FOOD ADDITIVE AND ADULTERANTS /SECB2

1. ROLE OF SACCHARINE ON GUT MOCROBIOTA.

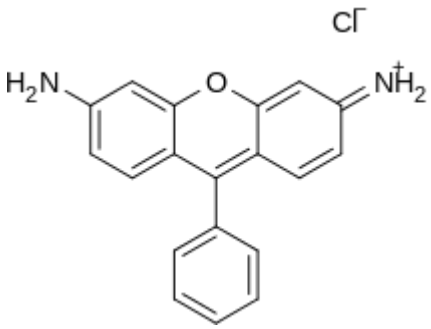
1. Saccharin is an artificial and non-nutritive sweetener used to produce various foods like jams, jellies, baked goods, chewing gum, low calorie candies, toothpaste, diet drinks, tinned fruit etc. It is an artificial sweetener with effectively no food energy. It is the oldest artificial sweetener, discovered in 1879.

2. The human body is inhabited by trillions of symbiotic microorganisms, mainly in the large intestine, and they are collectively called **gut -microbiota**. The gut microbiota are composed of several species of microorganisms, including more importantly bacteria, archaea, yeasts, and viruses, each individual being provided with a unique gut microbiota profile. Gut microbiota in humans is highly beneficial as it promotes degradation of undigested proteins and carbohydrates, monosaccharide fermentation, hydrogen disposal, bile-acid transformation, vitamin synthesis and stimulate immune system.

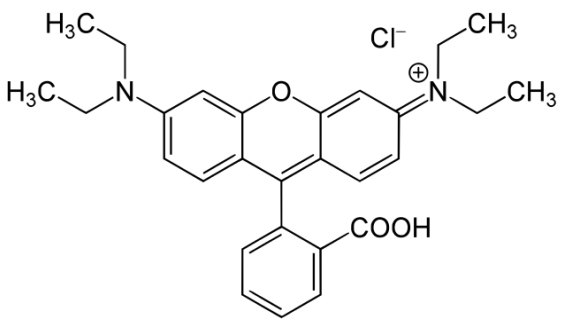
3. Long term intake of saccharine can damage the microbiota.
   - Studies have found very distinct changes in the composition and function of gut microbes and the molecules they secrete into peripheral blood. **Saccharin can cause beneficial bacteria** in the intestines such as E. coli (Escherichia coli) and E. faecalis (Enterococcus faecalis) **to become pathogenic.** These pathogenic changes include greater formation of biofilms and increased adhesion and invasion of bacteria into human gut cells.
   - Bacteria like E. faecalis can cross the gut wall and enter the blood stream, which can lead to a life-threatening condition like inflammation and sepsis.
   - Biofilms (clusters of bacteria) are produced in greater amount in gut that secrete toxins and become less sensitive to antimicrobial resistance treatment, potentially increasing the risk of other disease.
   - The ingestion of saccharin by animals and humans showed alterations in metabolic pathways linked to glucose tolerance and dysbiosis in humans. Excess saccharine has shown to cause weight gain.

2. IS RHODAMINE A XANTHENE DYE? EXPLAIN.

Dyes that contain a xanthene core are called xanthene dyes. Examples include fluorescein, eosins, and rhodamines. Rhodamine b is a synthetically prepared xanthene cationic dye and widely used for paper printing and as a colorant in textile and food stuff.



Xanthene dye



Rhodamine b

3. NAME ONE SIMPLE METHOD OF CHICORY DIFFERENTIATION FROM COFFEE POWDER

Tumbler test:

Take a transparent glass. Fill it with water. Gently sprinkle the coffee powder sample.

- If the powder floats for some time before sinking, it is coffee.
- If the powder sinks quickly, it is chicory or some other seed.
- If it readily diffuses to form brownish or yellowish colour, it contains Chicory or Caramel.

4. STATE PATHOLOGICAL SIDE EFFECTS OF UREA.

Urea is a white crystalline powder with a cooling saline taste. Urea occurs naturally in mammals and is an excretory end-product of amino acid metabolism. Urea is used as fertilizer, animal feed, as flavoring agent in food, in fermentation etc. Chewing gum may contain up to 3% urea. Urea is used in adulterated milk to increase the density. Urea is a common ingredient of a many cosmetic formulations.

Pathological side effects of urea:

- Exposure to exogenous urea can occur via inhalation, dermal contact, and oral intake through consumption of food and drinking water. The available toxicity information on this chemical suggests that the **liver and kidney** could be possible target organs. Toxicity studies in mice has revealed degenerative and necrotic changes in hepatocytes. Fatty changes in the perirenal tissue of kidney, mild necrosis, glomerulitis, leukocytic infiltration and a prolonged clearance time for glucose was observed in experimental animal after as little as 7 days of dosing.
- Sudden accidental inhalation of urea in workers has shown to develop nausea and persistent vomiting 3–5 hours after exposure, followed by convulsions accompanied with urination. However, none of the workers died, and all completely recovered within a few days.
- Prolonged bleeding was noted in patients with exposure to high doses of urea. A drastic reduction of the blood platelet adhesiveness was observed in them.
- No definite proof of carcinogenicity is available. However, increased frequency of interstitial adenomas in testes of male rats and an increase in malignant lymphomas in female rats have been observed in a dose -dependent fashion. Reports of low birth weights have been found in exposed cases.

5. MENTION ANY TWO HAZARDOUS EFFECTS OF ALUMINUM FOIL

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 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.

6. 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.

- **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 remodelling 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.

7. 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 µg/L or (0.01 ppm).
Permissible limit of arsenic in India in absence of an alternative source is 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.

8.. WHAT ARE AZO DYES?

Azo dyes are the largest group of synthetic dye which has azo (N=N) functional group. Azo dyes are the largest group of artificial food dyes, including 70% of the organic dyes generated in the world.

There are currently 11 azo dyes that are typically used in foods: The commonest azo dyes in the food industry have been the yellow dyes (sunset yellow and tartrazine) and red dyes (azorubine, ponceau, amaranth, and Allura red).