in the 1910 to 1940's with industry arguing a hormesis case, without any substantiating studies, that

low amounts of ionising radiation were actually good for one's health. This was done for example, by highlighting specific study findings of say, dogs exposed showing increased DNA repair and cell proliferation and ignoring they also found higher rates of leukemia longer term (Seed 1992). This attitude saw the medical establishment at the time back the deliberate addition of radium to bottled drinking water, chocolate bars, coffee, toys, toothpaste, and cosmetics (Crezo 2012).

In a large metanalysis (Møller 2013), significant negative effects from environmental radiation were found in a range of categories, including immunology, physiology, mutational and disease occurrence. The frequency of negative effects was beyond that of random chance. As described by the authors (USC 2012):

There's been a sentiment in the community that because we don't see obvious effects in some of these places, or that what we see tends to be small and localized, that maybe there aren't any negative effects from low levels of radiation... But when you do the meta-analysis, you do see significant negative effects.

It also provides evidence that there is no threshold below which there are no effects of radiation. A theory that has been batted around a lot over the last couple of decades is the idea that is there a threshold of exposure below which there are no negative consequences. These data provide fairly strong evidence that there is no threshold — radiation effects are measurable as far down as you can go, given the statistical power you have at hand.

Surely if there are effects at these low levels, then one wonders why aren't the regulations for exposures reined in, especially for intentional ones, like the nuclear power plant emissions, medical procedures, and airport x-ray machines, etc.?

This section has touched on a complex area of environmental effects on health, from ionising radiation. It was not intended to comprehensively cover this area, only enough to illustrate the immense amount of public ignorance and undone science on its health effects.

In the MCS chapter, in the section titled: EHS Overlaps with MCS, it was mentioned that there is evidence that heavy metal residues compound the health effects of EMF (non-ionising

radiation). So another area of undone science is in respect to radioactive heavy metal synergism.

Sunscreens

An example of undone science on a common commercial product we commonly expose ourselves to, especially children, is that of sunscreens. The situation will be briefly considered.

Skin applications of sunscreen can typically result in many grams applied, especially over many hours with many re-applications through holiday periods (Wang 2019). However, although they are widely used, little is known about the dermal absorption effects of the sunscreen constituents (USFDA 2019). While the scientific testing of sunscreens has been significant in finding the effectiveness of sunscreens in preventing sunburn, other effects of the sunscreen constituents on the health of the individual to which they are applied has had little attention. This aspect represents a significant area of undone science.

The US FDA's guidelines require nonclinical (estimated, not tested-for) toxicity assessments where the absorption of the sunscreen chemicals produce a steady-state blood level greater than 0.5 ng/mL. This level was arbitrarily chosen from the FDA's acceptance levels for food packaging substances, not chemicals that are absorbed through the skin (Matta 2019).

Chemicals that migrate from packaging into food are ingested, whereas sunscreen chemicals are absorbed through the skin directly into the bloodstream, bypassing the digestive tract, which can filter out some of the toxins. This level has no basis for safety. The products in common use then have simply been assessed as safe by sunscreen industry toxicologists on the basis that absorption is below such levels, despite such chemicals themselves having little research done on their health effects other than basic testing such as lethal dose tests on mice etc.

Sunscreens are composed of a complex chemical mix and commonly use active ingredients such as cinoxate, dioxybenzone, ensulizole, homosalate, meradimate, octinoxate, octisalate, octocrylene, padimate O, sulisobenzene, oxybenzone, avobenzone and aluminium (Matta 2019, Tietz 2019). Most of these ingredients do not have even basic assessment data such as for carcinogenicity, developmental and reproductive aspects.

A recent study on four common sunscreens measured levels of: avobenzone, oxybenzone, octocrylene, and ecamsule, and found steady state blood levels of each of these greater than 0.5 ng/mL. Ecamsule was an interesting inclusion since at the time of this study it was under a new drug application and not included in approved sunscreen ingredients for over the counter sales (Matta 2019). This study is but one of a number that have found absorption of such chemicals in significant levels in the bloodstream (Klimová 2015, Janjua 2008, Gonzalez 2006).

Of the little science that has been performed on these active ingredients, the following can be found for just one of them: oxybenzone:

- Has been found in human breast milk (Schlumpf 2001).
- Was found in amniotic fluid, urine, and blood (USFDA 2019).
- Is a phototoxicant: it has adverse effects when exposed to light (Downs 2016).
- Is neurotoxic (Ruszkiewicz 2017).
- Links with endocrine activity (USFDA 2019, Krause 2015).
- Significantly lowers testosterone levels in adolescent boys (Scinicariello 2016)
- Reduces sperm count in men (ES 2016).
- Increases male infertility by affecting calcium signalling in sperm (Rehfeld 2018)
- Alters hormone levels in men, especially testosterone, estradiol and inhibin B (Janjua 2004).
- Is linked to endometriosis in women (Kunisue 2012)
- Oxybenzone and several other active ingredients in sunscreens enhance the ability of other chemicals to penetrate your skin, including toxic herbicides pesticides and insect repellents (Pont 2004).
- Oxybenzone concentrations were about 400 to 500 times higher than the presumed safety threshold after just a couple of days' use. (Mata 2020, Splete 2020).

In an alarming study for the presence of benzene in sunscreens, 27% of the samples tested, 78 popular US products, contained it especially in the aerosol type sunscreens (Valisure 2021).

Benzene has been linked to several health effects such as bone marrow suppression, decrease in red blood cell numbers, immune system suppression, irregular menstrual periods, decrease in ovary size, and leukemia.

While traditionally titanium dioxide and zinc provided the best sun protection they are now starting to be present as nanoparticles. Their use as nanoparticles for more transparency in the latest sunscreens remains another significant area of undone science in view of their possible absorption into the body enabled by the accompanying chemical mix.

Covered in other areas of this thesis is the manufactured ignorance production by the sunscreen industry funded studies on the effects of sunscreens on vitamin D production. Vitamin D is an important hormone which influences 3,000 of the 25,000 human genes substantially affecting one's health. Some independent studies show long term effects (Matsuoka 1988) while other studies show no effect (Marks 1995) on vitamin D production. But there have been no studies of the effects of high sun protection factor sunscreens now widely used (Neale 2019) on vitamin D production and absorption. This is added to by the general public ignorance surrounding the sun protection factors (SPF) on sunscreens: The SPF values are based only on UVB, so in a high SPF sunscreen there could be negligible protection against UVA which is also linked with skin cancer induction (Agar 2004, Burke 2009, Beani 2014).

The push for sunscreen use and that sun exposure is dangerous has been highlighted to be paradigmatic (Jacobsen 2019, Lindqvist 2016, 2018). Most medical practitioners will promote total sun avoidance due to fears of skin cancer, whereas others argue that it is essential to have small amounts of regular direct sun exposure for vitamin D production and health in general. Some studies have found that the risk of melanoma does increase with sun exposure. One significant study found the risk increases by 75% in relative terms, but in absolute terms the risk of developing melanoma is just 0.3% for those who tanned frequently (Veierød 2003, Christensen 2010). It has been suggested that for every death caused by diseases related to excessive sun exposure, such as melanoma, there are 328 deaths caused by diseases of sunlight deprivation (Sorenson 2018).

This section has discussed the undone science concerning environmental effects on health. It has been shown that industry interests simply focus on the desired areas of knowledge which are positive for their products. Industry priority is on the sale of their products rather than investigating whether such products are detrimental to public health. This sees undone science actively maintained. It can also be used to their advantage as a delaying tactic when

undesirable aspects may be highlighted in the future about their product: that there are insufficient studies done to prove any detrimental effects. Two examples were covered, ionising radiation and sunscreens, to highlight undone science concerning these technologies which are commonly regarded as proven and helping us achieve a high standard of living. These examples illustrated the lack of studies done of effects on human health, an area of undone science about which the public is generally ignorant.

THE RECOGNITION OF IGNORANCE

Nickerson (1998) reviewed evidence that most people form a confirmation bias in creating an initial hypothesis. Evidence in support is then looked for. As they are cognitively anchored to the hypothesis, alternative ones are not considered and contradicting evidence is not sought. Even when such an anchor point is clearly random, evidence is typically gathered that comfortably supports their hypothesis.

To illustrate the aspects of recognition of ignorance Roy & Zeckhauser (2015) used the concept of a consequential amazing development (CAD). “A CAD must be not only amazing but also consequential to the decision maker. To amaze, a CAD must be an outlier such that even an individual who has long contemplated the future might not have envisioned such an outcome”. This is what Gross (2010) has termed ‘nescience’.

The cognitive anchoring to the initial hypothesis severely limits the contemplation of consequences of any CADs or uncertainty and results in a complementary failing due to such bias or beliefs (Michotte, 1963; Rips, 2011). This may also lead to failure to learn or the possibility of overlooking evidence of causality, or on the other hand concluding more than the evidence supports.

To avoid a state of consequential ignorance, a self awareness and acknowledgement of CADs is required. However this is hindered by overconfidence and salience (Roy 2015), typically shows vulnerability, and influenced by information processing and storage psychology :

Overconfidence

Roy & Zeckhauser use the example of Shakespeare's King Lear for overconfidence leading to extreme CADs: To retire and divide his kingdom among his three daughters, King Lear decides to test their loyalty and feelings for him. Due to his overconfidence he uses the very weak test of simply asking. The two elder daughters respond with profuse professions of affection but the youngest, his favourite, is measured in her response which angers him. So he divides the kingdom between the two eldest daughters and soon finds that he is treated in a peremptory, and autocratic manner. Lear did not ever contemplate the CADs that then occur. His eldest daughters' exhibit hypocrisy and abuse and end up plotting against each other while Lear has been made virtually homeless. Lear and the youngest daughter Cordelia are captured with Cordelia ultimately hanged: Lear unable to save her. Lear then fully realises his overconfidence in the first place in relying on their professions of affection. The betrayal by the two eldest daughters represents a CAD for Lear.

Salience

It has been put forward (Higgins 1996, Roy 2015) that people identify states that are salient: experiences (on a recognition bias) or recallable instances (accessibility) after an external stimulus. As a result the likelihood of the events is overestimated (availability heuristic) in considering the future.

Roy & Zeckhauser (2015) use the example of Gulliver from Jonathan Swift's novel 'Gulliver's Travels' (2001). Gulliver is faced with extreme CADs and situations of ignorance which he must deal with. Rather than being a children's book it was intended as a satire on 18th century politics and religion. But it is an example of behaviour when faced with ignorance. With his past experiences having no relevance, Gulliver miscalculates and misses various cues, and assesses the situation incorrectly due to no prior experience.

Vulnerability

The study of vulnerability is for the understanding of the social causes of conditions resulting in increased exposure, and lack of resources to anticipate, cope with, resist and recover from types of disasters.

Vulnerability is not just a condition that limits us but one that can enable us. As potential, vulnerability is a condition of openness, openness to being affected and affecting in turn (Gilson 2011p310).

Current vulnerability studies however rely on the understanding of the expectable, or known knowns. Most vulnerability studies do not engage with ignorance. Yet when an unexpected disaster occurs, hindsight often unveils not only that weak signals existed, but that vulnerable conditions were systematically overlooked and ignored by responsible authorities (Weick 2007). The discovery of nescience marks the precondition for a radical surprise (Gross 2007). After a disaster, the responsible organisations try to deflect oversight on their part. This is usually in the form of showing they were acting per the rules, attributing to individual oversights, associating with misconducts or wrong decision making (Hood 2002, Rothstein 2006).

The strategic utilisation of ignorance led to the fatal gas leak in 1984, at Union Carbide in Bhopal, India. Some of the aspects that led to, and amplified the disaster were (ICFTU 1985, UCC 1985, Varadarajan 1985):

- Use of hazardous chemicals, such as the toxic methyl isocyanate rather than less dangerous ones.
- Large tank storage of these chemicals rather than steel drums.
- The possible presence corroding material in pipelines.
- Poor maintenance of the plant which closed in the early 1980s.
- Several safety system failures (due to poor maintenance and regulations).
- Some safety systems had been shut down to save money, including the failed tank refrigeration system which would have prevented the disaster.
- Cheap plant modifications to conform to government regulations.
- The plant was located near a densely populated area.
- No disaster plans for plant leakages.
- Health care and rehabilitation shortcomings for such a disaster.

After the disaster there was denial of the toxicity of the leaked substance, what happened on site, as well as legal responsibilities of actors, led to the complexity of determining

responsibilities and consequences (Jasanoff 2007). Outrage was inhibited by combinations of evidence cover-up, victim devaluation, event reinterpretation, utilisation of official investigations in interpreting the events and the intimidation and bribery of the witnesses, supporters and victims, (Engel 2006). Union Carbide even attributed to an act of sabotage by a "disgruntled worker" (Kalelkar 1988).

Another example was the Minamata, Japan, situation where from the mid-1950s the local population experienced severe health effects, birth deformities, deaths, etc. from exposure to methyl mercury traced to a local chemical plant. Here the local population were dependent on a marine-based diet which made them vulnerable to health effects from toxin ingestion. The chemical plant continued discharging this toxin from 1932 to 1968. The ignorance about health effects on subsequent generations was downplayed by health authorities where due to undone science, there was no evidence that the toxin could cross the placenta. The famous reporter Eugene Smith captured the immortal image called 'Madonna of Minamata', showing a mother beside her grossly deformed son. Smith died a few years after a mob of chemical plant thugs had bashed him within an inch of his life when he tried visiting the plant to interview the manager (Smith 1975).

Kuhlicke (2015) used an example of the 2002 flood of the Mulde River in Germany. The residents were totally surprised and unprepared for such a situation which was quite disastrous for them. The towns had been well-protected by dikes, drainage systems and upstream retention basins since the 1950s. Furthermore, the residents downstream did not act on warnings from flooded residents upstream as the flood peak progressed along the river. The extensive engineering in the 1950s had appeared to have adequately solved the problems of flooding since then and falsely promoted certainty, stability and security for the population. Paradoxically, the previous effective control of the river set the situation for the 2002 flood being a surprise and hence the population vulnerable to such a disaster.

In the case of New Orleans after Hurricane Katrina, most of the population was vulnerable to the disaster due to poverty, home ownership, poor English language proficiency, ethnic minorities, immigrant status, and high-density housing (Zoraster 2010). Afterwards comprehensive soil tests for toxic chemical residues were not done in all neighbourhoods by the government departments. This limited the ability of the residents to react to the situation.

The locations that were tested seemed to be related to cultural inequalities (Frickel 2014a). So knowledge inequalities were related to cultural inequalities.

The vulnerability of the population to the effects of GMOs is similar to the above Mulde River flood example, where the people think there is a regulatory system that protects the public from any adverse effects from new food technologies. The ignorance of the regulatory authorities alone on the possible consequences for the release of such organisms into the environment on long term food security aspects remains to be seen and experienced by the whole population.

Throughout the research for this thesis on the environmental effects on health, in study after study there has been a clear vulnerable group of people affected by the cumulative types of ignorance that has been highlighted: that is on babies from the prenatal and postnatal/infant stage of human life.

Information Processing and Storage Psychology

Before we progress into looking how various forms of ignorance play a part in the aspects of environmental effects on health it is pertinent that we consider how human psychology on information processing can also contribute to ignorance.

It has been proposed that there is a dual nature of information processing and storage in human psychology (Chaiken 1999, Kahneman 2003,2011). In this concept, the mind is cast as having two systems for processing information and formulating judgements on such:

System 1

This is a quick and intuitive system driven by things as they first appear with judgement driven by the immediate environment and the immediate reactions experienced.

Answers to complex problems for example would be quickly guessed at via this system.

System 2

This is a slow and deliberate process where the new information is compared with stored memories and evaluated via logic and inference principles. It requires effort, motivation and ability. Answers to complex problems would be endeavoured to be accurately calculated or answered by this system.

Prentice (2015) identifies some implications for ignorance and knowledge in the human thought process:

- Knowledge can be separate between the two systems, for example, system 2 may know that sun exposure causes skin cancer, yet system 1 sees other people go out in the sun and partake in an activity, and so one goes out with the others anyway.
- Both systems have different ways of knowing. System 2 knowledge has been gained by learning, thought and logic. System 1 knowledge is quickly concluded based in immediate information experienced, taken at face value without scrutiny (Gilbert 1991). Prentice points out that knowledge comes so naturally in system 1 that it is suggested it cannot experience ignorance.
- As system 1 judgements are easy and undemanding compared to the effort required for system 2 judgements, system 1 is a more reliable guide to behaviour than system 2.

Traditionally, modes for behaviour change have focussed on system 2 approaches but system 1 has been increasingly utilised. An example is where factual information, perhaps with statistical and educational methods, were formerly used to discourage smoking. Today, simple messaging, such as a horrific picture of a lung cancer sufferer coupled with a simple message, is aimed to 'nudge' behaviour in the desired direction (Shafir 2012, Sunstein 2013). If the system 1 approach fails this reflects that the assumption about shared values of the population was incorrect rather than the choice of using a system 1 approach.

A complexity is generated by a system 1 approach. Its knowledge is simply formed by any information readily at hand which may be partial, biased and shallow. Such knowledge can interfere in putting system 2 knowledge into action or simply mask it. Such dynamics underlie the pluralistic ignorance soon to be covered. Orthodox doctors discussing a patient that claims to be sensitive to everyday chemical products or electromagnetic fields will not question a common general attitude that they should be treated psychologically. In looking to others in a group situation, the system 1 response is to take other people's action at face value as an indication of what they think and feel. The action, or inaction, of the other people in the group per a system 1 approach is then interpreted as an appropriate response. Ignorance, which may have created the behaviour, is perpetuated by the system 1 generated mis-knowledge.

Pluralistic ignorance (see more detail on this in a later section of this chapter) can generate a perceived need to educate system 2. This has been illustrated in an example by Prentice (2008) on excessive drinking by college students. Public concern over the pluralistic phenomenon where students are in drinking situations, displaying pleasure and camaraderie has initiated a variety of educational campaigns about excessive drinking aimed directly at system 2. However the student pluralistic ignorance in drinking situations is via system 1 response. Prentice observes that the best way to change pluralistic ignorance behaviour is to address both the ignorance of system 2 and the misknowledge of system 1. This can be through promotions to increase bystander action such as in an emergency check the victim is OK and dial emergency, “be an upstander not a bystander”, etc. together with group sessions discussing the dynamics leading to the misinterpretation of others’ behaviour. For the case of doctors recognising MCS, mentioned before, the required leadership to initiate such education/discussion is absent due to the paradigmatic nature of medicine today, as promoted and reinforced by the pharmaceutical industry maintaining orthodox doctor pluralistic ignorance.

Knowledge to prevent ignorance can be circumvented as Thaler & Sunstein (2008) illustrated by the example of how to induce people going through a cafeteria line to buy more healthy food. The placement of the healthier food first plays on the tendency for people to load their plate more from the earlier foods than the later foods. Further, as Johnson & Goldstein (2003) illustrated, in some countries despite various campaigns to highlight the importance of organ donations, only about 15% of the population registered. However when it was made such that all people were organ donators unless they opted out, more than 85% of the population did not opt out. This, and many other opt-out approaches, such as to children’s vaccines, produce better results than opt-in procedures.

The key question from the above examples which rely on system 1 thinking, is whether ignorance is needed to be effective. Do these remain effective when system 2 education and reasoning occurs? Certainly those who have educated themselves on the benefits of healthy food would already be choosing their food wisely in a cafeteria line. This is in line with dual process theory which would predict that system 2 based behaviour would override such behavioural nudges. However, as Prentice (2015p271) comments: “it is important to note how rare System 2 thinking is in these domains. Indeed, even if nudge strategies are effective only when people are ignorant, they will still be effective most of the time.”

There are strategies that go further than a 'nudge' for a system 1 effect. This can be achieved by approaches such as in social norms marketing. This is where the individual is compared with others, gaining the attention of system 1 with the motivation of conformity influencing employment of system 2 resources to achieve such. This is best illustrated in power bills that have comparison graphs to other power consumers with perhaps the use of smiley faces or other methods for low power consumers to override the inclination to conform by increasing consumption (Schultz 2007). This type of approach has the potential to promote new habits and long-term behavioural change. However used in other areas it may exhibit its shortcomings which are the absence of functional behaviour, use of the right values, and socially beneficial self-esteem. Often a normative component is missed in these programs such as information on the energy conservation program in the above example.

In conclusion, although appealing for excusing dysfunctional behaviour, ignorance cannot be used as a complete explanation. This is due to the importance of values in influencing one's behaviour. People generally do things not caring about downstream and collective consequences of their actions. Perhaps this is also added to from the perspective of others in viewing such behaviour in the immediate time frame.

The following sections will now consider various forms of ignorance involved in the environmental effects on health topic. The classifications overlap significantly and are far from distinct and isolated forms of ignorance.

MANUFACTURED IGNORANCE

If a company has a product which it has spent money developing and marketing it is a usual strategy to portray to the public how good it is. Any negative concerns about the product must be addressed or avoided, so that there is no effect on the product's sales. For environmental health effects from industrial products, such as pesticides, food additives or electromagnetic fields from phones, there can be scientific studies published which link health problems to them. In such situations a common industry response is to manufacture uncertainty/doubt about such studies to enable the continuation of their product sales and protect themselves

from potential liabilities if it is later deemed a dangerous good. When a company realises that there will be health consequences from its own research or evaluations this usually creates an incentive to manufacture ignorance. There have been numerous stories of companies hiding data, falsifying evidence, and selectively using findings for their advancement. The tobacco industry is the classic case which saw actions to generate uncertainty, doubt, and ignorance through advertising, misleading press releases, support for alternate research, establishment of research institutes, and funding of supportive research (Proctor 2008). The net result of such actions is the manufacture of ignorance among the general public, as well as the regulatory authorities, to effectively maintain the company's bottom line by keeping such products on the market and maintain their use.

One of the common measures used to downplay published studies that are counter to the interests of industry is to employ doubt alongside the manufacture of ignorance. Two prominent books that have highlighted this avenue are *Merchants of Doubt* (Oreskes 2011) and *Doubt is their Product* (Michaels 2008b). They show how detrimental product study results are put into doubt and promote favourable results through government committees, think tanks, and all types of media including scientific journals.

There are many ways of manufacturing ignorance and we will consider only three examples in a brief form below without pursuing details. There were however other examples that were investigated so these are summarised after the three main examples and simply listed in a table for generalised references.

The first example is the pesticide Roundup produced by Monsanto. In this example recent court cases where exposed victims have successfully connected their cancers with this product, have uncovered internal Monsanto documents. These documents have shown that the company had been familiar with severe health problems being linked with their product's exposure for many decades yet hid such. It also illustrates how they have proactively tried to discredit those organisations that have recognised the dangers of their product mentioning the use of seemingly independent organisations as well as ghostwriting scientific articles to use on a 'weight of evidence basis' for the evidence-based medicine approach used by the orthodox medical system.

The following example concerns the drug Phenylpropanolamine (PPA). In the early 1970s reports began of haemorrhagic strokes in young women following the taking of the drug. Twenty years later the US FDA raised concerns on the safety of the drug. These were rejected by the pharmaceutical companies. However in an unusual compromise between industry and the FDA, an independent investigator, the Yale University School of Medicine was used to investigate whether there was any causal relationship with the drug. In 1999 the study confirmed a causal relationship which was published the year after (Kernan 2000). At that time the drug had \$500 million per annum sales for the manufacturer. From an FDA analysis, the drug was responsible for between 200 and 500 strokes per year in the US (Michaels 2008a). Yet rather than drug withdrawal the manufacturer turned to a product defence firm who then attacked the Yale study on “bias and areas of concern” (Michaels 2008a). After stalling the FDA process for about a year, the FDA finally asked the manufacturer to cease marketing PPA in November 2000, almost 30 years after causal relationships were noticed. When this happened the manufacturer shipped a reformulated product immediately, having used many years to develop such a product.

The last example is on the toxic chemical BPA. It is an example of a very toxic chemical that has remained in use in our society for decades due to industry’s use of manufactured ignorance. It highlights how industry can infiltrate review panels and demand definitive proof before a decision is made, thus initiating further studies, enabling the product to remain being used while that happens. This is again similar to one of the tobacco industry strategies.

An indication of the success of the industry’s efforts to manufacture ignorance is when media are compelled to refer to the issue as a ‘debate’ or that they feel compelled to air ‘both sides’ of the story or argument. This happens commonly when environmental exposures are trying to be attributed to the cause of health problems.

In scientific circles where there are many studies done on certain subjects, such as chemicals in industrial products, some researchers conduct meta-analyses or scientometric analyses of the literature. These however can be used as a tool for the manufacture of ignorance since if the database has highly flawed studies (produced by industry) then a summary of the literature to determine conclusions or ways forward, will have unknown reliability and credibility. This is seen in many of the examples mentioned.

At the end of this section on manufactured ignorance is a short section which mentions the most-common manufactured ignorance approach encountered in this thesis research: the approach to put blame on individuals for their health problems.

Roundup Herbicide: Glyphosate Active Ingredient

This example illustrates how companies can hide knowledge of their product's health problems yet criticise published studies finding the problems. Industry's political influence and regulatory capture sees the public left to initiate action to ban this toxic chemical.

Orthodox views on Roundup are generally that it is a very safe herbicide and constitutes a backbone of the orthodox agricultural industry which relies on the product and therefore economical to use generally. Some examples of such attitudes:

When equal amounts were given orally and compared, it took less acetic acid to kill rats in the laboratory test that it did glyphosate. The acetic acid in even household vinegar was MORE toxic than Roundup! (Smith 2015)

Monsanto insists Roundup is not carcinogenic, says it has no plans to pull it from the market and is appealing the verdicts. "It's clear these products are safe when used as directed," said Rakesh Kilaru, a Washington attorney for Monsanto. (Higgins 2019).

Alex Berezow, PhD, is vice president of Scientific Affairs for the American Council on Science and Health, a pro-science consumer group.... "Glyphosate is safe to use, regardless of the brand," Berezow told Healthline. "The people who are exposed to the highest doses are farmers. But studies show that farmers don't have increasing rates of cancer despite the fact that more and more glyphosate has been used over the years (Plater 2019).

Recently in the US, Edwin Hardeman took Monsanto to court alleging that they concealed the risks of Roundup Herbicide causing non-Hodgkins lymphoma. He was successful with the case with his attorney stating:

It is clear from Monsanto's actions that it does not care whether Roundup causes cancer, focusing instead on manipulating public opinion and undermining anyone who raises genuine and legitimate concerns about Roundup. (Levin 2019).

The company's statement included:

...this verdict does not change the weight of over four decades of extensive science and the conclusions of regulators worldwide that support the safety of our glyphosate-based herbicides and that they are not carcinogenic.

Yet the jury had been presented evidence that between 1980 and 2012, Monsanto was aware of 7 animal studies, 5 epidemiological studies, 3 oxidative stress studies and 14 genotoxicity studies linking Roundup with cancer.

During the case it was revealed that Monsanto had only ever done testing on one component of the product Roundup: glyphosate. This also meant that regulatory authorities around the world had accepted such selective testing and represents selective ignorance on their part. The toxicity of the full formulations, of which glyphosate is a small part, has been found to be more toxic than glyphosate on its own (Mesnage 2013, Peixoto 2005). One of the ingredients for example, the surfactant polyethoxylated tallowamine (POEA), has been found to have various toxic effects (Brausch 2007, Mesnage 2013, 2019).

After the (IARC) had classified Roundup as a probable carcinogen, Monsanto orchestrated a critical review and alternative conclusion to the IARC assessment (Williams 2016). This expert opinion study declared:

...each individual participated in the review process and preparation of this paper as an independent professional...Neither any Monsanto company employees nor any attorneys reviewed any of the Expert Panel's manuscripts prior to submission to the journal."

Documents revealed by the trial showed that the data and study had been developed by Monsanto, and the journal that published the article used a Monsanto scientist for review. But the media had picked up on this article and had innocently assisted Monsanto counter the bad publicity since the IARC report.

A journalist attending the trial documented (Jaxen 2019):

The judge is allowing portions of a 2015 internal Monsanto email in which company scientist Bill Heydens discusses plans to ghost write a series of new scientific papers that

will contradict IARC's classification of glyphosate because in that email, Heydens remarks on how this plan is similar to the ghost writing of a scientific paper published in 2000 that found glyphosate to be safe.

A deposition from a Monsanto executive showed that \$16-17 million had been allocated in 2016 for an effort to combat IARC's report (Jaxen 2019). Some of this had also been given to a non-profit organization claiming to be independent: the American Council on Science and Health (ACSH). This was an industry front group which claimed IARC's findings were "scientific fraud" and called the cancer agency a "fringe group, seemingly more interested in scaring people than identifying actual health threats" (Berezow 2017).

Other documents revealed during the discovery process of lawsuits:

- Monsanto's "intelligence fusion center" targeted Journalist Carey Gillam from the nonprofit U.S. Right to Know (USRTK) and singer-songwriter Neil Young. Prior to the release of Gillam's book "White Wash" it was planned to discredit it through negative customer reviews and paying Google to preference search results negative to Gillam.
- Monsanto reported on USRTK's activities and developed a detailed plan for handling their Freedom of Information Act (FOIA) requests.
- The agrichemical industry hosts a private email server for co-ordination of its lobbying activities: AgBioChatter. Members include pro-industry academics, agrichemical industry staff and public relations staff. The members were advised to delete emails when there had been concern that an FOIA request would occur for the emails
- Monsanto's strategy for discrediting IARC, which included sending their team of lobbyists to speak with staff at the EPA, USTR, USDA and State Department and placing advertisements in Capital Hill newspapers (Fang 2019).

In a more recent cases the courts concluded:

We find that substantial evidence supports the jury's verdicts. Monsanto's conduct evidenced reckless disregard of the health and safety of the multitude of unsuspecting consumers it kept in the dark. This was not an isolated incident; Monsanto's conduct involved repeated actions over a period of many years motivated by the desire for sales and profit.

Monsanto acted with “willful and conscious disregard for the safety of others” and “failed to conduct adequate studies on glyphosate and Roundup, thus impeding discouraging or distorting scientific inquiry concerning glyphosate and Roundup.” (USRTK 2021).

As can be seen from the above, industry can actively manufacture ignorance to promote its products.

No individuals, such as directors or managers in Monsanto have been held responsible. No-one has been held responsible for adversely affecting other people’s lives: rather the company entity simply must pay the fine.

The above contrasts to research showing glyphosate has multi-generational effects. Pregnant rats exposed to low levels of glyphosate saw second and third generation offspring with high rates of prostate disease, obesity, kidney disease, ovarian disease, and birth defects (Kubsad 2019).

In sailing the political winds, the US EPA has changed from its 2015 “might cause cancer” position to today’s “not likely” stance. Due to its failure to act to protect the public, the Center for Food Safety on 17 Dec 2020, representing farmers, farmworkers, and conservationists, commenced a lawsuit against the EPA alleging that they ignored glyphosate’s health risks, disregarded its ecological impact, and failed to consider side effects such as drift damage.

This example illustrated how companies can hide knowledge of their product’s health problems while criticising published studies finding such problems. Industry’s political influence and regulatory capture of the regulating departments sees the public left to initiate action to ban this toxic chemical.

Phenylpropanolamine (PPA)

PPA is an over the counter drug for appetite suppression and a decongestant.

In the early 1970s reports began of haemorrhagic strokes in young women following the taking of the drug. Twenty years later the US FDA raised concerns on the safety of the drug. These were rejected by the pharmaceutical companies. However in an unusual compromise between industry and the FDA, an independent investigator, the Yale University School of Medicine was

used to investigate whether there was any causal relationship with the drug. In 1999 the study confirmed a causal relationship which was published the year after (Kernan 2000).

When the study's findings were made known it had \$500 million per annum sales for the manufacturer. From an FDA analysis, the drug was responsible for between 200 and 500 strokes per year in the US (Michaels 2008a). Yet rather than drug withdrawal the manufacturer turned to a product defence firm who then attacked the Yale study on "bias and areas of concern" (Michaels 2008a). After stalling the FDA process for about a year, the FDA finally asked the manufacturer to cease marketing PPA in November 2000, almost 30 years after causal relationships began to be noticed. When this happened the manufacturer shipped a reformulated product immediately, having used the intervening time for such product development. The product defence company boasted about this case on their website. The write up concluded:

The unique ability of the experts at the Weinberg Group to combine their expertise in epidemiology and biostatistics with strategic thinking enabled them to lead the pharmaceutical company's effort in their dispute with the FDA (Michaels 2008ap98).

Bisphenol A (BPA)

BPA is a chemical that has been used extensively to harden plastics for many decades. It is present in water bottles, dental sealants, medical products, compact discs, canned food linings, etc. Most of the population has BPA residues in their bodies from eating foods from containers made with BPA, and also from the air, dust, and water.

Some orthodox views on the safety of this chemical are:

FDA's current perspective, based on its most recent safety assessment, is that BPA is safe at the current levels occurring in foods (FDA 2018).

Some studies have raised potential concerns that BPA exposure may cause health problems. However the overwhelming weight of scientific opinion is that there is no health or safety issue at the levels people are exposed to (FSANZ 2018).

In the last decade or so the public started to gain some knowledge of the dangers of BPA and saw baby and children products and baby bottles change to 'BPA-free' products, such as baby

formula cans, sippy cups, etc. The BPA replacement, Biphenol S, just like BPA when introduced, has not been extensively tested for health effects and is already starting to be found to be just as toxic (Mao 2020).

The toxicity of BPA has been illustrated in other chapters. The mounting evidence that this chemical should be phased out was resisted by a stalling effect by industry. In the US in 2000, a large peer review was done through the National Toxicology Program of the National Institute of Environmental Health Sciences. Thirty-six scientists looked at the low dose effects of EDCs. They concluded “there is credible evidence that low doses of BPA (bisphenol A) can cause effects on specific endpoints” (Melnick 2002p428) and also noted that “it is not persuaded that a low-dose effect of BPA has been conclusively established as a general or reproducible finding” (Melnick 2002p429). As one of its main recommendations, to narrow the uncertainties, the panel composed an extensive research agenda to fully elaborate the mechanism at the molecular level of low-dose interactions which would take decades to carry out. This reflected the tobacco industry’s approach when large amounts of evidence had accumulated and they stalled further progress by emphasising the need for definitive studies. A discussion on the absolute certainty in medicine and public health is discussed later in this chapter and continues the example of BPA.

The American Plastics Council funded its own study which found positive reproductive and developmental toxicity studies on BPA as “questionable” (Gray 2004).

BPA becomes an example of a very toxic chemical that has remained in use in our society for decades due to industry’s use of manufactured ignorance.

Other Examples Summarised

As mentioned previously there were other examples of manufactured ignorance investigated but will only be briefly mentioned below and in the following table

Drug companies spend millions of dollars to get their drug approved and so have a huge financial interest in ensuring the drug has large sales in being extensively used. It perhaps represents an extreme in the manufacture of ignorance. Poor drug quality, bribery, fraud, conflict of interest, cover-ups, and price-fixing are just a part of the myriad of potentially illegal and unethical approaches used by the pharmaceutical industry (Clinard 1980, Braithwaite 1984, Dukes 2014, Gøtzsche 2013, Martin 2018). The prominent pharmaceutical companies have all

been fined millions of dollars almost each year for serious illegal activities on different occasions, being regarded as recalcitrant and displaying a willingness to use “illegal inducements as a core business strategy for selling its prescription drugs” (Kelton 2013).

The classic tobacco industry strategy must also be mentioned. With links to cancer being made early to mid last century, this industry has pioneered the use of ignorance such that their products are still being sold today.

Another example concerns the possible reaction between a health condition called Reye’s Syndrome and the use of drugs such as Aspirin. Since the fatality rate can be 1 in 3 in children it was important that warnings be sent out and present on packaging. In the early 1980s studies found a link between salicylate use in drugs such as aspirin, the presence of a virus in the consumer, and deaths in children. The delay in the US FDA’s regulation was due to the pharmaceutical companies insisting that the main four studies indicating the link were unclear, incomplete, or uncertain. They put-forward 17 flaws of the studies and called for more studies to be conducted (FDA 1982). This effectively kept the drugs on the market and prevented FDA warnings being issued. This situation continued for years until a community group pursued litigation for the government not acting to prevent more deaths from happening. This illustrates that even drugs, which are supposed to be regulated more thoroughly than many other chemical products such as pesticides, have ignorance manipulated by industry to keep their product on the market.

Another example concerns beryllium exposure, which highlights the unscientific nature of workplace safety levels. Apart from causing a condition of chronic beryllium disease, it was also being linked to lung cancer. A product defence firm which had done significant work for the tobacco industry (Michaels 2008a), in 2002 published a re-analysis of one of the pertinent Centers for Disease Control studies (Levy 2002). The reanalysis showed how the funding of studies can affect the outcome (compared to numerous studies concluding differently). In changing some parameters of the studies the lung cancer rates became statistically insignificant. This was then used by industry to question the validity of the link with lung cancer. This becomes an example of the industry approach of disputing data, calls for reanalysis, animal data argued as being irrelevant, human data faulty, unreliable exposure data, so more research is called for. It shows how a purely arbitrary allowable exposure level,

established in 1948 without any scientific studies, was retained by the US OSHA due to manufactured ignorance until 2017 when it was reduced ten-fold. This came at a cost of tens of thousands of deaths in the interim, again with no one particularly held responsible.

Breast cancer can be used as another example. It illustrates a manufactured ignorance that plays on public fear. Cancer is a multi-billion-dollar industry. The undone science is in the areas of the initiators of the cancer, identifying the beginning of dangerous forms of cancer and the side effects of mammography checks etc. Unnecessary breast cancer treatments can be promoted as saving lives if sufficient public ignorance is attained. Furthermore a false positive test and subsequent orthodox treatment can be utilised as a false example where early diagnosis has saved lives due to general public ignorance of the actual situation.

A further example considers ocean red tides, so named due to the colour of the toxic red algae that floats near the top of the water. These are found at many locations around the world but a good situation to consider is the red tides off the US east coast. There, the clearly most-prominent research organisation on these tides, MOTE Marine Labs, has published many scientific studies yet is openly sceptic about any environmental links. Any study showing environmental links is met with comments from the organisation that “the issue is too complex to draw conclusions,” “more research needs to be conducted,” and “the science is not settled.” Or calling for more research: “Full characterization of cellular consequences of brevetoxin exposure is critical to fully understand the impact of recurrent red tide events on human health” (Fleming 2011) which helps to raise more donations and delay conclusions. Millions of dollars have been given to the organisation over the years from government grants, public donations, and corporate sponsorships. The Mosaic Corporation is one of their sponsors, one of Florida’s most heavily fined polluters, and is their "corporate benefactor" on their website. It illustrates how industry can effectively use a seemingly independent front organisation to maintain ignorance.

Undone science areas of vaccine efficacy and safety studies are a further example. Only one study was able to be found which uses a true placebo. Normally the placebo is simply another vaccine which is administered to the control group. Most of the studies also use simple, short term health endpoints to monitor. One most recent studies referenced by the Australian National Centre for Immunisation Research and Surveillance as substantiating the effectiveness

and safety of various vaccines is a good example and illustrates the non-use of a true placebo as well as deceiving comments in the abstract claiming that health effects were studied for 6 months after vaccine administration. Furthermore the study exclusion criteria were far in excess normal contraindications, ensuring that only a healthy group was studied. Further undone science areas contribute to substantial medical and public ignorance on some safety aspects of vaccines. Vaccines illustrate how ignorance can be actively created by presenting findings as if sound science has been utilised. The abstract can be very deceptive.

Another example is aspartame which is an artificial sweetener 200 times sweeter than sucrose and is frequently a sugar substitute in beverages and foods. It has commonly been promoted as beneficial to dieters due to its sweetener characteristic without affecting blood sugar level. However there are findings indicating that it is not beneficial for weight control or a diabetes aid. Front groups for the industry are utilised to downplay many health effect concerns from the chemical. It highlights how a consistent marketing campaign based on one fact (that it's not sugar) together with an unsubstantiated attractive claim (beneficial to dieters) can maintain large sales of the product.

Biosludge is another example. This product illustrates how government departments can actively take part in the suppression of detrimental environmental health effect knowledge by manufacturing ignorance. An ex-USEPA senior researcher has been a whistle blower on these aspects. His book title explains: "Science for Sale: How the US Government Uses Powerful Corporations and Leading Universities to Support Government Policies, Silence Top Scientists, Jeopardize Our Health, and Protect Corporate Profits".

Community water fluoridation is another example. It highlights the original manufactured ignorance on the benefits of fluoride with industry studies showing advantages against teeth decay yet a growing number of independent studies over the years have not found such. The original studies are also typical of considering only one end point, tooth decay, and leaving as undone science whether such a toxic chemical will have any other side effects on human health.

Manufactured Ignorance Example	Description	References
Pharmaceutical Companies	These companies spend millions of dollars to get their drugs approved and so have a huge financial interest in ensuring the drugs have large sales. It perhaps represents an extreme in the manufacture of ignorance. Poor drug quality, bribery, fraud, conflict of interest, cover ups, and price fixing are just a part of the myriad of potentially illegal and unethical approaches used by this industry	Rawlinson 2017, Clinard 1980, Braithwaite 1984, Dukes 2014, Gøtzsche 2013, Martin 2018, Kelton 2013, Tombs 2015, Peters 2014, Pearlman 2017, Shearer 2016, Swithers 2013, Yang 2010, Husten 2014, ESC 2009, Husten 2011,
The Tobacco Industry	In the face of increasing evidence that cigarettes were killing many smokers, in the early 1950s the industry launched a multimillion dollar campaign to assure consumers that there was no 'proof' of these hazards. The industry's strategies after the first links of cigarettes with cancer were published in scientific journals, almost 70 years ago, have become a model for other industries. Cigarette manufacturers continue to sell and market a product that kills millions of people per year worldwide.	Proctor 2008, Oreskes 2008, Callahan 2012, Michaels 2008a, Christensen 2008,
Breast Cancer	This example illustrates a manufactured ignorance that plays on the public fear of cancer. It is to the cancer industry's advantage that breast screening is standard practice leading to many false positive diagnoses that require medical attention. The undone science is in the identification of the beginning of dangerous forms of cancer with possible environmental causes, and the side effects of mammography checks.	Bleyer 2012, Gøtzsche 2013, Davis 1993, Calaf 2020, IBCERCC 2013,
Red Tides (harmful algae blooms)	Red tides are increasingly occurring worldwide. Water colour varies according to the species involved and may make the water red, brown, green, or purple, while others, also toxic, do not discolour the water. There is wide ranging debate on what causes the blooms. It is ripe for manufactured ignorance with the 'cause' often attributed only to the algae and phytoplankton that are present, rather than why they grow so profusely in the first place.	Kirkpatrick 2004 & 2010, Fleming 2011, Glibert 2009 & 2018, Clark 1989, Jeong 2005, Bigelow 2014, Brand 2007

Vaccines	<p>The issues surrounding vaccines are highly controversial. The proponents and critics of vaccines are polarised, which is indicative of true paradigmatic beliefs being present. The concerns raised about vaccines need to be considered since it is a normal scientific approach to always question the science involved: scientific knowledge is never finalised. The non-use of true placebo controls in studies is one of many concerns.</p>	<p>Rogers 2019, Cowling 2012, Doshi 2020, Steinhoff 2012, Schlegel 1999, AHRP 2018, Cutts 2005, NCIRS 2020, Gatti 2016, Baylis 2003, Lyons-Weiler 2020, MCT Lawyers 2018, Lyons-Weiler 2016p186, Yuan 2016, Huynh 2008, Moseman 2012, Crone 1992, Poland 2012, Mathias 1989, Victoria 2010, Baylis 2011, McClenahan 2011, Petricciani 2014, Heckenlively 2017, Gentempo 2017, Pierson 2015,</p>
Aspartame	<p>Aspartame is an artificial sweetener. It is 200 times sweeter than sucrose and commonly used as a sugar substitute in foods and beverages. It is usual in sugar-free ice cream, diet soda, reduced-calorie fruit juice, chewing gum, yogurt, sugarless candy, and many other products promoted as being without blood sugar boosting refined sugars. It is extensively used in food products and promoted by industry and their front groups despite there being serious health concerns. It is a good example of where manufactured ignorance has enabled continuance and expansion of the use of a chemical over many decades.</p>	<p>Han 2019, Pepino 2013, Feijó 2012, Kroger 2006, Giacaman 2013, Soffritti 2006 2007, Schernhammer 2012, White 2004, Rycerz 2013, Olney 1996, Iyyaswamy 2012, Smith 2001, Holton 2012, Gross 1975,1976,1985, 1987a,1987b, Roberts 1997</p>
Biosludge	<p>The use of biosludge (or biosolids, or sewerage sludge) on playgrounds, farms, forests, parks, etc. has been occurring in the US and some other countries for many years. According to Lewis (2014), information about health hazards has been suppressed, to the advantage of industry.</p>	<p>Lewis 2014,</p>
Public Water Fluoridation	<p>Fluoride is added to public water supplies in some parts of the world with the aim of decreasing the rate of tooth decay in children. There is vast literature on the benefits, focused on dental health, and arguments condemning detractors, arguably in defence of a paradigm. It is typical of manufactured ignorance and a paradigmatic belief system considering the accumulating independent studies looking into the undone science on health effects of the chemical.</p>	<p>Newbrun 1999, Yeung 2008, Colquhoun 1985 & 1997, Hirzy 1999, Herbert 1992, NRC 2006b, Galletti 1958, Douglass 2006, Ortiz-Pérez 2003, Jianjie 2016, Bassin 2006, Kharb 2012, Malin 2019a,b, Yiamouyiannis 1977, Kim 2015, Blomberg 2017, Riddell 2019, Choi 2012, Grandjean 2014, Tang 2008, Till 2020, Bashash 2017, Oxford 2021, García-Pérez 2013, Neurath 2019, Wiener 2018</p>

The Most-Common Manufactured Ignorance Attitudes on Health Issues from Environmental Exposures

Although manufactured ignorance can take many forms, as illustrated in the above examples, there are some common results for most environmental exposure crises. The main approach is that of the industry and government blaming of the victims. The crises can last for decades before any action is taken, commonly via legal channels by public groups. In the lead issues blame ranged from the children's bad behaviour through to the parents' lack of discipline, lack of moral values and even being single parents (Warren 2000). The asbestos mine workers with lung problems were blamed for smoking or having an unhealthy lifestyle (Schneider 2004). After health warning labels were put on cigarette packets the blame was put on smokers for ignoring the warning (Michaels 2008a).

Another commonality is that after legal discovery in legal cases, as seen in the Roundup example above, but also with others such as asbestos, lead, tobacco, etc., industry documents show clearly that the companies involved knew the extent of the problems and deliberately covered-up such knowledge and actively criticised any similar knowledge in the public domain.

SELECTIVE IGNORANCE (STRATEGIC OR STRUCTURED IGNORANCE)

Selective ignorance occurs when specific information on a topic is produced but there is a failure to produce other information on the topic. This is unavoidable in practice since every aspect of a topic cannot be studied in detail due to time and funding. Full information on a topic can influence major public policy or cultural forces. It therefore becomes attractive for powerful interest groups to utilise selective ignorance on socially relevant topics and it is important for society to recognise this.

This form of ignorance is produced most often through (Frickel 2014a):

- "Secrecy (Rappert 2014)
- Censorship (Galison 2008)
- Deceit & suppression (Proctor 1995, Markowitz 2002)
- Denial (McGoey 2012b)

- Doubt (Oreskes 2011, Michaels 2005)”

In its simplest form selective ignorance could arise from clear social decisions to fund one area of research and not another, for example, cancer research rather than long term non-chemical based agricultural practice. Elliott (2015) and Hess (2016) point out that it is made complex due to the following:

- Mobilised counter-publics seek the prohibition of certain research, such as stem cell and cloning research (Kempner 2005).
- Scientists themselves may boycott work such as on weapons and armaments (Moore 2008).
- Religious or animal rights groups, etc. may require the banning of certain research.
- For complex topics, selective ignorance stems from a decision to study a topic in one way rather than others. For example, the chemical companies will readily fund studies on agricultural strategies using pesticides and fertilisers rather than more environmentally friendly methods (Elliott 2013, Lacey 1999). Health effect research from mobile phone EMR is not desirable and will be avoided by telecommunications companies.
- Selective ignorance can be created by collecting specific types of information, like a product’s benefits, rather than other information, such as harmful side effects.
- Selective ignorance can be generated by deciding to distribute, or publicise, certain information. This can explain why pharmaceutical companies spend much time and money in ‘publication planning’ to control the flow of information about a new product to decision makers (Sismondo 2007).
- Sometimes research is not explicitly banned but is selectively avoided by scientists who realise results/findings may be counter to the existing paradigm of the industry and have their funding reduced or knowing their research integrity may be criticised or defamed by industry-backed colleagues or vested interest parties.
- Some philosophers periodically note that there are many explanations of complex phenomenon by various scientists. Here, each scientist highlights one feature studied or understood by them while failing to cover other features with specialised understanding by others (Elliott 2013).

- Individual and societal avoidance of problematic information was highlighted by Heffernan (2012) through her work on wilful blindness.

Frickel (2014a) has illustrated “knowledge sequestration” where some knowledge is prevented from being circulated. There is a significant body of research which highlights how the industry and government have endeavoured to stop the flow of information in research and media (Martin 1986,1996,2007, Delborne 2008, Stocking 2015). Frickel (2014b) also highlighted political practices using poor methodology and lack of proper data storage.

However, as Frickel (2014a) points out, ignorance can also stem from “unintended consequences of social action.” The concern is that ignorance is produced by deviant science: “politically motivated or otherwise self-serving. The assumption leads logically to the troubling conclusion that more public transparency or scientific autonomy (depending of the source of interest) will reduce ignorance by rendering science less deviant.” But it must also be recognised that ignorance may be the “product of the structural pressures, institutional arrangements and the normative cultures that order everyday scientific practice and decision-making.” Examples of this are research funding, academic tenure funding, Honeybee Colony Collapse Disorder case (see later section in this chapter), and Frickel’s (2014b) Hurricane Katrina contaminated soil situation.

Frickel (2014a) cites examples of institutional production of ignorance in referring to the barriers encountered in dealing with the ‘sniff test’ done by the public for single point air samples (Mayer 2014), the seafood safety controversy following the BP oil spill in 2010 (Deepwater Horizon), and various other writings on disaster situations. The approach by the government authorities addressing such situations is not through the use of latest and appropriate scientific testing and specific evaluations, but rather through the use of off the shelf, traditional methods, some of which are many decades old. The shortcomings of such approaches leave the public generally in a “knowledge vacuum” as will be seen in the following examples.

Elliott (2015) has suggested some ways of avoiding selective ignorance in studies by firstly recognising the nature and significance of selective ignorance.

- This type of ignorance can be produced from study design aspects such as what end points to test for, the statistical analysis method, the terminology to use. These can result in knowledge in particular areas and ignorance in other areas.

- The reliance on traditional practices, while appearing to be value-free, may result in marginalised groups not being considered (Code 2012).
- Although selective ignorance can be produced by not researching particular areas, it can also be produced by the failure to make known particular information.
- Societal impacts can be significant, for example, in agriculture, the decision by farmers to use antibiotics and systemic pesticides in animal meat production exposes the consuming public to these, yet the farmers are only focused on animal sales. In pollution it is quite often selective to ignore population exposure to toxins, such as was seen in the biosludge example previously mentioned where the US EPA decided to selectively ignore possible health effects from the biosludge reuse.

Some ways to avoid selective ignorance in the environmental health area can be:

- By promoting social activism on science and technology. Public activist groups are generally acutely sensitive to areas of undone science.
- By encouraging new paradigm development in research decisions. For example, in the case of pollution, a ‘cradle to cradle’ design approach can eliminate waste (McDonough 2002) and ‘safety by design’ can reduce the use of industrial chemicals (Maynard 2006).
- By providing government funding for socially relevant research, and by collaborative research planning between industry, government and NGO representatives.

Some examples of selective ignorance follow. These examples firstly cover specific cases and lastly touch on evergreen science policy and biomedical adverse event under-reporting.

Agricultural Research

Most agricultural research to date has been focussed on how to increase crop yields. Orthodox views are usually to the effect:

Our research is improving productivity, profitability and sustainability in cropping, livestock production, aquaculture, horticulture and the food industry (CSIRO 2021)

In predominantly concentrating on productivity, ignorance can be generated on how agricultural practices affect rural communities, their environment and the nutritional value of the crops grown. Reports on agricultural contribution to Gross Domestic Product can be viewed

as one way to measure this area but ignores impacts on social equity and the environment (IAASTD 2009p7). This was recently highlighted in a report by the International Assessment of Agricultural Knowledge, Science, and Technology for Development (IAASTD 2009). The decision to attention agricultural problems via scientific and technological studies rather than political or social changes can also create ignorance in how current problems may be generated from social structures (Elliott 2013, Patel 2007p121).

The drive by agriculture for increasing yields from the land has seen hybridization for size and growth rates, the use of pesticides, synthetic fertilisers, and GMOs. Farmland is no longer rested, being fed with an artificial chemical cocktail to enable the continual tilling, producing serious mineral depletion. Pesticides such as Roundup have, at least initially, been effective against pests, resulting in farmers becoming complacent. So rather than crop rotation, a traditional practice to reduce pests and replenish the soil, the same crops have been planted year after year, relying on pesticides to keep pests at bay. Systemic insecticides, promoted as far less toxic to the environment than pesticides of the past, further add to this phenomenon. But they have been suspected of causing long term colony collapse disorder decimating the bees, essential for crop pollination.

The agricultural pesticides “resulted in widespread contamination of agricultural soils, freshwater resources, wetlands, non-target vegetation and estuarine and coastal marine systems”, meaning that many organisms in those habitats were “repeatedly and chronically exposed to effective concentrations of these insecticides” (Van der Sluijs 2015p148). We now not only have food from such farming practices with chemical residues but also the contaminated marine food sources from the run-off of such agricultural land.

The drive to increase yields in food production has seen yields on onions and nectarines up about 200%; celery and garlic, 250%; broccoli, beets and cantaloupe, 300%; tomatoes and almonds, 500%; strawberries, 550%, and similar increases in other crops (Schatzker 2015). The result is a “dilution effect” recognising that water and carbohydrate content has risen, with protein, vitamins and minerals substantially reduced in most of these crops, e.g., reductions in calcium, magnesium, copper reduced by 80% between mid to end 1900s (Mayer 1997). Researchers studying the drop in nutrient levels in food hypothesise it is due to lack of soil microbial diversity due to pesticides, GM plants, soil erosion, seed quality decline, and air and

water pollution. This results not only in contaminated food, but also low quality in respect to nutrient and mineral content for optimal health.

The potential effects on health from residues and the low mineral and nutrient contents in agricultural crops represents ignorance areas in the modern farming aims of only focusing on productivity and profitability. It represents an example of selective ignorance.

Air Pollution

A typical helpful website comment in respect to air pollution is:

If the air quality is especially poor, it may take a few days for your body to recover. And if you're regularly exposed to high levels of unhealthy air, the health consequences can linger for months or even years (NIH 2011).

In 2018, the US EPA released its 2014 National Air Toxics Assessment (USEPA 2018). Many areas in the US were found to have significant air pollution levels, especially those with chemical plants nearby. To some of the identified communities such pollutant levels explained the high rates of health problems including cancers. Lerner (2019) studied the US EPA on its actions on the problem in the affected communities. Two areas were compared. One was the affluent community of DuPage County which had cancer incidences higher than the EPA's "upper limit of acceptability": 100 cancers per million people. The county, in its highest polluted area, had 282 per million. The most prominent pollutant was ethylene oxide, a colourless gas. This could be tracked to a local plant. The chemical is linked to problems in the respiratory tract, reproduction problems, memory loss, headaches, and certain cancers such as non-Hodgkin lymphoma, leukemia, and breast cancer. In one suburb, Willowbrook, the EPA briefed local officials the same day the report was published and in less than a week distributed information to the residents on the risks they faced.

The report however indicated the most highly affected area was St. John the Baptist, a small African-American community of low socio-economic status, on the Mississippi River in Louisiana. The report indicated a cancer rate from the ethylene oxide gas of 317 per million. However in the worst area of this parish there were 45 pollutants that caused serious health problems, including cancer. Combined, these chemicals gave the residents the highest risk of cancer in the US: 1,505 per million.

But the St John residents didn't learn about these risks until eight months after the release of the report. There were no EPA representatives briefing the local officials and public or information distributed to the public as done for Willowbrook. Residents of St Johns were victims of selective ignorance.

Synthetic Vitamin Supplements: B12

The area of vitamin supplements is another area where selective ignorance is used by the industry concentrating only on delivery of the vitamin molecule. Many commercial vitamin supplements may theoretically contain the correct, or similar, vitamin molecule however they are usually delivered attached to other molecules to ensure the product's stability.

Most orthodox approaches for vitamin B12 supplementation are as below:

Cyanocobalamin injection is used to treat and prevent a lack of vitamin B12 that may be caused by any of the following: pernicious anemia (lack of a natural substance needed to absorb vitamin B12 from the intestine); certain diseases, infections, or medications that decrease the amount of vitamin B12 absorbed from food; or a vegan diet (strict vegetarian diet that does not allow any animal products, including dairy products and eggs). Lack of vitamin B12 may cause anemia (condition in which the red blood cells do not bring enough oxygen to the organs) and permanent damage to the nerves. Cyanocobalamin injection also may be given as a test to see how well the body can absorb vitamin B12. Cyanocobalamin injection is in a class of medications called vitamins. Because it is injected straight into the bloodstream, it can be used for people who cannot absorb this vitamin through the intestine (MedlinePlus 2010).

Most commercial forms of B12 supplement, especially those made by larger pharmaceutical companies, are in the form of a synthetic molecule: cyanocobalamin. This consists of a cyanide molecule bound to the cobalamin (B12) molecule. In this form, it is relatively cheap as it can be recovered from sewage sludge or tissue of mammals using potassium cyanide (Miner 1953, Melle 1959) or produced through total chemical synthesis. In this form, such a molecule will be broken down inside the body producing the B12 molecule, but also the cyanide molecule which is quite toxic, and if a person's detoxification mechanism is overloaded with other toxins or is epigenetically underperforming this may result in serious problems. It is interesting to note that

if one is poisoned by cyanide, a common treatment is to administer hydroxocobalamin which readily binds to cyanide to form cyanocobalamin for easier elimination from the body (Shenvi 2016).

The toxicity of cyanocobalamin has been highlighted in the past (Freeman 1992, Matthews 1997). It is classified as a hazardous air pollutant known or suspected to cause serious health problems (Toxnet 2019). It was linked with aggravation symptoms of bowel inflammation by shifting the microbiota towards a more inflammatory profile (Zhu 2018, Xu 2018).

Dietary contributions of B12 seem to be far superior (Greibe 2018), and probiotic supplementation to enable bacteria to produce B12 has also been indicated as preferable (Molina 2009). Natural sources include white button mushrooms, spirulina and chlorella.

Some causes of B12 deficiency include acid blockers for reflux, and microwave cooking due to its significant deactivation of B12 (Watanabe 1998) and many other vitamins.

As seen from the above, selective ignorance has been employed to sell a synthetic version of a vitamin that can be safely obtained from more natural sources. The ignorance has been generated by ignoring other possible side effects from the synthetic vitamin, in the case of B12, by its binding to a toxic molecular structure for its stabilisation.

Rational Selective Ignorance

The terminology that calls ignorance rational may at first seem paradoxical. How could it be rational to avoid knowledge? But in today's lifestyle it is quite normal to employ rational ignorance so that one's limited energy, time and cognitive capacity can be applied to activities deemed the most important. In employing rational ignorance in this way it becomes beneficial. It becomes a "knowable known unknown" (Roberts 2015p362). However there are times when the decision to forego knowledge acquisition can be harmful.

Somin (2015p274) makes the following definition:

A person is rationally ignorant whenever he or she has decided not to learn some body of knowledge because the costs of doing so exceed the benefits, based on the decision-maker's own objectives. Finding, studying, and assimilating information is a costly activity, not just in terms of money, but in terms of time and effort as well.

Rational ignorance has no tie with moral principles. The rationality is simply relative to the decision maker in terms of maximising their objectives (Becker 1976). Somin (2015) gives the example of a mafia boss devoting his time and energy to aspects that will enable his protection racket to operate efficiently rather than studying for example, ethical philosophies that could make him a better person.

The regulatory practice of extrapolation of data is usually attributed to governmental underfunding but is commonly favourable to industry. Frickel (2014b) highlighted this in the US EPA's analyses of soil contamination following Hurricane Katrina: the risk assessments of 70% of the 141 contaminants were extrapolated from a relatively small number of tests (most of the chemicals had never been tested for cancer risk). Another example is in nanotechnology: the nanoscale chemicals are presumed safe by the regulatory authorities due to their 'similar' structure to existing chemicals (Hess 2010, Lamprou 2011).

In studying the risk assessment for the Hurricane Katrina resultant land contamination in New Orleans, Frickel (2014a p271) noted that there was a large amount of undone science on cancer risks for many of the chemicals that were present. The regulatory authorities had assumed numerical values for the lifetime carcinogenic risks. These were then used and in the risk assessment became indistinguishable from values established from actual studies: "This is one way that ignorance is simultaneously produced, hidden from view, and institutionalised as meaningful regulatory science".

There are two types of ignorance that should be distinguished as similar to but different from rational ignorance: inadvertent and irrational ignorance.

Inadvertent ignorance

This is where a body of knowledge is overlooked by a person who has no idea that it exists (Friedman 2005). This is frequently experienced by many MCS sufferers, at first unaware of the MCS condition, who are treated for their symptoms by doctors, also ignorant of the officially unrecognised condition. Here, rational decisions to avoid information on MCS were never deliberately made due to their general ignorance of such a health condition.

A good example of inadvertent ignorance is that of fragrances. In 2015, an award-winning film, 'Stink!' by Jon Whelan, explored why there are toxins and carcinogens legally hidden, by design, in consumer products. In one example, as synthetic clothing has an obnoxious smell,

dangerous fragrance chemicals are normally added to the clothing. Laundry detergent and fabric softeners also add fragrance to clothing. While this may be a dual example of inadvertent ignorance on the part of the clothing manufacturers, it is normally the case for the public. Fragrance chemicals remain unregulated in most countries.

A fragrance product is usually made from between 100 to 350 ingredients. These products are present in detergents, air fresheners, cleaners, lotions, perfumes, nail enamel, baby wipes, shampoos, and other fragranced consumer products. Many of these ingredients are chosen for their ability to readily vaporise into the air to produce/distribute the scent. Most of these then attach themselves to the surroundings including clothing and hair. The ingredients include many toxic chemicals, for example, diethyl phthalate is used to make the scents last longer: it is a skin sensitizer, a reproductive toxin, and has exposure limits due to its use as a toxic plasticiser in plastic products. However, manufacturers are not required to disclose the ingredients as the industry has argued that it is proprietary information.

Most, and sometimes all, of the testing of fragrance chemicals is done by the industry and not published. The European Commission Scientific Committee on Consumer Safety (SCCS) reviewed studies on some fragrance materials submitted by the industry and found scientific shortcomings, such as invalid test protocols, incomplete data, test substance lack of identification, , appropriate controls not used, etc. The SCCS many times commented that a reliable conclusion of safety could not be formed from the data submitted (SCCS 2014, 2015). This was highlighted by the Women's Voices for the Earth community group (WVE 2015p9). They looked at 3000 fragrance ingredients with some of the findings being:

- “190 fragrance chemicals have been assigned the signal word ‘danger’ for their Safety Data Sheet”.
- “1,175 fragrance chemicals have been assigned the signal word ‘warning’.
- “44 fragrance chemicals require pictogram GHS06 of a skull and crossbones to indicate acute toxicity”.
- “97 fragrance chemicals require pictogram GHS08 indicating the chemical is a hazard to human health”.
- “The International Agency for Research on Cancer (IARC), a division of the World Health Organization, lists seven of the ingredients as possible human carcinogens.”

- “15 fragrance chemicals are prohibited from use in cosmetics in the EU”

With the inherent toxicity and level of fragrance exposures in modern living, it becomes a nightmare for those who are sensitive to these xenochemicals. A recent study has found in four developed countries that 32% (1 in 3) of the population report fragrance sensitivity, 26.0% have asthma/asthma-like problems, of which 58% have fragrance sensitivity. The report of autism/ASDs in 4.5% of the population, saw 76% of these having fragrance sensitivity. In this same study 19.9% of the population reported chemical sensitivity generally and of these 82% had fragrance sensitivity with 44.1% with chemical sensitivity experiencing severe and disabling health effects from fragrances (Steinemann 2019).

Yet, the majority of the population who do not experience, or link, health problems with fragrances, are inadvertently ignorant of such a sensitive significant part of the population (one in every three), or the toxicity of the chemical mixtures that make such fragrances.

The above consideration of fragrances represents examples of inadvertent ignorance where most users are not aware of the effects of such products on their own health as well as others, especially those that are sensitive to xenochemical exposures

Irrational Ignorance

Irrational ignorance occurs when an individual avoids information that may be counter to their beliefs or goals. ‘Motivated scepticism’ may lead an individual to avoid information that simply first appears to be counter to their belief yet may have been useful to achieve their goals (Taber 2006).

In the early 17th century, ‘scientific’ concepts were based mostly on theological and philosophical explanations, with the concept of ‘experimentation’ still in its formative stage. When the physician William Harvey announced his theory that blood circulates through the body based on his experimentation, comparative anatomy and calculations, it caused much controversy. Beliefs prevailing at the time were based on 1400-year-old teachings of Galen, denying the presence of circulation, so many opposed Harvey’s theory. Experimentation was still questionable as a concept, there was no direct proof that capillaries existed and the clinical applications of the theory were not realised (Lubitz 2004). Personal resentment and professional "territorialism" also played a role, and so was an example of irrational ignorance.

Given the vast amounts of knowledge available today it is very difficult to become an expert in more than one field, especially when the fields are not directly related. This has led to specialisation, especially in medicine. The specialisation leads to economy of information gathering for society members and the resultant recognition of experts in the various fields. This is to such an extent in society today that public ignorance in some scientific areas is quite significant. For example, in a survey of a sample of Americans and Europeans it was found that 20% do not realise the earth revolves around the sun (Somin 2013). This degree of ignorance can be rational for these people as they can progress their daily lives without such knowledge.

The extent of rational ignorance of the public on medical matters has been exploited by the pharmaceutical and chemical industries. The public reliance on the government to regulate the industries and ensure the public is protected is highly related to their rational ignorance on such matters.

This thesis has repeatedly demonstrated the inter-relationships between body systems and organs producing multiple symptoms from an environmental exposure. Yet the conventional medical system is so specialised that each specialist looks at the effects only from their specialised viewpoint. One such example is with MCS which spans many specialist areas such as:

- Toxicologists miss the myriad of subtle flow-on and long term effects, as they focus on short term physical effects found from lab test animals.
- Mental health practitioners who use talk based therapies do not consider interrelated pathological routes.
- Allergists treat some symptoms displayed, which can be typical of IgE mechanistic systems, even though the same symptoms may be caused through other mechanisms.

The medical system appears at times too specialised, creating elements of rational and irrational ignorance, to put together the whole story of integrated and interrelated effects of environmental exposures.

The following are examples of rational ignorance in respect to environmental effects.

Food Preservatives

There was an era where the food industry applied rational ignorance only so as not to interfere with profitability and is different today only that the rational ignorance now overlaps with of other types: manufactured, organisational, and selective ignorance generated by the food industry.

In the late 1800s, food companies started to make use of industrial developments in the field of chemistry. The transport of food without refrigeration in those days presented quite a large problem, so the use of chemicals which could preserve food for days/weeks was very attractive. This saw the use of chemicals such as formaldehyde, borax, and salicylic acid. It is estimated that thousands of children every year in New York alone, were killed by the formaldehyde in milk.

Furthermore, additional uses for chemistry started to be used such as saccharin to replace sugar, acetic acid instead of lemon juice, lab-created alcohols dyed and flavoured to mimic aged whiskeys and wines, glucose being sold as expensive maple syrup, etc. (Blum 2018). As there was a stark absence of any government regulations on such aspects, the industry had free run.

Early in the 1900s the US Agriculture department conducted an experiment on a group of young men who were fed a diet of food with typical preservatives to monitor their effects on human health long term. They became known as the Poison Squad. By the termination of this group's existence, those that hadn't already withdrawn due to various sicknesses, were very unwell. The formaldehyde used in dairy products, had strained their kidneys, with other chemicals, such as benzoates causing extreme weight loss and blood vessel damage. One of the members subsequently died from tuberculosis which, according to his family, was due to him being considerably weakened by these experiments.

In her book, Blum covered the subsequent battle by citizens/activists, journalists, scientists, and women's groups with industry in finally achieving the US 1906 Food and Drug Act. But unfortunately, due to various loopholes in these regulations, there are social and political challenges being faced today where there are thousands of chemicals added to foods creating rational and irrational forms of ignorance depending on the frame of reference: rational

ignorance from the chemical additives industry, and irrational from the consumer viewpoint in knowing chemical additives are being used but believing they must be OK for us to consume, otherwise they would not be used.

Estimates indicate that today the average person consumes 3.6 to 4.5 kg of food additives per year, but actual values are likely to be greater (Zengin 2011). Some examples of additives are:

- Sodium benzoate (additive E211). This was one of the chemicals found to have effects as far back as the poison squad yet is still being extensively used. This chemical is used for its antibacterial and anti-fungal effects in preserves, sauces, beverages, fruit juice, condiments, dairy products such as ice cream, yoghurt, pudding, etc. It can cause sickness such as nausea, vomiting, rhinitis, diarrhea, migraine, bronchospasm, anaphylaxis, and hyperactivity in children (Tuormaa 1994). It has been shown to be linked to human DNA damage (Zhang 2013, Pongsavee 2015). It is considered genotoxic, clastogenic, and neurotoxic (Linke 2018).
- Potassium bromate (additive E924), is typically used as a flour improver to strengthen the dough for higher rising to fluff up breads, buns, and pizza crusts. It is rated as a 2B human carcinogen. It can produce toxicity in multiple organs in both humans and animals (Farombi 2002, Kujawska 2013, Ahmad 2015) and is irritating and injurious to various other tissues such as in the kidneys and central nervous system (Robert 1996). Effects shown in animals include cancer and mutagenic effects (Kurokawa 1987). It has been banned in several countries. There is clear evidence that it degrades the main vitamins in bread thereby degrading its nutritional quality (Sai 1992).
- Nitrates and Nitrites (249 Potassium nitrite, 250 Sodium nitrite, 251 Sodium nitrate (Chile saltpetre), 252 Potassium nitrate (saltpetre)). These are used in processed meats such as ham, bacon, corned beef, saveloys, hot dogs, salami-type sausages, devon, luncheon rolls, fish, cheese, poultry products, etc. as a preservative. There are diverse health effects linked to nitrates and nitrites:
 - The American Medical Association has highlighted that due to the gastric tract composition in infants, they are particularly vulnerable to methemoglobinemia (USFDA 2011). This is a blood disorder in where an abnormal quantity of methemoglobin is produced: a form of haemoglobin: a red blood cell protein distributing oxygen.

- The American Medical Association also linked gastrointestinal and neural cancer to these chemicals.
- The International Agency for Research on Cancer classified processed meat (including cured, salted, or otherwise altered meat for preservation or flavour) as “carcinogenic to humans” (Bouvard 2015).
- Higher risks of childhood brain tumours in offspring have been linked to maternal consumption of nitrite-cured meats (Grosse 2006, Pogoda 2009).
- A non-monotonic dose response curve is applicable to nitrate exposure. It is also an endocrine disruptor producing effects depending on species, age, reproductive state, sex, exposure duration, dose, rate of metabolism, excretion ability, habituation, environmental conditions such as hypoxia and acidity, method of testing (in vitro or in vivo), and end points being studied (Edwards 2018).
- Human mania and brain gene expression and behaviour in rats have been associated with nitrated meat products (Khambadkone 2018).
- Processed meat consumption and mortality, in respect to cardiovascular diseases and cancer have been linked (Rohrmann 2013).
- The risk of colorectal cancer was linked with processed meat consumption and people with certain genotypes. In one study on diet effects, in this case with processed meat, a novel gene-diet interaction was shown. This highlights that genetic variants may be modified by diet and affect disease risk (Figueiredo 2014).
- Increased risk of heart failure was shown in men with low to moderate consumption of processed red meat compared to unprocessed red meat (Kaluza 2014).
- The exacerbation of asthma symptoms has been linked to cured meat intake (Li 2017).

Health authorities regard nitrates and nitrites as necessary due to their inhibition of toxic micro-organisms. Their argument is that without such there would be a higher death rate from food poisoning. Alternatives have not actively been pursued although there is some evidence of various spices as well as food-radiation treatment are being evaluated.

Industry spokespersons can sometimes point out that the recommended beet health drinks also contain high nitrate levels. However nitrates in cured or processed meats have converted to the dangerous nitrosamines after heating or cooking. In consuming beets, the body converts the nitrates into nitric oxide, or NO, through what's termed the 'nitrate-to-nitrite-to-nitric oxide (NO) pathway' which is vital for one's health (Gallardo 2018).

The above examples of the many food preservatives show that there was an era originally where the food industry applied rational ignorance only so as not to interfere with profitability. Today however, the food industry can be said to apply a small degree of rational ignorance to produce long shelf life, but it now, due to the increased knowledge of preservatives' toxicity to human health, significantly overlaps with of other types: manufactured, organisational, and selective ignorance.

AGGREGATED IGNORANCE

I have developed the term 'aggregated ignorance' due to its relevance to this thesis topic. This ignorance form is bidirectionally used by industry and government departments depending on the problem. On one hand results or statistics are aggregated together to dilute problem areas/results with non-problem areas/results. This was seen in the previous example of localised environmental effects on the population near the Louisiana petrochemical industry along the Mississippi river, where "industrial parishes are 'diluted' by non-industrial parishes, making the determination of elevated cancer rates near chemical plants impossible to decide" (Allen 2005p469). On the other hand, it may become advantageous not to aggregate effects, through similar chemical properties or possible synergism when commenting on exposures to a number of toxins. It is deemed better to consider each toxin on its own in order to progressively dismiss any possible health effects: this is the approach of the xenochemical regulators as well as in situations of chemical contamination or pollution.

Aggregated ignorance also has the potential to be created through autocorrelation effects. This can be initiated where one expert is not aware of an undone science area in the study of safety aspects of a chemical exposure and has offered an opinion of safety with that particular

chemical. This can then act as a springboard for other experts if also unaware of such undone science, to form similar views and look less critically at what studies have been done. This may be especially so where such experts may be influenced by industry interests either initiating or progressing the adoption of similar views. So potential consensus situations may develop representing another form of aggregated ignorance.

Frickel and Kinchy (2015) mention a case where ignorance can be generated by the size of one's reference frame on certain topics. The example of the gas fracking issue was used to highlight this. Initially the move from coal to gas as an energy resource was endorsed as a move in the right direction by environmental groups in relation to greenhouse gases and climate change. Prominent environmental activists originally only spoke of gas use on national and international levels. It was not until localised problems, visible at a scale of everyday rural life: wells for drinking water, streams, dams, roads, and landscapes, that the prior ignorance started to become visible. The large environmental groups then had to admit their former support had been based on ignorance of the consequences of fracking at a regional or local scale.

The industry response on the fracking issue was different. The health authorities and industry avoided studies which aggregated water quality results. This way larger, perhaps clearer, patterns were not obvious and problems were isolated and stayed local.

Yet industry and health authorities frequently work in the opposite direction of the avoidance described above for other problems. This can occur for localised environmental effects on the population as seems to be present with the Louisiana petrochemical industry being concentrated along the Mississippi river (Allen 2003, 2005, Frickel 2015). In this situation the cancer incidence data, collected in detail by government health authorities, is divided into ambiguous disease categories, averaged over time and combined into multi-parish regions. "Industrial parishes are 'diluted' by non-industrial parishes, making the determination of elevated cancer rates near chemical plants impossible to decide" (Allen 2005p469). To add insult, the health authority's website offers a comparison with state and national data to suggest that living near petrochemical industry in Louisiana is less risky for most cancers nationwide. This top-down system used by health authorities will erase knowledge about vulnerable communities defined by geography and occupation and based on personal biological data (Roberts 2015).

This type of ignorance generated by the very authorities that are charged in protecting the public is seen time and time again. The most common theme encountered is the consideration of the effect of thousands of xenochemical exposures on human health by considering each on an individual basis ignoring combined effects, potentiation, and synergism in the real world. Here the aggregation of all exposures is avoided so that comparison to ideal lab environments of test results of each chemical on its own can be made to selectively focus on chosen health effects, ignoring hundreds of other effects such as endocrine system disruption. Each xenochemical is progressively dismissed with the huge amount of undone science contributing. This also carries through to the legal system.

Conversely, aggregating chemicals together is seen through the human biomonitoring results from the CDC, through the National Health and Nutrition Examination Survey. Whilst acknowledging the amount of chemicals the population today is exposed to and retains, its lack of geographic or worker-specific, etc. break-up implies we are all exposed fairly equally yet this may be far from the actual case.

Consequences of Aggregated Ignorance

As highlighted in other sections in this chapter, once one toxic product has finally been studied enough to convince the regulators to act, it is quickly replaced by one or more new chemicals. One example that has been unfolding in recent years has been the striking similarity between PCBs banned in many countries, and the subsequently introduced polybrominated diphenyl ethers (PBDE) group of chemicals. The authorities have simply ignored the similarity of these two groups of chemicals, acting on the earlier PCBs, but waiting more than forty years for a 'weight of evidence' to accrue on the new chemical group.

The two groups of chemicals, PCBs and PBDEs, are so chemically similar that one wonders why regulatory authority scientists did not suspect similar health problems with the new chemical group as per the former PCBs (Cranor 2020).

Children are particularly susceptible to PCBs during the prenatal and postnatal periods (Jacobson 1994). Neurological problems such as lower IQs, as well as hearing problems are known effects from PCB exposure. There are various studies, linking mothers who consumed PCB contaminated fish, to children with learning and memory problems (Kuratsune 1972). Thyroid uptake in pregnant women can also be affected by PCBs also indirectly adversely

affecting the neurological development of developing fetuses (Woodruff 2008). But the most significant aspect was that the effects eventually seen in the children were seen in animal studies: mice, rats and monkeys. In-utero and neonatal animal exposures demonstrated impaired cognitive function and learning, sensory deficiencies and even ADHD (Kodavanti 2005).

After decades it is emerging that PBDEs do have similarities to the PCBs in relation to chemical and biological activity. Animal studies done on the effects of PBDEs resemble the effects of PCBs with a body of scientific evidence on this (Kodavanti 2005). In 2009 there was evidence that PBDEs could be four times more concentrated in Australian children than adults (Toms 2009). High concentrations of PBDEs have been found in breast milk, dust and food (Costa 2007). Although few studies have been conducted on children, it has been found that prenatal exposure significantly correlated with decreased cognitive function, decreased motor function, and increased behaviour problems (Hudson-Hanley 2018). Even the ability to fall pregnant has been associated with PBDE presence in women (Cranor 2014, 2020). The companies producing PBDEs had argued that animal studies were not conclusive proof that the same effects will happen in humans, arguing for more testing. This delayed action on this chemical group for years with the US EPA only recently showing concern over the chemical group after human studies started to mount showing similar effects in humans as it did in animals, as it had with PCBs (Cranor 2020). PBDEs are now ubiquitously present in the environment and in everyone's body (Schechter 2005). When they were first proposed to be used, when there were none in human bodies, this could have been avoided.

But PBDEs are also similar in many respects to other chemicals such as hexabromocyclododecane (HBCDD) and the PFAS/PFOS groups. One may beg to ask the question as to how many children must be affected for action to be taken, and why the precautionary principle wasn't applied in view of the chemical similarities to prevent such suffering in the first place. The scientific approach employed by the authorities to study one chemical at a time, appears to have significant shortcomings.

Further effects of aggregation ignorance are seen globally by the contamination of the global environment with persistent chemicals taking decades or centuries to degrade, for example DDT, BPA, glyphosate, mercury, and flame retardants. If a developed country starts to ban a

certain chemical the industry usually starts manufacturing in a less regulated developing country with the chemical still entering, distributing and contaminating the global environment including eventually the country that banned it. The US banned DDT in 1972, yet in 1996 it was exporting 1 ton of DDT per day (Wargo 1996). DDT then returns as imported food residues.

Localised Environmental Disasters

Aggregation is also seen in chemical disasters, such as Bhopal, Minamata, Seveso, and in local contaminated site issues. In these situations both industry and government treat them as a local issue, yet such chemicals will eventually disperse through the environment and add to the global persistent residues. A company may be prosecuted for polluting a local river but not for adding to the pollution of the planet or the human species. The treatment of each contamination case, that the public is usually responsible for highlighting, on a case-by-case basis will not solve the aggregated effect of filling the planet with rising background levels of many chemicals. The public is never given a choice in contamination. As industry is not 'affected' by contamination, the governments are the logical actor. More visionary and co-operative international action is required earlier rather than later.

The Stockholm Convention on Persistent Organic Pollutants was established in 2001 by the UN to address the chemicals that persist in food and environment. The Convention has 182 parties involved. After almost half a human generation only 28 substances have been banned or restricted from the tens of thousands of toxic and carcinogenic chemicals released globally. This however only applies to those countries who have signed and ratified the convention.

In the USA, the EPA has banned five chemicals in a 34-year period (Ruiz 2010) from the approximate 84000 used in public use available chemical products.

Cribb (2014p173) fittingly sums up:

Regrettably, the widely used phrase 'dirty dozen' appears to have created an impression in the minds of many citizens that the number of toxic chemicals is very small, and hence fairly easily reined in: however in reality there are in excess of 30000 known or suspected toxic substances totalling ten million tonnes in volume (according to the UNEP's 2012 data), plus a host of unintended pollutants created by mining, manufacturing, energy production and other activities.

Contamination keeps being regarded as a specific localised problem. But all persistent chemical uses keep adding to the global background levels and is the reason for POPs found in such places as the ocean depths and Antarctica. Cribb (2014p181) likens the situation as follows:

The task of international regulation of dangerous chemicals may in a sense be compared with an attempt to regulate traffic flow in a large city one vehicle at a time, instead of requiring road rules and traffic lights to be observed by all.

The Cooperative Research Centre for Contamination Assessment and Remediation of the Environment (CRC CARE) “brings together industry, government, science and engineering to prevent, assess and clean up environmental contamination.” One of its founders, Professor Naidu (2018) has stated:

Soil pollution is particularly insidious. It harms us when we eat food grown in contaminated soil, it poisons water that flows into dams and catchments, and people working with soil – or children playing in it – can be exposed directly.

Contaminated soil also disrupts the ecosystem services – the benefits that humans freely gain from the natural environment, such as water supply, erosion control and food production – upon which we rely for our quality of life.

We must also place soil pollution in the context of chemical contamination of our environment more generally, including air and water. In its scale and global implications, it is the most underrated, under-investigated and poorly understood of all the essential risks facing humans in the 21st Century.

I estimate that the impact of chemical contamination upon Earth is five times as large as that of climate change. Humans are the cause of this problem, and humanity must work together to solve it before it is too late.

Chemical disasters such as Minamata, Bhopal, Seveso, Erin Brockovich’s chromium VI in drinking water, and Fukushima have typically caused deaths and suffering of thousands of people. If one person poisons another then the offender may be punished by imprisonment. In contrast, corporation management and government officials responsible for such disasters are rarely brought to justice. This has been due to the typical situation where the poisoned victims must establish a clear technical link between the chemicals released and the health problems

experienced against the combined efforts of industry and government to hinder and discredit them.

The typical situation in a chemical disaster, as mentioned by Cribb (2014) is:

- The offending company “digs in, denies responsibility and fights through the courts”
- This leads to frustration, anger and hostility from the public and environmentalists
- “governments frequently seek to muddy the waters for their own protection, rather than seeking to resolve the problem.”

The above situation combined with the suffering and death of victims, the real or perceived lack of justice, and the costs to fight a legal battle, not surprisingly results in an outraged public and angry green groups. In this situation of wrath, blame and confrontation it is difficult to arrive at satisfactory solutions for all sides. The affected public often sees no compromise due to the perceived injustices experienced by the victims. Frequently while all this happens, the products that have caused harm may remain in use and circulation. At a personal level, is it perceived as fair to seek a compromise with a person who has been has poisoned, or a child affected the rest of their life? The directors and managers of the offending companies are far removed from being personally responsible: it is the company that is fined, no person from the companies are jailed or individually fined.

So aggregated ignorance has been extensively used by industry and government to suit the case at hand and becomes an important ignorance type in the area of environmental effects on health.

ORGANISATIONAL IGNORANCE

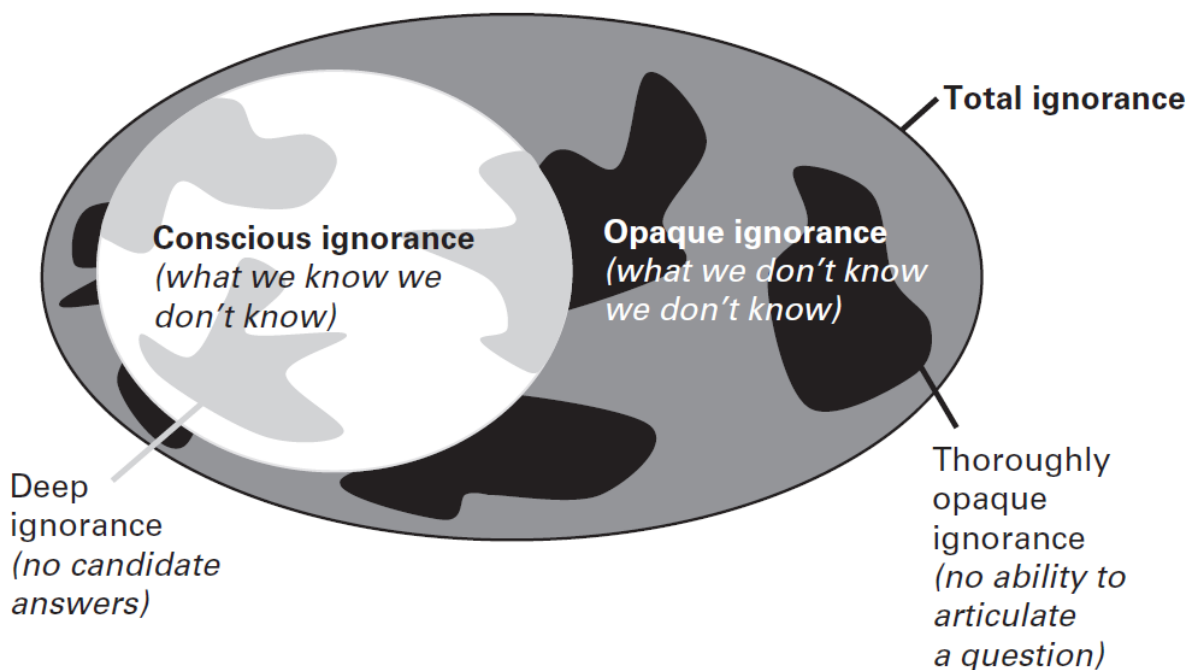
As seen from the above discussion of various forms of ignorance, there are many overlaps. This will again be seen in this section which covers ignorance from a social organisation viewpoint yet there are distinct characteristics. It is pertinent since it is both the industry and governmental organisations that are central to the topic this thesis covers.

If an organisation’s management has no awareness of the various forms of ignorance, then they will lack the foundational first step that is needed to manage unknowns.

In the business world there have been many views that the global financial crisis and the collapse of Enron were due to intelligent people choosing to ignore consequences of their collective behaviour (Elkind 2005). Individual and societal avoidance of problematic information was highlighted by Heffernan (2012) through his work on wilful blindness. Let us consider some of the types of ignorance information that can arise, pertinent to this thesis topic.

Unknown Unknowns

This type of ignorance is a state of total absence of knowledge and there is no awareness of such ignorance. It has been recognised for some time and has been originally referred to as “ignorance squared” or “ignorance of ignorance” (Ravetz 1993), also “opaque ignorance” (Wilholt 2019) or simply unknown unknowns. The earlier shown figure by Wilholt is reproduced below.



The above figure represents the landscape of ignorance from Torsten Wilholt (2019 fig8.1). Discussion of the opaque area (unknown unknowns) is present in the Organisational Ignorance section further on.

Unknown unknowns (or opaque ignorance) can exist for any organisation and represents ignorance at a point of time usually for organisation members. In many companies dealing with

high-risk research and development, such as in aerospace, an awareness has long been realised and sometimes referred to as “unk unks” (Longstaff 2005).

Unknown unknowns can be discovered through the organisation embarking on research and development, employment of new staff, new equipment or consultants. Feedback from customers or suppliers or observing competitor actions can also lead to such discovery. Speculative thinking and scenario and foresight studies by management can also transform them into known unknowns (Shoemaker 1995, Loveridge 2009).

The employment of new staff particularly ignorant of established knowledge in an organisation can be used to advantage. For example, Orson Welles produced *Citizen Kane* (1941) without any previous filmmaking experience. His ignorance enabled his work without boundaries of existing knowledge to achieve innovative approaches still used to the current day (Wheldon 2002). New staff, employed for their specialist knowledge and ignorance of the organisation procedures and approaches may question these in a way that normal staff would not. In recognising the potential of resultant questions and comments, it may lead to beneficial changes and knowledge creation. In these examples however ignorance combined with existing organisational knowledge has been key to good outcomes.

Unknown unknowns can be a relative concept. For example, a pesticide or food additive may have undergone risk assessment or cost-benefit analysis based on current knowledge and known unknowns, however when a health or environmental crisis occurs this is usually attributed to unknown unknowns by the assessment authorities. Yet it may have originated due to undone science: the known unknown areas of study. The public may be given incorrect facts and half-truths about the situation. This common situation is where knowable unknowns are claimed to be unknowable unknowns as a public relations strategy.

Known Unknowns

This ignorance is where there is knowledge on knowledge limits: there are aspects that we know we have no knowledge about.

Known unknowns can be handled by organisations similarly to unknown unknowns, for example, R&D, employment of new staff, new equipment or consultants, customer or supplier feedback and observing competitors.

This ignorance enables organisations to focus its resources: its priorities can then determine how much of such ignorance should be addressed. At a simplistic level this may entice the organisation to sub-contract activities which are outside its management knowledge.

For the case of products potentially causing public health problems, it may be decided, commonly by industry and regulatory authorities, that such known unknown areas may be best left with no research undertaken.

Knowable Known Unknowns

Such a situation occurs where something is knowable if sufficient resources, or even motivation, is allocated, rather remaining simply as a known unknown. This is rational ignorance.

Mobilised publics will readily recognise and highlight areas of undone science, which can be thought of as knowable known unknowns. In the case of GMOs and nanoparticles for example, environmentalists want more research done on long term health effects whereas industry views such knowledge as undesirable. Although both sides acknowledge the knowable known unknowns, it is then politicised whether research funding in such areas is worthy or not (Hess 2016).

Ignorance About Existing Knowledge and from Suppressing Knowledge

With the aim of completeness, the various other forms of organisational ignorance, not all being directly relevant to this thesis, are summarised by Roberts (2015p364) in his following table and are self-evident. In this thesis only the most relevant forms are covered.

<i>Source of Ignorance</i>	<i>Type of ignorance</i>	<i>Example</i>
Absence of knowledge	Unknown unknowns	Ignorance that is beyond anticipation. Recognized in high risk sectors such as aerospace, where they are known as 'unk unks'.
	Known unknowns	A known incompleteness of knowledge, at various levels of the organization, which can lead to the outsourcing of exposure to such ignorance or can direct R&D efforts.
Ignorance about existing knowledge	Knowable known unknowns	Knowledge that is not central to the organization's core competencies. Access to such knowledge can be outsourced to save on the organization's cognitive resources. For instance, many organizations outsource business services like advertising and legal service.
	Unknown knowns	Unrecognized tacit knowledge, such as that embedded in routines and practices. We often know more than we can articulate – such knowledge may be evident in intuition, instinct, and business hunches.
	Errors	Mistakes caused by human error or systems failures. For instance, one of the most common medical errors is the prescription of the wrong dose of medicine. In certain circumstances, this type of ignorance can be fatal.
Ignorance from suppressing knowledge	Taboos	Organizational cultures can enforce certain behaviours and knowledge to the detriment of others making some behaviours or knowledge taboo.
	Denials	The refusal to recognize major changes in the business context, which require the adoption of new business models. For instance, many retailers that denied the importance of online shopping in the early years of the Internet are now finding that their competitiveness is severely curtailed because of this earlier denial.
	Secrecy	Trade secrets, like the recipes for Coca-Cola or Kentucky Fried Chicken.
	Privacy	Confidentiality agreements with employees, customers and suppliers.

Secrecy

Within an organisation, secrecy, deliberately creating areas of ignorance, affects the distribution of organisational power (Dufresne 2008). Trade secrets on environmental products strategically employ ignorance to suppress knowledge (Proctor 2008). As mentioned elsewhere, the classification of trade secrets on products is frequently upheld by regulatory departments without question and in so doing suppresses public knowledge. This situation ultimately leads to the public being denied knowledge of what they are being exposed to and what safety tests have been done: quite an illogical situation but made supposedly acceptable by the inherent forms of ignorance that the public gains in its place.

A typical example is secrecy in respect to ingredients in a chemical product such as in fragrances or cosmetics, but also applies to processed foods and pesticides, etc. There are many laboratories that have advanced equipment that can fully analyse ingredients in chemical mixtures. To with-hold such information from the public is simply due to the desire to produce ignorance.

Apart from the secrecy surrounding pesticides, designed to kill living organisms, industry's secrecy veil is also applied to harmful effects of medicines. One study (Torka 2019) looked at 'direct health professional communications' (DHPCs) which are letters for advising clinicians of newly revealed evidence of problems and are initiated by regulators and manufacturers. In Australia, the Therapeutic Goods Administration (TGA) does not make these publicly available. The researchers requested all DHPCs from manufacturers issued in Australia from 2007 to 2016 inclusive for 207 drugs which had advisories despatched. They also submitted a freedom-of-information (FOI) request with the TGA. Despite following up non respondents, at the end they had no information for 170 of 207 (82%) of the drugs. The TGA also refused the FOI request. This led to their conclusion:

Our experience highlights unacceptable secrecy concerning safety warnings previously sent to thousands of Australian clinicians. In the absence of explicit regulatory policy supporting disclosure, companies differed in their response. These letters warn of serious and often life-threatening harm and guide safer care; full ongoing public access is needed, ideally in searchable online databases (Torka 2019p551).

It is not difficult to see that a similar statement applicable to many xenochemicals used in the modern environment.

Government organisation secrecy can be seen when considering the emerging 'superbug' issue for micro-organisms developing medication resistance as demonstrated in the following example.

A Fungus Called Candida Auris (C.Auris)

A new 'superbug', a fungus *Candida Auris*, which has been around for hundreds of years, has suddenly started to have devastating effects on people if they have depressed or developing (as for babies) immune systems. It is but one of many that have been emerging (Fisher 2018). Fungi, similar to bacteria, are evolving resistance to

medical treatments of anti-fungal medicines. One of the front-line anti-fungals is itraconazole which is identical to azole pesticides applied to agricultural crops, accounting for about a third of all fungicide sales. It seems that there is a possibility that we have created an environment, through agricultural usage, that has caused the fungi to evolve to survive such toxins (Chowdhary 2013).

In the last few years, a hushed panic has been happening in hospitals around the world, with governments being informed but no public announcements made. Dr S Schelenz, Royal Brompton Hospital's infectious disease specialist saw the lack of interest "very, very frustrating" (Richtel 2019). "They obviously didn't want to lose reputation," Dr. Schelenz said. "It hadn't impacted our surgical outcomes."

The Brompton hospital eventually closed its I.C.U. for 11 days and the story eventually told by *The Daily Telegraph* newspaper. However, internationally it remained quiet, despite appearing in many countries such as at the Hospital Universitari I Politeenie La Fe in Spain where within 30 days, 41% of infected patients died. One hospital spokesperson said that they did not speak to the media since it "is concerned about the public image of the hospital" (Richtel 2019). This however does not help patients to make informed decisions on non-urgent matters, for example, whether to have elective surgery. The same officials would likely not hesitate to tolerate secrecy about a food poisoning outbreak in a particular restaurant.

It has been illustrated in this section on organisational ignorance that it is a form of ignorance that is in our present systems of xenochemical approvals. Decisions by industry and regulatory authorities to research some areas and not others produces unknown unknowns, knowable unknowns, and knowable known unknowns. Furthermore, the automatic adherence by regulatory authorities, to industry secrecy classifications produces considerable ignorance for the public who must use and live with the consequences of such products.

CRIMINAL IGNORANCE

This ignorance classification is very pertinent to the industry and regulatory authority combination which has produced ignorance and undone science in environmental areas contributing to much suffering by the public that has been illustrated in all chapters of this thesis.

The initial subsection of this type of ignorance is focused on delinquents, where there are established psychological studies. Although delinquents are the main subject, the substitution of the offender with industry/medical system/regulatory authority should be evident. The moralities of the employees in organisations involved in the deliberate generation of ignorance have parallels to delinquency.

In the later subsections, the focus on the effects from the presence of power is covered in a subsection titled 'Denial through the presence of power'. This section considers how powerful organisations can evade moral and legal reactions. Such power and influence can shape actions of the supposed perpetrators, influence social reaction, evade or censor, or by manufactured ignorance, reframe the morality of the actions. Powerful organisations can shape, construct and enable collective and individual denial.

A following subsection considers large organisation obedience. Modern states have perfected effective manipulation of military and other personnel (Collins 2008). Bureaucratic organisations by their nature provide the 'will to ignorance' (McGoey 2007) and 'cover up' (Katz 1979), for example, the top members of the hierarchy claim no knowledge of what those at the bottom are doing and those at the bottom were only following orders. Furthermore the division of labour and specialists enable other organisational members to deny injurious activity. The reluctance of the legal system to take action against the managers and directors of companies enable deliberate ignorance strategies to consistently occur. One of the largest pharmaceutical companies in the world, Pfizer, has criminal track record, a pattern of continual fraud and dishonesty so longstanding that it can only be understood as an intentional business model. The many court cases lost in fines by Pfizer has led some observers to conclude "...both criminal and civil penalties appear to be, to Pfizer at least, a business expense worth incurring" (Evans 2010p21).

State denial is subsequently covered where the public faces of states try to modify, reframe and hide knowledge of illegal and detrimental events. This is followed by a subsection titled 'Official acknowledgement' asserting that to awaken people from the suppressive aspects of denial, it is not a case of imparting knowledge to the people themselves, but that the neutralisations and normalisations surrounding potentially detrimental actions need ceremonial and collective renouncement.

It was shown long ago that delinquents commonly expressed guilt and shame about their actions (Sykes 1957). Most of their time was spent not being delinquent, they valorised "certain conforming figures" and recognised the difference "between appropriate and inappropriate targets" (Sykes 1957p666) in respect to their crimes. Sykes and Matza likened youth offenders as being overcome with a broad morality that was being exhibited by others at the time and used "techniques of neutralisation" to disconnect from their own morality. As concluded by Thiel (2015), "These techniques have the effect of neutralizing inter-subjective and external disapproval of the deviation through linguistic strategies that deny, ignore or transform knowledge of the morally transgressive nature of an action or of one's ability to have been able to avoid it."

Sykes and Matza described five types of neutralisation as well as Cohen's (2001) additional "I don't know" type:

- **Denial of responsibility**

This denial involves claims that they were unable to prevent the transgression. Reasons can be that they were drunk or high and not in control but at times be attributed to being led-on by the crowd or they knew no different due to their background. In the case of workers in the industry/medical system/regulatory authority these last two are of course important. Some knowledge is utilised to create a form of ignorance of intention: a denial of their ability to prevent the actions. There are repeated examples through recent history where industry and regulatory authorities have allowed toxic chemical products to enter the living environment due to being perceived essential commercially. But later, due to public casualties and deaths, the products were found not to have been properly studied originally (DDT, BPA, mercury fillings, etc.).

- **Denial of injury**

This denies hurt due to the transgression, for example, the man attacked was a seasoned fighter, or the car wrecked was insured. This is similar to the manufactured ignorance produced by industry on the effects from their chemical products.

- **Denial of the victim**

This denial can range from the victim being thought to be of no moral worth, to actually being a perpetrator. Alternatively, “they started it” so the transgressive act morally allowed certain behaviours normally deemed immoral or illegal. The implication is that there was not really a victim so no injury caused. This is similar to industry finding other causes of the symptoms encountered by people affected likely from other products or actions, for example, the tobacco industry bringing up all other possible causes of lung cancer and highlighting no definitive proof of their product being responsible.

- **“I don’t know”**

This account for their delinquency implies an ignorance of one’s motivations which evades moral precepts linguistically. This is like industry, government and medical associations denying a problem with environmental exposures causing many chronic health problems.

- **condemnation of condemners**

This is where an implication is made that those condemning the delinquent behaviour are transgressive and so do not have any moral right to pass judgement: “who are they to say I’m wrong”. This would correspond to the smear campaigns employed by industry against the scientists, activists and whistle blowers trying to highlight industry’s transgressions.

- **Appeals to higher loyalty**

This is where it is implied that the actions were not from personal desire but were from a loyalty to others such as religion, family or a gang. This can correspond to the government regulators being captured by the industry they are supposed to be regulating.

The last two of the above neutralisation types do not deny moral responsibility but rather are the placement of certain morals above others to minimise the general wrongfulness.

Thiel (2015) points out that the neutralisation theory suggests that mainstream norms do not have to be rejected, or new ones learnt. It is more that the growth of deviation or criminality

results from the flexibility and narrative of the law and custom. Through such flexibility, the lack of morality in the actions can be denied and rationalised through embracing or ignoring forms of non- and novel knowledge for the transgression to be morally justified.

The neutralisation theory has been used in the study of many different forms of criminal behaviour: youth violence, fiddling and pilfering at work, domestic violence, genocide and war crimes, etc. Thiel (2015p258) summarises: “It can be used to reflect on and understand the ‘banality of evil’: that good people can and do engage in horrific acts – not necessarily because they are different from the rest of ‘us’, but, in particular situations and circumstances, people use the inherent flexibility of common cultural guidelines to deny – to themselves and others – the intentions and outcomes of their actions”. On an individual level, this can be achieved through a highly paid career in industry. Those that may have realised morality issues will leave or not be promoted.

We will consider below the various forms of ignorance and motivational actions that can cause in general, criminal ignorance in respect to the thesis subject.

Pluralistic Ignorance

First written about by Schanck (1932) and Matza (1964), pluralistic ignorance in delinquency was introduced. This refers to group individual members believing that the group holds certain norms and values, whereas in reality no individual member holds them. Alternatively, individual members believe they hold beliefs different to everyone else, but in fact others do share them. The resultant anxiety that individuals experience due to this situation is not discussed due to fear of showing a weakness or coming under ridicule. The anxiety may be transformed into a form of mockery of other members: termed ‘sounding’ or even ‘piss taking’ (Willis 1977). The sounding enables anxiety to be temporarily alleviated but reinforces a group normative framework that individuals may privately disbelieve. The group norms may then be misperceived, through neutralisation techniques, individuals may act out such norms. This may be self-reinforcing since by acting out the perceived norms, it is reinforced to other group members.

The above was illustrated in a study of university students (Prentice 1993) where pluralistic drinking norms were found. The students adjusted their behaviour to support the imagined high alcohol consumption that their private beliefs did not support.

This type of ignorance seems to apply to the scientists who perform the various studies in support of the chemical or pharmaceutical companies as has been repeatedly referenced in many parts of this thesis.

Gøtzsche (2013p38) included a quote from Peter Rost, a former global vice president of marketing for Pfizer then whistle-blower, after the company would not listen to his illegal marketing complaints:

It is scary how many similarities there are between this industry and the mob. The mob makes obscene amounts of money, as does this industry. The side effects of organized crime are killings and deaths, and the side effects are the same in this industry. The mob bribes politicians and others, and so does the drug industry ... The difference is, all these people in the drug industry look upon themselves - well, I'd say 99 percent, anyway - look upon themselves as law-abiding citizens, not as citizens who would ever rob a bank ... However, when they get together as a group and manage these corporations, something seems to happen ... to otherwise good citizens when they are part of a corporation. It's almost like when you have war atrocities; people do things they don't think they're capable of. When you're in a group, people can do things they otherwise wouldn't, because the group can validate what you're doing as okay.

Denial Through the Presence of Power

Society, through criminology, sometimes perceives injurious street crimes with more attention than highly injurious and illegal state crimes. Juvenile offenders, coming from relatively powerless positions, have their neutralisation experiences judged negatively as excuses and lies. Sometimes in these cases neutralisation may incur increased penalties (Maruna 2005). These aspects are in contrast to powerful organisations where neutralisation can evade moral and legal reactions. Such power and influence can shape actions of the supposed perpetrators, influence social reaction, evade or censor, or by manufactured ignorance, reframe the morality of the actions. We have seen this through history in many examples, some of which were

discussed in the manufactured ignorance section, such as for the tobacco industry. Cohen (2001) looked at how modern states manage and construct knowledge of their links with illegal wars, genocides, torture, terrorism and lack of human rights. He attributed denial for understanding the normalisation and the use of neutralisation for state-induced suffering. However in this case the denial is not non-knowledge of things wrong or injurious, but is a simultaneous condition of both knowing and not knowing. He termed the 'denial paradox' as the situation where in order to evade knowledge of something, there must be some knowledge of what to evade. So in order to achieve denial, there must be partial knowledge which is not attended to or acknowledged.

Powerful organisations can shape, construct and enable collective and individual denial. This may be by exploiting individual psychological inability to live life with full knowledge. Examples include where family members cannot comprehend intra-familial child abuse (Russell 1986), or where the nature and scale of the holocaust was not accepted by the European Jews in the 1940s and 50s (Cohen 2001).

Large Organisation Obedience

Modern states have perfected effective manipulation of military and other personnel (Collins 2008). Bureaucratic organisations by their nature provide the 'will to ignorance' (McGoey 2007) and 'cover up' (Katz 1979), for example, the top members of the hierarchy claim no knowledge of what those at the bottom are doing and those at the bottom were only following orders. Furthermore the division of labour and specialists enable other organisational members to deny injurious activity.

Conditions of 'crimes of obedience' were identified by Kelman & Hamilton (1989). These were organisational conditions that enabled state crime perpetrators to deny moral aspects of their actions.

- Authorisation: This allows individuals to personally deny responsibility as they acted under orders from the higher authority.
- Routinisation: This acts to normalise morally questionable behaviour. For example, questioning one's previous murderous routine would involve questioning the morality of such actions, so it is simpler to remain in denial and carry on (Bauman 1989).

- Dehumanisation: By regarding certain people as lowly, unwanted, or as animals, denies their moral worth and humanity, and so permits their ill-treatment.
- Pluralistic ignorance (Thiel 2015): In not discussing morality of their actions, the group normalises and routinises such behaviour.

The above approaches enable people with good moral backgrounds to neutralise, deny and ignore moral concerns (Alvarez 1997, Arendt 2006, Browning 2001). For example, many of the soldiers involved in the Holocaust had psychological breakdowns, through to committing suicide afterwards, but they carried out the murderous activity.

State Denial

The public faces of states try to refract, reframe and hide knowledge of illegal and detrimental events. Thiel (2015) highlighted various forms of denial by states:

- Literal denial: it did not happen.
- Interpretative denial: where literal denials have come under scrutiny. Here it may be admitted that it happened but it is not what it seems.
- Implicatory denial: the facts are acknowledged but not the implications.
- Condemnation of the condemners: due to exceptional circumstances the state had to act. This also involves discrediting the moral character of the people affected, denying their humanity and may even be partly applied to the condemners.
- Responsibility denial: they did not intend the injuries that occurred.
- Implicatory and interpretive denial: smokescreen-type responses to reframe public knowledge.

Cohen highlights how the developed world is a passive bystander to the suffering of the less-developed world, war zones, etc. Thiel (2015) attributes this to “state manipulation of knowledge, the human ability to deny, and readily available techniques of neutralization have, at least to some degree, normalized barbarism and suffering in the contemporary world.”

Official Acknowledgement

Cohen (2001) asserts that to awaken people from the suppressive aspects of denial is not a case of imparting knowledge to the people themselves. The neutralisations and normalisations surrounding potentially detrimental actions need ceremonial and collective renouncement. Berger (1991) attributes all knowledge as requiring public agreement and ceremony which is why state denial is so powerful. This makes public knowledge hear-say, rumour, beliefs,

speculation, etc. until officially transformed lowering the acceptance thresholds. An example of this is the denials made about torture in the war on terror. Little ceremonial denouncement of such has occurred enabling the perpetrator's status to remain intact with the denials accepted enabling what Cohen (2006) labels the new paradigm of 21st century: torture.

In the UK, in 2004, after being "questioned repeatedly about the effect of the drug industry on doctors' prescribing, medical education, scientific research, and drug evaluation, government officials told a parliamentary inquiry last week that there was no evidence of unhealthy influence" (Moynihan 2004p641). The British House of Commons Health Committee then reported in 2004-5, (HCHC 2005) on the pharmaceutical industry and found its influence as enormous and out of control (Collier 2006). It was found that it influenced doctors, charities, patient groups, journalists and politicians. It also found that the Department of Health represented the interests of the drug industry. But after having been shown clear evidence of unhealthy industry influence on public health, government officials decided there was no unhealthy influence, and the government did nothing.

State-Corporate Collaboration and Corruption

The manufactured ignorance section used some clear examples of industry manufactured ignorance such as the American Beverage Association purporting to have found that diet soda enables more weight to be lost than not drinking soda (Peters 2014). These findings contradicted a huge body of research showing that artificial sweeteners disrupt body metabolism and are causal for weight gain (Pearlman 2017, Shearer 2016, Swithers 2013, Yang 2010). Another example is the fraudulent beta blocker study which was attributed to leading to the deaths of as many as 800 000 Europeans (Husten 2014). The discredited paper had influenced the formulation of a "standard of care" for noncardiac surgery patients in the use of beta blockers (ESC 2009). The chairman of the committee that formulated the guideline that benefitted the industry was Poldermans who had been fired for scientific misconduct in 2011 (Husten 2011). Yet the government departments that should be taking action on such claims/products have been totally silent.

In another example, a US government agency, the EPA, without making any public announcements, approved the use of glutamic acid (MSG) as a crop pesticide and fertiliser. The product registration for such uses should have, by law, been announced through the Federal Register. However in 1997 only a notice was published that firstly it was going to be applied

without restriction for experimental purposes and a month later that an application for registration as a pesticide was being made. This initially saw thousands of people allergic to MSG, suddenly react after eating lettuce and potatoes which had residues present from spraying applications while growing. An exemption from establishment of an acceptable exposure tolerance limit and allowance to be used on many crop types was granted by the EPA. When the manufacturer wanted to market the product as a fungicide and the application was mentioned in the Federal Register in 2001, the community group, Truth in Labelling Campaign, filed a formal objection to the EPA (TLC 2019). No reply from the EPA was received until 2004 when EPA officials together with lawyers rang the community group in a conference call advising that their objections had been discounted and the product registration as a fungicide would be published in the Federal Register.

In conclusion to this section on criminal ignorance it should be noted from the above and throughout this thesis research on many problems with industrial products, whether they be pesticides, fragrances, food additives, cosmetics, drugs, etc., there has been blatant disregard shown by government departments about the number of people who have been adversely affected. As mentioned before, if this had been by one individual on another then the perpetrator would have been jailed, yet government departments allow industry to continue, mostly without actions taken let alone legal action for such assault on the population. Criminal ignorance is an apt terminology to describe such generated ignorance.

EPISTEMOLOGY, IGNORANCE AND TOXICOLOGY

The standards for toxins required by regulators reflect research norms for toxicologists. Good laboratory practice (GLP) is a standard for how experiments for regulators are to be designed, conducted, and reported (Nature 2010). Factors that directly cause a specific outcome are required to be determined by tightly controlled experimental designs. This means that such experiments must have the precise exposures determined without any confounding elements. In most health effect experiments, this essentially means a sterile environment other than the xenochemical that is to be studied. It also means that the test subjects are all 100% healthy. As

will be pointed out in chapter seven, such laboratory situations are far removed from the real world of thousands of environmental exposures encountered by people who are in a variety of ages and health conditions. There may be factors in the real world that could contribute to a certain outcome, even a synergistic effect with another environmental exposure. This produces a disconnect between a theoretical testing situation and the real-world situation potentially producing different results. One may logically think that real-world experience would therefore add to such studies and be actively pursued. The standardisation of such theoretical approaches without any real-world experience therefore becomes a significant source of ignorance. This can be illustrated through Kleinman and Suryanarayanan's (2015) account of honeybee colony collapse disorder (CCD). This is followed by a section considering how there can ever be absolute certainty in health effect research studies.

Honeybee Colony Collapse Disorder (CCD).

In the winter of 2004-5 professional beekeepers across the US noticed honeybee colonies they maintained were inexplicably collapsing. Bees affected by common problems such as parasites, viruses, and direct pesticide exposure would normally see bee bodies near the hives, yet this was not the case. Colony Collapse Disorder (CCD) was utilised as a name for this problem which saw hives vacated by the bees.

Colony health is important to beekeepers as otherwise their income could be affected if the bees are not in good condition. So over decades a great deal of knowledge on the complex aspects of colony health has been generated by beekeepers. Epistemically the beekeepers are non-reductive and attentive to the complex, dynamic, local and environmental conditions. But as this knowledge is generated far differently from normal laboratory experimental conditions, from the viewpoint of traditional academic entomology, beekeeper knowledge is imprecise and is not recognised for making definitive causal claims.

Many experienced beekeepers came to a hypothesis that the newly introduced systemic insecticides, the neonicotinoids played a major role in CCD. The plant seeds may be soaked in such chemicals with them being absorbed and retained in the whole plant when it grows.

When conventional toxicological experiments were carried out to simulate the exposure levels of the neonicotinoids, little measurable effect was found. However, since the regulators have their standards for testing reflecting epistemic toxicology through an isolated exposure, direct

effects approach, ignorance may be generated about the subtler, long-term and interactive effects. This was evidenced by some independent research (Pettis 2012) where three bee generations in a colony were raised where some hives had a sub-lethal pesticide (the most-common neonicotinoid) environment, then all were exposed to a gut parasite. The bees in the pesticide environment, although having undetectable levels of the pesticide, had significantly higher levels of the pathogen. It was concluded that “interactions between pesticides and pathogens could be a major contributor to increased mortality.”

In response to bee deaths raised by French beekeepers in the mid-90s the pesticide maker, Bayer, performed standard toxicological testing to determine LD50s (the lethal dosage that kills 50% of the test population). But, they did not move to understand the complex causal pathways the beekeepers proposed for their bee deaths.

Later, Bayer set up some field studies where hives were placed next to pesticide treated sunflower crops with enclosed tents for several metres. Bayer found no measurable bee effects and found very low levels of insecticide in the sunflowers: well below the no observable effect levels established in lab trials. Bayer then concluded that “there is no definitive evidence of the company’s systemic insecticides contributing to elevated levels of honey bee deaths” (Kleinman 2015p186). This was contrary to field bee studies by beekeepers and various lab studies by academic scientists, Bayer had taken a false negative approach. The company then argued in the following ways:

- Dismissed the beekeeper and academic research by saying it did not conform to the institutionalised epistemic toxicological research approach.
- Attributed the main causes of CCD to mites, nutritional deficiencies, pathogens and beekeeper use of unapproved miticides (Fischer 2009).
- Highlighted that the pesticides had been used in US agriculture for more than a decade before the first report of CCD
- Claimed that farmers release clouds of pesticide into the air and the paths of the bees.

After Bayer’s patent for its main neonicotinoid product finished a new set of systemic insecticides, the ketoenols was released. The US EPA however approved the release of the main product “Movento” conditional on bee toxicity testing in the field. So, in 2009, Bayer approached a beekeeper organisation to assist them in the field experiment. However, Bayer

controlled the experiment rather than the beekeepers having any significant collaboration in the experiment design or its conduct. The experiment concluded that there was no chronic effects of the insecticide and that it could be applied to blooming crops, the time when bees foraged on the plants. The beekeepers were quite dissatisfied with the experiment and pointed shortcomings as below:

- The citrus was sprayed during bloom, a period of 2 weeks, which is also the period they exposed the hives. This meant that any content in the nectar and pollen was from the immediate spray rather than the long-term systemic effects which is the main pesticide function. So it was no surprise that the sampled nectar, pollen and blossoms had low pesticide levels.
- The beekeepers had asked for a 12-month study, whereas Bayer wanted 2 months. Finally Bayer used 7 months at which point there were no observed differences, yet when the participating beekeepers inspected the hives at 9 and 10 month time points, there were significantly fewer pesticide exposed hives alive compared to controls. These results were dismissed by Bayer attributing the results to different beekeeper managements practices.

The beekeepers had been obliged to assist-only in the experiment due to the EPA and dominant players insisting that the beekeepers' knowledge, their expertise, was not credible. The epistemic form of testing that Bayer relied on ignored other aspects the beekeepers had raised of bio-accumulation and other environmental interactions.

In another later development of studies of CCD, to increase honey yields, high fructose corn syrup is made available for the colonies to feed on through the winter due to all the honey being taken. When the corn seeds were treated with neonicotinoids, CCD was observed up to 23 weeks in the hives supplied corn syrup from such systemically treated corn (Chensheng 2014).

An article later published (Siviter 2021) identified specific pesticide combinations that pose a substantial threat to bees, causing more damage than previously thought concluding that when bees interact with multiple agrochemicals bee mortality can increase significantly.

Situations such as the CCD issue can be highlighted to the authorities to right the wrongs as the beekeepers have been trying to do for the last couple of decades. But given the close

connections between the government and corporations, and the massive resources available to corporations such as Bayer, the regulations need only be enforced. As they are based on an epistemic form of knowledge creation, Bayer needs only assert that these be adhered to. In doing so, they contribute to the production of ignorance by arguing that the knowledge and data produced by beekeepers is not knowledge at all. There is also a “sound science” approach to regulation by the US EPA which prohibits use of observational data in federal rulemaking (Edwards 2008, Kleinman 2015p189). Furthermore, most regulatory policy is based on preference for false-negative over false-positive results: a willingness not to remove a product that may be doing harm, rather than restricting a chemical that could be safe.

In 2018, the EU Commission restricted three neonicotinoid compounds for use only on seeds and plants in greenhouses. The Australian regulators argue that no such restrictions are required as CCD has not been documented in Australia. It is however acknowledged that as with all environmental effects on health, there can be other factors also contributing to the above effects. As with any scientific study it is always good to restrict the number of exposure variables in the study to make such studies more simple and less expensive. When another common chemical in the environment, like glyphosate, is considered, then another contributor to the above CCD can be seen. Glyphosate is not just a herbicide, it was also patented as an antibiotic. This means that bees in contact with plants treated with glyphosate, will act on the important bacteria in the bee’s digestive tract, leaving it more vulnerable to illness due to effects on the immune system (Di Prisco 2013, Brandt 2017). This has been covered in chapter five on its same effects on humans. The microbiome can affect health, mood and even weight gain.

If one takes one step back from the specifics of CCD, let us briefly reflect on the principle of systemic pesticides and some later studies which have come out on the neonicotinoid group of pesticides. The CCD situation can be regarded as another canary in the coal mine situation for environmental effects on human health.

Consider firstly the principle of systemic pesticides: they diffuse throughout the plant to be effective as a pesticide should insects try to eat the plant and clearly they are persistent for the entire plant life cycle from seed to harvest. The fact that they are also in the pollen and nectar is not surprising although being also found in wildflowers surrounding such fields is surprising

(Botías 2015). In light of their persistence, there are now background levels throughout the world (Mineau 2013). They have even been found in US congressional cafeteria food (ABC 2015), demonstrating just how pervasive they have become in our food supply. It seems obvious they also have potential to accumulate in the animals and humans that are continually consuming such plants and that these would likely not be good for us in light of their effect on insects.

It seems that there is almost a monopoly in the seed industry, backed by the insurance companies who see neonicotinoid use as a guarantee for crop success against pests. This leaves farmers with little choice in their use. It also extends to most garden plants (FOTE 2013), further adding to the CCD situation.

Neonicotinoids have also been shown to have toxic effects on invertebrates, birds and other wildlife consuming insects (Eng 2019, Mineau 2013).

It has been estimated that toxicity levels for insects on American agricultural land is about 48 times more toxic than it was two decades ago. Furthermore, the neonicotinoid pesticides accounted for 92% of this toxicity load between 1992 and 2014 (DiBartolomeis 2019). However the situation becomes tragic when there is emerging evidence that neonicotinoid-treated seeds provide insignificant financial or agricultural benefits overall for farmers in soybeans, for example (USEPA 2014, Mourtzinis 2019).

This example has shown that although it is generally accepted that knowledge and power are related, ignorance and power can also be linked. Industry in the example was empowered to produce ignorance by the established norms and practices of institutionalised epistemic toxicology: the social organisation of science. This, plus having resources to impress its position on the stakeholders and the public enables industry interests to prevail.

[Absolute Certainty in Health Research Studies](#)

Although the above CCD situation coverage highlights differences in real life to standardised lab testing, let us put this aside for the time being and simply consider how absolute certainty can be achieved in lab-based health research on xenochemical exposure effects.

The U.S. Academy of Sciences has pointed out

[I]t is neither practical nor desirable to attempt to test every chemical (or mixture) against every end point during a wide range of life stages. The committee recommends

toxicity screening of every agent to which there is a strong potential for human exposure. A well-designed tiered strategy could help to set priorities among environmental agents for screening and could identify end points of mechanisms of action that would trigger more in-depth testing for various end points or in various life stages (NRC 2007).

Such a statement highlights that there will always be ignorance present with health effects from xenochemicals whether individually or as mixtures. The push by industry to get their product approved and sold produces a very tight window for such testing and approval. From the published literature, low dose health effect studies are clearly difficult to replicate. Sometimes epidemiologic studies are performed opportunistically when a chemical spill occurs but is done only on a small percentage of these situations. For example, in the New Orleans contamination issue covered by Frickel (2014a), mentioned previously in this chapter, the regulatory authorities were more consumed in seemingly reassuring the public that the contamination was at safe levels despite the many shortcomings of their assessment, rather than use the opportunity to study any health effects experienced.

Passive forms of ignorance generation have been identified by Kourany and Carrier (2019) in respect to the framework and methodologies used in research studies. Framework influence was illustrated in cancer research where there had been significant anomalies in the somatic mutations approach to cancer research and it was realised that clear correlations of genetic alterations had been overlooked (Bedessem 2019). The physician Emil Lou, who first noticed the alterations said "It's right in front of our face, but if that's not what people are focusing on, they're going to miss it" (Callier 2018). Elliott (2014, 2016) drew highlighted that typical laboratory guidelines provide large scope in study design in respect to choosing test animals, adjusting doses, or selecting statistical procedures. He illustrated this in testing for the risks associated with genetically modified Bt maize, the standard required pure Bt protein rather than the maize itself. This may obscure the possible toxic effects on the maize plant from the genetic modification (Elliott 2016). Methodology-created ignorance is illustrated in considering the two methods of treating patients. The orthodox evidenced-based approach is based on trials on large numbers of animals or people with the conclusions for treatment simply averaged. However treatment, especially by drugs, has various effects in different people. A personalised medicine approach can be more reliable and relevant.

Some studies utilize tens of thousands of animals and are rarely replicated due to the expense. In a study on dibenzopyrene, 42000 trout were fed very low doses, .45ppm, to detect one additional cancer in 1000 trout. This is because studies using a small number of test animals can be confounded due to non-uniform feed, different animal strains, the lab environment varying between the studies, etc. This can lead to problems in replicating such studies.

Industry has used the difficulties in replication to argue against low dose effects of chemicals such as BPA covered in the Manufactured Ignorance section previously. Some further examples are:

- A study by Ryan (2010) where rats were fed BPA during pregnancy and lactation showed no effects on either the male or female offspring, was contrary to other studies that had. This then saw debate about whether the rat strain used had been appropriate.
- A 24-hour human urine and serum profiling of BPA in high dietary exposure (Teeguarden 2011) which was criticised as lacking in a number of areas some of which were (vom Saal 2012):
 - The dosage fed in the meals to the small number of participants was not defined and suspected to be very low, conflicting with “High Dietary Exposure” as used in the title.
 - The urine BPA residues detected were lower than the NHANES US population survey results.
 - The authors withdrew BPA containing food from five families and found that BPA levels dropped to a third of those previous and returned to original levels when no food restrictions were made. This conflicts with the authors’ statement that in the real world, there is little BPA exposure.
- The first study to link BPA to prostate enlargement (vom Saal 1998) was attempted to be replicated by two labs using the same mice strain and same research design. Neither lab found changes in sperm production or prostatic size. The original researcher responded: “A critical issue in experiments concerning effects of low doses of estrogenic chemicals is that a common rodent feed used in toxicological studies has been reported by investigators at the National Institute of Environmental Health Sciences (Thigpen 2003) to be highly variable in estrogenic activity ... raising the possibility that endocrine-

disrupting components in this feed played a role in the failure of these studies to show low-dose effects of BPA” (vom Saal 2005p929).

- Nagel (1997) found mouse prostate effects and when another lab attempted the same study didn't find the same results but found that different effects occur with different mice strains (Ashby 2001).
- Sharpe (1995) conducted a study with phthalates on rats exposed in the womb, finding reduced testicular weights, yet he could not replicate the experiment (Sharpe 1998).

The common trend in most of the studies done on BPA was a biasing effect of industry funded studies: “As of the end of 2004, we are aware of 21 studies that report no harm in response to low doses of BPA. Source of funding is highly correlated with positive or negative findings in published studies, 94 of 104 (90 percent) report significant effects at doses of BPA, 50 mg/kg/day. No industry funded studies (0 of 11, or 0 percent) report significant effects at these same doses” (vom Saal 2005p928).

Following his early BPA prostate studies, vom Saal reported: “Dow Chemical sent a guy down here and he said we can arrive at a mutually beneficial outcome, where you don't publish this work on bisphenol A until the chemical industry has replicated your study, and approval for publication was received by all the plastic manufacturers” (Krimsky 2000).

It has been mentioned numerous times in this thesis that the source of funding affects the outcome of a study. This has been shown in biomedical science (Krimsky 2003), in toxicology (Michaels 2008a,b), public health (McGarity 2008), global warming (Gelbspan 1997), nutrition (Nestle 2001; Levine 2003) and “almost any academic discipline with strong commercial ties” (Krimsky 2014p246). Due to the sensitivity required in low dose studies, it is expected that the ‘funding effect’ will hamper recognition of low dose effects in the science.

There seems to be a growing trend, influenced mainly by the chemical and pharmaceutical industry, to demand proof rather than the use of precaution in approaches to public health. Yet absolute proof in public health and medicine is rarely achieved. Those affected say, that for any public health incident, especially with xenochemical exposures or pollution, the knee-jerk response from health authorities is usually that the situation is safe and there is no clear proof or evidence to warrant concern.

The study of disease in humans is complex. One cannot subject people to suspected toxic chemicals to determine effects or dosages that may cause an effect. As a result, animal testing is employed which may not directly represent equivalent effects on people. This point is argued as positive or negative by industry from time to time as suits their case at hand.

Another way is to study accidental exposures in the human population or even after a chemical has been released for use, although the latter usually only occurs when a problem has started to happen, after people have been detrimentally affected. This, as mentioned at the start of this section for disaster situations, is rarely made use of by government authorities due to their usual rush to downplay the incident. The original epidemiologic and lab studies, with their uncertainties and shortcomings, must be extrapolated by the regulatory authorities without absolute certainty. Since the chemical products have the benefit of being innocent until 'proven' otherwise the regulatory authorities cannot be effective if such 'proof' is required. "The best available evidence must be sufficient" (Michaels 2008ap91).

BIOMEDICAL ADVERSE-EVENT UNDER-REPORTING

Throughout this thesis there are many examples where there is evidence that various forms of ignorance have been deliberately created by combinations of industry and government. If adverse reports of health effects from various products can be suppressed, ultimately resulting in ignorance of any problems, then this can be a preferred method of ignorance production. This section briefly considers this issue.

Kostoff's (2016) look at the under-reporting of adverse events in the biomedical literature virtually established this matter-of-factly (Kostoff 2016p11):

Government, industrial, and foundation sponsors have both missions and agendas. Sometimes, in order to further specific agendas, the integrity of the research product may have to be compromised. For example, critical research may go un-funded (Frickel 2010), research findings may be suppressed (Martin 1999a), and research may be 'manufactured' (Gøtzsche 2013, Kassirer 2005). Some of the incentives for suppressing dissent in research are summarized by Delborne (2016), Martin (1999b), and Schumm (2015), and some specific examples of suppression of dissent in science include the

research of Hess (1999), Kuehn (2004), Martin (2015), and McCulloch and Tweedale (2007).

Some of the reasons that industrial research tends to favour industrial products are shown in Amiri et al. (2014) and Krimsky (2003). Collusion among government, industry, and research performer organizations has been reported extensively, and valuable summaries can be found in Dickson (1984), Lewis (2014), and Primack and von Hippel (1974).

Kostoff (2015) lists literature claiming under-reporting of adverse events. One of the most concerning ways to under-report is by deliberate suppression of research findings due to:

- Negative results making the sponsoring organisations, journals, or the authors themselves reluctant to publish
- Adverse events that make industry or the government reluctant to publish
- Possible commercial effects to industry
- The lack of ethics in the research

Some of the incentives to produce inadequate literature or undone science (Martin 1999a) are:

- The benefits to industry in concealing adverse effects
- Donor pressures on governments
- Industry growth advantages in respect to employment improvements for governments
- Professional journals/societies industry-sponsored have journalist editors who must maintain industry positive literature
- Journal editors who are pressured to produce industry positive (selective) literature due to advertising revenue
- Researchers depending on grants/sponsorships from industry are influenced to research only topics of interest and results attractive to industry. This may also be related to future employment by industry.

As mentioned above, while there are many studies showing how the science has been distorted, and elsewhere in this thesis by biased review committees and media, these are only the ones that are fortunately found out. The industry and government go to great lengths to hide such activities and there would likely be the majority that are never realised. Most of the

exposures result from court cases and whistle-blowers. Few people are prepared to become whistle-blowers due to the resulting professional and financial suicide (Lewis 2014, Martin 2008) and so the majority of factual distortions remain hidden.

The above considerations are further developed in the next chapter which will take the above situation into account and will critique standard knowledge-producing processes, consider vested interests (funding effect), bias, undone science, lack of public reporting, lobbying and related regulation failures.

THE NEED FOR EVERGREEN SCIENCE POLICY ILLUSTRATED BY CALCITONIN HORMONE

The length of time on the market is frequently used by health and regulatory authorities in assurances of safety of various synthetic products. However “a prolonged period on the market is no guarantee of safety” (Lexchin, 2014p18). Such assurances of safety then create ignorance of possible long-term effects. Consider for example, a drug registered decades ago for increasing calcium levels in the bones and decreasing the levels in the blood. Synthetic “calcitonin is used as a nasal spray to treat osteoporosis (loss of calcium in bones), an injection to treat Paget’s disease (a chronic bone disorder) and hypercalcemia (high blood calcium)” (Health Canada 2013). However long-term use of the synthetic hormone leads to increased risks of various types of cancer: long term use was not studied in respect to cancer (EMA 2020).

The need for what Reyner (2015) termed “evergreen science policy” where non-knowledge and knowledge are “placed on probation with respect to potential new knowledge or ignorance” is needed in respect to post-market surveillance of chemical products. The validity of the product’s approval would therefore not rest in knowledge stability but would remain in a probationary state for anticipated changes. However, in practice this is not a simple task to pursue in post-market surveillance due to the conflicting aspects of power in decision making and its implementation (Carpenter 2006, Light 2010) as will be seen in a previous section.

CONCLUSION

This chapter has illustrated that most of the problems in recognising that environmental exposures cause health effects can be explained by undone science and the generation of ignorance by industry and the government. Ignorance, sometimes aided by undone science, has been used to hide or mislead the public on health effects resulting from the use of many industry products.

It has been said that “[i]gnorance can be made or unmade and science can be complicit in either process” (Proctor 2008p3). Experience by authors such as Kleinman and Suryanarayanan (2015p183) shows that manufacturing uncertainty is a strategy used by corporations to advance their interests: “The stories of companies hiding data, falsifying evidence, or selectively using findings to advance their interests are legend.”

Research into the health effects on humans from the modern environment they live in does not interest industry in general. In today’s technology-based society, it is an advantage for industry to invest in researching innovative technologies, such as biotechnology, communication technology and nanotechnology, to ensure its financial success. It has little desire to know about the environmental, health, safety and societal implications of those technologies. Since there is no drive to research such aspects, ignorance in these areas is socially produced.

Within agnotology, undone science is commonly classified as “known unknowns”, although in researching areas of undone science, nescience (or knowledge we discover as a surprise) can result (Gross 2010). Social movements can view future research areas as positive non-knowledge whereas industry may view them as negative non-knowledge. Undone science typically involves a systematic underfunding of certain research areas. Those few scientists who do research in those topics can experience various industry-backed reactions to their work. If there are social benefits in not pursuing a line of research, social movements can also create undone science (Frickel 2010, Kempner 2005), e.g., animal testing.

Conflicts have occurred over decades between social movements and government and/or industry on the credibility of information which fed into a risk the assessment of existing or emerging technologies or products. Broader epistemic conflict also results from how knowledge, or the lack of it, is translated into policy.

In issues related to environmental effects on health, industry and government departments frequently cite studies that illustrate their claims of little or no risk. At the same time, they may even claim there is not enough research to substantiate any safety-assessment modification or regulatory action. So apart from simply representing an absence of knowledge, undone science can represent a structured absence (Hess 2016) created by social inequality which determines which areas of research are to be funded. Any analysis of undone science is perspectival due to the inherent differences over the importance of getting such research done.

Undone science does draw attention to an epistemic dimension of all social movements. After people experienced health effects apparently associated with environmental exposures, they may look for research into the issues. Because little or no research has been carried out, those affected may be dismissed by the orthodox system. When social movements then call for research, and it isn't funded or carried out, this leads to undone science.

This chapter has shown that industry interests frequently use undone science to focus on the desired areas of knowledge that are positive for their products. Industry's priority is the sale of its products rather than investigating whether such products are detrimental in any way, especially on public health. This sees the active generation of undone science which can also be used to industry's advantage as a delaying tactic. When undesirable aspects of a product may be highlighted in the future, industry can argue that insufficient studies done to prove any detrimental effects. Two examples were covered to demonstrate this, ionising radiation and sunscreens, to highlight the amount of undone science about technologies that are commonly regarded as proven and helpful in achieving a high standard of living. These examples illustrated the lack of studies done in respect to their effects on human health: the undone science which the public is generally ignorant of.

To illustrate aspects of recognising ignorance, Roy and Zeckhauser (2015) used the concept of a consequential amazing development (CAD), what Gross (2010) had termed "nescience". Cognitive anchoring to an initial hypothesis severely limits the contemplation of consequences of any CADs or uncertainty, and results in a complementary failing due to such bias or beliefs (Michotte 1963, Rips 2011). This may also lead to failure to learn, the possibility of overlooking evidence of causality, or on the other hand concluding more than the evidence supports. To avoid a state of consequential ignorance, self-awareness and acknowledgement of CADs is

required. However this is hindered by overconfidence and salience (Roy 2015). A CAD typically shows vulnerability and is influenced by information processing and storage psychology.

A common industry response to any detrimental studies of its products is to manufacture uncertainty or doubt around those studies, to enable continued product sales and to protect itself from potential liabilities if the product is later deemed a dangerous good. When a company realises from its own research or evaluations that there will be health consequences, this usually creates an incentive to manufacture ignorance: there are numerous stories of companies hiding data, falsifying evidence and selectively using findings for their advancement. The tobacco industry is the classic case, which saw actions to generate uncertainty, doubt, and ignorance through advertising, misleading press releases, support for alternative research, establishment of research institutes and funding of supportive research (Proctor 2008). The net result of such actions is the creation of ignorance in the general public and regulatory authorities, to effectively maintain the company's bottom line by keeping such products on the market and in use.

One of the common measures used to downplay published studies that are counter to industry interests is to employ doubt alongside the manufacture of ignorance. Detrimental product study results are put into doubt by showing contrary results through government committees, think tanks and all types of media (including scientific journals). The many ways of manufacturing ignorance were covered by considering examples from pharmaceutical companies, the tobacco industry, Roundup herbicide, aspirin and Reye's syndrome, berylliosis, phenylpropanolamine, breast-cancer, red tides, vaccines, aspartame, Splenda, baby powder, biosludge, fluoridation of drinking water, and bisphenol A (BPA). Although manufactured ignorance can take many forms, as illustrated in these examples, it has common results for most environmental-exposure crises. A frequent approach is to blame the victims. Crises can last for decades before any action is taken. With lead issues, blame ranged from the children's bad behaviour through to the parents' lack of discipline, lack of moral values and even being single parents (Warren 2000). Asbestos mine workers with lung problems were blamed for smoking or having an unhealthy lifestyle (Schneider 2004). After health-warning labels were put on cigarette packets, blame was put on smokers for ignoring the warning (Michaels 2008a).

After legal discovery in legal cases, as with asbestos, lead, tobacco, glyphosate, etc., industry documents clearly show that the companies involved knew the extent of the problems, deliberately covered that knowledge up and actively criticised any similar knowledge in the public domain.

Selective ignorance occurs when specific information on a topic is produced but there is a failure to produce any other information. This is unavoidable in practice since not every aspect of a topic can be studied in detail due to time and funding limitations. Full information on a topic can influence major public policy or cultural forces. It therefore becomes attractive for powerful interest groups to use selective ignorance on socially relevant topics and it is important for society to recognise this. It is produced most often through (Frickel 2014a): secrecy (Rappert 2014), censorship (Galison 2008), deceit and suppression (Markowitz 2002, Proctor 1995), denial (McGoey 2012a), and doubt (Michaels 2005ab, Oreskes 2011). In its simplest form, selective ignorance could arise from clear social decisions to fund one area of research and not another, e.g., cancer cures rather than cancer prevention. The complexities of this type of ignorance were covered in this chapter.

It also covered knowledge sequestration (Frickel 2014a), where some knowledge is prevented from being circulated. There is a significant body of research which highlights how industry and government have endeavoured to stop the flow of information in research and media (Delborne 2008, Martin 1986,1996,2007, Stocking 2015).

Ignorance can also stem from the “unintended consequences of social action” (Frickel 2014ap270). Ignorance can be produced by politically or self-serving deviant science, perhaps leading to a conclusion that greater public transparency or scientific autonomy would reduce ignorance. But it must also be recognised that ignorance may be produced by “structural pressures, institutional arrangements and the normative cultures that order everyday scientific practice and decision-making” (Frickel 2014ap270), e.g., research funding and academic tenure. The cases of honeybee colony collapse disorder (CCD) and Frickel’s (2014b) Hurricane Katrina contaminated-soil-situation were discussed.

Frickel (2014a) cites examples of the institutional production of ignorance like the barriers encountered in dealing with the “sniff test” done by the public for single-point air samples (Mayer 2014), the seafood safety controversy following the BP Deepwater Horizon oil spill in

2010, and various other writings on disaster situations. The approach of government authorities in addressing such situations is not to use the latest and most-appropriate scientific testing and specific evaluations, but rather to use off-the-shelf, traditional methods, some of which are many decades old. The shortcomings of such approaches generally leave the public in a “knowledge vacuum” as seen in the examples. Other examples of selective ignorance include agricultural research, GMOs, titanium dioxide, vaccine safety, air pollution, synthetic vitamin supplements, and salt.

Rational selective ignorance is commonly employed so that one’s limited energy, time and cognitive capacity can be applied to activities deemed the most important. In employing rational ignorance in this way it becomes beneficial, a “knowable known unknown” (Roberts 2015p362). However there are times when the decision to forego knowledge acquisition can be harmful. Rational ignorance has no tie with moral principles. Its rationality is only relative to the decision-maker maximising their objectives (Becker 1976). Somin (2015) gives the example of a mafia boss devoting his time and energy to aspects that will enable his protection racket to operate efficiently rather than studying ethical philosophies that could make him a better person.

Rational selective ignorance was illustrated in the regulatory practice of extrapolating data, usually attributed to governmental underfunding but commonly favourable to industry. Fricke (2014b) highlighted method in this in the US EPA’s analyses of soil contamination following Hurricane Katrina: the risk assessments of 70% of the 141 contaminants were extrapolated from a relatively small number of tests (most of the chemicals had never been tested for cancer risk). Another example is in nanotechnology: nanoscale chemicals are presumed safe by regulatory authorities due to their “similar” structure to existing chemicals (Hess 2010, Lamprou 2011).

Two types of ignorance were distinguished as similar to but different from rational ignorance: inadvertent ignorance, where a body of knowledge is overlooked by a person who has no idea that it exists (Friedman 2005), and irrational ignorance, when an individual avoids information that may be counter to their beliefs or goals. “Motivated scepticism” may lead an individual to avoid information that first appears to be counter to their belief yet may have been useful to achieve their goals (Taber 2006). Various examples of rational ignorance in respect to health effects were given related to various food preservatives.

Aggregated ignorance (introduced in this thesis) is generated by the very authorities charged with protecting the public, as seen repeatedly in this thesis research. The most common theme encountered is examining the effect of thousands of xenochemical exposures on human health by considering each on an individual basis ignoring combined effects, potentiation and synergism in the real world. Studying aggregation of all exposures is avoided, allowing only the comparison of test results (conducted in ideal lab environments) of each chemical on its own on selectively chosen health effects. This ignores hundreds of other effects like endocrine system disruption. Usually each xenochemical is progressively dismissed as causing any effects in low concentrations with the huge amount of undone science remaining. This approach also carries through to the legal system.

The aggregation issue can also be used in an opposite way that proves convenient for health authorities to water down any hot-spots. A top-down system used by health authorities can erase vulnerable communities if defined by geography, occupation or personal biological data (Roberts 2015). This avoids exposure effects for chemicals, individuals or groups that may be present in certain geographical locations. The example in this chapter illustrated how communities next to petrochemical plants along the Mississippi River have their health data combined with that from communities living in areas well away from them, so the localised health problems are not highlighted.

The same effect is seen through the human biomonitoring results of the CDC, sourced through the National Health and Nutrition Examination Survey (NHANES). While acknowledging the quantity of chemicals the population of today is exposed to and retains, its lack of geographic or worker-specific break-up etc. implies we are all exposed fairly equally. However, this may be far from the actual case. Individual examples of chemical products were given to demonstrate this, including localised environmental disasters.

As has been shown, there is significant overlap between the various forms of ignorance. This was also seen in organisational ignorance, but it was still pertinent to include as both industry and governmental organisations are central to this thesis. If an organisation's management has no awareness of the various forms of ignorance, they will lack the foundational first step needed to manage unknowns.

In the business world many believe that the global financial crisis and Enron's collapse were due to intelligent people choosing to ignore the consequences of their collective behaviour (Elkind 2005). Individual and societal avoidance of problematic information was highlighted by Heffernan (2012) through her work on wilful blindness. The types of organisational ignorance that can arise were discussed in this thesis: unknown unknowns, known unknowns, knowable known unknowns and secrecy.

This thesis considers the category of criminal ignorance as pertinent to the industry and regulatory authority close links that have produced ignorance and undone science on environmental effects, and contributed to much public suffering. This type of ignorance was introduced by focusing on established psychological studies of delinquents. Although juvenile delinquents were the main subject, the same analysis can be applied to authorities in industry, the medical system and regulatory bodies, which might be called institutional delinquents. The morality of employees in organisations involved in the deliberate generation of ignorance has parallels to delinquency. Various forms of ignorance and motivational actions that can cause criminal ignorance were considered: pluralistic ignorance, denial through the presence of power, large organisation obedience, state denial, official acknowledgement and state-corporate collaboration and corruption.

The section Denial Through the Presence of Power considered how powerful organisations can evade moral and legal reactions. Such power and influence can shape the actions of the supposed perpetrators, influence social reaction, evade or censor, or (by manufactured ignorance) reframe the morality of the actions in question. Powerful organisations can shape, construct and enable collective and individual denial.

The following subsection considered large organisation obedience. Modern states have perfected the effective manipulation of military and other state-employed personnel (Collins 2008). Bureaucratic organisations, by their nature, provide the "will to ignorance" (McGoey 2007) and "cover up" (Katz 1979). For example, the top members in the hierarchy can claim no knowledge of what those at the bottom are doing, and those at the bottom were only following orders. Furthermore, the division of labour and specialists enable other organisational members to deny injurious activity.

State denial was also covered under criminal ignorance, where the public faces of states try to refract, reframe and hide knowledge of illegal and detrimental actions. This was followed by a subsection titled Official Acknowledgement, which asserted that to awaken people from the suppressive aspects of denial, it is not a case of imparting knowledge to the people themselves. Instead, the neutralisations and normalisations surrounding potentially detrimental actions need ceremonial and collective renouncement.

The common types of neutralisation methods were discussed: denial of responsibility, denial of injury, denial of victim, the “I don’t know” approach, condemnation of condemners, and appeal to higher loyalties. The last two types do not deny moral responsibility but rather show the placement of certain morals above others to minimise the general wrongfulness. Thiel (2015) pointed out the neutralisation theory suggests that mainstream norms do not have to be rejected, or new ones learnt. It is more that the growth of deviation or criminality results from the flexibility and narrative of law and custom. Through such flexibility, the lack of morality in the actions can be denied and rationalised by embracing or ignoring forms of non- and novel knowledge for the transgression to be morally justified.

The neutralisation theory has been used to study many different forms of criminal behaviour: youth violence, fiddling and pilfering at work, domestic violence, genocide and war crimes, etc. Thiel (2015p258) mentioned that “good people can and do engage in horrific acts – not necessarily because they are different from the rest of ‘us’, but, in particular situations and circumstances, people use the inherent flexibility of common cultural guidelines to deny – to themselves and others – the intentions and outcomes of their actions”. On an individual level, this can be achieved through a highly paid career in industry. Those who may have realised moral issues will leave or not have been promoted.

The chapter also considered toxicology standards required by regulators that reflect the research norms for toxicologists. Good laboratory practice (GLP) is a standard for how experiments for regulators are to be designed, conducted and reported (Nature 2010). Factors that directly cause a specific outcome must be determined by tightly controlled experimental designs, so such experiments must have the precise chemical exposures determined without any confounding elements. In most health-effect experiments, this essentially means a sterile

environment other than the xenochemical being studied. It also means that the test subjects are all 100% healthy.

Such laboratory situations are far removed from the real world of thousands of environmental exposures encountered by people of a variety of ages and health conditions. There may be real-world factors that could contribute to a certain outcome, even a synergistic effect with another environmental exposure, which produces a disconnect between the results in a theoretical testing situation and the real-world situation. One may logically think that real-world experience would add to such studies and be actively pursued. The standardisation of such theoretical approaches without any real-world experience therefore becomes a significant source of ignorance through related undone science. This was illustrated through Kleinman and Suryanarayanans' (2015) account of CCD.

This thesis has provided many examples where various forms of ignorance have been deliberately created by combinations of industry and government. If adverse reports of health effects from various products can be suppressed, ultimately resulting in ignorance of any problems, then this can become a preferred method of ignorance production.

Length of time on the market is frequently used by health and regulatory authorities when they assure the safety of various synthetic products. However "a prolonged period on the market is no guarantee of safety" (Lexchin, 2014p18). Such assurances of safety then create ignorance of possible long-term effects as illustrated by the case with the hormone calcitonin. This drug has been used for decades for osteoporosis (loss of calcium in bones), Paget's disease (a chronic bone disorder) and hypercalcemia (high blood calcium) (Health Canada 2013). However, long-term use of the synthetic hormone leads to increased risks of various types of cancer; long-term use has not been studied in respect to cancer (EMA 2020).

What Reyner (2015) termed "evergreen science policy" where non-knowledge and knowledge are "placed on probation with respect to potential new knowledge or ignorance" is needed in respect to post-market surveillance of chemical products. The validity of the product's approval would therefore not rest in knowledge stability but would remain in a probationary state for anticipated changes. However, in practice this is not a simple task to pursue in post-market surveillance due to the conflicting aspects of power in decision-making and its implementation (Carpenter 2006, Light 2010).

The evidence presented of environmental effects on health in chapters 2 and 3 shows that a significant amount of such evidence exists. But why, then, does nothing appear to be done about the situation? Is there really a problem if no explanation is put forward at such an impasse: why is there no concern for or action on the situation by medical and health authorities? This chapter's consideration of ignorance in its many forms has helped explain the many facets of this complex issue. In so doing, the generation of ignorance in our society becomes an important consideration in respect to industry and government actions in general. It becomes a tool to be applied in other social issues such as climate change and farming sustainability. If the public were more aware of the concept of intentionally generated ignorance, broader attitudes could change on many issues. However, due to the ignorance generated, the public remains insulated from even questioning many situations as the lack of information deceives them in the first instance. Many people, for example, have the utmost respect for a doctor's individual opinion and will not even question it.

The dominant form of ignorance that ultimately prevails against change is the criminal ignorance displayed by those in power. As mentioned, it is not only a case of imparting knowledge to the people themselves to awaken them, but that the neutralisations and normalisations surrounding the potentially unethical actions of those in power need ceremonial and collective renouncement. This can be seen in the issue of climate change. There is great public awareness yet relatively insignificant actions are taken, not through general apathy, but from the seeming deliberate lack of acknowledgement and inaction by those in power.

The production of ignorance while being the main tool to hide environmental health effects, may only be one of many tactics applied by those in power over time. Once the extent of environmental contamination and background levels and their health effects are realised and finally acknowledged, will we be told (as seems to be the case with climate change) that we simply must "live with it"? Increased public awareness could, however, potentially achieve better long-term health outcomes by inducing change gradually, as people may search for practitioners who are also knowledgeable in such areas.

This chapter's classification of different forms of ignorance has enabled better understanding of the various approaches of industry and government on this issue. The many ways that ignorance is created and used have been demonstrated. In so doing, environmental effects on health are shown to be an issue that is determined and influenced by the social construction of ignorance, not just an application of science.

The next chapter, chapter 5, will take the effects of generating ignorance further and consider aspects of knowledge manipulation in environmental effects on health.

REFERENCES

ABC, Food addiction and the brain, Health Report, Radio National, Monday 5 August 2013 5.40pm
<https://www.abc.net.au/radionational/programs/healthreport/food-addiction/4865260> accessed 17 Mar 2019

AG: Australian Government: Department of Infrastructure, Transport, Cities, and Regional Development: Road Trauma Australia—Annual Summaries
https://www.bitre.gov.au/publications/ongoing/road_deaths_australia_annual_summaries accessed 21 Dec 2019. (1100 deaths/yr, 25 million population = .37% over 85 years).

Agar N, Halliday G, Barnetson R, Ananthaswamy H, Wheeler M, Jones A, The basal layer in human squamous tumors harbors more UVA than UVB fingerprint mutations: A role for UVA in human skin carcinogenesis, *Proc of the Nat Acad Sc*, Apr 2004, 101, 14, 4954-59..

Ahmad M, Khan A, Ali S, Mahmood R, Chemoprotective effect of taurine on potassium bromate-induced DNA damage, DNA-protein cross-linking and oxidative stress in rat intestine, *PLoS One*, 2015 Mar 6;10(3):e0119137.

AHRP: Alliance for Human Research Protection, Website page for Dr Jacob Puliyeel
<http://ahrp.org/jacob-puliyel-md/> accessed 21 Sep 2018.

Allen B, *Uneasy Alchemy: Citizens and experts in louisiana's chemical corridor dispute*. Cambridge: MIT Press, 2003.

Allen B, The problem with epidemiology data in assessing environmental health impacts of toxic sites, in M. M. Aral, C. Brebia, M. L. Maslia, and T. Sinks (eds.), *Environmental Exposure and Health*, Billerica, MA: Computational Mechanics Inc., 2005, 467–75.

Alvarez A, Adjusting to genocide: The techniques of neutralization and the holocaust, *Social Science History*, 1997, 21, 2, 139–78.

Amiri A, Kanesalingam K, Cro S, Casey A, Does source of funding and conflict of interest influence the outcome and quality of spinal research? *Spine Journal*, 2014, 14, 2, 308–14.

Anderson B, Preemption, Precaution, Preparedness: Anticipatory Action and Future Geographies. *Progress in Human Geography*, 2010, 34, 777–98.

Arendt H, Eichmann in Jerusalem, New York, Penguin, 2006 [1963].

Ashby J, Testing for endocrine disruption post-EDSTAC: extrapolation of low-dose rodent effects to humans, *Toxicology Letters*, 2001, 120, 233–43.

Bashash M, Thomas D, Hu H, Angeles Martinez-Mier E, Sanchez B, Basu N, Peterson K, Ettinger A, Wright R, Zhang Z, Liu Y, Prenatal fluoride exposure and cognitive outcomes in children at 4 and 6–12 years of age in Mexico. *Environmental health perspectives*, 2017 Sep 19,125, 9, 097017.

Bassetti M, Merelli M, Ansaldi F, de Florentiis D, Sartor A, Scarparo C, et al. Clinical and therapeutic aspects of candidemia: a five year single centre study. *PLoS One*. 2015, 10, e0127534.

[Bassin E](#), [Wypij D](#), [Davis R](#), [Mittleman M](#), Age-specific fluoride exposure in drinking water and osteosarcoma (United States), *Cancer Causes Control*, 2006 May, 17, 4, 421-8.

Baylis S, Shah N, Jenkins A, Berry N, Minor P, Simian cytomegalovirus and contamination of oral poliovirus vaccines, *Biologicals*, 2003 Mar 1, 31, 1, 63-73.

Baylis S, Finsterbusch T, Bannert N, Blümel J, Mankertz A, Analysis of porcine circovirus type 1 detected in Rotarix vaccine, *Vaccine*, 2011 Jan 17;29, 4, 690-7.

Bauman Z, *Modernity and the Holocaust*, Cambridge, Polity, 1989.

Beani J, Ultraviolet A-induced DNA damage: role in skin cancer, *Bulletin de L'academie Nationale de Medecine*, 2014 Feb, 198, 2, 273-95.

Becker G, *The economic approach to human behavior*, Chicago, IL, University of Chicago Press, 1976.

Becker H, *Outsiders*, Glencoe, Free Press, 1963.

Berezow A, Glyphosate-Gate: IARC's scientific fraud, American Council on Science and Health, 24 Oct 2017 <https://www.acsh.org/news/2017/10/24/glyphosate-gate-iarcs-scientific-fraud-12014> accessed 3 May 2021.

Berger P, Luckman T, *The social construction of reality*, London, Penguin, 1991 [1966].

Bigelow Laboratory for Ocean Sciences, Nutrients that feed red tide 'under the microscope' in major study. *ScienceDaily*, 2014, Nov 6, Retrieved August 8, 2018 from www.sciencedaily.com/releases/2014/11/141106132319.htm

Blomberg B, Thomassen A, de Jong P, Lam M, Diederichsen A, Olsen M, Mickley H, Mali W, Alavi A, Høilund-Carlsen P, Coronary fluorine-18-sodium fluoride uptake is increased in healthy adults with an unfavorable cardiovascular risk profile: results from the CAMONA study, *Nucl Med Commun*, 2017 Nov, 38, 11, 1007-14.

Botías C, David A, Horwood J, Abdul-Sada A, Nicholls E, Hill E, Goulson D, Neonicotinoid residues in wildflowers, a potential route of chronic exposure for bees, *Environmental science & technology*, 2015 Nov 3, 49, 21, 12731-40.

Bleyer A, Welch H, Effect of three decades of screening mammography on breast-cancer incidence, *New England Journal of Medicine*, 2012, 367, 21, 1998-05.

Blum D, *The poison squad: one chemist's single-minded crusade for food safety at the turn of the twentieth century*, Penguin Press, 2018.

Bouvard V, Loomis D, Guyton K, et al, International Agency for Research on Cancer Monograph Working Group, Carcinogenicity of consumption of red and processed meat. *Lancet Oncol*, 2015,16, 16, 1599–00.

Braithwaite J, *Corporate Crime in the Pharmaceutical Industry*, London: Routledge & Kegan Paul Books, 1984.

Brand L, Compton A, Long-term increase in *karenia brevis* abundance along the Southwest Florida Coast, *Harmful Algae*, 2007, 6, 2, 232–52.

Brandt A, Grikscheit K, Siede R, Grosse R, Meixner M, Büchler R, Immunosuppression in honeybee queens by the neonicotinoids thiacloprid and clothianidin, *Scientific reports*, 2017 Jul 5, 7, 1, 1-2.

Brausch J, Smith P, Toxicity of three polyethoxylated tallowamine surfactant formulations to laboratory and field collected fairy shrimp, *Thamnocephalus platyurus*, *Arch Environ Contam Toxicol*. 2007 Feb, 52, 2, 217-21.

Brenner D, Elliston C, Estimated radiation risks potentially associated with full-body CT screening, *Radiology*, 2004 Sep, 232, 3, 735-8.

Brown G, Denning D, Gow N, Levitz S, Netea M, White T, Hidden killers: human fungal infections. *Sci Transl Med*, 2012, 4,165rv13.

Brown P, *No safe place: Toxic waste, leukemia, and community action*, Berkeley, CA, University of California Press, 1990.

Browning C, *Ordinary Men*, London, Penguin, 2001.

Burke K, Wei H, Synergistic damage by UVA radiation and pollutants. *Toxicology and Industrial Health*, 2009, 25, 4-5, 219-24.

Busby C, Schnug E, Advanced biochemical and biophysical aspects of uranium contamination, In: (Eds) De Kok, L, Schnug E, *Loads and fate of fertilizer derived uranium*, Backhuys Publishers, Leiden, The Netherlands, 2008, ISBN/EAN 978-90-5782-193-6.

Busby C, Very Low dose fetal exposure to chernobyl contamination resulted in increases in infant leukemia in europe and raises questions about current radiation risk models, *International Journal of Environmental Research and Public Health*, 2009, 6, 12, 3105-14 <http://www.mdpi.com/1660-4601/6/12/3105>.

Calaf G, Ponce-Cusi R, Aguayo F, Muñoz J, Bleak T, Endocrine disruptors from the environment affecting breast cancer (Review), *Oncology Letters*, 2020, 20, 19-32.

Callahan P, Roe S, Big Tobacco wins fire marshals as allies in flame retardant push, *Chicago Tribune*, 8 May 2012 <http://www.chicagotribune.com/ct-met-flames-tobacco-20120508-story.html>

Callier V, Cells talk and help one another via tiny tube networks, *Quanta Magazine*, 23 April 2018. <https://www.quantamagazine.org/cells-talk-and-help-one-another-via-tiny-tube-networks-20180423/> Accessed 6 Jan 2022

Carpenter D, Reputation, gatekeeping and the politics of post-marketing drug regulation, *Virtual Monitor*, *Ethics Journal of the American Medical Association*, 2006, 8, 403–03.

Chaiken S, Trope Y, (Eds.), *Dual-process theories in social psychology*, New York, Guilford Press, 1999.

Chensheng L, Warchol K, Callahan R, Sub-lethal exposure to neonicotinoids impaired honey bees winterization before proceeding to colony collapse disorder, *Bulletin of Insectology*, 2014, 67, 1, 125-30.

- Choi A, Sun G, Zhang Y, Grandjean P, Developmental fluoride neurotoxicity: a systematic review and meta-analysis, *Environmental health perspectives*, 2012, 0, 10, 1362-8.
- Chowdhary A, Kathuria S, Xu J, Meis J, Emergence of azole-resistant *aspergillus fumigatus* strains due to agricultural azole use creates an increasing threat to human health, *PLoS Pathog* 2013, 9, 10, e1003633.
- Christensen J, Smoking out objectivity: Journalistic gears in the agnogenesis machine, In: *Agnotology, The making and unmaking of ignorance*, Stanford University Press, 2008, Chap 12, 266-82.
- Christensen P, Tanning beds: What do the numbers really mean? *Ass Health Care Journalists*, May 7, 2010 <https://healthjournalism.org/blog/2010/05/tanning-beds-what-do-the-numbers-really-mean/> accessed 11 Nov 2020.
- Clark L, *Acceptable risk: Making decisions in a toxic environment*. Berkeley, CA, University of California Press, 1989.
- Clinard M, Yeager P, *Corporate Crime*, New York, The Free Press, 1980.
- Code L, Thinking Ecologically: The Legacy of Rachel Carson, In: Kabesenche B, O'Rourke M, Slater M (eds.), *Environment: Philosophy, Science, and Ethics*. Cambridge, MA, MIT Press, 2012.
- Cohen S, *States of Denial*. Cambridge, Polity, 2001.
- Cohen S, Neither honesty nor hypocrisy: The legal reconstruction of torture, In: Newburn T, Rock P, (eds), *The politics of crime control*, Oxford, Oxford University Press, 2006.
- Collier J, Big Pharma and the UK Government, *Lancet*, 2006, 367, 96-8.
- Collins R, *Violence: A Micro-sociological Theory*, Princeton, NJ, Princeton University Press, 2008
- Colquhoun J, Why I changed my mind about water fluoridation, *Perspectives in Biology and Medicine*, 1997, 41, 1, 29-44.
- Colquhoun J, Influence of social class and fluoridation on child dental health, *Community Dent Oral Epidemiol*, 1985, 13, 1, 37-41.
- Costa L, Giordano G, Developmental neurotoxicity of polybrominated diphenyl ether (PBDE) flame retardants, *Neurotoxicology*, 2007, 28, 6, 1047-67.
- Cowling B, Fang V, Nishiura H, Chan K, Ng S, Ip D, Chiu S, Leung G, Peiris J, Increased risk of non-influenza respiratory virus infections associated with receipt of inactivated influenza vaccine, *Clinical Infectious Diseases*, 2012, 54, 12, 15, 1778-83.
- Cranor C, Reckless Laws, Contaminated People Science Reveals Legal Shortcomings in Public Health Protections. In: *Powerless Science? Science and Politics in a Toxic World*, Eds Boudia S, Jas N, Berghahn, New York, 2014, Chap 9, 195-214.
- Cranor C, How the Law Promotes Ignorance: The Case of Industrial Chemicals and Their Risks, Chap 7, Kourany J, Carrier M, eds. *Science and the Production of Ignorance : When the Quest for Knowledge Is Thwarted*. Cambridge, MIT Press, 2020.
- Crezo A, We used to put radium in coffee, *Health*, The Atlantic, 10 Oct 2012 <https://www.theatlantic.com/health/archive/2012/10/we-used-to-put-radium-in-coffee/263408/> accessed 18 Dec 2019.
- Cribb J, *Poisoned Planet: How constant exposure to man-made chemicals is putting your life at risk*, Allen & Unwin, Sydney, 2014.

- Cronkite E, Brecher G, The protective effect of granulocytes in radiation injury, *Ann N Y Acad Sci*, 1955, 59, 815–33.
- Crone N, Reder A, Severe tetanus in immunized patients with high anti-tetanus titers, *Neurology*, 1992, 42, 4, 761-4.
- CSIRO, Agriculture and Food <https://www.csiro.au/en/Research/AF> accessed 10 Jan 21
- Cutts F, Zaman S, Enwere G, Jaffar S, Levine O, Okoko J, Oluwalana C, Vaughan A, Obaro S, Leach A, McAdam K, Efficacy of nine-valent pneumococcal conjugate vaccine against pneumonia and invasive pneumococcal disease, In: The Gambia, randomised, double-blind, placebo-controlled trial. *The Lancet*, 2005, 365, 9465, 1139-46.
- Dadachova E, Casadevall A, Ionizing radiation: how fungi cope, adapt, and exploit with the help of melanin. *Current opinion in microbiology*, 2008, 11, 6, 525–31.
- Davis D, Bradlow H, Wolff M, Woodruff T, Hoel D, Anton-Culver H. Medical hypothesis: xenoestrogens as preventable causes of breast cancer, *Environmental health perspectives*, 1993, 101, 5, 372-7.
- De Santis M, Cesari E, Nobili E, Straface G, Cavaliere AF, Caruso A, Radiation effects on development. *Birth Defects Research Part C, Embryo Today, Reviews*, 2007, 81, 3, 177-82.
- Delborne J, Suppression and dissent in science. In: Bretag T (Ed), *Handbook of Academic Integrity* Singapore: Springer, 2016, 943–56.
- Delborne J, Transgenes and transgressions: scientific dissent as heterogeneous practice, *Social Studies of Science*, 2008, 38, 4, 509-41.
- Di Bartolomeis M, Kegley S, Mineau P, Radford R, Klein K. An assessment of acute insecticide toxicity loading (AITL) of chemical pesticides used on agricultural land in the United States, *PloS one*, 2019 14, 8, e0220029.
- Dickson D, *The new politics of science*. New York, Pantheon, 1984.
- Diniz C, Pellini A, Ribeiro A, Tedardi M, de Miranda M, Touse M, Baquero O, dos Santos P, Chiaravalloti-Neto F, Breast cancer mortality and associated factors in São Paulo State, Brazil: an ecological analysis, *BMJ open*, 2017, 7, 8, e016395.
- Di Prisco G, Cavaliere V, Annoscia D, Varricchio P, Caprio E, Nazzi F, Gargiulo G, Pennacchio F, Neonicotinoid clothianidin adversely affects insect immunity and promotes replication of a viral pathogen in honey bees, *Proceedings of the National Academy of Sciences*, 2013, 110, 46, 18466-71.
- Doll R, Wakeford R, Risk of childhood cancer from fetal irradiation, *British J Rad*, 1997, 70, 830, 130-9.
- Doshi P, Pfizer and Moderna’s “95% effective” vaccines—let’s be cautious and first see the full data, Comment and opinion from The BMJ’s international community of readers, authors, and editors, 26 Nov 2020 [Peter Doshi: Pfizer and Moderna’s “95% effective” vaccines—let’s be cautious and first see the full data - The BMJ](#) accessed 26 Jan 2021.
- Douglass C, Joshipura K Caution needed in fluoride and osteosarcoma study’, *Cancer Causes & Control*, 2006, 17, 4, 481-2.
- Downs C, Kramarsky-Winter E, Segal R, Fauth J, Knutson S, Bronstein O, Ciner F, Jeger R, Lichtenfeld Y, Woodley C, Pennington P, Cadenas K, Kushmaro A, Loya Y, Toxicopathological effects of the sunscreen UV filter, oxybenzone (benzophenone-3), on coral planulae and cultured primary cells and its

environmental contamination in Hawaii and the U.S. Virgin Islands, *Arch Environ Contam Toxicol*, 2016, 70, 2, 265-88.

Dufresne R, Offstein E, On the Virtues of Secrecy in Organizations, *J Management Inquiry*, 2008, 17, 2, 102–6.

Dukes G, Braithwaite J, Moloney J, *Pharmaceuticals, Corporate Crime and Public Health*. Cheltenham, England, Edward Elgar, 2014.

Edwards D, Director, Office of Pesticide Programs, US Environmental Protection Agency, Letter to the Sierra Club Genetic Engineering Committee, 10 Oct 2008). (referenced in Kleinman 2015p189).

Edwards T, Hamlin H, Reproductive endocrinology of environmental nitrate, *General and comparative endocrinology*, 2018, 265, 31-40.

Elkind P, McLean B, *The Smartest Guys in the Room: The amazing rise and scandalous fall of Enron*. London: Penguin Books, 2005.

Elliott K, Selective ignorance in environmental research. In: *Routledge International Handbook of Ignorance Studies*, (eds) Gross M, McGoey L, Routledge, 2015, Chap 17, 165-73.

Elliott K, Selective ignorance and agricultural research, science, technology, & human Values, 2013, 38, 328–50.

Elliott K, Financial conflicts of interest and criteria for research credibility, *Erkenntnis*, 2014, 79, S5, 917-37.

Elliott K, Standardized study designs, value judgments, and financial conflicts of interest in research, *Perspectives on Science*, 2016, 24, 5, 529-51.

Elliott T, Ledney G, Shoemaker M, Knudson G, Management of post irradiation infection: lessons from animal models, *Mil Med*, 2004, 169, 194–7.

EMA: European Medicines Agency, Medicine, Calcitonin
<https://www.ema.europa.eu/en/medicines/human/referrals/calcitonin> accessed 27 Dec 20.

Eng M, Stutchbury B, Morrissey C, A neonicotinoid insecticide reduces fueling and delays migration in songbirds, *Science*, 2019, 365, 6458, 1177-80.

Engel S, Martin B, Union Carbide and James Hardie: Lessons in politics and power, *Global Society, J Interdisciplinary Int Rel*, 2006, 20, 4, 475-90.

ES: Endocrine Society, Some Sunscreen Ingredients May Disrupt Sperm Cell Function, Press Release 1 Apr 2016 <https://www.endocrine.org/news-and-advocacy/news-room/2016/some-sunscreen-ingredients-may-disrupt-sperm-cell-function> accessed 12 Nov 20.

ESC: The Task Force for Preoperative Cardiac Risk Assessment and Perioperative Cardiac Management in Non-cardiac Surgery of the European Society of Cardiology, Guidelines for pre-operative cardiac risk assessment and perioperative cardiac management in non-cardiac surgery, *European Heart Journal*, 2009, 30, 2769a.

Evans R, Tough on Crime? Pfizer and the CIHR, *Healthc Policy*, 2010, 5, 4, 16-25.

Fairlie I, Radioactive spikes from nuclear plants - a likely cause of childhood leukemia, *The Ecologist*, 29 Sep 2014 <https://theecologist.org/2014/sep/29/radioactive-spikes-nuclear-plants-likely-cause-childhood-leukemia> accessed 23 Dec 2019.

Fang L, Emails show Monsanto orchestrated GOP effort to intimidate cancer researchers: Documents suggest the firm has antagonized regulators and applied pressure to shape research of the world's leading herbicide, *The Intercept*, 23 Aug 2019 [Monsanto Email Drafting Letters to the Senate Agriculture Committee Through Third Parties to Defend Glyphosate \(documentcloud.org\)](#) accessed 23 Dec 20.

Farombi E, Alabi M, Akuru T, Kolaviron modulates cellular redox status and impairment of membrane protein activities induced by potassium bromate KBrO₃ in rats, *Pharmacol Res*, 2002, 45, 63-68.

FDA, Labelling for salicylate-containing products: advanced notice of proposed rulemaking, *Federal Register*, 1982, 47, 57886.

FDA: US Food and Drug Administration, Petitions, Bisphenol A (BPA): Use in Food Contact Applications 2018 <https://www.fda.gov/food/food-additives-petitions/bisphenol-bpa-use-food-contact-application> accessed 10 Jan 21.

Feijó F, Ballard C, Foletto K, Batista B, Neves A, Ribeiro M, Bertoluci M, Saccharin and aspartame, compared with sucrose, induce greater weight gain in adult Wistar rats, at similar total caloric intake levels, *Appetite*, 10 Oct 2012, Epub 2012 Oct 19.

Figueiredo J, Hsu L, Hutter C, Lin Y, Campbell P, Baron J, Berndt S, Jiao S, Casey G, Fortini B, Chan A Genome-wide diet-gene interaction analyses for risk of colorectal cancer, *PLoS genetics*, 2014, 10, 4, e1004228.

Fischer D, Response to National Honey Bee Advisory Broad Letter on Imidacloprid Registration Review, *Comp Bee Culture's Catch The Buzz*, 17 Jul 2009 (referenced in Kleinman 2015p187).

Fleming L, Kirkpatrick B, Backer L, Walsh C, Nierenberg K, Clark J, Reich A, Hollenbeck J, Benson J, Cheng Y, Naar J, Review of Florida red tide and human health effects, *Harmful Algae*, 2011, 10, 2, 224-33.

FOTE: Friends of the Earth, Gardeners beware: bee-toxic pesticides found in "bee-friendly" plants sold at garden centers nationwide, 2013 [Gardeners-Beware-Report-11.pdf \(foe.org\)](#) accessed 28 Dec 20.

Freeman A, Cyanocobalamin--a case for withdrawal: discussion paper, *J R Soc Med*. 1992, 85, 11, 686-7.

Frickel S. Not here and everywhere: The non-production of scientific knowledge, In: *Routledge Handbook of Science, Technology, and Society*. London, 2014a, Chap 15, 263-76.

Frickel S, Gibbon S, Howard J, Kempner J, Ottinger G, Hess D, Undone Science: Social movement challenges to dominant scientific practice, *Science, Technology, and Human Values* 2010, 35, 4, 444-73.

Frickel S, Edwards M, Untangling ignorance in environmental risk assessment, In: *Powerless science? Science and politics in a toxic world*, ed. Jas N, Boudia S, Berghahn, 2014b, Chap 10, 215-33.

Frickel S, Kinchy A, Lost in Space: Geographies of ignorance in science and technology studies, In: *Routledge International Handbook of Ignorance Studies*, (eds) Gross M, McGoey L, Routledge, 2015, Chap 18, 174-82.

Friedman J, Popper, Weber, and Hayek: The Epistemology and Politics of Ignorance, *Critical Review* 2005, 17, 1-2, 1-58.

Galison P, Removing Knowledge: The Logic of Modern Censorship, In: *Agnology: The Making and Unmaking of Ignorance*, ed. Proctor R, Schiebinger L, Chap 2, 37-54. Stanford, CA, Stanford University Press, 2008.

Gallardo E, Coggan A, What Is in Your Beet Juice? Nitrate and nitrite content of beet juice products marketed to athletes, *International journal of sport nutrition and exercise metabolism*, 2018, 9, 00, 1-5.

Galletti P, Joyet G, Effect of fluorine on thyroidal iodine metabolism in hyperthyroidism, *J Clin Endocrinol Metab*, 1958, 18, 10, 1102-10
<https://pdfs.semanticscholar.org/cf2c/d1812505389cd3ec7d8cd2c6cf8574730bad.pdf> accessed 1 June 2019.

García-Pérez A, Irigoyen-Camacho M, Borges-Yáñez A, Fluorosis and dental caries in Mexican schoolchildren residing in areas with different water fluoride concentrations and receiving fluoridated salt. *Caries Res*, 2013, 47, 4, 299-08.

Gatti A, Montanari S. New quality-control investigations on vaccines: Micro-and Nano contamination. *Int J Vaccines Vaccin*, 2016, 4, 1, 00072.

Gelbspan R, *The Heat is On*, New York: Perseus, 1997.

Gentempo P, (interviewer), *Vaccines Revealed*, Episode 2, Interviews, 2017.

Giacaman R, Campos P, Muñoz-Sandoval C, Castro R, Cariogenic potential of commercial sweeteners in an experimental biofilm caries model on enamel, *Arch Oral Biol*, 2013, 58, 9, 1116-22.

Gilbert D, How mental systems believe, *American Psychologist*, 1991, 46, 107–19.

Glibert P, Burkholder J, Kana T, Alexander J, Skelton H, Shilling C, Grazing by *Karenia brevis* on *Synechococcus* enhances its growth rate and may help to sustain blooms. *Aquat Microb Ecol*, 2009, 55, 17-30.

Glibert P, Beusen A, Harrison J, Dürr H, Bouwman A, Laruelle G, Changing land-, sea-, and aircapes: sources of nutrient pollution affecting habitat suitability for harmful algae, In: *Global Ecology and Oceanography of Harmful Algal Blooms*, Springer, Cham, 2018, 53-76.

Gilson E, Vulnerability, ignorance and oppression, *Hypatia*, 2011, 26, 2, 309–32

Gonzalez H, Farbroth A, Larkö O, Wennberg A, Percutaneous absorption of the sunscreen benzophenone-3 after repeated whole-body applications, with and without ultraviolet irradiation. *Br J Dermatol*, 2006, 154, 2, 337-40.

Gøtzsche P, *Deadly medicines and organized crime. How big pharma has corrupted healthcare*. New York, Radcliffe, 2013.

Gray G, Cohen J, Cunha G, Hughes C, McConnell E, Rhomberg L, Sipes I, Mattison D, “Weight of the Evidence Evaluation of Low-Dose Reproductive and Developmental Effects of Bisphenol A, Human and Ecological Risk Assessment, 2004,10, 875–21.

Greibe E, Nymark O, Fedosov S, Heegaard C, Nexø E, Dietary intake of vitamin b12 is better for restoring a low b12 status than a daily high-dose vitamin pill: an experimental study in rats, *Nutrients*. 2018, 10, 8, pii, E1096.

Gross A, Memorandum from Mr. Adrian Gross, Scientific Investigations Staff to Mr. Carlton Sharp, Chairman, Searle Investigation Task Force, 29 Sep 1975, Reprinted in U.S. Senate Joint Hearings before the Subcommittee on Health of the Committee on Labor and Public Welfare and the Subcommittee on Administrative Practice and Procedure of the Committee on the Judiciary, "Preclinical and Clinical Testing by the Pharmaceutical Industry, 1975, Part 1," No. Y4.L11/2:P49/2/975, CIS# S541-16, p440+

Gross A, Memorandum from Mr. Adrian Gross, Scientific Investigations Staff to Mr. Carlton Sharp, Chairman, Searle Investigation Task Force, 15 Mar 1976, Reprinted in U.S. Senate Joint Hearings before the Subcommittee on Health of the Committee on Labor and Public Welfare and the Subcommittee on Administrative Practice and Procedure of the Committee on the Judiciary, "Preclinical and Clinical

Testing by the Pharmaceutical Industry, 1976, Part 3," No. Y4.L11/2:P49/2/976/pt.3, CIS# S541-12, 310-76.

Gross A, Statement from Dr. Andrian Gross, Former FDA Investigator and Scientist, "Aspartame Safety Act," Congressional Record, Volume 131, No. 106, 1 Aug 1985, S10835-40.

Gross A, Letter from Dr. Andrian Gross, Former FDA Investigator and Scientist to Senator Howard Metzenbaum regarding pre-approval tests by G.D. Searle, 30 Oct 1987a, Reprinted in US Senate 1987, 430-39.

Gross A, Letter from Dr. Andrian Gross, Former FDA Investigator and Scientist to Senator Howard Metzenbaum regarding pre-approval tests by G.D. Searle, 3 Nov 1987b, Reprinted in US Senate 1987b, 443-53.

Gross M, The unknown in process: Dynamic connections of ignorance, non-knowledge and related concepts. *Current Sociology*, 2007, 55, 5, 742–59.

Gross M, *Ignorance and Surprise: Science, Society, and Ecological Design*, Cambridge, MA, MIT Press, 2010.

Grosse Y, Baan R, Straif K, Secretan B, El Ghissassi F, Cogliano V, WHO International Agency for Research on Cancer Monograph Working Group, Carcinogenicity of nitrate, nitrite, and cyanobacterial peptide toxins, *Lancet Oncol.* 2006, 7, 8, 628–29.

Grotz K, Genitsariotis S, Vehling D, Al-Nawas B, Long-term oral *Candida* colonization, mucositis and salivary function after head and neck radiotherapy, *Support Care Cancer*, 2003, 11, 717–21.

Han P, Bagenna B, Fu M, The sweet taste signalling pathways in the oral cavity and the gastrointestinal tract affect human appetite and food intake: A review. *International journal of food sciences and nutrition*, 2019, 70, 2, 125-35.

HCHC: House of Commons Health Committee, *The Influence of the Pharmaceutical Industry*, Fourth Report of Session 2004–05, Volume I, Report, together with formal minutes Ordered by The House of Commons, printed 22 March 2005.

Health Canada, Press Release: Important Changes to the Availability and Conditions of Use for Drugs Containing Calcitonin, Ottawa, Government of Canada, 2013.

Heckenlively K, Mikovits J, *Plague, One Scientist's Intrepid Search for the Truth about Human Retroviruses and Chronic Fatigue Syndrome (ME/CFS), Autism, and Other Diseases*, Skyhorse; Reprint edition 2017.

Heffernan M, *Willful Blindness: Why We Ignore the Obvious at Our Peril*, New York, Walker and Company, 2012.

Herbert J, *EPA Ordered to Reinstate Whistleblower*, The Associated Press, 18 Dec 1992

<http://fluoridealert.org/articles/ntp10/> accessed 17 Feb 2019.

Hess D, Suppression, bias, and selection in science: The case of cancer research. *Account Research*, 1999, 6,4, 245–57.

Hess D, *Alternative Pathways in Science and Industry*, Cambridge, MA, MIT Press, 2007.

Hess D, The Potentials and Limitations of Civil Society Research: Getting Undone Science Done, *Sociological Inquiry*, 2009, 79, 3, 306–27.

- Hess D, Environmental reform organisations and undone science in the united states: Exploring environmental, health and safety implications of nanotechnology, *Science as Culture*, 2010, 19, 2, 181-14.
- Hess D, Undone Science and Social Movements: A review and typology. In *Routledge International Handbook of Ignorance Studies*, (eds) Gross M, McGoey L, Routledge, 2015, Chap 15, 141-54.
- Hess D, Undone science: social movements, mobilised publics, and industrial transitions, MIT Press, 2016, Chap 1.
- Higgins A, Whether or not Roundup is safe, the gardener has better options, *Washington Post*, 18 Sep 2019 https://www.washingtonpost.com/lifestyle/home/whether-or-not-roundup-is-safe-the-gardener-has-better-options/2019/09/17/8ccb8a5e-ca95-11e9-a1fe-ca46e8d573c0_story.html accessed 9 Jan 21.
- Higgins E, Knowledge activation: Accessibility, applicability, and salience, In: Higgins E, Kruglanski W, (eds) *Social Psychology: Handbook of Basic Principles*, New York: Guilford Pres, 1996.
- Hirzy J, The National Treasury Employees Union. Why EPA's headquarters union of scientists opposes fluoridation. *Fluoride*, 1999, 32, 179–86
https://www.juneau.org/beta_transfer/clerk/boards/Fluoride/Pages1494-1501_Exhibit_G.pdf accessed 17 Feb 2019
- Hood C, The risk game and the blame game. *Government and Opposition*, 2002, 37, 1, 15–37.
- Holton K, Taren D, Thomson C, Bennett R, Jones K, [The effect of dietary glutamate on fibromyalgia and irritable bowel symptoms](#), *Clin Exp Rheumatol*, Epub 4 Jul 2012.
- Hudson-Hanley B, Irvin V, Flay B, MacDonald M, Kile M, Prenatal PBDE exposure and neurodevelopment in children 7 years old or younger: a systematic review and meta-analysis, *Current Epidemiology Reports*, 2018, 5, 1, 46-59.
- Husten L, Medicine or mass murder? guideline based on discredited research may have caused 800,000 deaths in europe over the last 5 years, *Forbes*, 15 Jan 2014
<https://www.forbes.com/sites/larryhusten/2014/01/15/medicine-or-mass-murder-guideline-based-on-discredited-research-may-have-caused-800000-deaths-in-europe-over-the-last-5-years/?sh=78a934b547ec> accessed 16 Nov 20.
- Husten L, ESC may re-examine guidelines in wake of poldermans dismissal, *Forbes*, 23 Nov 2011
<https://www.forbes.com/sites/larryhusten/2011/11/23/esc-may-re-examine-guidelines-in-wake-of-poldermans-dismissal/?sh=275f0d5f51c0> accessed 16 Nov 20.
- Huynh W, Cordato D, Kehdi E, Masters L, Dedousis C, Post-vaccination encephalomyelitis: literature review and illustrative case. *Journal of clinical neuroscience : official journal of the Neurosurgical Society of Australasia*, 2008 15, 12, 1315–22 <https://doi.org/10.1016/j.jocn.2008.05.002>.
- Hvistendahl M, Coal ash is more radioactive than nuclear waste, *Scientific American*, 13 Dec 2007
<https://www.scientificamerican.com/article/coal-ash-is-more-radioactive-than-nuclear-waste/>
- IAASTD: International Assessment of Agricultural Knowledge, Science, and Technology for Development, *Agriculture at a crossroads: executive summary of the synthesis report*, Washington, DC, Island Press, 2009.
- IAEA: International Atomic Energy Agency, *Radiation in everyday life*
<https://www.iaea.org/Publications/Factsheets/English/radlife> accessed 13 Dec 2019.

IBCERCC: Interagency Breast Cancer and Environment Research Coordinating Committee, Breast cancer and the environment: Prioritizing prevention, 2013
https://www.niehs.nih.gov/about/assets/docs/breast_cancer_and_the_environment_prioritizing_prevention_508.pdf accessed 16 Mar 2019.

ICFTU: International Confederation of Free Trade Unions, The trade union report on bhopal: the report of the ICFTU-ICEF mission to study the causes and effects of the methyl isocyanate gas leak at the union carbide pesticide plant in Bhopal, India on December 2-3, 1984, published jointly by the ICFTU and the ICEF, 1985.

Iyyaswamy A, Rathinasamy S, Effect of chronic exposure to aspartame on oxidative stress in the brain of albino rats, *J Biosci.* 2012, 37, 4, 679-88.

Jacobsen R, Is sunscreen the new margarine? 10 Jan 2019
<https://www.outsideonline.com/2380751/sunscreen-sun-exposure-skin-cancer-science> accessed 15 May 2019.

Jacobson J, Jacobson S, The effects of perinatal exposure to polychlorinated biphenyls and related contaminants, In: Prenatal exposure to toxicants, Ed: Needleman H, Bellinger D, 1994, 130-47. Baltimore: Johns Hopkins University Press.

Janjua N, Kongshoj B, Andersson A, Wulf H, Sunscreens in human plasma and urine after repeated whole-body topical application. *J Eur Acad Dermatol Venereol*, 2008, 22, 4, 456-61.

Janjua N, Mogensen B, Andersson A, Petersen J, Henriksen M, Skakkebaek N, Wulf H, Systemic absorption of the sunscreens benzophenone-3, octyl-methoxycinnamate, and 3-(4-methyl-benzylidene) camphor after whole-body topical application and reproductive hormone levels in humans. *J Invest Dermatol*, 2004, 123, 1, 57-61.

Jasanoff S, Bhopal's trials of knowledge and ignorance, *ISIS*, 2007, 98, 2, 344–50.

Jansen-van der Weide M, Greuter M, Jansen L, et al, Exposure to low-dose radiation and the risk of breast cancer among women with a familial or genetic predisposition: a meta-analysis, *Eur Radiol* 2010, 20, 2547–56.

Jaxen J, Ghostwriting science, corporate malfeasance lead to \$80m jury decision in second roundup cancer trial, *Greenmedinfo*, 29 Mar 2019 [http://www.greenmedinfo.com/blog/ghostwriting-science-corporate-malfeasance-lead-80m-jury-decision-second-roundup-?utm_source=Daily+Greenmedinfo.com+Email+List&utm_campaign=456bd1f8e1-roundup&utm_medium=email&utm_term=0_193c8492fb-456bd1f8e1-91371377&ct=t\(roundup\)&mc_cid=456bd1f8e1&mc_eid=05bfd7e656](http://www.greenmedinfo.com/blog/ghostwriting-science-corporate-malfeasance-lead-80m-jury-decision-second-roundup-?utm_source=Daily+Greenmedinfo.com+Email+List&utm_campaign=456bd1f8e1-roundup&utm_medium=email&utm_term=0_193c8492fb-456bd1f8e1-91371377&ct=t(roundup)&mc_cid=456bd1f8e1&mc_eid=05bfd7e656) Accessed 11 Apr 2019.

Jeong H, Park J, Nho J, Park M, Ha J, Seong K, Jeng C, Seong C, Lee K, Yih W, Feeding by red-tide dinoflagellates on the cyanobacterium *Synechococcus*, *Aquat Microb Ecol*, 2005, 41, 2, 131-43.

Jhally S, Lewis J, Morgam M, The gulf war: a study of the media, public opinion and public knowledge, Research report, Centre for the Study of Communication, Doc #P-8, Feb 1991.

Jianjie C, Wenjuan X, Jinling C, Jie S, Ruhui J, Meiyan L. Fluoride caused thyroid endocrine disruption in male zebrafish (*Danio rerio*), *Aquatic Toxicology*, 2016, 171, 48-58.

Johnson E, Goldstein D, Do defaults save lives? *Science*, 2003, 302, 1338–39.

Kahneman D, A perspective on judgment and choice: Mapping bounded rationality. *American Psychologist*, 2003, 58, 697–20.

- Kahneman D, Thinking fast and slow, New York, Straus & Giroux, 2011.
- Kalelkar A, Little A, Investigation of large-magnitude incidents: Bhopal as a case study. London, AD Little; May 1988.
- Kaluza J, Åkesson A, Wolk A, Processed and unprocessed red meat consumption and risk of heart failure: prospective study of men. *Circulation, Heart Failure*, 2014, 7, 4, 552-7.
- Kaptschuk T, International Ignorance: A History of Blind Assessment and Placebo Controls in Medicine, *Bulletin of the History of Medicine*, 1998, 72, 3, 389–33.
- Karamoskos P, Nuclear power & public health, Evatt Foundation <https://evatt.org.au/papers/nuclear-power-public-health.html> accessed 28 Dec 2019.
- Kassirer J, On the take: How medicine's complicity with big business can endanger your health. Oxford, Oxford University Press, 2005.
- Katz J, Concerted ignorance: The social construction of cover-up, *Urban Life*, 1979, 8, 3, 295–16.
- Kelman H, Lee H, Crimes of obedience: toward a social psychology of authority and responsibility, New Haven, CT, Yale University Press, 1989.
- Kelton E, Is Big Pharma addicted to fraud? *Forbes*, 29 July 2013. <https://www.forbes.com/sites/erikakelton/2013/07/29/is-big-pharma-addicted-to-fraud/#2bde7e8d15fe> accessed 13 Oct 2018.
- Kempner J, Perlis C, Merz J, Forbidden Knowledge, *Science*, 2005, 307, 854.
- Kernan W, Viscoli C, Brass L, et al., Phenylpropanolamine and the risk of hemorrhagic stroke, *NEJM*, 2000, 343, 1826-32.
- Khambadkone S, Cordner Z, Dickerson F, Severance E, Prandovszky E, Pletnikov M, Xiao J, Li Y, Boersma G, Talbot C, Campbell W, Nitrated meat products are associated with mania in humans and altered behavior and brain gene expression in rats. *Molecular psychiatry*, 2018, 1.
- Kharb S, Sandhu R, Kundu Z, Fluoride levels and osteosarcoma, *South Asian J cancer*, 2012, 1, 2, 76–77.
- Kim J, Kwon W, Rahman M, Lee J, Yoon S, Park Y, You Y, Pang M, Effect of sodium fluoride on male mouse fertility, *Andrology*, 2015, 3, 3, 544-51.
- King B, Soule S, Social movements as extra-institutional entrepreneurs: the effects of protests on stock price returns, *Administrative Science Quarterly*, 2007, 52, 3, 412–43.
- Kirkpatrick B, Pierce R, Cheng Y, Henry M, Blum P, Osborn S, Nierenberg K, Pederson B, Fleming L, Reich A, Naar J, Kirkpatrick G, Backer L, Baden D, Inland transport of aerosolized florida red tide toxins, *Harmful Algae*, 2010, 9, 2, 123–242.
- Kirkpatrick B, Fleming L, Squicciarini D, Backer L, Clark R, Abraham W, Benson J, Cheng Y, Johnson D, Pierce R, Zaias J, Bossart G, Baden D, Literature review of florida red tide: implications for human health effects, *Harmful Algae*, 2004, 3, 2, 99–115.
- Kleinman D, Suryanarayanan S, Ignorance and industry: Agrichemicals and honey bee deaths, In: *Routledge International Handbook of Ignorance Studies*, (eds) Gross M, McGoey L, Routledge, 2015, Chap 19, 183-91.

- Klimová Z, Hojerová J, Beránková M, Skin absorption and human exposure estimation of three widely discussed UV filters in sunscreens—in vitro study mimicking real-life consumer habits, *Food Chem Toxicol*, 2015,83, 237-50.
- Kodavanti P, Neurotoxicity of Persistent Organic Pollutants: Possible Mode(s) of Action and Further Considerations, *Dose-Response* 2005, 3, 273–305.
- Kostoff R, Under-reporting of adverse events in the biomedical literature. *Journal of Data and Information Science*, 2016, 1, 4, 10-32.
- Kostoff R, Pervasive causes of disease. Georgia Institute of Technology: Atlanta, GA, 2015. Retrieved on 22 Sep 2016 from <http://hdl.handle.net/1853/53714>.
- Kourany J, Carrier M, The introduction in: Science and the production of ignorance: when the quest for knowledge is thwarted, Cambridge, MA, MIT Press, 2019.
- Kramer R, Michalowski R, Kauzlarich D, The origins and development of the concept and theory of state-corporate crime, *Crime and Delinquency*, 2002, 48, 2, 263–82.
- Krause M, Klit A, Blomberg J, et al., Sunscreens: are they beneficial for health? an overview of endocrine disrupting properties of UV-filters, *Int J Androl*, 2012, 35, 3, 424-36.
- Krimsky S, Science in the private interest: Has the lure of profits corrupted biomedical research? Lanham, MD, Rowman & Littlefield, 2003.
- Krimsky S, *Hormonal Chaos*, Lanham, MD, Johns Hopkins University Press, 2000.
- Krimsky S, Low Dose Toxicology: Narratives from the science - transcience interface. In *Powerless Science? Science and Politics in a Toxic World*, Eds Boudia S, Jas N, Berghahn, New York, 2014, Chap 11, 234-53.
- Kroger M, Meister K, Kava R, Low-calorie sweeteners and other sugar substitutes: a review of the safety issues, *Comprehensive Reviews in Food Science and Food Safety*, 2006, 5, 2, 35-47.
- Kroll-Smith S, Floyd H, *Bodies in protest: environmental illness and the struggle over medical knowledge*. New York: NYU Press, 2000.
- Kubsad D, Nilsson E, King S, Sadler-Riggelman I, Beck D, Skinner M, Assessment of Glyphosate Induced Epigenetic Transgenerational Inheritance of Pathologies and Sperm Epimutations: *Generational Toxicology*, *Scientific Reports*, 2019, 9, 6372.
- Kuehn R, Suppression of environmental science. *American Journal of Law & Medicine*, 2004, 30, 2–3, 333–69.
- Kuhlicke C, Vulnerability, ignorance and the experience of radical surprises. In: *Routledge International Handbook of Ignorance Studies*, (eds) Gross M, McGoey L, Routledge, 2015, Chap 25, 239-46.
- Kujawska M, Ignatowicz E, Ewertowska M, Adamska T, Markowski J, Jodynys-Liebert J, Attenuation of KBr O3-induced renal and hepatic toxicity by cloudy apple juice in rat, *Phytother. Res.*, 2013, 27, 1214-19.
- Kunisue T, Chen Z, Louis G, Sundaram R, Hediger M, Sun L, Kannan K, Urinary Concentrations of Benzophenone-type UV Filters in U.S. women and their association with endometriosis, *Environmental Science & Technology*, 2012, 46, 8, 4624-32.

Kuratsune M, Yoshimura T, Matsuzaka J, Yamaguchi A, Epidemiologic study on yusho, a poisoning caused by ingestion of rice oil contaminated with a commercial brand of polychlorinated biphenyls, *Environmental Health Perspectives*, 1972, 1, 119–28.

Kurokawa Y, Takayama S, Konishi Y, Long term in vivo carcinogenicity tests of potassium bromate, sodium hypochlorite and sodium chlorite conducted in Japan, *Environ. Health Prospect.*, 1987, 69, 221-36.

Lacey H, *Is Science Value Free?* London, Routledge, 1999.

Lamprou A, *Nanotechnology regulation: policies proposed by three organizations for the reform of the toxic substances control act.* Chemical Heritage Foundation, Center for Contemporary History and Policy, 2011.

Latané B, Darley J, *The Unresponsive Bystander*, New York, Appleton-Century-Crofts, 1970.

Lerner S, A tale of two toxic cities: the epa's bungled response to an air pollution crisis exposes a toxic racial divide, *The Intercept*, 25 Feb 2019 <https://theintercept.com/2019/02/24/epa-response-air-pollution-crisis-toxic-racial-divide/> accessed 24 Mar 2019.

Levin S, Monsanto found liable for California man's cancer and ordered to pay \$80m in damages, Agrochemical corporation found responsible for Roundup weedkiller's health risks in 'bellwether' federal trial, *Guardian*, 29 Mar 2019. <https://www.theguardian.com/business/2019/mar/27/monsanto-trial-verdict-cancer-jury#maincontent> accessed 18 Apr 2020.

Levine J, Gussow J, Hastings D, Eccher A, Authors' financial relationships with the food and beverage industry and their published positions on the fat substitute olestra, *American Journal of Public Health*, 2003, 93, 664–69.

Levy P, Roth H, Hwang P, Powers T, Beryllium and lung cancer: a reanalysis of a NIOSH cohort mortality study, *Inhalation Toxicology*, 2002, 14, 1003-15.

Lewis D, *Science for sale: how the us government uses powerful corporations and leading universities to support government policies, silence top scientists, jeopardize our health, and protect corporate profits*, New York, NY, Skyhorse Publishing, 2014.

Lexchin J, How safe are new drugs? market withdrawal of drugs approved in canada between 1990 and 2009, *Open Medicine*, 2014, 8, e14–e19.

Li Z, Rava M, Bédard A, Dumas O, Garcia-Aymerich J, Leynaert B, Pison C, Le Moual N, Romieu I, Siroux V, Camargo C, Cured meat intake is associated with worsening asthma symptoms. *Thorax*. 2017, 72, 3, 206-12.

Light D, *Bearing the Risks of Prescription Drugs*. In: Light D, (ed.) *The Risks of Prescription Drugs*, New York, Columbia University Press, 2010.

Lindqvist P, Epstein E, Nielsen K, Landin-Olsson M, Ingvar C, Olsson H. Avoidance of sun exposure as a risk factor for major causes of death: a competing risk analysis of the Melanoma in Southern Sweden cohort, *Journal of internal medicine*, 2016, 280, 4, 375-87.

Lindqvist P, The winding path towards an inverse relationship between sun exposure and all-cause mortality, *Anticancer research*, 2018, 38, 2, 1173-8.

Linke B, Casagrande T, Cardoso L, Food additives and their health effects: A review on preservative sodium benzoate, *African Journal of Biotechnology*, 2018, 17, 10, 306-10.

- Longstaff P, Security, resilience, and communication in unpredictable environments such as terrorism, natural disasters, and complex technology, Nov 2005, Center for Information Policy Research, Harvard University, http://pirp.harvard.edu/pubs_pdf/longsta/longsta-p05-3.pdf accessed 22 March 2014.
- Loveridge D, *Foresight: the art of and science of anticipating the future*, New York, Routledge, 2009.
- Lubitz S, Early reactions to Harvey's circulation theory: the impact on medicine, *Mt Sinai J Med*, 2004, 71, 4, 274-80.
- Lyons-Weiler J, Do matching autologous and foreign antigen peptide sequences contribute to vaccine-induced acute disseminated encephalomyelitis, In: *The Environmental and Genetic Causes of Autism*, Skyhorse Publishing NY, 2016.
- Lyons-Weiler, J, Thomas P, Relative incidence of office visits and cumulative rates of billed diagnoses along the axis of vaccination, *Int. J. Environ. Res. Public Health*, 2020, 17, 8674.
- Magnus D, Risk management versus the precautionary principle: agnotology as a strategy in the debate over genetically engineered organisms, In: *Agnotology, The making and unmaking of ignorance*, Stanford University Press, 2008, Chapter 11, 250-65.
- Malin A, Lesseur C, Busgang S, Curtin P, Wright R, Sanders A, Fluoride exposure and kidney and liver function among adolescents in the United States: NHANES, 2013–2016, *Environment international*, 2019a, 132, 105012.
- Malin A, Bose S, Busgang S, Gennings C, Thorpy M, Wright R, Wright R, Arora M, Fluoride exposure and sleep patterns among older adolescents in the United States: a cross-sectional study of NHANES 2015–2016, *Environmental Health*, 2019b, 18, 1, 106.
- Mao J, Jain A, Denslow N, Nouri M, Chen S, Wang T, Zhu N, Koh J, Sarma S, Sumner B, Lei Z, Bisphenol A and bisphenol S disruptions of the mouse placenta and potential effects on the placenta–brain axis, *Proceedings of the National Academy of Sciences*, 2020, 117, 9, 4642-52.
- Markey P, Bystander intervention in computer-mediated communication. *Computers in Human Behavior*, 2000, 16, 2, 183-8.
- Markowitz G, Rosner D, *deceit and denial: the deadly politics of industrial pollution*, Berkeley: University of California Press, 2002.
- Marks R, Foley P, Jolley D, Knight K, Harrison J, Thompson S, The effect of regular sunscreen use on vitamin D levels in an Australian population: results of a randomized controlled trial, *Archives of dermatology*, 1995, 131, 4, 415-21.
- Martin B, Sticking a needle into science: The case of polio vaccines and the origin of AIDS, *Social Studies of Science*, 1996, 26, 2, 245-76.
- Martin B, Suppressing research data: Methods, context, accountability, and responses. *Accountability in Research*, 1999a, 6, 333–72.
- Martin B, Suppression of dissent in science. *Research in Social Problems and Public Policy*, 1999b, 7, 105–35.
- Martin B, *Justice ignited: The dynamics of backfire*, Rowman & Littlefield, 2007.
- Martin B, Enabling scientific dissent. *New Doctor*, Dec 2008, 88, 2-5.

Martin B, On the suppression of vaccination dissent, *Science and engineering ethics*, 2015, 21, 1, 143–57.

Martin B, Fraud and the pharmaceutical Industry, (list only)
<https://www.uow.edu.au/~bmartin/dissent/documents/health/pharmfraud.html> accessed 13 Oct 2018.

Martin B, Baker A, Manwell C, Pugh C, Intellectual suppression, Angus & Robertson, 1986.

Maruna S, Copes H, What have we learned from five decades of neutralization research?, *Crime and Justice*, 2005, 32, 221–320.

Mathews D, Forsythe A, Brady Z, Butler M, Goergen S, Byrnes G, Giles G, Wallace A, Anderson P, Guiver T, McGale P, Cancer risk in 680 000 people exposed to computed tomography scans in childhood or adolescence: data linkage study of 11 million Australians. *BMJ*, 2013, 346, f2360.

Mathias R, Meekison W, Arcand T, Schechter M, The role of secondary vaccine failures in measles outbreaks, *AJPH*, 1989, 79, 4, 475-8.

Matsuoka L, Wortsman J, Hanifan N, Holick M, Chronic sunscreen use decreases circulating concentrations of 25-hydroxyvitamin D: a preliminary study, *Archives of dermatology*, 1988, 124, 12, 1802-4.

Matta M, Zusterzeel R, Pilli N, et al. Effect of Sunscreen Application Under Maximal Use Conditions on Plasma Concentration of Sunscreen Active Ingredients: A Randomized Clinical Trial, *JAMA*, 2019, 321, 21, 2082–91.

Matta M, Florian J, Zusterzeel R, et al. Effect of sunscreen application on plasma concentration of sunscreen active ingredients: a randomized clinical trial, *JAMA*, 2020, 323, 3, 256–67.

Matthews J, Cyanocobalamin [c-lactam] inhibits vitamin B12 and causes cytotoxicity in HL60 cells: methionine protects cells completely, *Blood*, 1997, 89, 12, 4600-7.

Matza D, *Delinquency and Drift*, New York, Wiley, 1964.

Mayer B, Bergstrand K, Running K, Science as comfort: The strategic use of science in post-disaster settings. In: *Routledge Handbook of Science, Technology, and Society*. London. 2014, Chap 24, 419-32.

Mayer A, Historical changes in the mineral content of fruits and vegetables, *Brit. Food J.* 1997, 99, 207–211.

Maynard A, et al. Safe Handling of Nanotechnology, *Nature* 2006, 444, 267–69.

McClenahan S, Krause P, Uhlenhaut C, Molecular and infectivity studies of porcine circovirus in vaccines, *Vaccine*, 2011, 29, 29–30, 4745-53.

McCulloch J, Tweedale G, Shooting the messenger: The vilification of Irving J. Selikoff. *International Journal of Health Services*, 2007, 37, 4, 619–34.

McCurry J, Fukushima: Japan will have to dump radioactive water into Pacific, minister says, *The Guardian*, 10 Sep 2019 <https://www.theguardian.com/environment/2019/sep/10/fukushima-japan-will-have-to-dump-radioactive-water-into-pacific-minister-says#:~:targetText=Fukushima%3A%20Japan%20will%20have%20to%20dump,water%20into%20Pacific%2C%20minister%20says&targetText=Tepeco%20has%20attempted%20to%20remove,a%20radioactive%20isotope%20of%20hydrogen>.

McCurry J, Fukushima: Japan announces it will dump contaminated water into sea, *The Guardian*, 13 Apr 2021 <https://www.theguardian.com/environment/2021/apr/13/fukushima-japan-to-start-dumping-water-pacific-ocean>

McDonough W, Braungart M, *Cradle to cradle: remaking the way we make things*. New York: North Point Press, 2002.

McGarity T, Wagner W, *Bending Science*, Cambridge, MA, Harvard University Press, 2008.

McGoey L, The logic of strategic ignorance, *British Journal of Sociology*, 2012b 63, 3, 553–76.

McGoey L, Strategic unknowns: toward a sociology of ignorance, *Special issue of Economy and Society*, 2012a, 41, 1.

McGoey L, On the will to ignorance in bureaucracy, *Economy and Society*, 2007, 36, 2, 212–35.

MCT Lawyers Website, What is acute disseminated encephalomyelitis or ADEM? <https://www.mctlawyers.com/vaccine-injury/acute-disseminated-encephalomyelitis-adem/> accessed 17 Sep 18.

MedlinePlus: NIH, US National Library of Medicine, Drugs, Herbs and Supplements, Cyanocobalamin Injection, review 2010 <https://medlineplus.gov/druginfo/meds/a605007.html> accessed 11 Jan 21.

Melnick R, Lucier G, Wolfe M, Hall R, Stancel G, Prins G, Gallo M, Reuhl K, Ho S, Brown T, et.al. Summary of the National Toxicology Program’s Report of the Endocrine Disruptors Low-Dose Peer Review, *Environmental Health Perspectives*, 2002, 110, 427–31.

Mesnage R, Bernay B, Séralini G, Ethoxylated adjuvants of glyphosate-based herbicides are active principles of human cell toxicity. *Toxicology*, 2013, 313, 2-3, 122-8.

Mesnage R, Benbrook C, Antoniou M, Insight into the confusion over surfactant co-formulants in glyphosate-based herbicides, *Food Chem Toxicol*, 2019, 128, 137-45.

Michaels D, Doubt is their product, *Scientific American*, 2005b, 292, 6, 96-101.

Michaels D, *Manufactured uncertainty: Contested science and the protection of the public’s health an environment*, In: *Agnotology, The making and unmaking of ignorance*, Stanford University Press, 2008a, Chapter 4, 90-107.

Michaels D, *Doubt is their product: how industry’s assault on science threatens your health*. Oxford, Oxford University Press, 2008b.

Michaels D, Monforton C, *Manufacturing uncertainty: contested science and protection of the public’s health and environment*, *American Journal of Public Health* 2005a, 95, S1, S39–48.

Michotte A, *The perception of causality*, trans. Miles T, Miles E, New York, Basic Books, 1963.

Nickerson R, Confirmation bias: A ubiquitous phenomenon in many guises, *Review of General Psychology*, 1998, 2, 2, 175–220.

Miglioretti D, Lange J, Van Den Broek J, Lee C, Van Ravesteyn N, Ritley D, Kerlikowske K, Fenton J, Melnikow J, De Koning H, Hubbard R, Radiation-induced breast cancer incidence and mortality from digital mammography screening: a modeling study, *Annals of internal medicine*, 2016, 164, 4, 205-14.

Mineau P, Palmer C, The impact of the nation’s most widely used insecticides on birds, *American Bird Conservancy*, March 2013 [Neonic FINAL.pdf \(abcbirds.org\)](#) accessed 27 Dec 20.

Miner J, Bernard W, inventors; Sewerage Commission Of Cit, assignee. Process of preparing vitamin b12-active product from sewage sludge. United States patent US 2, 646, 386. 21 Jul 1953.

Molina V, Médici M, Taranto M, de Valdez G, Lactobacillus reuteri CRL 1098 prevents side effects produced by a nutritional vitamin B deficiency, *J Appl Microbiol*, 2009, 106, 2, 467-73.

Møller A, Mousseau T, The effects of natural variation in background radioactivity on humans, animals and other organisms, *Biological Reviews*, 2013, 88, 1, 226-54.

Mooney C, *The republican war on science*, New York, Basic Books, 2005.

Moore K, *Disrupting science: Social movements, American scientists, and the politics of the military 1945-1975*, Princeton University Press, 2008.

Moseman E, Iannacone M, Bosurgi L, Tonti E, Chevrier N, Tumanov A, Fu Y, Hacohen N, von Andrian U, B cell maintenance of subcapsular sinus macrophages protects against a fatal viral infection independent of adaptive immunity, *Immunity*, 2012, 36, 3, 415-26.

Mourtzinis S, Krupke C, Esker P, et al. Neonicotinoid seed treatments of soybean provide negligible benefits to US farmers, *Sci Rep* 2019, 9, 11207.

Moynihan R, Officials reject claims of drug industry's influence, *BMJ*, 2004, 329, 7467, 641.

Nagel S, vom Saal F, Thayer K, Dhar M, Boechler M, Welshons W, Relative binding affinity-serum modified access (RBA-SMA) assay predicts the relative in vivo bio-activity of the xenoestrogens bisphenol a and octylphenol, *Environmental Health Perspectives*, 1997, 105, 70–6.

Nature: The Weight of Evidence, *Nature* 2010, 464, 7292, 1103-4.
<https://www.nature.com/articles/4641103b> accessed 26 Sep 18

Naidu R, Ignore soil pollution at humanity's peril, CRC-CARE, Press Release, 5 Dec 2018
<https://www.crccare.com/news/ignore-soil-pollution-at-humanity-s-peril> accessed 12 Mar 2019.

NCIRS: National Centre for Immunisation Research and Surveillance, Publications, Vaccine Trials
https://www.ncirs.org.au/publications?field_archive_value=2017&field_publication_category_target_id=8&combine= accessed 27 Nov 20.

Neale R, Khan S, Lucas R, Waterhouse M, Whiteman D, Olsen C, The effect of sunscreen on vitamin D: a review, *British Journal of Dermatology*, 2019, 181, 5, 907-15.

Nestle M, Food company sponsorship of nutrition research and professional activities: a conflict of interest?" *Public Health Nutrition*, 2001, 4, 1015–22.

Netea M, Brown G, Kullberg B, Gow N, An integrated model of the recognition of *Candida albicans* by the innate immune system, *Nat Rev Microbiol*, 2008, 6, 67–78.

Neurath C, Limeback H, Osmunson B, Connett M, Kanter V, Wells C, Dental Fluorosis Trends in US Oral Health Surveys: 1986 to 2012, *JDR Clin Trans Res*, 6 Mar 2019.

Newbrun E, Horowitz H, Why we have not changed our minds about the safety and efficacy of water fluoridation, *Perspectives in Biology and Medicine*, 1999, 42, 4, 526–43.

NIH: US National Institutes of Health, NIH News in Health, Bad Air Day: Air Quality and Your Health, July 2011 <https://newsinhealth.nih.gov/2011/07/bad-air-day> accessed 10 Jan 21.

NRC: National Research Council, Toxicity testing for assessment of environmental agents: interim report. Washington, National Academy Press 2007.

NRC: National Research Council. Health risks from exposure to low levels of ionizing radiation: BEIR VII phase 2, National Academies Press, 2006a.

Odds F, Hanson M, Davidson A, Jacobsen M, Wright P, Whyte J, et al. One year prospective survey of Candida bloodstream infections in Scotland, J Med Microbiol, 2007, 56, 1066–75.

Olney W, Farber N, Spitznagel E, Robins L, Increasing brain tumor rates: is there a link to aspartame? J Neuropathol Exp Neurol, 1996, 55, 11, 1115-23.

Oreskes N, Conway E, Challenging knowledge: how climate science became a victim of the cold war, In: Agnotology, The making and unmaking of ignorance, (eds) Proctor R, Schiebinger L, Stanford University Press, 2008, Chap 3, 55-89.

Oreskes N, Conway E, Merchants of doubt: How a handful of scientists obscured the truth on issues from tobacco smoke to global warming, NY: Bloomsbury Press, 2011.

Ortiz-Pérez D, Rodríguez-Martínez M, Martínez F, Borja-Aburto V, Castelo J, Grimaldo J, de la Cruz E, Carrizales L, Díaz-Barriga F, Fluoride-induced disruption of reproductive hormones in men, Environmental Research, 2003, 93, 1, 20-30.

Ottinger G, Refining expertise: how responsible engineers subvert environmental justice challenges. New York, New York University Press, 2013.

Oxford Languages, Fluorosis Definition

<https://www.google.com/search?q=fluorosis+definition&oq=Fluorosis+def&aqs=chrome.0.0i457j69i57j0l2j0i395j0i22i30i395i5.8087j1j15&sourceid=chrome&ie=UTF-8> accessed 13 Jan 21.

Patel R, Stuffed and starved: the hidden battle for the world's food system, Brooklyn, NY, Melville House, 2007.

Pattison J, Hugtenburg R, Green S, Enhancement of natural background gamma-radiation dose around uranium microparticles in the human body, Journal of the Royal Society Interface, 2010, 7, 45, 603-11.

Pearlman M, Obert J, Casey L, The association between artificial sweeteners and obesity, Curr Gastroenterol Rep 19, 2017, 64.

Peixoto F, Comparative effects of the Roundup and glyphosate on mitochondrial oxidative phosphorylation, Chemosphere, 2005, 61, 8, 1115-22.

Pepino M, Tiemann C, Patterson B, Wice B, Klein S, Sucralose Affects Glycemic and Hormonal Responses to an Oral Glucose Load, Diabetes Care, 2013 Apr 30, Epub.

Peters J, Wyatt H, Foster G, Pan Z, Wojtanowski A, Vander Veur S, Herring S, Brill C, Hill J, The effects of water and non-nutritive sweetened beverages on weight loss during a 12-week weight loss treatment program, Obesity, 2014, 22, 6, 1415-21.

Petricciani J, Sheets R, Griffiths E, Knezevic I, Adventitious agents in viral vaccines: Lessons learned from 4 case studies, Biologicals, 2014, 42, 5, 223-36.

Pettis J, Johnson J, Dively G, Pesticide exposure in honey bees results in increased levels of the gut pathogen Nosema, Naturwissenschaften, 2012, 99, 2, 153-8.

- Pierson B, Merck accused of stonewalling in mumps vaccine antitrust lawsuit, Westlaw News June 5, 2015 <https://www.reuters.com/article/health-vaccine-idUSL1N0YQ0W820150604> accessed 2 Dec 20.
- Plater R, Planning to Use Roundup on Your Lawn? Here's What You Need to Know, Healthline, 9 Apr 2019 <https://www.healthline.com/health-news/is-it-safe-for-you-to-use-roundup-weed-killer-on-your-lawn-this-spring> accessed 9 Jan 21.
- Plummer K, Labelling theory revisited: Forty years on. In: Peters H, Dellwing M (eds) *Langweiliges Verbrechen [Boring Crimes]*, Weisbaden: VS Verlag, 2011.
- Pogoda J, Preston-Martin S, Howe G, et al. , An international case-control study of maternal diet during pregnancy and childhood brain tumor risk: a histology-specific analysis by food group, *Ann Epidemiol*, 2009, 19, 3, 148–60.
- Poland G, Jacobson R, The re-emergence of measles in developed countries: time to develop the next-generation measles vaccines? *Vaccine*, 2012, 30, 2, 103–4.
- Pongsavee M, Effect of sodium benzoate preservative on micronucleus induction, chromosome break, and Ala40Thr superoxide dismutase gene mutation in lymphocytes, *BioMed research international*, 2015, 103512.
- Pont A, Charron A, Brand R, Active ingredients in sunscreens act as topical penetration enhancers for the herbicide 2,4-dichlorophenoxyacetic acid, *Toxicol Appl Pharmacol*, 2004, 195, 3, 348-54.
- Prentice D, Miller D, Pluralistic ignorance and alcohol use on campus: Some consequences of misperceiving the social norm, *Journal of Personality and Social Psychology*, 1993, 64, 243–56.
- Prentice D, Mobilizing and weakening peer influence as mechanisms for changing behaviour: Implications for alcohol intervention programs. In: Prinstein M, Dodge K, (Eds), *Understanding peer influence in children and adolescents* (p161–80), New York, Guilford Press, 2008.
- Prentice D, Targeting ignorance to change behaviour, In *Routledge International Handbook of Ignorance Studies*, (eds) Gross M, McGoey L, Routledge, 2015, Chap 28, 266-73.
- Primack J, von Hippel F, *Advice and dissent: Scientists in the political arena*, New York, Basic Books 1974.
- Proctor R, Agnotology: a missing term to describe the cultural production of ignorance (and its study). In: Proctor R, Schiebinger L, (eds) *Agnotology: The making and unmaking of ignorance*, Stanford, CA, Stanford University Press, 2008, p1–33.
- Proctor R, *Cancer Wars*, New York, Basic Books, 1995.
- Rappert B, Present Absences: Hauntings and Whirlwinds in “-graphy,” *Social Epistemology*, 2014, 28, 1, 41–55.
- Ravetz J, The sin of science: ignorance of ignorance, *Science Communication*, 1993, 15, 2, 157–65.
- Ravetz J, Usable knowledge, usable ignorance incomplete science with policy implications. In: *Sustainable development of the biosphere*, (eds) Clark W, Munn R, Cambridge University Press, Cambridge, 1986, p415–32.
- Ravetz J, From Descartes to Rumsfeld: The rise and decline of ignorance-of-ignorance. In: *Routledge International Handbook of Ignorance Studies*, (eds) Gross M, McGoey L, Routledge, 2015, Chap 6, 53-60.

- Rawlinson P, Immunity and impunity: corruption in the state-pharma nexus, *International Journal for Crime, Justice and Social Democracy*, 2017, 6, 4, 86-99.
- Redding S, Zellzars R, Kirkpatrick W, McAtee R, Caceres M, Fothergill A, et al. Epidemiology of oropharyngeal *Candida* colonization and infection in patients receiving radiation for head and neck cancer. *J Clin Microbiol*, 1999, 37, 3896–900.
- Rehfeld A, Egeberg D, Almstrup K, Petersen J, Dissing S, Skakkebaek N, EDC IMPACT: Chemical UV filters can affect human sperm function in a progesterone-like manner. *Endocrine connections*, 2018, 7, 1, 16–25.
- Reyner S, To know or not to know? A note on ignorance as a rhetorical resource in geoengineering debates. In: *Routledge International Handbook of Ignorance Studies*, (eds) Gross M, McGoey L, Routledge, 2015, Chap 32, 308-27.
- Richtel M, Jacobs A, A Mysterious Infection, Spanning the Globe in a Climate of Secrecy: The rise of *Candida auris* embodies a serious and growing public health threat: drug-resistant germs. *The New York Times*, April 6, 2019 <https://www.nytimes.com/2019/04/06/health/drug-resistant-candida-auris.html> accessed 9 Apr 2019.
- Riddell J, Malin A, Flora D, McCague H, Till C, Association of water fluoride and urinary fluoride concentrations with attention deficit hyperactivity disorder in Canadian youth. *Environment international*, 2019, 133, 105190.
- Rips L, Causation from perception, *Perspectives on Psychological Science*, 2011, 6, 77–97
- Robert I, William B, Carcinogenicity of potassium bromate in rabbit, *Biol. Edu*, 1996, 34, 114-20
- Roberts J, Organisational Ignorance. In *Routledge International Handbook of Ignorance Studies*, Routledge, (eds) Gross M, McGoey L, 2015, Chap 37, 361-9.
- Roberts H, Aspartame and brain cancer, *Correspondence, Lancet*, 1997, 349, 9048, 362.
- Robinson G, Multiple causation in tort law: reflections on the DES cases. *Virginia Law Review*, 1982, 68, 713–69.
- Rogers T, *The Political Economy of Autism*, PhD Thesis, Department of Political Economy, School of Social and Political Sciences, Faculty of Arts and Social Sciences, University of Sydney, 2019.
- Rohrmann S, Overvad K, Bueno-de-Mesquita H, Jakobsen M, Egeberg R, Tjønneland A, Nailler L, Boutron-Ruault M, Clavel-Chapelon F, Krogh V, Palli D, Meat consumption and mortality-results from the European Prospective Investigation into Cancer and Nutrition, *BMC medicine*, 2013, 11, 1, 63.
- Rothstein H, Irving P, Walden T, Yearsley R, The risks of risk-based regulation: Insights from the environmental policy domain, *Environment International*, 2006, 32, 8, 1056–65.
- Roy D, Zeckhauser R, The anatomy of ignorance: Diagnoses from literature. In *Routledge International Handbook of Ignorance Studies*, (eds) Gross M, McGoey L, Routledge, 2015, Chap 7, 61-113.
- Ruiz R, Industrial chemicals lurking in your bloodstream, *Forbes*, Jan 21, 2010 <https://www.forbes.com/2010/01/21/toxic-chemicals-bpa-lifestyle-health-endocrine-disruptors.html#43757bcbbb91> accessed 20 Mar 2019.
- Russell D, *The Secret Trauma*, New York, Basic Books, 1986.

Ruszkiewicz J, Pinkas A, Ferrer B, Peres T, Tsatsakis A, Aschner M, Neurotoxic effect of active ingredients in sunscreen products, a contemporary review, *Toxicology Reports*, 2017, 4, 245-59.

Ryan B, Hotchkiss A, Crofton K, Gray E, In utero and lactational exposure to bisphenol A, in contrast to ethinyl estradiol, does not alter sexually dimorphic behavior, puberty, fertility, and anatomy of female LE rats, *Toxicological Sciences*, 2010, 114, 133–48.

Rycerz K, Jaworska-Adamu J, Effects of aspartame metabolites on astrocytes and neurons, *Folia neuropathologica*, 2013, 51, 1, 10-7.

Saadatmand S, Bretveld R, Siesling S, Tilanus-Linthorst M, Influence of tumour stage at breast cancer detection on survival in modern times: population based study in 173 797 patients, *BMJ*, 2015, 351, h4901.

Sai K, Hayashi M, Takagi A, Hasegawa R, Sofuni T, Kurokawa Y, Effects of antioxidants on induction of micronuclei in rat peripheral blood reticulocytes by potassium bromate, *Mutat. Res.*, 1992, 269, 113-18.

SCCS: Scientific Committee on Consumer Safety, Opinion on the fragrance ingredients *Tagetes minuta* and *T. patula* extracts and essential oils (phototoxicity only), 2015
http://ec.europa.eu/health/scientific_committees/consumer_safety/docs/sccs_o_172.pdf accessed 18 Feb 2019.

SCCS: Scientific Committee on Consumer Safety, Opinion on vetiveryl acetate (fragrance ingredient) 2014 http://ec.europa.eu/health/scientific_committees/consumer_safety/docs/sccs_o_167.pdf accessed 18 Feb 2019.

Schanck R, A study of community and its groups and institutions conceived of as behaviours of individuals, *Psychiatry Monographs*, 1932, 43, 2, 1–33.

Schatzker M, *The Dorito Effect: The surprising new truth about food and flavour*, Simon & Schuster, 2015.

Schechter A, Papke O, Tung K, Joseph J, Harris T, Dahlgren J, Polybrominated Diphenyl Ether flame retardants in the US population: current levels, temporal trends, and comparison with dioxins, dibenzofurans, and polychlorinated biphenyls, *J Occ Env Med*, 2005, 47, 3, 199–211.

Schernhammer E, Bertrand K, Birmann B, Sampson L, Willett W, Feskanich D, Consumption of artificial sweetener- and sugar-containing soda and risk of lymphoma and leukemia in men and women, *Am J Clin Nutr*, 24 Oct 2012 Epub.

Schlegel M, Osterwalder J, Galeazzi R, Vernazza P, Comparative efficacy of three mumps vaccines during disease outbreak in eastern Switzerland: cohort study, *BMJ*, 1999, 319, 7206, 352.

Schlumpf M, Cotton B, Conscience M, Haller V, Steinmann B, Lichtensteiger W, In vitro and in vivo estrogenicity of UV screens, *Environmental health perspectives*, 2001, 109, 3, 239-44.

Schneider A, McCumber D, *An air that kills: How the asbestos poisoning of Libby Montana, uncovered a national scandal*, New York, NY, G. P. Putnam's Sons, 2004.

Schultz P, Nolan J, Cialdini R, Goldstein N, Griskevicius V, The constructive, destructive, and reconstructive power of social norms, *Psychological Science*, 2007, 18, 5, 429–34.

Schumm W, Navigating treacherous waters—one researcher's 40 years of experience with controversial scientific research, *Comprehensive Psychology*, 2015, 4, 24. Retrieved on September 22, 2016, from <http://cop.sagepub.com/cgi/content/full/4/0/17.CP.4.24>.

Scinicariello F, Buser M, Serum testosterone concentrations and urinary bisphenol A, benzophenone-3, triclosan, and paraben levels in male and female children and adolescents: NHANES 2011–2012, *Environmental health perspectives*, 2016, 124, 12, 1898-904.

Seed T, Kaspar L. Acquired radioresistance of hematopoietic progenitors (granulocyte/monocyte colony-forming units) during chronic radiation leukemogenesis, *Cancer research*, 1992, 52, 6, 1469-76.

Senn J, McCulloch E, Radiation sensitivity of human bone marrow cells measured by a cell culture method, *Blood*, 1970, 35, 56–60.

Shadad A, Sullivan F, Martin J, Egan L, Gastrointestinal radiation injury: symptoms, risk factors and mechanisms, *World J Gastroenterol*, 2013, 19, 185–98.

Shafir E, *The behavioral foundations of public policy*. Princeton, NJ: Princeton University Press, 2012.

Sharpe R, Fisher J, Millar M, Jobling S, Sumpter J, Gestational lactational exposure of rats to xenoestrogen results in reduced testicular size and sperm reduction, *Environmental Health Perspectives*, 1995, 103, 1136–43.

Sharpe R, Turner K, Sumpter J, Endocrine disruptors and testis development, *Environmental Health Perspectives*, 1998, 106, A220–A221.

Shearer J, Swithers S, Artificial sweeteners and metabolic dysregulation: Lessons learned from agriculture and the laboratory, *Rev Endocr Metab Disord*, 2016, 17, 179–86.

Shenvi C, Hydroxocobalamin: Turning cyanide into vitamin B12, *Emergency Physicians Monthly*, 16 Feb 2016 <http://epmonthly.com/article/hydroxocobalamin-turning-cyanide-into-vitamin-b12/> accessed 11 Apr 2019.

Shoemaker P, Scenario planning: A tool for strategic thinking, *Sloan Management Review*, 1995, 37, 2, 25-40.

Sismondo S, Ghost management: How much of the medical literature is shaped behind the scenes by the pharmaceutical industry? *PLoS Medicine*, 2007, 4, 9, e286, 1429–33.

Siviter H, Bailes E, Martin C, et al, Agrochemicals interact synergistically to increase bee mortality, *Nature* 2021, 596, 389-92.

Soffritti M, Belpoggi F, Esposti D, Lambertini L, Tibaldi E, Rigano A, First experimental demonstration of the multipotential carcinogenic effects of aspartame administered in the feed to Sprague-Dawley rats, *Environ Health Perspect*, 2006, 114, 3, 379-85.

Soffritti M, Belpoggi F, Tibaldi E, Esposti D, Lauriola M, Life-span exposure to low doses of aspartame beginning during prenatal life increases cancer effects in rats, *Environ Health Perspect*, 2007, 115, 9, 1293-7.

Sorenson M, Grant W, *Embrace the Sun*, 1st ed, 2018, ISBN-10: 069207600X, ISBN-13: 978-0692076002.

Smith E, Smith A, *The story of the poisoning of a city, and the people who chose to carry the burden of courage*, Holt, Reinhart and Winstron, NY, 1975.

Smith S, *Vinegar: is it a “safer” herbicide?* Ohio State University, College of food, agricultural and environmental sciences, 21 Jul 2015 <https://fairfield.osu.edu/news/vinegar-it-%E2%80%9Csafer%E2%80%9D-herbicide> accessed 8 Jan 21.

- Smith J, Terpening C, Schmidt S, Gums J, Relief of fibromyalgia symptoms following discontinuation of dietary excitotoxins, *Ann Pharmacother*, 2001, 35, 6, 702-6.
- Smithson M, *Social Theories of Ignorance*. In: *Agnotology, The making and unmaking of ignorance*, Stanford University Press, 2008, Chap 9, 209-29.
- Somin I, *Rational Ignorance*. In: *Routledge International Handbook of Ignorance Studies*, (eds) Gross M, McGoev L, Routledge, 2015, Chap 29, 274-81.
- Somin I, *Democracy and Political Ignorance: Why Smaller Government Is Smarter*, Stanford, CA, Stanford University Press, 2013.
- Spellberg B, Ibrahim A, Edwards J, Filler S, Mice with disseminated candidiasis die of progressive sepsis, *J Infect Dis*, 2005, 192, 336–43.
- Spelte H, FDA supports sunscreen safety studies, *Dermatology News*, MD Edge, 22 Jan 2020 <https://www.mdedge.com/dermatology/article/215926/mixed-topics/fda-supports-sunscreen-safety-studies?> Accessed 11 Nov 2020.
- Steinemann A, International prevalence of chemical sensitivity, co-prevalences with asthma and autism, and effects from fragranced consumer products, *Air Qual Atmos Health*, 2019, Feb, 1-9.
- Steinhoff M, Omer S, Roy E, El Arifeen S, Raqib R, Dodd C, Breiman R, Zaman K, Neonatal outcomes after influenza immunization during pregnancy: a randomized controlled trial, *Canadian Medical Association Journal*, 1 Jan 2012, cmaj-110754.
- Stocking S, Holstein L, Purveyors of ignorance: Journalists as agents in the social construction of scientific ignorance. In: *Routledge International Handbook of Ignorance Studies*, (eds) Gross M, McGoev L, Routledge, 2015, Chap 12, 105-13.
- Sunstein C, *Simpler*, New York, Simon & Schuster, 2013.
- Swithers S, Artificial sweeteners produce the counterintuitive effect of inducing metabolic derangements, *Trends in Endocrinology & Metabolism*, 2013, 24, 9, 431-41
- Sykes G, Matza, D, Techniques of neutralisation: A theory of delinquency, *American Sociological Review*, 1957, 22, 6, 664–70
- Taber C, Lodge M, Motivated skepticism in the evaluation of political beliefs, *American Journal of Political Science*, 2006, 50, 755–69
- Teeguarden J, Calafat A, Ye X, Doerge D, Churchwell M, Gunawan R, Graham M, 24-hour human urine and serum profiles of bisphenol A during high dietary exposure, *Toxicol. Sci*, 2011, 123, 48–57
- Thaler R, Sunstein C, *Nudge: Improving decisions about health, wealth, and happiness*, NewHaven, CT, Yale University Press, 2008
- Thiel D, *Criminal Ignorance*. In: *Routledge International Handbook of Ignorance Studies*, (eds) Gross M, McGoev L, Routledge, 2015, Chap 27, 256-65
- Tietz T, Lenzner A, Kolbaum A, *et al*. Aggregated aluminium exposure: risk assessment for the general population, *Arch Toxicol*, 2019, 93, 3503–21.
- TLC: Truth in Labelling Campaign, MSG, MSG in Agriculture, Details from the Federal Register, https://truthinlabeling.org/crops_details.html accessed 27 Feb 2019

Tombs S, Whyte D, The corporate criminal: why corporations must be abolished, London, Routledge, 2015

Toms L, Sjödin A, Harden F, Hobson P, Jones R, Edenfield E, Mueller J, Serum polybrominated diphenyl ether (PBDE) levels are higher in children (2–5 years of age) than in infants and adults, Environmental health perspectives, 2009, 117, 9, 1461

Torka M, Mintzes B, Bhasale A, Fabbri A, Perry L, Lexchin J, Secret safety warnings on medicines: A case study of information access requests, Pharmacoepidemiology and drug safety, 6 Mar 2019

Toxnet: US National Library of Medicine, NLM Toxicology Data Network, CYANOCOBALAMIN CASRN: 68-19-9 <https://toxnet.nlm.nih.gov/cgi-bin/sis/search/a?dbs+hsdb:@term+@DOCNO+2850> accessed 11 Apr 2019

Trenberth K, Check with Climate Scientists for Views on Climate, Wall Street Journal, 1 Feb 2012 <http://online.wsj.com/article/SB10001424052970204740904577193270727472662.html>.

Tuormaa T, The adverse effects of food additives on health: a review of the literature with special emphasis on childhood hyperactivity, Orthomolecular Medicine, 1994, 9, 225–43

UCC, Bhopal Methyl Isocyanate Investigation Team Report, Union Carbide Corporation, Danbury, Connecticut, U.S.A., March 1985

USC: University of South Carolina, Even low-level radioactivity is damaging, scientists conclude, ScienceDaily, 13 Nov 2012 www.sciencedaily.com/releases/2012/11/121113134224.htm accessed 23 Dec 2019

USEPA, Revised Human Health Risk Assessment on Chlorpyrifos [Overviews and Factsheets], 2018 [updated 2018-04-26; cited 2017 June 29]. <https://www.epa.gov/ingredients-used-pesticide-products/revised-human-health-risk-assessment-chlorpyrifos>. Accessed 26 Jan 2019

USEPA: US Environmental Protection Agency, Memorandum 15 Oct 2014, Benefits of Neonicotinoid Seed Treatments to Soybean Production [benefits_of_neonicotinoid_seed_treatments_to_soybean_production_2.pdf \(epa.gov\)](https://www.epa.gov/sites/default/files/2014-10/benefits_of_neonicotinoid_seed_treatments_to_soybean_production_2.pdf) accessed 27 Dec 20

USFDA: Food and Drug Administration, Background Document for the Food Advisory Committee. Certified color additives in food and possible association with attention deficit hyperactivity disorder in children. March 30–31, 2011. <https://wayback.archive-it.org/org-1137/20170406211659/https://www.fda.gov/downloads/AdvisoryCommittees/CommitteesMeetingMaterials/FoodAdvisoryCommittee/UCM248549.pdf>. Accessed 2 Dec 2018

USFDA: Food and Drug Administration: Sunscreen drug products for over-the-counter human use: proposed rule. Fed Regist. 2019, 84, 38, 6204-75 <https://www.govinfo.gov/content/pkg/FR-2019-02-26/pdf/2019-03019.pdf>. Accessed 1 Apr 2019.

USRTK: US Right to Know, 9 Aug 2021 [Appeals court rejects Bayer bid to overturn Roundup trial loss; cites Monsanto "reckless disregard" for consumer safety - U.S. Right to Know \(usrtk.org\)](https://www.usrtk.org/appeals-court-rejects-bayer-bid-to-overturn-roundup-trial-loss-cites-monsanto-reckless-disregard-for-consumer-safety-u-s-right-to-know-usrtk-org) accessed 21 Aug 2021.

Valisure. Valisure Detects Benzene in Sunscreen. Accessed May 30, 2021. <https://www.valisure.com/blog/valisure-news/valisure-detects-benzene-in-sunscreen/> accessed 7 Oct 21.

Varadarajan S, A scientific enquiry into the methyl isocyanate leak in Bhopal, Council of Scientific and Industrial Research, New Delhi, 1985

Veierød M, Weiderpass E, Thörn M, Hansson J, Lund E, Armstrong B, Adami H, A prospective study of pigmentation, sun exposure, and risk of cutaneous malignant melanoma in women, *JNCI: Journal of the National Cancer Institute*, 2003, 95, 20, 1530–1538

Victoria J, Wang C, Jones M, Jaing C, McLoughlin K, Gardner S, Delwart E, Viral nucleic acids in live-attenuated vaccines: detection of minority variants and an adventitious virus, *J Virol*, 2010, 84, 12, 6033-40

vom Saal F, Cooke P, Buchanan D, Palanza P, Thayer K, Nagel S, Parmigiani S, Welshons W, A physiologically based approach to the study of bisphenol A and other estrogenic chemicals on the size of reproductive organs, daily sperm production, and behavior, *Toxicology and Industrial Health*, 1998, 14, 239–60

vom Saal F, Hughes C, An extensive new literature concerning low-dose effects of bisphenol A shows the need for a new risk assessment, *Environmental Health Perspectives*, 2005, 113, 926–33

vom Saal F, Prins G, Welshons W, Letter to editor, *Toxicological Sciences*, 2012, 125, 1, 318-20

Wang J, Ganley C, Safety threshold considerations for sunscreen systemic exposure: a simulation study, *Clin Pharmacol Ther*, 2019, 105, 1, 161-7

Wareham S, The nuclear industry: a history of misleading claims, Briefing paper 20, Energyscience, The EnergyScience Coalition <http://www.energyscience.org.au/BP20%20Misleading.pdf> accessed 23 Dec 2019

Wargo J, *Our children’s toxic legacy: How science and law fail to protect us from pesticides*, New Haven, CT, Yale University Press, 1996

Warren C, *Brush with death: A social history of lead poisoning*. Baltimore, MD, Johns Hopkins University Press, 2000

Watanabe F, Abe K, Fujita T, Goto M, Hiemori M, Nakano Y, Effects of microwave heating on the loss of vitamin B12 in foods, *Journal of agricultural and food chemistry*, 1998, 46, 1, 206-10

Watts T, Mitochondrial Solutions, The Mitochondrial Summit interview, hosted by J Davidson, Transcript 2019

Woodruff T, Zeise L, Axelrad D, Guyton K, et.al. Meeting Report: Moving upstream—evaluating adverse upstream end points for improved risk assessment and decision-making, *Env Health Persp*, 2008, 16, 11, 1568-75.

Weber K, Rao H, Thomas L, From streets to suites: how the anti-biotech movement affected german pharmaceutical firms, *American Sociological Review*, 2009, 74, 1, 106–27

Weick K, Sutcliffe K, *Managing the unexpected: Resilient performance in an age of uncertainty*, (2nd Edn). San Francisco, CA, John Wiley & Sons, 2007

Wheldon H, The BBC Monitor Interview. Broadcast by the BBC on 13th March 1960. In: Estrin M (ed.) *Orson Welles: Interviews*, Jackson, MS, University Press of Mississippi, 2002, 77–95

White L, Dressendorfer R, Exercise and multiple sclerosis, *Sports medicine*, 2004, 34, 15, 1077-100

Wiener R, Shen C, Findley P, Tan X, Sambamoorthi U, Dental Fluorosis over Time: A comparison of national health and nutrition examination survey data from 2001-2002 and 2011-2012, *J Dent Hyg*, 2018, 92, 1, 23-29

Wilholt T, On knowing what one does not know: ignorance and the aims of research. In: Science and the production of ignorance : when the quest for knowledge is thwarted, Eds: Kourany J, Carrier M, Cambridge, MA, MIT Press, 2019.

Williams G, Aardema M, Acquavella J, Berry S, Brusick D, Burns M, de Camargo J, Garabrant D, Greim H, Kier L, Kirkland D, A review of the carcinogenic potential of glyphosate by four independent expert panels and comparison to the IARC assessment, *Critical Reviews in Toxicology*, 2016, 46, (sup1), 3-20

Willis, P. (1977) *Learning to Labour*. Farnborough: Saxon House

WVE: Unpacking the Fragrance Industry: Policy Failures, the Trade Secret Myth and Public Health, An investigative report by Women's Voices for the Earth November 2015

<https://www.womensvoices.org/wp-content/uploads/2017/10/FragranceReport2017Update.pdf>
accessed 18 Feb 2019

Xu Y, Xiang S, Ye K, Zheng Y, Feng X, Zhu X, Chen J, Chen Y. Cobalamin (vitamin B12) induced a shift in microbial composition and metabolic activity in an in vitro colon simulation, *Frontiers in microbiology*, 2018, 9, 2780

Yang Q, Gain weight by "going diet?" Artificial sweeteners and the neurobiology of sugar cravings, *Neuroscience* 2010, *Yale J Biol Med*, 2010, 83, 2, 101-8

Yeung C, A systematic review of the efficacy and safety of fluoridation, *Evidence-based Dentistry*, 2008, 9, 2, 39-43

Yiamouyiannis J, Fluoridation and cancer, *Lancet*, 1977, 2, 8032, 296

Yuan J, Wang S, Guo X, Hu W, Acute disseminated encephalomyelitis following vaccination against hepatitis b in a child: a case report and literature review, *Case reports in neurological medicine*, 2016, 2401809 <https://doi.org/10.1155/2016/2401809>

Zengin N, Yüzbaşıoğlu D, Ünal F, Yılmaz S, Aksoy H, The evaluation of the genotoxicity of two food preservatives: Sodium benzoate and potassium benzoate, *Food Chem. Toxicol*, 2011, 49, 4, 763- 9

Zhang G, Ma Y, Spectroscopic studies on the interaction of sodium benzoate, a food preservative, with calf thymus DNA, *Food Chem*, 2013, 141, 1, 41-7

Zhu X, Xiang S, Feng X, Wang H, Tian S, Xu Y, Shi L, Yang L, Li M, Shen Y, Chen J, Impact of cyanocobalamin and methylcobalamin on inflammatory bowel disease and the intestinal microbiota composition, *Journal of agricultural and food chemistry*, 2018, 67, 3, 916-26

Zoraster R, Vulnerable populations: Hurricane Katrina as a case study, *Prehospital and disaster medicine*, 2010, 25, 1, 74-8

Chapter 5

Manipulating Knowledge

Contents

Chapter 5.....	450
Manipulating Knowledge	450
INTRODUCTION.....	451
CONFLICTS OF INTEREST (COI).....	453
The Funding Effect	453
Journal Articles & Peer Review	459
Corrections & Retractions.....	462
INSTITUTIONAL CONFLICTS OF INTEREST	464
National Academies of Sciences, Engineering and Medicine (NASEM).....	466
American Association for the Advancement of Science (AAAS).....	467
Universities	469
Regulatory Authorities.....	469
Political COI.....	474
Medical Student Capture	474
Capture of Doctors.....	474
Medical Association Capture	475
Conclusion on Institutional Conflicts of Interest.....	475
STRATEGIES.....	476
Doubt as a Practice and a Strategy	476
Blame as a Strategy.....	477
Power to Enable Continuation Regardless.....	478
Industry Shills	479
Astroturfing Techniques.....	481
Smear Campaigns.....	483
Journalists As Agents In The Social Construction Of Scientific Ignorance	486
Fact Checking Companies	490
The Aspect of Reproducibility	491
False Advertising in Research Studies.....	491
When Knowledge Manipulation is Highlighted	492
CONCLUSION.....	494

REFERENCES.....	496
APPENDIX 1.....	507
References	512

INTRODUCTION

Chapter 4 covered different types of ignorance, and the manipulation of knowledge is complementary to generating ignorance. Knowledge manipulation can range from incorrect information being presented, as in faulty scientific studies, through to the outright suppression of information by various forms of power in our society. In the case of environmental exposures it can lead to ignorance of or false beliefs about health effects. It is therefore pertinent to consider common ways that knowledge can be manipulated. This chapter discusses ways this occurs, starting with coverage of conflicts of interest (COI). It will examine the funding effect and the manipulation of journal articles, and specific instances of various institutional COI, even those affecting seemingly independent organisations.

The funding effect refers to how directly and indirectly industry-funded studies are usually more favourable to industry interests. This has a carry-on effect in regulatory authority decisions, which are normally based on that industry-funded research.

The manipulation of knowledge will also be shown in journal articles, pointing out that the cornerstone of peer review is not as rigorous as might be expected. Journals are also unwilling to issue retractions for articles they know are not correct, demonstrating how their objectivity can be compromised by economic, personal and social factors.

The term “independent” is used extensively in this chapter. At its core, true independence in science today is rare: it is where a scientist or organisation conducts scientific studies using their own funds, and is removed from any direct or indirect industry influence. It can, however, apply in a modified way where scientists are less obviously subject to the influence of special interests. As will be seen, industry is aware of the strength attributed to seeming independence in studies on their products, so there is much difficulty in identifying how free studies are from

outside influence. One may sometimes be forgiven for simply classifying studies that identify problems with industrial products as being the independent ones but this is also problematic.

Examples of COI will be demonstrated in regard to the major regulatory bodies in the United States: the Centers for Disease Control and Prevention (CDC), Food and Drug Administration (FDA), National Institutes of Health (NIH) and Environment Protection Agency (EPA). Finally, COI of politicians, medical students, doctors and medical associations will also be illustrated.

Strategies used to manipulate knowledge will also be covered. One strategy is fostering doubt about negative findings on industry products.

A second one is to implicitly blame the sufferer for their condition, used by the medical profession to avoid research into the environmental causes of health conditions. This is usually done by attributing symptoms to a psychological problem or an individual's genetic make-up despite examples of epigenetic effects being commonly found through the literature.

In many situations maintaining ignorance about activities that affect people's health is achieved by the strategy of using "power" (as in influence within a society) to enable the continuance of activities. This will be illustrated with the example of asbestos mining.

Another strategy by industry is to use shills. If the number of public commentators reflecting the desired industry view can be multiplied and they appear to be independent, the greater the chance of success. However scientists, politicians, journalists or public activists whose studies have produced findings contrary to the current orthodoxy or who have spoken with contrary views can be subjected to smear campaigns by industry-backed organisations.

When industry needs to oppose proposed government legislation or regulations to prevent damage to the environment, it is more effective if the opposition comes from groups of experts or citizens who do not appear to be associated with the companies in question. When these groups or coalitions do not exist (so that the industry can foster them), public relations (PR) firms will create them to appear as a grass-roots populist movement.

Further, many strategies employ the journalistic values of objectivity, balance, fairness and free speech, which makes journalism vulnerable to being used as an accomplice in the deliberate cultural production of ignorance. A recent trend in the media industry is for supposedly neutral

fact-checking companies to be utilised by the public, yet many of these companies may be industry-backed and far from neutral in their assessments.

In addition, the traditional cherry-picking of positive data and discarding of negative trials, as allowed by many regulatory approval processes, has commonly been used to classify products as safe and effective.

Finally, when the manipulation of knowledge by industry is highlighted by researchers and writers, there is sometimes a significant legal response from industry to debunk their claims and to discourage others. Some examples will be discussed.

CONFLICTS OF INTEREST (COI)

The Funding Effect

There are many references in the biomedical literature to a ‘funding effect’ which relates to the tendency for a study funded by industry, to usually conclude positively or desirably for that industry (Krimsky 2004, 2005, Smith 2005, Michaels 2006, Capps 2016). Reviews in prominent biomedical journals have found pro-industry conclusions are more common when there is pharmaceutical company sponsorship (Bekelman 2003, Lexchin 2003). In industry funded studies, it has been noticed that even “the conclusions in negative trials are often presented in such a way that they appear to be more positive than they actually are” (Lundh 2012p3). Industry funded studies, compared to independent studies, produced conclusions consistent with the commercial interests of the funding company with the effect particularly in data collection and results interpretation (Krimsky 2010, Lundh 2012).

According to David Michaels (2008p103): “the opinions of virtually any scientist can be clouded by conflict of interest, even if it isn’t apparent to the scientist herself. Conflict of interest inevitably shapes judgement.” Michaels (2008p98) came to the conclusion that “it appears that the pharmaceutical industry is devoting sizable resources to the conduct of studies whose results will increase sales, but will not necessarily provide the information physicians need to select the best drug for their patients.” This was summarised by an ex-editor of the British Medical Journal (BMJ), Dr Richard Smith. He attributed companies getting the results they want

not so much by adjusting the research results, but by asking the right questions. He gave these examples:

- “Conduct a trial of your drug against a treatment known to be inferior.”
- “Trial your drugs against too low a dose from a competitor drug.”
- “Conduct a trial of your drug against too high a dose of a competitor drug (making your drug seem less toxic).”
- “Conduct trials that are too small to show differences from competitor drugs.”
- “Use multiple endpoints in the trial and select for publication those that give favourable results.”
- “Do multicentre trials and select for publication those that are favourable”
- “Conduct subgroup analyses and select for publication those that are favourable.”
- “Present results that are most likely to impress, for example, reduction of the relative rather than the absolute risk” (Smith 2005p365).

These methods can also be adapted to toxic exposure effects. A good example already aforementioned was in the studies on the risk of lung cancer in the workers exposed to beryllium. Here, an elevated risk was found in three government funded studies and one industry funded study did not. A specific example relative to the consideration of a chemically sensitive portion of our population is presented in Appendix 1.

Some industrial sponsors have been found to publish the same study in various journals with different author names and no cross-referencing. The effect then is that there are a number of independent studies with the same findings (Rennie 1999). This makes use of the orthodox weight of evidence or evidence-based approach.

A significant example of the funding effect was raised by Michaels (2008), in the case of bisphenol A (BPA). This chemical is found in epoxy resins and numerous other commercial products. It is also in food-contacting plastics: packaging, kitchenware, and inside coating of cans and jar caps. It is in children and infant products and toys, dental materials, healthcare equipment and thermal paper. After it was found in various studies that very low levels of BPA could alter endocrine functions, the American Plastics Council sponsored the Harvard Centre for Risk Analysis to perform a ‘weight of evidence’ toxicology review. The review found no consistent evidence of BPA effects at low doses by reviewing 19 animal studies (Gray 2004). Yet

industry on other occasions on the assessment of EDCs argued that animal studies cannot be used (Witorsch 2002).

Reacting to such a conclusion, a group of scientists reviewed many published studies. They found that 90% (94 of 104) of studies funded by government found an effect and none of the 11 industry funded studies found an effect (Vom Saal 2005).

Industry funded studies are mostly relied on by our regulatory authorities. The industry has strong incentives to suppress adverse effects. A basic fundamental of scientific work is violated without research independence: that the research be disinterested (Merton 1973). The shortage of resources sees regulatory authorities unable to replicate the little research that is performed. As a result, industry usually has a monopoly on their product scientific information.

Regulators have ignored ethical and safety issues involved in industry's extensive use of Contract Research Organisations (CROs). "Foreign trials offer the freedom to adjust the protocols to achieve the preconceived answer one seeks" (Mirowski 2011p375). One interviewed CRO scientific officer answered:

In my recruitment strategy, I can use subject inclusion criteria that are so selective I can "engineer out" the possibility of adverse events being seen.

Or, I can demonstrate that my new drug is better by "engineering up" a side effect in another drug. That is the big game of clinical trials (Mirowski 2011p375).

About 80% of all clinical trials are performed by CROs in foreign countries, China being the most common (Mirowski 2011p232). In the past falsified test results were identified by US CRO's as will be discussed in the chapter seven.

Mirowski suggests, "The current modern regime of science organization in many respects is not a new knowledge economy as much as it is an engine of agnogenesis" (Mirowski 2011p318).

Unless studies are replicated, falsification of research cannot easily be detected by regulators. The authorities rely on the fraud penalties to deter industry from doing such. Yet falsification has occurred, for example, in the 1970s in testing for pesticide manufacturers (McGarity 2002) where testing laboratories used falsified test data (IBT Laboratories on Glyphosate). This will be discussed further in chapter seven. Furthermore, falsification of mandatory self-monitoring

measurements occurred, such as for the US Mine Health and Safety Act (1969) where the mine operator was required to send the results of air samples to a regulatory authority and faced citation and monetary penalties if they were above the permissible levels (Wagner 2004).

But there are other features of the pharmaceutical industry approach than deciding what data and conclusions are published in journal articles as that is simply one step in an extensive marketing approach. Below are some industry practices to obscure important information, influence clinical trial research, and influence medical literature (Ross 2012):

Seeding trials: Clinical trials of a drug or device on human subjects for promoting the drug or product with doctors under the guise of them being part of the trial, without disclosing the marketing objectives.

Publication planning: Organizing and planning medical journal articles for maximum commercial value. High-profile journals are targeted for high market impacts and lower profile journals for market-focused articles.

Key messaging: Establishment of key messages and themes to promote sales and the incorporation into the publications.

Ghostwriting: The write-up of a study by a company employee with university scientists adding their names to the article as its authors

Guest authorship: Designation of an author, such as an academic investigator not employed by industry, to confer external objectivity.

Selective publication: As mentioned above, the delayed publication or nonpublication of clinical trials that have findings that do not support a drug or device or that may decrease the commercial value of the product.

Selective reporting: The partial or incomplete reporting of clinical trial findings not supporting the product.

Ambiguous reporting: Reporting of findings that do not support a product in a way that is misleading or less likely to attract public attention.

Government-funded drug studies usually have data available for private party inspection and re-analysis. Industry has no obligation to publish their studies' raw data.

As sponsored studies have been generally regarded by the scientific community to be less credible than truly independent studies, the industry has developed ways to 'launder' its funding through non-profit organisations to create the illusion of independent results that support their interests. Examples include the tobacco industry-sponsored research through the Centre for Indoor Research (Barnes 1996), and the Society for Women's Health Research supporting industry research for hormone replacement therapy (Clark 2003). Another strategy by industry is to sponsor review studies, such as what the tobacco industry did on passive smoking effects (Barnes 1998). Also, expert panels (hand-picked) to supposedly summarise research on a topic such as:

- The Council for Tobacco Research with the front of providing independent research, yet internal documents reflected it was "for public relations...to convince the public that the hazards of smoking had not been definitively proven" (Glantz 1996p33, see also Kluger 2010).
- The Fen-Phen drug manufacturer convening an expert review panel to review their drug, yet most of the experts had some links with the company (Mundy 2010).

When research results are adverse to industry interests, the studies can be discontinued, cancelled, said to be flawed, incomplete, subject to further confirmatory research, etc. The suppression of potentially damaging information has "substantially harmed public health" (Wagner 2004p126). Examples cited by Wagner are:

- Johnson and Johnson: suppression of consumer complaints and other information (West 1985). The suppression of the knowledge of asbestos in their talcum powder and the links with ovarian cancer.
- A.H. Robins, the manufacturer of the Dalkon Shield:
 - Numerous studies discontinued or concealed, and the burning of hundreds of such documents (Tetuan v A.H. Robins, 738 P.2d 1210, 1240 (Kan. 1987)).
 - An internal memo that research funding was "to make available for publication extremely good Dalkon Shield results." As a result, a 2 year study on baboons showing problems was not released (Mintz 1985).

- Merrell Dow and its breast implants:
 - Concealment of adverse affects. One study on dogs found no problems at 6 months but after 2 years significant inflammation was found demonstrating immune system reactions. This study was withheld for several years, and when finally released, only the 6 month positive results were present (Hopkins v Dow Corning Corp., 33 F.3d 1116, 1127-8 (9th Cir. 1994)).
- Asbestos Industry:
 - The attempts of the asbestos manufacturers' concealment and downplay of hazards is well-documented, such as in Brodeur (1985).
 - Asbestosis animal studies in the 1930s: the results were decided to be investors property.
 - Corporate policies of not educating sick employees on the nature of their illness in case they would pursue compensation claims and lawsuits.
- Vinyl chloride and lead:
 - Decades of concealment and downplay of hazards is well-documented (Markowitz 2013)
- Tobacco:
 - The tobacco industry is the classic case which saw actions to generate uncertainty, doubt, and ignorance through advertising, misleading press releases, support for alternate research, establishment of research institutes, and funding of supportive research (Proctor 2008).

Regulation deficiencies are found through the literature as well as the lack of oversight on the regulators such as the US CDC. The CDC has received regular donations, Congress-approved, from various pharmaceutical companies. Controversial recommendations on screening tests and drugs have been noted to have been made by the CDC, as well as the CDC overseeing studies with 'conditional' industry funding (Lenzer 2015).

The funding effect has been noticed in many scientific fields, as listed by Krinsky (2017): pharmaceuticals (Stelfox 1998), chemical toxicology (Vom Saal 2005), tobacco (Bero 2005), surgery (Lopez 2015), mobile phones (Van Nierop 2010), nutrition (Bes-Rastrollo 2013), and genetically modified crops (Diels 2011, Guillemaud 2016). Occasionally the funding effects are

highlighted in the media, such as the side effects, particularly suicidal inducement in young people, from antidepressant drugs (Vedantam 2004). However, the media interest seems to depend on the advertising sources and the drama content.

As can be seen from the above brief coverage, the funding effect is well-recognised and must be considered as an influence contributing to the manipulation of knowledge.

Journal Articles & Peer Review

At the core of scientific knowledge is how it becomes published for others to see and becomes a basis for other knowledge and study generation as well as provide a direction for the doctors to take in diagnosis or treatment. This section will briefly consider how knowledge may be manipulated in journal articles and how the cornerstone of peer review is not as rigorous as the expectation is.

In considering this area, Rogers (2019) brings up pertinent references, many of which are drawn on in this section.

There has been much written about the quality and bias of studies as seen in the previous section, due to funding origins. In a joint statement in 2001 editors of various biomedical journals put forward that sponsor control of publications “erode[s] the fabric of intellectual inquiry that has fostered so much high quality clinical research” (Davidoff 2001p854).

In his prominent essay titled ‘Medical Journals are an Extension of the Marketing Arm of Pharmaceutical Companies’, Smith argues that peer review is “ineffective” and “prone to bias and abuse” (Smith 2005p365). Smith suggests that “journals should perhaps stop publishing [clinical] trials” altogether and instead “concentrate on critically describing them.” This is reflected in comments from editors of the most prominent journals on this situation:

The pharmaceutical companies, by their arrogant behaviour and their naked disregard for the well-being of the public, have lost our trust. The FDA, by spinelessly knuckling under to every whim of the drug companies, has thrown away its high reputation, and in so doing, forfeited our trust: Drummond Rennie (2007p1011), who for 30 years was

firstly deputy editor of The New England Journal of Medicine (NEJM) and, after, the Journal of the American Medical Association (JAMA).

The case against science is straightforward: much of the scientific literature, perhaps half, may simply be untrue. Afflicted by studies with small sample sizes, tiny effects, invalid exploratory analyses, and flagrant conflicts of interest, together with an obsession for pursuing fashionable trends of dubious importance, science has taken a turn towards darkness: Richard Horton (2015p1380) editor-in-chief of The Lancet.

It is simply no longer possible to believe much of the clinical research that is published, or to rely on the judgment of trusted physicians or authoritative medical guidelines. I take no pleasure in this conclusion, which I reached slowly and reluctantly over my two decades as an editor of The New England Journal of Medicine: Marcia Angell (2009para29).

The profession of medicine in every aspect — clinical, education, and research — has been inundated with profound influence from the pharmaceutical and medical device industries. This has occurred because physicians have allowed it to happen, and it is time to stop: DeAngelis and Fontanarosa (2008p1833) editor in chief and executive deputy editor respectively of JAMA.

With science and medicine having tremendous influence and power in our society it is amazing to see doubts expressed by such prominent people about the validity of so much scientific literature. Author COI contributes to this situation. In analysing 61134 articles in the 1396 highest ranked journals, (only about 200 had COI policies), only 327 (0.5%) had a reference to author financial interests in relation to the publication subject matter (Krimsky 2001). It was later concluded that “journals neither police nor evaluate author compliance with their guidelines” (Krimsky 2004p171). With drug companies spending US\$326 million on medical

journal advertising just in 2010 (Kornfield 2013p2), the journals clearly would experience much pressure to publish articles from their clients. It is estimated that “approximately 40% of journal reports of clinical trials of new drugs” are ghostwritten by the pharmaceutical companies, not the author listed on the published article (Sismondo 2010p277, 2018).

Further to the above, with the published scientific literature being fully peer-reviewed this illustrates gross shortcomings with the peer review system. Smith after his investigations on journal article peer review, concludes:

Peer review is impossible to define in operational terms (an operational definition is one whereby if 50 of us looked at the same process we could all agree most of the time whether or not it was peer review). Peer review is thus like poetry, love, or justice.... [W]e have little evidence on the effectiveness of peer review, but we have considerable evidence on its defects. In addition to being poor at detecting gross defects and almost useless for detecting fraud, it is slow, expensive, profligate of academic time, highly subjective, something of a lottery, prone to bias, and easily abused (Smith 2006p178).

The ‘Matthew Effect’ (Merton 1968) takes its name from the Biblical passage (New Revised Standard Version), Matthew 13:12: “For to those who have, more will be given, and they will have an abundance; but from those who have nothing, even what they have will be taken away.” This was illustrated in a study conducted by Peters and Ceci (1982) by selecting 12 papers published in prestigious journals, and changing the names and institutions listed for the authors, and “resubmitted [them] to the same journals that had originally refereed and published them” finding that “Sixteen of the 18 referees (89%) recommended against publication and the editors concurred” (Peters 1982p187).

One study reviewed three past experiments on the ability of reviewers to detect errors by inserting deliberate errors, and found the majority of the reviewers did not pick them up (Schroter 2008). The researchers then sent a test paper to 607 peer reviewers at the BMJ. The test papers had 9 major errors inserted but the reviewers only found an average of 2.58 errors. This process was repeated with a second and third paper after a short training course was performed with the reviewers, but the average number of errors found only marginally improved to 2.71 and 3.0 for the second and third papers respectively.

Two pertinent conclusions:

If peer review is good at anything, it appears to be keeping unpopular ideas from being published (Wilson 2016para19).

So peer review is a flawed process, full of easily identified defects with little evidence that it works. Nevertheless, it is likely to remain central to science and journals because there is no obvious alternative, and scientists and editors have a continuing belief in peer review. How odd that science should be rooted in belief (Smith 2006p182).

It has been illustrated in this section that knowledge may be manipulated in journal articles and the cornerstone of peer review is not as rigorous as might be expected.

Corrections & Retractions

Corrections and retractions are an important indicator of knowledge manipulation, whether the original studies were deliberately incorrect or simply indicate a lack of good science. Drug studies are used as examples, as these supposedly have the highest acceptance standards for industrial products released in our environment in the most-controlled way.

For experimental work, various cell lines are cultured for use in various experiments. Some of the cell lines have been used for decades, such as the HeLa cell line since the 1950s. However due to various circumstances, including poor laboratory practices, "... more than 400 cell lines either lack evidence of origin or have become cross-contaminated with human or other animal cells at some point in their laboratory lineage" (Oransky 2016para4). "Recent estimates suggest that between 20 percent and 36 percent of cell lines scientists use are contaminated or misidentified — passing off as human tissue cells that in fact come from pigs, rats, or mice, or in which the desired human cell is tainted with unknown others. But despite knowing about the issue for at least 35 years, the vast majority of journals have yet to put any kind of disclaimer on the thousands of studies affected" (Oransky 2016para2). This contamination means that thousands of studies based on these cell lines have invalid conclusions. For just two of these

cell lines (HeLa/Hep-2 and HeLa/INT407), from the approximate 400 affected, there have been 7136 articles published in over 1200 journals. These have been cited a combined 214,000 times (Oransky 2016para4).

An analysis was undertaken of 2047 articles that were retracted in the PubMed database as of May 3, 2012. Only six papers were retracted due to contamination of cell lines or inappropriate cell use (Casadevall 2014). Based on Oransky and Marcus's findings, there should have been tens of thousands of retracted papers.

A significant study published almost 2 decades ago found that use of lab test mice rather than wild mice would "...likely overestimate cancer risks and underestimate tissue damage and consequent accelerated senescence" (Weinstein 2002p615). The lab mice, the majority of which came from a single source, were found to have greatly elongated telomeres. Telomeres are found at the ends of the chromosomes and play an important role in ensuring DNA gets copied properly when cells divide. As their telomeric failsafe has been disabled, the animals become unreliable for normal senescence and tumour formation. This effectively invalidated tens of thousands of studies based on these lab mice and should have seen all those studies retracted, however all journals have simply decided to ignore this finding.

These situations highlight that journals are unwilling to issue retractions for articles they know are not correct, and demonstrate that objectivity is outweighed by economic, personal and social factors.

Of the drug studies that have been retracted, an astounding 72% have been due to fabrication, unethical conduct, data falsification, plagiarism and the like (Samp 2012). A recent example was the study of the effectiveness of the drug hydroxychloroquine for treatment of COVID (Mehra 2020). The Lancet and later NEJM published a study that found it was not effective. However after an investigative Guardian article had shown serious errors in the data and raised questions about the data collection company and its CEO, a subsequent investigation saw the Lancet withdraw the article.

It is expected that doctors keep abreast of latest studies. This situation must cause confusion as their advice may be seriously flawed in reading such studies.

INSTITUTIONAL CONFLICTS OF INTEREST

Although there is much written about the funding effects on individual researchers there is an increasing awareness of the effects of bias generated from institutional conflicts of interest (COI). This can occur when an “institution’s own financial interests or those of its senior officials pose risks of undue influence on decisions involving the institution’s primary interests” (Inst Med 2009p218).

In this section, firstly considered will be both sides of the COI argument: whether advantageous or not. The remainder of the section will then bring up various COI of two seemingly independent organisations as examples that seeming independent organisations can suffer from COI. This will be followed by examples of COI from the major regulatory bodies in the US: the CDC, FDA, NIH and EPA. Finally a generalised discussion will be presented on COI of politicians, medical students, doctors and medical associations.

In studying the arguments put forward to accept seeming COI, Davis (2013) identified the following points, in summarised form:

- Academia, corporations and the government all want access to top researchers and scientists so are simply recruiting from the same, finite talent pool.
- Experts on advisory panels will not change their voting patterns if there is COI.
- COI rules would delay access to life saving treatments.
- COI rules would increase the cost of developing treatments.
- Government departments would benefit from the collaboration with conflicted experts.

Arguments in favour of strong COI rules:

- There is much evidence that COI affects voting patterns and therefore can affect the public good.
- The rules would prevent experts from one company influencing a decision that could affect competing companies.
- Industry costings on drug development costs are overestimated. Typically pharmaceutical companies spend 19 times more on marketing than scientific research.

- There is no evidence that a shared expert pool dilemma exists. Thirty to 50% of academic researchers do not have COI.
- Rushing products to approval without adequate evaluations can come at large societal costs.

On a personal level, there are studies that point to a general human trait that sees people generally unaware of bias and so make no effort to correct for it or avoid COI initially (Dana 2003). Furthermore, there are studies pointing to the ineffectiveness of the disclosure of COI (Camerer 1989, Wilson 1994, Cain 2005). To the extent that Elliot (2004p22) comments:

It is time to admit that as a remedy for conflict of interest, disclosure has been an utter failure. Disclosure is an empty ritual designed to ease the consciences of academics unable to wean themselves from the industry payroll. Its only purpose is to serve as a warning signal, like a fire alarm in a burning building. Disclosure does nothing to fix the underlying problem of pharma funding, which is not secrecy but power. It does patients no good to be told that doctors, researchers, and regulators are all in pharma's pocket if there is nothing they can do about it.

Rogers (2019p176) summarises:

Disclosure as a remedy for bias persists precisely because it is ineffective. If disclosure reduced bias then pharmaceutical companies would not be getting a return on their \$24 billion a year investment in marketing to physicians and there would be no point in making such expenditures in the first place.

In the financial and judicial systems COI is not acceptable: companies are expected to be audited for investor confidence, and a judge, lawyer or juror in the court system simply cannot have COI. In Australia, to maintain the financial system we have a regulatory system policed by the Australian Securities and Exchange Commission (ASIC). The contrast can be seen with our health effect assessment system if one were to substitute ASIC's main functions which are based on a fully audited situation, with that shown in brackets:

“The ASIC Act requires us to:

- Maintain, facilitate and improve the performance of the financial [regulatory] system and entities in it.
- Promote confident and informed participation by investors [the public] and consumers in the financial [regulatory] system.
- Administer the law effectively and with minimal procedural requirements
- Enforce and give effect to the law.
- Receive, process and store, efficiently and quickly, information that is given to us.
- Make information [safety studies] about companies and other bodies [their products] available to the public as soon as practicable.
- Take whatever action we can, and which is necessary, to enforce and give effect to the law” (ASIC 2019).

National Academies of Sciences, Engineering and Medicine (NASEM)

This is the first of two examples of where a seemingly independent organisation clearly has COI.

The US National Academies of Sciences, Engineering and Medicine (NASEM) has been a well-respected organisation over the years and has various guidelines and protocols for members of its committees in respect to COI. The NASEM originally did not have any policies on institutional COI despite being a private institution able to receive industry donations from sources that may have a financial interest in its work. In recent years one of its committees, with 20 members, authored a comprehensive report on GMO crops (NASEM 2016) with few concerns about adverse effects. With some of its past reports in the area of agricultural biotechnology drawing alleged COI (Peterson 2016, Wadman 1999, Strom 2016), Krinsky & Schwab (2017) decided to examine the financial ties of the committee members.

While the report declared no conflicts of interest, the examination “found that six out of twenty committee members had financial COI. Five individuals received research funding from for-profit companies related to the subject matter of the report and five had patents or patent applications on the subject matter of GE crops. Four panel members had two financial COI. In total there were ten financial COI among the six committee members, and it would appear that most of these conflicts meet the NASEM’s own standards for financial COI.” Krinsky & Schwab

found these from open literature and excluded possible COI through research grants or personal investments. All the COI found were from industries that could financially benefit from agricultural biotechnology: there were none with a COI in limiting such technology.

To exacerbate the situation with the report, those identified as having financial COI, made up all of the committee's expertise in several areas (Gould 2016). As is common in such committees, chapters are written by members who have the expertise on the chapter subject, so possibly complete chapters were written by such members.

Institutional COI was present, yet not mentioned until months later. Monsanto, Dupont and Dow had each recently donated up to \$5million each to the organisation (NAS 2014, 2015, NARA 1999). The NASEM Board of Agriculture, which oversaw the report, included a Monsanto representative as well as a number of feed and food companies such as Nestle, Cargill, Novus and Purina (NASEM 2016). Many guidelines of boards of colleges and universities require board members having COI to exclude themselves from involvement (AGBUC 2007).

[American Association for the Advancement of Science \(AAAS\)](#)

This is the second of two examples of where a seemingly independent organisation clearly has COI.

The AAAS is one of the largest scientific societies and publishes journals such as 'Science'. Since 1980 it has annually awarded Scientific Freedom and Responsibility recognition to "scientists, engineers or their organizations, whose exemplary actions have demonstrated scientific freedom and responsibility in challenging circumstances." This is outlined on the AAAS website (AAAS 2019a):

The types of actions worthy of this award include acting to protect the public's health, safety or welfare; focusing public attention on important potential impacts of science and technology on society by their responsible participation in public policy debates; or providing an exemplary model in carrying out the social responsibilities of scientists, engineers or in defending the professional freedom of scientists and engineers.

Some awardees have risked their freedom and even physical safety by their actions, while others have been honoured for their advocacy and their leadership.

On the 4 Feb 2019, the winners of this award were announced by the AAAS (AAAS 2019b) to be Dr Sarath Gunatilake and Dr Channa Jayasumana. These human health researchers published papers showing links between glyphosate exposure and chronic kidney disease of unknown etiology (CKDu) in Sri Lankan farmers. Three papers were published with the third finding that people who drank water from wells having higher glyphosate and heavy metal concentrations had a fivefold increased risk of CKDu (Jayasumana 2014, 2015a,b). However, on the 6 Feb 2019 the announcement was removed from the AAAS website.

The authors were well known to the herbicide manufacturers as they had a recent history of being critical of glyphosate-based herbicides, such as when Gunatilake said that adjuvants added to glyphosate-based herbicides “are 1,000 times more toxic than glyphosate itself” in an interview (Kumarasinghe 2018). He continued, “Over the last 25 years, the pesticide industry had us hoodwinked by referring only to glyphosate and not to the adjuvants or additives included in these herbicides.” Jayasumana, had testified at the year-long International Monsanto Tribunal asserting that glyphosate use had resulted in ecocide (IMT 2017).

Jessica Wyndham, director of the AAAS Scientific Responsibility, Human Rights and Law Program, had said (AAAS 2019b):

To right a wrong when significant financial interests are at stake and the power imbalance between industry and individual is at play takes the unique combination of scientific rigor, professional persistence and acceptance of personal risk demonstrated by the two scientists recognized by this year’s award.

On the same day the award announcement was deleted the AAAS CEO announced his retirement (AAAS 2019c).

A frequent pro-industry product spokesperson, Prof. Folta criticised the pair’s 2014 paper the day after the award announcement, as merely presenting “a hypothesis. There were no data. There were no experiments. It was a semi-well-crafted hypothesis that could be tested” (Folta 2019). However an inspection of this and the subsequent papers finds data from epidemiological and case-control studies, and geographical surveys. The authors clearly point

to an association (not causation) between glyphosate exposure and chronic kidney disease which technically remains a hypothesis until long term controlled studies are undertaken, although the funding for such a study would be difficult.

Prof Jack Heinemann appropriately commented (2019):

Whether or not the link between glyphosate (or formulation) and kidney disease is right misses the point. A scientific freedom award is given for persecution. If you only give it for proven science, it would be delayed decades and it would only benefit those who persecute.

Universities

Prospects for appointment, tenure and promotion can be improved by receiving industry research grants, guest authorship and consulting opportunities. Success in these areas affects standing and respect from colleagues. In such a situation where industry funding is key, those who do not participate can be left behind.

Some of the major sources of financial COI for contemporary academic and government researchers include consulting fees with private companies, grants or contracts to fund university research projects, honorariums, gifts, equity holdings, managements positions with start-up companies, and revenue streams from intellectual property (Elliott 2008p4).

Regulatory Authorities

Rogers (2019) has recently collected numerous cases where the main US regulatory authorities have been shown to have conflicts of interest (COI). Some of his examples are as below combined with others. As there are many COI indications on these government departments, only some COI instances are listed below in point form for brevity:

US Centers for Disease Control and Prevention (CDC)

- One government report found 97% of disclosure forms submitted by advisory committee members were incomplete. 13% of the members had not filed any such form (Levinson 2009).
- The CDC has a non-profit foundation allowing private donations, typically from the large pharmaceutical companies. These donations can “endow positions at the CDC, and even place individuals to work at the CDC, paid through ‘private funding’” (ICAN 2017p27). In 2016 over US\$42million was accepted (CDC Foundation 2016). Since its beginning (1995), the CDC Foundation has received \$161 million from private corporations (KEILC 2019). The CDC in accepting funding becomes a conflicted organisation with doubts cast on its scientific integrity.
- The CDC foundation accepted a directed donation from Roche for the CDC’s past Take 3 flu campaign (Lenzer 2015). Its Step 3 told the public to “take antiviral medicine if your doctor prescribes it”. The CDC’s substantiation for recommending antiviral drugs was observational and industry-funded studies, and a meta-analysis (Dobson 2015) which it described as an “independent” study. However, “the study was sponsored by Roche, and all four authors had financial ties to Roche, Genentech, or Gilead: the first two sell oseltamivir and Gilead holds the patent” (Lenzer 2015p2). Oseltamivir (Tamiflu) is Roche’s antiviral drug.
- Twenty thousand young sugar cane field workers died in Central America in an “epidemic of chronic kidney disease” (Ramirez-Rubio 2013p123). Agrochemicals and shocking working conditions had been suspected (Ordunez 2018). Yet after the sugarcane industry donated \$1.7million to the CDC, it decided to conduct a longitudinal study of no determined period, “to examine genetics and biomarkers in children that might predispose them to later kidney failure” (Lenzer, 2015p351). “Researching long-shot genetic explanations over and against investigating environmental causes that might threaten corporate interests has become standard practice at the CDC” (Rogers 2019p150).
- In 2018, it was revealed the head of the CDC, Dr. Fitzgerald, had purchased tobacco company shares about one month from taking office. This purchase was part of other investments, including drug companies (Ehley 2018).

- The CDC director of its Division for Heart Disease and Stroke Prevention was found to have repeatedly organised meetings with the WHO for Coca Cola representatives to attempt to relax their 2015 sugar guidelines that targeted sugary beverages, that labelled them as a major cause of childhood obesity (Howard 2019).
- CDC Directors have a history of crossing into significant roles and ties with the pharmaceutical companies. Dr. Julie Gerberding, head of the CDC until 2009, left and a short while later joined Merck as president of the vaccine division (England 2018). CDC director Brenda Fitzgerald resigned after it was revealed she purchased shares in tobacco, drug and food companies while the director of the CDC (Cancryn 2018).
- There has been much controversy on the vaccine side of the CDC's responsibilities. As one of the central missions of the CDC is to promote vaccines there appears to be some COI in CDC reviews done on vaccines, such as on their mercury content and its health implications (Dórea 2018, Hooker 2014p7).

US Food & Drug Administration (FDA)

- In studying a two-year period, the FDA adopted advisory committee recommendations in all instances except one where the committee recommended against approving a flu drug, yet it was approved (Cauchon 2000).
- In the same two-year period, the FDA granted 803 COI waivers, as seems to be routine (Cauchon 2000).
- One study found that “more than half of the experts hired to advise the [U.S.] government on the safety and effectiveness of medicine have financial relationships with the pharmaceutical companies that will be helped or hurt by their decisions” (Cauchon 2000p1).
- Consumer and patient representatives on advisory committees “often receive drug company money” (Cauchon 2000para6).
- The voting outcomes of 1,379 FDA advisory committee members over a 14-year period showed that those “who served on advisory boards solely for the sponsor were significantly more likely to vote in favour of the sponsor” (Pham-Kanter 2014p447).

- A survey in 2002 found, “one-third of staff members surveyed did not feel comfortable expressing contrary scientific opinions”; “a third felt negative actions against applications were ‘stigmatized’ within the agency”; and various drug reviewers said, “that decisions should be based more on science and less on corporate wishes” (Adams 2002).
- In another survey, 66% of reviewers in the FDA’s Center for Drug Evaluation and Research “lacked confidence in the FDA’s safety monitoring of marketed prescription drugs” and “18% had felt pressure to approve a drug despite reservations about its quality, efficacy, or safety” (HHS 2003).
- The Union of Concerned Scientists conducted a survey of almost 6000 FDA scientists in 2006 and found aspects such as:
 - 18.4% said they “have been asked, for non-scientific reasons, to inappropriately exclude or alter technical information or their conclusions in a FDA scientific document.”
 - 61% knew of cases where “Department of Health and Human Services or FDA political appointees have inappropriately injected themselves into FDA determinations or actions.”
 - 70% feel there are insufficient resources for the FDA mission of “protecting public health and helping to get accurate science-based information they need to use medicines and foods to improve their health” (UCS 2006).
- Many past FDA Commissioners (9 of last 10) have taken jobs with pharmaceutical companies. A recent FDA Commissioner, Scott Gottlieb, was no exception. While he was in office he instigated policies for increased speeds of drug approvals and encouraged greater use of biosimilars, generic copies of more expensive biologic drugs used to treat autoimmune diseases and some cancers (Foley 2019, Anderson 2020). Pfizer, the company in which Gottlieb became a director of (Gottlieb 2019), is a manufacturer of these drugs.

US National Institutes of Health (NIH)

- Many “top scientists at NIH received substantial honoraria and stock options from biomedical firms that, in some cases, doubled their government salaries” (Krimsky

2004pxi). It was found that: “NIH officials now allow more than 94% of the agency’s top-paid employees to keep their consulting income confidential...A survey by the [Los Angeles] Times of 34 other federal agencies found that all had higher percentages of eligible employees filing reports on outside income.... The trend toward secrecy among NIH scientists goes beyond failure to report outside income. Many of them routinely sign confidentiality agreements with their corporate employers, putting their outside work under tight wraps” (Willman 2003). Congressional hearings encouraged the Department of Health and Human Services to foster disclosure, but allowed such situations for senior officials (Krimsky 2004).

- Like the CDC, the NIH also has a ‘Foundation’ which accepts corporate donations.

US Environmental Protection Agency (EPA)

- As appropriately summarised by Rogers (2019p163):
To briefly sketch the problem: ‘There are more than 80,000 chemicals available for commercial use; the EPA over the past 30 years has assessed the health risks of only 570; these scientific assessments are necessary before any new regulation can be enacted’ (Heath 2015). ‘700 new chemicals hit the market each year’; ‘a typical review takes six to eight years’; and ‘it took 27 years for the agency to issue a partial assessment of dioxin’ (Heath 2013). When EPA Administrator Lisa Jackson took office in 2009 she pledged to complete 50 assessments a year. The EPA has never come close to that goal. It completed 14 toxic risk assessments in 2009, 14 in 2010, six in 2011, and just three in 2012 (Heath 2013). In 2014, the EPA completed just one toxic risk assessment (Heath, 2015). In 2015, ‘the EPA missed its own deadlines for completing risk assessments for atrazine, glyphosate, and imidacloprid’, three widely used toxic agrichemicals (Burd 2015). Compare that with the FDA’s near perfect record of completing drug reviews on time over that same period and one starts to get a more complete picture of corporate influence on the regulatory process. To put it simply: pharmaceutical companies want fast approvals and chemical companies want slow or no regulations and both industries get exactly what they want under the current system.

Political COI

The corporate donation situation to political parties is well known. The lobbying of politicians is also significant with 1440 pharmaceutical lobbyists employed in the US in 2018 (CRP 2018). The corporate effect on the courts in the US is also significant in tort cases (Mirowski 2011, Cranor 2016). It is estimated that the pharmaceutical industry contributes 70% of advertising revenue of traditional media in non-election years (Kennedy 2014). This assists to carry over the desired political paradigms.

Medical Student Capture

Aside from courses extensively incorporating pharmaceutical drug treatments and little coverage of nutritional effects on the body for example, the pharmaceutical industry sponsors lunches, courses, issues textbooks and journal reprints. One survey found 90% of students had received educational style materials from the industry (Austad 2011).

Capture of Doctors

The pharmaceutical industry in the US put at least \$27 billion into drug promotion in 2012: 89% of this was spent on marketing to doctors and 11% on consumer advertising (Pew 2013). A study found that 94% of doctors have financial relationships with pharmaceutical and medical device industries (Campbell 2007).

Two comments from studies that summarise the situation well:

Why is it so hard to bring about change? Partly because change is in nobody's financial interest. In the case of medical education, the funding operation is seamless. Not only does pharma fund the medical education and communications companies who organize the continuing medical education (CME), the academics who deliver the CME, and the offices that certify the CME, it also funds the professional societies that require the CME. Specialty groups like the American Psychiatric Association and the American Academy of Family Physicians are heavily dependent on industry funds. Pharma even

helps write the accreditation guidelines. Nearly half of the membership of the task force which produced the original ACCME [Accreditation Council for Continuing Medical Education] standards governing industry support of CME came from industry itself (Elliott 2004p21).

...doctors are rarely outside of an ontological and epistemological reality created by pharmaceutical companies to serve their profit interests (Rogers 2019p172).

Medical Association Capture

Coca Cola revealed that in the period of 2010-2015 it gave \$21.8 million to research and \$96.8 million to health and well-being partnerships such as large donations to the American College of Cardiology, the American Academy of Family Physicians, the American Academy of Pediatrics (O'Connor 2015). Most of the professional medical associations receive the majority of their income from industry advertising (in their publications, etc.) and corporate sponsorship (Lexchin 2006).

Guidelines for patient diagnosis and treatment distributed by these associations are not impartially written. One investigation found that 35% of clinical guideline authors had “declared financial links to relevant drugs companies, with around 70% of panels being affected” and that “this is likely an underestimate as it relies on authors’ own declarations” (Taylor 2005p1070).

Conclusion on Institutional Conflicts of Interest

From the above material on institutional conflicts of interest, the health care system – at least in the US-- clearly has endemic conflicts of interest. Yet the corruption is rarely discussed and investigated, let alone attentioned. It remains an “open secret” (García 2019). While addressed, in the previous chapter, various ways ignorance has been employed in the application of science in the health industry, it has perhaps only been simply covering a symptom rather than a root cause. Corruption needs to be treated as an important research area in the same way research is conducted into the causes of disease.

Corruption is an open secret known around the world that is systemic and spreading. Over two-thirds of countries are considered endemically corrupt according to Transparency International ... Corruption in the health sector is more dangerous than in any other sector because it is literally deadly ... Corruption is an ignored pandemic." (García 2019p2119).

In her paper, García (2019) reflects on the history of health care corruption and comes to one common sense rule that the less transparent a health care system is, the more corrupt it becomes. From this viewpoint, the Australian health system becomes highly suspect due to its inherent lack of transparency, although this could be extrapolated from its many examples of mirroring the US system's decisions.

García also referred to an equation for corruption for any given system:

Monopoly (M) on the supply of a product or service

The level of discretion (D) enjoyed by suppliers

The supplier's accountability (A) to others

The amount of corruption (C) can be expressed conceptually as: $C = M + D - A$.

Since in the regulatory system, the pharmaceutical and chemical industries effectively act as one in lobbying government departments, 'M' is effectively high. As we have seen, the extensive use of ignorance in affecting scientific conclusions and risk assessments provides the industry with a high level of 'D'. Since the accountability of the industry is to its shareholders, not public health, 'A' effectively becomes zero in respect to public health. Net result is a high level of corruption.

Institutional conflict of interest becomes a key cause of the present situation with environmental effects on health.

STRATEGIES

Doubt as a Practice and a Strategy

The coverage of doubt in chapter four does not need to be repeated, but is mentioned here for completeness. The strategy of doubt was pioneered by the tobacco industry and subsequently employed by other industries on the risks of their products. Scientific activity however involves epistemological scepticism (Ogien 2015). Research results must always be subject to subsequent verification. Explanation is always based on the current state of science for result interpretation. Scientists must also handle issues of error, plagiarism, fraud, and conflict of interests. Doubt can therefore be a practical consequence of science.

New research with findings not consistent with current paradigmatic views, such as climate change, can falter in its effect if the researchers involved do not acknowledge an element of doubt: charges of dogmatism and censorship that the paradigmatic opponents allege may turn into rumour or not adequately rebutted. An effective method is to endorse the supposedly discrediting information as valid doubt due to science never producing 100% accurate results, implying those that ask for certainty are not familiar with science.

Blame as a Strategy

It was highlighted in chapter 3, on MCS, how the medical profession would avoid research into environmental causes of the health condition by implicitly blaming the sufferer for their condition. This was usually done by attributing to a psychological problem or one's genetic make-up.

Many doctors encourage patients to blame themselves for obesity and diabetes. These conditions have significantly increased over the last few decades. They are commonly attributed to too many calories, a lack of exercise, as well as genetic predispositions. However, there is an increasing number of studies attributing the initiators of these diseases to environmental exposures, such as:

Environmental chemicals with hormone-like activity can disrupt programming of endocrine signalling pathways during development and result in adverse effects, some of which may not be apparent until much later in life. Recent reports link exposure to environmental endocrine disrupting chemicals during development with adverse health consequences, including obesity and diabetes (Newbold 2010p206).

In their review of the science linking human environmental exposures with obesity and diabetes, Porta and Lee (2012) found many positive studies which indicated weight gain in laboratory animals. Although there is much undone science in identifying chemicals that can increase obesity risk, chemicals such as POPs, PCBs, most pesticides, flame retardants, BPA, phthalates, lead, nicotine, diesel exhaust and prescription drugs have been linked to this risk. The mechanistic and animal studies illustrating this are increasing in number and consistency.

Porta and Lee also reported that in respect to type 2 diabetes:

Evidence suggesting a relationship between human contamination with environmental chemicals and the risk of Type 2 diabetes has existed for over 15 years, with the volume and strength of the evidence becoming particularly persuasive since 2006. Chemicals linked to Type 2 diabetes in human studies are POPs (including dioxins), PCBs, and some organochlorine pesticides and brominated flame retardants), arsenic, BPA, organophosphate and carbamate pesticides, and certain phthalates (Porta 2012p7)

This is further magnified, as mentioned in other parts of this thesis, by epigenetics to subsequent generations. The more the parent/s are overweight, the higher the risk of obesity in the next generation (Holliday 2006, ABC 2013).

Power to Enable Continuation Regardless

Some of those who initially speak out in environmental crises are vilified and experience severe economic and psychological harm. This then acts to instil fear in others that could speak out. In the case of asbestos, the mining and manufacturing companies knew their workers were dying due to asbestos effects on their lungs. The company doctors, insurers, and community leaders knew this but did nothing about it (Schneider 2004).

...nearly 80 percent of world asbestos production in the twentieth century was produced after the world learned that asbestos could cause mesothelioma! (McCulloch 2008p14).

Production continuation despite this knowledge is due to power:

...asbestos has proved to be so enduring, because the industry was able to mount a successful defence strategy for the mineral — that still operates in some parts of the world (McCulloch 2008p15).

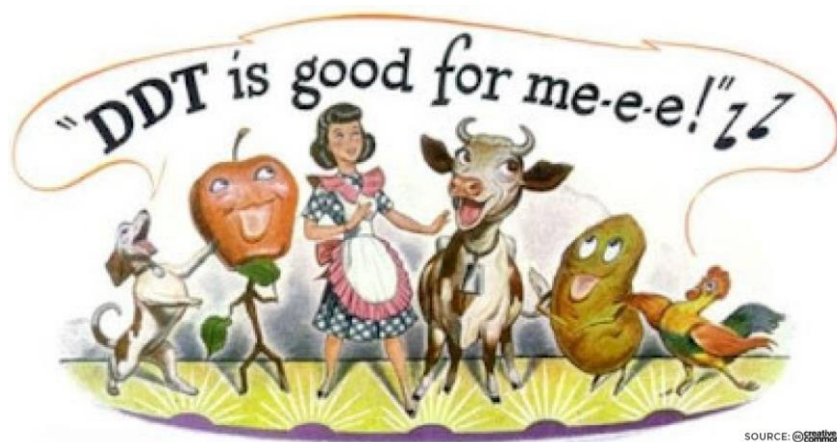
The strategy was of

...concealment and, at times, misinformation that often amounted to a conspiracy to continue selling asbestos fibre irrespective of the health risks (McCulloch 2008p15).

McCulloch and Tweedale's (2008) analysis suggests that in some ways we live under a sort of modern feudalism, where a handful of powerful actors can engage in long term toxic trespass, as long as they have enough resources to hire skilled product defence attorneys and a good public relations team (Rogers 2019p100).

Industry Skills

The coverage of industry front groups and shills by Gary Ruskin's book (2015) shows good examples of standard public relations tactics of the tobacco, chemicals, and fossil fuels industries to further their goals for legislation, regulation and public relations. In general, if the number of speakers in the public scene can be multiplied reflecting the desired industry view and appear to be independent, then the more chance of success there is. If their conflicts of interest can be hidden, then such speakers may command more credibility. The media can, once again, be complicit in the process. One of the old DDT advertisements that seeks to push aside the undone science and impose ignorance on the public, endorsed by shills:



(Ruskin 2015p29)

An example of an agrichemical industry shill is Henry Miller. The coverage of this man's activities, with references, is done well by Ruskin (2015p32) as summarised here:

- “He is the ‘the Robert Wesson Fellow in Scientific Philosophy and Public Policy at the Hoover Institution.’ He was the founding director of the FDA’s Office of Biotechnology. He has written numerous articles and op-eds in the Wall Street Journal, New York Times, Forbes and other news outlets in support of genetically engineered food, and against its labelling. He was even featured in TV advertisements against Proposition 37, a ballot initiative for labelling of genetically engineered food in the State of California.”
- Although now fired from Forbes, his former bio on the Forbes website included: “I debunk junk science and flawed public policy.” However through much of his life it seemed that he actually presented “an agile defense of junk science and flawed public policy.”
- He was regarded as “a key supporter” of the tobacco industry. He would for example write that “nicotine ... is not particularly bad for you in the amounts delivered by cigarettes or smokeless products.”
- He “is a member of the ‘scientific advisory board’ of the George C. Marshall Institute, which is famous for its oil and gas industry funded denials of climate change” and claimed that “the reality is that honeybee populations are not declining” in his defence of neonicotinoid pesticides.
- He “has repeatedly argued for the re-introduction of DDT, a toxic pesticide banned in the United States since 1972, which has been linked to pre-term birth and fertility impairment in women.”
- “In 2011, after the Japanese tsunami and radiation leaks at the Fukushima nuclear power plants, Miller argued in Forbes that ‘those ... who were exposed to low levels of radiation could have actually benefitted from it.’ At the same time, he wrote an article titled ‘Can radiation be good for you?’”
- “In an article in Forbes, Miller defended the use of the endocrine disruptor bisphenol A (BPA), which is banned in Europe and Canada for use in baby bottles.”
- “Miller was a trustee of the infamous industry front group American Council for Science and Health, according to the ACSH website.”

As can be seen from just one example, an industry shill may 'shoot off the hip' in their statements to the public and work by the principle that 'if one throws enough mud, some will stick'. This, combined with the public ignorance on such subjects, can produce results for the industry.

Astroturfing Techniques

Corporations frequently need to oppose proposed government legislation or regulations to prevent damage to the environment, so that the company is successful in its work, projects or products. They may oppose regulations in their company name but it is more effective if the opposition comes from groups of experts or citizens, or coalitions of such, who seem to be not associated with the companies. When these groups or coalitions do not exist so that the company can foster them, there are public relations (PR) firms that will create them. By linking company goals with what would then appear as a grassroots populist movement influencing governments these corporations "can profit handsomely" (Poole 1992).

For example, as far back as 1991, Dow Chemical contributed to 10 front groups including (Megalli 1992):

- Alliance to Keep Americans Working,
- the Alliance for Responsible CFC Policy,
- the American Council on Science and Health,
- Citizens for a Sound Economy,
- the Council for Solid Waste Solutions.

In the last five decades, the rise of public interest and environmental groups highlighting actions by large corporations against public interests has led to scepticism about claims made by business entities. So if Coca Cola said that sugary drinks are nutritious and good for you, then the more-informed consumers would tend to be sceptical. If however the American Council on Science and Health with its hundreds of 'expert' scientists said that there was nothing wrong with sugary drinks then many consumers would be more inclined to listen.

By sounding scientific the American Council on Science and Health can manipulate the public trust. This organisation has received “funds from food processing and beverage corporations including Burger King, Coca-Cola, PepsiCo, NutraSweet and Nestlé USA, as well as chemical, oil and pharmaceutical companies such as Monsanto, Dow USA, Exxon, Union Carbide and others. Its executive director, portrayed in the mass media as an independent scientist, defends petrochemical companies, the nutritional values of fast foods, and the safety of saccharin, pesticides and growth hormones for dairy cows. She claims that the U.S. government spends far too much on investigating unproven health risks such as dioxin and pesticides because of the public’s ‘unfounded fears of man-made chemicals and their perception of these chemicals as carcinogens’” (Beder 1998p2; see also Bleifuss 1995).

The public relations firms can also make hundreds of phone calls and/or mailed letters to politicians to create an impression of widespread support for their client’s case. This is enabled through today’s information technology and phone banks. Use of public relations firms has become standard procedure for large corporations (Faucheux 1995, Beder 1998). The incentives can be clear, with the Consumer Reports magazine (1994) reporting that they can receive up to \$500 “for every citizen they mobilize for a corporate client’s cause.” (Beder 1998p23).

Public database companies, especially in these days of Google and Facebook, can provide lists of people most likely to be persuaded to come on board or endorse the desired view. Even in a three day phone-up program feedback can be built-on as to what approach to the targeted public is working best to motivate them and improve response rates (Stauber 1995).

One of the past leading public relations companies providing grassroots support for clients was Bonner and Associates. With a list of clients being the who’s who in multibillion dollar companies, they managed to influence the amendments to the US clean air act in 1990 in favour of their clients. This was done by persuading citizen groups the proposed amendments would prevent large vehicles from being built. Six states were scoured for grassroots voices and these were coached on the issue’s ‘facts’, paying for their phone calls and airfares to Washington where a hall was hired for a press conference (Greider 1992). The PR company had 300 phone lines out of their Washington office and an elaborate computer system. Their staff phoned citizens throughout the country searching for those that would support the corporate

agendas. They also targeted members of congress who were unfamiliar with the issue or wanted justification in voting the desired way.

An expert in one of the PR companies, John Davies, commented on letter-writing campaigns that it was “not actually to get a majority of the people behind a position and to express themselves on it – for it would be virtually impossible to whip up that much enthusiasm – but to get such a heavy, sudden outpouring of sentiment that lawmakers feel they are being besieged by a majority. The true situation may be quite the contrary” (Sherrill 1990p376).

Dillon’s (1993) coverage of a PR firm organising front environmental groups to support Hydro Quebec that wanted to build dams included this comment: “The masquerade is part of the game. Burson-Marsteller and companies like it have become masters of manipulation. If a pro-utility group calls itself by a nice, green-sounding name, if speakers at public forums are not identified as being on the Hydro Quebec payroll, and if supposed activists are really moles for the opposition, image triumphs and truth becomes a casualty”.

Sharyl Attkisson (2017p7), an award winning journalist, remarks: “We’re living amid an artificial reality, persuaded to believe it’s real by astroturf engineered to look like grassroots. Success of the paid forces hinges on their ability to remain virtually invisible. To disguise what they do and make it seem as if their work is neither calculated nor scripted. It must appear to be precisely what it is not.”

Smear Campaigns

Extensions of astroturfing are smear campaigns aimed at individuals. Such individuals may be scientists who have produced study findings contrary to the current orthodoxy or who have spoken with contrary views, but also extend to politicians, journalists and public activists.

Propaganda campaigns were established leading up to the second world war in Germany.

Goebbels’ propaganda approach yielded three general principles:

- the larger the lie, the more people will believe it
- a lie repeated often enough becomes the truth
- points used to convince must be few in number and repeated persistently.

Today these principles are frequently employed on targeted individuals.

In discussing her experience with smear campaigns Attkisson (2017) outlines several examples of the extent PR firms will go by:

- digging up “dirt to use against their targets.” If there is insufficient information, they will use unproven or discredited points
- Their message is amplified via simultaneous, relentless attacks to give the impression there are many sources, thus numbers.
- Opinions threatening their agenda are made controversial
- They locate and organise seemingly qualified people to give speeches, hold press conferences, issue position papers, write blogs, write letters to the editor, exploit social media, serve as experts in their organised ‘think tanks’, get interviewed on TV and radio, etc.

The largest PR company in the world in 2016, Edelman, had an income of US\$854 million in 2015 employing six thousand people. Astroturf smear campaigns may be identified as significant areas in their financial statements as “crisis and risk”, “strategic communications”, “rapid response”, or “reputation management” (Attkisson 2017).

A variation of the above can be public shaming by social media, as has been highlighted by Ronson (2016). This can be triggered by the victim making a casual comment as in the Don Imus case and it be taken out of context and broadcast via social media and lead to massive abuse by people reading the distributed quote perhaps with some accompanying comments.

Martin (2015) highlights that the shaming can be used positively as a method of rehabilitation for those who have broken the law and hurt others. However on the negative side shame can be debilitating for the victims who have been a focus of attack. Ronson attributes a mob aspect to the phenomena in being part of a crowd with a common purpose of righteousness. It can also be part of the psychological process of ‘projection’ as Martin points out where a negative characteristic of the abusive individual is disowned through attacking the victim via the abusive comments. Ronson points out that the togetherness in righteousness highlights a societal attitude that everyone must conform or risk assault by the crowd.

PR firms can be behind online newspapers such as the Richmond Standard in the US which blends real news with the odd story on Chevron developments or community actions. Its newsroom consists of an account executive from the PR company who is both the reporter and editor (Attkisson 2017). It is completely financed by Chevron, a large oil company.

The vaccine debate has seen a situation where the pharmaceutical industry has for decades marketed vaccines as critical for the population to be protected from communicable diseases. In Australia in 2009, a citizens' group was formed to silence vaccine critics: Stop the Australian Vaccination Network (SAVN). As the name suggests, it was set up to campaign against the Australian Vaccination Network which is a community based group supporting people affected by vaccines which spoke out about the vaccine problems and the manufactured ignorance. Tactics employed by the SAVN were primarily of denigration and harassment of the critics. Martin (2018) analysed the situation where although very crude forms of denigration were used by SAVN, there were continued attempts to smear the vaccine critics. Whilst never entering into a public debate about vaccines, the SAVN tries to tie-up the AVN by complaining to various government departments and medical organisations who then question and restrict the AVN.

In general, there are often indicators of smear campaigns from an astroturf group or paid industry shill. The most common trait is the various accusations: that a doctor is a 'quack', or that someone is using 'pseudoscience', or that something is 'science based' or that 'the science has been settled', or referring to the 'weight of evidence', or someone is a 'crank', or using the word 'pseudo', or that people's views represent a 'conspiracy'.

The book "The Pesticide Conspiracy" was published in 1978 by Robert van den Bosch. Bosch recounted case after case where scientists who documented negative effects of pesticides, had their reputations and work attacked.

In 2019, an investigation by French prosecutors was launched into Monsanto's alleged watch lists which collated private information on about 200 people. Bayer, Monsanto's new owner, admitted they kept such 'watch lists' (Kresge 2019, Leverkusen 2019, Phys Org 2019, Burger 2019). Bayer indicated, for the time being, it had ceased communications with the public relations agency Fleishman Hillard, which seems to have been involved in originating

Monsanto's hit lists. Fleishman Hillard defended their work, in that it has been "mischaracterized," and added:

Corporations, NGOs and other clients rightfully expect our firm to help them understand diverse perspectives before they engage. To do so, we and every other professional communications agency gather relevant information from publicly available sources.

Those planning documents are fundamental to outreach efforts. They help our clients best engage in the dialogue relevant to their business and societal objectives (Saunders 2019).

Smear campaigns are now highly organised by PR firms acting on behalf of the industry. The resources able to be assembled by such organisations are extremely difficult to counter. When members of the public are not familiar with the topic, these campaigns are especially effective.

Journalists As Agents In The Social Construction Of Scientific Ignorance

Journalistic involvement in the tobacco industry's strategy has been covered well by Jon Christensen (2008). He maintained that corporation public relations approaches "could use journalistic values to fatally undermine public understanding and encourage ignorance in even the most clear cut of public health cases."

The journalistic values of objectivity, balance, fairness and free speech makes journalism vulnerable to be used as an accomplice in the deliberate cultural production of ignorance. With the tobacco industry deliberately trying to make the subject of health effects controversial, this also created a natural attraction for journalistic coverage. But the tobacco industry machine had demanded such from the journalists: Phillip Morris Corp demanded that their points be published to newspapers, for example, if ever there were any negative news on smoking. They set up an early warning and rapid response division which was substantially funded to keep an eye on the media so that anything published could immediately be countered (Christensen 2008).

In 1990, for example, Phillip Morris allocated \$2.5 million for an environmental tobacco smoke communication plan for:

- maintaining the issue as controversial and correcting 'misinformation' in public and scientific meetings, interviews, media briefings, etc.
- science based journalist conferences,
- publishing reports on sick building syndrome around the world.

A subsequent budget included the sponsoring of educational programs for journalists on shortcomings of risk assessment and how to accurately report on such matters. Phillip Morris also identified journalists and publications "opposed to government regulations that are based on inaccurate science" (Christensen 2008). Such campaigns were supplemented with networks of public relations consultants who monitored the press and co-ordinated letters to the editors etc.

After studying how journalism was utilised, Jon Christensen (2008p279) concluded:

I would argue that this half century public relations war (by the tobacco industry) has proven that these journalistic standards render journalism constitutionally unfit, in general, to deal effectively with this kind of strategic manipulation of journalistic standards in the service of agnogenesis.

Stocking and Holstein (2015) claim that over the last two decades it has become accepted that scientific ignorance, like scientific knowledge, is socially constructed, as concluded in chapter 4. Both have focused on two aspects of research on the social construction of scientific ignorance with emphasis on the role journalists have played: through claims-making, and through the suppression of threatening or uncomfortable science.

In attempting to further scientific knowledge and present themselves as credible investigators, scientists regularly make claims about ignorance in their communications with other scientists. They claim that gaps in knowledge exist, issue caveats in-anticipation of criticism, etc.

A good example of ignorance claims was when two government sponsored studies indicated environmental damage and health effects to neighbours from large industrial hog farms in the US (Wing 2000). In similar tactics to the tobacco industry, the North Carolina Pork Council issued news releases and had interviews with journalists where they claimed the study research was flawed, misleading, incomplete, "pseudo-science", and claims that the lead

researcher was biased, politically motivated and incompetent. None of these claims was empirically based. Without countering knowledge of the environmental and health effects the Pork Council simply claimed ignorance on these aspects.

Covering this issue, journalists were classified into “attitudinal clusters” by Weaver and Wilhoit (1996) as follows:

- The disseminator: a reporter for Associated Press simply presented one claim after the other with the attitude that both sides of story were objectively presented. This approach has been one of the most common over the years where equal weight is given to both the scientists’ and industries’ claims even though the claims of science are the most soundly studied, logical and/or independently consensus based. Such approaches were present in the tobacco and global climate change situations. This approach is from the blind application of journalistic practices of maintaining objectivity and the news value of an apparently controversial issue.
- Interpretive/investigative: a writer for editorial pages took time to assess the science on his own. An editorial resulted that backed the scientists’ claims against the pork industry’s claims.
- The populist-mobilizer, seeking to give voice to audiences and set public agendas. Manufactured scientific controversies distort public agendas by preventing proper political debate due to the inverted and perceived scientific concerns (Weinel 2012).
- Adversarial: a reporter believing the fulfilling of the ‘watchdog’ role involved the distrust of both industry and academic claims. In treating the scientists claims with scepticism the trust of the scientists’ findings was undermined thereby giving credence to the Pork Council claims.

The effect of the above journalistic approaches assisted the Pork Industry to provide a hearing for its ignorance claims even without any countervailing scientific evidence.

In the background of the industry efforts to suppress these scientific studies were tactics such as (Wing 2002, Stocking 2015):

- Criticism the researcher being sent to all levels of the university bureaucracy: from the department head to the board of Trustees.

- Threatening the researcher with a defamation lawsuit.
- Summoning the researcher for questioning by a legislative committee of the US General Assembly.
- Demanding a North Carolina Congressional delegation investigate the National Institutes of Health funding for one of the scientist's studies in an attempt to shut it down.
- Using the state Freedom of Information Act to gain access to questionnaire data of people who lived around the hog farms. These people had been assured of human privacy protection. This action tied up the research as investigators cleansed all identifying markers.

Most of the journalists that Stocking and Holstein (2015) studied did not cover these industry tactics. One news item appeared about the threatened lawsuit (Wagner 1999) and was circulated by the Associated Press. An editorial appeared (News & Observer 1999) condemning such tactics. It seems that it is preferred by media to cover new stories about new research findings rather than the difficulties of covering these industry tactics (Clark 2006).

Stocking and Holstein (2015) suggested a number of aspects in their study covering journalists as agents in the social construction of scientific ignorance. They felt that it had been well-established in the literature that ignorance is socially constructed and time was needed to study and highlight the many ways it is constructed and the influencing factors. As previously covered, perceptions of individual journalists can affect the reporting of the strategic ignorance tactics. Their study also suggested that the journalists' perceptions of the audience and their understanding of science and how it works also can have a large influence.

In earlier work, Stocking (1999) suggested the following factors that influence journalists in their coverage of efforts to construct scientific ignorance:

- "individual characteristics of journalists, such as education, experience, and allegiance to professional standards and values (journalists' perceptions of their professional roles operate at this level);
- media routines, such as the balancing routine for controversial stories, but also including routines for defining news; and

- organizational demands, such as the need to tailor stories to particular audiences and to take into account advertiser and ownership pressures.” (Stocking 2015p110)

Weaver and Willnat (2012) proposed that it was likely that culture and ideology, intertwined with ownership and politics, influence how journalists cover industry claims. There are many examples that illustrate these effects, however examples will not be pursued here.

In conclusion, the above discussion has illustrated that journalists can be agents in the social construction of scientific ignorance.

Fact Checking Companies

A recent trend in the media industry is for supposedly neutral fact checking companies to be utilised by the public. An example is NewsGuard, a self-appointed internet watchdog and arbiter selling a browser plugin that rates websites on nine criteria of credibility and transparency. The Publicis Groupe, a global communications company, provided most of the start-up funding for NewsGuard. Their health subsidiary, Publicis Health, has large pharmaceutical companies as clients, such as Lilly, Abbot, Roche, Amgen, Genentech, Celgene, Gilead, Biogen, Astra Zeneca, Sanofi, Bayer. In 2018, GlaxoSmithKline placed its \$1.5 billion media account with Publicis (Bulik 2018). It then handed over to Publicis Media the former Pfizer Consumer Healthcare Brands worth an estimated \$400 million (Bulik 2020). There is a general lack of transparency in NewsGuard as evidenced by their decline to disclose their revenue on the US Securities and Exchange Commission Form D filed March 5, 2018, and its claim for a Rule 506(b) exemption which enabled an unlimited number of investors. This contrasts with the 100% transparency they expect from the subjects of their work. NewsGuard tries to maintain free subscriptions for its service for schools and libraries.

The extremes of fact-checking actions have recently seen some reactions from those affected. There are two recent examples with respect to Facebook. Firstly, they had been taken to court by television journalist John Stossel. He claimed that facebook’s fact-checkin was defamation when it labelled his content as false, which would result in viewers to doubt its integrity. Stossel's lawsuit related to a video he had on Facebook about the 2020 California wildfires. In the video it was suggested that government mismanagement, rather than climate change, was

a significant factor in causing the fires. The downplay of climate change resulted in Stossel's video being labelled as "misleading". In the court hearings facebook representatives said that information was censored not based on facts but opinions without verification of facts.

The second example was when Facebook censored the British Medical Journal (BMJ) over an article that highlighted potential problems with Pfizer's COVID jab trial. The BMJ is one of the oldest and most respected peer-reviewed medical journals. The BMJ published a whistleblower report (Thacker 2021) that claimed there were serious data integrity issues in the Pfizer COVID jab trial. The article was censored by Facebook and labelled variably as either "False," "Partly false" or "Missing context." The BMJ had hired an investigative reporter to write the story, which was peer-reviewed, and legally reviewed. The BMJ had dozens of internal documents, photos, email, and recordings, that backed up their article (Godlee 2021).

The Aspect of Reproducibility

In an effort to reproduce significant studies in the field of preclinical cancer, Begley and Ellis repeated 53 published studies over the course of a decade. For some cases the original lab under the same original investigators were used. But in only 6 out of the 53 cases, were the scientific findings confirmed (Begley C, 2012, Begley S, 2012).

For one case C. Begley commented "... we re-did their experiment 50 times and never got their result. He said they'd done it six times and got this result once, but put it in the paper because it made the best story" (Begley S, 2012). It has been pointed out by Cosgrove, et al. that this cherry-picking of positive data and discarding of negative trials is allowed by the US FDA regulatory approval process for the classification of products as being safe and effective (Cosgrove 2016).

False Advertising in Research Studies

False advertising is a term used by Carrier (2019) referring to studies where there can be differences between the conclusions derived strictly from the design of a study and those mentioned or intimated. This can take the form of abstracts and conclusion sections being incongruous with the study design and/or results. The example in appendix 1 shows an

underpowered epidemiological study with many shortcomings, but due to its abstract and conclusion, it was used as a reference to counter claims of increased health effects when the petrol/gasoline formulation was changed in the US.

When Knowledge Manipulation is Highlighted

There are consequences from a century of relatively unrestrained industrial development. A book that highlighted knowledge manipulation by a number of chemical companies, 'Deceit & Denial' by Gerald Markowitz and David Rosner, came to the fore when it started to be used in a court case in the US for compensation of workers, including deaths, from chemical exposures (Subpoena 2004). The Chronical of Higher Education (Guterman 2004p1) published an article on the situation beginning as follows.

Lawyers representing more than 20 chemical companies have taken the unusual step of issuing subpoenas to five peer reviewers of a scholarly book as part of litigation over the alleged health risks of a widely used chemical compound... The book's publishers also received subpoenas, several months ago, to provide information about early drafts of the book and its peer review.

The legal action was taken two years after the publication of the book's first edition. These actions were the chemical company lawyers attempting to destroy the credibility of the book, its authors and the peer review process.

The book had outlined the knowledge manipulation of some chemical companies on the effects of lead and vinyl chloride chemicals.

The chemical company lawyers enlisted help from 'qualified' people in their attempt to discredit the authors. One of these was P. Scranton, director of the Hagley Museum and Library's Center for the History of Business, Technology, and Society and University Board of Governors, Professor of History at Rutgers University in Camden, New Jersey. He accused the authors of not having adequate documentation, yet the book had eighty-one pages of footnotes and references. His main argument was in respect to the Statement of Professional Conduct of the American Historical Association (AHA) and attempted to show they had violated principles of historiography. Specifically, he accused that the authors "repeatedly violated the

AHA's and the NCPH's [National Council on Public History] guidelines in six specific areas: I. Integrity and Accuracy; II. Misrepresentation and Omission; III. Advocacy and Oversimplification; IV. Inadequate Documentation; V. Qualification and Interpretation; and VI. Professional Ethics." (Scranton 2004). To this Roy Rosenzweig, the vice president for research of the AHA, Professor of History at George Mason University, responded, "I've read the AHA Statement on Standards, . . . I see nothing in Markowitz and Rosner's book that's a violation of the AHA Standards. In my opinion, the book represents the highest standards of the history profession. Scranton should be embarrassed to make the claim that there's an ethical violation here as opposed to the claim that he disagrees with their interpretation" (Wiener 2005). Later the authors were awarded the American Industrial Hygiene Association's Upton Sinclair Award and the New York Committee on Occupational Safety and Health honoured them for "outstanding scholarship exposing the deadly politics of industrial pollution" as well as their "advocacy on behalf of every person's right to an environment and a workplace that is safe and healthful."

In Gerald Markowitz and David Rosner's epilogue in the latest edition of their book *Deceit & Denial*, they commented that their involvement in this case made them appreciate the value of studying history. The ongoing media coverage of the court case had enabled the population to realise the injustice that had affected thousands of their children. The authors relate their sense of achievement in this regard in seeing a demonstration by mothers of affected children outside the courthouse with banners including 'they knew for decades.'

In another lawsuit, one of the first of its kind, a US State Department of Health applied health prevention principles to force polluting companies to remove lead paint from Rhode Island's housing. This was due to the ongoing chronic lead poisoning of the state's children. The State argued that lead paint was a 'public nuisance' and an injury waiting to happen. The industry's knowledge of lead's dangers was central to the case.

The State Department of Health won the case however the industry immediately launched intense lobbying with blog posts, editorials, and articles attacking the decision. After seven years of resultant legal wrangling, the Rhode Island Supreme Court overturned the decision by saying that nuisance law could not be applied to such a broad environmental disaster.

The book 'Golden Holocaust' by Robert Proctor (2011) followed the tobacco industry's manipulated knowledge of cigarette health effects to the extent that he too was asked to appear in court, by lung cancer victims. He was forced to hire lawyers for his defence against industry attempts to investigate his past and present research.

It has been shown by many examples through this thesis that industry has applied particular types of ignorance to its advantage. So it is interesting to note evidence of knowledge manipulation in considering the amount of fines that the pharmaceutical industry has encountered on the supposedly most thoroughly studied end of the environmental exposure spectrum: where xenochemicals are deliberately prescribed for the public: medical drugs. Just in the US, Merck and its subsidiaries since 2000, have been fined 42 times with fines totalling US \$3,023,907,640. Some of the offences are for unapproved promotion of medical products, off-label uses, false claims, safety violations, kickbacks and bribery, environmental violations, foreign corrupt practices, etc. These of course are the ones that have been discovered and successfully proven. This continues for many of these companies for example Bayer has 102 fines for similar offences in the same period (GJF 2019).

The above situation with pharmaceutical drugs contrasts with other industries which add numerous toxic chemicals to our food, cosmetics, etc. as well as EMF to our living environments. Apart from little, if any, testing done before exposing the public to such environmental exposures, there are no systems to record effects, let alone the problems in differentiating particular effects to the soup of chemicals and radiation that are now in our living environment.

CONCLUSION

Chapter two highlighted studies on the effect of various low-level environmental exposures, and extensive studies were found that showed many effects on health despite significant areas of undone science. Chapter 3 considered the significant percentage of the population most affected by environmental exposures and discussed multiple chemical sensitivity (MCS). People with MCS are like canaries in the coal mine indicating that low-level environmental exposures can have mild-to-incapacitating health effects. The orthodox medical system commonly dismisses them as having psychological problems with a general attitude that low-level

exposures do not cause any health effects. Chapter 4 then considered how the orthodox medical system and the general public remain ignorant about environmental effects, especially in light of the evidence in chapter 2. It was shown how industry can generate ignorance about the effects of environmental exposure.

Part of the process of generating ignorance is manipulating knowledge. As the research community is heavily reliant on industry's financial support, many conflicts of interest (COI) occur. It was shown that the source of funding has a clear effect on the results presented and conclusions made in scientific studies. This in turn affects journal articles and regulatory authority decisions which are normally based on industry-funded research. In addition, objectivity was shown to be outweighed by economic, personal and social factors in journal peer review and the retraction of incorrect articles rarely occurred.

Many examples of institutional COI were demonstrated in the major US regulatory bodies: the CDC, FDA, NIH and EPA. These institutional COI extended through the health system, as shown with examples of politicians, medical students, doctors and medical associations.

The strategies used in the manipulation of knowledge were also covered. Fostering doubt is used extensively by industry as a response to negative findings about their products, while another seen frequently is to blame the sufferer for their health condition.

Enabling the continuation of activities that affect people's health can sometimes be achieved by using the social "power" of the organisations involved to create ignorance of the real situation, as happened in the case of asbestos mining.

Further industry strategies include the use of shills and smear campaigns. Industry shills can be effective as they usually appear independent and their numbers are significant in the public-speaking arena. Yet anyone who presents a negative view on the industry or its products might be targeted by a smear campaign from industry-backed organisations.

Industry-commissioned PR firms can create what appear to be grass roots populist movements in order to oppose any proposed government legislation or regulations, and the journalistic values of objectivity, balance and fairness are often manipulated to serve industry interests. The latest trend is for industry to back fact-checking companies for manipulation purposes.

An age-old strategy, still very pertinent, only uses study data that supports the safety and effectiveness of the industrial product being reviewed. This is especially the case in submissions requesting regulatory approval. Also as a strategy, industry is prepared to pursue legal action. This can be to debunk findings or comments by researchers or activists, thereby discouraging others.

As this summary demonstrates, this chapter has considered how knowledge can be manipulated and distorted by industry and government entities. The importance of generating ignorance through knowledge manipulation is obvious for industry financial interests. The evidence of the extensive manipulation of knowledge to create ignorance around environmental effects on health, as shown in this chapter, further supports the central argument of this thesis.

REFERENCES

AAAS 2019a: Award for Scientific Freedom and Responsibility <https://www.aaas.org/page/aaas-award-scientific-freedom-and-responsibility-0> accessed 27 Feb 2019.

AAAS 2019b: Global fight against lethal herbicides earns 2019 AAAS Scientific Freedom and Responsibility Award, Adam D. Cohen, Office of Public Programs AAAS Annual Meeting Newsroom, AAAS, 4 February 2019 <https://www.aaas.org/news/global-fight-against-lethal-herbicides-earns-2019-aaas-scientific-freedom-and-responsibility> [as of 6 Feb 2019 this press release has been removed from the AAAS section of the Eurekalert site]: copied at <https://www.gmwatch.org/en/news/latest-news/18748> accessed 27 Feb 2019.

AAAS 2019c: AAAS Announces Rush D. Holt to Retire in 2019, Search Underway for Next CEO, 6 Feb 2019, <https://www.aaas.org/news/aaas-announces-rush-d-holt-retire-2019-search-underway-next-ceo> accessed 27 Feb 2019.

ABC, Food addiction and the brain, Health Report, Radio National, 5 August 2013 5:40PM <https://www.abc.net.au/radionational/programs/healthreport/food-addiction/4865260> accessed 17 Mar 2019

Adams C, FDA searches for an elixir for agency's attrition rate. The Wall Street Journal, section A:4, 2002 Retrieved from: <http://www.wsj.com/articles/SB1029711786987700275>

AGBUC: Association of Governing Boards of Universities and Colleges, Board policy on conflicts of interest, 17 Jan 2007. Available: <http://agb.org/statements/2007/agb-statement-on-board-accountability> Accessed 19 Jan 2019.

Anderson L, What Are Biosimilars? Top Facts You May Not Know, Drugs.com, 22 Jul 2020 [What Are Biosimilars? Top Facts You May Not Know \(drugs.com\)](#) accessed 5 Jan 21

Angell M, Drug companies and doctors: A story of corruption. The New York Review of Books, 2009, January 15.

ASIC: Australian Securities and Investments Commission, Our Role, What we do <https://asic.gov.au/about-asic/what-we-do/our-role/#what> accessed 7 June 2019.

Attkisson S, The Smear: How shady political operatives and fake news control what you see, what you think, and how you vote, Harper Collins, NY, 2017.

Austad K, Avorn J, Kesselheim A, Medical students' exposure to and attitudes about the pharmaceutical industry: a systematic review, PLoS medicine, 2011, 8, 5, e1001037.

Barnes D, Bero L, Industry-funded research and conflict of interest: an analysis of research sponsored by the tobacco industry through the Center for Indoor Air Research, Journal of Health Politics, Policy and Law, 1996, 21, 3, 515-42.

Barnes D, Bero L, Why review articles on the health effects of passive smoking reach different conclusions, Jama, 1998, 20, 279, 1566-70.

Beder S. Public relations' role in manufacturing artificial grass roots coalitions. Public Relations Quarterly, 1998, 43, 2, 21-7.

Begley C, Ellis L, Raise standards for preclinical cancer research. Nature, 2012, 483, 531–33.

Begley S, In cancer science, many 'discoveries' don't hold up. Reuters, 29 Mar 2012. Retrieved from: <http://www.reuters.com/article/us-science-canceridUSBRE82R12P20120328> Accessed 27 Jun 21.

Bekelman J, Li Y, Gross C, Scope and impact of financial conflicts of interest in biomedical research: a systematic review, Jama, 2003, 289, 4, 454-65.

Bero L, Tobacco industry manipulation of research, Public Health Reports 2005 Mar–Apr; 120:200–208.

Bes-Rastrollo M, Schulze M, Ruiz-Canela M, Martinez-Gonzalez M, Financial conflicts of interest and reporting bias regarding the association between sugar-sweetened beverages and weight gain: a systematic review of systematic reviews, PLoS Med, 2013, 10, 12, 1–9.

Bleifuss J, Science in the Private Interest: Hiring Flacks to Attack the Facts, PR Watch 1995, 2, 1, 11-12. Anon, Public Interest Pretenders, Consumer Reports 1994, 59, 5, 319. Anon, Misguided Health Priorities Could Affect Economy, International Insurance Monitor 1987, 41, 6, 16-17. Anon, Dr. Blasts US Health Care Priorities, Cash Flow 1987, 91, 47, 28-29.

Brodeur P, Outrageous misconduct: The asbestos industry on trial, 1985: referenced in (Wagner 2004p127).

Bulik B, GlaxoSmithKline taps Publicis—and its 'platformGSK' setup—for \$1.5B media account, Fierce Pharma, Oct 8, 2018 [GlaxoSmithKline taps Publicis—and its 'platformGSK' setup—for \\$1.5B media account | FiercePharma](#) accessed 7 Jan 21.

Bulik B, GlaxoSmithKline consolidates Pfizer consumer brands under Publicis Media specialty unit, Fierce Pharma, Jan 3, 2020 [GlaxoSmithKline consolidates Pfizer consumer brands under Publicis Media specialty unit | FiercePharma](#) accessed 7 Jan 21.

[Burger](#) L, Bayer says Monsanto likely kept files on influential people across Europe, Reuters, Business News, May 14, 2019 <https://www.reuters.com/article/us-bayer-france-monsanto-europe/bayer-says-monsanto-likely-kept-files-on-influential-people-across-europe-idUSKCN1S1JOY> accessed 4 June 2019.

Cain D, Loewenstein G, Moore D, Coming clean but playing dirtier: The shortcomings of disclosure as a solution to conflicts of interest. In Moore D, Cain D, Loewenstein G, Bazerman M, (Eds.). *Conflicts of interest: Challenges and solutions in business, law, medicine, and public policy* (104–125), Cambridge, United Kingdom: Cambridge University Press, 2005.

Camerer C, Loewenstein G, Weber M, The curse of knowledge in economic settings: An experimental analysis. *Journal of Political Economy*, 1989, 97, 1232–54.

Campbell E, Weissman J, Ehringhaus S, Rao S, Moy B, Feibelmann S, Goold S, Institutional academic-industry relationships, *JAMA*, 2007, 298, 15, 1779–86.

Cancryn A, Haberkorn J, Why the CDC director had to resign, Brenda Fitzgerald's departure quickly followed the arrival of HHS Secretary Alex Azar. *Health Care, Politico*, 31 Jan 2018 [Why the CDC director had to resign - POLITICO](#) [Back Button](#) [Search Icon](#) [Filter Icon](#) accessed 5 Jan 21.

Capps B, Can a good tree bring forth evil fruit? The funding of medical research by industry. *British Medical Bulletin*, 2016, 118, 5–15.

Carrier M, Agnotological challenges: how to capture the production of ignorance in science, Chapter 3. In: Kourany J, Carrier M, eds. *Science and the Production of Ignorance : When the Quest for Knowledge Is Thwarted*. Cambridge, MIT Press, 2020.

Casadevall A, Steen R, Fang F, Sources of error in the retracted scientific literature. *The FESEB (Federation of Experimental Societies for Experimental Biology) Journal*, 2014, 28, 3847–55.

Cauchon D, FDA advisers tied to industry, *USA Today*, 25 Sep 2000, 10A. <https://archive.commondreams.org/views04/headlines/092500-01.htm>

CDC Foundation: Centers for Disease Control Foundation, Year in review, 2016 <https://www.cdcfoundation.org/FY2016#year-review>

Christensen J, Smoking out objectivity: Journalistic gears in the agnogenesis machine, In: *Agnotology, The making and unmaking of ignorance*, Stanford University Press, 2008, Chapter 12, 266-82

Clark J, Marketing: a hot flush for big pharma, *BMJ*, 2003, 327, 7411, 400.

Clark F, Illman D, A longitudinal study of The New York Times Science Times section, *Science Communication*, 2006, 27, 4, 496–513.

Cosgrove L, Vannoy S, Mintzes B, Shaughnessy A, Under the influence: The interplay among industry, publishing, and drug regulation, *Accountability in Research*, 2016, 23(5), 257–279.

Cranor C, *Toxic torts: Science, law, and the possibility of justice* (2nd ed.), Cambridge, United Kingdom, Cambridge University Press, 2016.

CRP: Center for Responsive Politics, Industry Profile: Pharmaceuticals/health products, 2020 [Pharmaceuticals/Health Products Lobbying Profile • OpenSecrets](#) accessed 6 May 2021

Dana J, Loewenstein G, A social science perspective on gifts to physicians from industry, *JAMA*, 2003, 290, 2, 252–55.

Davidoff F, DeAngelis C, Drazen J, Hoey J, Højgaard L, Horton R, Kotzin S, Nicholls M, Nylenna M, John A, Overbeke P, Sponsorship, authorship, and accountability, *The Lancet*, 2001, 358, 9285, 854-6.

DeAngelis C, Fontanarosa P, Impugning the integrity of medical science: The adverse effects of industry influence, *JAMA*, 2008, 299, 15, 1833–35.

Diels J, Cunha M, Manaia C, Sabugosa-Madeira B, Silva M, Association of financial or professional conflict of interest to research outcomes on health risks or nutritional assessment studies of genetically modified products, *Food Policy*, 2011, 36, 2, 197–203.

Dillon J, PR Giant Burson-Marsteller thinks global, acts local: poisoning the grassroots, *Covert Action*, 1993, 44, 30-8.

Dobson J, Whitley R, Pocock S, Monto A, Oseltamivir treatment for influenza in adults: a meta-analysis of randomised controlled trials, *Lancet*, 2015, 385, 1729-37.

Dórea J, Low-dose Thimerosal (ethyl-mercury) is still used in infants vaccines: Should we be concerned with this form of exposure?, *Journal of Trace Elements in Medicine and Biology*, 2018, 49, 134-9.

[Ehley B, Karlin-Smith S, CDC director who traded tobacco stock resigns, Politico, 31 Jan 2018](#)
<https://www.politico.com/story/2018/01/31/cdc-director-resigns-over-financial-conflicts-380206>

Elliott C, Pharma goes to the laundry: Public relations and the business of medical education, *Hastings Center Report*, 2004, 34, 5, 18–23.

Elliott K, Scientific judgment and the limits of conflict-of-interest policies, *Accountability in Research*, 2008,15, 1, 1–29.

England C, Former CDC Director that Approved Gardasil Vaccine and Became Head of Merck's Vaccine Division Named "Woman of the Year", *Health Impact News*, 26 Jan 2018 [Former CDC Director that Approved Gardasil Vaccine and Became Head of Merck's Vaccine Division Named "Woman of the Year" \(healthimpactnews.com\)](#) accessed 5 Jan 21

Foley K, Trust issues deepen as yet another FDA commissioner joins the pharmaceutical industry, *Quartz*, 2 Jul 2019 [Yet another FDA commissioner joins the pharmaceutical industry — Quartz \(qz.com\)](#) accessed 5 Jan 21

Folta K, AAAS fumble? Prestigious scientists' organization endorses 'data-less' study suggesting links between glyphosate and kidney disease in Sri Lanka, *Genetic Literacy Project*, 5 Feb, 2019
<https://geneticliteracyproject.org/2019/02/05/aaas-fumble-prestigious-scientists-organization-endorses-data-less-study-suggesting-links-between-glyphosate-and-kidney-disease-in-sri-lanka/> accessed 27 Feb 2019.

García P, Corruption in global health: the open secret, *Lancet*, 2019, 394, 10214, P2119-24,

GJF: Good Jobs First: tracking subsidies, promoting accountability in economic development violation tracker <https://violationtracker.goodjobsfirst.org/parent/merck>
<https://violationtracker.goodjobsfirst.org/prog.php?parent=bayer> accessed 2 July 2019

Glantz S, Slade J, Bero L, Hanauer P, Barnes D, eds: *The Cigarette Papers*, Berkeley, University of California Press, c1996 1996. <http://ark.cdlib.org/ark:/13030/ft8489p25j/>

Godlee F, Rapid Response: Open letter from The BMJ to Mark Zuckerberg, *BMJ* 2021;375:n2635

[Gottlieb S, on Twitter 28 Jun 2019: "I'm honored to be joining the board of directors of #Pfizer and working together with more than 90,000 Pfizer colleagues to promote medical innovation, advance patient care, and secure access to better healthcare outcomes for families around the world. @pfizer https://t.co/RgzYbPZci7" / Twitter](#) accessed 5 Jan 21

Gould F, Public release event for genetically engineered crops: experiences and prospects, National Academies of Science, Engineering and Medicine Keck Center, 500 5th St NW, Washington, DC, 17 May 2016 <https://nas-sites.org/ge-crops/2016/04/27/report-release/> Accessed 19 Jan 2019.

Gray G, Cohen J, Cunha G, et al. Weight of the evidence evaluation of low-dose reproductive and developmental effects of bisphenol A, *Hum Ecol Risk Assess*, 2004, 10, 875–921

Greider W, *Who will tell the people: the betrayal of American democracy*, Simon & Schuster, New York, 1992, p37.

Guillemaud T, Lombaert E, Bourguet D. Conflicts of Interest in GM Bt crop efficacy and durability studies, *PLoS One*, 2016, 11, 12.

Guterman L, Peer reviewers are subpoenaed in cancer lawsuit against chemical companies, *Chronicle of Higher Education*, 19 Nov 2004.

Heath D, Obama's EPA breaks pledge to divorce politics from science on toxic chemicals. Center for Public Integrity. 23 Jan 2015, Retrieved from:

<https://www.publicintegrity.org/2015/01/23/16641/obamas-epa-breaks-pledgedivorce-politics-science-toxic-chemicals> accessed 24 Jun 21.

Heath D, Greene R, EPA contaminated by conflict of interest, Center for Public Integrity, 13 Feb 2013, Retrieved from:

<http://www.pbs.org/newshour/spc/multimedia/epa-corporate/> accessed 24 Jun 21.

Heinemann J, a professor at the University of Canterbury, NZ, Twitter 9:49 AM - 8 Feb 2019

https://twitter.com/Jack_Heinemann/status/1093929931413819392 accessed 27 Feb 2019.

HHS: US Dept of Health and Human Services, FDA's review process for new drug applications: a management review (Publication no. OEI-01-01-00590), Office of U.S. Inspector General, 2003

<https://oig.hhs.gov/oei/reports/oei-01-01-00590.pdf>

Holliday R, Epigenetics: a historical overview, *Epigenetics*, 2006, 1, 2, 76-80.

Hooker B, Kern J, Geier D, Haley B, Sykes L, King P, Geier M, Methodological issues and evidence of malfeasance in research purporting to show thimerosal in vaccines is safe, *Bio Med Research International*, 2014, 247218, 8 pages

Horton R, What is medicine's 5 sigma? *Lancet*, 2015, 385, 9976, 1380.

Howard J, Old emails hold new clues to Coca-Cola and CDC's controversial relationship, CNN January 29, 2019 <https://edition.cnn.com/2019/01/29/health/coca-cola-cdc-emails-study/index.html>

ICAN: Informed Consent Action Network, Introduction to vaccine safety science & policy in the United States, 2017. Retrieved from <https://icandecide.org/wpcontent/uploads/whitepapers/VaccineSafety-Version-1.0-October-2-2017.pdf>

IMT: Summary of the advisory opinion of the International Monsanto Tribunal Delivered on the 18th of April 2017 in The Hague, Netherlands.

https://en.monsantotribunal.org/upload/asset_cache/1016160509.pdf accessed 27 Feb 2019.

Institute of Medicine (US) Committee on Conflict of Interest in Medical Research, Education, and Practice; Lo B, Field M, eds, *Conflict of Interest in Medical Research, Education, and Practice*, Washington (DC), National Academies Press (US), 2009, 8, Institutional Conflicts of Interest.

Jayasumana C, Gunatilake S, Senanayake P, Glyphosate, hard water and nephrotoxic metals: are they the culprits behind the epidemic of chronic kidney disease of unknown etiology in Sri Lanka? *International journal of environmental research and public health*, 2014, 11, 2, 2125-47.

Jayasumana C, Gunatilake S, Siribaddana S, Simultaneous exposure to multiple heavy metals and glyphosate may contribute to Sri Lankan agricultural nephropathy, *BMC Nephrology*, 2015a, 16, 103.

Jayasumana C, Paranagama P, Agampodi S, Wijewardane C, Gunatilake S, Siribaddana S. Drinking well water and occupational exposure to Herbicides is associated with chronic kidney disease, in Padavi-Sripura, Sri Lanka, *Environmental Health*, 2015b, 14, 1, 6.

KEILC: Knowledge Ecology International Liberty Coalition Project on Government Oversight Public Citizen U.S. Right to Know, Letter 5 Nov 2019 [Petition-to-CDC-re-Disclaimers.pdf \(usrtk.org\)](#) accessed 31 Dec 2019

Kennedy R, Ed, Thimerosal: Let the science speak. New York, Skyhorse Publishing, 2014.

Kluger R, *Ashes to ashes: America's hundred-year cigarette war, the public health, and the unabashed triumph of Philip Morris*, Vintage, 26 May 2010.

Kornfield R, Donohue J, Berndt E, Alexander G, Promotion of prescription drugs to consumers and providers, 2001–2010, *PLoS ONE*, 2013, 8, 3), e55504.

Kresge N, Bayer Hires Lawyers as Monsanto Spying Probe Widens, *Bloomberg, Technology*, 21 May 2019 <https://www.bloomberg.com/news/articles/2019-05-21/bayer-hires-sidley-austin-as-monsanto-spying-probe-widens> accessed 4 June 2019

Krimsky S, *Science in the private interest: Has the lure of profits corrupted biomedical research?* Rowman & Littlefield, 2004.

Krimsky S, Rothenberg L, Conflict of interest policies in science and medical journals: Editorial practices and author disclosure. *Science and Engineering Ethics*, 2001, 7, 2, 205–18.

Krimsky S, The funding effect in science and its implications for the judiciary, *JL & Pol'y*, 2005,13, 43.

Krimsky S, Schwab T, Conflicts of interest among committee members in the National Academies' genetically engineered crop study, *PloS one*, 2017, 12, 2, e0172317.

Krimsky S. Combating the funding effect in science: what's beyond transparency. *Stan. L. & Pol'y Rev.*. 2010;21:81.

Kumarasinghe K, Glyphosate without adjuvants not very useful: Prof. Sarath Gunathilake, *Daily Mirror*, 27 Jun 2018 <http://www.dailymirror.lk/article/Glyphosate-without-adjuvants-not-very-useful-Prof-Sarath-Gunathilake-151867.html> accessed 23 Jun 21.

Lenzer J, Centers for Disease Control and Prevention: protecting the private good? *BMJ (Online)*, 15 May 2015, 350.

Leverkusen, Bayer commissions external law firm to investigate Monsanto's stakeholder mapping project and reaffirms its commitment to transparency and fair dealings with all stakeholders, *Bayer Global, Media Release*, May 12, 2019 <https://media.bayer.com/baynews/baynews.nsf/id/Bayer-commissions-external-investigate-Monsantos-stakeholder-mapping-project-reaffirms-commitment?Open&parent=news-overview-category-search-en&ccm=020> accessed 4 June 2019

Levinson D, CDC's ethics program for special government employees on federal advisory committees. Department of Health and Human Services, Office of Inspector General, Dec 2009. <https://oig.hhs.gov/oei/reports/oei-04-07-00260.pdf>

Lexchin J, Bero L, Djulbegovic B, Clark O, Pharmaceutical industry sponsorship and research outcome and quality: systematic review, *BMJ*, 2003, 326, 7400, 1167-70.

Lexchin J, Light D, Commercial influence and the content of medical journals. *BMJ*, 2006, 332, 1444-47.

Lopez J, Lopez S, Means J, Mohan R, Soni A, Milton J, Tufaro A, May J, Dorafshar A, Financial conflicts of interest: an association between funding and findings in plastic surgery, *Plastic and Reconstructive Surgery* 2015, 136, 5, 690e-97e.

Lundh A, Sismondo S, Lexchin J, Busuioac O, Bero L, Industry sponsorship and research outcome, *Cochrane Database Syst Rev*, 12 Dec 2012, 12, 12.

Markowitz G, Rosner D, Deceit and denial: The deadly politics of industrial pollution, Univ of California Press, 2013.

Martin B, Emotions, mobbing: Dealing with shaming, 5 May 2015 <http://comments.bmartin.cc/2015/05/05/dealing-with-shaming/> accessed 6 Oct 18

Martin B, Fraud and the Pharmaceutical Industry, <https://www.uow.edu.au/~bmartin/dissent/documents/health/pharmfraud.html> accessed 13 Oct 2018.

McCulloch J, Tweedale G, Defending the indefensible: The global asbestos industry and its fight for survival, Oxford, UK, Oxford University Press, 2008.

Megalli M, Friedman A, Masks of Deception: Corporate Front Groups in America, *Essential Information*, 1991, p4.

Mehra M, Desai S, Ruschitzka F, Patel A, Hydroxychloroquine or chloroquine with or without a macrolide for treatment of COVID-19: a multinational registry analysis, *Lancet*. 2020 May 22, (Retracted).

Poole W, Neither Wise nor Well, *Sierra*, 1992, 59-61, 88-93.

Merton R, The normative structure of science, in: *The Sociology of Science: Theoretical and Empirical Investigations*, Guston J, ed, 1973, 267, 275.

Merton R, The Matthew Effect in science. *Science, New Series*, 1968, 159, 3810, 56-63.

Michaels D, Manufactured uncertainty: Contested science and the protection of the public's health an environment, In: *Agnotology, The making and unmaking of ignorance*, Stanford University Press, 2008, Chap 4, 90-107

Michaels D, Manufactured uncertainty. *Annals of the New York Academy of Sciences*, 2006, 1076, 149-62.

Mintz M, At any cost: corporate greed women and the Dalkon Shield, 1985: Bibliographic Citation New York: Pantheon Books, 1985, p308; also referenced in (Wagner 2004p127).

Mirowski P, *Science-mart: Privatizing American science*. Cambridge, MA, Harvard University Press, 2011.

Mundy A, *Dispensing with the truth: The victims, the drug companies, and the dramatic story behind the battle over Fen-Phen*, St. Martin's Press, 2010, 119.

NARA: National Academies Roundtable Agenda. <http://nas-sites.org/publicinterfaces/files/2014/07/PILS-02-GMO-Interface-agenda10.pdf>. Accessed 19 Jan 2019.

NAS: National Academy of Sciences, Report of the Treasurer of the National Academy of Sciences for year ended December 2014-2015. <http://www.nap.edu/read/21779/chapter/4>. Accessed 19 Jan 2019.

NAS: National Academies of Science, Engineering and Medicine: Public Interface in Life Sciences Roundtable. Public engagement on genetically modified organisms: when science and citizens connect, a workshop summary, Washington DC, National Academies Press, 2015. <http://www.nap.edu/read/21750/chapter/1>. Accessed 19 Jan 2019.

NASEM: National Academies of Science, Engineering and Medicine, Committee on Genetically Engineered Crops: Genetically Engineered Crops: Experiences and Prospects. Washington DC, National Academies Press, 2016. <http://www.nap.edu/catalog/23395/genetically-engineered-crops-experiences-and-prospects>.

Newbold R, Impact of environmental endocrine disrupting chemicals on the development of obesity, *Hormones*, 2010, 9, 3, 206-17.

News & Observer, Pork or porcupine? editorial, Raleigh NC, 14 May 1999, A18.

O'Connor A, Coca-Cola funds scientists who shift blame for obesity away from bad diets. *New York Times*, 9 Aug 2015. <http://well.blogs.nytimes.com/2015/08/09/coca-cola-funds-scientists-who-shiftblame-for-obesity-away-from-bad-diets>

Ogien A, Doubt, Ignorance and Trust: On the unwarranted fears raised by the doubt mongers. In: *Routledge International Handbook of Ignorance Studies*, Routledge, 2015, Chap 20, 192-8

Oransky I, Marcus A, Thousands of studies used the wrong cells, and journals are doing nothing. *STAT*, 21 Jul 2016. Retrieved from <https://www.statnews.com/2016/07/21/studies-wrong-cells/>

Ordunez P, Nieto F, Martinez R, Soliz P, Giraldo G, Mott S, Hoy W, Chronic kidney disease mortality trends in selected Central America countries, 1997–2013: clues to an epidemic of chronic interstitial nephritis of agricultural communities. *J Epidemiol Community Health*, 2018, 72, 4, 280-6.

Peters D, Ceci S, Peer-review practices of psychological journals: the fate of submitted articles, submitted again, *Behavioral and Brain Science*, 1982, 5, 2, 187–255.

Peterson M, Biotech expert's new job casts a shadow on report, *New York Times*, 16 Aug 1999 <http://www.nytimes.com/1999/08/16/us/biotech-expert-s-new-job-casts-a-shadow-on-report.html>. Accessed 23 June 21

Pew, Persuading the prescribers: Pharmaceutical industry marketing and its influence on physicians and patients. *Pew Prescription Project*, 2013, 441. <http://www.pewtrusts.org/en/research-and-analysis/factsheets/2013/11/11/persuading-the-prescribers-pharmaceutical-industry-marketing-and-its-influence-on-physicians-and-patients>

Pham-Kanter G, Revisiting financial conflicts of interest in FDA advisory committees, *The Milbank Quarterly*, 2014, 92, 3, 446-70.

Phys Org, Bayer admits Monsanto may have other 'watch lists', *Phys Org, Biology*, 12 May 2019. <https://phys.org/news/2019-05-bayer-monsanto.html> accessed 4 June 2019.

Porta M, Lee D, Review of the science linking chemical exposures to the human risk of obesity and diabetes, *Chem Trust UK*, 2012. <https://www.chemtrust.org/wp-content/uploads/CHEM-Trust-Obesity-Diabetes-Summary-Report.pdf> accessed 17 Mar 2019

Proctor R, Agnotology: a missing term to describe the cultural production of ignorance (and its study). In: Proctor R, Schiebinger L, (eds) Agnotology: The making and unmaking of ignorance, Stanford, CA, Stanford University Press, 2008, p1–33.

Proctor R, Golden holocaust: origins of the cigarette catastrophe and the case for abolition, Univ of California Press, 2011.

Ramirez-Rubio O, McClean M, Amador J, Brooks D, An epidemic of chronic kidney disease in Central America: an overview, Postgraduate medical journal, 2013, 89, 1049, 123-5.

Rennie D, Fair conduct and fair reporting of clinical trials, Jama, 1999, 282, 18, 1766-8.

Rennie D, When evidence isn't: Trials, drug companies and the FDA, Journal of Law and Policy, 2007, 15, 3, 991–1012.

Rogers T, The Political Economy of Autism, PhD Thesis, Department of Political Economy, School of Social and Political Sciences, Faculty of Arts and Social Sciences, University of Sydney, 2019.

Ronson J, So you've been publicly shamed, Riverhead Books, 2016.

Ross J, Gross C, Krumholz H, Promoting transparency in pharmaceutical industry–sponsored research, American journal of public health, 2012, 102, 1, 72-80.

Ruskin G, Seedy Business: What Big Food is hiding with its slick PR campaign on GMOs, Chapter 6: The agrichemical industry's key front groups and shells aren't trustworthy, US Right To Know, 2015 <file:///C:/Temp/seedybusiness.pdf>

Samp J, Schumock G, Pickard A, Retracted publications in the drug literature: Pharmacotherapy, J Human Pharmacology and Drug Therapy, 2012, 32, 7, 586-95.

Saunders J, Statement from John Saunders, President and CEO of FleishmanHillard, May 28, 2019 <https://fleishmanhillard.com/2019/05/statement-from-john-saunders-president-and-ceo-of-fleishmanhillard/> accessed 26 Jun 21.

Schneider A, McCumber D, An air that kills: How the asbestos poisoning of Libby Montana, uncovered a national scandal, New York, NY, G. P. Putnam's Sons, 2004.

Schroter S, Black N, Evans S, Godlee F, Osorio L, Smith R, What errors do peer reviewers detect, and does training improve their ability to detect them? Journal of the Royal Society of Medicine, 2008, 101, 10, 507–14.

Scranton P, Affidavit, August 3, 2004, in Douglas M. Spann et al., v. Airco Inc. et al., Case No. 3:02-CV-1645WS, U.S. District Court for the Southern District of Mississippi, available at <www.deceitanddenial.org>.

Sherrill R, Why They Call it Politics: A Guide to America's Government, 5th ed., Harcourt Brace Jovanovich, San Diego, 1990, p376.

Sismondo S, Doucet M, Publication ethics and the ghost management of medical publication. Bioethics, 2010, 24, 273-283.

Sismondo S, Ghost-managed Medicine: Big Pharma's Invisible Hands, Mattering Press, 2018.

Smith R, Medical journals are an extension of the marketing arm of pharmaceutical companies, PLoS Medicine, 2005, 2, 5, e138, 364-6

Smith R, Peer review: A flawed process at the heart of science and journals. *Journal of the Royal Society of Medicine*, 2006, 99, 178–182.

Stelfox H, Chua G, O'Rourke K, Detsky A, Conflict of interest in the debate over calcium-channel antagonists, *N Engl J Med*, 1998, 338, 101–106.

Stocking S, How journalists deal with scientific uncertainty', in: Friedman S, Dunwoody S, Rogers C, eds, *Communicating Uncertainty: Media Coverage of New and Controversial Science*, Mahwah, NJ, Lawrence Erlbaum, 1999.

Stocking S, Holstein L, Purveyors of ignorance: Journalists as agents in the social construction of scientific ignorance. In: *Routledge International Handbook of Ignorance Studies*, Routledge, 2015, Chap 12, 105-13.

Stossel J, Case 5:21-cv-07385-VKD Document 1 Filed 09/22/21 John Stossel v. Facebook, Inc. <https://digitalcommons.law.scu.edu/cgi/viewcontent.cgi?article=3543&context=historical> accessed 4 Jan 22.

Strom S, National Biotechnology Panel Faces New Conflict of Interest Questions, *New York Times*, 27 Dec 2016. https://www.nytimes.com/2016/12/27/business/national-academies-biotechnology-conflicts.html?_r=0. Accessed 23 Jun 21.

Stauber J, Rampton S, *Toxic Sludge is Good For You! Lies, Damn Lies and the Public Relations Industry*, Common Courage Press, Monroe, Maine, 1995, p84.

Subpoena, November 4, 2004, in Douglas M. Spann et al., v. Airco Inc. et al., Case No. 3:02-CV-1645WS, U.S. District Court for the Southern District of Mississippi.

Tannenbaum M, Hepler J, Zimmerman R, Saul L, Jacobs S, Wilson K, Albarracín D, Appealing to fear: A meta-analysis of fear appeal effectiveness and theories, *Psychol Bull*, 2015, 141, 6, 1178-204.

Taylor R, Giles J, Cash interests taint drug advice, *Nature*, 2005, 437, 7062, 1070–71.

Thacker P, Covid-19: Researcher blows the whistle on data integrity issues in Pfizer's vaccine trial, *BMJ*, 2 Nov 2021, 375.

UCS: Union of Concerned Scientists, How Dow Chemical Influenced the EPA to Ignore the Scientific Evidence on Chlorpyrifos <https://www.ucsusa.org/ignoring-scientific-evidence-dangerous-pesticide-chlorpyrifos#.W9crNvZFxVI> accessed 2 Mar 19.

UCS: Union of Concerned Scientists, FDA scientists pressured to exclude, alter findings; scientists fear retaliation for voicing safety concerns, 20 Jul 2006.
<http://scienceblogs.com/grrlscientist/2006/07/20/fda-scientists-pressure-toexc/>

Van Nierop L, Rössli M, Egger M, Huss A, Sources of funding in experimental studies of mobile phone use on health: update of systematic review, *J of C.R. Physique* 2010, 11, 622–27.

Vedantam S, Antidepressant makers withhold data on children, *Washington Post*, 29 Jan 2004, A01.

Vom Saal F, Hughes C, An extensive new literature concerning low-dose effects of bisphenol A shows the need for a new risk assessment, *Environmental Health Perspectives* 2005, 113, 8, 926–33.

Wadman M, GM advisory panel is slanted, say critics, *Nature*, 1999, 399, 6731, 7.

Wagner J, Under the dome: hog study raises legal hackles, *The (Raleigh, NC) News & Observer*, 11 May 1999, A3.

Wagner W, Michaels D, Equal treatment for regulatory science: extending the controls governing the quality of public research to private research, *American journal of law & medicine*, 2004, 30, 2-3, 119-54.

Weaver D, Willnat L, eds, *The Global Journalist in the 21st Century*, New York, NY: Routledge, 2012.

Weaver D, Wilhoit G, *The American Journalist in the 1990s: U.S. News People at the End of an Era*, Mahwah, NJ: Earlbaum, 1996. (see also Weaver D, Beam R, Brownlee B, Voakes P, Wilhoit G, *The American Journalist in the 21st Century: U.S. News People at the Dawn of a New Millennium*, Mahwah, NJ: Earlbaum, 5th edn 2006).

Weinel M, Expertise and inauthentic scientific controversies: what you need to know to judge the authenticity of policy-relevant scientific controversies, in: Goodwin J, ed, *Between Scientists and Citizens*, Ames, IA, Great Plains Society for the Study of Argumentation, 2012.

Weinstein B, Ciszek D, The reserve-capacity hypothesis: evolutionary origins and modern implications of the trade-off between tumor-suppression and tissue-repair, *Experimental gerontology*, 2002, 37, 5, 615-27.

West v Johnson & Johnson Prods. Inc., 220 Cal. Rptr.437, 445 (Cal. Ct App.1985): referenced in (Wagner 2004).

Wiener J, Cancer, Chemicals and History, *The Nation*, 20 Jan 2005. www.thenation.com/article/cancer-chemicals-and-history accessed 8 Jan 21.

Willman D, How a new policy led to seven deadly drugs. *Los Angeles Times*, 20 Dec 2000. <https://www.latimes.com/nation/la-122001fda-story.html> accessed 5 Jan 2021.

Willman D, Records of payments to NIH staff sought, *Los Angeles Times*, 9 Dec 2003 <https://www.latimes.com/archives/la-xpm-2003-dec-09-na-nih9-story.html> accessed 5 Jan 21.

Wilson T, Brekke N, Mental contamination and mental correction: Unwanted influences on judgments and evaluations, *Psychological Bulletin*, 1994, 116, 117–42.

Wilson W, Scientific regress. *First Things*, May 2016. Retrieved from: <https://www.firstthings.com/article/2016/05/scientific-regress>

Wing S, Wolf S, Intensive livestock operations, health and quality of life among Eastern North Carolina residents, *Environmental Health Perspectives*, 2000, 108, 3, 233–8

Wing S, Social responsibility and research ethics in community-driven studies of industrialized hog production, *Environmental Health Perspectives*, 2002, 110, 5, 437–44.

Witorsch R, Low-Dose In Utero Effects of Xenoestrogens in Mice and their Relevance to Humans: An Analytical Review of the Literature, *Food and Chemical Toxicology*, 2002, 40, 905–12.

APPENDIX 1: GENERAL ANALYSIS OF A PUBLISHED STUDY CONSIDERING THE CHEMICALLY SENSITIVE PORTION OF THE POPULATION

A study funded by industry in 2000 (Fiedler) was one of the first epidemiological studies on a chemical product to consider the sensitive portion of the population. It illustrates how choosing the study structure and restricting end points can provide a favourable outcome for its petroleum industry major funder: Lyondell Petroleum. It was then used as a reference to counter claims of increased health effects when the petrol/gasoline formulation was changed in the US.

Prior to the change in the US Clean Air Act in 1990 which required prescribed oxygenation levels in petrol/gasoline, methyl tertiary butyl ether (MTBE) had been used as an octane enhancer, however after the Act was changed the MTBE content was increased from 11% to 15%. After this there were some reports of health problems that were attributed to this change (Moolenaar 1994, White 1995, HEI 1996).

The study by N Fiedler et.al. (2000) was designed as an exposure study to “compare the symptoms, psychophysiologic reactivity, and neurobehavioral performance of persons who reported sensitivity to MTBE and controls in response to four controlled exposure conditions: clean air, gasoline, gasoline with 11% MTBE, and gasoline with 15% MTBE” (Fiedler 2000p753). To their credit, they used two control groups, although too small in number to be significant, a “self reported sensitivities” (SRS) group of 12 and a group of 19 controls who did not have any sensitivities. For the SRS group, 800 introductory letters had been sent to interested people responding to advertisements. From gasoline exposure these people had experienced, combinations of headache, cough, nausea, daytime sleepiness, burning sensation in nose or mouth, losing balance or dizziness, difficulty concentrating. These were then filtered down to a group of 12. The control group, from 287 applicants, were also filtered the same as the SRS group as below:

...individuals with any of the following medical conditions were excluded from participation: neurologic disease or history of brain injury, significant exposure to neurotoxicants, stroke or cardiovascular disease, serious pulmonary disease (e.g., asthma), liver or kidney disease, serious gastrointestinal disorders, nasal polyps, nasal

surgery, sinus disease, chronic fatigue syndrome, multiple chemical sensitivities, or major psychiatric disorders including psychoses, bipolar disorder, alcoholism, or drug abuse. Smokers and pregnant or lactating women were also excluded, as were individuals taking beta and alpha blockers, anxiolytics, antidepressants, and steroids (Fiedler 2000p754).

The testing was done in a controlled environmental facility with blood levels of MTBE and one of its metabolites, tertiary butyl alcohol (TBA), measured before and after each exposure of 15 minutes. The exposure tests were separated by approximately one week. A symptom questionnaire was filled in immediately before and after each double blind exposure test. During the exposure the participant was occupied by

...a computer monitor, where a vigilance task randomly displayed bars on the screen and a tone sounded occasionally to maintain the subject's attention. The subject was instructed to keep a mental count of the bars and then report the total number when asked at the end of the experimental session (Fiedler 2000p757).

The study concluded:

Despite increased symptoms in response to gasoline with 15% MTBE, SRSs did not show any concomitant decrements in simulated driving performance tests, and did not manifest any significant psychophysiological responses with exposure. Furthermore, the present study did not support a dose response to MTBE exposure nor the specific symptoms associated with MTBE that were suggested in previous epidemiologic studies (Fiedler 2000p762).

So essentially the study pointed out that even for the sensitive portion of the population, the increase from 11% to 15% MTBE did not present increased driving risks nor significant psychophysiological effects to counter the prior studies which attributed health effects.

There are many shortcomings of the study in coming to such a conclusion as well as the incorrect perceptions that would be marketed to the public. Many of these shortcomings were

referenced by the authors in their discussion section as if they were quite aware of them in the extrapolation they had done to achieve a favourable conclusion for their sponsor.

- People with MCS were excluded from the SRS group. Some people with MCS can have very quick severe reactions to exposures, even within minutes from exposures as was the case here which showed mild reactions. Having said this, it would have been dangerous, even life threatening, to have exposed people with MCS to these fumes so from this viewpoint it is understandable to exclude them.
- Further to the above, sensitive people in general can have varied reaction times up to hours or days after, especially if they encounter another environmental exposure shortly after, such as a lady wearing strong perfume in a car/lift/small room. An exposure to just one chemical is rare today.
- A comment was made that “neither SRSs nor controls were able to distinguish whether MTBE was present at 11% or 15%; and a majority of both groups thought MTBE was present when they were exposed to gasoline only” (Fiedler 2000p761). This is not surprising as most people would not know what MTBE vapour smelled like on its own let alone mixed with the many components of gasoline. For the study to then pick up more health effects from the 15% MTBE than the 11% content by self-reporting is counter to the safety of the higher content gasoline. This is first admitted in the first part of the conclusion but is then glossed over by the last part of the conclusion as quoted above.
- MTBE has become increasingly present in the environment and it is pertinent to note that it was present along with one of its breakdown products tertiary butyl alcohol (TBA), in the blood of all participants before being exposed in the controlled environment for the tests. This should have raised a concern due to its significant toxicity from inhalation and links with cancer (Franklin 2001). A mention was made of this in the discussion however was dismissed as simply “suggesting a heightened sensitivity”.
- Other than the blood tests for the two chemicals of concern, there were no other pathology results reported.
- MTBE breaks down differently at atmospheric conditions such as filling the fuel tank, and after combustion. The study considered one of the breakdown products at

atmospheric conditions, tertiary butyl alcohol (TBA), but did not measure the other major breakdown product: formaldehyde from oxidation of evaporative emissions or tailpipe emissions of unburned fuel. The addition of MTBE to fuels has increased the formaldehyde warm motor emissions from tailpipes by 12 to 13% (Burns 1995, Kirchstetter 1999). This by-product therefore becomes important for a driver in busy traffic and is well known for its high toxicity. No tailpipe gas compositions were tested for.

- There was no comment that compared the exposure concentrations used in the experiments to allowable weighted average or maximum ceiling levels. The concentrations of the exposures were not determined which is unusual for a study based on exposures to chemicals.
- It is suggested that the SRS group did not contain people that were very chemically sensitive. Although the study clearly excluded those with MCS, quite a significant percentage of the population either are not aware of MCS as a health condition or are in the broad group that is in the transition between MCS and a healthy person. The lack of sensitivity is shown by the following statement in the study. A person close to having a condition of MCS even after a few minutes of exposure to gasoline would experience very severe, even life threatening conditions.

Despite increased symptoms during exposure to gasoline with 15% MTBE, SRSs were not impaired relative to controls in overall performance of a driving simulation task, nor did they show significant psychophysiologic responses early or late in the exposure period. Thus, controlled exposure to gasoline with 15% MTBE, while producing more symptoms, did not cause impaired performance or psychophysiologic changes.

The above statement indicates that their selection process had been toward the least sensitised end of the spectrum.

- Further to the above point, and to their credit, the authors acknowledge “the understandable reticence of affected individuals to undergo exposure to the

chemical of concern” and later, “those most sensitive to MTBE may have been least likely to volunteer for an exposure study. This self-selection bias could partially explain the present findings”. This is the pertinent point that anyone with chemical sensitivities would avoid such exposures and never have applied to be a test subject in the first place.

- Health effect questionnaires were completed immediately after each test and the computerised assessment of the psychophysiological aspects for driving capability were done during the exposures. As one may drive for one to two hours at a time, any effects at another later time would have been missed.
- Further to the above point, if the study simulates a person filling their car with fuel, this is far removed from that person then getting in their car and driving in heavy traffic and being exposed to the combustion products if their windows are down or the air conditioner draws in air from the outside (not recirculated). Yet the study conclusion and the abstract are written as being more generalised giving the impression that the 15% MTBE mixture does not in general, and for sensitive people, have detrimental effects. The conclusion and abstract conflict with the authors’ admission at the end of their discussion:

Finally, it is also possible that use of longer exposure durations or exposure conditions that reflect ongoing exposure while driving may have shown greater effects on performance. For example, anecdotal reports indicate that SRS subjects reported symptom exacerbation while driving in traffic where exhaust emissions are prevalent. The latter could not have been completed in a CEF study because of the high levels of carbon monoxide in the exhaust and the complex composition of exhaust in the atmosphere, (e.g., fresh versus aged exhaust that includes particles and gases).

- The comment in the discussion section: “Although SRSs also reported more symptoms than controls when exposed to gasoline with 15% MTBE relative to gasoline alone, this difference was not statistically significant”. This is most part irrelevant to the sensitive population as their heightened sensitivity can be triggered

by a combination of chemicals such as in gasoline. No clear tabulation of the reported symptoms was presented.

This industry funded study was one of the first epidemiological studies on a chemical product to consider the sensitive portion of the population and for this it is commendable. It illustrates an impractical and ethical difficulty in trying to include a truly sensitive group of chemically sensitive people. From the industry-funded perspective it shows a tailored study structure, restricted results, and use of endpoints to provide a favourable outcome for its funder. Its conclusion is surprising considering the discussion section in the paper, which shows that the scientists involved were aware of the study shortcomings.

While being an underpowered epidemiological study with many shortcomings, the study, due to its abstract and conclusion, was used as a reference to counter claims of increased health effects when the petrol/gasoline formulation was changed in the US.

References

Burns V, Rapp L, Koehl W, Benson J, Hochhauser A, Knepper J, Leppard W, Painter L, Reuter R, Rippon B, Rutherford J, Gasoline Reformulation and Vehicle Technology Effects on Emissions-Auto/Oil Air Quality Improvement Research Program, SAE transactions, 1 Jan 1995, 1817-39.

Franklin P, Koshland C, Lucas D, Sawyer R, Evaluation of combustion by-products of MTBE as a component of reformulated gasoline, Chemosphere, 2001,42, 5–7, 861-72.

Fiedler N, Kelly-McNeil K, Mohr S, Lehrer P, Opiekun RE, Lee C, Wainman T, Hamer R, Weisel C, Edelberg R, Liroy PJ. Controlled human exposure to methyl tertiary butyl ether in gasoline: symptoms, psychophysiologic and neurobehavioral responses of self-reported sensitive persons, Env health persp, 2000, 108, 8, 753-63.

HEI: Health Effects Institute, The potential health effects of oxygenates added to gasoline. A review of the current literature, Topsfield, MA, 1996.

Kirchstetter T, Singer B, Harley R, Kendall G, Traverse M, Impact of California reformulated gasoline on motor vehicle emissions, 1, Mass emission rates, Env Sc & Tech, 1999, 33, 2, 318-28.

Chapter 6

Conclusion

This thesis has focused on the area of chemical and radiation exposures from industrial and commercial products. As a broad area of study, it was not possible to define or analyse all environmental influences let alone all the mechanisms involved. This thesis showed that the modern living environment subjects everyone to millions of non-natural, manufactured chemicals. Although orthodoxy regards many exposures as low level, they can still have impacts as both continual exposures and bioaccumulation over long periods of time magnify health effects. Since there are myriad real-life exposures and stressors, studying each environmental exposure separately has little real-world relevance. The complicit medical system, influenced by the ignorance manufactured by the pharmaceutical/chemical industry, has become a tool to promote public ignorance around these issues.

Personal, deliberate environmental exposures from diet, alcohol, smoking, recreational drugs, etc. have not been considered in this thesis. Instead, exposures about which the public is generally ignorant, or where they have no choice in their exposure, have been the focus.

The condition of multiple chemical sensitivity (MCS) was used in this thesis as a key health condition showing health effects from environmental exposure. No attempt was made to undertake specific analyses of events or occasions of divergent views in respect to MCS sufferers; these are available in the references cited. Consideration of the variation in medical paradigms over time was also not pursued, because the paradigmatic approach to chemical sensitivities (as promoted by industry) has been much the same over the last century. Further, instead of substantiating the argument of the thesis by extrapolating from a small number of detailed analyses, extensive evidence from the literature was used to support the work.

SUMMARY OF CHAPTERS

Chapter 2 illustrated the considerable evidence showing links between environmental exposures and human health conditions, especially chronic diseases. In contrast to an orthodox approach, the chapter considered the effects of low concentrations of xenochemicals and radiation in the environment, rather than higher levels like those allowed in the workplace. The chapter found that many published studies indicating the importance of exposures have been relatively neglected in scientific circles, where many research findings are ignored or dismissed.

The orthodox medical and health system has not explained the causes of many chronic health conditions, let alone their increasing incidence. At best, it picks up the symptoms when the body is heading down the track of a chronic health condition, which usually initiates a pharmaceutical treatment of such symptoms. Root causes from exposure to industrial products, technology or pollution releases can be linked to many chronic conditions. These effects, however, are usually not short term, but medium to long term (e.g., cancers can have a lead up period from initiation, of up to 20 years).

Yet the public remains generally ignorant of environmental effects on health. It's almost blind faith in the orthodox regulatory and medical systems is the main contributor to such ignorance. With the health -- or rather the sickness -- industry projecting to the public that uncompromising science-based approaches are being employed, that ignorance is reinforced. This also applies to the practitioners of orthodox treatments themselves. The latest trend is to apply evidenced-based approaches or a meta-analysis of published studies, yet most practitioners have little time and awareness to recognise industry-backed studies that can be designed to negate independent studies that find environmental effects. It was shown that an evidence-based or meta-analysis approach needs to assess shortcomings and inconsistencies rather than simply accepting a study's conclusions. Furthermore, the evidence-based approach has stifled doctors' ability to use what they themselves can learn through observation and experience.

In researching the environmental effects on health it becomes clear that industrial products, pollution and technology are prime contributors. It is ingrained in society that everyone benefits from technological development, yet the perceived higher living standards come at a

great price long term. The development of industrial products and technology has the main aim of making money: a company's focus is the bottom line of the balance sheet, providing an incentive to dismiss any perceived health effects as insignificant or low risk. No new product development within industry wants to be bogged down in years of studies around possible health effects.

The beginning of chapter 2 offered examples of many technological developments which have led to unexpected environmental and health problems or disasters. In all those development "advances", the regulatory authorities did not take actions to prevent such problems, but simply assessed and "controlled" the perceived risks to reduce the exposures of each individual chemical as if it was the only focus. The disasters resulting from the introduction of these technologies were not the risks that regulators, policy makers and health authorities had originally identified. The originally considered risks related to the perceived hazard to be controlled to reduce exposure, or to a complicated calculation of health or economic risks. The general lack of understanding of the consequences of many technological developments outpaced the perceived advantages of the technology and resulted in the public living in a constant state of risk. The few studies that were originally performed served to legitimate the new technologies and ultimately became incapable of ensuring public safety. These studies were also done in isolation, then their results were applied to hundreds of thousands of xenochemicals. There are now established background levels of many chemicals such as dichlorodiphenyltrichloroethane (DDT), p,p'-dichlorodiphenyldichloroethylene (DDE), bisphenol A (BPA), glyphosate and poly-fluoroalkyl substances (PFAS), in locations from Mt Everest to the deepest parts of the ocean.

A significant health condition caused by environmental exposures exists, which by some estimates affects up to 27% of the population: multiple chemical sensitivity (MCS). This condition was explored in chapter 3. Many MCS sufferers can feel an immediate health effect from exposures: an exposure that would not affect most people – e.g., to perfumes --sees mild to debilitating effects in MCS sufferers. The illness is clearly epigenetic as these people had usually led normal lives until, through repeated low-level exposures or one significant exposure, their body systems suddenly left them chemically sensitive. Sufferers establish clear links between their condition and many environmental exposures, realising that by avoiding such exposures they can achieve improved health. It is not simply an academic argument

requiring many studies to establish cause and effect, rather one of the observation, consistency and experience of the patients themselves. MCS is a key illness that this thesis has used to illustrate environmental effects on human health.

Since MCS sufferers react to different exposures in different ways and with different intensities, and possibly in multiple organs, the complexity and absence of clear pathology make it complex to establish diagnostic criteria. Furthermore, the overlaps of this condition with others such as chronic fatigue syndrome (CFS), chronic inflammatory response syndrome (CIRS), or electromagnetic hypersensitivity syndrome (EHS) sees orthodox medicine poorly equipped to deal with such an illness. Even concepts of individual sensitisation and environmental triggering are not in the vocabulary of most doctors. As these aspects are different to conventional diseases, sufferers cannot be “normalised” and the problem is perceived to be with the individual rather than the environment they are exposed to. Since MCS cannot fit into orthodox models of illness and standard diagnostic criteria, it is usually written off with a psychological diagnosis. Strangely though, psychological conditions do not usually have clinical signs for diagnosis either. The orthodox treatment of these conditions usually involves the prescription of low-level xenochemicals, in the form of medications, although this occurs in these people’s cases due to the non-belief that low-level xenochemicals can have health effects.

Added to orthodox medicine’s lack of recognition of MCS as a legitimate illness, is the industry desire to discredit it. Recognition of an illness that clearly shows health effects from industrial products and technology is not welcome. Industry produces ignorance on MCS that in turn, aided by a neoliberal approach, influences governments and society.

It is a human trait for a majority to trivialise a problem experienced by a minority: the victims are blamed for their problem and metaphorically “cast out”. This has occurred with asthma, for example, failing to make any link with the environmental exposures possibly having subclinical effects on many other people, e.g., loading up their immune system to suppress similar effects. Childhood asthma has also substantially increased, increasing to one in three over the last four decades, but it continues to be regarded as a problem for this growing minority only. However, the links with effects from environmental influences are becoming more extensive. One only needs to consider the myriad of other allergic reactions to food contents, air pollutants, etc.,

yet they are not linked to a logic that could indicate that environmental influences may also be an initiator of the increase in incidence for many of other unexplained diseases. In this vein, MCS may be a key, not for recognising the condition itself as a group of genuinely unhealthy people, but that the “normal” environment itself is unhealthy and that MCS is simply a condition, a canary in the mine, that signals danger.

In considering epigenetics and non-monotonic dose responses to endocrine disrupting chemicals which can affect future generations, serious research should be conducted into MCS and environmental effects in general, as a matter of utmost urgency. But to understand why there is no alarm, chapter 4 considered some types of ignorance on these concerns fostered and generated by industry.

That chapter illustrated how “undone science” was used by industry to foster various forms of ignorance in many areas. Industry focuses on the desired areas of knowledge that benefit their products. Undone science is perspectival however, due to the inherent differences over the importance of getting such research done. It can also represent a structured absence due to social inequality which determines which areas of research are to be funded. It can also later be used to industry’s advantage as a delaying tactic when undesirable aspects of their products are highlighted: a common argument is that insufficient studies have been done to prove any detrimental effects. It can take many years of research to accumulate proof sufficient to satisfy companies.

Chapter 4 showed that when a company realises, from its own research or evaluations, that there will be health consequences of their new development, this usually creates an incentive to manufacture ignorance. The net result of such actions is the creation of ignorance for the general public as well as regulatory authorities. This effectively keeps the products on the market. Detrimental product-study results are brought into doubt by highlighting contrary decisions or results through government committees, think tanks, and all types of media including scientific journals. Chapter 5 then considered how knowledge can be manipulated showing how the industry use of doubt makes use of epistemological scepticism of scientific activity and research results. It was also shown that a common approach of the medical and health orthodoxy is blaming the victims.

The regulatory practice of extrapolating data using rationalised selective ignorance was illustrated, e.g., in the US EPA's analyses of soil contamination post-Hurricane-Katrina. This highlighted a common practice of making gross assumptions in risk assessments of contaminated sites, due to undone science. The assumptions effectively cannot be seen by the public once the reports are issued.

Introduced in this thesis was a reversible form of ignorance that can be seen in many situations. Termed "aggregated ignorance", it was seen from one perspective as a tool for the progressive dismissal of many chemical exposures. Each xenochemical is considered on an individual basis, ignoring combined effects, potentiation and synergism in the real world. Furthermore, health effects may be selectively chosen for study, ignoring hundreds of other effects such as endocrine-system disruption, may be considered only. Each xenochemical can then be progressively dismissed as causing any significant effects. In the alternative perspective, aggregated ignorance combines severe health effects with negligible health effects in an averaging effect. This avoids revealing the effects of exposure to specific chemicals, individually or in groups, that may be present in certain geographical locations, population groups, etc. This dilution process can then show that exposures are not a concern.

It is not difficult to argue that a significant percentage of the population has been, and is being, severely affected by various environmental exposures. This can occur gradually over the long term, or occur over a short period (as with chemical spills/disasters). While various forms of ignorance originating from industry could explain how much has been initially allowed to occur before a disaster or major problem happens, there must be people involved in the industry and government regulators who can see the public potentially or gradually being affected. Another form of ignorance was therefore considered: criminal ignorance. The morality of employees in organisations involved in the deliberate generation of ignorance has been compared to delinquency. Furthermore, it was shown that powerful organisations can evade moral and legal reactions. Such power and influence can shape the actions of supposed perpetrators, influence social reactions, evade or censor known or growing public health problems, or, by manufactured ignorance, reframe the morality of the actions themselves. State denial was also covered, where the public representatives of states try to refract, reframe and hide knowledge of illegal and detrimental actions.

Following chapter 4, discussing the various forms of ignorance employed by industry, this thesis then considered how knowledge was manipulated. A funding effect was demonstrated on the outcome of scientific studies, which then affects the outcomes of evidence-based medicine and meta-analyses that are considered the cornerstones of the scientific method. The peer-review process was found to be ineffective, particularly in journal articles and journals were unwilling to issue retractions for articles they know were incorrect. The use of shills or groups of experts or citizens secretly associated with industry to influence the public was also discussed, as was the use of public relations firms to create grassroots populist approaches and manipulate media.

Smear campaigns, another common strategy, have been applied to prominent people who may speak up against an industry interest, which can range through to legal actions.

Conflicts of interest were also demonstrated in the US regulatory bodies, politicians, medical students, doctors and medical associations.

From this chapter summary, it can be seen that there is ample evidence to substantiate that:

Environmental exposures can have significant effects on people's health but industry, via undone science and the generation of ignorance, suppresses much potential knowledge about these effects. The orthodox medical system and government contribute to, or do little to oppose, this process.

LIMITATIONS

A limitation to this research was that it concentrated predominantly on the situation in the United States, due to the largest chemical and pharmaceutical companies being based there. Thus their effects were more pronounced there. However many other countries, like Australia, appear to have simply fallen in line with the American example. This is evidenced by the same products being allowed to be sold in those countries despite sometimes alarming concerns.

It is unfortunate that through studying undone science and the generation of ignorance, the cracks in generally accepted models of the scientific approach have become more pronounced. These include the distortion of scientific studies via the funding source, the ineffectiveness of the peer review and journal publishing process (especially on emerging diseases) and the

resulting, distorted evidenced-based medicine approach which uses those published articles at face value. All study approaches considering the environmental effects on health that were found in this research had been influenced by industry.

A thesis about undone science and ignorance generation is itself based on current knowledge and known unknowns. Despite my extensive survey of research, if there are unknown unknowns then my assessment will become subject to these eventually becoming known. For example, the condition of MCS has been explained as not just one consistent condition but a group of similar, environmentally related conditions, precipitating in different ways and intensities in each sufferer. There does not appear to be a clear, simple mechanism involved. However it remains possible, with future studies, that some common mechanism may link everything together, for example, one of the millions of viruses, fungi or parasites that are yet to be documented and defined.

FUTURE STUDIES

There are many directions for future studies in this area. During my investigation, the shortcomings of the regulatory system became obvious and one could have proposed that it was not adequately protecting the public: this needs attention as a clear matter of public protection.

Other topics that I examined were shaken baby syndrome and sudden infant death syndrome (SIDS). These tragic conditions are subject to considerable undone science and evidence of ignorance being generated. Psychological conditions such as depression and attention deficit hyperactivity disorder (ADHD) were found to be areas where orthodox (paradigmatic) approaches were present and selective ignorance was being displayed by the health and medical systems. In addition, there are the many examples of everyday products, of which only a few were appeared in this thesis, where the industry's generation of ignorance sees the public blissfully unaware of the health consequences of their use. This extended through to the food produced through modern processes, which link to the direction in which farming methods have headed over the last century. Agriculture is focused only on pest resistance, yield and appearance for selling, with no weighting or focus on the resultant effects on human health.

The consideration of ignorance in its many forms in chapter 4 and the manipulation of knowledge discussed in chapter 5 have helped explain the many facets of the complex topic covered in this thesis. In so doing, the generation of ignorance in our society becomes an important consideration with respect to industry and government actions overall. It becomes a tool to be applied to other social issues such as climate change, vaccine effectiveness, COVID and farming sustainability. If the public were more aware of the concept of the intentional generation of ignorance, general attitudes could change on many issues. However due to the generation of ignorance, the public remains insulated from even questioning many situations since in the first instance they are deceived by the lack of information. Many people, for example, have the utmost respect for a doctor's individual opinion and will not even question it.

As seen from the above, there are almost limitless areas for future research.

IMPLICATIONS AND WAYS FORWARD

The increasing rates of chronic diseases, through to even cancer rates in children do not have any established causes. Yet as shown in chapter 2, there are many environmental effects that can be clear causal contributors. The delayed effects, as well as continuing exposures in subsequent generations, are a serious concern. The high toxic loads and radiation exposure during conception and early pregnancy produces compounding effects for each generation. Endocrine disruption from many environmental effects is particularly concerning; it has implications for reduced fertility and is consistent with declining fertility worldwide.

Big money is being made from sickness. Pharmaceutical companies profit directly from the sickness industry and chemical companies have been shown to be more concerned with selling new products than the health effects that may result from them. It is to industry's advantage to maintain the current trends, and hence to create public ignorance. With the orthodox medical system taught from the start to treat symptoms and to be virtual drug outlets, the medical system is ignorant of their ignorance. The regulatory capture by industry, repeatedly shown throughout this thesis, sees the fox being put in charge of the hen house.

It would be easy to say, as Nishtar (2018) does, that more robust laws and regulations are needed to prevent harm. However this thesis has illustrated how governmental departments have repeatedly disregarded existing regulations and public health protection through industry

capture and employee conflicts of interest. The orthodox health and medical system, fully influenced by the interests of the pharmaceutical industry, is doing little to address this situation.

In his recent book *The Triumph of Doubt: Dark Money and the Science of Deception*, Michaels suggests that scientific studies be paid through or by attorneys who act for a client. Also, that there be full disclosure of conflicts of interest in journals, in public comments on rulemaking proposals, and editorials. Those scientists evaluating health effects from products must be unconflicted and favour transparency over confidential industry data. Furthermore, litigation process discovery documents on safety should be publicly available (Michaels 2020).

If an adequate regulatory system were operating, then – at a bare minimum -- restrictions similar to those proposed by Krinsky (2014p248) should be present. This would disallow:

- (1) chemicals that bioaccumulate in the body;
- (2) synthetic chemicals that attach to hormone receptors;
- (3) synthetic chemicals that leach into food in quantities that are hazardous to test animals;
- (4) synthetic chemicals that interact with important human biochemical pathways;
- (5) synthetic chemicals that cross the placenta and expose the foetus.

Radiation (ionising and non-ionising) restrictions should be in place that provide rights to people not to be exposed rather than allow based on heating body cells (by one degree centigrade) ignoring all other effects. A human right should be in place: a right not to be poisoned or receive radiation.

It is an injustice that companies can avoid their managers, directors and shareholders taking individual accountability. If an individual poisoned or otherwise detrimentally affected the health, or caused the premature death of another person, they would be imprisoned, yet there are so many examples where thousands of people have been detrimentally affected by company products and no-one is individually accountable: it's just a matter of the company paying the fine. This is especially prevalent with pharmaceutical drugs and vaccines but applies to many chemical products (see discussion in chapter 4 on compensation cases from Roundup exposure and localised disasters). Yet all decisions for company actions are made by individuals within the company's management. Any affected people are left to carry out the undone science to prove that the chemical exposures caused specific effects in a court of law.

If such a human right not to be poisoned or receive radiation was recognised in law, and more accountability was legally required of company management, then quite a different society would evolve.

It was mentioned in chapter 4 that it is not only a case of imparting knowledge to the public to awaken them to environmental effects, but that the neutralisations and normalisations surrounding the potentially unethical actions of those in power need ceremonial and collective denouncement. Even with climate change, there has been a deliberate lack of acknowledgement and action by those in power, despite public awareness of the issue. The production of ignorance, while being a main tool, may merely be one of many tactics applied by those in power over time. Once the extent of environmental contamination, background levels and their health effects are realised and finally acknowledged, will they say, as seems to be the case with climate change, that the public must simply 'live with it'?

The most promising driver of a paradigm shift on the issue of environmental effects on health is the public's gradual awareness of the situation. Change can be initiated by consumers sending economic incentives for industry to adopt non-toxic products and processes, zero waste, green chemistry, cradle to cradle production, product stewardship, and for the public searching for knowledgeable practitioners, etc.

Rather than the public needing to understand the technicalities of how environmental exposures affect health, they can simply be aware of the concepts of undone science and the creation of ignorance. This is far easier for the public to grasp and to begin to realise how the present situation has occurred and is maintained. Members of the public are unlikely to be interested in the complicated technical explanations of how the biological effects of various technologies can be dangerous. I have found when trying to talk about this subject that most people glaze over after even the first few technical terms. I hope that introducing the basic concepts of undone science and ignorance, with simple examples, will help people more easily initiate questioning and be interested in looking further for another side of the story.

REFERENCES

Krimsky S, Low Dose Toxicology: Narratives from the science - transcience interface. In: Powerless Science? Science and Politics in a Toxic World, Eds Boudia S, Jas N, Berghahn, New York, 2014, Chap 11, 234-53.

Michaels D, *The triumph of doubt: dark money and the science of deception*, Oxford, UK: Oxford University Press, 2020, 344pp.

Nishtar S, Niinistö S, Sirisena M, Vázquez T, Skvortsova V, Rubinstein A, Mogae F, Mattila P, Seyyed Hassan G, Kariuki S, José N, Adewole I, Adboulaye D, Kim Y, Saia Ma'u Piukala, et al., Time to deliver: report of the WHO Independent high-level commission on NCDs, *Lancet*, 2018, 392, 10143, 245-52.