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DISEASE AND DEVELOPMENT

Patterns of Chronic Disease and Industrial Production Offer Clues to Making Development Sustainable

Devra Lee Davis
and Hillary J. Stainthorpe

Public health is a negative. When it is at its best, nothing happens: there are no epidemics, food and water are safe to consume, the citizens are well informed regarding personal habits that affect their health, children are immunized, the air is breathable, factories obey workers safety standards, there is little class-based disparity in disease or life expectancy, and few members of the citizenry go untreated....

Laurie Garrett, *Betrayal of Trust*

I. Introduction

In most developed countries, the leading causes of death and disability are associated with where and how we live, work, and play. Because the impact of infectious diseases throughout life has generally receded, people are living longer than ever. Although this fact seems promising, cardiovascular incidents, heart disease, respiratory ailments, and cancer kill nearly two million people annually in the United States and leave many more disabled. In many developed countries, tobacco use, alcohol consumption, obesity, and other bad habits remain the most important causes of many chronic diseases and are being addressed by public policies. Despite many advances, increasing rates of cancer, asthma, neurodegenerative diseases, reproductive disorders, and other chronic diseases are occurring in both developed and developing countries.

Despite a number of major improvements in global health conditions over the past several decades, such as broader access to sanitation, avoidable factors not addressed by the health care system remain major determinants of death and disease. Public health reflects a complex interplay between the socioeconomic system that constrains the quality of life and the health sector. The major sectors determining dis-

ease, death, and injury in any given society lie far beyond the reach of the health care system. These sectors include transportation, household and industrial energy, water and waste, housing, education, agriculture, industry, and the military. The concept of sustainable development provides a rhetorical framework for thinking about the interconnections between various economic sectors and human health.

Sustainable development has been broadly cast as development that meets the needs of the present without compromising the ability of future generations to meet their needs. The term has been defined by Reed as both people-centered, insofar as it aims to improve the quality of human life, and conservation-based, insofar as it is conditioned by the need to respect nature's ability to provide resources and life-supporting services. In this regard, sustainable development means improving the ability of humans to thrive within the carrying capacity of supporting ecosystems. As delimited in a number of United Nations reports, sustainable development means leaving future generations and ecosystems with as many or more options as in the present.¹ The first principle of the Rio Declaration [1993] states that "human beings are at the center of concerns for sustainable development. They are entitled to a healthy and productive life in harmony with nature." These goals clearly cannot be achieved when there is a high prevalence of debilitating illness, when there are growing socioeconomic inequities, and when natural resources are being depleted at a rate incommensurate with their continued use.

Within an economic framework, sustainable development requires that the value of natural resources, including the potential health of human capital, does not decline over time.² If a given course of development contributes to a notable decline in population health or natural resources, then this development cannot be thought of as sustainable. This definition identifies sustainable development in terms of what it is not, rather than what it is. The implications of the lack of a concrete definition have been noted by Cohen:

One consequence of the ascendancy of sustainable development, which once referred to the radical restructuring of society in accordance with ecological limits, is that the idea no longer has any concrete meaning. Diverse parties now confer on the virtues of environmental sustainability without any shared understanding of what this concept means or how individuals would have to change their production and consumption patterns (if they have to be changed at all) to achieve such a goal....

One astute commentator has remarked that “sustainable development” is a term that everyone likes, but nobody is sure of what it means.³

In an effort to clarify how environment and health affect sustainable development, this essay addresses potentially controllable causes of ill health linked with environmental conditions, in addition to those tied with tobacco, nutrition, and alcohol. Environment, here, is understood to encompass the full array of exogenous factors and impacts on natural resources that can arise with modern development, interfere with normal human functioning, and affect human and planetary well-being. In most developed countries, campaigns to make individuals aware and responsible for their health have made much headway. These individually based programs can easily obscure the need to address broad scale social and environmental factors that also affect health.

Efforts to promote healthier menus, more exercise, and responsibility and accountability for individual health are being engaged at many different levels. At the same time, as the threat of bioterrorism makes chillingly clear, there are some health problems that affect entire populations and can only be addressed by major institutional responses.⁴ Biological warfare and terrorism, like water and air pollutants, pesticides, toxic products in the workplace and home, and global climate change, constitute health risks that cannot be remedied by individuals acting alone, no matter how aware they may be of such hazards.⁵ These issues are what economists refer to as classic externalities, meaning that the problems are caused by the actions of groups of individuals, governments, and businesses. Because of this, even the most conservative philosophy grants that in these cases government actions are required in order to promote health, prevent disease, and ensure sustainable development.⁶

This three-part essay focuses chiefly on the health component linked with toxic chemicals and other aspects of industrial growth and sustainable development. It briefly highlights the individualized approach to health in contrast to the population-based approach. The medical model of disease, with its concentration on individualized treatment and diagnoses, is discussed in the context of recent patterns in the production of some known chemical and environmental hazards. While in many countries much progress has been made with respect to traditional hazards linked with individual behaviors or established workplace risks (such as lead and asbestos), many newer

hazards, such as those tied with endocrine disrupting agents and global climate change, remain unaddressed. In addition, rapidly developing economies often rely on various hazardous materials and the depletion of natural resources, practices that have long been banned in industrial economies. The second part highlights unexplained patterns in male reproductive health, respiratory and neurological disease, asthma, and cancer, in order to bring attention to these puzzling and growing modern ailments.⁷ Finally, the closing section provides strategic advice about public policies that should be developed to ensure that development remains “sustainable,” as that term is continuing to evolve.⁸

II. Medical Model of Disease and the Growth of Industrial Society

With the advent of scientific medicine over the past two centuries, medical practices have focused on diagnosing and curing diseases in individuals, rather than on preventing diseases from arising in populations overall. Some historians of medicine have commented that if most illnesses were not, in fact, self-limiting, medicine would never have achieved the prominence that it did.⁹ The history of infectious disease control in the 19th century provides an important model for understanding how to address major sources of chronic illness. By the middle of that century, long before the advent of antibiotics or the germ theory of disease, deaths from infectious diseases had begun to decline. This advance in health arose largely because of improvements that provided cleaner water and better food, housing, and working conditions. Reductions in the number of people suffering from these diseases stemmed not only from treatments given to individuals, but also from the public health efforts that kept these afflictions from occurring in the first place. Medicine acquired high public standing as the triumph of new individualized interventions, such as anesthesia, sterilization, and immunization, became evident. Still, it is important to note that the development of immunizations and later of antibiotic therapy extended and solidified the public health gains that were originally caused by some basic improvements in sanitation, not by the individually based treatments.¹⁰

In contrast to the practice of clinical medicine, which necessarily focuses on individuals, the public health approach arose from observations that trends in population health occur in patterns reflecting social and economic trends. The field of public health was born at the turn of

the 18th century, when scientists began to notice how health related to environment. In Italy, Bernardo Ramazzini noted that where men worked accounted for many of their health problems. Around the same time, in England, Percival Pott observed that young men who worked as chimney sweeps had much higher rates of a peculiar type of scrotal cancer. By relating health conditions to external factors, great headway was made in reducing or even eliminating many diseases. In the 19th century, for example, regional urban epidemics of cholera were stopped by closing off contaminated water sources in London.¹¹

Today, much public health research concentrates on identifying individual risk factors for diseases, such as those tied to tobacco and alcohol. In this regard, modern epidemiology has in large part lost its connection to the work that originally gave impetus to the field, namely, studies of the patterns and trends in diseases in time and space. In doing so, it has neglected the fact that many determinants of health stem from broad scale factors, such as those affecting the quality of air, water, workplace, household environments, and the global climate.

The apparent success of mass immunization and available antibiotics in controlling certain infectious diseases became the rationale for the creation of more complicated medical technologies, all focusing on individual ailments and remedies. The reasoning behind the belief in individualized treatments is straightforward. Providing treatment to individuals works quite well in many situations, but may not be sufficient when the underlying conditions arise from social and economic factors that cannot be controlled by individual behavior.¹² Thus, a lead poisoned child or a worker who develops solvent toxicity can be treated with drugs that will remove lead or counteract those exposures to some extent. If that child or worker is returned to the environment that provided this toxic exposure, however, then the problem will recur.¹³

As the burden of disease shifts from traditional hazards (such as infectious disease and accidental injury) to modern ailments (such as cancer, asthma, neurological disorders, and heart disease), the individualized medical approach that we so admire becomes less and less functional. These conditions arise from social and economic factors that cannot be controlled by individual behavior and, therefore, individualized treatments are not sufficient.¹⁴ Modern environmental health consequences are the chronic diseases that arise from the sum total of experiences occurring over an individual's lifetime. Inherited

genetic defects play a much smaller role than is widely believed, accounting for fewer than one in ten cases of cancer, for instance. Diet, exercise, smoking, alcohol, workplace, endogenous and exogenous hormones, bacterial and viral contacts, and environmental exposures all interact with genes to affect the chances that any given individual will acquire specific illnesses. Finally, several important health problems are associated with global climate change, and can be confronted only through institutional policies that ensure that development in the future remains sustainable.¹⁵

Of course, there are more cases of cancer and chronic diseases today because more people are living long enough to acquire such diseases. In addition, techniques for detecting such ailments have improved. Yet several studies that adjust for the increased survival of older persons have found that the rate of all new cases of cancer, excluding that of the lung, has grown about 35 percent since 1950. For young men today, rates of cancer are triple that of their grandfathers, while sperm counts are significantly lower. Women born in the 1960s have 50 percent more cancer than did their grandmothers.¹⁶ In light of these trends, the question of the sustainability of current patterns of industrial society looms large.

The tide has turned in regard to the nature of health threats facing the global community. Excepting HIV/AIDs, infectious diseases are no longer the dominant hazard in the world. Even in those regions where infectious diseases are predominant, such as sub-Saharan Africa, the medical model, with its emphasis on the treatment of the diseased individual and his or her symptoms, cannot provide adequate remedies. Widely shared health problems require broad socioeconomic strategies that cut across economic sectors and involve education, poverty alleviation, and development to address the social contexts that give rise to these problems. For HIV, as for most chronic diseases, individualized treatments for most of the health problems discussed later cannot succeed if the underlying socioeconomic conditions that give rise to these problems are not factored in. Our focus must shift from improvements in efficiency of treatment to primary prevention. Primary prevention, in turn, requires a literate population, with sufficient resources to make decisions about how best to reduce toxins and other damaging forces within the environment. Health-based standards for technologies that will be used as societies evolve must play a central role in the planning and execution of economic development. Those technologies that are less toxic and do not deplete natural

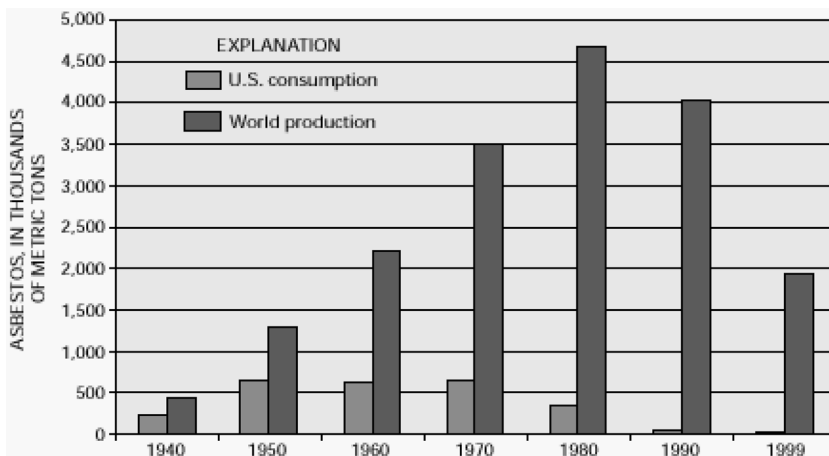
resources or human capital should be accorded a higher priority. The increased incidence of disease across populations must be considered an indicator of non-sustainable development.

III. Patterns of Production of Known Chemical Hazards

Recent studies suggest that exposure to toxins in the air we breathe, the water we drink, and the food we eat cause a variety of adverse health effects, including reproductive, developmental, behavioral, neurological, endocrinological, and immunological problems.¹⁷ Because diseases associated with these exposures are common, it is difficult to discern the relative contribution of such exposures to these ailments. Despite growing evidence that some chemicals are toxic to wildlife and humans, and despite some successes in reducing exposures to a portion of these, the production and industrial use of many suspect chemicals continues, including those linked with deficits in intelligence and development.¹⁸ Continued use of these chemicals is driven by their easy availability, durability, and profitability. We will focus on three examples— asbestos, lead, and arsenic—that illustrate problematic exposures of the modern chemical environment.

Modern use of asbestos grew dramatically during World War II. Initially, this fiber appeared to be a wonder material that successfully solved many difficult problems, such as making the braking systems in automobiles more reliable or insulating homes more effectively. As shown in figure 1, the peak year of asbestos use in the United States was 1973, when approximately 719,000 tons were used for manufacturing friction products, flooring, caulks, gaskets, packings, electrical and heat insulation, plastics, roofing, textiles, and a host of other consumer and commercial products. However, around the same time, health studies that had secretly been conducted for years by industry became public, implicating asbestos as a harmful airborne material. In collusion with officials in the federal government, the industry had suppressed evidence of the hazardous nature of asbestos since the 1930s.¹⁹ Humans who breathe in high levels of asbestos have an increased risk for asbestosis (a painful scarring of lung tissue), lung cancer, and mesothelioma (a rare cancer that affects the lungs, chest, and abdomen). Effects are not limited to highly exposed workers, but have been found in the children of workers,²⁰ other relatives, and those living in close proximity to factories. Based on the public disclosure of these effects and global efforts to reduce the use of this highly persis-

Figure 1: Summary of Asbestos Production from 1940–1999



Source: U.S. Geological Survey Fact Sheet FS-012-01: Asbestos

tent material in many developed countries, asbestos use has dramatically decreased, but it is still used for a few commercial purposes in the U.S. and elsewhere.

Like asbestos, the heavy metal lead has many beneficial physical and chemical characteristics, including corrosion resistance and malleability, which have fueled its widespread use throughout the developing world. Yet lead has long been considered one of the most easily avoidable environmental toxins in the world. It is known to be extremely toxic to the brain, the kidneys, and the reproductive and cardiovascular systems. Exposure to the developing organism can produce permanent neurological damage, while exposure to adults can produce renal damage, along with neuropathological effects.²¹ One of the most environmentally significant uses of lead was its addition to gasoline. In the 1920s, auto manufacturers discovered that adding lead to gasoline significantly increased power.

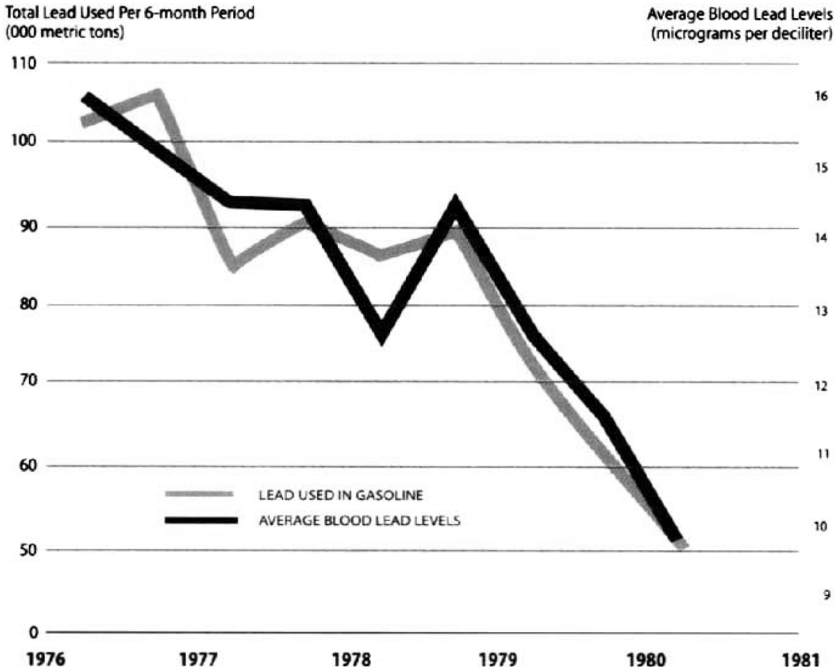
Public health scientists, such as Alice Hamilton, warned at the time that this use of lead would result in massive, widespread exposures with unknown consequences. Their warnings were ignored.²² Researchers conducted studies of workers exposed for a few days to emissions of leaded gasoline. When none of the workers appeared to become ill, the researchers concluded that lead was safe. This was an early instance of looking for acute and short-term toxic effects, when longer term, cumulative, or chronic effects proved much more insidi-

ous and important. While short exposures to lead in air are not lethal, over the longer term lead supplants calcium, is absorbed into the blood, brain, and bone, and causes subtle and permanent neuro-developmental damage. The developing brains of children are at the highest risk from such exposure. Adults who are regularly exposed to lead in the workplace are also especially vulnerable to negative health impacts.

Another disturbing property of lead arises from the fact that once emitted, heavy metals can reside in the environment for hundreds of years. Because lead is highly persistent, its widespread use in the past has created extensive regions where exposures are still well above those recommended by the Centers for Disease Control and Prevention. The widespread introduction of lead into gasoline caused an enormous jump in the amount of free floating lead that people inhale, as well as the amount of lead that would eventually settle in the land and pollute the water supply. The Environmental Protection Agency accelerated the phase-out of leaded gasoline in the mid-1980s, and between 1976 and 1990, average blood lead levels in the U.S. population declined from 14.5 to 2.8 micrograms per deciliter, as figure 2 shows. Unfortunately, there are at least 55 nations that still allow relatively high concentrations of lead in gasoline, i.e., above 0.65g/l.²³ Recent studies have found that lead used in wheel weights can also enter the environment, as they are cast off deteriorating equipment. In some areas, levels reported from this use alone would qualify certain street corners for treatment as hazardous waste sites.²⁴

Arsenic, another heavy metal, has been linked to some of the same human health threats as lead, but it is still commonly used in wood preservatives in both developed and developing countries. In addition, more than fifty million persons in Asia regularly drink water containing toxic levels of arsenic. This has occurred because surface waters became too contaminated from agricultural and industrial activities, so drinking water wells had to be set deeply into aquifers that contain high levels of arsenic. The effects of arsenic range from skin disorders, including skin cancer, to lung cancer, and include a host of neurological and developmental problems. A recent study in Bangladesh investigated the effects that exposure to arsenic in the drinking water has on pregnancy outcomes.²⁵ The study recruited 192 married women (15–49 years), divided them into exposed and unexposed groups, and analyzed whether the exposure affected the pregnancy outcomes in terms of live birth, stillbirth, spontaneous abortion,

Figure 2: Decreases in Blood Lead Values and Amounts of Lead used in Gasoline in the United States, 1976-80
Benefits of Reducing Lead in Gasoline



Source: Valerie M. Thomas, "The Elimination of Lead in Gasoline," *Annual Review of Energy and the Environment* 20 (1995): 307

and preterm birth. The 96 exposed women came from Samta, where 87 percent of the tube wells had an arsenic content greater than 0.05mg/L, and the 96 unexposed women came from Katiarchar, where all of the tube wells had an arsenic content of less than 0.02mg/L. There was no statistically significant difference ($p > 0.05$) for socioeconomic status, age at marriage, or education between the two groups. The study found that rates of spontaneous abortion, stillbirth, and preterm birth were 2.9, 2.24, and 2.54 times higher, respectively, in the exposed group than in the unexposed group. These differences are statistically significant ($p = 0.008$ for spontaneous abortions, $p = 0.046$ for stillbirths, and $p = 0.018$ for preterm births). The range of negative health effects linked with exposure continues to increase. Despite this, public policy actions to remediate or prevent this exposure have yet to be proposed.

The nature of the effects produced by any given toxic exposure will vary with the timing and the amount of exposure involved. Arsenic exposure to young men and women seeking to become parents can interfere with their ability to reproduce. Prenatal arsenic exposure causes low birth weight and premature birth. Exposures later in life have been linked with skin disorders, neurological deficits, and lung cancer. Thus, for many toxic chemicals, the timing of exposure can be even more important than the amount of exposure in determining the biological impact. Safe levels of exposure to chemical hazards should be based on effects of long term exposure to the most susceptible sub-populations, in many cases, children or fetuses.

Reduction of such known chemical hazards by government regulation in developed countries has been in progress for years and has generally been successful. In contrast, regulation in developing nations has been minimal. For instance, asbestos use has remained prevalent in developing countries, where it has a strong market as an inexpensive, durable construction material. The governments of such countries may recognize the hazards associated with the use of such materials, but often lack economically viable alternatives to their use. In other countries, policymakers may be unaware of the lethal legacy of these materials. If sustainable development is to occur, information on health risks must be shared among nations and made available to policymakers and individuals. People's health must not be seen as a trade-off for economic gains. Instead, a long-term balance between health and profits must be sought, as both are essential to the success of future generations. Such a balance may be encouraged by trade regulations that provide incentives for the use of more sustainable materials.

IV. Air Pollution and Human Health

The Industrial Revolution has produced much material benefit, but has also resulted in unprecedented growth in the combustion of fossil fuels. One consequence of this growth is the fact that atmospheric levels of carbon dioxide and other greenhouse gases (GHG) are now higher than at any other time in planetary history during which human habitation has been possible. Increased levels of GHGs are projected to be associated with major changes in global climate, including altered mean temperatures and sea levels, increased extremes in weather, radical shifts in the ecology of infectious diseases, and/or land use patterns.²⁶ The implications of such effects for health are

immense. Incidence of vector-borne diseases, such as malaria and dengue fever, would dramatically increase as the range of the vectors widened, especially in areas lacking the infrastructure to cope with such developments. Environmental refugees, displaced from their homes by rising sea levels, would increase crowding and poverty in remaining cities. The majority of the world population lives in coastal regions, many of which could be lost to rising sea levels. Overcrowding and poverty tend to lead to poor health. Crop production would be altered, yielding changes in the availability of nutritious food.

While debates about energy choices, long-term climate change impacts, and the capacity to adapt to those impacts continue to evolve, there is little doubt that air pollution from current patterns of fossil fuel use for electricity generation, transport, industry, and housing are already sickening or killing millions throughout the world.²⁷ Increasing power generation by conventional fossil fuel combustion further threatens human health and welfare by increasing air pollution. It has been estimated that reducing emissions from older coal-fired power plants in the United States could provide substantial benefits to public health, including the avoidance of 18,700 deaths, three million lost work days, and sixteen million restricted activity days each year.²⁸ By reducing emissions from nine older coal plants in the Midwest, roughly 300 deaths, 2000 respiratory and cardiac hospital admissions, 10,000 asthma attacks, and 400,000 person-days of respiratory symptoms could be avoided each year.²⁹ Globally, the burden of air pollution is already substantial. Deaths from air pollution, including both indoor and outdoor sources, have been ranked as one of the top ten causes of disability by the World Health Organization (WHO).³⁰ In 1995, the WHO estimated that 460,000 avoidable deaths occur annually as a result of suspended particulate matter, largely from outdoor exposures in the urban environment.³¹ In 1997, the WHO and others estimated that air pollution accounts for nearly 700,000 deaths annually and for about eight million avoidable deaths that will occur worldwide by 2020.³²

While the use of fossil fuels provides numerous health and lifestyle benefits to society, efforts to promote cleaner and less carbon-intensive energy have both short- and long-term advantages. One example of the immediate benefits of improved air quality was documented during the Olympic Games held in Atlanta, Georgia in 1996. The impact of reduced air pollution on asthma was derived by comparing the average morbidity rates during the 17 days of the games (19 July to 4

August 1996) with the four-week time periods of the year before and afterwards.³³ When alternative transportation policies during the Games reduced vehicle exhaust and related air pollutants (such as ozone) by about 30 percent, the number of acute asthma attacks and Georgia Medicaid claims fell by 40 percent, and pediatric emergency admissions dropped 19 percent.

Reductions in GHG emissions can similarly reduce associated co-pollutants that affect human health, provided these reductions are based on lowered fossil fuel combustion. In addition, if climate change is avoided as a result of mitigation efforts, then related air quality shifts (such as rising ozone air pollution from higher temperatures) can also be avoided.³⁴ There are now hundreds of reports from both developed and developing countries consistently showing that short- and long-term exposure to current air pollution levels of particulate matter and ozone affect death rates, hospitalizations and medical visits, complications of asthma and bronchitis, days of work lost, restricted activity days, and a variety of measures of lung damage in children and adults.³⁵

A recent joint industry-government and private sector multi-city analysis, conducted for the Health Effects Institute (HEI) by scientists from Johns Hopkins University, found that a daily increase of 20 $\mu\text{g}/\text{m}^3$ in particulate matter (PM_{10}) that can be inhaled increases the death rate by about 1 percent.³⁶ In addition, an HEI reanalysis of two key studies confirmed that a 25 $\mu\text{g}/\text{m}^3$ increase in lifetime average concentration of fine particles ($\text{PM}_{2.5}$) in a city increases the overall total annual death rate by some 15 percent.³⁷ The Ontario Medical Association reported that for every death from air pollution, there are an additional 5.1 hospital admissions, 6.8 emergency room visits, and 24,128 minor illness days. For the year 2000, about 1,900 premature deaths associated with air pollution occurred in Ontario, as well as 47 million minor illness days.³⁸

Several new studies of air pollution also reveal the exquisite sensitivity of the fetus and newly born. Late fetal loss in Sao Paulo increased nearly 20 percent in areas with the highest combined index of air pollution, in contrast to zones that were the cleanest.³⁹ Maternal cord blood analysis showed that carboxyhemoglobin levels were also elevated in mothers living in these more polluted areas, providing a biological marker of air pollutant exposure. Other studies have indicated that the risk of infant death is doubled in some regions, such as the Czech Republic⁴⁰ and Mexico City,⁴¹ when pollution levels have been

highest. A recent report from the United States found that infants born between 1994 and 1996 in Northeastern urban areas, where exposure to air pollution is high, had a significantly lower than average birth weight. Low birth weight is a factor well recognized to place infants at risk for a host of health problems.⁴²

It is clear that children are at enhanced risk from pollution for a number of important reasons. Physiologically, their organ systems continue to develop through their first few years of life. A child's lungs, for example, grow most rapidly in the first two years of life, and continue to grow until the late teen years.⁴³ Developing organs can be extremely sensitive to the toxic effects of pollutants. Children also tend to absorb pollutants more readily than adults, and retain them in the body for longer periods of time. While the average active adult inhales about 10,000–20,000 liters of air per day, a three-year-old child takes in twice the amount of an adult per unit body weight.⁴⁴ That child therefore absorbs double the amount of pollutants for its weight than an adult. In a study of infant deaths in their first month of life and particulate air pollution in the U.S., those living in areas with greater PM10 exposure encounter a 45 percent higher risk of dying from respiratory illness than those living in less polluted areas.⁴⁵

Especially worrisome is the threat of diesel exhaust, one of the most common exposures in the urban environment, as an endocrine disrupter. One study conducted on school children living in Harlem with no known unusual exposures found that three out of every four of them had urinary metabolites, indicating exposure to diesel exhaust.⁴⁶ Under some proposed energy scenarios, diesel transportation can be expected to expand, and with it, the potential for increased exposures to children and young adults who will later become parents.

In addition to the direct hazards of pollution tied with fossil fuels, two major influences on the capacity of nations to achieve sustainable development are technology and terrorism, the combined effects of which have become painfully evident. Technology developed for burning fossil fuels has dramatically broadened humanity's range of possibilities for transportation, industrial production, and meeting basic everyday needs, including cooking and heating. Fossil fuel use to provide centralized power can reduce traditional risks, such as those tied with indoor air pollution associated with the burning of wood, biomass, or other older technologies for cooking and heating homes. However, fossil fuel use has been clearly linked with a host of new health problems as discussed above. In addition, centralized forms of

fossil fuel use and nuclear energy also pose unique opportunities for those determined to subvert modern societies. The potential for sabotage of fuel reserves and the power infrastructure remains great. The public health impact, if any of these threats were to materialize, cannot be gauged easily but is likely to swamp most other imagined scenarios. The fear and terror prompted by these threats are giving rise to renewed pressure to reduce the dependency on conventional energy sources.

For all these reasons, both the Right and Left have renewed interest in reducing dependence upon natural resources from unstable regions of the globe, such as oil production from the Middle East. Global politics, along with basic public health concerns, make it clear that developed nations should plan to reduce their reliance on fossil fuel use. Cleaner energy technologies that are not dependent on fossil fuels, such as solar and wind power, must be made available to developing nations as alternatives to fossil-fuel-based industrial development. They must also be aggressively pursued by developed countries, given the new geopolitical realities.

V. Mitigation Benefits in Four Major Cities

To investigate the immediate health benefits of GHG mitigation measures, Cifuentes and colleagues evaluated the reductions in adverse health effects that might be achievable over the next two decades in Mexico City, Mexico; New York City, U.S.A.; Santiago, Chile; and Sao Paulo, Brazil. These cities have a combined population of 45 million persons.⁴⁷ Researchers developed quantitative estimates of the change in selected health end points that can be projected to occur per million people exposed to a given unit of particulate matter and ozone. Various subpopulations were treated separately, using coefficients derived from local health studies, to take into account differences in response to a given level of pollution due to age or other demographic factors. Based on projected emission and population patterns for the time period 2001 – 2020, researchers calculated potential health benefits of using existing, readily acquirable technologies that would reduce GHG emissions from fossil fuels.

The study found that adoption of GHG mitigation technologies would also reduce particulate matter and ozone ambient concentrations by about 10 percent, thereby avoiding some 64,000 (95% CI: 18,000 to 116,000) premature deaths (including infant deaths), 65,000

(95% CI: 22,000 to 108,000) chronic bronchitis cases, and 37 million (95% CI: 27 to 47 million) person-days of restricted activity or work loss in these four cities alone through 2020.⁴⁸ These findings illustrate that GHG mitigation can provide considerable local air pollution-related public health benefits to countries that choose to abate GHG emissions by reducing fossil fuel combustion. These reductions can be achieved either by increased energy efficiencies or substitution of alternative fuel sources.

The potential public health benefits projected are likely to be conservative due to the exclusion of the impact of other toxic air pollutants (such as benzene or polycyclic aromatics), and because many effects are not yet quantifiable on the basis of available literature. Related impacts on other media, such as water systems, wildlife, and agriculture, are likewise unaccounted for in this study. Even with these limitations, the results suggest that a major public health opportunity is afforded by taking mitigation steps now, rather than waiting for a crisis. In general, the more GHG abatement a country achieves, the more air pollution-related health benefits will accrue. These results should apply throughout the developed and developing world.

VI. Unexplained Trends in Reproductive Abnormalities

With respect to chronic disease patterns that remain unexplained, recent research indicates that over the past several decades, the proportion of male births has declined, incidence of testicular cancer has increased, sperm quality has declined, and male genitourinary birth defects have become more frequent. The causes of these trends are not known. However, it has been suggested that these reproductive abnormalities stem from a common cause affecting early prenatal development.⁴⁹ There is growing concern that these changes in reproductive and metabolic function may be caused by increased exposure to environmental estrogens.⁵⁰ Both the body's own estrogen and those ingested or transformed from exogenous sources can bind with the body's estrogen receptors. The total body burden of hormones can be affected by these combined exposures. Numerous chemicals used in pesticides and industrial processes are known or suspected environmental estrogens.

Elevated levels of maternal circulating estrogens produce negative effects on the developing male fetus. Sexual differentiation occurs during the first trimester of pregnancy, at which time the fetus has a uni-

sex pair of gonads and two sets of ducts, referred to as wolffian and mullerian ducts (mesonephric and paramesonephric ducts). The default developmental pattern appears to produce female offspring through the disappearance of the wolffian ducts and the differentiation of mullerian ducts into oviducts. In the male fetus, the SRY gene on the Y chromosome, assisted by several effector and autosomal genes, induces the fetal gonads to develop into a testis.⁵¹ Sertoli cells in the developing fetal testis produce mullerian inhibiting substance (MIS), which triggers the disappearance of the mullerian ducts. The disappearance of the mullerian ducts allows for the descent of the testis into the scrotum, regulation of the development and function of leydig cells (cells that secrete testosterone, inducing further differentiation of the wolffian ducts into male genitalia), and support for the germ cells that will eventually mature into spermatozoa. Sertoli cells in the developing testis are the weak point in susceptibility to adverse developmental effects of estrogens. Sertoli function and proliferation are controlled by follicle stimulating hormone (FSH), which is regulated through negative feedback by estrogens. Elevated estrogen levels lead to reduced levels of FSH, and in turn to diminished function and multiplication of sertoli cells.⁵² Disruptions during this critical period of sexual differentiation could affect the phenotypic determination of sex, resulting in various degrees of feminization of male offspring as well as subsequent development of offspring.⁵³

While development of sertoli cells occurs only during prenatal and early neonatal development, disruption of their development may have far-reaching negative effects on male reproduction later in life.⁵⁴ Each sertoli cell can support only a finite number of germ cells so reduced numbers will mean less sperm production throughout life. Because sertoli cells also control descent of the testis and development of external male genitalia, impaired function may lead to an increased incidence of cryptorchidism (undescended testis) and hypospadias (displacement of the urethral opening towards the scrotum). Further, because sertoli cells regulate the developmental cascade leading to testosterone production, impaired function may inhibit this production and, therefore, the development along the male physiological pathway. In the absence of properly functioning sertoli cells, a genetic male could potentially develop in utero according to the default female pathway. In later life, those males that have incurred disruption of sertoli development are also likely to have reduced sperm quality and quantity and to be at increased risk for testicular cancer as young

adults.⁵⁵ This risk of increased cancer arises because alterations in functioning can lead to reduced cell repair, alterations in gap junction cell communication, and other distortions of the cell cycle conducive to the expression of malignancy.⁵⁶

Table 1 shows reported increases in several types of male reproductive defects for which explanations are not clear. For instance, analysts in several developed countries have reported increases in cryptorchidism (figure 3) and hypospadias (figure 4). The primary explanation advanced for these trends is that they are artifacts.⁵⁷ The definition of hypospadias may have become more inclusive over the last few decades: deviations from the normal position of the urogenital opening that were formerly considered natural variation may now be considered hypospadias. This hypothesis is consistent with social trends in that doctors today face increased pressure from malpractice suits, and hypospadias can complicate circumcisions. Doctors may be reporting more deviations as hypospadias to protect themselves legally. However, if this hypothesis were true, we would expect an increase in the ratio of mild to severe instances of hypospadias. Many studies have found the opposite effect: that mild hypospadias account for a smaller percentage of overall cases today than 25 years ago.⁵⁸ Cryptorchidism is similarly subject to the effects of changes in diagnosis and reporting in that it has been sought out more aggressively in recent decades due to the apparent relationship between this condition and testicular cancer. Undescended testes are at increased risk of cancer, and early correction of the abnormality may reduce this risk.⁵⁹ The validity of such a reporting artifact as an explanation for increased incidence of cryptorchidism fails to account for the common etiology suspected of cryptorchidism and testicular cancer. If these two conditions share a common cause, then we would expect trends in incidence of the two to coincide. Testicular cancer is also on the rise, suggesting that the trend seen in cryptorchidism is real and not artificial.⁶⁰ Other explanations offered for the increased incidence of congenital male reproductive defects include maternal illness, reduced fertility of parents, maternal hormone treatment during pregnancy, and parental age.⁶¹

The link between such anomalies and general environmental exposures remains a matter of speculation. However, recent molecular biological analyses from a study of adolescent health in the Netherlands provide strong indications that such a link exists.⁶² Defects in testosterone metabolism and testosterone receptors in males with hyospa-

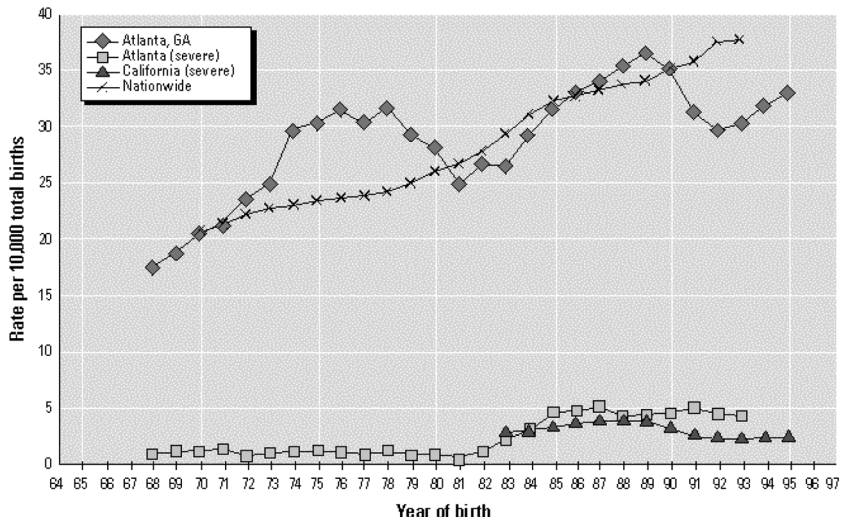
Table 1: English-language Published Reports of Upward Trends in the Prevalence of Hypospadias and Cryptorchidism

Year	Location	Birth years covered	Reference
Hypospadias			
1975	Norway	1967–1973	Bjerkedal and Bakkeiteig (9)
1979	Sweden	1965–1977	Källén and Winberg (10)
1982	Sweden	1965–1979	Källén and Winberg (11)
1985	England and Wales	1964–1983	Matlai and Beral (12)
1985	Hungary	1971–1983	Czeizel (13)
1986	Denmark	1970–1981	Källén et al. (14)
1991	United States	1974–1988	ICBDMS (15)
1991	Strasbourg	1982–1988	ICBDMS (15)
1991	Italy	1981–1988	ICBDMS (15)
1997	United States	1970–1993	Paulozzi et al. (16)
1997	Atlanta	1968–1995	Paulozzi et al. (16)
Cryptorchidism			
1984	England and Wales	1952–1977	Chilvers et al. (17)
1985	England and Wales	1969–1983	Matlai and Beral (12)
1986	England (Oxford)	1950s vs. 1984–1985	JRHCSG (18)
1992	England (Oxford)	1950s vs. 1984–1988	JRHCSG (18)

Abbreviations: ICBDMS, International Clearing house for Birth Defects Monitoring Systems; JRHCSG, John Radcliffe Hospital Cryptorchidism Study Group.

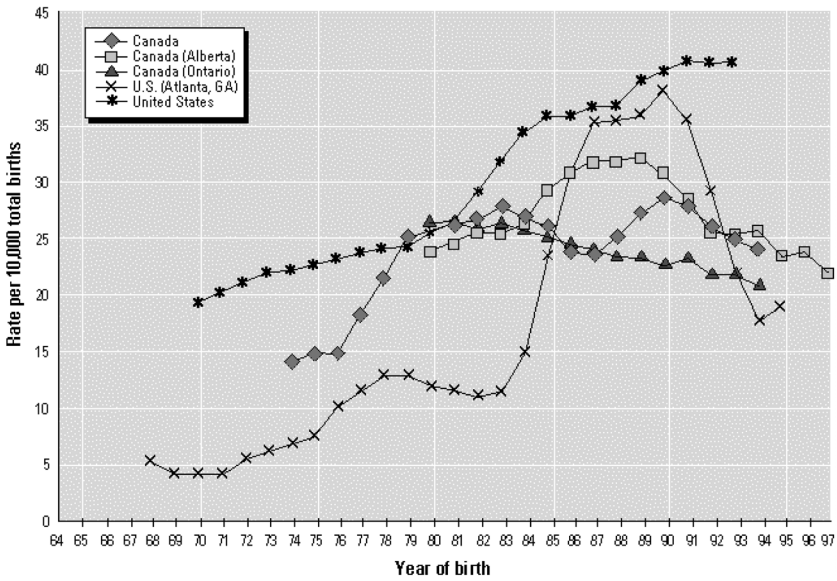
Source: Paulozzi, *Environmental Health Perspectives* 107, no. 4, April 1999

Figure 3: Cryptorchidism Rates by System and Year of Birth (1968-1997), 3-year moving averages, United States and Commonwealth groups



Source: Paulozzi, *Environmental Health Perspectives* 107, no. 4, April 1999

Figure 4: Hypospadias Rates by System and Year of Birth (1968-1995), 3-year moving averages, United States group

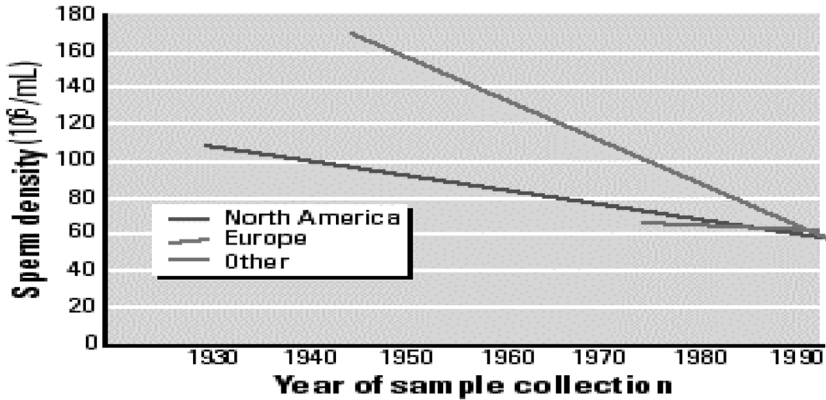


Source: Paulozzi, *Environmental Health Perspectives* 107, no. 4, April 1999

dias suggest that abnormal endocrine factors may influence the development of hypospadias.⁶³ Additionally, increased incidence of hypospadias and cryptorchidism has been noted in offspring of women treated with diethylstilbestrol, an estrogenic drug formerly used under the mistaken notion that it would reduce the risk of spontaneous abortion.⁶⁴

Some studies have found that sperm counts have also declined in many countries, although the extent of this decline is widely debated. Figure 5 shows an aggregated analysis, with European countries having the sharpest decline (more than 50%); those in North America dropping less precipitously (by about 40%); and those in other countries not declining as much. Whether these measurements were obtained under comparable circumstances and whether comparisons can readily be made between countries with respect to sperm density measurements are issues that continue to be debated. What remains clear is that various measures in many countries show drops in sperm quality and density. The reasons are not known at this time, but increased exposure to estrogenic substances could be a factor due to

Figure 5: Regression Model for Mean Sperm Density by Year and Geographic Region, after controlling for proven fertility, abstinence time, age, specimen collection method, method of counting sperm, whether the study was included by Carlsen et al. (1), and interaction of region and study year

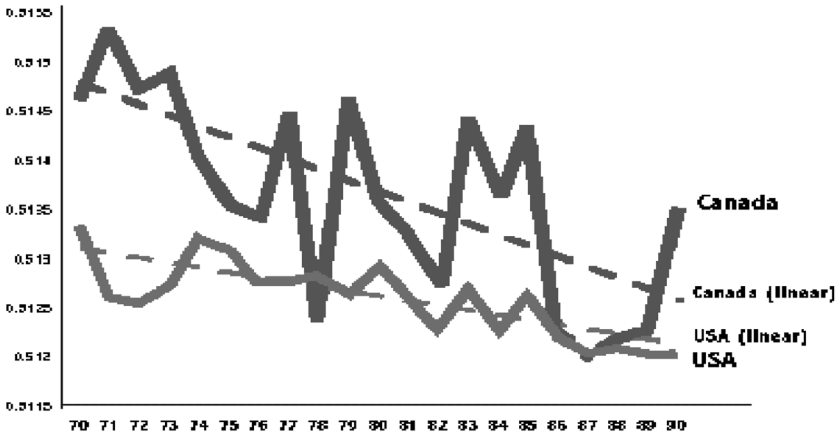


Source: Swan, *Environmental Health Perspectives* 108, no. 10, October 2000

their potential for disruption of male sexual development.

Another measure of reproductive health, which is as controversial as sperm density, is sex ratio at birth. This is typically expressed as the ratio of males to females born, or as a fraction of the total that is male, or male proportion. While illness and age of mother are believed to play some role, the sex of offspring is chiefly determined in mammals by male health. The SRY gene on the male chromosome determines whether the conceived embryo will be male or female. We have previously reported that there is a significant decline in the U.S. and Canada of the birth of boys relative to girls, as illustrated in figure 6.⁶⁵ The reasons are not known. Studies of workers who are highly exposed to some organochlorine compounds, such as the pesticide dibromochloropropane, have significantly fewer baby boys.⁶⁶ In addition, young parents who incurred high exposures to dioxin from a chemical plant explosion in Seveso, Italy, have also been found to have deficits of male births. Of the ten births that occurred to parents most highly exposed for the decade following the explosion, not a single one yielded a male child. The effect persisted, even taking into account marriage outside of the local community, if the father had been exposed during late adolescence. Ongoing work suggests that this downward trend is continuing in the U.S. and a number of other coun-

Figure 6: Proportion of Male Births in U.S. and Canada, 1970-1990



tries. Factors known to affect sex ratio include race, parental age, birth order, the timing of conception, maternal illness, reduced parental fertility, and maternal hormone treatment. Yet none of these factors can explain the steady decline in sex ratio over the past thirty years.⁶⁷ It is interesting to note that several of these factors are also known to influence rates of hypospadias, lending support to the notion of a common etiology. A conclusive explanation of the cause of the declining sex ratio has not yet been put forth.

As reported in *Environmental Health Perspectives*, June 2001, by Axelrod and his colleagues, animal studies of the sensitivity of the developing organism have repeatedly confirmed that susceptibility to toxicants depends on the rate of cell division going on at the time of exposure. The synthetic estrogen diethylstilbestrol (DES) produces structural defects in the seminiferous tubule structure of male offspring, following maternal exposures that take place during critical phases of organogenesis.⁶⁸ A single dose of exposure to 17-B estradiol early in gestation can create defects that do not occur with exposure later in pregnancy or neonatally.⁶⁹ Moreover, exposure early in gestation to 17 alpha estradiol induces defects in offspring, while exposure to the same compound later in life produces no effect.⁷⁰ Tween 80, a chemical widely used in industry, has been shown to have similar effects. Exposure during a critical period of development results in abnormalities, while adult exposure results in no effects.⁷¹ Aracadi and colleagues reported that DEHP produces testicular damage in male

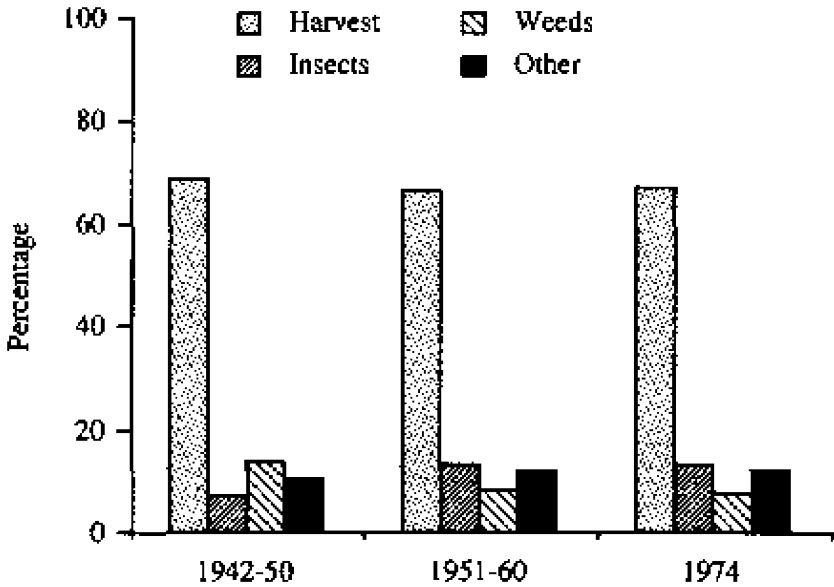
offspring of female rats exposed to an estimated 3.0–3.5 milligrams per kilogram body weight daily in drinking water.⁷²

Two recent studies indicate that prenatal exposures to some common plastics and vehicle exhausts can increase development of reproductive defects in male offspring.⁷³ A new study assessed effects of in utero and lactational exposure to the most commonly encountered plastic, a phthalate ester, di(2-ethylhexyl) phthalate (DEHP), on male reproductive system development and sexual behavior. Male Sprague-Dawley rats dosed with corn oil or DEHP (0, 375, 750, or 1,500 mg/kg/day) from gestation day 3 through postnatal day (PND) 21, developed dose-related effects, including reduced anogenital distance, areola and nipple retention, undescended testes, and permanently incomplete preputial separation. Testis, epididymis, glans penis, ventral prostate, dorsolateral prostate, anterior prostate, and seminal vesicle weights were reduced at PND 21, 63, and/or 105–112. Additional dose-related effects included a high incidence of anterior prostate agenesis and a lower incidence of partial or complete ventral prostate agenesis.⁷⁴

With respect to diesel exhaust—one of the most common exposures in the urban environment today—rats exposed during days 7–20, which are a critical phase of pregnancy, exhibit a number of profound alterations in sexual functioning and development.⁷⁵ Three groups of exposures were studied: a group exposed to total diesel engine exhaust containing 5.63 mg/m³ particulate matter, 4.10 ppm nitrogen dioxide, and 8.10 ppm nitrogen oxide; a group exposed to filtered exhaust without particulate matter; and a group exposed to clean air. With more exposure, differentiation of the testis, ovary, and thymus is delayed and disturbed. Thymus weight is significantly reduced. Testosterone levels are increased and estradiol decreased, resulting in masculinization of the fetus, following exposure during critical phases of organogenesis of reproductive organs and functions.

A number of studies have consistently found that paternal and maternal genomes both make important contributions to health problems of offspring, including germ cell cancers in infants. Paternal exposures prior to conception also play an important and widely underestimated role in the health of offspring. For instance, Garry et al. found that children of male pesticide workers were at increased risk of birth defects. The risks varied with the amount of reported spraying. Timing of pesticide exposure also played an important role in risk of birth defects, with exposures occurring early in pregnancy leading to

Figure 7: Pre-harvest Yields and Losses to Insects, Weeds, and other Causes, over the Period when Insecticides became Widely Used



Source: Pimentel 1976

more birth defects than exposures late in pregnancy. Male offspring were at greater risk than females for a number of birth defects.⁷⁶ Thus, the importance of the timing of exposure extends to the period of spermatogenesis, some 62 days prior to conception, and males appear more vulnerable than females to certain types of damage. It is interesting to note that there is no clear evidence that pesticide use increases net crop yields or reduces loss of crops to insects. Figure 7 illustrates this point. Their economic benefit therefore lies in their production and sales, not in their use.

Sometimes health effects arise and the critical period of exposure remains unknown. For instance, premature sexual development in girls as young as 22 months has been found to occur in those with higher exposures to phthalates in a case series reported from Puerto Rico.⁷⁷ Other studies show that biomarkers of volatile compounds, such as benzene, increased in adolescents who have reduced sexual development, renal function, and increased genetic damage, and who live in close proximity to incinerators operating at legal limits.⁷⁸ It is unclear whether prenatal or early life exposures are more critically

involved in these instances, or whether male-mediated teratogenesis may also be involved.

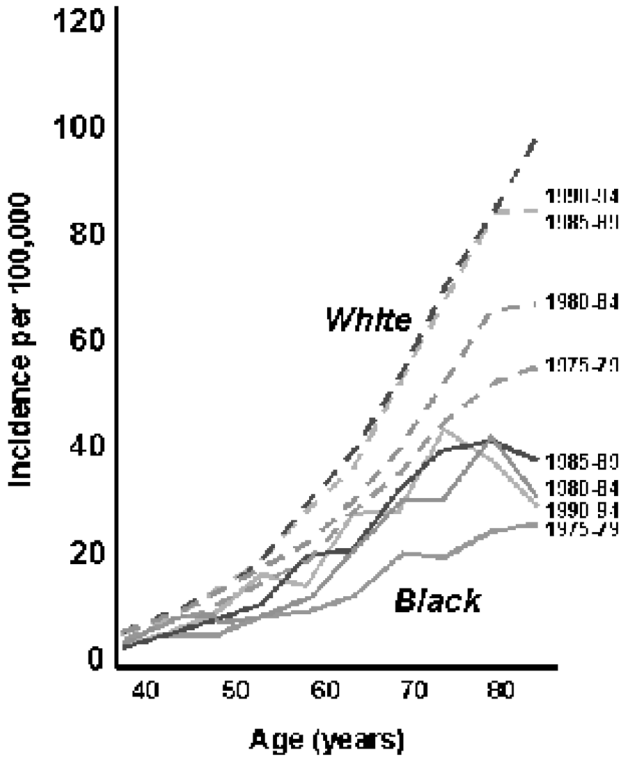
VII. Cancer

Cancer patterns are among the most complex confronting public health today.⁷⁹ The most common and virulent form of cancer is that of the lung. While 80 percent of lung cancer in many societies is tied with smoking, the remaining proportion occurs in those who are not smokers. Passive smoking is an important exposure, as is radon in some cases. But, the nonsmoking fraction of lung cancer is so large that it easily constitutes the sixth most frequent type of cancer death.⁸⁰ A number of recent studies indicate that urban air pollution increases the risk of lung cancer in nonsmokers. The studies also implicated traffic fumes, particularly diesel exhaust.⁸¹

Nine-year trends (1988–1996) show changes in incidence at the state level that account for the year-to-year variability and the fact that most underlying changes in the natural history of a cancer occur slowly (e.g., changes in exposure or risk factors such as smoking). The Minnesota Cancer Registry (MCR) reports that incidence of several cancers not known to be linked to smoking rose in the nine-year period.⁸² Non-Hodgkin's lymphoma among males and females, for example, increased in the state and in the entire country (figure 8). To assess whether any of these changes are real (i.e., not due to chance variation), the MCR used the "estimated annual percent change" (EAPC) statistic. The EAPC is a straight line (regression) that best approximates all of the data points. For example, an EAPC of 2.1 percent means that, on the average over the nine-year period, the cancer incidence increased 2.1 percent per year. After the EAPC is calculated, the next question is whether the EAPC is statistically different than zero. That is, is there evidence that the apparent change is real or is it consistent with the normal variability of the cancer incidence and no change in incidence has occurred?

Cancers strongly related to smoking have generally decreased among males and increased among females, with the notable exception of esophageal cancer. These trends are thought to reflect the changing smoking patterns of males and females over time. Among Minnesota males, the incidence and mortality of cancers of the lung and bronchus have decreased an average of about one percent per year, and laryngeal cancer incidence has declined 3.27 percent per

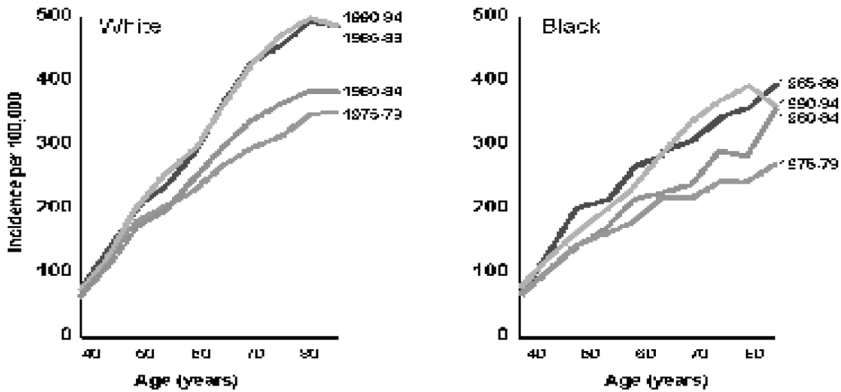
Figure 8: Non-Hodgkins Lymphoma in U.S. Women



year. Oral cavity cancers have also decreased among males (a significant 2.65 percent per year for incidence and a nonsignificant 2.44 percent per year for mortality). Curiously, the incidence of cancer of the esophagus has increased in males an average of 3.27 percent per year. Nationally, esophageal cancer incidence rates for males have increased since 1988 as well, with the suggestion of a plateau starting in 1994. Among Minnesota females, incidence and mortality of cancers of the lung and bronchus have increased significantly, about 3 percent per year, and cancers of the larynx have increased nonsignificantly, an average of one to two percent per year. Incidence and mortality rates for cancer of the oral cavity and cancer of the esophagus among females show little significant change over the nine-year period.

Trends in breast, cervical, and ovarian cancers are notable. Breast cancer incidence in females increased an average of 0.2 percent per year. Although this finding is not significant, it is comparable to national estimates. Between 1950 and 1991, there was no evidence that

Figure 9: U.S. Breast Cancer Incidence by Race



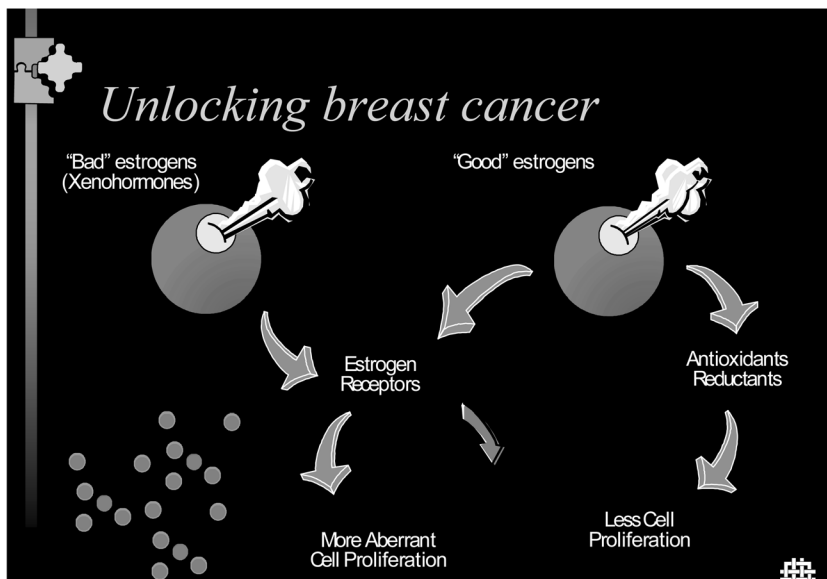
breast cancer mortality in females changed. This was true nationally, as well as in Minnesota. The lack of improvement in mortality experience was puzzling since it was hoped and believed that screening would produce a more favorable outcome. In 1992, breast cancer deaths decreased for the first time. The mortality rate has continued to decline in Minnesota, yielding a statistically significant decline in breast cancer mortality for a short period of time.

National figures for breast cancer are similar. Another way of looking at these data is to consider time periods of diagnosis, as in figure 9. These show that mortality stopped increasing in white women after 1986–93, but continued to increase in African American women.

It is clear that breast cancer continues to pose serious challenges, as most women who develop the disease have none of the known risk factors. Mortality has not appreciably declined in more than two decades for African American women. Even though nonwhites develop the disease less often than do whites, more nonwhites die of it. This suggests that there are major cultural and socioeconomic factors that affect prognosis. Access to care is obviously important.

A number of researchers have advanced the theory that endocrine disruptors, environmental chemicals that function like hormones, could also be affecting trends in a number of hormonal cancers, including breast, prostate, testicular, and ovarian cancer.⁸³ The concept is essentially the same for cancer as for reproductive hazards previously discussed, and is illustrated in figure 10. Chemicals in the environment function like estrogen in the endocrine system, and increase the total hormone burden on the body. Women who sustain greater lifetime

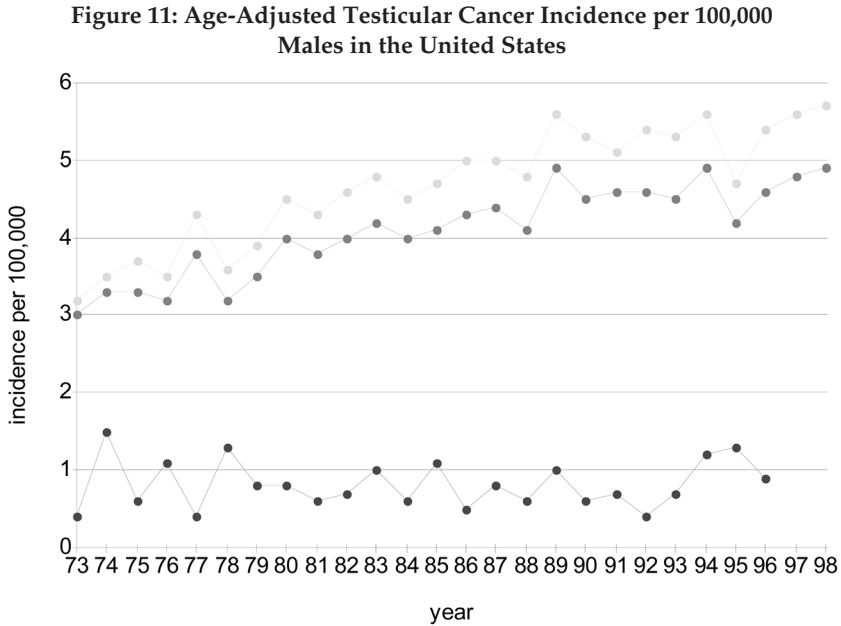
Figure 10: Unlocking Breast Cancer



exposure to hormone mimicking compounds appear to be at increased risk for breast cancer as well as other diseases.

Testicular cancer has increased in incidence in most industrial countries (see figure 11). Mortality has fallen dramatically, chiefly due to advances in treatment. The underlying trends remain unexplained though. Several studies have found that workers with higher exposures to organic solvents have an increased risk of testicular cancer, with risks being highest in those with the greatest exposures. In addition, prenatal exposure to the hormone diethylstilbestrol (DES) has been linked with a variety of male reproductive tract abnormalities, and also appears associated with increased risk of testicular cancer.

Recent studies make clear that the study of hormonal cancers, such as testicular and breast, requires careful consideration of the timing of exposure, along with dose. Studies of persistent organic pollutants, such as DDE and PCBs in adults at the time of diagnosis,⁸⁴ cannot shed light on their role in causing cancer when exposure occurs at earlier stages of development.⁸⁵ In addition, studies of these persistent compounds cannot assess the role of agents known to cause breast and testicular cancer, such as methylene chloride, benzene, and some phthalates and chlorinated organic solvents, nor can they clarify the impact of prescription pharmaceuticals that have been identified as



Source: SEER Cancer Statistics Review, 1973-1998

tumor promoters. These compounds leave no residues that can be detected months or years after exposures have occurred.⁸⁶ For many reproductive outcomes, no direct measures of prenatal exposure are readily available.

VIII. Trends in Neurodegenerative Diseases

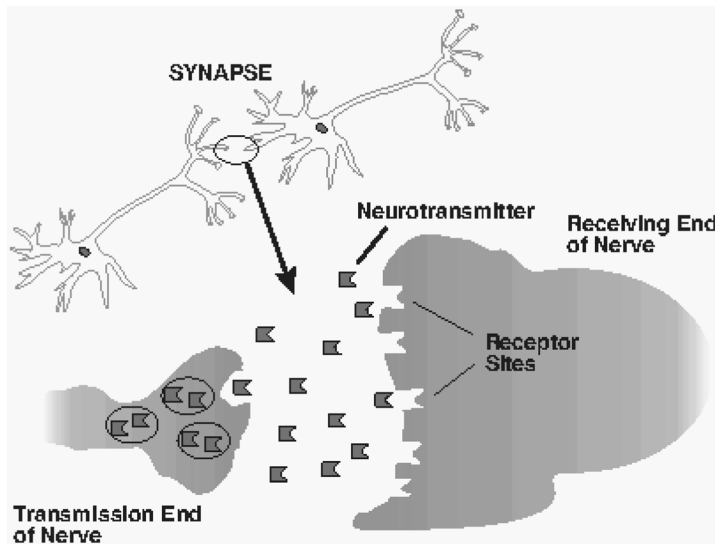
In recent decades, we have witnessed an unexplained increase in neurological disorders in people of all ages. Neurotoxins are suspected of playing an important role, although research remains preliminary. A surprising number of children suffer from a variety of neurological disorders, including attention deficit/hyperactivity disorder (ADHD), learning disabilities (such as dyslexia), mental retardation, autism, and emotional/behavioral problems.⁸⁷ We also see an increase in diseases that typically occur at the end of life, such as Alzheimer's and Parkinson's disease.⁸⁸ It is especially disconcerting to observe that the average age of onset for these diseases has been decreasing. While some of this decrease could be due to improved surveillance and ascertainment, it does appear that there is a real increase in the incidence of the diseases

and a reduction in the average age of diagnosis. All of these diseases seem to be caused by either a degradation in a particular area of the brain or a disruption within certain neural networks.⁸⁹ In both cases, the neurological malfunction appears to stem from the fact that neurotoxins inhibit the ability for neurotransmitters, our brain's messengers, to travel across a synapse and communicate with other neurons. This pathway to neurological disorders is parallel to that noted for breast cancer, where some compounds that are xenoestrogens have been found to inhibit gap junction cell communication.⁹⁰

The human brain consists of billions of neurons that integrate, regulate, and control our daily activity through a complex interplay of chemical and electrical processes, as illustrated by figure 12. Each neuron has a cell body, an axon that extends from the cell body, and many dendrites that branch off from the axon. To communicate with other neurons, an electrical charge builds up within the cell body and travels along the axon until it reaches a synapse, the gap between itself and the dendrites of another neuron. This electrical charge then fuels a chemical reaction to release neurotransmitters that travel across the synapse and bind to specific receptor sites on the postsynaptic, or receiving neuron's, membrane. The neurotransmitters alter the membrane to initiate or prevent the same process from happening in the receiving neuron. Although different functions in the brain rely on different types of neurotransmitters, this electrical and chemical process allows signals to pass from neuron to neuron and accomplish the brain's diverse range of activities. However, neurotoxins can greatly impair, and sometimes permanently damage, these intricate processes.⁹¹

Organochlorines, organophosphates, dioxin, mercury, and lead are among the most commonly cited neurotoxic chemicals.⁹² These substances are incorporated in widely used products including pesticides and solvents, as well as released into the environment as industrial effluent. Both animal and human studies have implicated these and other common chemical exposures to neurodevelopmental, learning, and behavioral disabilities. Exposure to high levels of methyl mercury caused severe disturbances in motor coordination, gait, and language in adults and children, and severe mental retardation and cerebral palsy-like symptoms in prenatally exposed children.⁹³ Further studies have shown neurological impairment at even "safe" levels of mercury exposure.⁹⁴

Figure 12: Depiction of how Neurotransmitters are Received by Receptors on the Postsynaptic Membrane



Source: http://kaboom.chem.uwm.edu/toxicology/neurotoxins/neuro_intro.htm

Organochlorines interfere with neuron communication by directly binding to the receptor sites and blocking out the natural neurotransmitters; for example, polychlorinated biphenyls (PCBs) interfere with an enzyme necessary for the synthesis of dopamine, a neurotransmitter that plays an important role in learning and memory. A landmark study by Jacobson and Jacobson in 1984 confirmed neurological impairment in children who were exposed to PCBs in the womb. The study enlisted mothers who ate Great Lakes fish, which contain significant levels of PCBs and other chemical contaminants,⁹⁵ at least 2–3 times per month in the six years before pregnancy. PCBs accumulated in the mother and were passed to her child in the womb as well as through breast milk. Jacobson and Jacobson found that the fish-eater mothers gave birth to babies who suffered from more neurological impairment than their nonfish-eater counterparts. The study described how the children were affected throughout early development from their in utero exposure. At birth, the fish-eaters' children had lower birth weight and head circumference size, as well as weak reflexes and unbalanced movements. At seven months, the infants did not exhibit

Figure 13: Summary of Chemical Contaminants in Adults, Highlighting Differences associated with Gender and Consumption of Great Lakes Fish

Table 3. Summary statistics for PCB, DDE, lead, and mercury by sex, age, and fish-eating status.

Sex	Age (years)	Exposure	Fish eaters				Non-fish eaters			
			No.	Mean ± SD	Median	Min-Max	No.	Mean ± SD	Median	Min-Max
Male	< 60	PCB ^a	16	15.88 ± 14.8	10.60	ND-65.20	11	6.11 ± 4.2	5.00	ND-15.30
		DDE ^b		12.26 ± 9.2	9.60	1.80-36.70		7.12 ± 5.9	4.80	ND-17.40
		Lead ^c		4.40 ± 1.5	5.00	2.00-7.00		3.45 ± 1.6	3.00	ND-6.00
		Mercury ^d		3.18 ± 4.8	2.40	ND-20.50		2.05 ± 1.9	2.10	ND-5.00
	60-69	PCB	16	23.03 ± 19.1	17.25	ND-65.20	12	6.82 ± 3.5	5.95	3.40-15.50
		DDE		18.12 ± 17.7	8.70	ND-52.70		9.99 ± 8.0	7.95	1.80-31.50
		Lead		5.13 ± 1.8	5.00	3.00-9.00		5.08 ± 4.1	4.00	ND-15.00
		Mercury		3.96 ± 2.7	3.75	ND-9.80		ND	ND	ND-4.00
	≥ 70	PCB	14	24.69 ± 19.3	19.95	ND-75.00	7	5.66 ± 2.0	4.90	4.00-10.00
		DDE		28.09 ± 37.4	13.05	5.40-145.00		8.03 ± 6.4	6.90	3.00-21.40
		Lead		4.38 ± 1.7	4.00	2.00-7.00		3.67 ± 1.6	3.50	2.00-5.00
		Mercury		3.06 ± 2.9	2.85	ND-7.40		ND	ND	ND-3.50
Female	< 60	PCB ^a	21	8.67 ± 4.6	7.60	ND-23.00	19	4.64 ± 2.2	ND	ND-9.20
		DDE ^b		7.09 ± 4.6	6.20	ND-15.90		4.94 ± 4.3	4.50	ND-19.60
		Lead ^c		2.45 ± 1.5	2.00	ND-7.00		2.47 ± 2.2	2.00	ND-9.00
		Mercury ^d		ND	ND	ND-3.00		ND	ND	ND-4.10
	60-69	PCB	17	14.04 ± 6.8	12.40	4.00-26.30	16	8.71 ± 8.5	6.10	ND-25.90
		DDE		11.50 ± 6.3	10.00	2.90-26.40		8.46 ± 7.9	6.10	1.60-32.50
		Lead		3.29 ± 1.3	3.00	ND-6.00		2.75 ± 1.3	2.00	2.00-6.00
		Mercury		ND	2.00	ND-6.00		ND	ND	ND-3.30
	≥ 70	PCB	17	13.52 ± 10.8	12.10	ND-49.00	13	5.05 ± 3.8	4.70	ND-11.00
		DDE		22.63 ± 16.1	16.20	7.00-62.80		7.22 ± 6.1	5.80	ND-20.90
		Lead		3.38 ± 1.9	3.00	ND-7.00		3.54 ± 1.4	3.00	2.00-6.00
		Mercury		ND	ND	ND-8.00		ND	ND	ND-3.00

Abbreviations: Max, Maximum; Min, minimum; ND, below the limit of detection.
^aDetection limit = 3.0 ng/g; ^bDetection limit = 1.0 ng/g; ^cDetection limit = 2.0 µg/dL; ^dDetection limit = 2.0 ng/g.

Source: Schantz, S., et al., *Environmental Health Perspectives* 109 (6): 605-611

the normal behavior of staring at new images more than familiar images; and at age four, the children had lower scores in verbal and memory tests. Studies in North Carolina, New York, and Denmark support these findings⁹⁶

A study released this past June reveals that PCBs have measurable detrimental effects on adults as well. In a similar study design, Schantz et. al. recruited 180 adults, ranging from 49 to 86 years old, and divided them into fish-eaters (consumed greater than 24 lb/year) and nonfish-eaters (consumed less than 6 lb/year). A blood sample from each subject was analyzed for PCBs and ten other contaminants, and levels of both PCBs and DDE were higher in the 101 fish-eaters than the 79 nonfish-eaters. On average, males had higher PCB levels than females (mean: males 15.2 versus females 9.2; p=0.01), and PCB levels were also associated with increasing age (chi-square test, p=0.04). The subjects performed a battery of cognitive tests, including tests of memory and learning, executive function, and visual-spatial function. PCBs, but not DDE, were found to be associated with the decreased memory and learning; however, they were not found to have any effect on an adult's executive function and visual-spatial function. The authors conclude that fish consumption advisories should be expanded from targeting pregnant women to targeting fish-eaters of all ages. They feel that further studies should explore the impact of

Figure 14: Summary of Association between PCB Level and Cognitive Ability Tests in Adults

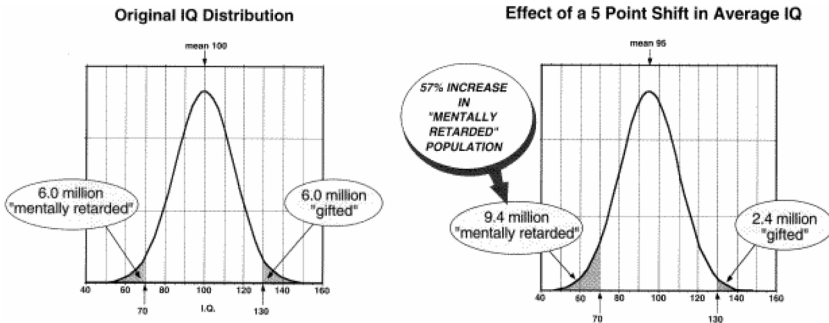
Table 5. Memory and learning test scores by PCB exposure quartile.

PCB Level (ng/mL)	WMS logical memory, delayed recall			CVLT Semantic Cluster Ratio			CVLT List A, Trial 1		
	No.	Observed (mean ± SD)	Predicted mean	No.	Observed (mean ± SD)	Predicted mean	No.	Observed (mean ± SD)	Predicted mean
ND ^a -4.6	44	9.67 ± 3.4	9.72	43	2.41 ± 0.9	2.31	44	7.64 ± 2.1	7.53
4.7-7.8	45	9.09 ± 5.2	9.26	44	2.20 ± 0.9	2.29	45	7.31 ± 2.3	7.26
7.9-13.8	45	8.39 ± 4.2	8.28	45	2.13 ± 0.8	2.12	45	6.91 ± 2.0	7.10
13.9-75.0	45	7.66 ± 3.9	7.54	43	1.88 ± 0.7	1.89	43	6.14 ± 1.5	6.11

^aBelow the detection limit of 3.0 ng/mL.

Source: Schantz, S., et al., *Environmental Health Perspectives* 109 (6): 605-611

Figure 15: Effect of a Five Point Shift in Average IQ



Source: Schettler, T., et al., *In Harms Way: Toxic Threats to Child Development*, Cambridge, Mass.: Greater Boston Physicians for Social Responsibility, 2000

occupational exposure to PCBs, as workers have been reported to have serum PCB levels 10- to 100-fold higher than the levels that are typical in Great Lakes fish-eaters.

Vast amounts of neurotoxic chemicals are released into the environment each year, and often lead to human exposures with potentially harmful effects. Our scientific understanding of the effects of neurotoxic exposures is inadequate for predicting the effects of a given chemical exposure, and has failed to protect populations from harmful consequences. As with endocrine disruptors, precautionary policy must be instituted to protect populations from effects that might jeopardize the potential for success of future generations. Figure 15 shows the effect of a small shift in average IQ that might result from widespread exposure to neurotoxins. A downward shift of five points would result in more than a 50 percent increase in the number of men-

tally retarded individuals and a decline of almost 50 percent in gifted individuals.⁹⁷ The implications of such a shift are worrisome indeed.

IX. Occupational Hazards and their Implications

Modern society has seen an unprecedented growth in the production, use, and disposal of synthetic organic chemicals, including many highly persistent materials, now known to have a detrimental impact on humans. While current methods of production in large-scale systems are highly mechanized, individual exposure of workers can still be especially high in industrial countries. In rapidly growing economies in transition, workers are often on the front line for exposure to materials, many of which have been banned in developed economies.

Less than 10 percent of all chemicals in commerce have been tested for their toxicological properties. While industry is now mounting an impressive campaign to remedy this deficit in information, for nearly half a century, human exposure to such materials has taken place on a scale unprecedented in history. Moreover, there are some classes of chemicals for which use is equivalent to disposal. Chemicals, such as a number of organochlorine compounds and heavy metals, are highly persistent. Once they enter the environment, they tend to bioaccumulate and move up the food chain.

The relationship between health and the economy is truly reciprocal. Economic activities across sectors have a wide range of positive and negative impacts on health. The bulk of this essay has addressed potential negative impacts of economic sectors on health, but the health of populations will also affect all sectors, as illustrated by Table 2. These effects can be direct, as in the case of cancer leading to a reduced workforce and more sick days, or indirect, as in the case of reduced food supply resulting from fewer agricultural workers or degraded natural environments. The indirect effects of health on the economy can extend much farther, although the magnitude of these impacts declines with each step away from the cause. For example, a reduced or weakened agricultural workforce, afflicted with a high rate of HIV or other ailments, will make large-scale monoculture a more attractive farming method because it is less reliant on workers. Under our current energy scheme, industrialization of agriculture increases fossil fuel emissions, which, in turn, increases the incidence of respiratory infections, cancer, and reproductive disorders. Under some condi-

Table 2: Cross-Sectoral Health Effects

“C” tells how each sector may cause health effects.

“E” tells potential effects of health conditions on each sector.

	Agriculture	Energy	Transportation	Industry
Neurological Damage	C: neurotoxic compounds used in pesticides E: fewer able workers, reduced technological innovation due to lower average IQ, possibly reduced food production	C: E: fewer able workers, reduced technological innovation due to lower average IQ, possibly inadequate energy supply	C: E: fewer able workers, reduced technological innovation due to lower average IQ, possibly greater incidence of traffic accidents	C: neurotoxic metals used in industrial processes and products E: fewer able workers, reduced technological innovation
Reproductive Dysfunction	C: estrogenic compounds used in pesticides E: reduced workforce in future generations, possibly reduced food production	C: diesel exhaust may act as endocrine disrupter E: reduced workforce in future generations	C: diesel exhaust may act as endocrine disrupter E: reduced workforce in future generations	C: estrogenic solvents and agents used in industrial processes and products E: reduced workforce in future generations
Respiratory Illness	C: fossil fuel exhaust E: reduced work force, more sick days, possibly reduced food production	C: fossil fuel exhaust E: reduced work force, more sick days	C: fossil fuel exhaust, fine particulates E: reduced work force, more sick days	C: fossil fuel exhaust, fine particulates, other air pollutants, asbestos E: reduced work force, more sick days
Cancer	C: pesticides linked with hormonal cancers E: death of workers, more sick days, possibly further decreased workforce in future generations and/or reduced food production	C: fossil fuel exhaust E: death of workers, more sick days, possibly further decreased workforce in future generations	C: vehicle exhaust, carcinogenic agents used in vehicle production E: death of workers, more sick days, possibly further decreased workforce in future generations	C: carcinogenic agents used in industrial processes and products E: death of workers, more sick days, possibly further decreased workforce in future generations
HIV	C: E: death of workers, more sick days, possibly further decreased workforce in future generations and/or reduced food production	C: E: death of workers, more sick days, decreased workforce in future generations	C: E: death of workers, more sick days, decreased workforce in future generations	C: E: death of workers, more sick days, decreased workforce in future generations

tions, monoculture could also lead to nutritional imbalances, leaving the population more susceptible to a variety of infectious agents and pollutants. Further, large-scale monoculture can also deplete soil nutrients, increase groundwater degradation, and diminish the quality and quantity of water available for human use.

X. Education

Education, especially literacy for young girls, remains central to achieving sustainable development for several reasons. In a complex world, some degree of literacy is virtually required to understand the dangers posed by rapid industrialization. Educated people can more easily find the pertinent information, assuming it is available at all, to make choices about how to live in a healthful manner. Being able to read allows individuals to decipher directions when using potentially hazardous chemicals, which Table 3 makes clear. There is also a strong correlation between increased years of schooling and lower infant

Table 3: Number and Proportion (%) of Respondents who Prepared Agrochemicals according to the Instructions, Wore Protective Clothing while Spraying, and Took Precautions when Handling Chemicals by Level of Education. Kenya.

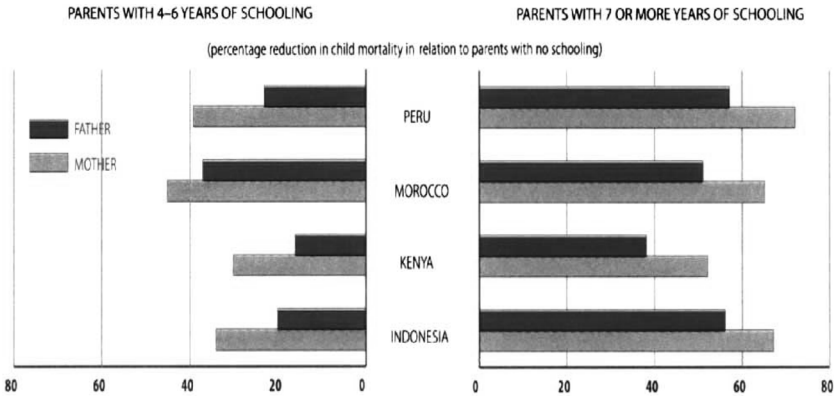
Level of education	Followed instructions	Wore protective clothing	Caution with —		Sample size
			All chemicals	Certain chemicals	
None	64 (15.0)	96 (22.5)	116 (27.2)	259 (60.8)	426
Primary	424 (44.0)	342 (22.5)	217 (22.5)	699 (72.6)	963
High school and over	256 (62.7)	167 (40.9)	98 (24.0)	289 (70.8)	408
Total	744 (41.4)	605 (33.7)	431 (24.0)	1 247 (69.4)	1 797

Source: Source: Impact of Pesticide Use on Health in Developing Countries, Part I, IDRC, 1993

mortality rates. As figure 16 demonstrates, even a few years of formal education can lead to large advances toward this end. A reduction of infant mortality by an average of nearly 40 percent was seen in women with only 4–6 children. The average reduction for women with seven or more years of schooling was closer to 60 percent.

A growing literature indicates that female literacy is the key to sustainable development. Teaching girls to read allows them to learn how to control their personal and reproductive health. Literate girls, in turn, can learn to engage in environmentally sound practices in their immediate environments. Armed with education and information about expanding opportunities, women in a number of developing economies have been able to advance their welfare and that of their families. Female literacy thus remains central to efforts to promote healthier environments and growth. An excellent illustration comes from the Grameen Bank initiative. It provided modest funding to thousands of landless, rural women, who used these funds to begin small businesses in Bangladesh and more than forty other impoverished zones. This program provided micro-credit and self-employment strategies to adopt information technologies, which now extends to 241 programs in 58 different countries on four continents. The poor do not lack survival skills; in fact, the poor have extraordinary skills just in order to survive. Giving poor women the capital to apply these skills has proven far more profitable than was ever anticipated. The Grameen Bank is not a charity. To date, all of the Bank’s \$2 billion in loans have been repaid.

Figure 16: Effect of Parents' Schooling on the Risk of Death by Age 2, Selected Countries, Late 1980s
Education and the Link with Family Health



Source: The World Bank, *World Development Report 1993: Investing in Health*, World Development Indicators, New York: Oxford University Press, 1993, p. 43

XI. Implications and Conclusions

The complex developments underway in society make it difficult to pinpoint causal connections between these developments and specific health consequences. In part, this is because many chronic diseases have multiple causes and because exposures that take place at different points in life can result in different outcomes. Despite these difficulties, the lessons of the last two centuries are clear. Basic policies to clean up the environment, workplace, household, air, and water will result in improvements to public health, the full extent of which cannot be precisely estimated. Research is needed to clarify the links between specific exposures and outcomes. However, while that research continues to evolve, public policies need to be developed to reduce use of and exposure to agents suspected of exacerbating many chronic ailments that are unexplained today. Moreover, children remain at special risk for reasons that have been well articulated.⁹⁸

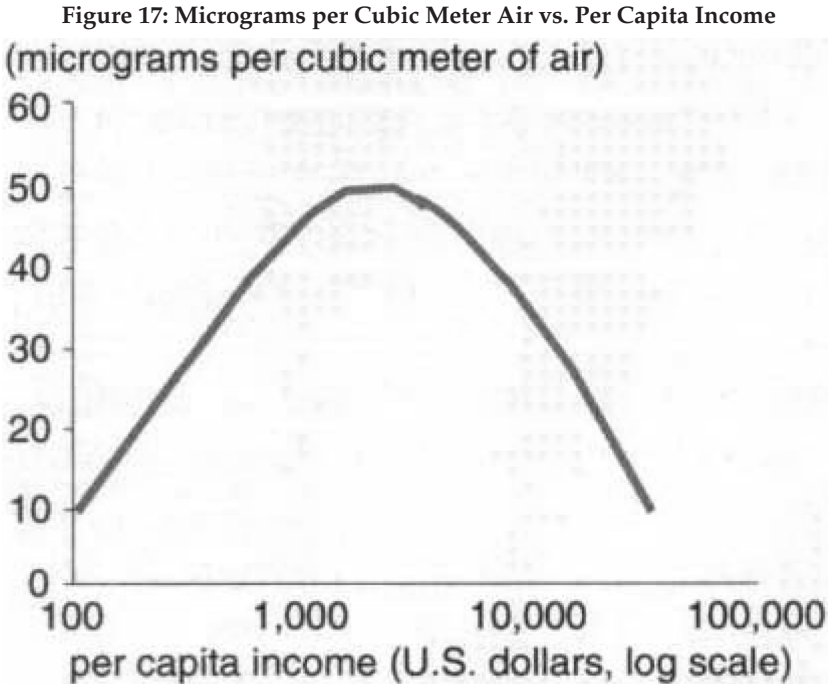
Today, most of the world's children live in the rapidly developing world. Children living in urban areas are at double jeopardy from poverty and degraded environments. Many do not have access to basic health care, such as immunizations, and a significant number of them suffer from malnutrition and infectious diseases. Environmental pollu-

tion only adds to the burden of food deprivation, microbial diseases, and lack of preventive care and medical treatment. It has long been known that air pollution can aggravate illnesses such as bronchitis, asthma, and chronic obstructive pulmonary disease. Children with diets deficient in vitamins, minerals, and protein are especially vulnerable to the toxic effects of chemicals. When their immunity is reduced, their bodies cannot easily transform pollutants into more benign substances, and tend to retain toxic materials for longer periods. Less well appreciated is the fact that nutritional, workplace, and other exposures of young people affect their ability to have children as well as their long-term health.

Obviously, by-products of industrialization pose real risks to human well-being. Yet there is still no widely recognized measure of economic activity that incorporates health as well as environmental degradation into its calculations. Efforts are underway to develop such a system. "Green accounting," a relatively new concept used by the World Bank and other lending institutions, is a good start. Methods are now being generated for assessing the long-term costs of natural resource degradation and for devising economic policies that address these issues. Unfortunately, this expansion of the definition of economic progress does not integrate the accounting of public health costs and benefits. In many transitional economies, for example, the capacity to control, monitor, and use new transportation and energy technologies without endangering workers and communities is limited or, in many cases, nonexistent.

In an appealing analysis, Kuznets has suggested that as countries develop and per capita GDP rises above \$6,000, a demand for improved environmental quality naturally arises. Indeed, one large statistical study found that ambient environmental pollution in countries tends to be reduced once GDP approaches this level. This conclusion is based on an assessment of historical trends in regions that have undergone major transitions, and some aggregate analyses of overall global trends. An unfortunate implication of this conclusion is that relatively poor countries must merely wait until their development has grown to per capita GDP \$6,000 before investing in environmental protection. Another is that these countries presumably cannot afford to invest in basic improvements until GDP has attained that critical level. This effect is illustrated in figure 17.

What is wrong with this picture? Despite its obvious allure, the argument that development axiomatically leads to environmental pro-



Source: World Resources 1996-97—A Guide to the Global Environment—The Urban Environment, WRI, 1996, Part I, Ch. 1, fig. 1.3

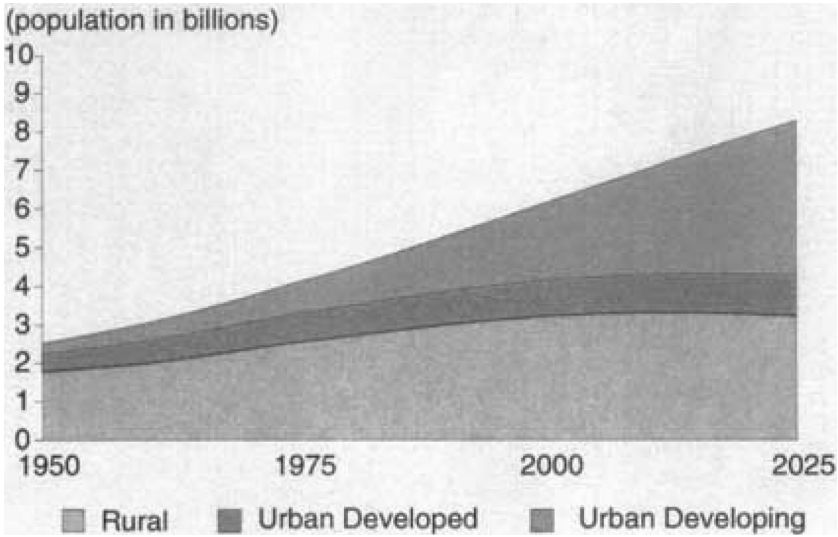
tection flies in the face of what is happening in many rapidly growing centers. It fails to take into account how different the world of today is from that of a few decades ago. Effectively, those who would apply Kuznet's historical analysis to future projections do not consider four fundamental facts of rapidly industrializing regions.

First of all, the density of population in developing megacities, shown in figure 18, means that any given exposure will hit a much greater proportion of the population. The poor remain predominantly female and young. Many poor children and women in cities lack the most basic services and health care.

Second, the globalization of trade has substantially expanded markets for industrial pollution. Development is occurring in places where people simply do not have the resources to protect themselves from such agents. There is no routine labeling of hazards, even with pictographs, and, in some areas, the majority of people are only semiliterate and lack information about potential risks.

Third, measures of GDP are now widely understood to exclude many key factors, or what economists refer to as externalities. Thus, if

Figure 18: Population in Rural, Urban Developed, and Urban Developing Regions over Time



Source: United Nations (U.N.) Population Division, *World Urbanization Prospects: The 1994 Revision*, New York: U.N., 1995, pp. 86-101.

a man marries his housekeeper, he is reducing GDP. If five people drive five cars to a meeting, rather than sharing a ride, then GDP is increased, even though pollution is worsened. If, on their way to the meeting, they get into a serious accident and need extensive and costly medical treatment, then GDP is also increased. There is no question that as conventionally measured, GDP cannot directly reflect the full quality of life.

Finally, some of the long-term effects of pollution cannot be fixed, no matter how much money is thrown at them after the fact. Air and other toxic pollution can permanently reduce lung growth, damage the brain, and/or affect the body's ability to resist infectious agents. Pollution may also reduce the ability to produce healthy children. Thus, the failure to prevent harmful exposures from occurring, especially in rapidly growing urban areas, can permanently damage public health and welfare, and, in doing so, reduce human capital. The presumption that the eventual growth in per capita GDP will ultimately allow countries to address environmental health and welfare issues does not consider this fundamental point. From an economist's perspective, activities that permanently damage or deplete natural resources and

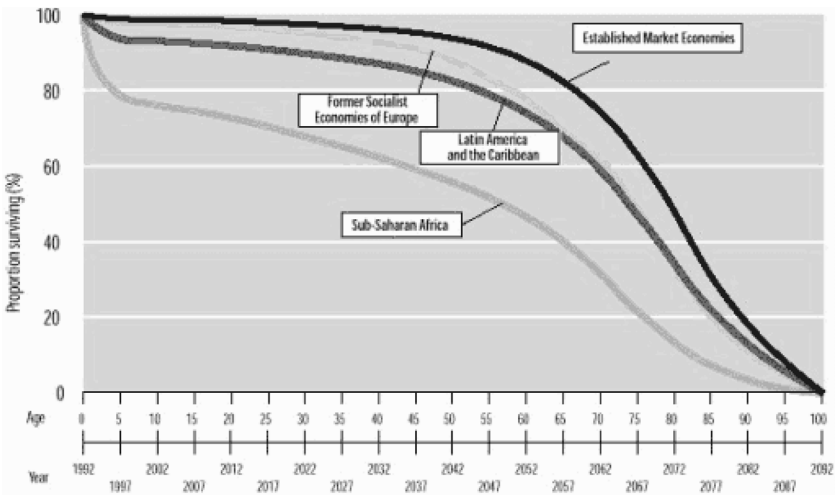
human capital can result in real and irrevocable declines in national wealth. The potential for a given activity to reduce wealth in this way must be examined along with its potential to create new capital and increase national wealth.

Within the discipline of economics, the notion of green GDP accounting has been bandied about for some time but consensus has not been reached on how to put the idea into operation. Without concrete methods for valuing green activities, GDP remains a crude measure of economic activity that fails to take into account positive and negative impacts on natural resources and human populations. Pressure to think more broadly about the socioeconomic consequences of development are likely to foster political changes, the full extent of which will only become clear when today's rapidly growing regions have reached maturity. Whether the citizens of these regions will be made to pay the price of past developed regions through the continued use of inefficient technologies is one of the key questions that challenges us today. There is no doubt that if such a price were exacted, the toll would be more massive than at any point in human history.

While economic growth and development have produced important benefits to modern society, the positive and negative health consequences of this development have not been fully assessed. Life expectancy has risen. Infant mortality has dropped. As figure 19 shows, however, the world's populations do not share equally in these improvements. In some regions, incongruities in economics and health have increased. Women sustain the greatest disadvantage in the poorest regions. Girls are at the bottom of the social order in terms of access to food, education, and health care. Whether the recent health advances that have occurred in many countries can be sustained in a world undergoing increased environmental degradation is one of the most important challenges of this new century.

This broad discussion makes clear that technology decisions are also fundamentally public health decisions. Depending on how they are devised, policies to mitigate GHG and reduce environmental contamination can yield substantial and immediate benefits to the three billion people currently residing in urban areas throughout the world. Moreover, these largely unappreciated health benefits could be a strong motivator for a variety of mitigation actions, such as reducing the overall use of carbon-based or toxic materials. The challenge to the policymaking community will be to forge specific practical strategies to encourage the funding and adoption of more efficient, less polluting

Figure 19: Life Expectancy Curves for Developed Market Economies, Former Soviet Union, Latin America, and Sub-Saharan Africa



Source: Calculated from data in Murray & Lopez, 1996a. Health and Environment in Sustainable Development, WHO 1997, ch. 1, fig. 1.2

technologies in the workplace and in the general environment. If the substantial public health impacts charted here become more widely recognized and their full economic and social impact are integrated into discussions of public policy, a major rethinking of today’s technology decisions could be brought forth. Sustainable development can only be achieved when information is made broadly available on the full range of potential health impacts of development decisions on equity and resources. ●

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