MODEL ANSWER

PHYSIOLOGY HONOURS /SEMESTER 6/PAPER CC14

(ENVIRONMENTAL POLLUTANTS AND HUMAN HEALTH)

1. Differentiate between ionizing and non-ionizing radiation with suitable examples (4/2022).

ANSWER:

IONISING RADIATION	NON-IONIZING RADIATION
1. Higher energy electromagnetic wave or particles which can emit electrons from orbit and can ionize atoms or molecules. Example: α, β, γ, cosmic rays, X-rays, high energy UV-rays etc.	1. Lower energy electromagnetic wave that does not have enough energy to dislodge electron from its orbit. They cannot ionize atoms or molecules. Example: low frequency radio wave, visible light, microwave, infra-red etc.
2. They have shorter wavelength but higher frequency	2. They have longer wavelength but lower frequency
3. They have very high penetration power.	3. They have comparatively lower penetration power.
4. They can damage the living cell by ionization including damage to DNA, denaturation of proteins, cancer etc.	4 They are not as dangerous as the ionizing radiation but too much exposure can damage tissue. Heating is the most common effect of non-ionizing radiation.

2. What is non-ionizing radiation? Describe briefly the long-term effects of non-ionizing radiation on human body. (2+4/2021)

ANSWER: Lower energy electromagnetic wave that does not have enough energy to dislodge electron from its orbit is called non-ionizing radiation. They cannot ionize atoms or molecules and have less penetration power. Example: Spectrum of ultraviolet (UV), visible light, infrared (IR), microwave (MW), radio frequency (RF), and extremely low frequency (ELF).

LONG TERM CHANGES:

a) Non-ionizing radiation is basically non-penetrating. There are two primary hazards to non-ionizing radiation; tissue heating (thermal effects) and photochemical reactions to the skin and retina of the eye.

b) **On Eye:** The eye is particularly sensitive to thermal effects because the low volume of blood flow through the eye prevents rapid dissipation of absorbed heat following exposure to the radiation. Serious exposure may increase eye temperature which eventually produces irreversible protein denaturation and the subsequent formation of opacities in the lens of the eye (cataracts). This effect will slowly reduce eyesight and may turn it into partial blindness. However, if the retina is damaged, it can result in permanent blindness. Less penetrating radiation, such as, UV may cause damage to cornea and under acute exposure will cause inflammation of these corneal tissues. Exposure to UV may result in photokeratitis which is commonly referred to as 'snow blindness'. Photokeratitis is an inflammation of the conjunctiva.

c) **On skin:** Exposure to non-ionizing UV light is responsible for developing skin cancer (especially non-melanoma skin cancers), sunburn and premature aging of skin. This effect is largely mediated via the production of unstable ROS. Intracellular ROS can cause damage to both proteins and DNA. The chronic effect of repeated exposure to UV results in a thickening of the upper layers of the skin and the production of the UV-absorbing melanin. Melanin is what gives sun tanned skin its color.

d) **Heating effect:** The heating of tissue is the most important effect of exposure to radiofrequency energy. At prolonged temperatures above about 43 ºC, proteins begin to denature and coagulate, which may lead to damage to the cell membrane and cell death. Temperatures above about 60 ºC produce burns.

3. What do you mean by Half-life of radio-active molecules? (2/2021)

ANSWER: A radioactive substance is a substance which is highly unstable and loses energy by the process of decaying. Time taken for the substance to reduce to half its initial amount is called 'half- life' of the substance. The following equation is an expression for the substance present (left) at time t.

Suppose, initial amount of the substance be N_0., Let the amount of substance left at time 't' be N and λ is the decay constant.

$$N=N_0e^{-\lambda t}$$

4. What protective measures should be taken by human being while working with radioactive elements? (4/2021)

Radiation workers/individuals can control and limit their exposure to penetrating radiation by taking advantage of time, distance, and shielding.

a) **Reduce Time:** Time directly influences the dose received: By reducing the time of exposure to a radiation source, one can minimize the dose received.

b) **Increase distance**: For gamma rays and X-rays, the radiation intensity is inversely proportional to the square of the distance from the source (i.e., the inverse square law). So, by increasing the distance between the subject and the radiation source can reduce the dose received.

c) **Use shielding:** The third exposure control is based on the proper radiation shields, automatic interlock devices, use of lead or lead equivalent aprons, radiation monitoring instruments, concrete walls next to a radiation oncology accelerator etc. Graded-Z shielding is a laminate of several materials with different Z values has been designed to protect against ionizing radiation. Compared to single-material shielding, the same mass of graded-Z shielding has been shown to reduce electron penetration over 60%.

d) **Engineering control: ALARP** stands for "As Low As Reasonably Practicable". **ALARA** stands for "As Low As Reasonably Achievable". Both ALARA and ALARP are important radiation-protection programs in the workplace which can minimizes radiation doses and releases of radioactive materials using all reasonable methods available. Employers should use engineering controls to maintain occupational radiation doses within applicable regulatory dose limits.

e) **Administrative Controls**: This can generally supplement engineering controls. Examples of administrative controls include signage, warning systems, and written operating procedures to prevent, reduce, or eliminate radiation exposure.

5. What are the industrial sources of lead exposure? Name the human system which are affected by lead poisoning. Why should human bone be a tissue of interest in lead toxicity? (2+2+2/2022)

Answer:

a) Industrial sources of lead exposure:

- Ore and metals processing
  Aircraft operating on leaded aviation fuel.
- Waste incinerators, utilities, and lead-acid battery manufacturers.
- Lead-Based Paint.
- Children's Toys and Jewelry.
- Lead Glazed Ceramic Ware, Pottery and Leaded Crystal. Etc.

b) **EFFECTS OF LEAD POISONING:** Lead is the most important toxic heavy element in the environment. There is almost no function in the human body which is not affected by lead toxicity.

- **On Nervous system**: Of all the organs, the nervous system is the mostly affected target in lead toxicity, both in children and adults. One of the mechanisms by which lead interferes with cognition is that it acts as a potent reversible and selective blocker of voltage-dependent calcium -channels in brain at low concentrations. Lead poisoning also causes loss of neuron myelin sheath, reduction in the number of neurons, it interferes with neurotransmission and decreases neuronal

growth. Signs and symptoms of chronic exposure include loss of short-term memory or concentration, depression, loss of coordination, problems with sleep, low IQ, ADHD, slurred speech etc.

- **On Blood:** Long-time exposure to lead has been reported to cause anaemia, along with an increase in blood pressure. Lead inhibits ALAD, porphobilinogen synthase and ferro-chelatase, which prevents heme synthesis. Lead alters the permeability of blood vessels and collagen synthesis. Lead also disrupts the maintenance of the cell membrane. Red blood cells with a damaged membrane become more fragile, resulting in anaemia.
- **On Kidney:** Severe damage to kidneys, both in adults and children, were found to be linked to exposure to heavy lead levels resulting in death. Chronic lead nephropathy occurred due to years of lead exposure manifested by moderate focal atrophy, loss of proximal tubules and interstitial fibrosis.
- **On reproductive system:** In pregnant women, high exposure to lead may cause miscarriage, prematurity, low birth weight, and problems with development during childhood. Chronic lead exposure was found to reduce fertility in males. Activities like motility and the general morphology of sperm are affected with chronic exposure.
- **On bone:** Bone act as a reservoir of lead in body. Chronic lead exposure causes a significant reduction in the bone calcium content. Osteopenia, osteoporosis, and osteomalacia with increased bone fragility have been observed in humans following chronic lead exposure.

c) **Bone is a tissue of interest in lead toxicity because** bone is a reservoir of systemic lead, 90-95% of the total lead burden is contained within bone in non-occupationally exposed adults. The total lead content of bone has been reported to be up to 200 mg in 60–70-year-old men, less in women. Lead forms highly stable complexes with phosphate and can replace calcium in the calcium-phosphate salt that comprises the primary crystalline matrix of bone. As a result, lead deposits are formed in bone during bone growth and remodeling and is lead is released to the blood during the process of bone resorption which, in turn, may contribute to an increase in the concentration of lead in blood.

6. How can fluorine and arsenic affect human health? (3+3/21, 2022)

EFFECT OF FLUORINE: Fluorine is a common element in the earth's crust. The major sources of systemic fluoride exposure are the diet (food and water).

- **Dental fluorosis** (DF) is an undesirable developmental defect of tooth enamel attributed to high fluoride exposure during critical periods of amelogenesis. DF is characterized by opaque white patches in the enamel, which may become stained yellow to dark brown, increased porosity with a loss of enamel translucency and marked pitting and brittleness of teeth.
- **Skeletal fluorosis**: Chronic exposure of fluoride leads to detectable increases in bone mass characterized by sporadic pain and stiffness of joints and osteosclerosis of the pelvis and vertebral column. A second clinical phase associated with slight calcification of ligaments, and increased osteosclerosis of cancellous bones. Third stage is crippling skeletal fluorosis characterized by marked limitation of joint movements, considerable calcification of ligaments, crippling deformities of the spine and major joints, muscle wasting, and neurological defects associated with compression of the spinal cord. In bone, fluoride can lead to conversion of carbonated hydroxyl apatite crystals to carbonated fluorapatite.
- **Hypersensitivity** reactions have been reported following exposure to fluoridated water, toothpastes, and fluoride supplements. Symptoms include skin rash, inflammation of oral mucosa, gastrointestinal irritation, and headache, which subsided after discontinued use of these products.
- **Hematopoietic cells/hematopoiesis**: Effects vary depending upon fluoride dose, duration of exposure, and species, and include anemia and leukopenia. Fluoride exposure shifts the hematopoietic differentiation in bone marrow along the monocyte/macrophage lineage.
- **Acute toxicity:** Acute exposure at high concentration of fluoride leads to binding of fluorine with serum calcium and magnesium, vomiting, nausea, chronic convulsion, necrosis of the mucosa of the digestive tract and heart failure.

EFFECT OF ARSENIC ON HUMAN HEALTH: The World Health Organization (WHO) lists arsenic as one of the 10 chemicals of major public health concern. Long-term consumption of drinking water contaminated with naturally

occurring soluble inorganic arsenic leads to chronic arsenic poisoning, also called **arsenicosis.** West Bengal (WB) is an arsenic endemic state in India, with at least 9 out of 18 districts exposed to groundwater contaminated with arsenic (of geological origin) above the WHO's maximum permissible limit of 10 mcg/L. (0.01 ppm).
Permissible limit of arsenic in India in absence of an alternative source - 0.05 mg/l (50 µg/l).

- **Chronic arsenicosis** is a multisystem disorder. Apart from generalized weakness, appetite and weight loss, and anemia, patients had symptoms relating to involvement of the lungs, gastrointestinal system, liver, spleen, genitourinary system, hemopoietic system, eyes, nervous system, and cardiovascular system.
- **On skin:** Classic skin lesions such as **"rain drop pigmentation"** and **keratosis** are more commonly described in arsenicosis. Major dermatological signs are diffuse or spotted melanosis, leucomelanosis, and keratosis.
- **On Lungs** Symptoms of chronic lung disease, chronic cough, were present in people with chronic arsenic toxicity. Lung function tests carried out on patients showed features of restrictive lung disease
- **Gastrointestinal system:** Dyspepsia, gastroenteritis, nausea, diarrhea, anorexia, and abdominal pain was observed in patients with chronic arsenic toxicity studied in West Bengal. Patients developed features of portal hypertension with signs of liver fibrosis.
- **On cardiovascular system**: Blackfoot disease (BFD), a form of peripheral vascular disease, has been reported to be one of the important complications of chronic arsenic toxicity. Increased prevalence of hypertension among residents in the endemic area has been found. Mortality rate from ischemic heart disease was found to be increased with endemic arsenicosis.
- **On nervous system**: peripheral neuropathy and Peripheral neuritis characterized by paresthesia (tingling, numbness, limb weakness, and others) was present in patients of chronic arsenicosis. Irritability, lack of concentration, depression, sleep disorders, headache, and vertigo were reported in arsenicosis people showing features of neuropathy in West Bengal.
- **On reproductive system** High concentrations of arsenic (≥200 mg/L) during pregnancy were found to be associated with a six-fold increased risk for stillbirth.

7. Mention the importance of chelation therapy in acute arsenic toxicity. 4/21

a) **Chelation therapy** is a treatment for heavy metal poisoning. Heavy metals like lead, mercury, and arsenic serve no function in the human body. People with very high levels of these heavy metals are treated with drugs called "chelators". These chemicals bind to the metals in the blood stream and forms 'metal-chelator compound' which is then eliminated in the urine. A good chelating agent can compete with endogenous ligands and must have a relatively higher affinity and selectivity for toxic metals.

b) The employment of chelation mechanisms in the treatment of arsenical intoxication began during World War II, with the synthesis of **British Anti-Lewisite (BAL)** or **2,3-dimercapto-propanol**. BAL is formulated in peanut oil because of its lipophilic nature and administered intramuscularly at an initial dose of 3–5 mg/kg of body weight every 4 h. This chelating agent has ability to compete effectively with the thiol groups of proteins for binding to the As ion and forms metal-chelator complex which is then excreted in the urine. This chelator is highly effective but produces severe side effects.

c) Other Thiol-based metal chelators such as **DMSA, DMPS, and PCA** are mainly administered in acute As poisoning. DMPS and DMSA, due to their high water-solubility, low toxicity, and high therapeutic indexes, are superior to BAL in the treatment of acute As poisoning. DMPS should be selected as the first choice because of its greater antidotal potency. In acute As poisoning, DMPS is used as a 5% solution for intravenous and intramuscular injection, as well as tablets and capsules for oral administration.

d) Chelation therapy for acute As poisoning is recommended because the benefits exceed the risk of side effects. The application of **natural chemical chelators** is one of the traditional methods of removing toxic metals from the body. Natural antioxidants like flavonoids, lipoic acid, carotenoids, vitamin C and E, taurine, gallic acid, curcumin, etc. have shown beneficial effects in counteracting metal toxicity by increasing methylation capacity and free ROS scavenging. Flavonoids present in green tea leaves exerts a protective action against As-caused damage through their antioxidant and chelating ability.

8. Discuss briefly the effects of Lead and Aluminum poisoning on human health. (5/2021)

EFFECT OF ALUMINUM TOXICITY ON HUMAN HEALTH:

Aluminium is the most abundant metal and the third most abundant element in the Earth's crust. Diet is a significant contributor to the body burden of aluminum. (1 - 20 mg per day). Aluminium is an important component of many aerosol formulations of cosmetics, and particularly anti-perspirants, and these, especially through regular use, will contribute significantly to exposure to aluminum through breathing. it is also used in foil for wrapping of food, cooking utensils etc.

a) **Acute effects**: the initial contact can irritate skin and eyes and cause 'metal-fume-fever'. This is a flu like illness with symptoms of metallic taste in mouth, fever, chill, chest tightness and cough. It may last for day or two.
b) **Neurotoxicity:** Chronic exposure of Aluminum can cause specific encephalopathy and dementia. There is a significant association between aluminum and Alzheimer's disease. Aluminum can modify hippocampal calcium signal pathways that are crucial to neuronal plasticity and, hence, to memory. Cholinergic neurons are also particularly susceptible to aluminum neurotoxicity, which affect synthesis of the neurotransmitter acetylcholine.
c) People with kidney disease store a lot of aluminum in their bodies and sometimes develop bone or brain diseases which may be caused by the excess aluminum.
d) Aluminum in large amounts has been shown to be harmful to unborn and developing animals because it can cause delays in skeletal and neurological development.

9. What are organophosphate pesticides? Give one example. (2/2021, 2022).

ANSWER: Organophosphates (OP) are chemical substances produced by the process of esterification between phosphoric acid and alcohol. Organophosphates can undergo hydrolysis with the liberation of alcohol from the ester bond. These chemicals are the main components of herbicides, pesticides, and insecticides. OPs are also the main components of nerve gas. The most used organophosphate pesticides are Parathion, malathion, phosmet etc.

10. What are organocarbamate pesticides? Give one example. (2/2021, 2022)

ANSWER: Carbamates are a class of insecticides structurally and mechanistically similar to organophosphate (OP) insecticides. Carbamates are N-methyl carbamates derived from a carbamic acid and cause carbamylation of acetylcholinesterase at neuronal synapses and neuromuscular junctions. Examples include aldicarb, carbofuran, fenobucarb, trimethacarb etc.

11. Discuss briefly the effects of organophosphate and organocarbamate pesticides on human. (6/2022)

Answer:

a) **Acute effects or organophosphates**: Organophosphates can irreversibly bind to acetylcholine esterase enzyme and prevent their breakdown which leads to overstimulation of both the muscarinic and nicotinic Ach-receptors. The main effect of acute exposure to organophosphates is poisoning. Symptoms of poisoning includes muscular fasciculations and weakness, bronchospasm, tachycardia, respiratory arrest, and pulmonary edema. A life-threatening severity of poisoning is signified by loss of consciousness, seizures, and respiratory depression. The primary cause of death is respiratory failure.
b) **Acute effects or organocarbamate:** Carbamate also leads to reversible inhibition of acetylcholine-esterase enzyme present at parasympathetic and sympathetic ganglia, parasympathetic muscarinic terminal junctions, sympathetic fibers located in sweat glands, and nicotinic receptors at the skeletal neuromuscular junction. Persistently elevated acetylcholine levels lead to increased neurotransmitter signaling and produce confusion, delirium, hallucinations, tremor, muscle weakness and seizures.
c) It is important to remember that the adrenergic symptoms of tachycardia, hypertension, and mydriasis also may be present due to acetylcholine-dependent activation of nicotinic receptors in sympathetic ganglia. That is why mixed autonomic presentations are common.

d) **Delayed/chronic effects**: chronic effects of organophosphate are weakness, in proximal, cranial, and respiratory muscles, A few organophosphates may cause an axonal neuropathy that begins 1 to 3 weeks after exposure. Long-term, persistent effect of organophosphate poisoning may include cognitive deficits and parkinsonism. Carbamates do not lead to chronic toxicity because carbamate bonds are hydrolyzed from acetylcholine spontaneously and rarely cause symptoms after 24 to 48 hours.

12. Mention two sources of organocarbamate. (2/2022)

- The carbamates are mainly used in agriculture, as insecticides, fungicides, herbicides, nematocides, or sprout inhibitors. In addition, they are used as biocides for industrial or other applications and in household products. A potential use is in public health is vector control.
- Other potential causes of organophosphate or carbamate toxicity include ingestion of contaminated fruit, flour, or cooking oil, and wearing contaminated clothing.

13. Discuss briefly the effects of chlorinated hydrocarbons on human (5/2022)

Chlorinated hydrocarbon (CHC) is a generic term given to compounds containing carbon, chlorine, and hydrogen. Examples include organochlorine pesticides such as **lindane and DDT, industrial chemicals such as polychlorinated biphenyls (PCB), and chlorine waste products such as dioxins and furans**. Although the United States and most developed countries have banned the use of DDT, recently it has been used globally as an insecticide for control of vectors for malaria.

a) The direct **DDT** exposure produces toxic effects in humans include developmental abnormalities, reproductive disease, neurological disease, and cancer. Following exposure to high doses, human symptoms can include vomiting, tremors or shakiness, and seizures. DDT is considered a possible human carcinogen. The exposure DDT metabolite (DDE) also promotes health effects such as childhood diabetes and obesity. DDT has transgenerational effects in progeny and generations never directly exposed to DDT. Exposure to DDT/DDE has been linked with lymphoma, leukemia, and pancreatic cancer.

b) The **polychlorinated biphenyls (PCBs)** are synthetic organochlorine chemicals that were useful industrial products in the past, but their production was ended because they persist in both the environment and living organisms. PCBs are found primarily in lake and river bottom sediments and fatty tissues in fish. Eating contaminated fish remains the major route of exposure to PCBs. Toxic effects include hepatic damage, Chloracne and related dermal lesions, and respiratory problems. They can covalently bind to cellular macromolecules (e.g., protein, DNA, RNA) and induce DNA strand breaks and DNA repair, which can contribute to the toxic response of PCBs. In a person with PCB-induced chloracne, the acne-like lesions arise as a result of inflammatory responses to irritants in the sebaceous glands.

c) **Dioxins** are called **persistent organic pollutants (POPs)**, meaning they take a long time to break down once they are in the environment**.** The biggest source of dioxin is the pulp and paper and petrochemical industries, and incinerators. Dioxins are absorbed and stored in fat tissue and, therefore, accumulate in the food chain. More than 90 percent of human exposure is through food. Dioxins are highly toxic and can cause cancer, reproductive and developmental problems, damage to the immune system, and can interfere with hormones.

14. What do you understand by Snow-blindness? (2/2021)

Answer: Snow blindness is a form of photokeratitis that is caused by UV rays reflected off ice and snow. Eye damage from UV rays is particularly common in the North and South Pole areas or in high mountains where the air is thinner and provides less protection from UV rays. Expose to UV light may cause damage to cornea and will cause inflammation of these tissues. It can cause extreme pain, light sensitivity, and blurry vision.