COURSE TITLE: FOOD TOXICOLOGY

COURSE CODE: FST 516

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LECTURE 1

INTRODUCTION

MEANING OF TOXIC: A toxic substance or toxicant is described as one that could cause health hazards in human when ingested at a level beyond the standard safe-level.

The scientific study of the nature, effects and control of toxic substances on living systems is referred to as **TOXICOLOGY**.

A physician named Paracelsus (1493-1541), is considered to be the founder of toxicology as an objective science. He was the first that attributed adverse effects of certain substances to the substance itself and not to its association with an evil or angered spirit or god.

Basic Concept / Principle of Toxicity

Paracelsus stated the basic concept of toxicity as:

"All substances are poisons; there is none that is not a poison. The right dose differentiates the Poison from a remedy".

For example;

- water toxicity and its lethal effect and
- conversely, **botulinum toxin** produced by *Clostridium botulinum* bacterium and its therapeutic usefulness.

Branches of Toxicology

There are multidisciplinary application of toxicology; these include:

- 1. Clinical toxicology- Managmt of poisoning in clinical envrmt.
- 2. Forensic toxicology- Law enforcement detectn harmful chem.
- 3. Occupational toxicology- Work place toxic subtances and safety
- 4. Environmental toxicology- Natural/man made envmtl chemicals
- 5. Regulatory toxicology- Epidemiological/experimental data
- 6. Ecotoxicology- Distribtn & toxic effect within a defined ecostm
- 7. Food toxicology- Toxic substance occurred in foods

LECTURE 2

Food Toxicology

Food toxicology is the major area of interest in this course and it is concern with the analysis and toxic effects of bioactive substances as they occurred in foods.

By definition, **Food Toxicology** is the study of the nature and effects of toxic bioactive substances ingested through diet or food, their manifestation in humans.

Toxic bioactive substances may be natural endogenous products or may be introduced from contaminating organisms, or from food production, processing, and preparation.

Sources of Health Hazards Associated with Food

The US Foods and Drug Administration (FDA) has ranked the relative importance of health hazards associated with food in the following descending order:

- 1) Microbiological contamination
- 2) Inappropriate eating habits
- 3) Environmental contamination
- 4) Natural toxic constituents
- 5) Pesticides residues
- 6) Food additives / food processing

However, the public opinion polls shows that people rate **food additives** as one of their major concerns about the safety of the food supply.

Natural Toxic Constituents in Foods

The chemical toxicants in foods and the food sources are majorly of plant origin:

- Glycoside cassava, bitter almond, Sorghum, pulses
- Glycoprotein beans
- Goitrogenic glycosides cabbage, garden eggs, mustard seeds, Alligator pepper
- Saponins soyabeans, bitter leaf
- Gossypol cotton seed
- Lathyrogens *Lathyris sativas* (grass pea or Indian pea)
- Allergens grains, milk, fish, egg, etc.
- Vicine Fava beans
- Tannins malted grains like *Sorghum*
- Phytates oilseeds
- Protease inhibitor (Polyphenols) beans like cowpea
- Excess of vitamins- A, D, C, E, Niacin, Thianine, Folacin) excessive vit. supplement
- Antivitamins e.g. Ascorbates soya bean, kidney bean

LECTURE 3

Table 1a: Characteristics and Effect of Plant Food Toxicants

Plant Food toxicants	Characteristics	Effects
Glycoside (Cassava)	Staple food of many African	-Linamarin and Lotaustralin which yield HCN- a potent
	countries	respiratory inhibitor
	Some species have bitter taste	-Thioglycoside and Benzothio-glycoside which
		produce Benzylcyanate that are poisonous compound
		capable of anti-thyroid action
Glycoprotein (e.g.	Highly consume in the world	-Lecithins (Heamaglutinins) which agglutinate the red
Beans)	esp. India, Latin America and	blood cells, reduce growth, causes diarrhea, interfere
	Africa	with Biotin (B-vitamin)
		-Some prevent the absorption of food materials by the
		GIT enzymes
Goitrogenic	Cruciferous vegetables;	Giotrogens- inhibit synthesis of thyroid hormones by
glycosides (cabbage,	responsible for the pungent	thyroid gland resulted into goitre
garden egg, etc)	nature of some seeds like	
	mustard & Alligator pepper	
Saponins (soyabeans,	Bitter taste, profuse foaming,	Uronic acid which causes growth depression in poultry,
bitter leaf)	haemolysis RBC	goats, and swine

Table 1b: Characteristics and Effect of Food Toxicants

Gossypol	Toxic at levels of 0.4-0.7%	Reduce the nutritive value of cotton seed
(polyphenolic		flour, reduced growth, cardiac lesions, liver
cmpd)- cotton		damage, heamorage, & oedema in
seed		experimental animals
Vicine – Fava	Common in the	Favism is inborn deficiency of glucose-6-
beans	Mediterranean basin esp.	phosphate dehydrogenate leading to severe
	West Africa	heamolysis, vomiting, dizziness, postration,
	Sensitive in broad beans	haemolytic anaemia
Tannins	Astringent in nature	Decrease protein digestivity & a.a. absorption
(phenolic		Produce dhurrin which is carcinogenic in
compound)		experimental animals
Phytates (oil	Ex. chelates minerals like Ca,	Reduce mineral bioactivities i.e. anti-nutritive
seeds)	Mg, Zn, Fe	when in excess
Protease	Occurred in raw cowpea	Trypsin inhibitor (Ovomucin in egg)- Inhibits the
inhibitor	Nutritional value improves by	utilisation of protein by impairment of its
(Polyphenols)	heat treatment	digestion and absorption

Table 1c: Characteristics and Effect of Food Toxicants

Excess of vitamins- A,	Fat soluble vitamins occurred	Toxicity through the excessive vitamin
D, C, E, Niacin,	in subcutaneous part of the	supplement intake
Thianine, Folacin)	body	
Vit. A	RDA max. of 700-900 μ/day	Erythema (fast breathing), cerebrospinal fluid
	of vit. A	pressure increase, hepatic (liver) neurosis, &
		death
Vit. D	RDA max. of 15-40 μ/day of	Excess vit. D causes High serum Ca level and
	vit. D	accentuate (notice) the atherosclerotic process
Vit. C	RDA max. of 500 mg of vit C	Ex. vit C can cause diarrhea
Niacin		Ex. Niacin induces flushy, itching, skin rash,
		heart burn, vomiting, diarrhea, gastric and
		duodenal ulcer, hypo and hyperglycemia
Thiamine	RDA max. of 1 g/day of	Ex. Thiamine- hypersensitivity reactions, rapid
	Thiamine	pulse vaso-dialation, oedema, abnormal heart
		beat
Folacin		Ex. Folacin causes convulsion and neurological
		damage in patients 13

Table 1d: Characteristics and Effect of Food Toxicants

Antivitamins e.g	Normally added to food	Antivitamins chelates vitamins and
Ascorbates (in	to enzymatically destroy	reduce their bioavailability
soya bean, kidney	vits. In order to stop the	
bean)	physiological action of	
	vits.	
Lathyrogens –	Occurred in Lathyris	Interfere with the role of glutamic acid as
(grass pea or	sativas seed as β-amino-	an excitatory neurotransmitter in the
Indian pea)	propionitrite and β-N-	brain leading to Latyhrism- Neurological
	oxaly-L-α, β-diamino	disease characterised by muscular
	propionic acid	weakness, irreversible leg paralysis,
	Restricted to India	death
Allergens (grains,	Normal component of	Altered reactivity in individual
milk, egg, fish, etc)	foods	

LECTURE 4

TOXINS OF ANIMAL FOOD STUFFS

Poisonous animal are those whose tissues are toxic and cause adverse responses when eaten. Majority of such animals are restricted almost entirely, to marine forms.

Their presence among the edible species of marine animals create a problem as man turns to the oceans for additional source of animal protein.

The toxins causing these sea foods to be poisonous vary considerably in their **chemistry and toxicology**. Two main types of poisoning by marine animals are recognised:

- 1. Fish poisoning that resulted from eating fish containing poisonous tissues; this is known as **icthyotoxism** and
- 2. Shellfish poisoning which resulted from ingestion of shellfish that has concentrated toxins from plankton constituting their food supply is known as **paralytic shellfish poisoning.**

Icthyotoxism

About 500 species of marine fishes are known to be poisonous when eaten, and many of these are among the edible varieties. Poisoning can occurred following ingestion of a wide variety of commonly used food fishes, such as, grouper, sea bases, and snappers.

Paralytic shellfish poisoning

This syndrome is cause by eating clams or mussels that have ingested dinofllagellates and effectively concentrates them. Shellfish become toxic when local conditions favour the growth of the flagellates beyond their normal numbers; such circumstances are referred to as 'Red tides'.

Table 2: Characteristics and Effect of Animal Food Toxicants

Animal Food	Characteristics	Effects
toxicants		
Icthyotoxism	Marine origin e. g. groupers, sea basses,	-Poison the fish tissue
	snappers	-Indirectly cause food
	Toxicity is associated with food chain	poisoning in man
	relationship	
	Blue-green algae → herbivorous fish →	
	indirectly to man	
Paralytic	Marine origin e. g. clams, mussels (big)	-death through respiratory
shellfish	Toxic agent occurred in dinoflagillates with	failure
poisoning	the empirical formula: C ₁₀ H ₁₇ O ₄ .2HCl	
	-Purified toxin has an LD ₅₀ of 9µg/kg body	
	weight at the centre of the brain	
	It is stable to heat and cannot be destroyed	
	by cooking	1

METHODS OF FOOD DETOXIFICATION

- **Soaking** rafinnose oligosaccharides, tannin, heamagglutinin, trypsin and chymotrypsin inhibitors
- Cooking- trypsin and chymotrypsin inhibitors, HCN, saponin, phytates, allergens
- **Germination-** rafinnose oligosaccharides, phytates, heamagglutinin, trypsin inhibitors
- Fermentation- rafinnose oligosaccharides, phytates
- Extraction- heamagglutinin, tannin
- Filtration- phytates
- Enzymatic method- rafinnose oligosaccharides, HCN

LECTURE 5

Acute Toxicity Test and Evaluation of LD₅₀

Acute Toxicity Test- Single test conducted on essentially all chemicals of any biological interest.

- Chemical is administered to the animal on one or at most, two occasions.
- Determines the symptomatology consequence and LD₅₀ of the compound/chemical.
- No autopsy and no control group is used except when a foreign chemical is used as vehicle for the injection of the test chemical.
- Sequence of effects following the administration of the compound is observed.

Mode of Administration of the Chemical

Oral- Less toxic due to poor absorption from the GIT

Intramuscular- Less toxic as a result of detoxification effect of the liver

Intravenous- More toxic as a result of direct introduction to the blood stream

Acute Lethality of a toxin/compound is usually determined on the basis of death occurring in 24 h; a seven-day prolong observation gives better inference

Symptomatology Observations

Physical

Bizarre (strange/unusual) positions, Alterations in cardiac rate

rhythms Paralysis

Bizarre tail position Exploratory behaviour

Change in papillary size Sensitivity to pain

Inactivity Skin lesions

Convulsions, spontaneous Corneal opacities

Dyspnea Placing reflexes

Sedation, Righting reflexes

Nystagmus Grasping reflexes

Cyanosis Pineal reflexes

Abnormal excreta Nasal discharge

Salivation Altered muscle tone

Piloeroction Muscle tremors

Phonation Coma Coma Coma

Catatonia Death

Aggressiveness towards the experimental

Potency / LD 50 of Toxin

When suitably extensive observations of the symptomatology of animals used for acute toxicity tests are made, it is important to determine the dose levels;

minimal symptomatic dose- toxic dose
maximal tolerated dose- dose at which the animal recovered
completely from the effect of the chemical, and
no symptom at-all dose- No observable symptom or no toxic effect

Therefore, the dose level of a cmpd /chem/toxin that produce toxic and lethal effect on 50% of the experimented animal population is referred to as $\mathbf{LD_{50}}$

The higher the magnitude the less toxic is the substance.

Potency of Common Toxins

Agent	LD ₅₀ (mg/kg)	Toxicity
Ethyl alcohol	9,000	
Sodium chloride	4,000	
BHA/BHT(antioxidants	2,000	Slight
Morphine sulphate	900	Slight
Caffeine	200	Moderate
Nicotine	1	High
Cucane	0.5	High
Shellfish toxin	0.01	High
Dioxin	0.01	High
Botulinum toxin	0.00001	Extreme

Substances with LD_{50} values greater than 2 mg/kg body wt. generally are considered to be of slight toxicity. But, exposure to substances in the extreme category with LD50 < 1 mg/kg requires only a few drops or less to be lethal and may be a considerable hazard.

Toxicology Examination indices

The indices commonly employed in animal toxicology examination include;

- **Blood chemistry studies-** Sodium, potassium, blood urea nitrogen, and glucose levels in the blood
- Urine analysis (Urinalysis)- pH, protein, glucose, ketoses, crystals, blood cells and bacteria cells
- **Haematology-** Hematocrit- %total red blood cells, total and differential white blood cell counts, and thrombocytes counts
- Organ function- Liver and kidney functions
- Hepatological- Liver diseases include;
 Hepatitis A: Caused by ingestion of infected food; least serious
 Hepatitis B and C: Caused by infected blood and it is very serious

FIRST TEST ON FOOD TOXICOLOGY

DATE: 28-06-2021

Qn. 1. Aside food toxicology, mention four other areas of toxicology studies. 2 marks

Qn. 2. List five chemical toxicants that occurred naturally in foods with their corresponding food source(s). 5 marks

Qn. 3. Explain the toxicity strength of a toxicant in relation to its LD_{50} value. 3 marks

Qn. 4. Explain "Favism" and state its effects. 4 marks

Qn. 5. Enumerate the methods of toxin removal from food materials and briefly explain one of the methods. 6 marks

SECOND TEST ON FOOD TOXICOLOGY

DATE: 26-07-2021

- 1. Single test conducted on essentially all chemicals of any biological interest in toxicological study is referred to as............... Enumerate the steps involved in carrying out the mentioned test. **5 marks**
- 2. State the characteristic of oral, intramuscular, and intravenous administration of a chemical/toxin. **5 marks**
- 3. Mention ten symptomatology observations in text animals during toxicity test of a chemical. **5 marks**
- 4. Mention all and explain two of the indices commonly employed in animal toxicology examination. **5 marks**