1.7 Toxic agents

1.7.1 Pesticides

Pesticides can be defined as a class of compounds, which possess the ability to suppress or eliminate pests, without causing damage to the growth of desired organisms. For instance a farmer will not tolerate his crops being damaged by fungus so he will apply fungicides to eliminate the fungal population. Pesticides thus include fungicides, insecticides, nematicides, molluscicides, and rodenticides that are used on organisms that come under the category of pests. Pests are species whose existence conflicts with peoples profit, convenience or welfare (Clarke *et al.*, 1967).

Pesticides include a wide range of chemicals such as organo-chlorines, organo-phosphates, carbamates, thio-carbamates, triazines, amides, phenols, arsenic, copper, mercury and bromine compounds. These are sprayed in the air or on crops and find their way into the soil. The resistant nature of these compounds is a major problem and is included under persistent organic pollutants (POPS). All these chemicals are hazardous and reach lethal concentration through bioaccumulation and biomagnification. The situation is aggravated by the by-products from pesticide manufacturing industries such as dioxins, chlorine compounds and mercury, which are highly toxic.

The episode of toxic gas leakage in Bhopal, M.P., on December 3rd, 1984 involved methyl isocyanate (MIC), which is used in the manufacture of carbaryl or sevin. Though the cause of the tragedy is still shrouded in mystery, scientific explanation suggests that the accidental addition of water in MIC storage tanks could be the cause of the tragedy. MIC is stored with phosgene, which prevents its polymerization. Addition of water causes causes phosgene to react with water molecules to produce hydrochloric acid. HCl catalyses polymerization of MIC, a reaction that is highly exothermic. Accidental addition of water triggered the reaction, generating enormous amounts of heat releasing about 40 tons of MIC. MIC is an extremely poisonous substance and thus the leakage cause the death of about 2890 people and about the same number of cattle, while 200000 were affected with chronic poisoning (the symptoms included chest congestion and pain, breathing trouble and a burning sensation of the eyes. This gives us an idea of the substances that are being injected into the environment and the potential hazards that they can create.

Effects

Acute health problems may occur in workers that handle pesticides, such as abdominal pain, dizziness, headaches, nausea, vomiting, as well as skin and eye

problems. Studies have examined the effects of pesticide exposure on the risk of cancer. Associations have been found with: leukemia, lymphoma, brain, kidney, breast, prostate, pancreas, liver, lung, and skin cancers. This increased risk occurs with both residential and occupational exposures. Increased rates of cancer have been found among farm workers who apply these chemicals. A mother's occupational exposure to pesticides during pregnancy is associated with an increase in her child's risk of leukemia, Wilm's tumor, and brain cancer. Strong evidence links pesticide exposure to worsened neurological outcomes. The risk of developing Parkinson's disease is 70% greater in those exposed to even low levels of pesticides. People with Parkinson's were 61% more likely to report direct pesticide application than were healthy relatives. Both insecticides and herbicides significantly increased the risk of Parkinson's disease. There are also concerns that long term exposures may increase the risk of dementia Strong evidence links pesticide exposure to birth defects, fetal death and altered fetal growth. In the United States, increase in birth defects is associated with conceiving in the same period of the year when agrochemicals are in elevated concentrations in surface water. Agent orange, a 50:50 mixture of 2,4,5-T and 2,4-D, has been associated with increased birth defects in Vietnam. A number of pesticides including dibromochlorophane and 2,4-D has been associated with impaired fertility in males.

1.7.2 Heavy metals

Given below is a brief description of the source and effects of some heavy metals:

(i) Lead : Lead was used extensively in the 1950s for the production of water pipes, since then it has been replaced by other materials. The WHO- recommended guideline for lead in drinking water is 0.05 mg/l. At present the chief source of lead in water are the effluents of lead from paint industries, lead acid cells, and from the atmosphere with rains. The atmosphere receives lead through burning of fossil fuels and from automobile exhausts. Lead is generally added to petrol as an anti-knock agent. Nowadays lead free petrol is being used in order to check lead pollution.

Lead poisoning is common among both children and adults. The metal is absorbed either through the gastro-intestinal tract or the lungs. The slow buildup of the metal in the body is known as plumbism. More than 90 percent of the lead absorbed goes to the blood from where it is redistributed to the liver, kidney, and bones. Chronic lead toxicity may result in three types of effects:

(i) **Gastro-intestinal disorders**: accompanied by diarrhoea and often bleeding in severe cases. Finally there may be a failure of blood circulation and liver and kidney functions. The patient may pass into coma followed by respiratory-cardiac failure. The best known effect is chronic nephritis, a disease characterized by a scarring and shrinking of kidney tissue.

(ii) Neuromuscular effects: through degenerative changes in motor nerves and ganglia. This decreases the speed of conductance of the nerve impulses to and from the muscles. The individual may become dull, slow, and inactive.

(iii) Central nervous system effects: Lead may pass through the blood-brain barrier and affect the central nervous system. Two different mechanisms appear to be involved in lead encephalopathy, or brain damage; edema and direct injury to nerve cells. The walls of the blood vessels are affected so that the capillaries become too permeable; they leak causing edema (swelling of the brain tissue). Since the brain is enclosed in a rigid container, severe swelling destroys brain tissue. Moreover, it appears that certain brain cells may be directly injured, or their function inhibited by lead. In growing children degeneration of intellect and mental retardation may occur. In severe cases it may lead to coma and death.

Lead is also capable of passing through the placenta to the foetus to cause developmental abnormalities and stillbirths.

Lead is generally stored in the bones and as inclusion bodies in the kidneys. The clearest manifestation of the inhibitory effect of lead on the activity of sulphydryl – dependent enzymes is the disturbance it causes in the biosynthesis of heme. Heme requires six steps for its formation. Two of the steps are inhibited by the presence of lead. Lead is implicated specifically in the metabolism of delta – aminolevlinic acid (ALA: step 2) and in the final formation of heme from protoporphyrin . Both these steps are mediated by enzymes that are dependent on free sulphydryl groups for their activity and are there fore sensitive to lead. The functional effect of lead poisoning is anaemia. The decrease in heme synthesis leads at first to a decrease in the life span of red blood cells and in the amount of haemoglobin per cell (Chisolm, 1971).

(ii) Mercury: A lot of attention has been focused in recent years on the problems of mercury poisoning. Mercury enters the biosphere as a waste product from a number of industrial activities (paints, agriculture, dental, pulp and paper, pharmaceuticals, metallurgy and mining, caustic soda). The single largest use of mercury is in the manufacture of sodium hydroxide and chlorine by the electrolysis of brine, and the emission from these plants is quite moderate. Strict emission

controls have since been introduced, and have resulted in alternative processes of manufacture and the reduction in the amount entering the biosphere.

Inhalation of mercury vapour is injurious and in acute cases it causes irritation and destruction of lung tissues, with symptoms including chills, fever, coughing and a tight feeling of the chest, and there have been reports of fatalities from such exposure (Goldwater, 1972). The soluble inorganic salts of mercury (for instance, mercury bichloride) produce corrosion of the intestinal tract, injury to the kidneys, suppression of urine and ultimately death through kidney failure.

The inorganic mercury lost from various industrial processes enters he aquatic system where anaerobic bacteria convert it into methyl mercury. This is then converted into dimethyl mercury by aerobic bacteria. Methyl mercury is highly persistent and thus accumulates in the food chain. It is lipid soluble and so readily accumulates in fatty tissues. Mercury was responsible for the Minamata epidemic that caused several deaths in Japan. The tragedy was due to consumption of heavily mercury-contaminated fish (about 10-12 mg per kg of fish) by the people living in Minamata bay. The source of mercury in the bay was a single chloride plant using mercuric chloride as a catalyst.

The target organ of methyl mercury is the brain where it disrupts the blood brain barrier, upsetting the functioning of the nervous system. The neurons of the cerebral cortex undergo degenerative changes and necrosis. This results in tremors, inability to coordinate voluntary movement, paralysis, impairment of vision, loss of hearing, coma, and finally death. Methyl mercury has teratogenic effects and is capable of inducing abortions and other embryological effects (Goldwater, 1972). It also leads to the damage of chromosomes.

In agriculture mercuric chloride ($HgCl_2$) is used to infect seeds and to control diseases (principally to prevent fungal diseases in seeds). The largest epidemic of mercury poisoning occurred in 1971-1972 in Iraq when scarcity forced people to eat wheat grains treated ethyl mercury based fungicide (ethyl mercury p-toluene sulfonanilide. The average ethyl mercury content of these grains was about 4.8 - 14.6 mg per kg. Mothers exposed to sublethal doses gave birth to retarded offsprings and secreted mercury with their milk for long periods (Bakir *et al.*, 1973)

Deliberate or accidental ingestion of divalent mercury salts causes corrosive ulceration, bleeding and necrosis of the intestinal tract. Severe abdominal cramps, diarrhea, and suppression of urination follow. If the patient survives the gastro-intestinal damages, kidney failure occurs within twenty hours due to necrosis of proximal tubular epithelium.

(iii) Cadmium: Cadmium gains entry into the environment from mining and metallurgical operations, electroplating industry, industries manufacturing

polyvinyl chloride, plastics, nickel-cadmium cells but the main contributor is the electroplating industry. As cadmium is found associated with zinc, copper, and lead in mineral deposits, it is released into the environment during mining and processing operations of these metals.

Cadmium ranks next to mercury in its toxicity. The WHO recommended maximum is about 0.01 mg per litre. The toxic effects of cadmium received widespread attention as a result of the development of the disease Itai-Itai byo (ouch ouch disease) in Japan. The name comes from the severe pain developed by the sufferers as lumbago-type pain progresses to severe bone damage with multiple fractures of the softened bones. In severe cases skeletal deformities occur.

Cadmium is a potent enzyme inhibitor. It interacts with the sulph-hydryl groups of several enzyme systems and forms metal protein complexes within the cell, a property that is responsible for its accumulation in kidneys and other tissues without any obvious symptoms of toxicity. These cadmium protein complexes are termed as metallothienins. It has been suggested that the toxic effect occurs only when exposures exceed the capacity of the cellular system to provide enough proteins to bind the excess cadmium. It is free cadmium ions, which are responsible for the ensuing toxicity.

Cadmium is a slow acting poison. The toxicity curve for rainbow trout exposed to cadmium is linear over a concentration range between 1and 64 mg per litre and a time period of about six days. Continuing the test for 14 days revealed that cadmium continues to act lethally down to 0.01 mg per litre and the threshold concentration may lie as low as 0.008 mg per litre (Abel, 1989).

The mechanism of cadmium toxicity is probably due to its high affinity to sulph-hydryl groups and ligands containing nitrogen. Therefore, binding with such groups in chemical systems, makes the control function of organisms vulnerable to cadmium toxicity, even at low concentrations.

The main target organs for cadmium are the liver and the kidney. Critical effects occur when a concentration of 200µg per gram (wet weight) is reached in the kidney cortex. Since the excretion of this metal is slower than the rate of entry, it tends to bioaccumulate and is biomagnified as it moves through the food chain. It causes serious histological damage to the liver and ovary in fishes. Cadmium tends to accumulate in the liver, kidney, and thyroid.

In man, consumption of cadmium salts results in cramps, nausea, vomiting, and diarrhoea (Nordberg, 1972). Although the potential of cadmium for carcinogenic and teratogenic effects have been well documented in animals however, observations on human subjects are still inadequate.

(iv) Chromium: Chromium is generally an abundant element in the earth's crust, and occurs in oxidation states ranging from Cr^{2+} to Cr^{6+}. However, the trivalent

and hexavalent forms are industrially more important. Chromium metal and its salts are used in the production of stainless steel, chrome alloys, tanning, dyeing, and as an anti-corrosive agent.

Trivalent and hexavalent forms of chromium are of ecological significance. Trivalent chromium is the most common form of the metal in nature and it is in this state that the metal always occurs in the living system.

Acute chromium toxicity causes serious renal tubular necrosis. Exposure to hexavalent chromium has been found to cause dermatitis, allergic skin reactions, ulceration of the nasal septum and gastro-intestinal tract (Langard and Norseth, 1979). Chromium toxicity has been associated with carcinogenic and teratogenic effects. However, it is not certain as to which form of chromium is more potent. At present however, both forms are considered equally effective as a carcinogenic agent (Norseth, 1981).

(v) Arsenic: Arsenic is ubiquitous in distribution. In limestone and siliceous deposits its concentration ranges between 0.5-2.0 ppm while in volcanic rocks as much as 20 ppm of arsenic occurs. Natural processes such as weathering of rocks release about 80,000 tons of arsenic into the environment. Global emission of the metal due to anthropogenic activity has been estimated to be about 240,000 tons annually.

The WHO recommended maximum allowable concentration is about 0.05 mg per litre. However problems occur in areas where the concentration is higher and the intake increases to 0.20-0.35 mg per litre. Arsenic poisoning is frequently reported from West Bengal where not only surface water but also ground waters have registered high arsenic levels. A Rs. 1.5 crore multi-pronged training and awareness programme has been recently launched in West Bengal by the All India Institute of Hygiene and Public health in collaboration with WHO to tackle the arsenic problem. People residing in 61 blocks in six districts of the state have been affected by the arsenic contamination of ground water, while a much larger number are residing in the arsenic risk zones of the state.

Arsenic is a cumulative, potent, protoplasmic poison and inhibits SH- groups in enzymes. Arsenic is absorbed through the lungs, skin, and gastro-intestinal tract. It causes diarrhea, peripheral neuritis, conjunctivitis, hyperkeratosis, and lung and skin cancer. The carcinogenic properties of arsenic as a sequel to prolonged exposures to low doses have been well documented (Tseng, 1977). Chronic exposure to arsenic leads to eczema like changes characterized by a blackening and mottling of the skin and is known as the black foot disease.

(vi) Nickel: Nickel originates mainly from alloy producing, electroplating, and ceramic industries. Apart from these sources combustion of fossil fuel releases about 70,000 tons of nickel into the environment.

The most common effect of nickel exposure includes dermatitis and respiratory disorders. Nickel tetracarbonyl formed as a result of the reaction with carbon monoxide is highly carcinogenic. As is usual, tumour induction occurs as a result of prolonged exposure to such compounds.

(vii) Copper: Copper is widely used in the production of dyes, paints, pigments, ceramics, and pesticides. Copper contamination of the environment is largely due to its release by industrial units producing non-ferrous metals, fertilizers, and from mines. Copper sulphate and stabilized copper compounds are extensively used in the treatment of water reservoirs to control the growth of algae and diatoms. Copper may also enter the aquatic environment from agricultural units sprayed with pesticides such as Bordeaux (which contains copper). This may in turn enter nearby water bodies with runoff water. Copper is extremely poisonous to fishes.