***Environmental Toxicology***

It refers to the study of poisons/pollutants and their action on the environment.

**pollutant** may be defined as a substance or effect which adversely alters the environment by changing the growth rate of species, interferes with health, comfort, facilities or property values of the people.

In general pollution causes degradation and/or damage to the natural functioning of the biosphere which may involve vegetation, animals and all other species, crops, soil, water and ultimately altering the food chains.

The presence of these compounds in the environment may lead to:

•air pollution

•water pollution

•soil pollution

**AIR POLLUTANTS**

Air pollution is a reality of the twenty-first-century lifestyle. It can be classified into:

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| **1. Outdoor Air Pollution**  The ambient air is contaminated with mixture of gases including: Sulfur dioxide, carbon monoxide, ozone, nitrogen dioxide, lead and particulate matter.  **Sulfur Dioxide**  Sulfur dioxide is a water-soluble irritant gas that is absorbed predominantly in the upper airways and stimulates bronchoconstriction and mucus secretion in a number of species, including humans.  The penetration of SO2 into the lungs is greater during mouth as opposed to nose breathing. An increase in the airflow rate further augments penetration of the gas into the deeper lung. As noted with asthmatics, are likely to experience greater irritation. Once deposited along the airway, SO2 dissolves into surface-lining fluid as sulfite or bisulfite and is distributed readily throughout the body. It is thought that the sulfite interacts with sensory receptors in the airways to initiate local and centrally mediated bronchoconstriction and narrowing of the airways.  The conversion of SO2 to sulfuric acid is favored in the environment. |

Sulfuric acid irritates respiratory tissues by virtue of its ability to protonate (H+) receptor ligands and other biomolecules. This action can damage membranes directly or activate sensory reflexes that initiate inflammation.

Asthmatic persons appear to be somewhat more sensitive to the bronchoconstrictive effects of sulfuric acid than are healthy individuals. Asthma generally is characterized by hyperresponsive airways, and so the tendency of those airways to constrict at low acid concentrations is expected.

**Carbon Monoxide**

Carbon monoxide is a colorless, odorless and tasteless gas. It is produced by imperfect oxidation of carbonaceous materials. It is classed toxicologically as a chemical asphyxiant because its toxic action stems from its formation of carboxyhemoglobin, preventing oxygenation of the blood for systemic transport.

**Ozone**

It is one of the most intractable air pollutants. It is a gas formed by sunlight-driven reactions involving nitrogen oxides, which are released mostly by automobile exhaust. Together with oxides and fine particulate matter, it forms the "smog". Its toxicity is related to the formation of free radicals which injure epithelial cells along the respiratory tract and alveolar cells.

**2. Indoor air pollution**

There is growing awareness of the potential for indoor air pollution to elicit adverse health effects. The responses to indoor air pollution appear to be affected by ambient comfort factors such as temperature and humidity.

The suspected causes include combustion products, household chemicals, biological materials and vapors, and emissions from furnishings. The perception of irritancy to the eyes, nose, and throat ranks among the predominant symptoms and can become intolerable with repeated exposures.

Some toxic inhalants may be classified in this group, such as CO, NO2, and many VOCs (Volatile organic compounds are common to the indoor air arising from chlorinated water or dry-cleaned clothes).

**Nitrogen Dioxide**

Nitrogen dioxide is a deep lung irritant that can produce pulmonary edema if it is inhaled at high concentrations. Potential life-threatening exposure is a real-world problem for farmers, as significant amounts of NO2 can be liberated from silage. Nitrogen dioxide is also an important indoor pollutant, especially in homes with unventilated gas stoves or kerosene heaters. Side stream tobacco smoke also can be a source of indoor NO2.

Damage to the respiratory tract is most apparent in the terminal bronchioles. At high concentrations, the alveolar ducts and alveoli also are affected. There is also damage to epithelial cells in the bronchioles, notably with loss of ciliated cells, as well as a loss of secretory granules in Clara cells. The pattern of injury of NO2 is quite similar to that of O3, but its potency is about an order of magnitude lower.

**SOIL POLLUTANTS**

Soil contamination or soil pollution is caused by the presence of xenobiotic (human-made) chemicals or other alteration in the natural soil environment. It is typically caused by industrial activity, agricultural chemicals, or improper disposal of waste. The most common chemicals involved are petroleum hydrocarbons, solvents, pesticides, lead, and other heavy metals. Contamination is correlated with the degree of industrialization and intensity of chemical usage.

***Sources***

1. **Natural** –local accumulation of toxic minerals e.g. ferrous salts, radioactive minerals etc.

2.  **Man-made**–dumped wastes, domestic, industrial wastes, etc,

•Agricultural chemicals and pesticides (overuse, drift and persistence)

•Radioactive wastes from nuclear stations.

**WATER POLLUTANTS**

Water pollution is a major global problem which can be defined as the contamination of water bodies (e.g. lakes, rivers, oceans, and groundwater). This form of environmental degradation occurs when pollutants are directly or indirectly discharged into water bodies without adequate treatment to remove harmful compounds.

Water pollution affects the entire biosphere – plants and organisms living in these bodies of water. In almost all cases the effect is damaging not only to individual species and population, but also to the natural biological communities.

***Sources***

1. **Natural**–Eutrophication by leaking minerals from soil and leaving silting in lakes and rivers. Leaching of toxic ions e.g. Cu and Iron.

2. **Man-made**–

Domestic sewage

•Industrial effluents

•Heavy metal pollutants from industrial processes

•Eutrophication e.g. nitrates from fertilizers, phosphates from detergents,

•Thermal e.g. cooling water from power stations.

•Radioactive wastes and oil washings from oil tankers, accidents at sea.

**TEAR GASES and PEPPER SPRAY:**

The use of irritating vapors in warfare goes back to the time of the ancient Greeks. The age of modern riot control agents began in 1869. The lacrimating agents commonly used today are readily dispersed and easily decontaminated. Symptoms usually resolve rapidly when the victim is removed from the source of exposure.

**PATHOPHYSIOLOGY**

CS *(orthochlorobenzalmalononitrile)* and CN (*chloroacetophenone)* are highly reactive alkylating agents that attack sulfhydryl groups on crucial enzymes and coenzymes.

CS is more effective than CN in causing lacrimation and other disabling symptoms, but it is safer and causes less systemic and ocular toxicity. Corneal injuries caused by lacrimators usually heal rapidly by regeneration from adjacent epithelial cells. No permanent scarring occurs if structures under the epithelium are not affected. Injury to deeper layers may result in conjunctival edema, necrosis, and sloughing. An inflammatory reaction, ulceration, and revascularization may occur, leading to corneal opacification, iritis, and intraocular hemorrhage. In addition, increased intraocular pressure secondary to exposure may cause acute glaucoma.

**CLINICAL PRESENTATION**

Within seconds of exposure, lacrimators typically cause burning eye pain and irritation, uncontrollable blepharospasm, abundant tearing, edema, and conjunctivitis. Upper respiratory and oropharyngeal symptoms include nasal irritation, rhinorrhea, sore throat, burning sensation of the tongue, and increased salivation. Lacrimators can exacerbate underlying asthma or bronchitis, producing chest tightness, dyspnea, and wheezing.

After inhalation of the lacrimating agents, pneumonia, pulmonary edema, and chemical pneumonitis may occur.

Lacrimators are also primary skin irritants, producing immediate erythema and discomfort. Prolonged exposure can cause first- and second-degree burns.

Other signs and symptoms that can result from exposure to lacrimators include tachycardia and transient hypertension (which may be significant in patients with cardiac disease), photophobia, headache, abdominal pain, nausea, and vomiting. Despite the dramatic symptoms they produce, in general these agents have low systemic toxicity and rarely cause long-term morbidity or mortality unless the victims are exposed to high concentrations in an enclosed space.

**TREATMENT**

The most important immediate intervention is to move the exposed victim into fresh air. In most cases, symptoms from low-dose exposure (blepharospasm, blepharoconjunctivitis, eye pain) resolve quickly once exposure is terminated. All clothing should be removed and sealed in plastic bags. Heavy rubber gloves should be worn when handling contaminated clothing. Respiratory protection (properly fitted gas masks) should be worn if necessary.

For ocular exposure and symptoms, irrigation is done abundantly with water or normal saline.

Patients with respiratory symptoms should receive supplemental humidified oxygen; bronchodilators (inhaled β-adrenergic receptor agonists) can be used to treat wheezing or bronchospasm.

Exposed skin and eyes are decontaminated. The skin can be washed with mild soap and water. Persistent contact dermatitis can be treated with corticosteroid ointment.