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Pesticides and Human Health

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1. Introduction

Pesticides are those chemicals that are used to destroy unwanted forms of life or organisms. They include insecticides, rodenticides, herbicides, fungicides, fumigants etc. They are expected to have a selective action or toxicity to animals and man. This has made the government of many countries to introduce legislation which prevent the use of the more dangerous and persistent chemicals as pesticides and to demand withdrawal from the market any licensed chemical found to be harmful to animals or man.

In 1977, it was estimated that every year 20,000 fatalities result from the use of pesticides. Most of these occurred in developing countries (Forget, 1991). The 1981 estimate by OXFAM gave a figure of 40,000 fatalities from about two million cases of poison per year (Akubue, 1997).

Pesticides are those substances which, on entering the body by whatever route, e.g. ingestion, inhalation, or absorption through intact skin, produce harmful effects. The effect may be in the form of damage to the tissues or as a disturbance of the functioning of the body.

According to the project of cooperative extension officers of cornel, University, Michigan state university, the science of toxicology is based on the principle that there is a relationship between a toxic reaction (response) and the amount of the poison received (the dose). An important assumption in this relationship is that there is almost always a dose below which no response occurs or can be measured. A second assumption is that once a maximum response is reached any further increases in the dose will not result in any increased effect. In a particular instance, a dose – response relationship does not hold true in regard to true allergic relations. Allergic reactions are special kind of changes in the immune system; they are not really toxic responses. The difference between allergies and toxic reactions is that a toxic effect is directly, the result of the toxic chemical acting on the cells. Allergic responses are the result of a chemical stimulating the body to release natural chemicals which are in turn directly responsible for the effects seen. Thus in allergic reaction, the chemical acts as a trigger, not as bullet.

For all types of toxicity of chemicals to humans , knowing the dose response relationship is a necessary part of understanding the cause and effect relationship between chemical exposure and illness. As Paracelsus in the 16th century once wrote, “The right dose differentiates a poison from a remedy”. Note that the toxicity of a chemical is an inherent quality of the chemical and cannot be changed without changing the chemical to another form. The toxic effects on humans are related to the amount of exposure.

1.1 Measure of exposure

Exposure to poisons can be intentional or unintentional. The effects vary with the amount of exposure. Contamination of food or water with varying doses of chemicals can be obtained each time when contaminated food and drink are taken. Some commonly used measures for expressing levels of contaminants are;

Parts per million (PPM), which in metric equivalent is in milligrams per kilogram (mg/kg). This is approximately the amount in the water as one teaspoon per 1,000 gallons.

Parts per billion (PPB), which in metric equivalent is in micrograms per kilogram (μg/kg). This is approximately the amount in water as one teaspoon per 1,000,000 gallons.

However, as an example, individual's sensitivity to alcohol varies, as do individual sensitivity to other poisons. In testing the effects of poison to human health, lower animals are used.

In one particular measure of effects e.g. ED_{50} which means effective dose for 50 percent of animal tested. The ED_{50} of any poison varies depending on the effect measured. In general, the less severe the effect measure, the lower the ED_{50} for that particular effect. Obviously, poisons are not tested in humans in such a fashion. Instead animals are used to predict the toxicity that may occur in humans.

One of the most commonly used measures of toxicity is the LD_{50}. The LD_{50} (the lethal dose for 50 percent of the animals tested) of a poison is usually expressed in milligrams of chemical per kilogram of body weight (mg/kg). A chemical with a small LD50 (like 5mg/kg) is highly toxic. A chemical with a large LD_{50} (1,000 to 5,000 mg/kg) is practically non-toxic. The LD_{50} says nothing about non-lethal effects though. A chemical may have a large LD_{50}, but may produce illness at very small exposure levels. It is incorrect to say that chemicals with small LD_{50}'s are more dangerous than chemicals with large LD_{50}'s. They are simply more toxic. The danger or risk of adverse effect of chemicals is mostly determined by how they are used, not by the inherent toxicity of the chemical itself.

The LD_{50} of different poisons may easily be compared; however, it is always necessary to know which species was used for the tests, the age, sex and how the poison was administered (the route of exposure), since the LD_{50} of a poison may vary considerably based on these factors. Some pesticides (poisons) may be extremely toxic if swallowed (oral exposure) and not very toxic at all if splashed on the skin (dermal exposure). If the oral LD_{50} of a poison were 10mg/kg, 50 percent of the animals that swallowed 10mg/kg would be expected to die and 50 percent to live.

The potency of a pesticide is a measure of its strength compared to other poisons. The more potent the poison, the less it takes to kill; the less potent the pesticide, the more it take to kill. The potencies of pesticides are often compared using signal words or categories as 'Danger-poison' (skull and cross bones) – highly toxic.

Moderately toxic – WARNING

Slightly toxic – CAUTION

Practically non – toxic – none required.

Toxicity assessment is quite complex, many factors can affect the result of toxicity tests. Some of these factors include variables like temperature food, light, and stressful environmental conditions. Others factors related to the animals itself include age sex, health, and hormonal status.

The NOEL (No Observable Effect Level) is the highest dose or exposure level of a poison that produces no noticeable toxic effect on animals.

In toxicology, residue tolerance levels of poisons that are permitted in food or in drinking water for instance, are usually set from 100 to 1,000 times less than the NOEL to produce a wide margin of safety for humans.

The TLV (Threshold Limit Value) for a chemical is the airborne concentration of the chemical (expressed in PPM) that produces no adverse effects in workers exposed for eight hours per day, five days per week. The TLV is usually set to prevent minor toxic effects like skin or eye irritation.

Very often, people compared poisons based on their LD_{50}s and use the decisions about the safety of a chemical based on this number. This is over simplified approach to comparing chemical because the LD_{50}'s and base decisions about the safety of a chemical based on this number. This is an over simplified approach to comparing chemicals because the LD_{50} is simply one point on the dose-response curve that reflects the potential of the compound to cause death. What is more important in assessing chemical safety is the threshold dose, and the slope of the dose-response curve, shows how fast the response increases as the dose increases. It is quite possible that a chemical will produce a very undesirable toxic effect (such as reproductive toxicity or birth defects) at doses which cause no death at all.

A true assessment of chemical toxicity involves comparisons of numerous dose-response curves covering many different types of toxic effects. The determination of which pesticides will be restricted use pesticides involves this approach. Some restricted use pesticides have very large $_{LD50s}$ (low acute oral toxicity) however, they may be very strong skin or eye irritants and thus require special handling.

Although there is no direct extrapolation of animal studies to man, the knowledge gained from dose-response studies in animals is used to set standards for human exposure and the amount of chemical residue that is allowed in the environment. As mentioned previously, numerous dose-response relationships must be determined, in many different species. Without this information, it is impossible to accurately predict the health risks associated with chemical exposure. With adequate information, we can make informed decisions about chemical exposure and work to minimize the risk to human health and the environment.

Pesticides differ from any other chemical substances because they are deliberately spread into the environment. As a consequence, a great part of the human population may be exposed either in the general environment or in the working setting. The occupational exposure involving the manufacturing and the use of pesticides, takes place mainly through dermal or respiratory route. While the environmental exposure, involving general population is mainly due to the ingestion of the contaminated foods and water. The environmental and occupational exposure determines the detrimental effect that this exposure could have on reproductive function. In women, if primordial follicles are destroyed extensively, they cannot be regenerated. This can cause premature ovarian failure and early menopause.

Note the information contained in this topic is not a substitute for pesticide labels. The trade names used here are for convenience only, no endorsement of products is intended nor criticism of unnamed products implied.

In California, suspected pesticide-related illness and suspected work related illnesses and injuries are reportable conditions. In 1998, the occupational Health branch of the California department of health services (CDHS) received a report from California department of pesticide regulation (CDPR) of a pesticide exposure incident in fresno county involving 34 farm workers. CDHS investigated this incident by reviewing medical records of the 34

workers and interviewing 29. The finding indicated that the workers became ill after early re-entry into a cotton field that had been sprayed with a cholinesterase inhibiting carbamate pesticide.

1.2 Prenatal exposure

The study of the effects of low level exposure to environmental neurotoxic agents has been one of the most important aspects of environmental research. The developing organism may be more susceptible to many of these chemicals than the adults. This susceptibity varies during different stages of development, but severe behavioural neurochemical, and neurophysiological abnormalities can be measured in adults that were exposed during neuronal development. Apparently, one of the problems of toxicity testing is to correlate these behavioral alterations with the anatomical changes induced by the neonatal exposure.

The large scale pollution of our environment by chemicals released from industrial and other sources has resulted in many well-publicized episodes of mass poisonings of populations in modern world. While these episodes have highlighted the consequences of unchecked industrial growth, they have not shed light on what may be a much more urgent problem facing mankind: the problem of low level exposure to a variety of chemicals throughout the life cycle. This problem is of completely different nature from the problem of acute mass poisoning in that it not only exposes all age groups but also exposes these groups to several chemicals simultaneously whose individual toxicities even today are largely unknown. The realization that biological systems and ecosystem may not be able to cope with the continuously rising tide of chemical pollution has led government agencies to pass legislation before toxicity thresholds had been adequate determined. In fact, it has resulted in a dilemma for the toxicologist in that more is known about the toxicity of many of the chemicals from cases of human poisoning than is known from animal studies. The production of a new untested chemicals to the already existing pool at an ever-increasing rate also seem to the possibility of careful dose-response studies on identified toxic agents because of the pressure to respond to today's emergency rather than yesterday's head line.

In response to this emergency, biologists have tried to develop rapid screening tests that would serve as early indicators of toxicity. Such tests show a great deal of promise in the screening of potential carcinogens and of neurotoxic substances.

The minimata episode in the 1950's in Japan has made it clear that pregnant women who are exposed to low concentrations of pesticides (e.g methimercury) may experience few symptoms and yet can give birth to severely retarded children. This effect has since been documented in animal studies and has been shown to be due to the increased fetal brain concentration of mercury (null et al; 1973). As was stated earlier, the purpose of many animal studies, therefore, has been to define the threshold level of exposure to mercury below which no neurological abnormalities will be observed in the offspring.

2. Principles of biological tests for toxicity

Toxicology has been defined as the study of the effects of chemical agents on biological material with special emphasis on harmful effects. It basically involves an understanding of all effects of essentially all chemicals on all types of living matter. There is ample evidence to indicate that every chemical is capable, under some conditions of producing some type of effect on every biological tissue. Toxicologic tests are therefore the tests that define the conditions that must be present when a biological cell is affected by a given chemical entity,

and the nature of the effect which is produced. As far as the conditions that must be present are concerned, they may vary from being practically unattainable under ordinary circumstances to being so readily attained that simple exposure of living tissue to certain chemical produces destruction of the cells. As far as the nature of any effect of a chemical on living tissue is concerned, effects may be of such minor significance that the tissue is able to carry on its ordinary function in a normal manner so that it is only under conditions of stress or critical tests that a chemical induced effect is even detectable. Effects may result from small amounts of some chemicals and required to produce any positive findings. Generally it is a simple matter to separate those relatively few chemicals that in small amounts produce prompt effects that are distinctly harmful to living cells from those that are practically harmless when exposure is over a short period of time, but it becomes difficult to demonstrate that small amounts of some compounds do not produce some types of toxicity when animals are exposed over a long period of time.

Most of the biological methods which have been developed in toxicology are the result of the practical need to obtain as much information as possible about the effects of chemical in so far as they may be pertinent to man's continued physical well-being. The continuing economic progress of the human race has been accompanied by a continuing increase in the numbers of chemical entities to which man is either in intentionally or unintentionally exposed. A person may be exposed through direct industrial of domestic occupational contact, through contact with the clothes or devices he wears, the food, he used and drugs he consumes and the atmosphere he inhales. It is necessary not only to understand the toxicities that can occur but also to obtain assurance that exposure of man to large number of chemical entities will mot lead to obvious direct or insidious indirect detrimental effects. Consequently , it is essential that chemicals which are to be intentionally administered to man, such as food additives, food substitutes or drugs, it is necessary to obtain as much toxicity data as is economical possible.

Because of the moral ethical and legal restrictions regarding the use of humans for experimental purposes in order to acquire toxicological data, only limited amounts of such data are available. Information regarding the effects of chemicals on human is obtained only after a chemical is used by human or from limited types of experimental procedures that may be conducted on humans. Biological methods in toxicology therefore generally involve the use of expendable species of animals on the hypothesis that toxicity studies in suitable species have an extrapolative value for man.

Several of the procedures involved in testing for toxicity involve the use of non-mammalian species and even cell cultures. It would be of great advantage to be able to utilize such species as bacteria, neurospora, daphnia, drosophila, the various echinoderms or fish for evaluation of toxicity because of the economic advantage and abundance of such populations of living cells. Furthermore some of these species lend themselves to accurate and simple procedures such as those that make use of their accurately defined and measurable genetic characteristics, reproductive processes and enzymatic performance. The main drawbacks associated with the use of such species are the dissimilarities in translocation barriers as compared to man and differences in or the lack of biotransformation mechanisms that are present in man. These factors preclude extrapolation of the data obtained on most non-mammalian species to man. Never the less such tests serve the purpose of alerting the investigator to potential toxic hazards which can then be further studied in mammalian species.

However when any chemical is used in massive quantities such as in agriculture and becomes available in the general environment, it is necessary to evaluate the toxicity of that agent in many species which may directly or indirectly influence the overall welfare of man. It should be recognized that there are many variations in both short and long term chemical-induced toxicity between various mammalian species of animals, however careful complete evaluation of the effects of chemicals on animals have been shown to be the most rational, acceptable and successful means of determining most types of toxicity for purposes of extrapolation to man. The principal exception is the rather unsuccessful evaluation of immunogenic types of toxicity. It is interesting to know that many workers before the 19th century described the actions of poisons and their antidotes, these studies seemed to lack the scientific approach. The first to undertake scientific studies on the harmful effects of chemicals on biological systems was M.J.B. Orfilia (1787-1853) a Spaniard at the University of Paris (USA today, 1989). He is regard as the father of modern toxicology and was the first to introduce quantization in the study of the actions of chemicals on animals and to consider toxicology as a separate discipline from pharmacology. Orfilia was the author of the first book on harmful effects of chemicals (in 1815). He not only studied and reported on the effect of chemical but also on the treatment of poisoning due to such chemicals.

Toxicology as a science has its basis in the science of chemistry and biochemistry and is dependent on the knowledge of physiology. Pathology is often regarded as part of toxicology because the effect of the chemical on the biological system may appear as macro- or microscopical deviations of the normal cell or organ.

Toxicology is an offshoot of, and closely related to pharmacology because a pharmacologist attempts to understand the beneficial effects of the chemical when used therapeutically as well as its harmful or adverse effects. There are three main divisions of toxicology namely, economic, forensic and environmental toxicology, each with its own specialist toxicologist.

2.1 Economic toxicology

This concerns the harmful effect of chemicals, administered to man or animals in order to produce a specific effect. This includes drugs which are administered to modify physiological functions or eliminate some bacteria/parasitic organisms in the body. The study of drug toxicity (including tests for toxicity) is a major area of economic toxicology. It includes studies on the safety of food additives and cosmetics. Some chemicals have selective action on biological organisms and are used by man to eliminate pests and insects (as pesticides and insecticides) which become the uneconomic species. The human which is protected from the effect of the pest and insects becomes the economic species. The effect of the chemical on both economic and uneconomic species also forms a part of economic toxicology.

2.2 Forensic toxicology

This concerns the medical and legal aspects of the harmful effects of chemical on animals, including man. The medical aspects refer to the diagnosis and treatment of the effects of the chemical and the harmful effects produced by it. In forensic toxicology, an attempt is made to identify the chemical in the tissue by chemical analysis and in case of death, to establish the cause or circumstances of death. Both intentional and accidental exposures to the chemical are of interest to forensic toxicologists. They develop methods for the management

and treatment of acute and chronic poisoning including the use of antidotes. These are specifically referred to as clinical toxicologists.

2.3 Environmental toxicology

This deals with the harmful effects to man and animals of chemicals that are present as contaminants of the environment. The chemical may be present in the air, water, soil or food. In the urban centers, because of the industrial activities, the environment may be polluted by particles or by gasses. Environmental toxicology is concerned with hazardous substances in the air, water or soil, the disposal of industrial waste and the protection of the environment and peoples either at home or at manufacturing sites from industrial emissions. The environmental toxicologist deals with the evaluation of the effects of and the establishment of the limits of safety of exposure to these chemicals i.e. estimating the health risks of a particular chemical.

3. Factors that affect the toxicity of chemicals

Among the factors that affect the toxicity of a chemical are the chemical, biological, genetic factors and the route of administration or exposure to the chemicals.

3.1 Chemical factors

The chemical structure determines the ability of the chemical to interact with specific receptors responsible for the observed effects. Biotransformation mechanism is dependent on the structure and may produce a metabolite that is more toxic or less toxic than the parent chemical. It may influence the excretion process of the chemical or its metabolite. It is known that some chemicals do induce or inhibit metabolizing enzymes and in this way modify their activities and those of other chemicals metabolized by the same enzymes.

3.2 Biological factors

Here the factors include biotransformation and elimination mechanisms, plasma protein binding, storage of the biological membrane through which it passes. Each of this will influence the toxicity. Age, sex, nutritional status of the individual and species of aminal also play roles too.

3.3 Genetic factors

Genetic differences may have a great effect on toxicity. For example, some individuals are genetically deficient in blood psendocholinesterase enzymes. Thus succinylcholine (a muscle relaxant) normally hydrolyzed by psendocholinesterase, will induce prolonged muscle relaxation in a person genetically deficient in the enzyme contrary to expectation.

The anti-cholinesterase's used in therapeutics' are generally those which reversibly inactivate cholinesterase for a few hours. Insecticides of the carbamate type act by reversible inhibition of cholinesterase but organophosphorous insecticides inhibit the enzyme almost or completely irreversible so that recovery depends on formation of fresh enzymes. This process may take weeks although clinical recovery is usually evident in days. Cases of poisoning are usually, after agricultural, industrial or transport accidents. Substances of this type have also been studied for use in war (nerve gas). The prominence of individual effects varies with different agents, e.g. sweating and salivation are not usual in dyflos poisoning.

A typical case of poisoning by cutaneous absorption, will, perhaps after a delay, develop headache confusion, anorexia and a sense of unreality. The patient is often giddy, apprehensive and restless. Conspicuous salivation, rhinorrhoea and sweating follow, with respiratory wheeze and dyspnoea indicating the onset of broncho constriction and excessive bronchial secretion. Miosis may occur and cause the headache, but it is not invariable nor is it an index of severity, for it may be due to a local effect of the poison entering via the conjunctiva. Vomiting and cramping abdominal pains may lead to diarrhoea and tenesmus, and there may also be urinary incontinence. Muscle twitching typically begins in the eyelids, tongue and face, then extends to the neck and limbs and is accompanied by severe weakness. Progressive respiratory difficulty leads to convulsions and coma. Death is due to a combination of the actions in the central nervous system, to paralysis of the respiratory muscles by peripheral neuromuscular block, and to excessive bronchial secretions causing respiratory failure. At autopsy, ideal intrsusception are commonly found.

4. Route of administration or entry into the body

Chemicals may enter the body through inhalation, by contact with the skin and by oral route. In addition, drugs may be administered by porenteral routes. The toxicity of a chemical may be many times greater through one route than through the other. A typical example is curare which is not absorbed orally and hence induces no toxicity. Its toxicity manifests itself when administered parenterally.

5. Toxicological effects

The toxicological actions of a chemical or materials may induce acute or chronic toxic effects or poisoning. Acute effects arise form an exposure to a chemical or an over dosage of drugs and the poisoning may be accidental, suicidal or homicidal. This is a toxicological emergency and demands emergency management, care and treatment.

Chronic poisoning is caused by ingestion of or exposure to the chemical over a period. In certain cases of occupational poisoning, ingestion of polluted water or inhalation of insecticides by farm workers results in chronic poisoning. It takes times to manifest itself and an equally long time to treat.

However chronic poisoning may produce an acute toxic effect which will necessitate toxicological emergency action and a chronic management of the patient until the body load is reduced or poison eliminated form the body.

6. The environment

The human environment contains many chemicals that are toxic to man and animals. It is one of the sources of health hazard and is responsible for various acute toxicities and many chronic illnesses. The environmental chemicals may be present in the atmosphere as air pollutants, in soil or water including the under ground water as contaminants and in food as residue or contaminants.

The sources of environmental hazardous chemicals are two, namely, natural sources and man-made sources. For the sake of this topic, emphasis is laid on man-made source.

6.1 Man-made sources

These arise from human activities and include chemicals that reach the atmosphere as a result of industrial activities. There are many chemicals that are present or are used in work places and therefore constitute occupational hazard. Many industrial activities pollute the atmosphere with particulate matters and gasses like carbonmonoxide, sulphurdioxide, hydrocarbon e.t.c. Some factory smoke stacks release particles which are deposited onto vegetables crops that are consumed by man.

Some potential toxic chemicals are normally found in the home as drugs, as pesticides. These are responsible for accidental poisoning among children. Studies in developed countries have shown that poisoning is the second or third most important cause of fatal accidents in the home (Backett, 1965). Pesticides used by farmers for enhanced food production may appear in the food and the same applies to food preservatives. In Nigeria, some farmers and traders used pesticides to preserve grains like beans, maize, rice, etc, in spite of the fact that pesticides are not approved for the preservation of grains. Through this source, pesticides are consumed by the unsuspecting public with possible acute or often chronic consequences. The pesticides used in the farm may be carried by rain, run-off to contaminate the streams and rivers in countries without the necessary controls.

There are many activities that routinely release hazardous chemicals to the environment. The petrol station attendant inhales benzene as petrol is put in the car and benzene is also released to the air. The exhaust fumes form motor vehicles release a number of chemicals including particulate matters in to the atmosphere. Such substance in the air create problems in the some developed countries like the USA to control and set limits of emission from motor vehicles for permit to ply the road.

The Natural Resources Defense Council (NRDC) of the USA estimated in 1989 that industry is pumping more than 361 million pounds of cancer producing chemicals into the air yearly (USA Today, 1989)

Over the years, human beings have tended to depend on some sudden and unexpected episodes to realize the dangers of environmental pollution from man-made sources. There are many instances which point to the fact that pollution can cause serious illness or death. In 1952 in London, a dense fog (SMOG) due to environmental pollution settles over the city for 4 days. This resulted in about 3,500 to 4,000 deaths in greater London alone (Klassen, 1990). Ten years later a similar episode occurred in London and caused many deaths particularly amongst the elderly and children (Akubue, 1997)

In the Meuse valley in Belgium in 1930, a heavy fog associated with very stable air mass caused severe respiratory symptoms and death, the death rate in the community during the period was 10 times more than normal. The air pollution was said to have come from the industrial plants in the neighbor hood,

In June, 1996 in Santiago, Chile, there was part of SMOG as air pollution reached an alarming level. There were many deaths from respiratory diseases. The city authorities ordered 300,000 cars off the roads in an effort to improve the quanlity of air.

There are also many instances of major accidental release of toxic chemicals in to the atmosphere.

In 1976, there was a massive exposure of the city and people of seveso, in Italy to 2, 3, 7, 8-tetra chlorobenzodioxin (TCDD) (Ottobin, 1991). This was as a result of an explosion in a manufacturing plant. Many people particularly children suffered chlodacne and thousands of animals like chicken, birds, dogs and horse died a few days after the explosion. It is believed that dioxins contaminants of herbicide, agents' orange, were responsible for many

health problems of Vietnam Veterans (Ottoni, 1991). Dioxins of which TCDDTS, the most toxic and most persistent poison, are by-product contaminants of many chemicals reactions and are formed during the manufacture of chemicals like trichlorophenols and the combustion of waste materials. Studies have shown that if TCDD is not a cancer inducer, it is a promoter. In Nigeria, there is no doubt that TCDD is formed as a by-product of chemical processes being carried out in the industries and as a product of combustion of waste materials. Hence, TCDD is in the air around us. The question that needs to be answered is, what have we done to bring its level to the bear rest minimum?

An explosion at an industrial plant in Bhopal, India in 1985 released into the environment methyl-isocyanate and this was responsible for the death of about 1,500 people (Keritage, 1992). This would not have happened if adequate safety measures were in place in the industry. Other toxic chemicals worthy of note are discussed below;

6.1.1 Dichlorodiphenyltrichloroethane

DDT is an organochlorine insecticides (also referred to as halobenzene derivative) which was widely used in agriculture and in malaria control. It is highly soluble in fats and poorly soluble in water. In the body it is stored in the fat depot. It is only slowly eliminated from the body. DDT is usually used as solution in organic solvents, especially kerosene. It is established that DDT increases the incidence of liver cancer in mice (Innes, 1969) there is yet no evidence for such an effect in humans. However, its use has been limited or withdrawn in many countries because of uncertainly of the effect of prolonged exposure and storage in man, beside, some insects developed resistance to it.

6.1.1.1 Symptoms

Symptoms of acute poisoning include vomiting tremor, and convulsion. There is anesthesia of the tongue, lips and face with marked apprehension and excitement. Diarrhoea may occur.

A study indicated that carbonate pesticides namely aldicarb, aldicarb sulfoxide, baygon, penthiocarb, carbofuran, 3-hydrocarbofuram, carbaryl, desmedipham, methiocab, methomyl, thiodicarb, oxamyl, and propham was made in ground and surface water from an agricultural zone of the Yaqui valley located in northwest Mexico. From the result of trace determinations made by liquid chromatograph (LC) with post-Column fluorescence detection, it showed that the level of contamination with methiocarb was about 5.4 mg/l in a ground water sample and that for 3-hydroxycarbofuran was 18mg/l in a surface water sample (Garcia-de-Llasera, 2001).

Carbofuran was estimated for an Acceptable Daily intake (ADI) in 1978 and 1979 and a temporary ADI for man was estimated to be 0-0.003mg/kg body weight (FAO/WHO, 1977; FAO.1980). The available data reflected that carbofuran is a highly toxic carbonate ester whose metabolic profile has been well defined. Carbofuran is a potent, reversible cholinesterase inhibitor. Chlolinesterase inhibition and acute toxic signs of poisoning are subject to rapid spontaneous reversal and recovery. The measurement and evaluation of cholinesterase depression induced by carbofuran, because of the rapid reversibility, is difficult and required substantial care.

Similarly, in a group of mice (100 male and 100 females, Charles River CD-1mice/group were fed carbofuran in the diet of dosage levels of 0, 20, 125, or 500 mg/kg for two years. It was reported that a localized hair loss and reddening of the ear(s) frequently followed by

scabbing or sloughing of portions of the ears was noted with greater frequency in the treated mice. (Pesticide residues in food: 1950 evaluation)

In a similar study using fenamiphos, a carbonate pesticide like carbofuran, exposure of rats to technical grade fenamiphos (purity, 92.2%) diluted with a 1:1 mixture of ethanol and polyethylene glycol 400 for aerosolozation in a dynamic flow inhalation chamber at doses of 0,0.03,0.25, or 3.5 mg/l for 6hrs per day, five days per week for three weeks showed a significant decrease in (48-7.9%) in plasma cholinesterase activity and a slight decrease (9-18%) in erythrocyte acetyl cholinesterase activity in animals of each sex at 3.5μg/l (Thysen, 1979b)

6.1.2 Benzene Hexachloride and Lindane

Benzene hexachloride (BHC) is a mixture of eight isomers with gamma isomer (Lindane) being the most toxic and active but most rapidly excreted. The toxic effects of Lindane resemble those of DDT. It is a CNS stimulant and a potent inducer of hepatic microsomal enzymes. BHC is said to cause aplastic anaemia (Akubue, 1997)

6.1.3 Polycyclic chlorinated substance chlorinated cyclodienes

The substances are many with varying toxicities. Examples are aldrin, dieldrin, heptachlor, Endosulfan and chlordane. They stimulate the CNS and induce convulsion. Before this, there may be headache with nausea, dizziness, vomiting and mild chronic jerking, and tremor ataxia. The CNS stimulation may be followed by depression which may end in respiratory failure. The insecticides have potentials as carcinogens and cause haematoma in mice. Hence their use in some countries (e.g. USA) is banned.

6.1.4 Organophosphorous insecticide

Organophosphorous insecticides are extensively used in agriculture. Through they do not persist in the environment; they can cause serious toxic effect in man, being potent and irreversible inhibitors of cholinesterase. The symptoms of poisoning are due to muscarinic, nicotinic and CNS effects.

In acute (mild to moderate) poisoning from ingestion or inhalation, the following symptoms can be as observed (headache, dizziness, tremor of the tongue and eyelids, miosis and impaired vision. These symptoms are followed by nausea, vomiting, salivation, tearing, abdominal cramps, and sweating, slow, pulse and muscle fasciculation. In severe poisoning, diarrhoea, pinpoint pupils not reactive to light, respiratory difficulty, cyanosis, convulsion, coma and heart block may be observed.

Chronic poisoning may occur and inhibition of cholinesterase can persist for up to 6 weeks. Delayed neuropathy occasionally develops after poisoning.

7. Rodenticides

Many drugs had been used to kill small animal like rats and mice. Amongst these are fluoroacetate, x-naphthylthiourea(Antu) and pindone (warfarin- like anticoagulant).

7.1 Fluoroacetate sodium

Fluoro acetate sodium is extremely toxic to rodents and to man and other animals. Fluoroacetate is present in the plant known as *Dichapetalum cymosum* which grows in

Nigeria and is used as rat poison. Fluoroacetate is too toxic for use in the home. It has been withdrawn from the market in some countries but fluoro actamide, which has the same toxicity, is still on the market.

The most prominent effects of a cute poisoning in man from ingestion or inalation of fluoroacetate are vomiting and convulsions. It induces irregular heart beat, exhaustion and coma. Death is usually due to respiratory failure.**7.2**

Pindone

This is a coumarin anticoagulant with actions similar to warfarin which is also used as rodenticide. In large doses, they can cause vascular collapse.

8. Herbicides

Herbicides are now used extensively to destroy noxious weeds. The most common ones are the following;

- 2, 4-dichlorophenoxyacetic acid (2, 4-D)
- 2, 4, 5- trichlorophenoxyacetic acid (2, 4, 5-T)
- Dinitrophenols
- Paraquat.

8.1 Chlorophenoxy compounds (2, 4-D and 2, 4, 5,-T)

Chlorophenoxy compounds are used to control broad-leaf weeds. They rarely cause toxicity in man though contact dermatitis is known to occur. The effect is due to contaminant called TCDD(2,3,7,8- tetrachlorodibenzo-p-dioxin).

8.2 Dinitrophenols

Dinitrophenols are also used extensively in weed control. The toxic effect is due to the uncoupling of oxidative phosphorylation. Thus the metabolic rate is increased with a subsequent rise in body temperature. Other symptoms of acute poisoning include nausea, restlessness, sweating tachycardia, fever rapid respiration, fever and cyanosis.

9. Fungicide

Most fungicides that are used to control fungal diseases on seeds and plants are not particularly toxic except the mercurials. However, some do produce toxic effects in man. A good example is seen in carbamates e.g aldicarb, aminocarb, carbofuran, propoxur and bendiocarb. Health hazards to man are mainly as a result of occupational exposure.

9.1 Dithiocarbamates

Both dimethyldithiocarbamate and ethylenebisdithiocarbamate have low acute toxicity. However, thay may have teratogenic as well as carcinogenic effect and can produce disulfiram-like effects when alcohol is ingested. It is known that in humans, in the environment and during cooking of contaminated food, the ethylenebisdithiocarbamate is broken down to form ethylenethiourea, which is carcinogenic and teratogenic.

Other chemicals used as fungicides include hexachlorobenzene and penta chlophenol. The toxic effects of pentacholophenol resemble those of nitrophenols which increase the metabolic rate by uncoupling oxidative phosphorylation.

10. Fumigants

Funigants are used in gaseous form as pesticides to reach areas that are inaccessible. Examples include cynide, crabondisulphide, carbon tetrachloride, thylene oxide, methylbromide and phosphine.

10.1 Cyanide

Cyanide as hydrogen gas, is used as a fumigant in ships and buildings. It is also used in industry in chemical synthesis and electroplating and as household pesticides and rodenticide. Large doses of hydrogen cyanide can cause rapid respiration, convulsion. Loss of consciousness and death within 5minutes: lower doses produce headache, staggering gait, dilated pupils, palpitation, unconsciousness, violent convulsion and death. Combustion of plastic materials releases hydrogen cycanide. This was the cause of death of 303 pilgrims in 1980 in Riyadh, Saudi Arabia (wager, 1983)

Cyanide in the body complexes with cytochrome oxidase and is responsible for cellular oxygen transport. In this way it interferes with oxygen uptake by the body cells.

10.2 Methyl bromide

Methyl bromide is a colourless gas used as a fumigant for soil, stored dried food stuffs and for disinfection of fresh fruits and vegetables.

Methyl bromide causes headache, blurred vision, weakness, oliguria, anuria, confusion, drowsiness, convulsion, and coma. There may be circulatory collapse or respiratory failure. With low doses, the symptoms may take 12 to 24 hours to appear.

10.3 Phosphine (PH_3)

This is used to fumigate grains. It is slowly released from aluminum phosphine tablets in the presence of atmospheric moisture. Poisoning by inhalation may cause a fall in blood pressure, pulmonary oedema, collapse, convulsion and coma. The first sign of chronic poisoning is toothache followed by swelling of the jaw and necrosis of the mandible (phossy jaw). There may be anaemia and spontaneous fractures.

11. Carcinogens

Chemical substances have been associated with cancer in man long before it was demonstrated experimentally in animals. For example the high incidence of cancer of the scrotum was reported amongst chimney workers in 1775 (Wolf, 1953). This was thought to be due to exposure to soot. Cancer was also recognized as an industrial hazard among coal tar workers. It is now generally accepted thatmany human cancers are directly and indirectly due to environmental factors which induces exposure to chemicals and ionizing radiations (Tomatis, 1976)

Chemicals known to be responsible for certain human and animal cancer are referred to as carcinogens. Some of these chemicals produce the same type of cancer in animals and in man but with some other chemicals there is species variation for example, 2- methylamine causes bladder cancer in man and dogs (WHO, 1972). Occupational exposure to chemicals is known to cause cancer mostly that of the skin, bladder, lungs and sinuses (WHO, 1972).

Cancer develops slowly and there may be along latent period (15-40 years (before the cancer is detected. It can occur at the site of application or at a site far away from the site of

contact. Certain carcinogenic agents are not themselves carcinogenic until metabolized to an active product which is called proximate carcinogen.
It is also well established that chemical like polycyclic hydrocarbons e.g. 3.4- benpyrene present in coal tar) and 3, 4-benzphenanthrene azo dyes e.g. Dimethylnitrosamine are potent carcinogens. Thus a very wide range of chemical structures can induce carcinogenicity. This has made determination of the mechanism of induction rather difficult. It is believed that most organic carcinogens react covalently with macromolecules in the tissue. Others are converted to reactive metabolites which contain an electrophilic atom. It is this reactive (elctrophilic) group in the carcinogen or its metabolite that reacts with any nucleophilis atom of the target macro molecules of the cell. The nucleophilic site is on the DNA, RNA, and/ or protein. Carcinogens can react covalently with DNA in vivo directly or after conversion to the reactive group.
The problem of carcinogens is very great in developing countries where control measures and legislation are not available to protect the public from the industrial hazard of these chemicals and from the use of such chemicals in foodstuffs, cosmetic and agriculture. Many local industries do not even try to protect their workers and the workers, out of ignorance do not protect themselves. It may be realized that even when chemical is declared non-carcinogenic in other developed country, because of our genetic make-up and because of other promoting factors which may be presented in our environment may be carcinogenic in our surroundings. It is therefore imperative that all chemicals in use for whatever purpose should be monitored for possible carcinogenic effects (Akubue, 1997)

12. Chemical mutagen

A change in the hereditary constitution (otherwise called genotype) of the individual is known as mutation. This may be produced by chemicals and also by radiation and hence called mutagens. Mutation can occur spontaneously by unknown mechanisms.
Mutagens act by altering the genetic make-up of the individual and this can be handed down to the offspring during cell division. This means the new cell will have heritable characteristics. The mutation may occur as a result of alteration of one or more nucleotide or changes in number as structure of chromosomes.
In general, two kinds of mutation can be recognized: Chromosome and gene mutation. A chromosome mutation may be of several types e.g. Deletion, duplication inversion, translocation (Roberts, 1982).
However, on some occasions, two homologous chromosomes, instead of separating during cell division, called meiosis, go off into the same gamete. This phenomenon is known as non-disjunction and it results in half the gametes having two of the chromosomes whilst the other half has none. The fusion of the first kind of gamete with a normal gamete of the other sex will give an individual with three such chromosomes i.e. the normal pair plus an extra one. This condition is called trisomy.
Quite how important non-disjunction has been in generating useful genetic novelty is uncertain, but there is no doubt it can have profound effect on an organism's development. For example, mongolism (better called Down's syndrome after the clinician who first described it) is now known to be caused by the presence o f an extra chromosome in the cells, chromosome number 21 to be precise. This is one of the smallest of all the human chromosomes, and yet its presence plays havoc with the individual's normal development. Sufferers, form Down`s syndrome, if they survive at all, have a characteristically slit-eyed

appearance, reduced resistance to infection and are always mentally deficient, all because of one extra chromosome.
The short description above is a tip of an iceberg of the several havoc pesticides can cause to the body.
A gene mutation arises as a result of a chemical change in an individual gene. An alteration in the sequence of nucleotides in that part of a DNA molecule corresponding to a single gene will change the amino acids making up a protein, and this can have far- reaching consequences on the development of an organisms.
Drastic effects can some times be produced by a seemingly trivial change in the nucleotide sequence of a gene. This can be seen, for example in the formation of hemoglobin. In the inherited disease known as sickle cell anaemia, the red blood cells, normally biconcave discs, are sickle-shaped and the victims suffers all the symptoms of extreme oxygen shortage, weakness, emaciation, kidney and heart failure.
The anemia is not caused by a distorted shape of the red blood cells as such, but by the fact that they contain abnormal haemoglobin, haemoglobin S, which inefficient at carrying oxygen. Gene mutation can be by deletion, inversion substitution.
Note that although the addition of whole chromosome as seen above may have been disastrous more often than it has been helpful, it is highly likely that the addition of new genes within individual chromosomes (insertion) has been extremely important in promoting evolutionary novelty. In general terms it is important to appreciate that variation is not always disadvantageous (Roberts, 1982)
Where the action of the mutagen is in the human germ cells (i.e. spermatozoa or ova), the offspring will carry the mutant genes in its cells. This may result in death of the zygote) or abnormal offspring. The effect of the mutation appears only in the offspring and may take a few generations before it manifest itself.
Mutation may also affect somatic cells. The effect is not passed on to future generations but may be responsible for carcinogenesis. For example, the alteration in genetic material may cause cell division in cells that normally do not divide during adult life. Such a division will eventually lead to cancer.
Though it is not known how many gene mutations in man can be attributed to chemicals it is acknowledged that many chemicals can cause gene mutation in micro-organisms and in insects. Also many chemicals can cause chromosome aberrations in man and can interact with nucleic acids. This may be a pointer to the fact that there are many chemical mutagens in our environment.
Among the chemicals shown to be mutagenic in mammalian cells are ethyleneoxide, Ziridine, aminobiphenyl, dimethylsulphate, benzidine e.t.c (Fischbein, 1979)

13. Teratogens

Teratogens are agents that cause abnormalities of foetal development. They usually interfere with the development of the foetus at a dose that produce no serious toxic effect on the mother or impairment of placental function. However, in high doses most of them will cause foetal death followed by abortion or resorption of the foetus.
One of the earliest reports of environmental factors being responsible for the birth of malformed babies came form Australia 1940 (Gregg, 1941). Mothers who suffered form the mild virus disease (rubella) during the first trimester of pregnancy gave birth to blind or deaf children. It was found that if the infection occurred after the third mouth of pregnancy,

no abnormality developed. This was followed about twenty years later by the thalidomide disaster of the early sixties when a seemingly non-toxic drug produced teratogenic effect. Some environmental pollutions, like chemical defoliants, 2, 4, 5-T (2, 4, 5-trichlorophenoxyacetic acid), are teratogenic in animals. Their effects in man are not yet fully established.

Evidence for the teratogenic effect in man of organophosphorous insecticides is still inconclusive though such an effect in animal is well established. Lead which is an environmental pollutant may be teratogenic (Scalon, 1972).

From the above account it is obvious that a lot needs to be known about chemical teratogens. It is not unlikely that most of the congenital malformations encountered in a community may have been due to environmental chemicals (Akubue, 1997). As teratogenic effect cannot be reversed or treated, the only right approach is to take necessary steps to prevent it. This is best achieved by avoiding occupational exposure to environmental chemical during pregnancy, particularly during the first trimester.

Developed countries do monitor the activities of industrial companies with regard to pollution and waste disposal. In 1992, Dexter corporation in the USA was fined thirteen million dollars (US$ 13 million for activities in its manufacturing plant in Windsor Locks Connecticut, USA, (EPA, 1990) with regard to illegally disposing of carbondisulphide and discharging hazardous waste and waste water into a river.

The above instances of the effect of environmental pollution and the action taken by a government environmental protection agency against a manufacturing company are presented to draw the attention of all sundry to the time bomb we are setting on. Nigeria and all developing countries must learn from the mistakes of the developed countries. We try to industrialize, we must not repeat the mistakes which other countries made in the past and paid heavily for them.

Apparently, in developing countries like Nigeria where increasing use of pesticides is absolutely vital for it purpose as to protection of economically important crops such as tobacco, cotton, rice and so on with little attention paid to its deleterious effects (Carlson et al., 1998 Roex et al., 2001) and for feeding increasing large populations adequately, some recommendation have made by Iyaniwura, (1991b) in order to maximize the benefits at the least risk to human population. They include;

The increasing need for extension workers to embark on the training of farmers in the choice of pesticides, storage, applications, technique and the use and disposal of spent container become very important in this area.

The need for the development of preventive measures, diagnostic tests and treatment facilities in pesticide poisoning will go along way in helping farmers.

That training of physicians and health workers in high risk areas should includes instruction on the diagnosis and treatment of pesticide poisoning.

Trained scientist specialize in the treatment of victims of pesticide poisoning should be made available to health officials and hospital at those locations where pesticides are being used.

It is worthy of note here that regulations or restrictions of use and production of the chemical itself need to be enforced to avoid damage to man, animals, insects or wild life generally. In this regulations, various tests are performed including; chemical characteristics, toxicological characteristics physiological and biochemical, behavioral, environmental, ecological and tolerance assessment.

Pollution cause immediate health effects, in many cases, the effect may be delayed for 5 to 10 years or more. The greatest danger is that the effect may be unnoticed until irreversible

damage is done. This is worrying in our circumstances. In developing countries specifically, Nigeria, an average Nigerian does not feel threatened by any chemical or any operation that has no acute toxic effect. It is known that the health effect may appear long after the person has left the area of exposure. By this time, the health effect may not be associated with the exposure.

Some of the effects that are known to be induced by pesticides in humans includes cancer (various types) infertility, liver damage, kidney damage, premature death, hyperactivity in children, bronchitis, defective sight and blindness, birth defects, blood diseases, nervous system damage, heart defects and sudden death.

These illnesses are very devastating with high morbidity and mortality rates but as they are preventable, they are best prevented. The cost of prevention is next to nothing compared with the cost of treating the ailments or managing the in curable ones.

14. Biopesticides and human health

People have been using biotechnology for millennia. This technology is based on the use of microorganisms, which e.g. ferment the sugar in barley to alcohol during beer production. Other examples of everyday products that undergo biotechnological processing are cheese, yogurt, vinegar, wine, yeast, and sourdough. Without knowledge of the exact backgrounds, our ancestors used these methods to discover and improve a range of applications that made their life easier. Genetic engineering is a modern subspecialty of biotechnology. It is concerned with the targeted modification of the genetic material of bacteria or plants, for example to stimulate them to biosynthesize desired products. Today genetic engineering is primarily used in the field of medicine, but is also applied in industry and agriculture.

Biotechnology is the science that modifies the genetic composition of plants, animals and microorganisms. It is used to incorporate genetic material from one living organism to another. Biological pesticides are produced through the use of biotechnology by harnessing the pest-fighting abilities of existing plants and microbes. When these products have unique biological properties, they also pose unique regulatory challenges. In addressing these challenges, the Environmental Protection Agency (EPA), the U.S. Department of Agriculture (USDA), and the Food and Drug Administration (FDA) have shared responsibility for regulating agricultural biotechnology in the United States. For instance, EPA regulates pesticides created through biotechnology as a part of its regulatory jurisdiction over all pesticides marketed and used in the United States. As such, EPA has tailored its basic regulatory framework to fit the distinctive characteristics of these genetically engineered biological pesticides (EPA, 2003). In theory, biotechnology could be used to prevent pest problems and thus reduce the need for pest management and pesticide use. Since the beginning of agriculture, plant breeders have developed crop varieties that were resistant to or tolerant of particular pests. For example, cotton varieties with long, twisted bracts (called frego bracts) around the bolls are resistant to boll weevil damage and solid stemmed wheat varieties are not damaged by the wheat stem sawfly (Cox, 1993).

The tools of biotechnology could be used to make plant breeding easier and quicker. Genetically engineered crop plants will be in farmers' hands in the next few years. We are therefore at a critical point where we need to evaluate this new technology and the impact it will have to our agricultural systems and human health. What problems will the new technology bring? However, genetic engineering has the tendency to move agriculture in

the opposite direction, towards maintaining or increasing present pesticide use patterns. The total world production of biopesticides is over 3,000 tons/yr, which is increasing at a rapid rate. India has a vast potential for biopesticides as it utilizes more than 100,000 tons (1992-93) of pesticides annually. Most (80%) of the pesticides are used on cotton (45%), rice (30%) and vegetables (5%), the remaining crops receiving only 20% as share.

14.1 Benefits of biopesticides

International organisations and bodies such as the United Nations Food and Agriculture Organisation (FAO), the World Health Organisation (WHO) and the International Council for Science, as well as a number of national food safety authorities and medical associations, have all positively commented on the safety and/or benefits of agricultural biotechnology. There are direct environmental benefits which arise from the different management techniques plant biotechnology makes possible. For example, herbicide-tolerant crops facilitate the use of no-till agriculture, which reduces both soil erosion and energy inputs. At the same time, soil organic matter is maximized, which reduces agriculture's contribution to global emissions of greenhouse gases, linked to climate change. Pest-resistance reduces the need for spraying, with consequent benefits to non-target organisms and overall biodiversity. Also, by making farming more efficient on limited land area, plant biotechnology contributes significantly to preventing habitat destruction – the biggest single threat to biodiversity.

Organic food sales are increasing because the public is willing to pay more for pesticide-free vegetables, fruits and dairy products. Biotech based pesticides therefore are becoming viable alternatives to chemical pesticides. For instance a biocidal product of plant origin that can combat insects as well as fungus and bacteria would find ready acceptance in the hand of farmers for the control of diseases and pests. It could be grown by farmers themselves and the seeds can be turned to organic pesticides. Is it the spraying of yeast formulation on fruits before they are transported to selling centres to keep the fruits fresh? Biotech pesticides or biopesticides tend to harness nature for solving health problems of agricultural crops. These substances used in controlling pests in crops are ground water and environment friendly. They are used in small quantities. Crop yield are not affected by them. As they are of natural origin, they mutate hence their use result in substantial labor cost savings.

It is important to know the mode of action of biopesticide as compared to traditional pesticide prior to using it. A biopesticide cannot be considered as non toxic because it is natural in origin, but can generally be considered only as less toxic to humans.

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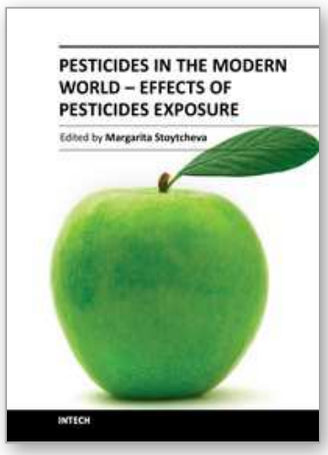

Pesticides in the Modern World - Effects of Pesticides Exposure

Edited by Dr. Margarita Stoytcheva

ISBN 978-953-307-454-2
Hard cover, 376 pages
Publisher InTech
Published online 12, September, 2011
Published in print edition September, 2011

The introduction of the synthetic organochlorine, organophosphate, carbamate and pyrethroid pesticides by 1950â€™s marked the beginning of the modern pesticides era and a new stage in the agriculture development. Evolved from the chemicals designed originally as warfare agents, the synthetic pesticides demonstrated a high effectiveness in preventing, destroying or controlling any pest. Therefore, their application in the agriculture practices made it possible enhancing crops and livestockâ€™s yields and obtaining higher-quality products, to satisfy the food demand of the continuously rising worldâ€™s population. Nevertheless, the increase of the pesticide use estimated to 2.5 million tons annually worldwide since 1950., created a number of public and environment concerns. This book, organized in two sections, addresses the various aspects of the pesticides exposure and the related health effects. It offers a large amount of practical information to the professionals interested in pesticides issues.

How to reference

In order to correctly reference this scholarly work, feel free to copy and paste the following:

Alewu B. and Nosiri C. (2011). Pesticides and Human Health, Pesticides in the Modern World - Effects of Pesticides Exposure, Dr. Margarita Stoytcheva (Ed.), ISBN: 978-953-307-454-2, InTech, Available from: http://www.intechopen.com/books/pesticides-in-the-modern-world-effects-of-pesticides-exposure/pesticide-and-human-health

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