FOOD MICROBIOLOGY AND TOXICOLOGY

M.Sc., FOODS AND NUTRITIONAL SCIENCES, II YEAR, Paper- III

Specialization: Food Science and Quality Control

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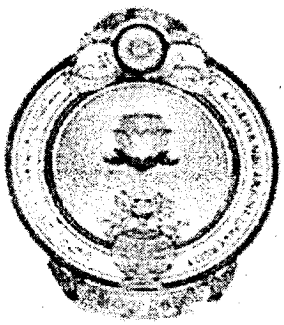

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FOREWORD

Since its establishment in 1976, Acharya Nagarjuna University has been forging ahead in the path of progress and dynamism, offering a variety of courses and research contributions. I am extremely happy that by gaining a B++ (80-85) grade from the NAAC in the year 2003, the University has achieved recognition as one of the front rank universities in the country. At present Acharya Nagarjuna University is offering educational opportunities at the UG, PG levels apart from research degrees to students from about 300 affiliated colleges spread over the three districts of Guntur, Krishna and Prakasam.

The University has also started the Centre for Distance Education with the aim to bring higher education within reach of all. The Centre will be a great help to those who cannot join in colleges, those who cannot afford the exorbitant fees as regular students, and even housewives desirous of pursuing higher studies. With the goal of bringing education to the doorstep of all such people, Acharya Nagarjuna University has started offering B.A and B.Com courses at the Degree level and M.A., M.Com., M.Sc, M.B.A. and LL.M. courses at the PG level from the academic year 2003-2004 onwards.

To facilitate easier understanding by students studying through the distance mode, these self-instruction materials have been prepared by eminent and experienced teachers. The lessons have been drafted with great care and expertise within the stipulated time by these teachers. Constructive ideas and scholarly suggestions are welcome from students and teachers involved respectively. Such ideas will be incorporated for the greater efficacy of this distance mode of education. For clarification of doubts and feedback, weekly classes and contact classes will be arranged at the UG and PG levels respectively.

It is my aim that students getting higher education through the Centre for Distance Education should improve their qualification, have better employment opportunities and in turn facilitate the country's progress. It is my fond desire that in the years to come, the Centre for Distance Education will grow from strength to strength in the form of new courses and by catering to larger number of people. My congratulations to all the Directors, Academic coordinators, Editors and Lesson - writers of the Centre who have helped in these endeavours.

Prof. K. Viyyanna Rao
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Acharya Nagarjuna University

M.Sc. (FOODS & NUTRITIONAL SCIENCES), SECOND YEAR
SPECIALIZATION - II : FOOD SCIENCE AND QUALITY CONTROL
Paper - 3: FOOD MICROBIOLOGY AND TOXICOLOGY
SYLLABUS

UNIT - I:

- Factors associated with food spoilage: Micro-organisms, contamination during processing and handling.
- Microorganisms important in food preservation: moulds yeast, bacteria, characteristics and identification.
- Principles underlying spoilage, chemical changes caused by microorganisms.
- Control of Microorganisms: Growth curves, physiochemical factors influencing destruction of microorganisms and their death time.
- Food enzymes: Factors relating in destruction of naturally present food enzymes.

UNIT - II:

- Mould and micotoxin contamination of foods: Classification of micotoxins, methods of detection and prevention.

UNIT - III:

- Toxicology: Definition, classification of food toxicants-factors affecting toxicity of foods and diseases out breaks.

UNIT - IV:

- Food toxin and implications on human health: Neuro toxicity, hepatic toxicity, Nephron toxicity, Haemo toxicity, skeletal toxicity, Reproductive toxicity, Allergenicity, Mutagenicity. Teratogenic effects, carcinogenic and miscellaneous manifestations.
- Incidence of mushroom poisoning and types of mush room poisoning
- Toxicology of marine foods: Mollusks, fish and marine algae.

UNIT - V:

- Derived food toxicants: Toxins formed from fats by oxidation and rancidity, toxins formed from proteins and amino acids by alkali treatment and produced by reacting with contaminants.

REFERENCES :

1. Food Toxicology part A. Principles and concepts by jose M.Canon, Marcel Dekker, Inc.New York, 1988.
2. Food Toxicology part B principles and concepts by jose M.Concon marcel Dekkher Inc, New York, 1988.
3. Food Additive Toxicology by Joseph A. Maga Anthony T.TU Marcel Dekker, Inc, New York, 1994.
4. Journal of Medical Microbiology.
5. Current Science.

QUESTION PAPER PATTERN

Each Paper carries 100 marks, 20 marks Internal assessment through assignments and 80 marks for year end examination. The duration of examination for theory papers will be three hours.

Pattern :

The question paper is divided into five Units. Unit I, II, III, IV and V

Unit - I : (1*16 = 16 marks)

The unit consists of 2 questions, out of which the candidate has to write 1 question, which carries 16 marks

Unit - II : (1*16 = 16 marks)

The unit consists of 2 questions, out of which the candidate has to write 1 question, which carries 16 marks

Unit - III : (1*16 = 16 marks)

The unit consists of 2 questions, out of which the candidate has to write 1 question, which carries 16 marks

Unit - IV : (1*16 = 16 marks)

The unit consists of 2 questions, out of which the candidate has to write 1 question, which carries 16 marks

Unit - V : (1*16 = 16 marks)

The unit consists of 2 questions, out of which the candidate has to write 1 question, which carries 16 marks

Food Microbiology and Toxicology

CONTENTS

Lesson – 1

FOOD SPOILAGE

1.0 Objective

After reading this chapter, you should be able to:

- To know different types of Micro-organism
- To understand contamination in food handling

Structure

1.1. INTRODUCTION

Food is considered contaminated when unwanted microorganisms are present. Most of the time the contamination is natural, but sometimes it is artificial. Natural contamination occurs when microorganisms attach themselves to foods while the foods are in their growing stages. For instance, fruits are often contaminated with yeasts because yeasts ferment the carbohydrates in fruits. Artificial contamination occurs when food is handled or processed, such as when fecal bacteria enter food through improper handling procedures.

Food spoilage refers to undesirable changes occurring in food due to the influence of air, heat, light, moisture, which foster the growth of microorganisms. Foods take different period of time to lose their natural form though spoilage. In context to food preservation foods are classified as perishable, (meat, fish, milk fruits and some vegetable), semi perishable (eggs, onions, potatoes, carrot, beans) and non-perishable (cereals, pulse nuts).

Moulds vary greatly in heat resistance but again the greatest risk with spoilage of acid foods is if they gain entry after heating is completed. Most vegetative and sporing forms of mould will be controlled in acid foods by temperatures greater than 85°C. A few moulds are extremely heat resistant and it is not practical to nominate processes which will kill these moulds in all acid foods without causing heat damage to the food itself. Commercially, one attempts to ensure that contamination with these moulds is avoided or raw material likely to be contaminated, e.g. some fruit pulps, is checked for their absence before use.

Definition: Micro-organisms are tiny organisms present almost everywhere,they occur in all fresh foods,in the soil,in dust,on all surfaces and on the skin of people handling food.

1.2 FACTORS ASSOCIATED WITH FOOD SPOILAGE:

Foods are spoilt by the action of: (1) Micro-organisms (2) Enzymes and (3) Insects.

Micro-organisms:

The micro-organisms responsible for food spoilage are moulds, yeast and bacteria.

Moulds:

Moulds are in the form of threads developed on perishable foods and are easily visible to the eye. They contain spores which can spread through the air and start new mould plants. When these moulds find a favorable environment, they germinate and produce a fluffy growth, often white or grey but sometimes bluish-green, red, orange or some other colour, depending upon the variety of the mould. Most moulds are not harmful. A relatively small proportion of the moulds, found on foods are capable of producing toxic materials known as mycotoxins of which aflatoxins is an example.

Moulds classified as fungi develop a multicellular structure visible to the naked eye. They grow from cells called spores present in the air. They settle and multiply on suitable foods. At this stage, they are visible as a fluffy coloured mass and the food is said to have gone mouldy. Moulds grow most readily in most conditions, at temperatures between 20oC and 40oC. They grow on a variety of foods, particularly meat, cheese, fruit and bread, especially if the food is stored in damp conditions. Moulds may remain active at the low temperatures of a refrigerator but they are destroyed by heat above 70oC. They also like a slightly acid medium and this is why they attack citrus and the surface of jams. Why don't you try and give examples of food that can be attacked by moulds. Let's continue.

Food that is contaminated with mould often appears to be safe to eat as only the outer part is affected by mould growth. However, recent research has shown that substances produced by the mould which migrate into the food could be harmful to many organs of the body. It is therefore advisable to discard mouldy food completely, rather than just to remove the mouldy part.

Moulds grow in filaments forming a tough mass which is visible as 'mould growth'. Moulds form spores which, when dry, float through the air to find suitable conditions where they can start the growth cycle again. Mould can cause illness, especially if the person is allergic to molds. Usually though, the main symptoms from eating mouldy food will be nausea or vomiting from the bad taste and smell of the moldy food.

Both yeasts and moulds can thrive in high acid foods like fruit, tomatoes, jams, jellies and pickles. Both are easily destroyed by heat. Processing high acid foods at a temperature of 100°C (212°F) in a boiling water canner for the appropriate length of time destroys yeasts and moulds.

Bacteria

Bacteria are the most widespread of the micro-organisms found in food. They are minute single cells of various shapes. Under ideal conditions, they divide into two every 20 minutes, consequently, millions of them may develop in contaminated food in a short time. They are more dangerous than moulds and yeast because food may be severely infected but not smell, taste or

look bad. Many types of bacteria present are harmless but some do cause illness. The bacteria that cause infections in humans are known as pathogens.

Bacteria are unicellular organism and are much smaller in size than either yeasts or moulds. They occur in different sizes and shapes and are classified as coccus (spheroidal), bacilli (cylindrical) or spirillae (spirillar) on the basis of their shape as seen under the microscope. They also vary in their requirement for food, moisture, acidity, temperature and oxygen. Bacteria can grow and develop rapidly between 20°C and 53°C. Bacteria are classified according to the temperature ranges that they need for growth:

1. A higher temperature than 45°C are known as thermopile, (e.g. in canning industry and milk processing plants).
2. Temperatures between 20-25°C are called Mesophiles.
3. Temperature less than 20°C are called psychrophites (e.g. in Refrigerator and in cold storages).

As all bacteria thrive in similar conditions, it is important to avoid conditions which favour their growth in order to prevent infection. They are active over a wide range of temperatures. Some like warmth and are active at 75oC. Others like cold conditions and grow at temperatures as low as 5oC. This may cause problems in storage of foods. Some bacteria can form resting bodies called spores to protect them when the conditions are unfavourable for normal growth, for example the wrong degree of acidity or alkalinity, temperature or lack of moisture. Although normal bacteria are destroyed during heat treatment by boiling, some spores survive boiling for hours. They can resume normal activity when conditions become more favourable and contaminate some preserved foods. Bacteria are lalled in an acid medium, and therefore they are not a problem in preserving fruits and making jams. The pasteurization of milk does not destroy all bacteria in milk but does destroy those bacteria likely to cause disease. Freezer temperatures must be low enough to prevent bacteria activity during storage. Although some bacteria die in the freezer, some remain inactive in the food and start to grow again when the food thus. The removal of moist are by drying or by addition of large quantities of sugar and salt make conditions unsuitable for bacteria, and these methods are therefore use in food preservation.

Yeasts:

Yeasts are tiny organisms which are not visible to the naked eye, but which can be seen through the microscope. They multiply very fast and cause fermentation by acting on certain components of the perishable foods like fruit juices, syrups etc. During yeast fermentation, the sugars present in the food are broken up to form alcohol and carbon dioxide. Foods liable to be spoiled by yeasts are fruit juices, syrups, molasses, honey, jams and jellies. Let's look at how they reproduce. Yeast cells reproduce by budding. At first a small projection appears at the edge of the parent cell and from this cytoplasm and nutrients flow. As the bud grows, the nucleus moves towards it and divides so that a new nucleus enters the bud. When the bud is almost as large as the parent cell, a wall forms separating if from the parent cell, and it then breaks away. When they are reproducing rapidly, the buds do not break away but continue to reproduce until long chains of yeast cells are formed.

Yeast cells grow and reproduce in conditions similar to those required by other fungi. They need oxygen, warmth, food and moisture in order to grow successfully. Yeast grow best at

temperatures between 25oC and 30oC. Extreme heat destroys all yeasts and most are destroyed at temperatures above 60oC.

Spoilage by Enzymes:

Enzymes are organic catalyst present in living cells. The life of every living cell depends upon the chemical reactions activated by these enzymes. Hence, they cause food spoilage due to the chemical reactions as in cutting apples; it becomes brown while tomato cause develops a black scum. Enzymes are sensitive to heat and are easily destroyed by heat. They can act from 0°C to 60°C; their optimum temperature of reaction is usually 37°C. All enzymes are inactivated by temperatures above 80°C. Therefore, enzyme activity can be prevented by heating foods to temperature which inactivate the enzymes. It can also be prevented by cooling (as in freezing and refrigerated).

Yeasts are microscopic fungi; they are found in the air and soil, and on the surface of fruit. Some are able to tolerate fairly high acidic, salt and sugar concentrations and can grow without the presence of oxygen. the activity of yeast is used in the baking and brewing industries to make bread, doughnut and alcoholic beverages through a process called fermentation. However, they can cause food spoilage in syrups, fruits, fruits juices and jam especially as they can survive without air.

Enzyme action in the food

Food spoilage can also come about through the action of enzymes presents in the food. Enzymes are chemicals which are present in all food. They speed up chemical changes that result in loss of flavour, colour and texture. As enzymes are mainly composed of protein, they are sensitive to heat. They are active in temperatures found in a kitchen on a warm sunny day. They can remain very slightly active at very low temperatures such as those found in the freezer. This is why there is a limit to the time food can be stored in a freezer. The activity of these enzymes stops when they are heated above 70 °C. Heat treatment by blanching (i.e. pouring boiling water on the food) is recommended. Some enzymes remain inactive until the food is harvested or slaughtered. Once activated, such enzymes speed up the process of decay by breaking down the tissues and components of the food in the various ways such as oxidation, browning and ripening.

Enzymes are proteins found in all plants and animals. If uncooked foods are not used while fresh, enzymes cause undesirable changes in colour, texture and flavour. Enzymes are destroyed easily by heat processing. The activity of enzymes in food makes it easier for the micro-organisms responsible for food spoilage to enter the food. Atmospheric oxygen can react with some food components which may cause rancidity or color changes. Infestations (invasions) by insects and rodents, which account for huge losses in food stocks. Low temperature injury - the internal structures of the food are damaged by very low temperature.

Spoilage by insects

Worms, bugs, weevils, fruit flies, moths cause extensive damage to food and reduce its nutritional value and make it unfit for human consumption.

- **Oxidation**

When Oxidation occurs (i.e. when food comes into contact with oxygen) the enzymes cause the destruction of certain nutrients e.g. vitamin c, thiamine and carotene.

- **Browning**

Enzymes again cause browning in certain foods the moment they are exposed to air. When you cut or bruise food such as apple or yam, the exposed surface will discolour and turn brownish due to the activity of enzymes.

- **Ripening**

Enzymes are involved in the process that causes ripening in certain foods such as fruits and vegetables. Unripe bananas for example contain starch which is gradually converted to sugars, until the banana becomes very sweet, and its skin colour changes from green to yellow. Eventually, the skin colour changes to dark brown and it is no longer fit to be consumed.

The activity of enzymes in food makes it easier for the micro-organisms responsible for food spoilage to enter the food. The main micro-organisms responsible for the contamination of food are bacteria, moulds and yeasts. These micro-organisms are invisible to the naked eye, but can be seen under a microscope. They are capable of multiplying very rapidly in the correct moisture, food and temperature conditions. These conditions must be avoided if the risk of food spoilage is to be reduced. Let's now look at each of these micro-organisms and how they operate.

Spore-forming bacteria

Some bacteria form heat resistant bodies as part of their normal life cycle. These heat resistant bodies are called spores and are important in determining the heating procedure necessary to produce a food which will not spoil.The best way to minimise the risk of spoilage of acid preserves by this type of organism is to maintain the pH as low as possible and preferably not greater than 4.0. Some manufacturers are reluctant to alter their formulations to reduce the pH and this means a more severe cook is required if the pH is in the 4.0 – 4.5 range and particularly if the pH is near 4.5.

It is a matter of judgment if the heat process given to acid foods should be designed to take account of spore-forming bacteria. The decision has to be made on the basis of the pH of the product and the association of spore formers with raw materials used in the product.

1.3 MICRO-ORGANISM

Micro-organisms are bacteria and viruses (more commonly known as germs), fungi or parasites. In most workplaces, the risk of catching an infection, such as a cold or flu, is no higher than in any other public place and you do not have to take any action. However, some people who work with animals, or provide care for people, or who clean up or handle waste materials, can be exposed to harmful micro-organisms.

These can cause an infection if they are breathed in, swallowed, or if they penetrate the skin, and can include some very serious illnesses. Some may in turn cause an allergic reaction or are toxic (they produce a poison).

Microorganisms are microscopic, living, single-celled organisms such as bacteria. Ubiquitous throughout the world, microorganisms play a vital role in supporting and maintaining nature and life. Although some bacteria are harmful, the vast majority are not harmful, but in fact beneficial. They keep nature clean by removing toxins from water and soil, and degrade organic matter from dead plants and animals. In the human body they aid in digestion and help prevent invasion by harmful bacteria. Without bacteria, life would not be possible.

Microorganisms were the first living creatures on earth. It is estimated there are a total of 5 nonillion microorganisms on earth (5x1020). You will find nearly 10 million a millilitre of ocean water and 40 million in a gram of soil. They are also ubiquitous on the human body. There are 40 million microorganisms in a millilitre of saliva and make up 10% of a human's total dry weight. In many cases, animals not only live with microbes, but our health is dependent on them (gut bacteria, immune system development, etc.). All living organisms are dependent on microorganisms and their biochemical process. Research has long suggested that microbes help humans by doing things like protecting us from allergies and preventing the spread of malaria. The more we learn about them the more phenomenal these microscopic, single celled-organisms become. They may even be the key to detecting landmines and making radioactive metals inert.

It is important to remember that the same food handling practices are used to prevent all food poisoning diseases. Washing your hands with soap and drying them on a paper towel or with a clean cloth is the best way to stop the spread of bad bacteria.

The four most common types of food poisoning bacteria are discussed below.

Staphylococcus

These bacteria are found on the skin, in sores, infected eyes and in the nose, throat, saliva and bowel of humans. There may be many of these bacteria in the yellow mucus (slimy substance) which comes from the nose or is coughed up when a person has a cold or a lung infection. Staphylococci do not cause illness until they get onto food and grow and multiply. While they are doing this they produce a toxin (poison). It is the toxin which causes the illness. The toxin is not destroyed by cooking the food.

Salmonella

There are hundreds of different types of salmonella bacteria but not all are harmful to humans. They are found mainly in the intestines, bowels and faeces of humans and other animals. It is the salmonella bacteria themselves which can cause salmonella food poisoning.

People can get salmonella food poisoning from:

- poor food handling practices in the home or in food outlets
- seafood caught in polluted water or eggs with dirty shells
- meat or poultry which has been contaminated by poor food handling before it gets to the food outlet, such as at the abattoir

Salmonella food poisoning takes up to 48 hours to develop after the food is eaten. Symptoms include nausea, stomach cramps, diarrhoea, fever and headache, and may last between 3 and 21 days. It can cause death in very young, weak or very old people. People who have cancer or are taking medication for serious health conditions such as heart, kidney or liver problems need to also be particularly careful that they eat safe food.

Clostridium

These bacteria are found in the soil and in the intestines of animals, including cattle, poultry, fish and humans. Food poisoning caused by clostridium bacteria is important to know about because these bacteria are common in the environment. People can get clostridium food poisoning from poor food handling practices in the home, in the factory or in a food outlet, especially relating to cooking and storage/refrigeration temperatures.

Clostridium food poisoning symptoms occur about 12 hours after eating the contaminated food and are similar but usually less severe than the other types. Symptoms include stomach pains, diarrhoea and sometimes nausea and vomiting. Symptoms last about 24 hours. One type of clostridium bacteria produces a very serious food poisoning disease called **botulism.** This

disease is caused by eating food which is contaminated with an extremely poisonous toxin produced by the bacteria Clostridium botulinum. Unless properly treated about one-third of people who get this disease die within 3-7 days.

Campylobacter

These bacteria are found in many animals including dogs, cats, cattle and poultry. The sources of infection from these bacteria are usually contaminated food and water.

People can get campylobacter from:

- ingestion of contaminated food or water (especially undercooked chicken & creek or river water)
- contact with infected animals (especially puppies or kittens with diarrhoea)
- poor food handling (especially by using the same chopping boards, knives and plates for raw and cooked chicken)

Campylobacter food poisoning symptoms usually last from 2 to 5 days. These include diarrhoea, severe abdominal pain, vomiting and fever. It is a serious disease in Indigenous communities because of the possibility of dehydration from diarrhoea.

Disease causing bacteria grow best when there is:

- warmth (37°C-38°C) (Note: human body temperature is 37°C)
- moisture
- food supply

In ideal conditions, bacteria double their numbers every 20 minutes. For example, if a piece of kangaroo meat infected with 100 food poisoning bacteria is left lying on a kitchen bench on a warm day, the bacteria will double their number every 20 minutes, and in 3 hours, the 100 bacteria will multiply to over 50,000 bacteria.

The following table shows how the bacteria will multiply on the meat over 3 hours

1.4 CONTAMINATION DURING HANDLING

- Pathogens can be introduced into food from infected humans who handle the food without thoroughly washing their hands.
- These pathogens are thus transferred from trace amounts of fecal matter present on hands to the food.
- Hand washing and Hand Hygiene information
- Food and kitchen tools and surfaces may become contaminated from raw food products (i.e., meat and poultry).
- Microbes can be transferred from one food to another by using the same knife, cutting board or other utensil without washing the surface or utensil in between uses.
- A food that is fully cooked can become re-contaminated if it touches other raw foods or drippings from raw foods that contain pathogens.Cross-contamination is the physical movement or transfer of harmful bacteria from one person, object or place to another.
- Many pathogens need to multiply to a larger number before enough are present in food to cause disease.
- In general, refrigeration or freezing prevents virtually all bacteria from growing.

- If food is heated sufficiently, parasites, viruses and most bacteria are killed.

1.5 SUMMARY

The control of microbiological spoilage requires an understanding of a number of factors including the knowledge of possible hazards, their likely occurrence in different products, their physiological properties and the availability and effectiveness of different preventative measures. *Food spoilage microorganisms* focus on the control of microbial spoilage and provide an understanding necessary to do this.

1.6 SELF ASSESSMENT QUESTIONS

1. What are the causes of micro-organism?
2. Explain reasons for food spoilage?
3. Describe about harmful bacteria in detail?

1.7 REFERENCE BOOKS:

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2. Journal of Medical Microbiology.

3. Current science.

Dr. Santhi Sri. K. V

Lesson – 2

FOOD PRESERVATION

2.0 Objective

After reading this chapter, you should be able to

To create awareness in Food preservation

To know the characteristics of Microorganism

Structure

2.1 INTRODUCTION

Foods and microorganisms have long and interesting associations which developed long before the beginning of recorded history. Foods are not only nutritious to consumers, but are also excellent source of nutrients for microbial growth. Depending upon the microorganisms present, foods may spoil or preserved by fermentation

Microorganisms can be used to transform raw foods into fermented delights including yoghurt ,cheese, sausages, tempeh, pickles, wine, beers and other alcoholic products. On the other hand, foods also can act as a reservoir for disease transmission, and thus detection and control of pathogens and spoilage organisms are important areas of food microbiology. During the entire sequence of food handling from the producer to the final consumer, microorganisms can affect food quality and human health

To keep microorganisms out of food, contamination is minimized during the entire food preparation process by sterilizing equipment, sanitizing it, and sealing products in wrapping materials. Microorganisms may be removed from liquid foods by **filtering** and sedimenting them or by washing

and trimming them. **Washing** is particularly valuable for vegetables and fruits, and **trimming** is useful for meats and poultry products.

Heat. When **heat** is used to preserve foods, the number of microorganisms present, the **microbial load**, is an important consideration. Various types of microorganisms must also be considered because different levels of resistance exist. For example, bacterial spores are much more difficult to kill than vegetative bacilli. In addition, increasing acidity enhances the killing process in food preservation.

Three basic heat treatments are used in food preservation: **pasteurization**, in which foods are treated at about 62°C for 30 minutes or 72°C for 15 to 17 seconds; **hot filling**, in which liquid foods and juices are boiled before being placed into containers; and **steam treatment** under pressure, such as used in the canning method. Each food preserved must be studied to determine how long it takes to kill the most resistant organisms present. The heat resistance of microorganisms is usually expressed as the **thermal death time**, the time necessary at a certain temperature to kill a stated number of particular microorganisms under specified conditions.

In the **canning** process, the product is washed to remove soil. It is then blanched by a short period of exposure to hot water to deactivate enzymes in the food. Diseased sections in the food are removed, and the food is placed into cans by a filling machine. Sealed cans are then placed into a sterilizing machine called a **retort**, and the food is processed for a designated time and temperature.

Low temperatures are used to preserve food by lowering Slow freezing and quick freezing are used for long-term preservation. Freezing reduces the number of microorganisms in foods but does not kill them all. In microorganisms, cell proteins undergo denaturation due to increasing concentrations of solutes in the unfrozen water in foods, and damage is caused by ice crystals. Several kinds of chemicals can be used for food preservation, including propionic acid, sorbet acid, benzoic acid, and sulfur dioxide. These acids are acceptable because they can be metabolized by the human body. Some antibiotics can also be used, depending upon local laws and ordinances. Tetracycline, for example, is often used to preserve meats. Storage and cooking normally eliminates the last remnants of antibiotic.

In many foods, the natural acids act as preservatives. In sauerkraut, for example, lactic acid and acetic acid prevent contamination, while in fermented milks (yogurt, sour cream), acids perform the same function. For centuries, foods were prepared in this manner as a way of preventing microbial spoilage.

Drying is used to preserve food by placing foods in the sun and permitting the water to evaporate. Belt, tunnel, and cabinet dryers are used in industry for such things as instant coffee and cocoa. Freeze-drying, a process calledlyophilization, is also valuable for producing a product free of moisture and very light.

Ultraviolet radiation is valuable for reducing surface contamination on several foods. This short-wavelength light has been used in the cold storage units of meat processing plants. Ionizing radiations such as gamma rays can be used to preserve certain types of vegetables, fruits, and spices, according to state and U.S. federal regulations.

Food is preserved by either slowing downtheactivity of disease causing bacteria or killingthe bacteria altogether Enzymes are a proteinnaturally found in food that can make it spoilor change color.

They are fragile. They aredestroyed when heated to 150 degrees F.Sterile food has no bacteria. Bacteria are found in food if it hasn't been sterilized.

2.3.1 Moulds

Moulds belong to a big group of Thallophytes called Fungi, which are not capable of carbon assimilation because they do not possess chlorophyll. Hence all fungi depend upon organic matter for their food-either dead organic matter or living plants and animals.

Occurrence:

Moulds, which are multicellular filamentous fungi, are very widely distributed in nature. They grow on any kind of dead organic matter or on living plants and animals.

Structure:

They differ from yeasts and bacteria in that they are multicellular the thallus consisting of thread-like filaments called hyphae, the entire plant body being called a mycelium. The hyphae may be septate (divided at intervals by cross walls) or non-septate (without any cross walls).

Nutrition:

Since they do not contain any chlorophyll, their mode of nutrition is different from that of green plants. Green plants are capable of building up carbohydrates from carbon-dioxide and water. Moulds on other hand, have to take in ready-made organic foods. When the food is absorbed from dead organic matter, the mode of nutrition is said to be saprophytic and when it is obtained from living hosts on which the moulds grow, the mode of nutrition is said to be parasitic.

Reproduction:

Moulds show three kinds of reproduction – vegetative, asexual and sexual. When a small bit of a mycelium is transferred to a new place, it grows into a new mycelium. This method is said to be vegetative. The main method of reproduction among moulds is however by means of asexual reproductive bodies called spores. These asexual spores are of two kinds-those formed inside sporesacs or sporangia and those which are not formed within sporangia and called conidia. Spores are much more resistant to unfavourable conditions than moulds. Spores are very light and are hence suspended in the air and when they settle on any kind of organic matter, germinate into new mycelia.

Economic importance of Moulds:

The biochemical activities of moulds are of great importance in the industrial world. Moulds are perhaps not so important as yeasts and bacteria in this respect, but they produce certain changes that the other two groups do not. Some of the more important biochemical changes brought about by moulds are given below:

1. **Alcoholic Fermentation:** Yeast is usually responsible for the production of alcohol from various sugars like sucrose (cane sugar) molasses and fruit juices. In addition to these sugars various starches (corn, potato, malt etc,) and cellulose are also employed. But before starches and other polysaccharides can be utilized by yeasts, they have to be hydrolysed to soluble sugars. A number of species of Mucor are capable of fermenting starch directly to alcohol. These moulds secrete both amylase (diastase) and a group of enzymes and coenzymes which were together

called "Zymase", earlier. Amylase hydrolyses starch to sugar and "Zymase" converts sugar into alcohol. Mucor rouzii is used in the orient for preparing alcoholic beverages from starch. The mould ix mixed with rice meal and sold as "Chinese" yeast.

2. **Citric Acid:** Citric acid is a natural constituent of citrus and other fruits. It was first isolated and crystallized by Scheele in 1784. Aspergillus niger is sometimes used to prepare citric acid. The carbohydrates that are used for citric acid fermentation are starch, sucrose, glucose, fructose and molasses.

3. **Fumaric acid:** Fumaric acid is an unsaturated acid produced by a number of moulds, specially some species of Rhizopus, the carbohydrates commonly used for this purpose being sucrose, maltose, molassess and starches.

4. **Cheeses** are sometimes ripened with the help of moulds. The soft Camembert cheese is ripened by the mould Penicillium camemberti and the green-streaked Roquefort cheese by P. Roqueforti.

5. **Antibiotics:** A few antibiotics are prepared from moulds e.g. Penicillin from penicillium notatum and P. Chrysogenum.

2.3.2 Yeast

Yeast is made up of a single-celled organism, *Saccharomyces cerevisiae*, which multiplies rapidly when fed sugar in a moist environment. One pound of yeast contains 3,200 *billion* yeast cells! Yeast also thrives on starch, which it converts to glucose, a simple sugar. This process ferments the sugar, which converts to alcohol and carbon dioxide. The carbon dioxide expands the baked goods to produce the light, fluffy textures.

Salt inhibits the growth of yeast. Never mix yeast into salted water. Since most tap water goes through a filtering process which utilizes salt as a refining/cleaning agent, many cooks use only distilled water for baking. However, if you are baking during the hot summer season and find your dough rising too much, the addition of a little extra salt can control that runaway yeast growth.

Most yeast is sold in single-use packets or bulk bags known as **dry active yeast.** Compressed yeast is not as widely available, but can be used to the ratio of one standard cake of compressed yeast to one scant tablespoon of dry yeast. If the dry yeast is stored in airtight packaging, in a cool dry place, it is not necessary to refrigerate it. Yeast should always be at room temperature to begin a recipe. Standard single-use packets contain about 2-1/2 teaspoons.

Now available on the market is fast-rising active dry yeast, which is smaller-grained than conventional active dry yeast and speeds rising times by as much as fifty percent, often eliminating the need for a second rising period. It may be used interchangeably, measure for measure, with active dry yeast. The best method for using this yeast is to mix it directly with the dry ingredients before adding liquid, instead of adding it to warmed liquid and then adding to dry ingredients.

Fresh yeast consists of 70 percent moisture and *must* be stored in the refrigerator. Compressed fresh yeast is highly perishable, as opposed to dry active yeast, and loses its vitality within 2 weeks, even when properly stored refrigerated in an airtight container. Compressed yeast can be

stored in the freezer, but should be defrosted at room temperature and then used immediately. Compressed fresh yeast is difficult to find in the United States.

Dried yeast has become the norm for its staying power in the pantry. Yet, even dry yeast must be stored in an airtight container, with no threat of moisture, and it will lose its life over time. Use 2 teaspoons of dried yeast to a 2/3 ounce compressed yeast cake as a substitution. Kneading also develops a firm gluten structure, providing the framework for the carbon dioxide bubbles.

Brewer's yeast has no leavening properties but is added to products for nutritive benefits, as it is rich in the B vitamins. It is also, of course, used in the brewing of beer.

Salt and Sugar in Yeast Baked Goods

Although salt does inhibit the growth of yeast, it does give a firmer crust, a finer crumb, and adds flavor. Sugars are not essential to leavened baked goods, but they make the product tenderer due to postponement of protein coagulation, allowing the dough/batter to grow to a greater volume before being frozen into stasis by the baking process, as well as adding to flavor. If too much sugar is used, it can slow down the growth of the yeast, with a low-rise result. The relationship of sugar to salt to leavening is crucial to a pleasing final product.

2.3.3 Bacteria:

Bacteria are often maligned as the causes of human and animal disease (like this one, *Leptospira*, which causes serious disease in livestock). However, certain bacteria, the actinomycetes, produce antibiotics such as streptomycin and nocardicin; others live symbiotically in the guts of animals (including humans) or elsewhere in their bodies, or on the roots of certain plants, converting nitrogen into a usable form. Bacteria put the tang in yogurt and the sour in sourdough bread; bacteria help to break down dead organic matter; bacteria make up the base of the food web in many environments. Bacteria are of such immense importance because of their extreme flexibility, capacity for rapid growth and reproduction, and great age - the oldest fossils known, nearly 3.5 billion years old, are fossils of bacteria-like organisms.

2.4 DIFFERENT WAYS TO PRESERVE FOODS :

Refrigeration and freezing : These are the most popular ways. Refrigeration slows bacteria to a crawl to prevent spoiling. Freezing stops bacteria action completely. It can be used on all foods with little effect to the taste, except for fruit.

Canning: This has been around since 1825. You boil food to kill the bacteria and then seal the container (that can be boiled). You seal it before or while the food is boiling. This will prevent any new bacteria from getting in. Once the can is opened, bacteria can get in and spoil it . That is why you refrigerate it after it has been opened. Canning will usually change the taste and texture of the food. Food will also lose nutrition through this process. In the **canning** process, the product is washed to remove soil. It is then blanched by a short period of exposure to hot water to deactivate enzymes in the food. Diseased sections in the food are removed, and the food is placed into cans by a filling machine. Sealed cans are then placed into a sterilizing machine called a **retort**, and the food is processed for a designated time and temperature.

Low temperatures: Low temperatures are used to preserve food by lowering microbial activity through the reduction of microbial enzymes. However, psychrophilic bacteria are known to grow even at cold refrigerator temperatures. These bacteria include members of the genera Pseudomonas, Alcaligenes, Micrococcus, and Flavobacterium. Fungi also grow at refrigeration temperatures.

Dehydration: Most bacteria will die when dehydrated. Drying will change the taste and texture of the food. Often it will change it altogether. An example of this would be a raisin.

Freeze drying: This is different than dehydration. Food is frozen and put in a strong vacuum. The water turns from ice to vapor. The most common use is for instant coffee.

Salting: This is an ancient way of preservation, especially of meat. The salt takes moisture out, so bacteria cannot grow. It is better to salt in cold weather. If it is hot, the meat will spoil quicker, and the salt would not have had time to work.

This method was replaced with refrigeration and freezing. The exception is when using it for taste in salt-cured meat: ham, beef and pastrami. For this method, they soak the meat in 10% salt water for several weeks.

Pickling: This was used for meat, fruit and vegetables in the past. Today, it is just for pickles. They use salt, acid and vinegar. Acetic acid also stops bacterial growth. For pickles you soak in 10% salt water brine for several days, rinse and store in vinegar. This can last for years.

Pasteurizing: When you boil food, you kill bacteria, making it sterile. You also change the taste and nutritional value. Pasteurizing is used for liquids. This will kill some bacteria and stop some enzymes through heating. Milk is the most commonly known product that is pasteurized. Juice and ice-cream are pasteurized also. It would take ½ hour at 145 degrees or 15 seconds at 163 degrees to pasteurize milk.

Fermentation: This will produce alcohol which will kill bacteria Carbonation. This method takes carbon dioxide gas that has been dissolved under pressure. By taking out the oxygen, bacteria stops growing. Carbonated drinks have a natural preservative. Cheese-making uses bacteria and enzymes, naturally forming acids to solidify milk proteins and fat and preserve them. Once turned into cheese, it can be preserved for years. Salt and acid are the main reasons why they can be preserved for so long.

Chemical preservation.

There are 4 kinds used:

benzoates

nitrates

sulfites

sorbic acid

They all either slow down or kill bacteria.

Irradiation: This kills bacteria without changing the food. You need to seal food in plastic, then radiate it. It will become sterile and can be stored on the shelf. It doesn't change the taste. This could prevent food poisoning, but it is not popular because of the radiation. People don't like that idea.

2.5 SUMMARY

Microorganisms play an important role in food handling. proper food preservation techniques should be followed such as Refrigeration and freezing, Canning, dehydration, salting and pickling. Preservation of food can be useful and more economy.

2.6 SELF ASSESSMENT QUESTIONS

1..Explain food preservation techniques in detail?

2. Describe the types of microorganisms in brief?

1.7 REFERENCES

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2. Journal of Medical Microbiology.

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Lesson – 3

MICROORGANISMS

3.0 Objective

After reading this chapter, you should be able to

To create awareness in microorganisms

To know the chemical changes caused by microorganisms

Structure

3.1 INTRODUCTION:

Food protection and food preservation have one aim in common they are intended to prevent contamination and spoilage of foods. Many of the methods of food protection and preservation used today are of ancient origin. Having an understanding of food microbiology in Study is important for food protection and preservation practice. In this study session, you will learn about the principles and methods of food protection and preservation, and also the details of safe ways of food procroom temperature allows microorganisms to start to grow and multiply; therefore, cooked food must be stored very carefully. If it cannot be eaten straight away, it should be kept as cold as possible, ideally in a refrigerator, to avoid growth of microorganisms.

If any food has to be reheated, this must be done thoroughly. If food is only warmed and not reheated properly, microorganisms will multiply in it, so you need to heat it enough to destroy them. Infant foods should not be stored at all, but must be used immediately assessing and preparation.

Food protection methods are measures taken to protect food from being contaminated by any agent. All food must be protected at all times during storage and preparation from the following contaminants:

- any water that is not known to be safe, including overhead leaks and drips
- dirty hands
- coughing and sneezing
- dust and soot
- flies, rodents and other vermin
- insecticides and other chemicals
- unclean utensils and work surfaces

- cigarette smoke.

These factors either affect the food directly to make it unsafe (such as cigarette smoke or soot), or, like coughs and sneezes or insects, contaminate the food with microorganisms. The most important way of preventing contamination is by adopting good **food handlers' hygiene.** This is the term for a group of practices that should be followed at all times by anyone handling food at any stage of the food supply process. Food handlers' hygiene in retail and commercial premises where food and drink is sold to customers is of critical importance and this is discussed further. The same principles also apply in domestic situations. The importance of promoting good food handlers' hygiene is

To prevent food contamination and spread of disease.
To ensure the good health of people eating the food.
To protect the health of the food handler.

Anyone handling food should avoid bad habits such as scratching, touching the hair, nose or mouth, having unclean hair, unclean and long fingernails, smoking, and coughing or sneezing in food handling and preparation areas. They should always wash their hands before starting to prepare food, and after every interruption, particularly after using the toilet. People who have skin infections, diarrhea or sore throats should avoid handling food.

There are other general principles for preventing food contamination:

- All water used in food preparation should be wholesome.
- All dishes, glasses and utensils must be kept clean by regular washing in clean water, and clean utensils should be kept covered.
- All surfaces that come into contact with food should be meticulously clean (Figure 10.1).
- Food storage, preparation and serving areas should be free of pets, rats, mice and insects.
- Food should be covered, and kept separate from chemicals and poisons (which should be clearly labeled).
- Cloths that come into contact with dishes and utensils, and that are used to cover food, need to be changed daily and boiled before use.

Principles of Food handling

The basic principles of food preservation primary involves the process of inhibiting

- The growth and activity of microorganism
- Activity of endogenous enzymes
- Chemical reactions which may deteriorate the quality of food
- Invasion and spoilage by insects and rodents

In addition, spoilage of food may be caused during mechanical handling, processing, packaging, storing and transportation. Appropriate care has to be exercised to prevent deterioration of quality of food.

Several methods are available for preservation of food based on the above principles, the method include:

- Preventing the accessibility of food to microorganism by asepsis and packaging
- Physical removal of microorganisms from food by filtration or centrifugation

- Hindering the growth and activity of microorganism by use of preservatives, use of low temperatures atmospheric control in packaging and storing of foods and decreasing water activity in foods by drying or evaporation
- Killing the microorganism by use of high temperature and ionizing radiation
- Inactivation of endogenous enzymes by moderate heating
- Inhibition of chemical reaction through the use of chemical additives
- Fermentation of foods to yield more stable or less perishable food product

Food preservation as it is practiced in the industry always involves the use of combination of methods for achieving maximum effectiveness.Asepsis or preventing the accessibility of food to microorganisms is well exemplified in nature, the protective covering in natural foods such as skins on fruits and vegetables, shells on eggs and nuts and skins and membranes on livestock and fish prevent the attack by microorganisms and maintain the living tissues in healthy condition.

Microbial attack is facilitated only after the death of the animal or when the skin is physically damaged. Packaging of foods and food products in a variety of materials such as metal cans, plastic films pouches, bags or boxes, paper bags or cartons and glass bottles provide effective protection against microbial attack.Filtration or centrifugation is adopted to physically remove microorganisms particularly in liquid foods such as milk, soft drinks, fruits juices and alcoholic beverages such as wine and beer.

Principle of Food Preservation.

Food is particularly vulnerable to contamination while it is being prepared for eating. It is important to remember food handlers' hygiene and to ensure that all surfaces and utensils are clean. Foods intended to be eaten raw, such as fruit and some vegetables, must be washed carefully in clean, safe water (Figure 10.2). Food that is to be cooked must be cooked thoroughly to kill all pathogenic microorganisms. All parts of the food must reach a temperature of at least 70°C. You cannot tell how hot the food is just by looking, so it is important to cook the food for long enough to make sure that it is all cooked through. Cooking, as well as being a very important part of food preparation, is also used for preserving food; this is the subject of the next section.

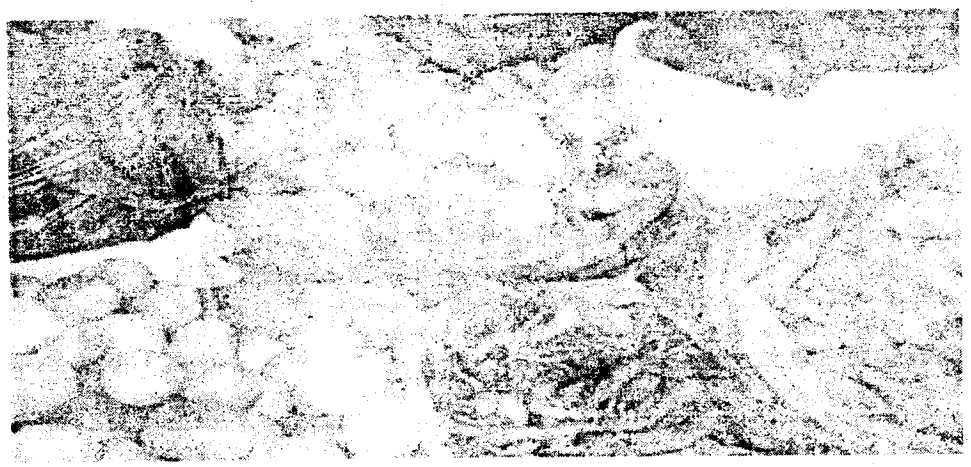

Figure 10.2 Vegetables must be clean before cooking. (Photo: Janet Haresnape)

3.2 FOOD PRESERVATION AND MICROORGANISMS:

Icludes a variety of techniques that allow food to be kept for extended periods of time without losing nutritional quality and avoiding the growth of unwanted microorganisms. There are three basic objectives for the preservation of foods:

- Prevention of contamination of food from damaging agents.
- Delay or prevention of growth of microorganisms in the food.
- Delay of enzymic spoilage, i.e. self-decomposition of the food by naturally occurring enzymes within it.

For storing or preserving food, one or several of the living conditions needed for the growth of microorganisms have to be removed. Like humans, microorganisms need a source of food and water; and they also need a suitable pH and temperature to grow, so food preservation techniques aim to target these requirements. Food preservation depends on procedures which effectively manage the microbial content of foods and on processes that aiter or delay the activities of enzymes in the food. The techniques may be applied separately or in combination. Their aims are to prevent contamination in the first place, to remove or reduce the numbers of contaminants, and to prevent microbial growth. We describe them below.

3.3 PRESERVATION OF CONTAMINATION (ASEPTIC TECHNIQUE)

This technique simply means to prevent contamination of the food by spoilage agents or by contact with them. The word 'aseptic' means free from harmful bacteria, viruses etc. The technique requires either using an artificial covering for the food, or keeping its natural protective covering if there is one. Examples of natural coverings are the shells of eggs, fat or skins in animals, and/or the skin or peel of fruits. Leaving the natural covering of the food intact, or applying a clean artificial cover, can prevent microorganisms from entering or dropping on to the food.

Removal or reduction of microorganisms

Microorganisms can be physically removed from food, or their numbers reduced, by techniques like washing, trimming, sieving and filtration. For example, vegetables and fruit should be washed in clean water; any damaged or dirty parts of vegetables should be trimmed off with a clean knife; flour can be sieved to remove any unwanted contaminants.

The use of temperature and role of Microorganism:

Heat is one of the oldest methods of destroying microorganisms in food processing and preservation. The greatest advance in food hygiene was inadvertently made when humans discovered the advantage of boiling, roasting, baking and other heat treatments of food, hence preserving the food for longer periods. Food is also rendered safe by the application of heat because most pathogenic microorganisms are comparatively heat-sensitive. Some of the methods of heat treatment used for food preservation are discussed below.

Boiling is the process of applying heat to water until the temperature reaches about 100°C. Boiling foods in water cannot completely destroy all microorganisms, but the vegetative cells of bacteria, yeasts and moulds are generally quickly destroyed at temperatures of 100°C or above. Spores of some bacteria are extremely resistant to heat and are not killed at this temperature, although their growth is prevented. For this reason, boiling food can rarely be relied upon to ensure complete destruction of all organisms. However, most pathogens are killed, provided that sufficient exposure time is maintained. Although the spores of *Clostridium botulinum*, which causes botulism, are extremely heat-resistant, the toxin produced by this organism is readily destroyed by boiling. However, some toxins produced by other bacteria such as staphylococci are not easily inactivated. Thermopiles (heat-loving) organisms may survive the effects of boiling and can cause food spoilage if environmental conditions are favourable for them.

Bacterial destruction by heat is affected by time and temperature variation. The higher the temperature, the more rapid is the destruction. On the other hand, as the temperature is lowered, the time of exposure (**holding time**) needs to be longer. Cooking can have some disadvantages. It can damage the food's appearance, texture and flavour, and may also destroy some important vitamins. Nevertheless, the advantages of cooking outweigh the disadvantages because it inhibits spoilage and possible disease transmission.

Pasteurisation is a process of heat treatment of milk, beer and some other beverages. It requires sufficient holding time to assure the thermal destruction of pathogens and organisms responsible for spoilage, without altering the nutritional value. It involves heating the food to a specific temperature for a specific time and then cooling rapidly. Pasteurisation kills most but not all of the microorganisms present. It is a very useful method when more rigorous heat treatment could harm the quality of the product, as in the case of milk, and when the aim is to kill only the pathogens that are not very heat-resistant.

The temperature applied and the holding time of pasteurisation vary with the equipment available and the type of food product. In milk pasteurisation, the time-temperature combination is selected on the basis of the thermal death time of the most resistant pathogens (TB bacilli) that may be present in raw milk, and the maximum temperature and time at which the taste, palatability and nutritive value of milk are maintained. Normally milk is pasteurised at 62.8°C for at least 30 minutes or at 71.7°C for at least 15 seconds, or, if using ultra-high temperature (UHT), at 135°C for 1–2 seconds. UHT milk is sterilised, meaning all forms of life are destroyed. This extends its storage time but does affect the taste.

Blanching is a mild pre-cooking operation which can reduce the bacterial load on vegetables by 90%. It means the application of boiling water or steam for a short time. It wilts some bulky vegetables and prevents discoloring of others. It cleans peas of the moist and sticky material around them. Blanching vegetables prior to canning, freezing or drying helps to remove soil, insects and microorganisms, and destroys or slows the action of enzymes. It sets the green colour and generally facilitates dicing, peeling and packing.

Canning is one of the most widely used modern methods of processing and preserving food. It involves the careful preparation of food packed into a sealed tin, glass or plastic container

which is subjected to defined high temperatures (above 100°C) for an appropriate period of time, and then cooled. Following the thermal (heat) processing, the sealed container must be cooled immediately to a temperature of about 38°C to prevent unnecessary adverse effects of heat on the texture, flavor or colour of the food.

The canning method involves the following steps: sterilizing the food to be canned, packing it in sterile, air-tight stainless metal, glass or plastic containers, and then hermetically sealing (i.e. with a complete, airtight seal) the containers to prevent contamination during handling and storage. In the heat process, all vegetative bacteria are destroyed and spores cannot grow. Any can that is damaged or swollen should not be used. A swollen, bulging can indicates that gas is being produced on the inside and demonstrates there is microbial activity in the food, so it would not be safe to eat.

Unlike high temperature, cold is not an effective means of destroying pathogenic bacteria, viruses and toxins in foods, but it can retard their multiplication and metabolic activities. No food or food product is rendered free from microorganisms by low temperature (by freezing or refrigeration). This explains the generally accepted danger of refreezing any kind of thawed foods. Certain parasites, such as *Taenia* cysts in beef and all stages of *Trichinella spiralis,* can be completely destroyed by storage of infected food at -18°C for periods of 20 to 30 days, depending upon the rate of cold penetration. The most important prerequisite for successful preservation by cold is that the food must be clean to start with.

Chilling involves reducing food temperatures, but only to approximately -1°C. Refrigerators for cold storage/chilling are normally used at 0°C to +8°C for preservation of a wide variety of food products (see Figure 10.3).

Freezing of food, when carried out properly, is one of the best methods of preserving foodstuffs in as nearly natural a state as possible. Freezing preserves the storage life of foods by slowing down enzyme reactions and the growth of microorganisms. A low storage temperature of at least -12°C is important if prolonged storage life is desired without losing flavour. Needless to say, freezing foods to preserve them is only possible with a freezer and reliable power supply. Vegetables with a high moisture content do not freeze well because cellulose (in plant cell walls) tends to be broken down by enzymes regardless of the rate of freezing, making the vegetables soft. Therefore, for such food items, blanching to destroy enzyme activity is required prior to freezing.

This is a dehydration process by which the water/moisture content of the food is removed or decreased. Pathogenic and other bacteria cannot multiply in the absence of water. Most tend to die in foods that have been dehydrated to a moisture content of 10–20% of weight. Drying, however, may not kill spores. Drying also achieves food preservation by inactivating enzymes.

Drying or evaporation methods have been applied to nearly every kind of watery food, including milk. Although the loss in vitamins and nutritional value is usually minor, some foods change physically and chemically, and are sometimes altered in natural colour and flavour. Other dried products do not compare favourably with their fresh counterparts due to difficulties in

reconstitution, i.e. adding water to return the food to its original form. One traditional form of dried food is *quanta* (Figure 10.4). *Quanta* is made from sliced meat which is hung in the air to dry.

Figure 10.4 *Quanta*: an example of food preserved by drying. (Photo: Pam Furniss)

Not all microorganisms are bad. Certain microorganisms are necessary in the preparation and preservation of many foods and beverages. Essentially, **fermentation** (a controlled microbial action) is a process of anaerobic or partially anaerobic oxidation of carbohydrates that produces acids and alcohol. It is one of the oldest methods of food preservation. In fermentation, food preservation is achieved by the presence of acid or alcohol, which creates unfavourable environmental conditions for decomposing and other undesirable bacteria.

Foods commonly processed and preserved by fermentation methods are milk and milk products, beef, vinegar, drinks like beer and wine, and pickled fruits and vegetables. Pickling is the process of preserving food by anaerobic fermentation either in brine (salt solution) or in an acid solution, usually vinegar. The concentrations of the pickling agents and the time needed for pickling are determined by the type of food. Fermented and/or pickled food products are semi-perishable and must be protected from moulds, which are able to attack the acids and permit the invasion of spoilage organisms.

It has been customary to classify chemicals incorporated into food for preservation purposes as 'intentional additives'. Additives used at food industry level include vitamins, mould inhibitors, bactericides, emulsifiers, minerals, food colouring, synthetic flavours and sweeteners. Chemicals that get into food accidentally are referred to as 'unintentional additives'. They include the unavoidable residues of agricultural chemicals, pesticides or antibiotics.

There are several traditional methods of food preservation used at the household level that can be classed as chemical methods. Substances such a sugar, salt, vinegar, spices and wood-smoke are generally regarded as safe and natural preservatives. Salting, sugaring and smoking are **all** methods of **curing** foods. Curing is a general term that covers all these types of food preservation.

Salting is the addition of salt (sodium chloride or NaCl) to food for the purpose of preservation. The growth of microorganisms is inhibited by the salt, which has the effect of drawing water out of the bacterial cells so they become dehydrated and die. In this manner, salt, in combination with other measures, acts as a preservative in many foods such as butter, cabbage, cheese, cucumber, meat and fish. It also gives a desired flavour to the food. Salting can be done by rubbing adequate quantities of dry salt into foods, or by immersion, where the food item is soaked in a concentrated salt solution (i.e. brine). For effective preservation, the concentration of the brine solution has to be maintained above 18%. This is approximately one cupful of salt to five cups of water.

Sugaring refers to the action of sugar in food preservation. It is similar to the action of salt in that it depends on the removal of water. In concentrations of at least 65%, sugar solution is widely used as a sweetening and preserving agent. However, care is needed because at low concentrations, sugar solution can support the growth of microorganisms. It has been found that microorganisms rarely survive in solutions above 20–25% sugar concentration.

Smoking is one of the oldest methods used to improve the quality of food and is commonly used to preserve meat and fish. The smoking process involves exposing food to smoke from burning or smouldering wood or other plant material. It partially preserves the food by surface drying, i.e. removing moisture from the surface of the food, but it is not a reliable method of preservation unless combined with some other method such as salting or drying.

Spices also have some uses in food preservation because they tend to inhibit the growth of staphylococci and other bacteria. However, they have a very limited application because they often get contaminated themselves by a number of bacteria. There are some other methods of food preservation that are used in the food industry and require special equipment, for example, irradiation and vacuum packing. Irradiation is the process of exposing food to ionising radiation in order to destroy microorganisms. Vacuum packing depends on the removal of oxygen from food packaging to prevent the growth of aerobic bacteria that will decompose the food.

Chemical change:

When something undergoes a "chemical reaction" and a new substance is formed as a result, we call this *chemical change*. In some instances, simply applying heat can cause a chemical change. Cooking pancakes, for instance, is an example of a chemical change; the pancake batter "changes" from a liquid to a solid. Rust forming on metal is another example of a chemical change. The iron reacts with water and oxygen to create a new substance — rust.

3.4 SUGGESTIONS:

1. The aim of food protection is to protect food from all possible sources of contamination at all stages, including storage and preparation.
2. It is essential that all food handlers are aware of the need for good personal hygiene to protect the food from contamination and prevent disease.

3. Food must be stored correctly, in an appropriate space, at the correct temperature and avoiding contact with any source of contamination.
4. Food preservation methods are used to keep foods safe for extended periods of time.
5. Recommended methods for safe food preservation are aimed at preventing contamination, reducing microbial numbers, preventing microbial growth and delaying self-decomposition.
6. There are many different methods of food preservation that can be used for different foods.

Food protection methods are measures taken to protect food from being contaminated by any agent. All food must be protected at all times during storage and preparation from the following contaminants:

- any water that is not known to be safe, including overhead leaks and drips
- dirty hands
- coughing and sneezing
- dust and soot
- flies, rodents and other vermin
- insecticides and other chemicals
- unclean utensils and work surfaces
- Cigarette smoke.

These factors either affect the food directly to make it unsafe (such as cigarette smoke or soot), or, like coughs and sneezes or insects, contaminate the food with microorganisms. The most important way of preventing contamination is by adopting good **food handlers' hygiene.** This is the term for a group of practices that should be followed at all times by anyone handling food at any stage of the food supply process. Food handlers' hygiene in retail and commercial premises where food and drink is sold to customers is of critical importance and this is discussed.

The same principles also apply in domestic situations. The importance of promoting good food handlers' hygiene is:

- To prevent food contamination and spread of disease.
- To ensure the good health of people eating the food.
- To protect the health of the food handler.

3.5 SUMMARY:

Anyone handling food should avoid bad habits such as scratching, touching the hair, nose or mouth, having unclean hair, unclean and long fingernails, smoking, and coughing or sneezing in food handling and preparation areas. They should always wash their hands before starting to prepare food, and after every interruption, particularly after using the toilet. People who have skin infections, diarrhea or sore throats should avoid handling food.

3.6 SELF ASSESSMENT QUESTIONS

1. Explain the principles of food spoilage?
2. What are the factors influencing in food spoilage?
3. Write short notes on food enzyme?

3.7 Reference books

1.Food toxicology part A.principles and concepts by jose M.concon marcelDekkher Inc, NEW YORK,1988.

2. Journal of Medical Microbiology

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Lesson – 4

CONTROL OF MICROORGANISMS

4.0 Objective

After reading this chapter, you should be able to

1. To create awareness in control of microorganism..
2. To plot standard growth curve
3. To determine the generation time of given bacteria.
4. To know the food enzymes present in food

Structure

4.1 INTRODUCTION:

With enough food and the right conditions, microbes will grow indefinitely. This can be useful in certain industrial processes, such as the production of insulin or biofuels. When the microbes cause diseases, however, uncontrolled growth can lead to people becoming more sick or dying. In laboratories that work with microbes, controlling their growth is essential for obtaining useful experimental results.

Sometimes controlling microbial growth means killing them outright. This is often done in hospitals to prevent infections caused by microbes living on medical equipment or other surfaces. These non-selective methods kill all microbes at once. Other times, specific microbes are targeted using a variety of chemicals and drugs. Laboratories use methods to kill microbes, but they also grow specific microbes under controlled conditions.

Sterilization is a method that kills or eliminates all of the microbes in a sample or on a surface. The purpose of sterilization is to kill all living microbes, along with any dormant ones. This

makes the sample or surface "sterile." Sterilization generally uses heat, chemicals, radiation or the direct removal of microbes. The method used depends upon the types of microbes present and the nature of the sample or surface. Radiation may work well in the laboratory, but may not be convenient — or safe — in the home.

Heat is the most common method. It is used in laboratories, hospitals and at home. Incineration uses open flames to kill microbes. This is done in laboratories to sterilize the equipment used to collect samples. Boiling is effective at killing microbes in water or other liquids. This can be used to sterilize drinking water while hiking. Steam heat is the basis of many sterilization procedures in laboratories and hospitals. This method uses an autoclave — like a pressure cooker — to kill microbes on equipment and clothing.

Many chemicals and gases are lethal to microbes. These can be used to sterilize equipment and surfaces in hospitals and laboratories. Some of these substances are also dangerous to humans, so special protection is required when using them. Ethanol — pure alcohol like that found in alcoholic drinks used to sterilize surfaces. Ethanol is also found in many commercial hand sanitizers and cleaning wipes. In order to be effective, though, the ethanol must be left on the surface long enough to kill the microbes.

Radiation is also effective at killing microbes. One common type of radiation used is ultraviolet, the same type that is found in sunlight. Special light bulbs that produce only ultraviolet light are used. These may be installed in a laboratory, or used as part of a handheld device. The ultraviolet light, if strong enough, damages the DNA of the microbes beyond repair. This kills the microbes. Antibiotic resistance in bacteria is a major problem for medicine and agriculture, where antibiotics are used widely to treat diseases. Normally, when an antibiotic is given to a patient, all of the bacteria that it targets are weakened or killed. Weakened bacteria are then killed by the patient's immune system. Resistance to antibiotics results from antibiotics being overused or misused. This may occur when a patient does not take all of the antibiotics prescribed by a doctor, or uses them to treat a cold caused by a virus. Antibiotics are not effective against viruses.

Some bacteria are naturally resistant to antibiotics. Others develop resistance when antibiotics are not used long enough or in the right amount. When this happens, some bacteria may survive. The survivors have developed a resistance to the antibiotic. The next time the drug is used, the bacteria will not be killed. Over time, this resistance is passed onto the bacteria's offspring. Bacteria can also transfer their resistance to other bacteria. Over time, more bacteria have grown resistant to one or more antibiotics. As this happens, doctors have fewer tools available to combat potentially-deadly infections.

4.2 PHYSIOCHEMICAL FACTORS INFLUENCING

Key to your industrial applications is having a database that is easy to find and easy to get. The TPC (Thermo-Physico-Chemical) property values of MOLINSTINCTS are determined based on two steps: Step 1 to apply our innovative computational technologies based on quantum mechanics, fundamental theories, and modern modeling approaches, and step 2 to perform manual inspection based on chemical analysis theories, e.g., similarity analysis. Most of the property data including physical, physicochemical, thermo chemical, thermodynamic, thermo physical, and transport properties are available in MOLINSTINCTS. Constant TPC Properties There is 35 important properties which are invariant with temperature. Critical properties for your reactions and chemical processes covered by MOLINSTINCTS includes, a centric factor, normal boiling

point, critical compressibility factor, critical pressure/temperature/volume, dipole moment, electron affinity, enthalpy (heat) of formation/combustion/fusion, entropy, flammability limit, flash point, Gibbs free energy, ionization potential, liquid molar volume, parachor, polarizability, refractive (refraction) index, Hildebrand solubility parameter, radius of gyration, Van der Waals area & volume, and magnetic susceptibility. Simply click and get the property as needed.

- Values obtained from other widely used methods as well as from MOLINSTINCTS are available for comparison. Deviations from the experimental data are also provided if the data are available in our collection for 3+ years.

- Temperature Dependent TPC PropertiesMOLINSTINCTS has 11 temperature dependent properties, and more systematic information is available for you. Industrially important properties, e.g., heat (thermal) capacity, enthalpy (heat) of vaporization, liquid density, thermal conductivity, vapor pressure, chemical (molecular) viscosity, second viral coefficient, and surface tension, are covered.

- Property values from MOLINSTINCTS and other methods are provided in a table, which can be copied and pasted into another program. The level of accuracy is again available compared to the experimental data if they are found in our collection. The property values are presented in a 2-dimensional chart as well for better analysis.

4.3 GROWTH CURVES:

Growth The increase in the cell size and cell mass during the development of an organism is termed as growth. It is the unique characteristics of all organisms. The organism must require certain basic parameters for their energy generation and cellular biosynthesis. The growth of the organism is affected by both physical and Nutritional factors. The physical factors include the pH, temperature, Osmotic pressure, Hydrostatic pressure, and Moisture content of the medium in which the organism is growing. The nutritional factors include the amount of Carbon, nitrogen, Sulphur, phosphorous, and other trace elements provided in the growth medium. Bacteria are unicellular (single cell) organisms. When the bacteria reach a certain size, they divide by binary fission, in which the one cell divides into two, two into four and continue the process in a geometric fashion. The bacterium is then known to be in an actively growing phase. To study the bacterial growth population, the viable cells of the bacterium should be inoculated on to the sterile broth and incubated under optimal growth conditions. The bacterium starts utilising the components of the media and it will increase in its size and cellular mass. The dynamics of the bacterial growth can be studied by plotting the cell growth (absorbance) versus the incubation time or log of cell number versus time. The curve thus obtained is a sigmoid curve and is known as a standard growth curve. The increase in the cell mass of the organism is measured by using the Spectrophotometer. The Spectrophotometer measures the turbidity or Optical density which is the measure of the amount of light absorbed by a bacterial suspension. The degree of turbidity in the broth culture is directly related to the number of microorganism present, either viable or dead cells, and is a convenient and rapid method of measuring cell growth rate of an organism. Thus the increasing the turbidity of the broth medium indicates increase of the microbial cell mass (Fig 1) .The amount of transmitted light through turbid broth decreases with subsequent increase in the absorbance value.

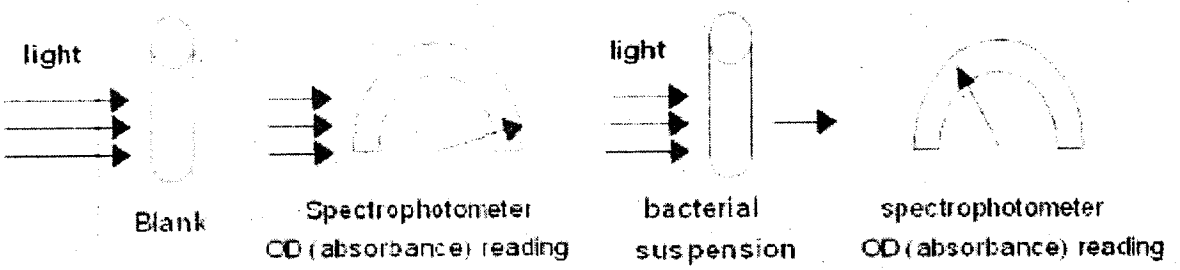

Fig 1: Absorbance reading of bacterial suspension

The growth curve has four distinct phases

1. Lag phase

When a microorganism is introduced into the fresh medium, it takes some time to adjust with the new environment. This phase is termed as Lag phase, in which cellular metabolism is accelerated, cells are increasing in size, but the bacteria are not able to replicate and therefore no increase in cell mass. The length of the lag phase depends directly on the previous growth condition of the organism. When the microorganism growing in a rich medium is inoculated into nutritionally poor medium, the organism will take more time to adapt with the new environment. The organism will start synthesising the necessary proteins, co-enzymes and vitamins needed for their growth and hence there will be a subsequent increase in the lag phase. Similarly when an organism from a nutritionally poor medium is added to a nutritionally rich medium, the organism can easily adapt to the environment, it can start the cell division without any delay, and therefore will have less lag phase it may be absent.

2. Exponential or Logarithmic (log) phase

During this phase, the microorganisms are in a rapidly growing and dividing state. Their metabolic activity increases and the organism begin the DNA replication by binary fission at a constant rate. The growth medium is exploited at the maximal rate, the culture reaches the maximum growth rate and the number of bacteria increases logarithmically (exponentially) and finally the single cell divide into two, which replicate into four, eight, sixteen, thirty two and so on (That is 2^0, 2^1, 2^2, 2^3.....2^n, n is the number of generations) This will result in a balanced growth. The time taken by the bacteria to double in number during a specified time period is known as the generation time. The generation time tends to vary with different organisms. *E.coli* divides in every 20 minutes, hence its generation time is 20 minutes, and for *Staphylococcus aureus* it is 30 minutes.

3. Stationary phase

As the bacterial population continues to grow, all the nutrients in the growth medium are used up by the microorganism for their rapid multiplication. This result in the accumulation of waste materials,

toxic metabolites and inhibitory compounds such as antibiotics in the medium. This shifts the conditions of the medium such as pH and temperature, thereby creating an unfavorable environment for the bacterial growth. The reproduction rate will slow down, the cells undergoing division is equal to the number of cell death, and finally bacterium stops its division completely. The cell number is not increased and thus the growth rate is stabilised. If a cell taken from the stationary phase is introduced into a fresh medium, the cell can easily move on the exponential phase and is able to perform its metabolic activities as usual.

4. Decline or Death phase

The depletion of nutrients and the subsequent accumulation of metabolic waste products and other toxic materials in the media will facilitates the bacterium to move on to the Death phase. During this, the bacterium completely loses its ability to reproduce. Individual bacteria begin to die due to the unfavorable conditions and the death is rapid and at uniform rate. The number of dead cells exceeds the number of live cells. Some organisms which can resist this condition can survive in the environment by producing endosperms.

4.4 DESTRUCTION OF MICROORGANISM:

Heat is lethal to microorganisms, but each species has its own particular heat tolerance. During a thermal destruction process, such as pasteurization, the rate of destruction is logarithmic, as is their rate of growth. Thus bacteria subjected to heat are killed at a rate that is proportional to the number of organisms present. The process is dependent both on the temperature of exposure and the time required at this temperature to accomplish to desired rate of destruction. Thermal calculations thus involve the need for knowledge of the concentration of microorganisms to be destroyed, the acceptable concentration of microorganisms that can remain behind (spoilage organisms, for example, but not pathogens), the thermal resistance of the target microorganisms (the most heat tolerant ones), and the temperature time relationship required for destruction of the target organisms. The extent of the pasteurization treatment required is determined by the heat resistance of the most heat-resistant enzyme or microorganism in the food. For example, milk pasteurization historically was based on *Mycobacterium tuberculosis* and *Coxiella burnetti*, but with the recognition of each new pathogen, the required time temperature relationships are continuously being examined.

A thermal death curve for this process is shown below. It is a logarithmic process, meaning that in a given time interval and at a given temperature, the same percentage of the bacterial population will be destroyed regardless of the population present. For example, if the time required to destroy one log cycle or 90% is known, and the desired thermal reduction has been decided (for example, 12 log cycles), then the time required can be calculated. If the number of microorganisms in the food increases, the heating time required to process the product will also be increased to bring the population down to an acceptable level. The heat process for pasteurization is usually based on a 12 D concept, or a 12 log cycle reduction in the numbers of the organism.

4.5 Food enzymes

This Regulation harmonises at Community level the authorisation procedure and the use of enzymes in foodstuffs. Its objective is to ensure the smooth functioning of the internal market while guaranteeing a high level of protection for human life and interests. The Regulation

aims to create a list of authorised enzymes, to lay down the conditions for the use of enzyme Enzymes are very specific. Each has a particular job it does and it does only that job. So you want to get the right type of enzymes for the right type of food or job you want the enzyme to work on. Enzymes must have the right shape and chemistry to function. Here are some animations that shows how the shape of the enzyme and the substrate are important.

Enzymes in food can help digest some types of foods. Examples include lactase supplements for lactose-intolerant people and alpha-galactosidase (Beano) for help digesting those beans and such. Enzymes themselves do not have much nutritional value beyond that of any other protein, though. Most ingested enzymes are broken down in the gut just like other proteins. The way microwaves affect food is through the heat they create when they are absorbed. In other words, microwaving food denatures food enzymes because it heats them. There is nothing special about the microwave; it is just a convenient way to heat food quickly. If you want to preserve enzyme activity in food, do not heat it.

This is the reason that jello will not set if you put fresh pineapple, kiwi, or papaya in it. The protein-degrading enzymes in those fresh fruits break down the gelatin. Cooking the fruits first (such as when they are canned) destroys this effect. To overstate digestive food enzymes benefits it's almost impossible. Presence or absence of enzymes in food is matter of life, or death. Yet, medical and nutritional fraternity totally ignores this and food manufacturing industry is systematically destroying every trace of enzymes in food. They have perfected science of enzyme destruction. In their ignorance they are proudly proclaiming the benefits of destroying enzymes. And countless billions of unsuspecting souls are supporting them in their Endeavour, not knowing that they are digging their own graves.

Enzymes are proteins used by the body to increase or decrease the speed of chemical reactions. Though there are many different kinds of enzymes, we commonly think of the digestive enzymes because they make it possible for our body to break down and assimilate the foods we eat. A diet rich in enzymes can increase energy and stamina, as well as support weight loss, healthy skin, and overall good health.

Papayas

Papayas are a tropical fruit. They contain large amounts of the enzyme known as papain. A plant-based enzyme, papain is referred to as a cysteine protease or proteolytic enzyme, which refers to the papaya's ability to break the peptide bonds of proteins, hydrolyzing them into smaller units known as amino acids. Eating raw papaya is a good source of this potent digestive and systematic enzyme. Chewable supplements are available in concentrated form Pineapples Raw pineapples contain the enzyme bromelain. Like papain, bromelain is a proteolytic enzyme. Besides contributing to the digestion of proteins, bromelain is a natural anti-inflammatory and anti-coagulant. The highest concentration of bromelain is found in the stem of the pineapple, which is edible, though hardly palatable. Some bromelain is found in the pineapple fruit. Those looking to supplement with larger amounts can find a bromelain enzyme supplement at any health food store.

Sprouts

Sprouts are the seeds of many different types of grains. They are packed with nutrients and may contain more than 100 times more enzymes than fruits and vegetables. Sprouts are most concentrated with active enzymes when they are germinated. Germination requires soaking the seeds in water to allow them to sprout. Once activated, the live enzymes are potent health-promoting factors for proper digestion and overall wellness.

Raw Nuts and Seeds

Nature wisely endowed all raw, natural foods with the proper enzymes for digestion. Nuts and seeds contain lipase, the enzyme that breaks down lipids or fats. Triglyceride fats are hydrolyzed to break the ester bonds, creating fatty acids and glycerol. Most fat is digested in the small intestine after bile from the gall bladder breaks large triglycerides into smaller units. The pancreas makes lipase; however, when dietary sources are deficient it puts added strain on this important organ. Roasting nuts and seeds destroys the live enzymes. Eat them raw for a healthy dose of essential enzymes.

Raw Fruits and Vegetables

Just as nuts and seeds contain lipase for fat digestion, fruits and vegetables contain enzymes to break down carbohydrates. Amylase is the main digestive enzyme for carbohydrate metabolism. It is present in our saliva, as digestion truly begins in the mouth. Amylase works to break carbohydrates into simpler sugars that can be used for energy and metabolism. Fruit and vegetable enzymes are devitalized by cooking above 118 degrees Fahrenheit. This means that steaming and microwaving destroys most of the enzymes, making foods harder to digest. Eating raw fruits and vegetables is a healthful way to improve overall digestion.

Food enzymes other than those used as food additives are not currently regulated or are regulated as processing aids under the legislation of the Member States. Differences between national laws concerning the assessment and authorisation of food enzymes may hinder their free movement within the internal market by distorting the rules of competition. This Regulation lays down the rules on food enzymes used in foods, including such enzymes used as processing aids. It does not cover food enzymes used in the production of food additives falling within the scope of **Regulation (EC) No 1333/2008,** or those used in the production of processing aids.

Community list of food enzymes

The creation of a list of authorised enzymes will make it possible to harmonise at European level all enzymes used as food additives. This list shall include all enzymes which perform a technological function in foods such as invertase (E 1103), lysozyme (E 1105), urease and betaglucanase. The establishment of the positive list of food enzymes will benefit the consumer because the list will lay down common rules for the assessment and authorisation of these products.

The list of enzymes should include:

- the name of the enzyme;
- Its specifications (including its origin, purity criteria, etc.);
- the foods to which it may be added;
- the conditions under which it may be used;
- restrictions on its sale;
- Specific labeling requirements.

Conditions for the inclusion of food enzymes in the Community list

An enzyme may be included in the Community list if and only if:

it does not pose a concern to the health of the consumer, in the concentration used and on the basis of the existing scientific information;

- its use is justified by a technological need;
- Its use does not mislead the consumer.

Plant enzymes are important because they are capable of digesting food before the body's own digestive process begins. In other words, plant enzymes can enhance the digestion of food and the delivery of nutrients to the blood even if you have a compromised digestive system. The same cannot be said of animal enzymes such as pancreatic. Everyone agrees that proper nutrition is crucial to the maintenance of a healthy body. However, most healthcare practitioners overlook the true cause of many nutritional disorders. It is assumed, quite mistakenly, that digestion occurs automatically and the correction of a nutritional disorder simply requires matching the right nutritional supplement to the condition. For example, vitamin C for colds, vitamin A for viruses and herbal laxatives for constipation. While this treatment may relieve patient symptoms, the relief is only temporary because the underlying problem of faulty digestion is ignored. Healthcare practitioners who want to effectively manage health problems that are related to nutritional imbalances must consider each person's ability to digest food. Unfortunately, most clinicians give little or no thought to the role of enzymes in digestion, despite overwhelming evidence of their importance.

Enzymes are present in all living animal and plant cells. They are the primary motivators of all natural biochemical processes. Life cannot exist without enzymes because they are essential components of every chemical reaction in the body. For example, they are the only substance that can digest food and make it small enough to pass through the gastrointestinal mucosa into the bloodstream. Three very broad classifications of enzymes are:

1. Food enzymes - occur in raw food and, when present in the diet, begin the process of digestion
2. Digestive enzymes - produced by the body to break food into particles small enough to be carried across the gut wall
3. Metabolic enzymes - produced by the body to perform various complex biochemical reactions.

4.5 SUMMARY

Destruction or removal of all viable organisms from an object or environment.killing, inhibition, or removal of pathogenic microorganisms (mainly pertains toinanimate objects)- prevention of microbial infection in living tissue reducing microbial populations to a safe level in accord with public healthstandards- a suffix indicating that the agent will kill the kind of organism in question a suffix indicating that the agent will prevent the growth of the type of organism inquestion (e.g., bacteriostatic) Microorganisms are not killed instantly when exposed to a lethalagent.Population death decreases by a constant fraction at constant intervals (exponential killing)Amicroorganism is considered dead when it is unable to grow in conditions that wouldnormally support its growth.

4.6 SELF ASSESSMENT QUESTIONS

1. What is the role of food enzymes?
2. Explain growth curve in detail?
3. Describe the Methods in controlling microorganisms?

4.7 REFERENCE BOOKS

1.Food toxicology part A.principles and concepts by jose M.concon marcelDekkher Jnc,NEW YORK,1988.

2. Journal of Medical Microbiology

3. Current science.

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Lesson – 5

MOULD AND MICOTOXIN CONTAMINATION

5.0 Objective

After reading this chapter, you should be able to:

- Know the Mould and micotoxin contamination through harmful effects
- To understand the classification of micotoxin contamination
- To create awareness in Mould and micotoxin detection and prevention

Structure

5.1 INTRODUCTION

A mycotoxin is a highly toxic by-product of mold growth in feed and grain. Myco means fungus and toxin means poison. They represent a broad spectrum of acute and chronic diseases in livestock. Mycotoxins can remain as a residue in meat and milk, posing a possible threat to human health. Mycotoxins are poisons generated from the secondary metabolic processes which occur naturally in a variety of molds. The amount and type of mycotoxin or secondary metabolite varies with environmental conditions such as temperature and humidity. Aspergillus, the mold which produces aflatoxins, grows in warm, dry conditions. Fusarium, which produces zearalenone and the "T" toxins, grows in cool, wet conditions. Fungi are major plant and insect pathogens, but they are not nearly as important as agents of disease in vertebrates, i.e., the number of medically important fungi is relatively low. Frank growth of fungi on animal hosts produces the diseases collectively called mycoses, while dietary, respiratory, dermal, and other exposures to toxic fungal metabolites produce the diseases collectively called mycotoxicoses. Mycotoxins are substances naturally produced by moulds and fungi that are normally present as some form of defence for the organism. All natural materials, and many man-made ones, are subject to contamination by moulds and fungi. Fungal spores are ubiquitous – which means that they are found everywhere without exception – and are unavoidable within the natural world. The growth of moulds and fungi is typically encouraged by heat and the

presence of moisture, however their toxin production is dictated by other factors, that induce stress including drought.

Toxins are naturally produced by all types of moulds and fungi. Hundreds of these mycotoxins exist, and, contamination of natural materials with multiple toxins, either from one or several fungi, is common. Mold is a term used to describe various fungi that grow in animal feed and degrade the quality of that feed. Mold growth is estimated to reduce feed value by at least 10%. Mold germinates at temperatures as low as 45°F, with most rapid growth at 75° to 90°F. They require humidity for germination, therefore, an upper limit of 14.5% moisture is recommended for stored corn or milo. Other grains should maintain moisture levels less than 13%.

Mycotoxins may be produced on bay, cereals, pastures, or fodder, or may be present in constituents used in the manufacture of meals or pelleted animal diets. Some animal diets, especially those containing grain or nuts, may contain several toxigenic species of mould, which may produce a number of mycotoxins having different toxic or pharmacological properties. Under these situations, clinical signs and lesions found In disease outbreaks may not conform to the usual descriptions of outbreaks, or to experimental findings in animals dosed with mycotoxins isolated from pure mould cultures.

5.2. MOULDS AND MICOTOXIN CONTAMINATION OF FOOD

Toxins may also assist with the organisms colonisation of the plant material. The use of toxins aids the competitiveness and success of the mould or fungi. The potential for fungal growth and toxin production can be limited by applying good management practices, but essentially it is impossible to guarantee that any naturally occurring materials will be free of mycotoxin contamination.

Host plant stress in the field: poor soil fertility, insect damage, high/low temperatures or moisture

Harvesting: late harvesting, dry crops, slow storage filling e.g. silage clamp, soil contamination.

Storage: wet grain, poor silage packing, incorrect fermentation of ensiled products

Finished feeds and forages: poor hygiene, exposure to air/moisture, incorrect storage (temperature/moisture)

Any growing crop, including forage and cereals, is susceptible to mould, with Fusarium types being the main concern. Fusarium moulds can produce mycotoxins on the growing plant. Whilst the moulds themselves may not survive the transition from field to feeding trough, the mycotoxins will remain intact, though invisible to the naked eye. Feeds may therefore appear and analyse as high quality, but may harbour a mycotoxin(s) challenge.

Signs of mould in feed:

Dustiness

Caking of feed

Poor feed flow out of bins

Feed refusal by animals for no apparent reason

Moldy, milde smell of feed grains

Darkening of feed and grain

What factors lead to mold growth and mycotoxin production in field crops?

Stress, such as water stress, high temperature stress, insect damage, and subsequent reduced vigor predispose crop plants to infestation, colonization and contamination by molds and mycotoxins. Effect of mycotoxin leads to destruction of nutrients and reduced palatability in human beings.Micotoxins can have a very wide range of effects on animals depending on quantitative and qualitative aspects of their production in animal feedstuffs. The diseases in animals caused by mycotoxins are referred to as mycotoxicoses..

Mycotoxins act within the animal to:

Modify nutrient quality, absorption and metabolism

Alter endocrine and neuroendocrine functions

Depress the immune response

While obvious clinical mycotoxicoses can result in lesions, abortion and even death, even moderate amounts frequently result in:

Lower feed consumption

Lower feed conversion

Reduced disease resistance

Increased reproductive problems.

Common Mycotoxins

Aflatoxin:

Aflatoxin has been the most heavily researched of the mycotoxins. Aflatoxin is produced from certain strains of the molds Aspergillus flavus and Aspergillus parasiticus. Aflatoxins are most abundant in hot, humid geographic areas. The effects in animals of ingesting excessive amounts of the toxin range from chronic health and performance problems to death. Aflatoxins' pricipal target within an animal is the liver. The damage to the liver results in abnormal blood clotting, development of jaundice, hemorrahaging, and reduction of immune response. Aflatoxin levels greater than 20 ppb constitute contaminated corn by the FDA. This is the maximum level that grain can contain when fed to dairy cattle. The aflatoxin level acceptable in milk is 0.5 ppb.

5.3 CLASSIFICATION OF MYCOTOXINS

Classified according to the main organ system, they affect.;

Hepatotoxins

- Sporidesmin
- Aflatoxins
- Luteoskyrin
- Cyclochlorotine
- Rubratoxins

Nephrotoxins
- Ochratoxin
- Citrinin

Neurotoxins
- Penitrema
- Patulin
- Citreoviridin
- Misc. neurotoxins

Cytotoxins (Alimentary Tract Toxins)
- Trichothecenes
- T-2 Toxin
- Diacetoxyscripenal
- Neosolaniol
- Nivalenol
- Diacetylnivalenol
- Deoxynivalenol (Don, Vomitoxin)
- HT2 toxin
- Fusarenon X

Estrogenic Mycotoxins
- F-2 Toxin (Zearalenone)

Other Mycotoxins
- Ergot
- Fescue
- Lupinosis

Discovered by:

Stevens et al. 1960: Turkey H disease outbreak Wannop, 1961; Abrams, 1965: duckling is more susceptible.Asao et al (1963): isolated the Chemical structure & referred to it as "Aflatoxin"Harley et al (1963): found FBl, FB2, FG1, and FG2 Aflatoxicosis increases the susceptibility of turkeys to candidasis. pasteurellosis and salmonellosis, and of chicks to coccidiosis and Marek's disease: Experimental work has revealed a significant interaction between aflatoxin and vitamin D with respect to bone calcification in chicks, this increasing their vitamin D requirements. (Bird, 1978). Vitamin A increases mortality rate in Aflatoxin chicks (Bryden et al., 1979) Affected:

Animals: Poultry ducks; turkey poults; pheasant chicks; chickens quail Mammals young pigs; pregnant sows; dogs; calves; cattle; sheep; cats; monkeys; man

Fish Lab. animals Cause(s): Some strains of Aspergillus flavus & most strains of A. parasiticus

Sign(s): a high mortality sudden onset inappetite dejected appearance

Farm animals:
- malabsorption of various nutrients
- coagulopathy
- less tissue integrity
- poor growth
- less FCR
- greater susceptibility to infection

- vaccine failures
- drug failures
- reproductive problems
- greater sensitivity to temperature extremes

Other animals:

Chicken:

depression, inappetence, lower growth rates, poor condition, bruising, lowered egg production, fertility, and hatchability, with high mortality. Also, ataxia, convulsion and opisthotonos are common signs.

less plasma testosterone

paralysis & lamenessTurkey poults & ducklings are particularly susceptible. In acute outbreaks, death occursafter only a short period of inappetence. Subacute symptoms are more usual.

- lower growth rate
- lower FCR
- depressed appetite
- interferes with absorption of food especially catotenoid
- with low level of aflatoxin:
- lower quantity of meat of carcass
- lower meat & bone ratio
- susceptability to body wands
- lower fat in liver
- destroyed protein synthesis in liver
- lowered resistance to diseases
- lower level of plasma amino acids

Fish

highly sensitive

1 ppb B1 - cancer in Rainbow trout(5)

Rat

highly sensitive p.(6)

1 ppb B1 - cancer in Fisher rat(5)

Severity ~ Biological Effect of Toxin (3)

Doskges:

Duration of Expose:

Species:

Sheep - highest resistance.

The interactions between 20 killer yeasts of various genera and species were examined. Ten distinct groups were recognized with respect to killer activity and 10 distinct groups with respect to resistance to killer action. Using both killing and resistance phenotypes, 13 classes of killer yeast were found. With the exception of Torulopsis glabrata NCYC 388, non-Saccharomyces strains of yeast were not killed by a member of the genus Saccharomyes. The killer character of the 3 killing groups of Saccharomyces identified could be cured by treatment with cycloheximide or incubation at elevated temperature and the effectiveness of these procedures was indicative of the category of killing. Killer-associated killer yeasts not belonging to the genus Saccharomyces could not be cured by these procedures. Double-stranded ribonucleic acids were extracted only from Saccharomyces kluyveri and the molecular weights of the species present were a function of the killer class to which a strain belonged. By an analysis of the effects of proteolytic enzymes,

temperature and pH on killer activity and by gel chromatography of crude preparations of killer factors, the toxins of different killer classes were shown to be biochemically distinct. However all toxins had certain properties in common consistent with there being a protein component essential to killer action.

A standardized LC-UV-MS micro-scale method for screening of fungal metabolites and mycotoxins in culture extracts is presented. The paper includes data for detection and dereplication of > 400 fungal metabolites to facilitate detection and identification when standards are not available. The data also shows the types of components that can be analysed by positive electrospray (ESI+) mass spectrometry (MS) along with common fragments and adducts of these, as well as giving suggestions on whether UV or ESI+-MS methods should be used. Examples of dereplication of penitrems and macro-cyclic ichothecenes, and detection of several novel compounds are shown. This was done by UV spectroscopy combined with accurate mass determination of adduct and fragment ions obtained by high-resolution orthogonal time-of-flight MS.

spergillus flavus is a common filamentous fungus that produces aflatoxins and presents a major threat to agriculture and human health. Previous phylogenetic studies of A. flavus have shown that it consists of two subgroups, called groups I and II, and morphological studies indicated that it consists of two morphological groups based on sclerotium size, called "S" and "L." The industrially important non-aflatoxin-producing fungus A. oryzae is nested within group I. Three different gene regions, including part of a gene involved in aflatoxin biosynthesis (omt12), were sequenced in 33 S and L strains of A. flavus collected from various regions around the world, along with three isolates of A. oryzae and two isolates of A. parasiticus that were used as outgroups. The production of B and G aflatoxins and cyclopiazonic acid was analyzed in the A. flavus isolates, and each isolate was identified as "S" or "L" based on sclerotium size. Phylogenetic analysis of all three genes confirmed the inference that group I and group II represent a deep divergence within A. flavus. Most group I strains produced B aflatoxins to some degree, and none produced G aflatoxins. Four of six group II strains produced both B and G aflatoxins. All group II isolates were of the "S" sclerotium phenotype, whereas group I strains consisted of both "S" and "L" isolates. Based on the omt12 gene region, phylogenetic structure in sclerotium phenotype and aflatoxin production was evident within group I. Some non-aflatoxin-producing isolates of group I had an omt12 allele that was identical to that found in isolates of A. oryzae.

Epipolythiodioxopiperazines (ETPs) are toxic secondary metabolites made only by fungi. The best-known ETP is gliotoxin, which appears to be a virulence factor associated with invasive aspergillosis of immunocompromised patients. The toxicity of ETPs is due to the presence of a disulphide bridge, which can inactivate proteins via reaction with thiol groups, and to the generation of reactive oxygen species by redox cycling. With the availability of complete fungal genome sequences and efficient gene-disruption techniques for fungi, approaches are now feasible to delineate biosynthetic pathways for ETPs and to gain insights into the evolution of such gene clusters.

Mycotoxins are low-molecular-weight secondary metabolites of fungi. The most significant mycotoxins are contaminants of agricultural commodities, foods and feeds. Fungi that produce these toxins do so both prior to harvest and during storage. Although contamination of commodities by toxigenic fungi occurs frequently in areas with a hot and humid climate (i.e. conditions favorable for fungal growth), they can also be found in temperate conditions. Production of mycotoxins is dependent upon the type of producing fungus and environmental conditions such as the substrate, water activity (moisture and relative humidity), duration of exposure

to stress conditions and microbial, insect or other animal interactions. Although outbreaks of mycotoxicoses in humans have been documented, several of these have not been well characterized, neither has a direct correlation between the mycotoxin and resulting toxic effect been well established in vivo. Even though the specific modes of action of most of the toxins are not well established, acute and chronic effects in prokaryotic and eukaryotic systems, including humans have been reported. The toxicity of the mycotoxins varies considerably with the toxin, the animal species exposed to it, and the extent of exposure, age and nutritional status. Most of the toxic effects of mycotoxins are limited to specific organs, but several mycotoxins affect many organs. Induction of cancer by some mycotoxins is a major concern as a chronic effect of these toxins. It is nearly impossible to eliminate mycotoxins from the foods and feed in spite of the regulatory efforts at the national and international levels to remove the contaminated commodities. This is because mycotoxins are highly stable compounds, the producing fungi are ubiquitous, and food contamination can occur both before and after harvest. Nevertheless, good farm management practices and adequate storage facilities minimize the toxin contamination problems. Current research is designed to develop natural biocontrol competitive fungi and to enhance host resistance against fungal growth or toxin production. These efforts could prevent toxin formation entirely. Rigorous programs for reducing the risk of human and animal exposure to contaminated foods and feed also include economically feasible and safe detoxification processes and dietary modifications. Although risk assessment has been made for some mycotoxins, additional, systematic epidemological data for human exposure is needed for establishing toxicological parameters for mycotoxins and the safe dose for humans. It is unreasonable to expect complete elimination of the mycotoxin problem. But multiple approaches will be needed to minimize the economic impact of the toxins on the entire agriculture industry and their harmfulness to human and animal health. Ancestral beta gamma-crystallin precursor structure in a yeast killer toxin. Implications of apoptosis for toxicity, carcinogenicity, and risk assessment: fumonisin B(1) as an example.

Thirty-one isolates of Stachybotrys chartarum from indoor and outdoor environments were analyzed for the presence of the trichodiene synthase (Tri5) gene, trichothecenes, boar sperm cell motility inhibition, and randomly amplified polymorphic DNA banding patterns (RAPDs). Twenty-two S. chartarum isolates tested positive for the Tri5 gene and nine were negative when tested using novel Tri5 gene-specific PCR primer pair. The Tri5 gene positive isolates contained satratoxins (five isolates) or the simple trichothecene, trichodermol (11 isolates). The Tri5 gene negative isolates did not produce satratoxins or trichodermol. Nineteen S. chartarum isolates, distributed among the Tri5 gene negative and positive groups, inhibited boar spermatozoan motility at concentrations of < or = 60 microg of crude cell extract/mL. The inhibition of motility was independent of satratoxins or atranones. Unweighted pair group method of arithmetic averages (UPGMA) cluster analysis of RAPD fragments clustered the 31 S. chartarum isolates in two distinct groups designated as RAPD groups 1 and 2. The grouping of S. chartarum isolates obtained by UPGMA cluster analysis of RAPD fragments was identical to the grouping obtained by Tri5 gene-specific PCR. This indicates that the S. chartarum isolates belonging to different groups were genetically distinct in a much wider area than just the Tri5 gene.

Mycotoxins are chemical compounds, produced by a variety of fungi, that can cause illness in humans and animals. This paper is a review of literature on mycotoxins with emphasis on mycotoxins in indoor air. Consideration is given to specific mycotoxins identified in indoor air, indoor sources of the mycotoxins, factors affecting mycotoxin production, potential health effects indicated by animal laboratory studies, and case studies of possible human inhalation health effects of these mycotoxins. Historically, mycotoxicoses have been associated with

consumption of moldy grain. In recent years, some attention has been given to mycotoxins in dust from agricultural environments, but relatively few studies have examined mycotoxins or mycotoxin-producing molds in indoor environments. The few indoor studies suggest that mycotoxicoses may occur in some indoor environments. More studies are needed to understand.

5.4 METHOD AND DETECTION:

The large and diverse group of microscopic food borne yeasts and molds (fungi) includes several hundred species. The ability of these organisms to attack many foods is due in large part to their relatively versatile environmental requirements. Although the majority of yeasts and molds are obligate aerobes (require free oxygen for growth), their acid/alkaline requirement for growth is quite broad, ranging from pH 2 to above pH 9. Their temperature range (10-35°C) is also broad, with a few species capable of growth below or above this range. Moisture requirements of foodborne molds are relatively low; most species can grow at a water activity (a_w) of 0.85 or less, although yeasts generally require a higher water activity.

Both yeasts and molds cause various degrees of deterioration and decomposition of foods. They can invade and grow on virtually any type of food at any time; they invade crops such as grains, nuts, beans, and fruits in fields before harvesting and during storage. They also grow on processed foods and food mixtures. Their detectability in or on foods depends on food type, organisms involved, and degree of invasion; the contaminated food may be slightly blemished, severely blemished, or completely decomposed, with the actual growth manifested by rot spots of various sizes and colors, unsightly scabs, slime, white cottony mycelium, or highly colored sporulating mold. Abnormal flavors and odors may also be produced. Occasionally, a food appears mold-free but is found upon mycological examination to be contaminated. Contamination of foods by yeasts and molds can result in substantial economic losses to producer, processor, and consumer.

Several foodborne molds, and possibly yeasts, may also be hazardous to human or animal health because of their ability to produce toxic metabolites known as mycotoxins. Most mycotoxins are stable compounds that are not destroyed during food processing or home cooking. Even though the generating organisms may not survive food preparation, the preformed toxin may still be present. Certain foodborne molds and yeasts may also elicit allergic reactions or may cause infections. Although most foodborne fungi are not infectious, some species can cause infection, especially in immunocompromised populations, such as the aged and debilitated, HIV-infected individuals, and persons receiving chemotherapy or antibiotic treatment.

The dilution plating and the direct plating methods may be used to detect fungi in foods. The direct plating method is more efficient than the dilution plating method for detecting individual mold species, including most of the toxin producers, but it is less effective in detecting yeasts. It is also used to determine whether the presence of mold is due to external contamination or internal invasion. Methodology for testing the ability of isolates of toxigenic mold species to produce mycotoxins on sterile rice water substrate is included here.

5.5 PREVENTION:

Prevention of mycotoxin formation is essential since there are few ways to completely overcome problems once mycotoxins are present. Ammoniation of grains can destroy some

mycotoxins, but there is no practical method to detoxify affected forages. Prevention of mycotoxins in silage includes following accepted silage making practices aimed at preventing deterioration, primarily by quickly reducing pH and the elimination of oxygen. Some additives are beneficial in reducing mold growth and therefore mycotoxin formation. Ammonia, propionic acid, sorbic acid and microbial or enzymatic silage additives are shown to be at least partially effective at inhibiting mold growth. Silo size should be matched to herd size to insure daily removal of silage at a rate faster than deterioration. Feed bunks should be cleaned regularly. Care should be taken to ensure that high moisture grains are stored at proper moisture contents and in a well maintained structure. Grains or other dry feed, such as hay, should be stored at a low moisture content (<14%) below which molds do not readily grow, and then protected to remain dry. Aeration of grain bins is important to reduce moisture migration and to keep the feedstuffs in a good condition. Obviously moldy feed should be avoided, if possible. If unacceptaObviously moldy feed should be avoided, if possible. If unacceptably high levels of mycotoxins occur, dilution or removal of the contaminated feed is prefer- able; however, it is often impossible to completely replace some feeds in the ration, particularly the forage ingredients. Increasing dietary levels of nutrients such as protein, energy and antioxidants may be advisable. Animals exposed to aflatoxin show marginal responses to increased protein. In some situa- tions, poultry respond to water soluble vitamins or to specific minerals. Acidic diets seem to exacerbate effects of mycotoxins, and therefore adequate dietary fiber and buffers are recommended. Favorable results have been seen when absorbent materials such as clays (bentonites), complex indigestible carbohydrates such as glucoman- nans or mannanoligiosaccharides, and other similar products are added to mycotoxin contaminated diets of rats, poultry, swine and cattle.

5.5 SUMMARY

Mycotoxins are prevalent in feedstuffs. Many different mycotoxins exist. They affect dairy cattle in many ways; the most important is perhaps immunosup- pression. Diagnosis of a mycotoxicosis is difficult and indirect, but mycotoxins should be considered as a potential cause of increased disease and loss of production. While mycotoxins can cause acute toxicity, they are more likely to cause chronic problems of increased disease and decreased milk production. Contamination of milk by aflatoxin can cause huge economic losses. Management of crops and feeds is important to reduce mycotoxin contamination. Certain feed additives are proved to be helpful in treatment.

5.7. SELF ASSESSMENT QUESTIONS

1. Explain the classification of Moulds and micotoxins in detail?
2. Describe the methods and prevention of Mould and micotoxins?
3. Write short notes on effect of moulds and micotoxins?

5.7 REFERENCE BOOKS

1.Food toxicology part A.principles and concepts by jose M.concon marcelDekkher Inc,NEW YORK,1988.

2. Journal of Medical Microbiology

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LESSON-6

TOXICOLOGY

6.0 Objective

After reading this chapter, you should be able to:

- Know the Mould and micotoxin contamination through harmful effects
- To understand the classification of micotoxin contamination
- To create awareness in Mould and micotoxin detection and prevention.

Structure

6.1 INTRODUCTION:

Toxicology is the study of the adverse effects of chemicals on living organisms. It is the study of symptoms, mechanisms, treatments and detection of poisoning, especially the poisoning of people. The relationship between dose and its effects on the exposed organism is of high significance in toxicology. The chief criterion regarding the toxicity of a chemical is the dose, i.e. the amount of exposure to the substance.

All substances are toxic under the right conditions. The term LD_{50} refers to the dose of a toxic substance that kills 50 percent of a test population (typically rats or other surrogates when the test concerns human toxicity).For example, toxic genomics involves applying molecular profiling approaches to the study of toxicology. Other areas include Aquatic toxicology, Chemical toxicology, Ecotoxicology, Environmental toxicology, Forensic toxicology, and Medical toxicology.

Chemical toxicology is a scientific discipline involving the study of structure and mechanism related to the toxic effects of chemical agents, and encompasses technology advances in research related to chemical aspects of toxicology. Toxicity is the degree to which something is able to produce illness or damage to an exposed organism. Toxicity can refer to the effect on a whole organism, such as a human or a bacterium or a plant, or to a substructure, such as a cell (cytotoxicity) or an organ (organotoxicity such as the liver (hepatotoxicity). By extension, the word may be metaphorically used to describe toxic effects on larger and more complex groups, such as the family unit or "society at large".

While the great importance of plants as a major source of human food is recognized, the inherent limitations associated with plant-derived food stuffs, as a result of the occurrence of various

antinutritional factors and toxicants, is reviewed. The quality and safety of food could be threatened by a host of factors, including a diverse range of natural compounds capable of precipitating deleterious effects in animals. The toxicants include, trypsin (protease) inhibitors, tannins and polyphenols, phytohemagglutinins (lectins), oxalate, phytic acid, cyanogenic glycosides, goitrogens, nitrates/nitrites, saponins, alkaloids and mycotoxins. The toxicological implications of these factors were highlighted. Mycotoxins from fungi that contaminate staple food grains are a danger to humans and animals alike and their economic importance in international trade is considerable.

Most companies will not submit an application, or will withdraw a submission, when they see that it will not meet the strict criteria outlined by Health Canada. For example, several years ago, research was conducted in order to improve the quality of soybean meal as an animal feed. This involved the transfer of genetic material coding for a storage protein from a Brazil nut to a soybean.

Since the Brazil nut is known to cause allergic reaction in a small number of sensitive individuals, laboratory tests using sera from Brazil nut-sensitive individuals were conducted in order to determine whether an allergenic protein had been transferred to the soybean. The results of the laboratory tests showed that the gene obtained from the Brazil nut likely encoded the major Brazil nut allergen and research on this product was discontinued.

Many plant foods contain natural or processing-induced constituents with biological activity in mammals. Among these are glycoalkaloids, amines, glucosinolates, cyanogenic glycosides, protease inhibitors, oxalates, coumarins, polyphenols, cyclopropenoid fatty acids, phytates, xanthines and essential oils. Recently, various lines of evidence have suggested that plant-derived constituents may play an important role in determining spontaneous rates of genetic damage and tumor incidence. Dietary antioxidants such as β-carotene, vitamins A and C, tocopherols, and glutathione have been suggested to protect against endogenous oxygen-radical damage. On the other hand, genotoxic constituents such as certain flavones, anthraquinones, browning products, benzoxazinones, and acetals have been identified in plant-derived foods, and unsaturated oilseed lipids have been found to increase cancers of certain sites, particularly intestine and breast, in laboratory animals. Whether these constituents play a significant quantitative role in human health is a key scientific issue which is at present not adequately resolved.

In the science of toxicology, toxicity is the degree of impact of an external substance or condition and its deleterious effects on living things: organisms, organ systems, individual organs, tissues, cells, sub cellular units is the subject of study. A central concept of toxicology is that effects are dose-dependent; even water – generally not considered to be toxic – can lead to water intoxication when taken in large enough doses, whereas for even a very toxic substance such as snake venom there is a dose below which there is no detectable toxic effect. Toxicity is the ability of a chemical or physical agent to induce detrimental temporary or permanent tissue change or to detrimentally interfere with normal biochemical processing.

6.2 CLASSIFICATION OF FOOD TOXICANTS

There are generally three types of toxic entities; chemical, biological, and physical.Chemicals include inorganic substancessuchas lead, hydrofluoricacid,and chlorine gas, organic compounds such as methyl alcohol, most medications, and poisons from living things.

Biological toxic entities include those bacteria and viruses that are able to induce disease in living organisms. Biological toxicity can be complicated to measure because the "threshold dose" may be a single organism. Theoretically one virus, bacterium or worm can reproduce to cause a serious infection. However, in a host with an intact immune system the inherent toxicity of the organism is balanced by the host's ability to fight back; the effective toxicity is then a combination of both parts of the relationship. A similar situation is also present with other types of toxic agents.

Physically toxic entities include things not usually thought of under the heading of "toxic" by many people: direct blows, concussion, sound and vibration, heat and cold, non-ionizingelectromagnetic radiation such as inirared and visible light, andionizing radiation such as X-rays and alpha, beta, and gammaradiation. Toxicity can be measured by the effects on the target (organism, organ, tissue or cell). Because individuals typically have different levels of response to the same dose of a toxin, a population-level measure of toxicity is often used which relates the probability of an outcome for a given individual in a population. One such measure is the LD 50. When such data does not exist, estimates are made by comparison to known similar toxic things, or to similar exposures in similar organisms. Then "safety factors" are added to account for uncertainties in data and evaluation processes. For example, if a dose of toxin is safe for a laboratory rat, one might assume that one tenth that dose would be safe for a human, allowing a safety factor of 10 to allow for interspecies differences between two mammals; if the data are from fish, one might use a factor of 100 to account for the greater difference between two chordate classes (fish and mammals). Similarly, an extra protection factor may be used for individuals believed to be more susceptible to toxic effects such as in pregnancy or with certain diseases. Or, a newly synthesized and previously unstudied chemical that is believed to be very similar in effect to another compound could be assigned an additional protection factor of 10 to account for possible differences in effects that are probably much smaller. Obviously, this approach is very approximate; but such protection factors are deliberately very conservative and the method has been found to be useful in a wide variety of applications.

Assessing all aspects of the toxicity of cancer-causing agents involves additional issues, since it is not certain if there is a minimal effective dose for carcinogens, or whether the risk. If a dose of toxin is safe for a laboratory rat, one might assume that one tenth that dose would be safe for a human, allowing a safety factor of 10 to allow for interspecies differences between two mammals; if the data are from fish, one might use a factor of 100 to account for the greater difference between two chordate classes (fish and mammals). Similarly, an extra protection factor may be used for individuals believed to be more susceptible to toxic effects such as in pregnancy or with certain diseases. Or, a newly synthesized and previously unstudied chemical that is believed to be very similar in effect to another compound could be assigned an additional protection factor of 10 to account for possible differences in effects that are probably much smaller. Obviously, this approach is very approximate; but such protection factors are deliberately very conservative and the method has been found to be useful in a wide variety of applications.

Assessing all aspects of the tcxicity of cancer-causing agents involves additional issues, since it is not certain if there is a minimal effective dose for carcinogens, or whether the risk is just too small to see. In addition, it is possible that a single cell transformed into a cancer cell is all it takes to develop the full effect (the "one hit" theory). It is more difficult to assess the toxicity of chemical mixtures than of single, pure chemicals because each component display its own toxicity and components may interact to produce enhanced or diminished effects. Common mixtures include gasoline, cigarette smoke, and industrial waste. Even more complex are situations with

more than one type of toxic entity, such as the discharge from a malfunctioning sewage treatment plant, with both chemical and biological agents.

Toxicity of a substance can be affected by many different factors, such as the pathway of administration (whether the toxin is applied to the skin, ingested, inhaled, injected), the time of exposure (a brief encounter or long term), the number of exposures (a single dose or multiple doses over time), the physical form of the toxin (solid, liquid, gas), the genetic makeup of an individual, an individual's overall health, and many others. Several of the terms used to describe these factors have been included here. ;chronic exposure: continuous exposure to a toxin over an extended period of time, often measured in months or years can cause irreversible side effects.

6.2 CLASSIFICATION OF FOOD TOXICANTS

A. Heavy Metals
B. Solvents and Vapors
C. Radiation and Radioactive Materials
D. Dioxin/Furans
E. Pesticides
F. Microbial toxins
G. Mushroom toxins
H. Plant Toxins
I. Animal Toxins
J. Subcategories of Toxic Substances

Heavy Metals

Metals differ from other toxic substances in that they are neither created nor destroyed by humans. Their use by humans plays an important role in determining their potential for health effects. Their effect on health could occur through at least two mechanisms: first, by increasing the presence of heavy metals in air, water, soil, and food, and second, by changing the structure of the chemical. For example, chromium III can be converted to or from chromium VI, the more toxic form of the metal.

Solvents and Vapors

Nearly everyone is exposed to solvents. Occupational exposures can range from the use of "white-out" by administrative personnel, to the use of chemicals by technicians in a nail salon. When a solvent evaporates, the vapors may also pose a threat to the exposed population.

Radiation

Radiation is the release and propagation of energy in space or through a material medium in the form of waves, the transfer of heat or light by waves of energy, or the stream of particles from a nuclear reactor.

Dioxin/Furans

Dioxin, (or TCDD) was originally discovered as a contaminant in the herbicide Agent Orange. Dioxin is also a by-product of chlorine processing in paper producing industries.

Pesticides

The EPA defines pesticide as any substance or mixture of substances intended to prevent, destroy, repel, or mitigate any pest. Pesticides may also be described as any physical, chemical, or biological agent that will kill an undesirable plant or animal pest.

Plant Toxins

Different portions of a plant may contain different concentrations of chemicals. Some chemicals made by plants can be lethal. For example, taxon, used in chemotherapy to kill cancer cells, is produced by a species of the yew plant.

Animal Toxins

These toxins can result from venomous or poisonous animal releases. Venomous animals are usually defined as those that are capable of producing a poison in a highly developed gland or group of cells, and can deliver that toxin through biting or stinging. Poisonous animals are generally regarded as those whose tissues, either in part or in their whole, are toxic.

All of these substances may also be further classified according to their: Effect on target organs (liver, kidney, hematopoietic system), Use (pesticide, solvent, food additive), Source of the agent (animal and plant toxins), Effects (cancer mutation, liver injury), Physical state (gas, dust, liquid Labeling requirements (explosive, flammable, oxidizer), Chemistry (aromatic amine, halogenated hydrocarbon), orPoisoning potential (extremely toxic, very toxic, slightly toxic).

There are several different types of food poisoning in the UK, all with slightly different symptoms - and levels of severity. Some can start soon after the food involved was eaten. Others take several days to take effect. While some forms of the illness clear up after a couple of days, others can linger for weeks and be carried in the body for months. Here is a guide to the five most common types of food poisoning in the UK, where they are most commonly caught and how to spot them from their symptoms.

Campylobacter This is the most common food poisoning bug in Britain and is nicknamed the 'barbecue bug'.

Mostly undercooked poultry and burgers and shellfish. Symptoms: Headaches, dizziness and fever followed by severe stomach pains and diarrhoea, which may be bloody. Vomiting is rare. Occasionally pains are so severe that sufferers are misdiagnosed as having appendicitis. How long before symptoms occur?: Anything from one to ten days. The illness can last from a day to more than a week. Full recovery can take several weeks.

Salmonella: Raw eggs, raw meat and poultry. Symptoms: Fever, vomiting and stomach pains. In severe cases septicaemia or peritonitis - an acute inflammation of the membrane that lines the abdomen. How long before symptoms occur?: Usually between 12 and 48 hours, but up to four days. You could be ill for up to three weeks and carry the bug for another three months.

E coli : Raw and undercooked meats, unpasteurised milk and dairy products, unpasteurised apple juice and raw vegetables. Symptoms: Diarrhoea, which may be bloody, stomach pains and sometimes vomiting. E coli 0157 produces a powerful poison called verocytotoxin which can cause kidney failure and death. How long before symptoms occur?: Between 12 hours and three days.

The length of time the illness lasts depends from person to person, but young children and the elderly are most likely to be badly affected.

Listeria : Cheeses, especially soft ripe cheeses such as Brie and Camembert, poultry, meat, pate and salads. Symptoms: Range from flu-like symptoms to meningitis and septicaemia. This bug is particularly dangerous for pregnant women as it can cause miscarriage or be passed on to the unborn baby. How long before symptoms occur?: From three to 70 days. Duration varies.

Viral food poisoning - (SRVSε) : Sources: Originates in humans, but is spread through uncooked foods or foods handled after cooking. Shellfish - particularly raw oysters, cockles and mussels - are also a common carrier of the virus as they may have been contaminated with human sewage in the sea. Symptoms: Nausea and projectile vomiting, diarrhoea. How long before symptoms occur?: Usually between 12 and 48 hours. Symptoms usually last two days.

Inherent plant toxicants are among the plant metaboliteswhich are claimed to have an ecological role in thephysiology, proliferation or defense of plants. Althoughsome constituents seem to be designed to deter feedingby mammals, which are thus toxic or otherwise unpleasantto humans, several constituents with possiblyother purposes (plant physiological, defense againstinsects, bacteria, fungi and viruses) may also just happento be toxic to humans (Harborne, 1988). Of mostsubstances, however, their precise function for planthealth and proliferation is not known. This makesmodifying their levels in plants in the pursuit of minimising risks of food poisoning a delicate matter.Some of these constituents are allellopatics orphytoalexin.

6.3 FACTORS AFFECTING IN FOOD TOXICITY

Dose is the primary concern; however, the exact intake of toxicant is seldom known. Duration and frequency of exposure are important. The route of exposure affects absorption, translocation, and perhaps metabolic pathways. Exposure of a toxicant relative to periods of stress or food intake may also be a factor. Following ingestion of some toxicants, emesis may occur if the stomach is empty, but if partly filled, the toxicant is retained and toxicosis can occur. Environmental factors, such as temperature, humidity, and barometric pressure, affect rates of consumption and even the occurrence of some toxic agents. Many mycotoxins and poisonous plants are correlated with seasonal or climatic changes. For example, the ischemic effects of ergot toxicosis are more often observed during the winter cold, and plant nitrate levels are affected by rainfall amounts.

Biologic Factors

Various species and strains within species react differently to a particular toxicant because of variations in absorption, metabolism, or elimination. Functional differences in species may also affect the likelihood of toxicosis, eg, species unable to vomit can be intoxicated with a lower dose of some agents.

Age and size of the animal are primary factors in toxicosis. Metabolism and translocation of xenobiotic agents are compromised by the underdeveloped microsomal enzyme system in young animals. Membrane permeability and hepatic and renal clearance capabilities vary with age, species, and health. The amount of toxicant required to cause pathology is generally correlated to body weight, but with greater body weight, a disproportionate increase in toxicity (per unit body weight) of a compound often occurs. Body surface area may correlate more closely with the toxic dose. No measurement parameter is consistent for every situation.

Nutritional and dietary factors, hormonal and health status, organ pathology, stress, and sex all affect toxicosis. Nutritional factors may directly affect the toxicant (ie, by altering absorption) or indirectly affect the metabolic processes or availability of receptor sites. The copper-molybdenum-sulfate interaction is an example of both.

Chemical Factors

The chemical nature of a toxicant determines solubility, which in turn influences absorption. Nonpolar or lipid-soluble substances tend to be more readily absorbed than polar or ionized substances. The vehicle or carrier of the toxic compound also affects its availability for absorption. Isomers, including optical isomers, vary in toxicity. For example, the ã isomer of hexachlorocyclohexane (lindane) is more toxic than other isomers.

Adjuvants are formulation factors used to alter the toxicologic effect of the active ingredient (eg, piperonyl butoxide enhances the insecticidal activity of pyrethrins). Binding agents, enteric coating, and sustained-release preparations influence absorption of the active ingredient. As absorption is delayed, toxicity decreases. Flavoring agents affect palatability, and thus the amount ingested.

6.4 FOOD TOXICITY AND DISEASES

Disease-Causing Organism & IncubationPeriod Main Sources of Infection (Food UsuallyContaminated) SymptomsSalmonellosis

Salmonella species 6 to 72 hours (usually 18 to 36 hours)Raw or undercooked poultry, meat, eggs,prepared food; meat contaminated by feces;food handlers with poor hygiene. Contactwith contaminated food (raw chicken, meat)can spread salmonella to other items (cookedor ready to eat dishes, salad) via countertops,cutting boards, utensils,hands.Survivesinadequate cooking and may grow andmultiply in cooked meat, poultry, stuffing,gravy and fish.Diarrhea, abdominal cramps, mild fever,nausea, most severe in the very young andelderly. Can be fatal in infants, the elderly andpeople with depressed immune systems. Mostsevere if acquired from fatty food (e.g., cheese,hamburger, salami, hot dogs, chocolate).

Staphylococcal Food Poisoning

Staphylococus aureus

1 to 8 hours (usually within 2 to 6 hours)Found in nose, throat, on skin, fingertips of30-50% of healthy people. Spread by foodhandlers, coughing, sneezing and otherunsanitary practices; also via dirty skin,pimples. Grows best on protein-rich food;meat, poultry, fish, milk, cheese, custards andmuch-handled food such as sandwiches, pasta,potato salad.Vomiting, nausea, abdominal cramps, diarrhea,chills, possibly weak pulse and shallowbreathing. Usually uneventful recovery 24-48hours.

CampylobacteriosisCampylobacter jejuni

2 to 7 days (usually 3 to 5 days)Found in gastrointestinal tracts of wild anddomestic animals. C. jejuni contaminates rawmeats and poultry during processing throughcontact with feces. Other sources ofcontamination: raw milk, untreated water,clams, undercooked beef, chicken.Fever, diarrhea, abdominal cramps, andpossibly bloody stool.

Bacteria and viruses commonly cause people to get sick from tainted food. The most commonly recognized food-borne infections are those caused by the bacteria campylobacter, salmonella and e. coli O157:H7 and by a group of viruses called calicivirus, also known as the Norwalk and Norwalk-like viruses, according to the Centers for Disease Control and Prevention.

If you or a loved one has been injured because of food poisoning, you deserve to have a strong legal advocate on your side. Contact Metzger Wickersham for a free and confidential consultation. You may be entitled to compensation for lost wages, medical expenses, pain and suffering, among other possible damages.

Campylobacter: A bacterial pathogen that causes fever, diarrhea and abdominal cramps. This is the most commonly identified bacterial cause of diarrheal illness in the world. These bacterial live in the intestines of health birds. Most raw poultry meat has campylobacter on it. Eating undercooked chicken or other food that has been contaminated with juices dripping from raw chicken is the most frequent source of this infection.

Salmonella: A bacterium that is widespread in the intestines of birds, reptiles and mammals. It can spread to humans through a variety of different foods of animal origin. The illness it causes, salmonellosis, typically includes fever, diarrhea and abdominal cramps. In people with poor underlying health or weakened immune systems, it can invade the bloodstream and cause life-threatening infections.

E. coli 0157:H7: A bacterial pathogen that has a reservoir in cattle and other similar animals. Human illness typically follows consumption of food or water that has been contaminated with microscopic amounts of cow feces. The illness it causes is often a severe and bloody diarrhea and painful abdominal cramps, without much fever. In 3-5 percent of cases, a complication called hemolytic uremic syndrome (HUS) can occur several weeks after the initial symptoms. This severe complication includes temporary anemia, profuse bleeding, and kidney failure.

6.5 SUMMARY

Food Poisoning and Food Toxicology is harmful to Human health. Pesticides, Microbial toxins, Mushroom toxins, Plant Toxins, Animal Toxins and Subcategories of Toxic Substances. Salmonella species, Staphylococus aureus, and E. col can cause diarrhea, coma and convelsions and death.

6.6 SELF ASSESSMENT QUESTIONS

1.Explain Food toxicity importance in detail?

2.Describe the classification of Food Toxicity?

3. Explain the various reasons for Food Toxicology?

6.7 REFERENCE BOOKS

1.Food toxicology part A [illegible] Dekkher Inc,NEW YORK,1988.

2. Journal of [illegible]

3. Current [illegible]

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Lesson – 7

FOOD TOXINS

7.0 Objective

After reading this chapter, you should be able to:

- Know the effect of Food Toxins on Human Health
- To create awareness regarding toxicity and harmful side effects in human beings.
- To educate the community through nutrition Knowledge.

STRUCTURE

7.1 INTRODUCTION

It would be nice if plants were made just to be our food. But what they have in mind is, in the first place, their own survival. Part of it is that they often contain what could be broadly called "natural pesticides": substances that are toxic to mold, insects and, sometimes, animals. Wild plants include foods that we eat almost daily: carrots (contain *carota toxin* and *myristicin*, nerve poison and hallucinogen, respectively), parsley, parsnip and celery (contain *psolarens*, increasing skin photosensitivity and vulnerability to cancer), black pepper and nutmeg (carcinogen*saffrol*), some herbs, like comfrey (contains *pyrrolizidine alkaloids*, inflicting liver damage), and many others.

Naturally occurring plant toxins, just as manmade pesticides usually disrupt metabolic processes by blocking certain enzymes. Possible effects range from hallucinogenic to degenerative and mutagenic. Fortunately, most of natural food pesticides are intended for much smaller predators, and don't pack enough of a punch to harm humans. However, prolonged use of little known herbal preparations and teas, carries the risk of harming health. Three major groups of natural food toxins are alkaloids,bioactive,and fungal toxins. Worth mentioning are also purines, salicylates, pyrrolizidine alkaloids and carrageenan.

Unfortunately, we humans are not spared from their toxicity. And plants from nightshade family are rather abundant in our diet. They include potato,tomato,pepper,eggplant,cayenne,chilli and paprika. Another well-known member of nightshade family is tobacco. One of the mechanism through which nightshade *glycol alkaloids* can affect health by interfering with the *acetyl cholinesterase* enzyme, a vital part of the neuromuscular function. You may get the picture of how important this enzyme is from the fact that some snake venoms, as well as some nerve gases (sarin, VX) work by blocking its function.

Nightshade glycol alkaloids are also capable of damaging cell membranes, from endoplasmic reticulum, needed for cellular detoxification and protein synthesis, to sodium and calcium channels, crucial for proper cardiovascular function. But all this may and may not affect you. The mechanism of action of these glycoalkaloids is determined by their level in the body, and individual mode and level of sensitivity. The rate of body accumulation from their very low food level also varies individually, which only makes more unpredictable when and how they will affect you - if ever Pesticide-like action of bio-active amines (chemical compounds also belonging to the broader group of alkaloids) is based on their chemical structure resembling that of some of our hormones, such as adrenaline. Bio-active amines affect either blood vessels (vasoactive amines) - causing changes in blood pressure and related symptoms like migraine headaches - or the nervous system, by affecting the level and function of neurotransmitters (psychoactive amines).

Pore-forming protein toxins (PFTs) are one of Nature's most potent biological weapons. An essential feature of their toxicity is the remarkable property that PFTs can exist either in a stable water-soluble state or as an integral membrane pore. In order to convert from the water-soluble to the membrane state, the toxin must undergo large conformational changes. There are now more than a dozen PFTs for which crystal structures have been determined and the nature of the conformational changes they must undergo is beginning to be understood. Although they differ markedly in their primary, secondary, tertiary and quaternary structures, nearly all can be classified into one of two families based on the types of pores they are thought to form: alpha-PFTs or beta-PFTs. Recent work suggests a number of common features in the mechanism Pore-forming toxins (PFT) are proteins able to produce well-structured holes in target cell membrane. They have a very broad taxonomic distribution being produced from bacteria to mammals. Depending on the secondary structure of the membrane-spanning region, these proteins are categorised into two classes: á-PFT and â-PFT. The pore structure of representative members of each class will be described. These proteins can be also classified according to their pore structure: barrel-stave andtoroidal protein–lipid pore. In the barrel-stave pore the protein molecules provide a continuous interface between the core of the bilayer and the channel lumen, whereas in the toroidal protein–lipid pore both polypeptide chains and polar phospholipid headgroups are involved in the building of pore walls. The stoichiometry and the pore diameter depend on the protein, thus the channels can allow leakage of ions, adenosine triphosphate (ATP), proteins and even bacteria. Attacked cells trigger different responses, some promoting recovery of membrane integrity, others

transition to a low-energy-consumption state, in addition to inflammatory responses and changes in gene transcription.

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7.2 NEUROTOXICITY:

The term Neuro toxic is used to describe a substance, condition or state that damages the nervous system and/or brain, usually by killing neuron. The term is generally used to describe a condition or substance that has been shown to result in observable physical damage. Neurologically damaged by exposure to "toxins", "chemicals", or other environmental agents. will seek confirmation and reassurance that their concerns are valid, that they have a definable illness, and that their condition will be treated. Considerable numbers are referred to a neurologist or psychiatrist for help. when the exposure to natural or artificial toxic substances, which are called neurotoxins, alters the normal activity of the nervous system in such a way as to cause damage to nervous tissue. This can eventually disrupt or even kill neurons.

Evaluating neurobehavioral function and the effects of toxic chemicals, drugs and substances (Neuro toxic agents) on the nervous system since 1979; serving the Courts (federal, state and administrative) as a forensic expert witness in both civil and criminal matters since 1983. The nervous tissue found in the brain, spinal cord and periphery comprises an extraordinarily complex biological system that largely defines many of the unique traits of individuals. As with any highly complex system, however, even small perturbations to its environment can lead to significant functional disruptions. Properties leading to the susceptibility of nervous tissue include a high surface area of neurons, a high lipid content which retains lipophilic toxins, high blood flow to the brain inducing increased effective toxin exposure, and the persistence of neurons through an individual's lifetime, leading to compounding of damages. As a result, the nervous system has a number of mechanisms designed to protect it from internal, and external insults, including the blood brain barrier.

The elimination of toxins from the body and the regeneration or rejuvenation of the immune system, the nervous system as well as blood, liver, and kidney, reveals three features of current popular thinking on toxins: a terrible ignorance of basic science, a poor understanding of the organization and function of the human body, and an irrational fear of the "chemicals" that prevent us from living to our full potential. Whatever our private thoughts on the expression and exploitation of these fears, we tend to forget that the most complete text on experimental and clinical neurotoxicology lists more than 350 compounds (synthetic and naturally occurring) known to cause functional or structural damage to the nervous system.

Definition:

Neuro toxicology is defined as the science that deals with the adverse effects of naturally occurring and synthetic chemical agents on the structure or function of the nervous system. Neurotoxin is a naturally occurring or synthetic chemical agent that can cause a functional or structural change in the nervous system.

7.3 MECHANISM OF NEUROTOXICITY:

Mercury in both organic and inorganic forms is neurotoxic. Methylmercury (MeHg) is a commonly encountered form of mercury in the environment. Early electrophysiological experiments revealed that MeHg potently affects the release of neurotransmitter from presynaptic nerve terminals.

Whether the primary cellular target of mercury is astrocytes or neurons, mercury can disturb cellular function through various mechanisms. Altering calcium ion homeostasis, depleting antioxidants, especially glutathione, and perturbing mitochondrial membrane potential have all been reported upon mercury exposure. Disturbed glutamate activity and enhanced formation of reactive oxygen species (ROS) are cellular effects of mercury toxicity. Mercury has been shown to inhibit Na+"K+"Cl" cotransportand to decrease adenosine triphosphate levels. These effects, both individually and in combination, can lead to cell death through apoptosis, necrosis, or both.

7.4 CLASSIFICATION OF NEUROTOXINS:

Neurotoxins are compounds which adversely affect the nervous system, a number of mechanisms through which they function are through the inhibition of neuron cellular processes. These inhibited processes can range from membrane depolarization mechanisms to inter-neuron communication. By inhibiting the ability for neurons to perform their expected intracellular functions, or pass a signal to a neighboring cell, neurotoxins can induce systemic nervous system arrest as in the case of botulinum toxin, or even nervous tissue death. The time required for the onset of symptoms upon neurotoxin exposure can vary between different toxins, being on the order of hours for botulinum toxins and years for lead.

7.5 SIGNS AND SYMPTOMS OF NEUROTOXICITY:

Brain damage, Memory loss, anxiety, depression, Impaired mental functioning, Limb weakness, Limb numbness, Impaired vision, Headache, Impaired cognitive function Behavioral problems and Sexual dysfunction. Symptoms may appear immediately after exposure or be delayed. They may include limb weakness or numbness, vision, and/or intellect, uncontrollable obsessive and/or compulsive behaviors, delusionscognitive and behavioral problems and sexual dysfunction. Individuals with certain disorders may be especially vulnerable to neurotoxins.

7.6TREATMENT:

Neurotoxicity can result from exposure to substances used in chemotherapy, radiation treatment, drug therapies, certain drug abuse, and organ transplants, as well as exposure toheavy metals, certain foods and food additives[citation needed], pesticides, industrial and/or cleaning solvents, cosmetics, and some naturally occurring substances. The name implies the role of a neurotoxin, although the term neurotoxic may be used more loosely to describe states that are known to cause physical brain damage but where no obvious neurotoxin has been identified.

The presence of neurocognitive deficits alone is not usually considered sufficient evidence of neurotoxicity, as many substances exist which may impair neurocognitive performance without resulting in the death of neurons. This may be due to the direct action of the substance, with the impairment and neurocognitive deficits being temporary, and resolving when the substance is metabolised from the body. In some cases the level or exposure-time may be critical, with some substances only becoming neurotoxic in certain doses or time periods. Some of the most common naturally occurring brain toxins that lead to neurotoxicity as a result of excessive dosage are Beta amyloid (Aâ), Glutamate and Oxygen radicals. When present in high concentrations they can lead to neurotoxicity and death (apoptosis). Some of the symptoms that result from cell death include loss of motor control, cognitive deterioration and autonomic nervous system dysfunction. Additionally, neurotoxicity has been found to be a major cause of neurodegenerative diseases such as Alzheimer's disease (AD).

Everybody knows that you need good elimination habits to function optimally but few have only a basic understanding of what that means. In a society that is becoming more focused on health and nutrition, a closer look at a group of toxins, called neurotoxins, is needed. While elimination routes include kidney, gastrointestinal, skin and exhaled air, the liver is the most vital of body functions in the process of elimination of neurotoxins. Common sources of neurotoxins include metals, biotoxins (viral, fungal or parasitical sources), man-made chemicals called xenobiotics that include pesticides, preservatives and excitatoxins such as MSG, aspartame, and food colorings.

Neurotoxins are absorbed by nerve endings and travel inside the neuron to the cell body in the mammal nervous system. As a result, they cause disruption in vital functions of the cell such as axonal transport(1) of nutrients, mitochondrial function and proper DNA transcription. In the liver, elimination of most all products are expelled with the bile into the small intestine. Unfortunately, because of the lipophilic and neurotropic nature of neurotoxins, most of these toxins are reabsorbed in the small intestinal wall by nerve endings of the enteric nervous system (ENS). The ENS tissue is the same as the brain in the embryonic stage and then separates, hence sometimes referred to as the brain away from the brain. Once these toxins are reabsorbed, they can be transported back to the brain, the liver, sub clavian vein or uptake by bacteria in the bowel resulting in cause or exacerbation of illness cause by neurotoxins.Obviously, the issue is complex. Risk factors that can contribute to the sluggishness of the liver include allergy, poor diet of high carbohydrate and low protein, occupational exposure, prolong illness, surgeries, constipation, metal absorption and genetics to name a few.

Solutions include evaluation of liver function for Phase I and Phase II detoxification, evaluation of toxic levels, and implementing a detoxification program. It must include proper protein, a good mineral base and balanced electrolytes which can help displace metals. Other nutrients and food sources, including a mercury-free EPA/DHA fish oil, aide in binding up these toxins so they can be eliminated. Improving the diet, elimination and reduction of metal sources and other risk factors are a start but it needs to be done right.

7.7 HEPATIC TOXICITY

The liver plays a central role in transforming and clearing chemicals and is susceptible to the toxicity from these agents. Certain medicinal agents, when taken in overdoses and sometimes even when introduced within therapeutic ranges, may injure the organ. Other chemical agents, such as those used in laboratories and industries, natural chemicals (e.g., microcystins) and herbal remidies can also induce hepatotoxicity. Chemicals that cause liver injury are called hepatotoxins.

Mounting evidence indicates that microglial activation contributes to neuronal damage in neurodegenerative diseases. Recent studies show that in response to certain environmental toxins and endogenous proteins, microglia can enter an overactivated state and release reactive oxygen species (ROS) that cause neurotoxicity. Pattern recognition receptors expressed on the microglial surface seem to be one of the primary, common pathways by which diverse toxin signals are transduced into ROS production. Overactivated microglia can be detected using imaging techniques and therefore this knowledge offers an opportunity not only for early diagnosis but, importantly, for the development of targeted anti-inflammatory therapies that might slow or halt the progression of neurodegenerative disease. Hepatotoxicity (from *hepatic toxicity*) implies chemical-driven liver damage.

More than 900 drugs have been implicated in causing liver injuryand it is the most common reason for a drug to be withdrawn from the market. Hepatotoxicity and drug-induced liver injury also account for a substantial number of compound failures, highlighting the need for drug screening assays, such as stem cell-derived hepatocyte-like cells, that are capable of detecting toxicity early in the drug development process Chemicals often cause subclinical injury to liver which manifests only as abnormal liver enzyme tests. Drug-induced liver injury is responsible for 5% of all hospital admissions and 50% of all acute liver failures.

7.8 MECHANISM OF LIVER DAMAGE:

Drugs continue to be taken off the market due to late discovery of hepatotoxicity. Due to its unique metabolism and close relationship with the **gastrointestinal tract**, the liver is susceptible to injury from drugs and other substances. 75% of blood coming to the liver arrives directly from gastrointestinal organs and then spleen via **portal veins** which bring drugs and xenobiotics in near-undiluted form. Several mechanisms are responsible for either inducing hepatic injury or worsening the damage process. Many chemicals damage **mitochondria**, an intracellular organelle that produce energy. Its dysfunction releases excessive amount of oxidants which, in turn, injure hepatic cells. Activation of some enzymes in the cytochrome P-450 system such as **CYP2E1** also lead to oxidative stress. Injury to **hepatocyte** and **bile duct** cells lead to accumulation of **bile acid inside the liver**. This promotes further liver damage. Non-parenchymal cells such as Kupffer cells, fat storingstellate cells, and leukocytes (i.e. neutrophil and monocyte) also have a role in the mechanism.

7.9 HEPATIC TOXICITY SYMPTOMS:

symptoms are the results of a drug-induced liver damage. If we're on the subject of hepatic toxicity, one should understand that the other name for it is a short word that is hepato toxicity. To date, this disease is increasing at an alarming rate and is now the leading cause of liver failure in different countries. Because hepatic toxicity is all about adverse drug reactions, the unwilling victim is none other than the liver, as drugs intake cause a gradual damage to it.

On a global scale, many hospital admissions both adults and children, are ascribed to harmful reactions to drugs. According to statistics, almost 40% of these medical cases have been classified as life threatening. That is why it's never an understatement to say the accurate pre-screening of drugs and other chemical compounds for hepa toxicity should be effectively adopted by the medical profession. Termed as toxicity testing, this will help many health institutions help in reducing the number of drug-induced liver damage patients in the world, in the light of growing dependence on pharmac symptoms are the results of a drug-induced liver damage. If we're on the subject of hepatic toxicity one should understand that the other name for it is a short word that is

hepatotoxicity. To date, this disease is increasing at an alarming rate and is now the leading cause of liver failure in different countries. Because hepatic toxicity is all about adverse drug reactions, the unwilling victim is none other than the liver, as drugs intake cause a gradual damage to it.

Web Site Today we live in an over medicated society which only confirms why there is a high incidence of drug-induced liver damage. It might not be known to everybody but the most singled out pharmaceutical compound accountable for drug-induce liver damage is an analgesic drug called acetaminophen. It has the same effect as aspirin and is a major component in many no-prescription medicines like Tylenol, Medicol and Midol, that can be purchased over-the-counter.

For instance, liver donors are very,very few and that if there's one available, a patient has to fall in line. Needless to say, depends on who is first in line. Oftentimes, patients are prioritized according to the gravity of their condition. Under this scenario, many people turn to holistic healing or alternative medicines. The most popular in this category is detoxification which is believed to cleanse the body of its accumulated toxic substances. Detoxification is actually done in a number of methods, among them chelation therapy, dietary therapy, fasting, hydrotherapy, nutritional supplementation and more. Detoxification is meant to target chemical toxins and other drug byproducts that induce harm to our body organs. Proponents believe in the efficacy of detoxification to cleanse, rejuvinates,purifies and diminish hepatic toxicity symptoms

Mounting evidence indicates that microglial activation contributes to neuronal damage in neurodegenerative diseases. Recent studies show that in response to certain environmental toxins and endogenous proteins, microglia can enter an overactivated state and release reactive oxygen species (ROS) that cause neurotoxicity. Pattern recognition receptors expressed on the microglial surface seem to be one of the primary, common pathways by which diverse toxin signals are transduced into ROS production. Overactivated microglia can be detected using imaging techniques and therefore this knowledge offers an opportunity not only for early diagnosis but, importantly, for the development of targeted anti-inflammatory therapies that might slow or halt the progression of neurodegenerative disease. Hepatotoxicity (from hepatic toxicity) implies chemical-driven liver damage.

The liver plays a central role in transforming and clearing chemicals and is susceptible to the toxicity from these agents. Certain medicinal agents, when taken in overdoses and sometimes even when introduced within therapeutic ranges, may injure the organ. Other chemical agents, such as those used in laboratories and industries, natural chemicals (e.g., microcystins) and herbal remediescan also induce hepatotoxicity. Chemicals that cause liver injury are called hepatotoxins.

More than 900 drugs have been implicated in causing liver injury[1] and it is the most common reason for a drug to be withdrawn from the market. Hepatotoxicity and drug-induced liver injury also account for a substantial number of compound failures, highlighting the need for drug screening assays, such as stem cell-derived hepatocyte-like cells, that are capable of detecting toxicity early in the drug development process. Chemicals often cause subclinical injury to liver which manifests only as abnormal liver enzyme tests. Drug-induced liver injury is responsible for 5% of all hospital admissions and 50% of all acute liver failures.

7.10 SUMMARY

Several mechanisms initiate liver cell damage and aggravate ongoing injury processes. Mitochondria are prominent targets for the hepatotoxicity of many drugs. Dysfunction of these vital

cell organelles results in impairment of energy metabolism and an intracellular oxidant stress with excessive formation of reactive oxygen species and peroxynitrite. In addition to mitochondria, induction of cytochrome P450 isoenzymes such as CYP2E1 also promote oxidant stress and cell injury. Once hepatocellular function is impaired, accumulation of bile acids causes additional stress and cytotoxicity. Cell injury, gut-derived endotoxin or a combination of both also activate Kupffer cells and recruit neutrophils into the liver. Although responsible for removal of cell debris and part of the host-defense system, under certain circumstances these inflammatory cells initiate additional liver injury. However, cell injury and death is not only determined by the nature and dose of a particular drug but also by factors such as an individual's gene expression profile, antioxidant status, and capacity for regeneration. Because of the many direct and indirect mechanisms of drug-induced cell injury in the liver, hepatotoxicity remains a major reason for drug withdrawal from pharmaceutical development and clinical use.

7.11 SELF ASSESSMENT QUESTIONS

1.Explain Neurotoxicity importance indetail?

2.Describe Hepatic Toxicity?

3 Write classification of Hepatic toxicity?

7.12 REFERENCE BOOKS

1.Food toxicology part A.principles and concepts by Jose M.Concon Marcel Dekkher Inc, NEW YORK,1988.

2. Journal of Medical Microbiology

3. Current science

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Lesson – 8

NEPHRON AND HAEMO TOXICITY

8.0 Objective

After reading this chapter, you should be able to:

- Know the effect of Food toxins on Human health
- To create awareness regarding Nephron and Haemo toxicity and harmful side effects in human beings.
- To educate the community through nutrition Knowledge.

Structure

8.1 INTRODUCTION

The kidneys are the primary organs of the urinary system, which purifies the blood by removing wastes from it and excreting them from the body in urine. Every day, the kidneys filter about 45 gal (180 l) of blood, about four times as much as the amount that passes through any other organ. Because of this high volume, the kidneys are more often exposed to toxic substances in the blood and are very vulnerable to injury from those sources.

Each kidney contains over one million structures called nephrons. Each nephron consists of two parts: the renal corpuscle and the renal tubule. The renal corpuscle is where the blood is filtered. It is made up of a network of capillaries (the glomerulus) and the structure that surrounds these capillaries (Bowman's capsule). Blood flows into the glomerulus, where the liquid part of the blood (plasma) passes through the walls of the capillaries and into Bowman's capsule (blood cells and some proteins are too big to pass through and therefore remain in the blood vessels). The plasma, now called filtrate, contains substances that the body needs, such as water, glucose, and other nutrients, as well as wastes, excess salts, and excess water. When the filtrate moves from

Bowman's capsule into the renal tubules, about 99% of it is taken back up as the action of the tubules allows beneficial substances to be reabsorbed into the blood stream. The remaining filtrate is then passed to the bladder as urine.

When the kidneys are exposed to a toxic agent, either accidentally or intentionally (as in a suicide attempt), damage can occur in a number of different ways, depending upon the agent. One toxin may directly affect the glomerulus or the renal tubules, causing the cells of these structures to die. Another toxin may create other substances or conditions that result in the same cell death. Nephrotoxic injury can lead to acute renal failure, in which the kidneys suddenly lose their ability to function, or chronic renal failure, in which kidney function slowly deteriorates. If unchecked, renal failure can result in death.

8.2 NEPHRON TOXICITY

Characteristic features of metal nephrotoxicity are lesionsthat tend to predominate in specific regions of the proximal tubule and in the glomerulus. This suggests thatthese regions of the nephron may be selectively sensitiveto specific metals. Regional variability in sensitivity tometals could result from the localization of transport andbinding ligands that deliver metals to targets within thenephron andor the localization **of** molecular targets incertain cell populations. Although significant progresshas been made in identifying various extracellular, membrane,and intracellular ligands that are important in theexpression of the nephrotoxicity of metals, the mechanismsby which metals induce renal injury remain poorlyunderstood. This is due, at least in part, to the complexityof the ligand interactions that dominate the disposition ofmetals in living organisms. For the most part, metals affectmolecular processes through binding interactionswith molecular targets, generally nucleophiles such asproteins and nucleic acids. These interactions may alterthe normal function of the target molecule by displacingessential elements from the molecule, altering its structureor changing its rate of metabolism or its interactionswith other important ligands. While it is possible to studyin great detail the binding interactions of metals and theconsequences of binding on the function **of** specific binding

ligands, it is extremely difficult to determine whichof these interactions contribute to the biochemical changes that give rise to toxicity. Thus, elucidating mechanismsof toxicity requires an understanding of the pathophysiology.

8.3 MECHANISM OF NEPHRON TOXICITY

Most cases of TDF-associated nephrotoxicity have been described in patients with HIV infection rather than hepatitis B; whether this reflects a genuine potentiation of TDF toxicity by the HIV or simply reporting bias is unclear. The proximal tubule is an important contributor to drug excretion from the body, and the potential thus exists for interactions between TDF and other agents in this nephron segment that could lead to the development of toxicity. TDF enters proximal tubule cells through basolateral organic anion transporters and exits using the apical transporter MRP4 (multidrug resistance-associated protein 4) (Fig 2). The NRTI didanosine also is a substrate for organic anion transporters, and potentiation of kidney toxicity between didanosine and TDF is well recognized. It therefore is not recommended that both drugs be used concomitantly in the same patient. With regard to TDF exit from proximal tubule cells, 19 of 22 patients with severe tubular toxicity in our case series also were using a ritonavir-boosted protease inhibitor, and this combination has been described in other case reports. Ritonavir is a substrate for MRP2, and it

has been suggested that it may potentiate the toxicity of TDF by inhibiting exit from proximal tubule cells and increasing the intracellular concentration.

8.4 SIGNS AND SYMPTOMS OF NEPHRON TOXICITY

Several different substances can be toxic to the kidneys. These include:

Antibiotics, primarily aminoglycosides, sulphonamides, amphotericin B, polymyxin, neomycin, bacitracin, rifampin, trimethoprim, cephaloridine, methicillin, aminosalicylic acid, oxy- and chlorotetracyclines.analgesics, including acetaminophen (Tylenol), all nonsteroidal anti-inflammatory drugs (e.g. aspirin, ibuprofen), all prostaglandin synthetase inhibitors.contrast agents used in some diagnostic tests, such as sodium iodideheavy metals, such as lead, mercury, arsenic, and uranium.Anti-cancer drugs, such as cyclosporin, cisplatin, and cyclophosphamide.Methemoglobin-producing agents.solvents and fuels, such as carbon tetrachloride, methanol, amyl alcohol, and ethylene glycol.herbicides and pesticidesoverproduction of uric acid.

Nephrotoxic injury is most commonly caused by drugs, primarily antibiotics, analgesics, and contrast agents. In some cases, such as with aminoglycosides and amphotericin B, the drug itself will damage the kidneys. In others, such as with methicillin, sulphonamides, and some contrast agents, the drug provokes an allergic reaction that destroys the kidneys. Some chemicals found in certain drugs and industrial agents damage the kidneys by converting the hemoglobin of red blood cells into methemoglobin, thereby interfering with the blood's transport of oxygen. In hospitals, the most common form of nephrotoxic injury is antibiotic nephropathy, which usually occurs when antibiotics are given to patients with already weakened kidneys. Analgesic nephropathy is another common form of nephrotoxic injury and occurs as a result of long-term abuse of analgesics, usually NSAIDs (e.g., ibuprofen). Analgesic nephropathy is most prevalent in women over 30. Lead nephropathy, arising from lead poisoning, and nephropathy, from ingestion of the solvent carbon tetrachloride, are also more common forms of nephrotoxic injury. Uric acid nephropathy is one form of nephropathy that is not caused by exposure to an external toxin; instead, it arises from the body's overproduction of uric acid, usually in persons with diseases of the lymph nodes or bone marrow.

Risk factors for nephrotoxic injury include:

The elderly are more likely to overdose on antibiotics or analgesics.Underlying kidney disease. Kidneys already weakened by conditions such as diabetes can be particularly susceptible to nephrotoxic injury.Severe dehydration.Prolonged exposure to heavy metals or solvents on the job or in the home.Presence of diseases that cause the overproduction of uric acid.

Symptoms of nephrotoxic injury are wide ranging and, in some cases, depend upon the type of toxin involved. In general, symptoms are similar to those of renal failure and include excess urea in the blood (azotemia), anemia, increased hydrogen ion concentration in the blood (acidosis), excess fluids in the body (overhydration), and high blood pressure (hypertension). Blood or pus may be present in the urine, as may uric acid crystals. A decrease in urinary output may also occur. If the toxin's effect on the kidneys remains unchecked, more serious symptoms of kidney failure may occur, including seizures and coma.

The most efficient way to prevent or mitigate nephrotoxicity is to have sensitive and specific biomarkers that can be used in animals early in drug development, well before clinical studies are underway. These biomarkers should be able to sensitively predict toxicity in preclinical models and clinical situations so that they can be used to efficiently guide drug developers to modify or discard the potential therapeutics and replace them with variants that affect the same target without the toxicity. However, it is important to recognize that safety concerns must always be incorporated into a general 'risk-benefit' analysis and that toxicity of a drug does not necessarily mean that it should not be developed or approved. Some examples of nephrotoxic drugs that have provided a very high therapeutic benefit are the aminoglycoside antibiotics, the cancer drug cisplatin and the antiviral tenofovir.

One approach to the early detection of kidney injury involves defining different biomarkers that rely on the mechanisms of toxicity of each drug or drug class. However, this approach can be problematic for the many clinically useful agents for which the mechanism of toxicity is not well established. An alternative approach, to which we subscribe, involves finding a limited number of biomarkers that identify injury to primary sites in the kidney, such as the glomerulus or the proximal tubule, which together represent the major sites of toxicity related to >90% of drugs. Drugs with different mechanisms of toxicity frequently affect different parts of the kidney, which shows the primary sites of nephron toxicity for various drugs. The most likely explanation for this observation is that different regions of the nephron are characterized by different transporters, metabolic characteristics, blood flow characteristics and oxygen tensions. Most drug-inuced renal injuries affect the proximal tubules. Drug toxicity initially targeted to the glomerulus or more distal parts of the nephron may also cause secondary injury to proximal tubules. Detection of proximal tubule injury might thus provide a sensitive way to monitor most, but not all, toxicities. After these markers of glomerular and proximal tubule injury are established, additional ones can be added to reflect abnormalities of the distal and collecting tubules and ducts or papillary injury.

Histopathological changes in the kidney are associated with drug toxicity. These changes have been well characterized in commonly used experimental animals, and they currently remain as the 'gold standards' against which biomarkers from body fluids are measured. Although histopathology is the gold standard to detect renal injury, it is not without its shortcomings, even in animals where the entire organ can be examined. For example, it does not identify non–histopathology-associated types of kidney disturbances such as either inhibition of transporters in the proximal tubule (resulting in glucosuria, aminoaciduria or hyperuricosuria) or inhibition of vasopressin action in the collecting duct (resulting in diabetes insipidus). Furthermore, a degree of subjectivity is associated with histopathological evaluation. Finally, use of histopathology inevitably introduces a delay in appearance of injury. Following exposure to nephrotoxicants, levels of at least some biomarkers are reported to appear before obvious changes in histology are evident.

The use of histopathology as a benchmark for kidney injury in humans is usually impractical, except in relatively rare instances where a kidney biopsy is performed for diagnostic purposes. Moreover, the pathophysiology of the toxicity is associated with spatial variations in injury to particular [illegible] susceptibility of the tubules to injury. As discussed above, [illegible] these factors complicate the interpretation of the histopathology. Furthermore, there are frequently [illegible] which complicate the evaluation of biomarkers data. For example, [illegible] can be [illegible] used

by vascular or blood cells in addition to kidney tubules may reflect systemic perturbation rather than kidney injury. The strong foundation provided by detailed understanding of the sensitivity and specificity of a biomarker in various contexts of injury is thus critical to its appropriate use in animals and/or humans.

It is important to consider that biomarkers for one type of kidney toxicity may not be as useful in another. A good biomarker for injury may not reliably indicate delayed repair; a biomarker that detects inflammation effectively may not be as sensitive in detecting early proximal tubule toxicity in the absence of inflammation. A biomarker of injury might not detect a functional defect, such as is observed in Fanconi syndrome or nephrogenic diabetes insipidus. And a biomarker useful in an animal model may or may not be useful in the same way in humans. Another question is whether panels of biomarkers will be more informative than a single biomarker. At first, this might seem logical because different biomarkers might be more sensitive or specific for different forms of injury. Nonetheless, if multiple biomarkers are used to detect a similar form of injury, an adjudication process will be necessary if the biomarkers suggest different outcomes.

8.5 EFFECT OF DRUG TOXICITY

The underlying characteristics of the offending agent also play an important role in the development of nephrotoxicity. From a practical standpoint, prolonged therapy at high doses with toxic substances enhances renal injury based on excessive renal exposure in the absence of other risks. Exogenous substances and their metabolites that are insoluble in human urine may cause renal injury. In addition to unique characteristics of the offending agent that induce insolubility, factors such as urine pH, sluggish tubular urine flow rates, and rapid parenteral or excessive dosing (high peak serum and urine concentrations) enhance risk for precipitation and crystal formation in distal nephron tubular lumens.Commonly used medications such as acyclovir, methotrexate, sulfadiazine, indinavir, atazanavir, and oral sodium phosphate solution are examples. Aminoglycosides with more positive charge are more likely to cause nephrotoxicity, perhaps due to enhanced interactions with negatively charged membrane phospholipids and megalin.This is reflected by the greater nephrotoxicity of the neomycin as compared with amikacin. Drug combinations also raise risk of nephrotoxicity. Combinations such as aminoglycosides and cephalothin, nonsteroidal anti-inflammatory drugs (NSAIDs) and radiocontrast, and cisplatin and aminoglycosides are a few examples of enhanced nephrotoxic risk with concurrent drug administration. As mentioned previously, various drugs and endogenously produced molecules can compete for transport proteins in the proximal tubular cells, thereby reducing renal elimination and increasing intracellular drug/toxin concentration. This favors development of nephrotoxicity.

Several drugs and toxins are more highly nephrotoxic and can promote renal injury, even with brief or low-level exposure. Examples include the aminoglycosides (in particular neomycin), amphotericin B, the polymyxins, zolendronate, and the antiviral agents adefovir and cidofovir Aminoglycosides are a classic nephrotoxin. Accumulation of high concentrations within lysosomes and release into the cell cytoplasm promotes phospholipid membrane interruption, oxidative stress, and mitochondrial injury that cause proximal tubular cell apoptosis and necrosis, leading to clinical AKI. Amphotericin B, and the lipid/liposomal formulations to a lesser degree, disrupt cellular membranes and increase permeability to cations, resulting in tubular dysfunction due to cell swelling and lysis. Colistin and polymyxin B are polymixin antimicrobials that are extremely nephrotoxic with a very narrow therapeutic window. Their toxicity appears to be due to the D-amino content and fatty acid component, which increase membrane permeability and influx of cations.As seen with amphotericin B, cell swelling and lysis results. The acyclic nucleotide phosphonates enter the cell

via basolateral HOAT-1 and promote cellular injury through multiple mechanisms. Mitochondrial injury, as manifested by mitochondrial enlargement, clumped cristae, and convoluted contours, occurs with adefovir through inhibition of DNA polymerase gamma, which is the sole DNA polymerase in mitochondria.Cidofovir, which forms cidofovir-phosphocholine (analog of cytidine 5-diphosphocholine) within cells, interferes with synthesis and/or degradation of membrane phospholipids, resulting in proximal tubular injury. Tenofovir also impairs cellular energetics through mitochondrial disruption and other intracellular processes.A unique and newly recognized form of nephrotoxicity has been described with anti-angiogenesis therapy.Vascular endothelial growth factor (VEGF), produced by adjacent podocytes, is required to maintain normal fenestrated endothelial function and is particularly important for normal functioning of the glomerular basement membrane . Reduction in VEGF or its effects by the various anti-angiogenic drugs leads to loss of the healthy fenestrated endothelial phenotype and promotes microvascular injury and thrombotic microangiopathy, causing proteinuria and kidney disease. Also, reduced nephrin expression in the slit diaphragms from these drugs may also contribute to proteinuria. While these drugs cause a number of renal lesions, endothelial injury and thrombotic microangiopathy are the most commonly noted.Renal Compartments and Nephrotoxin Injury Nephrotoxic substances produce disease in all compartments of the kidney. The entire nephron and collecting duct system are capable of being injured by various nephrotoxins. For ease of classification, they can be divided into the following renal compartments and the associated disease components: 1) Hemodynamic (prerenal disease); 2) Renal parenchyma (intrinsic renal disease); and 3) Collecting system (postrenal disease).

8.6 TREATMENT

Antibiotics and hypotensor

Whether the patients suffer from kidney problems after taking those drugs, it also depends on one's physical condition. In addition to this, drug abuse, overdose and drug combination account for its occurrence too. In some countries, self-medication happens commonly. Antibiotics are usually adopted but may induce nephrotoxicity. Its side effects develop extremely strong when one takes the medication for a really long time or overdose. Aminoglycoside antibiotics,â lactam antibiotic, tetracycline, amphotericin, rifampicin and sulfonamides.

Contrast agent

Together with the high occurrence of cardiovascular disease and cancer in recent years, contrast agent is widely used in clinic, for instance, angiography, IVP (intravenous pyelography) or NCCT. Contrast agent may cause some serious consequences, like acute Renal Failure. It is especially true among people with dehydration, diabetes, high blood pressure, chronic renal insufficiency.

Hypotensor

Hypotensor is widely adopted due to high blood pressure attacks widely. Along with the progression of one's disease, combination of multiple drugs would be adopted. ACEI and ARBs are common hypotensors but can cause damages to kidneys if one has a low blood flow.

Immunosuppressants

Immunosuppressants are often used for patients with kidney transplant or with lupus etc. cyclosporin A is one of them and a typical one characterized by nephrotoxicity.

Chinese medicine

Here, the Chinese medicine, I mean some certain herbal medicines instead of all Chinese medicines. Nephrotoxic herbal drugs should be well realized and avoided, they are: Aristolochia fangchi, Caulis aristolochiae manshuriensis, aristolochia contorta, herba aristolochiae, berba aristolochiae mollissimae, ciliatenerve knotweed root and birthwor root. Other potential herbal medicines include thunder god vine, Mangnolia officinalis and leonurus etc.

Because the liver is able to pull the toxins out of the body and excrete them into your bowel, the trick is to keep the toxins in your bowel so that they go out in the stool. Fortunately, there is an old medication called Questran (cholestyramine). It is a powder that acts like a sponge, and it used to be prescribed to pull cholesterol out of people's bodies. which people take one scoop or one packet four times a day (although not as effective, you can sometimes get by with 3 times a day, however). Within a month (usually within 3 days to 2 weeks), those who are going to improve with this treatment will often start feeling better or at least have their vision test improve. Insomnia, pain and spastic colon symptoms can sometimes improve in 1-3 days with this treatment.

Diagnosis

Damage to the kidneys is assessed through a combination of physical examination, blood tests, urine tests, and imaging procedures. Diagnosis of nephrotoxic injury as the underlying cause results from a thorough investigation of the patient's history. Information regarding preexisting conditions, current prescriptions, and environmental exposures to toxins aid the physician in determining what toxin, if any, has caused the kidneys to malfunction.

Treatment

Treatment of nephrotoxic injury takes place in the hospital and focuses on removing the toxin from the patient's system, while maintaining kidney function. Removal methods are targeted to specific toxins and may include the use of diuretics or chelates to enhance excretion of the toxin in urine, or, in extreme cases, the direct removal of toxins from the blood via hemodialysis or passing the blood over an absorbent substance such as charcoal. Success of treatment depends on the extent of damage to the organs and ranges from monitoring

Chelate — A chemical that binds to heavy metals in the blood thereby [illegible] excrete them in urine.

Contrast agent — Substance ingested so as to highlight [illegible] tests.

Diuretic — A drug that promotes the excretion of urine

Glomerulus — A network of capillaries [illegible] the bu[illegible]

Methemoglobin — A compound formed from hemoglobin by oxidation

Nephron — [illegible]

Nephrotoxin — Substance that is poisonous to the kidneys.

Renal failure — Disorder characterized by the kidney's inability to filter wastes from the blood. It may be acute (occuring suddenly and usually reversable) or chronic (developing slowly over time as a result of permanent damage).

Prognosis

The outcome of nephrotoxic injury is determined by the cause and severity of the damage. In cases where damage has not progressed beyond acute renal failure, kidney function can be fully restored once the toxin is removed from the system and equilibrium restored. However, if permanent damage has resulted in chronic renal failure, lifelong dialysis or a kidney transplant may be required.

Prevention

Exposure to nephrotoxins can be minimized several different ways. When taking antibiotics or analgesics, recommended dosages should be strictly followed. Also, elderly patients on these medications (for example, those taking aspirin for heart problems or NSAIDs for arthritis) should be closely monitored to prevent accidental overdose. Health care workers should be aware of any underlying conditions, such as diabetes or allergies to antibiotics, that may heighten the effect of a potential nephrotoxin. When using solvents or handling heavy metals, procedures regarding their safe use should be employed.

8.7 HAEMOTOXICITY

Hepatotoxicity is a state of toxic damage to the liver. Drug-induced toxicity is the leading cause of acute liver failure in the United States. Antidotes are available for only a few hepatotoxins with one being N-acetylcysteine for acetaminophen overdose, which is a common drug most often associated with hepatotoxicity. Drug hepatotoxicity is rare and may not occur during clinical trials due to limited participants, compared to the number of consumers once the drug is on the market. Signs and symptoms can vary from fatigue, nausea, upper quadrant abdominal pain, loss of appetite, to jaundice. Adults are more susceptible to hepatotoxicity than [illegible] and women are more susceptible than men. Some products [illegible] grapefruit juice induce hepatic [illegible] an[illegible] alcohol abuse also [illegible] drugs. Liver function [illegible] testing test results [illegible] taken into acc[illegible]

[illegible] and [illegible] that [illegible] ary painful [illegible] ed limb is possible

Hemotoxins are frequently employed by venomous animals, including vipers and pit vipers. Animal venoms contain enzymes and otherproteins that are hemotoxic or neurotoxic or occasionally both (as in the Mojave Rattlesnake, the Japanese mamushi,[1] and similar species). In addition to killing the prey, part of the function of a hemotoxic venom for some animals is to aid digestion. The venom breaks down protein in the region of the bite, making prey easier to digest.

8.9 SYMPTOMS OF HAEMO TOXICITY

Fang marks. This is usually a good indication of a potential envenomation. This could either be distinct puncture marks at the site, or merely a scratch.

* Minimal Edema (swelling). Victims may experience slight swelling around the bite site. This is not immediate, and may occur some times after a bite. Unlike cytotoxic venom the swelling does not progress further along the affected limb.

* Slight pain and discomfort may be experienced by the victim usually 1-3 hours after envenomation has occured.

* Continuous bleeding from bite mark. This occurs as a result of the venom destroying the coagulent properties of blood.

* Irregularities in victims blood.

* Bruising(echymosis) usually occurs as a result of the weakening of the capillary endothelium. The damaged capillaries allow blood to seep through into the surrounding tissue.

* Headaches.

* Overall general weakness.

* Nausea.

* Hematemesis (Vomiting blood). As the mucous membrane surfaces of the upper gastrointestinal tract (mouth, pharynx, oesophagus,stomach, and small intestine) begin to bleed it causes involuntary regurgitation causing the victim to vomit blood.

* Anaemia and shock may develop in certain bite victims.

* Bleeding from stractches and mucus surfaces.

* Victims may also experience Epistaxis (nose bleed).

* Another possible symptom of a haemotoxic envenomation is the presence of blood in the victims urine (hematuria).

* Mental confusion. Victims often experience feelings of disorientation, and their decision-making ability may become impaired.

* Increased sweating may occur.

* Hypertension, (increase in blood pressure) may occur in certain victims.

* In some cases the opposite occurs and victims may experience hypotension (decrease in blood pressure).

* Multiple organ dysfunction syndrome, previously known as multiple organ failure occurs if untreated.

* Convulsions.

* Unconciousness.

* Coma.

* Death.

8.10 SUMMARY

Nephrotoxins and Haemotoxins plays an important role in human bodies.toxicity is sometimes dangerous.prevention and tretment is very essential.If not it leads to coma or death.Proper precautions and remidies are also to be taken after diagnosing the condition.

Drug-induced nephrotoxicity plays an important role in the high incidence and prevalence of AKI and may serve as an important contributor to chronic renal disease. Current metrics, such as SCr and BUN, lack the sensitivity and/or specificity to adequately detect nephrotoxicity before significant loss of renal function. Better biomarkers will allow drug developers to make more informed decisions about which products to move forward in testing, the doses at which they should be used, and ways to design clinical trials that will provide clear information about product benefit and safety. Besides facilitating drug development, biomarkers shown to reliably predict kidney injury in experimental animals should eventually be evaluated for their utility in humans to promote patient safety and guide therapeutic decisions in the clinic.

8.11 SELF ASSESSMENT QUESTIONS

1.Explain Haemotoxicity importance in detail?

2.Describe Nephron Toxicity?

3. explain the prevention and treatment of Haemotoxicity?

8.12 REFERENCE BOOKS

1.Food toxicology part A.principles and concepts by jose M.concon marcelDekkher Inc, NEW YORK,1988.
2. Journal of Medical Microbiology
3. Current science.

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Lesson – 9

SKELETAL AND REPRODUCTIVE TOXICITY

9.0 Objective

After reading this chapter, you should be able to:

- Know the effect of Food Toxins on Human Health
- To create awareness regarding skeletal and reproductive toxicity and harmful side effects in human beings.
- To educate the community through nutrition Knowledge.

Structure

9.1 INTRODUCTION

The skeletal muscle is necessary for normal motor functions such as walking or breathing. It accounts for between 30 and 50% of total body weight in humans and is the most energy-consuming tissue in the body. Skeletal muscle is a unique tissue with intricate organization and arrangement, requiring precise regulation of intracellular ions and cooperation by a [illegible]. As a result, it also has vulnerabilities to a diverse array of toxic [illegible] can cause loss of movement, multiorgan [illegible] direct effects on skeletal muscle, this [illegible] and organs by release of [illegible] [illegible] important [illegible] target [illegible] muscle physiology [illegible] and function

Laboratory methods and diagnostic observations to be used for investigating skeletal muscle injury in a research and clinical setting are also discussed. Finally, we provide an in-depth outline of skeletal muscle toxicants arranged according to their proposed mechanism of action.But they can also be caused by an abundance of something, like too much fluoride, too much mercury, too much arsenic, or too much lead.

9.2 SKELETAL TOXICITY

Lead is a heavy metal, one that is not required by the human body for metabolic functions, unlike vitamins C or D. This element is found naturally in the environment, so it can be expected to find trace levels of lead in the skeleton of every individual, both ancient or modern. However, because of the physical properties of lead being malleable and easily worked people have been using it to create a large range of items and thus have been exposed to heavy metal toxicity for millennia. The dangers of lead were not fully appreciated until the second half of the 20th century, which was why lead began to be taken out of household goods such as paints and fuels.

9.3 SIGNS AND SYMPTOMS OF SKELETAL TOXICITY

The main danger of lead – the reason that it is toxic – is due to the way it interferes with the normal enzyme reactions within the human body. Lead actually mimics the properties of other metals that are essential to biological functioning. But as lead does not work the same way as those metals, the enzymatic reactions that depend on things like calcium, iron, and zinc are disrupted. The most damaging enzymatic reaction that lead affects is the production of haemoglobin, or red blood cell production, which can cause anaemia. So doctors in modern times often find anaemia in a person with lead poisoning. Lead is also particularly problematic because it stays in the body for a long time once it has been absorbed, inhaled, or ingested. Most of the lead is depos.. in our bones and teeth. However it can be removed from the body by excretion through the kidneys nd urine, but it is a very slow processwithoutmodern chelation therapy.

In modern society lead poisoning is diagnosed through a blood test to determine the level of lead in the body. With ancient remains however there is no blood to sample and so it is necessary to investigate lead levels in what can be measured in bone and enamel. Probably the first and only study to actually measure levels of lead in skeletons from Rome is the one that involved samples provided by the author from two cemeteries at Casal Bertone and Castellaccio Europarco (1st-3rd c AD) near Rome itself.The analysis was led by Janet Montgomery, now at Durham University, and also involved around 200 samples from Britain from the Neolithic to the Late Medieval periods (Montgomery et al. 2010).

9.4 TREATMENT

Skeletal disorders are often associated with advancingage, but these effects can be exacerbated by exposure tocancer and its treatment. This review will explore the cancer and cancer treatment-related causes of skeletal disorders.Methods.

9.5 REPRODUCTIVE TOXICITY

On the basis of current knowledge of reproductive biology and toxicology, it is apparent that chemicals affecting reproduction may elicit their effects at a number of sites in both the male and the female reproductive system. This multiplicity of targets is attributable to the dynamic nature of

the reproductive system, in which the hypothalamic-pituitary-gonadal axis is controlled by precise positive and negative feedback mechanisms among its components. Interference by a xenobiotic at any level in either the male or the female reproductive system may ultimately impair hypothalamic or pituitary function. Normal gonadal processes such as spermatogenesis or oogenesis, ejaculation or ovulation, hormone production by Leydig or granulosa cells, and the structure or function of the accessory reproductive structures (e.g., epididymis, fallopian tube) also appear vulnerable to xenobiotics. The reproductive system is a complex one that requires local and circulating hormones for control. This brief review illustrates a system for characterizing the mechanism of action of reproductive toxicants, as well as for defining the sites available for disruption of reproduction. Unfortunately, at present, data addressing the actual vulnerability of reproduction are sorely lacking. However, when experiments have been conducted and combined with epidemiologic data or clinical observation, it has been possible to demonstrate impairment of reproductive processes by xenobiotics. The role of environmental exposure to xenobiotics in the increase in infertility

The overall objectives of this research core are: (1) to enhance intellectual interactions and collaborative research in reproductive toxicology between the members of the research core as well as other center investigators through use of service core facilities; (2) to test scientific hypotheses and to undertake interdisciplinary research initiatives which are likely to result in new proposals for extramural funding.

General areas of overlapping research interests within this core unit include: (1) mechanistic studies of the action of environmental toxicants on reproductive cells at the cellular and molecular level, (2) application of biomarkers for reproductive toxicity for use in epidemiologic field studies, (3) identification of clinical populations and animal models that are relevant for understanding the adverse effects on reproduction that may result from environmental exposures. Specific research interests in the group are focused on the male, the female, the preimplantation embryo and the fetus. Environmental chemicals to be studied include the pesticides(DDT, 2,4D), herbicides (atrazine, simizine, molinate), fungicides (benomyl) and products of combustion (dioxin).

9.6 SYMPTOMS OF REPRODUCTIVE TOXICITY

Lead is a heavy metal, one that isn't needed by the human body, unlike vitamins C or D. This element is found in the environment naturally, so we do expect to find some amount of lead in the skeleton of every person, ancient or modern. But, because of the physical properties of lead - it can be made into hard, sharp things - people have been using it for millennia and thus have been exposed to heavy metal toxicity for millennia as well. The dangers of lead actually weren't well known until the second half of the 20th century, which was when lead was taken .The main problem with lead - the reason that it's toxic - is that it interferes with normal enzyme reactions within the human body. Lead can actually mimic other metals that are essential to biological functioning. But since lead doesn't work the same way as those metals, the enzymatic reactions that depend on things like calcium, iron, and zinc are disrupted. The most damaging enzymatic reaction that lead affects is the production of hemoglobin, or red blood cell production, which can cause anemia. So doctors in modern times often find anemia in a person with lead poisoning. Lead is also particularly problematic because it stays in the body for a very long time once it's absorbed, inhaled, or ingested. Most of it gets deposited in the bones and teeth. Lead can be removed from the body, excreted through the kidneys and urine, but it's a very slow process without modern chelation therapy.

Although chemical exposure may be one of the most preventable causes of reproductive disease, the scope of this problem has not been well defined and there have been only limited attempts at concerted research efforts to seek solutions. The current status of the field is such that only an integrated, multidisciplinary effort can make rapid and significant progress in understanding and mitigating the adverse effects of environmental factors on human reproduction. This effort will require the combined expertise of toxicologists, epidemiologists, reproductive and developmental biologists and specialized clinicians. Such a team has been formed in this research facilitation core and this group will address research problems with a range of research skills including cellular and molecular techniques, specialized expertise in endocrine biomarkers, clinical medicine and epidemiology.

Research into occupational exposures and effects on reproductive systems has made important scientific contributions in the past few decades. Early studies focused on possible effects on the foetus rather than the reproductive health of the woman. Later, it was realized that reproductive toxins may also induce hormonal alterations affecting other aspects of reproductive health such as the menstrual cycle, ovulation and fertility. Attention is now shifting from concern for the pregnant woman and the foetus, to the entire spectrum of occupational health hazards among women and the reproductive health of both genders.

It is defined as any biological index capable of being measured, which is associated with or indicative of a defined biological endpoint such as a developmental or disease stage. Identification and verification of anatomical, endocrine, cellular and molecular biomarkers is crucial for successful clinical diagnosis and treatment of toxicity and disease, as well as basic toxicological, epidemiological and other research. Various biomarkers of reproductive development and health have been identified, including those associated with pubertal development, adult reproductive health and pregnancy outcome.

Assessment by ContinuousBreeding (FACB or RACB), has been developed by the National Toxicology Program (NTP) (Lamband Chapin, 1985; Morrissey et al., 1989; Gulati et al., 1991). As originally described, this protocol(FACB) was a one-generation test. However, in the current design (RACB), dosing is extended intothe F1 generation to make it compatible with the EPA workshop recommendations for a twogeneration design (Francis and Kimmel, 1988). The RACB protocol is being used with both mice andrats. A distinctive feature of this protocol is the continuous cohabitation of male-female pairs (in the Pgeneration) for 14 weeks. Up to five litters can be produced with the pups removed soon after birth. This protocol provides information on changes in the spacing, number, and size of litters over the 14-week dosing interval. Treatment (three dose levels plus controls) is initiated in postpubertal males andfemales (11 weeks of age) seven days before cohabitation and continues throughout the test. Offspringthat are removed from the dam soon after birth are counted and examined for viability, litter and/or pupweight, sex, and external abnormalities and then discarded. The last litter may remain with the dam untilweaning to study the effects of in utero as well as perinatal and postnatal exposures. If effects onfertility are observed in the P or F generations, additional reproductive evaluations may be conducted,including fertility studies and crossover matings to define the affected gender and site of toxicity. The sequential production of litters from the same adults allows observation of the timing ofonset of an adverse effect on fertility. In addition, it improves the ability to detect subfertility due to thepotential to produce larger numbers of pregnancies and litters than in a standard single- ormultigeneration reproduction study. With continuous treatment, a cumulative effe Sexual behavior reflects complex neural, endocrine, and reproductive organ interactions and istherefore susceptible to disruption by a variety

of toxic agents and pathologic conditions. Interference24with sexual behavior in either sex by environmental agents represents a potentially significant humanreproductive problem. Most human information comes from studies on effects of drugs on sexualbehavior or from clinical reports in which the detection of exposure-effect associations is unlikely. Dataon sexual behavior are usually not available from studies of human populations that were exposedoccupationally or environmentally to potentially toxic agents, nor are such data obtained routinely instudies of environ Increase or decrease in ovarian weightC Increased incidence of follicular atresiaC Decreased number of primary folliclesC Decreased number or lifespan of corpora lutea. Evidence of abnormal folliculogenesis or luteinization, including cystic follicles,luteinized follicles, and failure of ovulation.Evidence of altered puberty or premature reproductive senescence mental ag The mammary glands of normal adults change dramatically during the period around parturitionbecause of the sequential effects of a number of gonadal and extragonadal hormones. Milk letdown isdependent on the suckling stimulus and the release of oxytocin from the posterior pituitary. Thus,mammary tissue is highly endocrine dependent for development and function (Wolff, 1993; Imagawa etal., 1994; Tucker, 1994).

Mammary gland size, milk production and release, and histology can be affected adversely by toxic agents, and many exogenous chemicals and drugs are transferred into milk (American Academyof Pediatrics Committee on Drugs, 1994; Oskarsson et al., 1995; Sonawane, 1995). Reduced growthof young could be caused by reduced milk availability, palatability or quality, by ingestion of a toxicagent secreted into the milk, or by other factors unrelated to lactational ability (e.g., deficient sucklingability or deficient maternal behavior). Perinatal exposure to steroid hor Significant effects on measures showing a decrease in the age of onset ofreproductive senescence in females should be considered adverse. Cessation of normal cycling, which is measured by vaginal smear cytology, ovarian histopathology, or an endocrine profile that is consistentwith this interpretation, should be included as an adverse effect. mones and other chemicals can ents with rost species. ct could female rats and mice, the age at vaginal opening is the most commonly measured marker ofpuberty. This event results.from an increase in the blood level of estradiol. The ages and weights offemales at the first cornified (estrous) vaginal smear, the first diestrous smear, and the onset of vaginalcycles have also been used as endpoints for onset of puberty. In [illegible] separation orappearance of sperm in expressed urine or ejaculates can serve as [illegible] of puberty. Body weight atpuberty may provide a means to separate specific delays in [illegible] those that are related togeneral delays in development. Agents may differentially [illegible] puberty onset.so it is useful to have inform For all examinations of [illegible] reproductive effects and potentially toxicexposures, defining the exposure [illegible] crucial. Theconcepti exposures ofeither parent and in utero exposures [illegible] examinedoutcomes (e.g., fetal loss, malformations, [illegible] Theseexposures, plus postnatal exposure via [illegible] may also be associatedwith postnatal developmental effects [illegible] on). A number of factors affect the [illegible] environmental exposures are typically lower than those found in industrial or agricultural settings. Howeve relationship may change as exposures are reduced in workplaces and as more is learned aboutenvironmental exposures (e.g., indoor air exposures, home pesticide usage). Larger populations arenecessary to achieve sufficient power in settings with lower exposures which are likely to have lowermeasures of risk (Lemasters and Selevan, 1984). In addition, exposure of individuals may change astheyage in and out of areas with differing levels and types of exposures, thus affecting the number of exposed and comparison events for study.Data on exposure from human studies are frequently qualitative, such as

employment orresidence histories. More quantitative data may be difficult to obtain because of the nature of certainstudy designs (e.g., retrospective studies) and limitations in estimates of historic exposures. Manyreproductive effects result from exposures during certain critical times. The appropriate exposureclassification depends on the outcomes studied, the biologic mechanism affected by exposure, and thebiologic half-life of the agent. The half-life, in combination with the patterns of exposure (e.g.,continuous or intermittent) affects the individual's body burden and consequently the actual dose duringthe critical period. The probability of misclassification of exposure status ation on more than one marker Teratogenic effects.

Birth defects are known to occur in 3-5% of all newborns. They are the leading cause of infant mortality in the United States, accounting for more than 20% of all infant deaths. Seven to ten percent of all children will require extensive medical care to diagnose or treat a birth defect. And although significant progress has been made in identifying the etiology of some birth defects, approximately 65% have no known or identifiable cause. It was previously believed that the mammalian embryo developed in the impervious uterus of the mother, protected from all extrinsic factors. However, after the thalidomide disaster of the 1960s, it became apparent and more accepted that the developing embryo could be highly vulnerable to certain environmental agents that have negligible or non-toxic effects to adult individuals.

A teratogen is a substance that can cause birth defects. Thus, something that can cause deformities to a fetus during pregnancy is teratogenic.Some drugs that are prescribed for bipolar disorder are known to be teratogenic. In particular, some of the anticonvulsant medications that are used as mood stabilizers have been found to carry a major risk of birth defects.Most bipolar disorder medications are in a category where the risk of birth defects in humans has not been studied, but animal studies suggest there may be such a risk. For these drugs, doctors and patients are urged to consider whether the potential risks to the infant outweigh the risks to the mother of discontinuing the drugs.

9.7 SUMMARY

The skeletal musculature is necessary for normal motor functions such as walking or breathing. It accounts for between 35 and 45% of total body weight in humans and is the most energetically demanding tissue in the body. Skeletal muscle is a unique tissue with intricate organizational arrangement, requiring precise regulation of intracellular ions and cooperation by a multitude of cellular proteins. As a result, it also has susceptibilities to a diverse array of toxic insults. Derangements of skeletal muscle function can cause loss of movement, multiorgan involvement and even organismal demise. In addition to direct effects on skeletal muscle, this tissue can also be responsible for damage to distant tissues and organs by release of large intracellular proteins into the vasculature.

9.8 SELF ASSESSMENT QUESTIONS

1.Explain Reproductive toxicity importance in detail?

2.Describe skeletal Toxicity?

3. Explain the prevention and treatment of Skeletal toxicity?

9.9 REFERENCE BOOKS

1.Food toxicology part A.principles and concepts by jose M.concon marcel Dekkher Inc, NEW YORK,1988.

2. Journal of Medical Microbiology

3. Current science.

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Lesson – 10

CARCENOGENIC MANIFESTATITIONS

10.0 Objective

After reading this chapter, you should be able to:

Know the effect of Food Toxins on Human Health

To create awareness regarding Carcenogenic manifestations and harmful side effects in human beings.

To educate the community through nutrition Knowledge.

Structure

10.1 INTRODUCTION

Although any food protein can be potentially allergenic, relatively few cause most allergic reactions. In addition, an allergenic protein can only induce an allergic reaction in an allergic person who has been sensitized to it. [*Sensitization* occurs when a person's immune system has responded to a food as if it were a foreign material that could pose a threat to the body.] Most of the severe allergic reactions to foods occur in response to a surprisinglysmall number of foods.

The foods most commonly associated with allergic reactions in children are: milk, egg, wheat, soy, peanut, tree nuts, fish and shellfish. Allergies to milk, egg, wheat and soy are usually outgrown in early childhood. Adults generally experience allergic reactions to the foods that tend to persist as allergens beyond infancy. These are: peanuts, tree nuts, shellfish and certain species of fish.

Lists of the most highly allergenic foods vary according to the source of the data. Ingeneral, the "top eight allergenic foods" include:Peanut and peanut products,Soy and soy products,Egg and egg products andMilk and milk products.Tree nuts and tree nut products: the most allergenic of these tend to be:Walnut,Fish and fish products (not all species of fish have the same allergenicity)Shellfish: Crustaceans (shrimp, prawn, lobster, crab, crayfish (crawfish)) andMolluscs

(clams, mussels, oysters, scallops),Wheat and wheat products.

10.2 ALLERGENICITY

Foods varies. For example:Peanuts, tree nuts, shellfish, fish, milk and egg account for most reported cases ofanaphylactic reactions in children and adultsSoy is less frequently reported as a highly allergenic food, although it is oftenassociated with severe cases of allergy and atopic dermatitis (eczema) in childhoodWheat allergy (quite distinct from gluten-sensitive enteropathy or celiac disease,which is not considered an allergic condition) is usually mild, and is omitted frommany "top allergen lists"Other allergenic foods, present on some lists, absent on others include:Sesame seed and products containing sesame seed,Mustard seed,Cod,Corn.Food additives rarely cause IgE-mediated hypersensitivity reactions, and therefore do notappear on allergen lists. The exception is sulfite, which is included on many food allergenlists.Cooking and processing of foods can affect their allergenicity: Some foods, especiallyvegetables and fruits, become less allergenic when cooked. The allergenicity of many otherfoods is unaffected by heat and they cause the same degree of reaction whether eaten raw orcooked.changed by heating as to no longer cause an allergic reaction, while others are unaffectedeven by boiling. Whether a person can tolerate boiled milk or not depends on the specificproteins to which they are sensitized: If a person is allergic to milk proteins that aredenatured by heat (heat labile proteins) they will tolerate boiled milk, but not milk that hasbeen insufficiently heated. When a person is sensitized to milk proteins that are unaffectedby heat (heat stable proteins), they will develop allergic symptoms after consuming milkregardless of whether it has been boiled or not.The method of cooking also seems to affect the allergenicity of some foods: Forexample, roasting peanuts, which is common in Western countries, tends to increase theallergenicity of the peanuts, whereas boiling or other methods of cooking, more common inOriental countries, either reduces, or does not affect peanut allergenicity.In addition, the ripeness of vegetables and fruits can affect their degree ofallergenicity.

During the ripening process the plant produces differentcomponents, some ofwhich may be less or more allergenic that the unripe form. Thus it is often not possible topredict whether a fruit or vegetable will be less or more allergenic as it ripens.An interesting example of a change in the "reactivity potential" of a plant product isthe tomato. In this case it appears that it is the histamine content of the fruit that changes,not the protein. The green tomato rarely causes symptoms in a histamine-intolerant person,whereas the ripe fruit does cause a reaction. Tomatoes release histamine during the processof ripening. Although this is not, strictly speaking, an example of a change in allergenicity,it is a very good illustration of how a food in one stage of maturation causes symptoms, butin a later stage does not.Despite the fact that every atopic individual will react differently to foods, with diversesymptoms of varying severity, and that they will develop symptoms in response to variousallergenic foods in the form of a scale of "relative reactivity". The Food Allergen Scaleprovided here (often referred to as the *Joneja Allergen Scale*) has been developed overfifteen years and summarizes data and information from many published articles as well asindividual allergists from several countries who have kindly shared their experiences in food.

allergy management of patients with the author.A scale of this type has several uses:Used as "reactivity chart" it allows a person to see the number of foods that areavailable to them after their allergens have been crossed off. So often a patient isoverwhelmed when informed that they must avoid a number of staple foods (forexample, wheat, milk and milk products and eggs), and the common response is,"But there is nothing left to eat!" A glance at the remaining foods on the chartreassures them, and counselling can proceed in a more amiable fashion thereafterThe foods

most likely to cause an allergic response (the "top eight") are apparent.This is useful for the atopic individual because so often chronic reactivity to thesefoods in particular seems to lead to "hidden food allergy". As a person begins tomake alternative choices, their symptoms are more readily identifiable when theculprit food is eaten less frequentlyIf there is a risk of severe or anaphylactic reactions, the culprit foods can be markedin red: this makes family members more aware of the allergic individual's needs andto be alert to sources of the problem food. Many people like to keep a copy of thescale on the fridge door to alert themselves and others to their danger. For thisreason the printed scale is limited to a single page.The *Food Allergen Scale* is based on the typical experience of persons eating a Westerndiet. In cultures where significantly different foods are commonly consumed, the chart.

The GHS defines a germ cell mutagen as a chemical that may cause mutations in the germcells of humans that can be transmitted to the progeny. A mutation is defined as apermanent change in the amount or structure of the genetic material in a cell. The termsmutagenic or mutagen are used to refer to those chemicals that cause an increasedoccurrence of mutations in populations of cells and/or.organisms. Genotoxic is a moregeneral term that applies to agents or processes which alter the structure, informationcontent, or segregation of DNA, including those which cause DNA damage by interferingwith normal replication processes, or which in a non-physiological manner temporarily alterits replication.

Children prenatally exposed to alcohol can suffer from serious cognitive deficits and behavioral problems as well as from alcohol-related changes in brain structure. Neuropsychological studies have identified deficits in learning and memory as well as in executive functioning both in children with fetal alcohol syndrome and in children with less severe impairments. Both groups of children also exhibit problem behaviors, such as alcohol and drug use, hyperactivity, impulsivity, and poor socialization and communication skills. Brain imaging studies have identified structural changes in various brain regions of these children including the basal ganglia, corpus callosum, cerebellum, and hippocampus that may account for the cognitive *deficits. Functional brain imaging studies also have detected changes in alcohol-exposed children indicative of deficits in information processing and memory tasks.*

10.3 TARATOGENIC EFFECTS

Prenatal alcohol exposure can have serious and permanent adverse effects on children. The extent and severity of a child' s condition depends on several factors, such as how much alcohol the pregnant mother consumed and how often and at what point during her pregnancy she drank. The most serious outcome is fetal alcohol syndrome (FAS) , the diagnosis of which is based on three criteria: (1) growth deficiency manifested by small overall height and small head size (i. e. , microcephaly) ; (2) central nervous system disorders; and (3) a distinctive pattern of abnormal facial features. Other children with histories of heavy prenatal alcohol exposure, however, often do not meet the diagnostic criteria of FAS. These children, who typically lack the characteristic facial features of FAS, have variously been labeled as having fetal alcohol effects (FAE) , alcohol-related neurodevelopmental disorder (ARND) , or prenatal exposure to alcohol (PEA) . Both children with FAS and those with related disorders can be born to women known to drink in a heavy episodic fashion or more regularly during pregnancy. For the remainder of this article, children with histories of prenatal alcohol exposure who do not meet the diagnostic criteria of FAS are referred to as either having FAE or PEA. When available, data from such children are noted; otherwise, the results presented in this article refer to children diagnosed with FAS.

Children with histories of heavy prenatal alcohol exposure show evidence of changes in brain structure and function as well as a variety of behavioral effects presumably resulting from this insult to the brain. Most of the research conducted among alcohol-exposed children and adolescents has focused on either the structural or behavioral effects. Only recently have studies begun to demonstrate the relationship between the two areas that changes in brain structure could negatively affect behavior. This article summarizes the results of neuropsychological studies analyzing alcohol' s teratogenic (i. e. , damaging to the developing fetus) effects on behavior and of brain imaging studies analyzing alcohol' s effects on brain structure. It then highlights the existing connections between those two areas of research. For more extensive coverage of these topics, the reader is referred to review articles by Mattson and Riley (1998) and Roebuck and colleagues (998).

Generally, heavy prenatal alcohol exposure is associated with deficits in a wide range of areas of function, including both cognitive functioning (e. g. , general intellectual functioning, learning of new verbal information, and performance on visual-spatial tasks) and fine-and gross-motor performance. Neuropsychological studies have analyzed the cognitive impairment of children with histories of prenatal alcohol exposure. Although many of these studies have focused on children diagnosed with FAS, several analyses have included children with FAE or PEA. Importantly, many studies show that strong similarities exist between children with FAS and children with FAE/ PEA.

For example, studies of overall cognitive ability in FAS children typically report average IQ scores in the borderline range of functioning (i. e. , in the low 70s) , although they can range from intellectually deficient ((IQ scores less than 70) to average (IQ scores between 90 and 109) . Children with FAE or PEA also show deficits in IQ scores, although these deficits typically are not as severe as in the children with FAS (Streissguth et al. 1991; Mattson et al. 1997).

In addition to overall intellectual or cognitive deficits, researchers have evaluated a broad range of cognitive functioning areas in children with FAS, FAE, or PEA, including language skills, visual-spatial functioning, fine-motor behavior, nonverbal learning, and academic performance. In general, alcohol-exposed children both with and without FAS show significant impairments in all neuropsychological areas with few qualitative differences observed between the FAS and PEA/ FAE groups. Similarly, high levels of prenatal alcohol exposure are related to an increased risk for cognitive deficits across a range of functioning areas, which again can occur in children both with and without a diagnosis of FAS.

Learning and Memory

Both anecdotal information and results from animal studies have indicated that prenatal alcohol exposure can affect learning and memory. Studies of children with FAS generally have supported this observation, although the deficits in memory may not be as global as was once thought. For example, one study investigated verbal learning and memory in children with FAS and in non-alcohol-exposed control children (Mattson et al. 1996 b) . The study found that although the FAS children demonstrated some deficits in memorizing verbal information, these deficits resulted from difficulties with the acquisition of the information rather than with the ability to remember the information over time. Other studies also have revealed similar deficits in the acquisition of nonverbal information in alcohol-exposed children (Mattson and Roebuck in press) , suggesting that learning deficits occur in both verbal and nonverbal arenas and are likely to cause significant impairment in diverse areas of functioning. It is unclear, however, whether the degree of impairment for each child differs between the verbal and nonverbal areas of function.

Some studies suggest that children with FAS can perform well when memory function is tested in a different way, for example in tests of implicit memory a type of memory that is not under conscious control. When subjects successfully perform implicit memory tests, they may use information from previous tasks without being aware that they have done so. In one study, investigators showed children with FAS lists of words and asked the children to rate those words on likeability (Mattson and Riley 1999) . (This rating component served to enhance the children' s attention to the words.) Later in the testing session, the children were asked to complete partial words (e. g. , MO or SM) with the first word that came to mind (e. g. , MOUSE or SMILE) . The children were not reminded of the previous words nor prompted to remember them by the examiner. Nevertheless, both FAS and control children were more likely to complete the partial words with words from the previous task than with new words. These results indicated that both groups of children used implicit memory and that prior exposure helped them learn and memorize the words. Taken together, these findings suggest that although children with FAS may have significant impairments in learning new information, their overall memory function is complex and may not be as globally affected as was commonly thought. Nevertheless, specific aspects of memory may be affected by prenatal alcohol exposure.

Executive Functioning

The term "executive functioning" refers to a group of higher-level cognitive abilities, such as solving problems, thinking abstractly, planning ahead, and being flexible in one' s thought processes. These types of skills are independent of overall intellectual function and influence whether and in what manner a person can complete a task. Conversely, tests of other cognitive abilities tend to assess how well, or at what level, a person performs a skill (Lezak 1995).

Children with heavy prenatal alcohol exposure (both with and without FAS) have demonstrated impairments on executive functioning tasks (Kodi - tuwakku et al. 1995; Mattson et al. 1999) . Importantly, in these studies the children' s deficits in executive function were unrelated to their overall intellectual levels. This finding is supported by a recent study among adults with FAS or FAE, which found that the subjects deficits in executive functioning were greater than would have been predicted if they were related to overall IQ scores (Connor et al. in press) .

Deficits in executive functioning can have real-life implications for people prenatally exposed to alcohol. For example, people with heavy prenatal alcohol exposure may act without first considering the consequences of their behavior or they may have difficulties with activities that require problem solving or with planning a sequence of activities. These types of deficits may explain why children with heavy prenatal alcohol expo-sure, even those with average IQ scores, have difficulty succeeding in school.

Psychosocial Deficits and Problem Behaviors

Studies involving parent reports and interviews have suggested that alcohol-exposed children with or without FAS not only have cognitive deficits but also are at high risk for problem behaviors that can interfere with their participation in home, school, and social environments. For example, these children appear to be at increased risk for psychiatric disorders, trouble with the law, alcohol and other drug abuse, and other maladaptive behaviors (Streissguth et al. 1996) . Moreover, they are more likely than non-alcohol-exposed children to be rated as hyperactive, disruptive, impulsive, or delinquent (Roebuck et al. 1999; Mattson and Riley 2000) . Similarly, on measures of adaptive ability and skills necessary to perform age-appropriate daily living activities,

adolescents and adults with FAS often exhibit poor socialization and communication skills. In addition, the majority of these adolescents and adults display significant maladaptive behaviors (e. g. , impulsivity) and are less likely to be living independently (Streissguth et al. 1991; Thomas et al. 1998) . It is noteworthy that these problems occur in people prenatally exposed to alcohol whether or not they meet the criteria of FAS and occur to a greater extent than would be predicted by the person s general intellectual functioning or demographic factors.

Results from Brain Imaging Studies

The neuropsychological and behavioral deficits described in the previous section represent real-life manifestations of the effects of prenatal alcohol exposure. Although deficits on these measures are thought to provide evidence of underlying changes in brain structure or function, they represent only indirect measures of such brain changes. Alcohol' s direct effects on brain development were already noted in the earliest reports of FAS (Jones et al. 1973) , however, and autopsy studies of brains from people with FAS noted numerous and wide-spread brain abnormalities. Because these cases represented only the most severely affected children, it is problematic to generalize the findings to all people living with FAS. With the advent of numerous structural imaging techniques, such as magnetic resonance imaging (MRI) , and functional imaging techniques, such as electroencephalography (EEG) , positron emission tomography (PET) , and single photon emission computed tomography (SPECT) , however, researchers can now study the living brains of alcohol-affected children in a relatively noninvasive fashion.

Structural Brain Imaging

Imaging studies using MRI have revealed several differences between the brains of alcohol-exposed and non-exposed individuals. Consistent with the characteristic small head size, which is one of the diagnostic criteria for FAS, imaging studies show a decrease in the over-all size of the brain of FAS children (Roebuck et al. 1998) . To determine whether this size reduction results from global and diffuse alcohol effects on all brain areas or is limited to specific regions, researchers have assessed specific structures in proportion to overall brain size. This approach can determine whether specific, disproportion-ate reductions occur in some brain areas. These investigations have focused on several brain areas, including the basal ganglia, corpus callosum, cerebellum, and hippocampus (see figure 1) .

Basal Ganglia. The basal ganglia are a group of nerve cell clusters (i. e. , nuclei) , including the caudate nucleus, putamen, and globus pallidus. They are involved in motor abilities and cognitive functions, such as the executive functions described earlier. MRI studies have revealed that the basal ganglia are affected by heavy prenatal alcohol exposure and are disproportionately reduced in volume in children with FAS and PEA. More de-tailed examination of the components of the basal ganglia found that the reductions are not uniform and that the caudate nucleus appears to account for most of the size reduction in the basal ganglia (Mattson et al. 1996 a ; Archibald et al. 2001) .

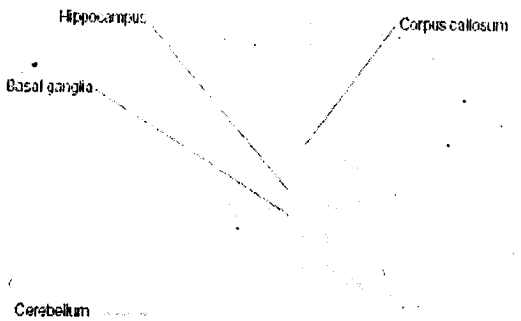


Figure1: Brain areas affected by prenatal alcohol exposure.

The caudate nucleus is the portion of the basal ganglia involved in cognitive functions. For example, skills such as the ability to shift from one task to another, inhibition of inappropriate behavior, and spatial memory, which are impaired in people with prenatal alcohol exposure, have been related to the basal ganglia in other populations, such as patients with Huntington' s disease (Mattson et al. 1996 a ; Mattson and Riley 1999; Archibald et al. 2001) . Accordingly, it is possible that the reductions in the caudate nucleus account for some of the cognitive deficits seen in people with prenatal alcohol exposure. This hypothesis is particularly appealing because the caudate nucleus also has extensive neural connections to the frontal lobes of the brain, which traditionally are thought to mediate higher cognitive and executive functions.

Corpus Callosum. The corpus callosum is a large bundle of nerve fibers connecting the two hemispheres of the brain, thereby allowing the left and right sides of the brain to communicate with one another. Corpus callosum abnormalities have been linked to deficits in attention, intellectual functioning, reading, learning, verbal memory, and executive and psychosocial functioning, all of which are impaired in alcohol-exposed people. MRI studies and autopsy reports suggest a vulnerability of the corpus callosum to prenatal alcohol exposure; such studies found that people with FAS exhibit abnormalities ranging from a thinning to complete absence (i. e. , agenesis) of the corpus callosum (Roebuck et al. 1998) . When specific regions of the corpus callosum were analyzed, researchers found that the front-most area the genu and the back-most areas the isthmus and splenium were disproportionately reduced in size (Riley et al. 1995) . Moreover, the rate of agenesis of the corpus callosum may be higher in people with FAS than with any other developmental disorder (Jeret and Serur 1991; Riley et al. 1995) .

Recently, researchers analyzed in more detail the shape and location of the corpus callosum of FAS and PEA children as well as of control children (Sowell et al. 2001) . The study not only confirmed that the corpus callosum was reduced in size, specifically in the splenium, but that it was also significantly displaced in three-dimensional space (see figure 2) . After equalizing all brains for brain size and the location of other structures located along the midline of the brain, the average location of the corpus callosum for the alcohol-exposed children was compared with the average location for the control children. This analysis found that the corpus callosum in the alcohol-

exposed children was displaced compared with the control children, with the biggest differences in the area of the isthmus and splenium, both of which are located in the back of the corpus callosum. Furthermore, this corpus callosum displacement was highly related to the children's performance on a verbal learning task. In other words, children with greater displacement exhibited more substantial performance impairments.

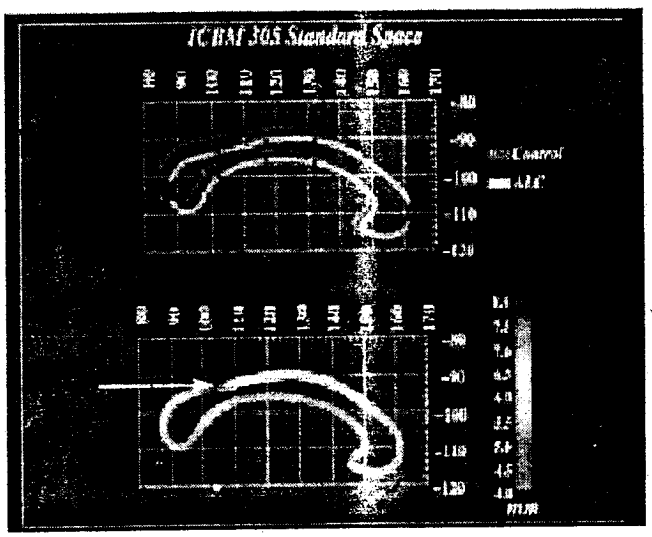


Figure 2 (Top) Average outlines of the corpus callosum (i. e. , the bundle of nerve fibers connecting the brain s right and left hemispheres) in alcohol-exposed subjects (ALC) and non-alcohol-exposed control subjects. The corpus callosum is oriented so that the front of the head is to the right and the back of the head is to the left. The figure shows that the corpus callosum of the ALC is displaced in three-dimensional space compared with that of the control subjects, with the greatest displacement occurring in the isthmus and splenium at the back of the corpus callosum. (Bottom) A map showing the average displacement in millimeters between the ALC and the control subjects. Darker area (see arrow) indicates greater displacement between the two groups. Greater displacement is associated with greater performance impairment in certain tasks.

SOURCE: Figure courtesy of Dr. Elizabeth Sowell.

Cerebellum. Another area of the brain that is affected by prenatal alcohol exposure is the cerebellum, which is involved in both motor and cognitive skills and is located at the base of the brain. For example, damage to the cerebellum has been implicated in learning deficits as well as in balance and coordination, all of which are impaired by prenatal alcohol exposure. A recent study found that the overall volume of the cerebellum was disproportionately reduced relative to overall brain size in people with FAS compared with control subjects (Archibald et al. 2001) . These findings partially replicate previous reports of reduced cerebellar size in FAS and PEA children (Sowell et al. 1996) . In addition to the overall reductions in the size of the cerebellum, studies conducted in both humans and animals suggest that a specific region of the cerebellum the anterior portion of the cerebellar vermis is particularly affected by alcohol exposure before or shortly after birth[1] (Goodlett et al. 1990; Sowell et al. 1996) ([1]The studies in animals, primarily rodents, were

conducted shortly after birth, a period that corresponds to the third trimester of gestation in humans with respect to brain development).

Hippocampus. The hippocampus is a structure that lies deep within the temporal lobe of the brain and is involved in memory. Although the precise function of the hippocampus in specific aspects of memory is controversial, it probably plays a role in the consolidation of memories. For example, in adults with hip pocampal damage, the most obvious effect is a loss of the ability to store new memories (i. e. , anterograde amnesia) . Animal studies have long suggested that this area is affected by prenatal alcohol exposure (Berman and Hannigan 2000) . Moreover, an MRI study of children with FAS documented volume asymmetries in the hippocampus, with the absolute volume of the hippocampus in the left temporal lobe smaller than that of the corresponding area in the right temporal lobe (Riikonen et al. 1999) . Although such differences also exist in adults with normal neurological function, the extent of the asymmetry was greater in the FAS children than in the control children. Conversely, another study found that the hippocampus was less affected than some other brain regions in FAS children (Archibald et al. 2001) . In that study, the reduction in the volume of the hippocampus was proportionate to the reduction in overall brain size, whereas other brain areas showed greater reductions in volume.

Behavioral studies have supported the hypothesis that the hippocampus might be affected in children with prenatal alcohol exposure. For example, people with prenatal alcohol exposure have been reported to exhibit deficits in spatial memory as well as other memory functions associated with the hippocampus (Uecker and Nadel 1996) . However, the memory deficits in alcohol-exposed children require more detailed study and should be integrated with information about the integrity of the hippocampus. This issue also points out a limitation of structural imaging, namely that this approach only determines the size of a particular brain structure but does not indicate whether the structure is functioning correctly. To determine how a particular brain area functions under different conditions and whether these functions are altered by prenatal alcohol exposure, researchers are turning to functional brain imaging approaches, discussed in the following section.

Functional Brain Imaging

Functional imaging techniques allow researchers to study how the brain works, either at rest or when presented with a task. Because some functional techniques are more invasive or technically difficult to conduct with children, only a small number of studies using these techniques have been conducted in FAS children. The most commonly used technique in these studies is electroencephalography (EEG) .

EEG. The EEG measures the brain s spontaneous electrical activity by recording signals from the brain with electrodes placed on the scalp. These signals can be visualized as waves with specific frequencies, such as alpha, beta, and theta waves. Early studies on infants suggested that EEG may be a sensitive measure of changes in brain function resulting from prenatal alcohol exposure (Ioffe and Chernick 1990) . More recent studies of children and adolescents with FAS found that approximately one-half of these subjects had clinically suspect EEG readings (Kaneko et al. 1996 b) . Furthermore, subjects with FAS exhibited reductions in the power or strength of the alpha frequencies, which is the predominant type of activity when a person is relaxed. These reductions were seen predominantly in the left hemisphere and suggest immature brain activity.

Using similar techniques, it is possible to measure the brain s electrical response to specific sensory stimuli (i. e. , event-related potentials) . These event-related potentials can be visualized as spikes in certain brain waves. One of these spikes is called P300, because it typically occurs

approximately 300 milliseconds after the stimulus; it appears to reflect the cognitive aspects of information processing. Using EEG analyses, researchers found that the P300 spikes occur with a delay (i. e. , have a prolonged latency) in a certain brain region, the parietal cortex, in FAS children (Kaneko et al. 1996 a , b) . This finding suggests that children with FAS may have deficits in information processing. Thus, electrophysiological measurements are powerful tools in the study of FAS; future studies combining them with localizing brain imaging may provide further information about brain function.

PET. The PET technique allows researchers to monitor the activity of specific brain regions by generating images of metabolic or physiologic processes, such as blood flow or breakdown of sugar molecules, in the tissue. For this approach, the subject is injected with small amounts of radioactive material so that brain activity in the region of interest can be measured while the subject performs a task. These tasks can range from the simple, such as moving a finger, to the complex, such as recalling information. One PET study assessed brain activity in adolescents and adults with FAS who showed no severe mental retardation (i. e. , who were high functioning) . The study revealed reduced metabolic activity in the caudate nucleus and in the thalamus when the subjects were at rest (Clark et al. 2000) . These functional data support the structural data, such as the reduced size of the caudate nucleus, suggesting that subcortical brain regions may be especially sensitive to prenatal alcohol insult.

SPECT. The SPECT technique is similar to PET, and although it is less powerful, it is more commonly available. However, only one study of FAS children has used this technique. In that study, the investigators found that FAS children exhibited similar metabolic activity in both hemispheres of the brain (Riikonen et al. 1999) . Normally developing children, in contrast, show greater resting activity in the left hemisphere than in the right hemisphere. These results are consistent with the EEG findings described above and may support verbal or language deficits in FAS children.

Functional Magnetic Resonance Imaging (fMRI) . The newest functional technique used to study activity in the living brain is fMRI. Its main advantage is that it is less invasive than PET or SPECT because it does not involve injecting the subject with radioactive substances; moreover, it is more commonly avail-able. Similar to PET and SPECT, fMRI allows researchers to visualize brain reports exist of fMRI studies in people with prenatal alcohol exposure; however, such studies are currently underway. One preliminary report described an fMRI study of working memory using information held in memory for a short period of time in four adults with FAS or FAE (Connor and Mahurin 2001) . The study revealed activation in an area called the dorsolateral prefrontal cortex in the FAS subjects but not in control subjects. This area is thought to play a role in higher cognitive functions, such as the executive functions described above. This result suggests that the working memory task was more difficult for the alcohol-exposed subjects and required greater involvement of this region of the frontal lobe compared with the control subjects.

New Image Analysis Techniques

In addition to improvements in brain imaging techniques, new ways of analyzing the data obtained with these techniques are providing scientists with insights about the damaging effects of prenatal alcohol exposure. One of those techniques is called brain mapping. It uses a structural MRI analysis but provides greater visualization of all brain structures. As a result, researchers can study the whole brain at once, rather than focus on specific brain regions, and therefore can localize brain abnormalities more easily than with previous techniques.

Sowell and colleagues (2001 b) have used the brain mapping technique to analyze and compare brain images of people with FAS or PEA and non-alcohol-exposed control subjects. Consistent with the results of Archibald and colleagues (2001) , the study detected disproportionate reductions in the brain s white matter, which contains the nerve cells extensions (i. e. , axons) that connect nerve cells with each other. Conversely, the brain' s gray matter, which contains the nerve cell bodies, showed reductions that were not as great. In addition, the parietal lobe, which is involved in visual-spatial processing and in the integration of sensory information, appeared to be especially susceptible to alcohol' s effects. Thus, once overall brain size was accounted for, both the volume (Archibald et al. 2001) and the density (Sowell et al. 2001 b) of white matter in this region were significantly reduced (see figure 3) . Conversely. the gray matter density in the parietal cortex was significantly increased (Sowell et al. 2001 b) . These findings lend additional support to the suggestion that alcohol s effect on the developing brain is not global in nature but, rather, affects specific brain regions selectively.

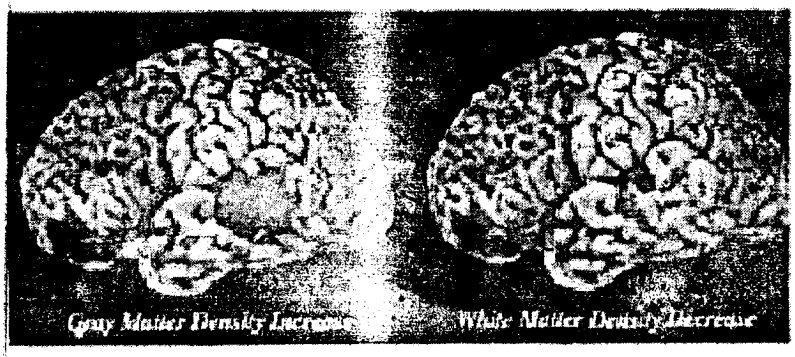


Figure 3 Changes in brain tissue density in children with heavy prenatal alcohol exposure. A representative brain is shown with the back of the brain facing the reader s right. Brain-mapping studies detected areas of increased gray matter density (shown in yellow on the left) as well as areas of reduced white matter density (shown in red on the right) in the parietal lobe.

SOURCE: Figure courtesy of Dr. Elizabeth Sowell.The studies reviewed here provide clear evidence that both brain structure and brain function are affected by heavy prenatal alcohol exposure. More recent studies indicate that the effects of this alcohol exposure are not global in nature but seem to affect certain areas more than others in both the neuropsychological and neuroanatomical arenas. Continuing studies are focusing on the relationship between neuropsychological and neuroanatomical data and hopefully will result in a clearer picture of the strengths and weaknesses of people with a history of heavy prenatal alcohol exposure, thereby allowing researchers and clinicians to develop more targeted and effective intervention approaches.

Most women with active epilepsy need treatment with antiepileptic drugs during pregnancy. Antiepileptic drugs are also frequently used for other indications, such as migraine, pain syndromes, and psychiatric disorders, which are prevalent among women of childbearing age. Possible teratogenic effects of antiepileptic drugs are therefore of wide concern and the risks imposed by the drugs must be weighed against the risks associated with the disorder being treated. Adverse drug effects on the fetus can present as fetal loss, intrauterine growth retardation, congenital malformations, impaired postnatal development; and behavioural problems. For optimum use of antiepileptic drugs in women of childbearing age and rational management of epilepsy during

pregnancy, a thorough understanding of the teratogenic effects of antiepileptic drugs and knowledge of the differences in risks between various treatment options are needed.

10.4 CARCENOGENIC AND MISCELLANEOUS EFFECTS

A carcinogen is a substance that is capable of causing cancer in humans or animals. If a substance is known to promote or aggravate cancer, but not necessarily cause cancer, it may also be called a carcinogen. Though there are many things that are believed to cause cancer, a substance is only considered carcinogenic if there is significant evidence of its carcinogenicity.

A carcinogen may act on deoxyribonucleic acid (DNA), causing dangerous changes, or it may work to increase the rate of cell division. This change in cell division may work to increase the probability of DNA changes. Some carcinogens promote the development of cancer in other ways as well.

It is important to note that carcinogens don't lead to cancer after every exposure. Some cause cancerous changes following high-level, prolonged exposure, while others may cause damage at lower levels and shorter exposure periods. Furthermore, an individual's unique genetic makeup may influence the body's response to a carcinogen.

Testing human subjects for carcinogenic behavior and properties is considered unethical, not to mention hazardous to the health of the test subjects. As such, animals are often used for carcinogen testing. Additionally, cell cultures from both humans and animals are used in testing. Scientists also consider the effects of substances at the molecular level in determining whether or not they are carcinogenic. Evidence of links between exposure to substances and the development of cancer is also considered.

Many substances have been identified as carcinogenic. Some commonly known carcinogens include asbestos, radon, certain pesticides, arsenic, and tobacco smoke. Smokeless tobacco is a known carcinogen as well.One major carcinogen originates from something vital to life. The sun emits ultraviolet rays that are carcinogenic.

10.5 SELF ASSESSMENT QUESTIONS

1.Explain Carcenogenic importance in detail?

2.Describe skeletal Toxicity?

3. explain the prevention and treatment of Skeletal toxicity?

10.6 REFERENCE BOOKS

1.Food toxicology part A.principles and concepts by jose M.concon marcelDekkher Inc,NEW YORK,1988.
2. Journal of Medical Microbiology

3. Current science.

V.CHINNARI HARIKA

Lesson – 11

INCIDENCE OF MASHROOM POISONING

11.0 Objective

After reading this chapter, you should be able to:

- Know the effect of Food toxins on Human health
- To create awareness regarding Mashroom toxicity and harmful side effects in human beings.
- To educate the community through nutrition Knowledge.

Structure

11.1 INTRODUCTION

Mushroom poisoning (mushroom toxicity) occurs after the ingestion of toxins synthesized by the mushrooms themselves. Mushrooms are the fruiting bodies of a group of higher fungi that have evolved contemporaneously with plants for millions of years. They are widely distributed throughout the world, and thousands of species have been identified.

About 100 species of mushrooms are poisonous to humans, and 15-20 mushroom species are lethal when ingested. No simple rule exists for distinguishing edible mushrooms from poisonous mushrooms. In more than 95% of mushroom toxicity cases, poisoning occurs as a result of misidentification of the mushroom by an amateur mushroom hunter. In less than 5% of the cases, poisoning occurs after the mushroom is consumed for its mind-altering properties.

The severity of mushroom poisoning may vary, depending on the geographic location where the mushroom is grown, growth conditions, the amount of toxin delivered, and the genetic characteristics of the mushroom. Boiling, cooking, freezing, or processing may not alter the mushroom's toxicity.

Variations in clinical effects may depend on an individual's susceptibility and on the presence of confounding factors such as contamination or coingestion. In general, children, older persons, and persons with disabilities are at greater risk for the development of serious complications with mushroom poisoning than healthy young adults are.

Mushroom poisoning in children is an infrequent but perennial problem for parents and clinicians. Parental anxiety is generally high because of fears of unknown or untoward effects. The challenges for clinicians are to identify such poisonings, to discern whether poisoning has taken place, to order appropriate diagnostic studies, and to prescribe reasonable therapy. The varied nature of mushroom toxicities, their ubiquitous distribution, and the relative infrequency of the ingestions make these challenges difficult to meet.

Mushroom poisoning (also known as mycetism) refers to harmful effects from ingestion of toxic substances present in a mushroom. These symptoms can vary from slight gastrointestinal discomfort to death. The toxins present are secondary metabolites produced in specific biochemical pathways in the fungal cells. Mushroom poisoning is usually the result of ingestion of wild mushrooms after misidentification of a toxic mushroom as an edible species. The most common reason for this misidentification is close resemblance in terms of colour and general morphology of the toxic mushrooms species with edible species. Even very experienced wild mushroom gatherers are upon rare occasion poisoned by eating toxic species, despite being well aware of the risks, through carelessness.

11.2 INCIDENCE OF MASHROOM POISONING

As soon as mushrooms start appearing in the woods the newspapers begin to report stories about mushroom poisoning. IN spite of the warning provided by these news items, year after year there are accidental poisoning caused by mushrooms. Yet there is no family that does not read at least one newspaper, and there is hardly anyone unaware of these reports especially in the age of Internet. If these accidents continue to happen, with a frequency proportional to the number of mushrooms grown during a given season, it means that most people do not benefit from newspaper and internet reports: they get a little frightened but its soon forgotten and that is all. Most people do not pay attention to the shape and characteristics of the dangerous mushrooms and continue to rely blindly n their own intuition or on some arbitrary and misleading homemade tests.

Many human life could be saved if an addition to the reports the reports of poisoning accidents, the newspapers and internet articles publishers publish the description of the mushrooms responsible for poisoning, and list of first aid procedures to help victims of such accidents. This would be a relatively easy task since, in comparison with several hundred species of edible mushrooms; there are only about a dozen really dangerous species. The publishers willing to carry such a service for its readers would probably add to its prestige and sales

11.3 TYPES OF MASHROOM POISONING

Deadly Mushrooms:

The most dangerous and the one responsible for most of the deaths resulting from mushroom poisoning is the Amanita phalloides, which is found very frequently in woods, in summer and autumn, especially in its forms or varieties viridisandviresens. The lethal dose for a man of medium size is twenty grams of freshAmanita Phalloides. Mushrooms as poisonous as Amanita

Phalloides areAmanita Bisporigera, Amanita verna, Amanita Virosa, Lepiota Helveola andCortinarius orellanus; these species are less common than Amanita Phalloides and completely absent in some regions.

Some deaths have also been attributed to Gyromitra esculenta, in spite of the fact that its name, esculenta, means edible. Once exsiccated, this mushroom is completely harmless; but when it is fresh it is not well tolerated by everybody, especially if it is only slightly cooked, consumed with the water in which it has been cooked, and eaten in a large quantity. For some people it is particularly harmful when eaten in successive meals. But over the years it ha been sold in many markets and consumed without harm by many people. However, one should pay attention to how it is cooked. It should be parboiled in water first then cooked well. The juice produced by the mushroom while cooking should not be used; it is also advisable to eat only small quantities of it and wait at least four days before eating more. The symptoms of poisoning from eating the deadly amanitas become evident only some time after eating, from 8 to 40 hours. It begins with vomiting, diarrhea, heavy perspiration, and consequent insatiable thirst; the feet and hands become cold, calf cramp develops; the eyes look deep sunk, the face is drawn and pale, and sometimes yellow, as in jaundice. This is followed by a state of anxiety, deep prostration and an imperceptible pulsebeat; and eventually rattling, paralysis, convulsive spasms, death. All this can last from 10 to 20 days. Sometimes, during the first 48 hours, there might be a slight improvement, but then the illness continues its inexorable course. Prompt and proper care can save adult and robust people; but it is much more difficult to save children. Poisoning symptoms from Lepiota helveola, from Vortinarius orellanus and Gyromitra esculenta are fundamentally similar to those caused by the deadly amanitas; the cure is also essentially the same. The symptoms of intoxication due to Cortinariusorellanus become evident only very late, from 3 to 14 days, after eating this mushroom and although very similar to those caused by the Amanita phalloides, there are a few differences.

The cure for which only a physician can assume full responsibility, aims to eliminate the poisonous substances from the organism through an evacuation of the digestive system and a stimulation of kidney activity; it aim to calm the pain and sustain the general condition, especially the heart. The cure should specifically fight against suffocation, nervous depression, and organism dehydration; it must protect kidneys and liver, which are directly threatened y the toxic substances of the mushrooms. Choline,and thioctic acid are found very useful in protecting the live in cases of mushroom poisoning.

Mushrooms harmful to the nervous system: Amanita musaria, Amanita pantherina, Inocybe Patouillardi, Clytocibe dealbata, and species related to them. In several areas of Italy, France, and Russia, Amantita muscaria is consumed regularly without harm, but one must consider that this might be due partly to the fact that often this mushroom is eaten after first being treated, which reduces its poison leel, and partly to the fact that the amount of toxic principle contained in this mushroom varies depending on the area and the season. Still we believe it better to avoid this mushroom. Luckily, the symptoms of intoxication caused by this group of mushrooms show up quickly: from half an hour to four hour after eating them. The principle symptoms are: abundant secretion of saliva, of nasal mucus, and tears; slowing down of the pulse and a sensation of suffocation. In these instances the doctor should administer a good laxative, some diuretic beverages in large quantities, including tea and coffee but not alcoholic beverages, and sometimes an injection of atropine if the subject is particularly weak.

Russula emetic is another mushroom containing substances toxic to the nervous system; but since its ingestion causes immediate vomiting, the subject eliminate the toxic principles and

there are no other consequences. At the same time it is so acrid to the taste that it is almost important to eat a large and dangerous amount of it. A mushroom that is known to cause serious intoxication and also lethal, although rarely, is Entoloma lividum. Those that can cause a strong intoxication include:Tricholoma pardimum, virgatum, and groanense, Clitocybe olearia, Boletus Satanas, and purpureus, Clavaria Formosa, and pallid, psalliota xanthoderma can cause a weak intoxication. The less these mushrooms are cooked the more serious is the resulting intoxication. Within one hour from ingestion the subject experiences nausea, colic, vomiting, diarrhea, and fainting. The cure is essentially identical to that used for poisoning by Amanita muscaria.

Almost all mushrooms belonging to the genera Peziza, Morchella, Helvella, those related to them and a few other species, such as Amanita rubescens andRhodopaxillus nudus. The cure is essentially identical to that for poisoning byAmanita muscaria. Sometimes the symptoms of the poisoning are slight and disappear spontaneously in a short time. Several other mushrooms belonging to the genera Russula and Lactarius are also toxic when eaten raw; but their taste, acrid and bitter, is sufficient to prevent people from eating them. Once cooked, they often lose not only their disgusting taste but also their toxic properties. Sometimes fermentation is sufficient to destroy the toxic substances: this method is used in different parts of East Europe and the people in those places eat, after the mushrooms are fermented, not only Lactarius torminosus, Lactarius piperatus, and Lactarius plumbeus, which are acrid when raw, but alsoLactarius rufus, which, when eaten raw, has a taste that can only be described as a very hot fire. The collector of edible mushrooms could follow a simple rule when picking russulas and lactarii, and that is to discard, when in doubt, all the specimens with acrid or bitter flavor, and to consider edible all the others, provided they are not too ripe or infested by larvae.

This is an edible mushroom which, however, can be quite harmful to some people, especially when one drinks alcoholic beverages or sometime even coffee or tea, while or after eating it. The most characteristic symptom is a deep reddening of the face, together with an acceleration of the pulse, a loss of strength and a cooling of hands and feet. These disturbances are not dangerous; they last only a short time and disappear without further consequence.

The proper cure requires the knowledge of the species of mushroom ingested by the poisoned person. Often the poor victim does not know their name himself. Therefore, at times, to identify the suspect mushroom it is necessary to examine the parts discarded in the garbage can, the remains left after the meal or even the vomit itself. As for vomiting, it is useful to remember that it is absolutely useless to induce it, if more than 8-10 hours have elapsed since the ingestion of the mushrooms, because the mushrooms are probably no longer in the stomach but in the intestine.

While waiting for the doctor, one could administer a laxative; but if the poisoned person has strong visceral pains, use castor oil instead of a salt type laxative, and use only a moderate dose: 30 grams for an adult, 15 grams for a child. You can administer beverages, even in large doses, such as milk, water with sugar, water with salt; or even tea or coffee, but in moderate doses. Keep the patient well covered quiet and in a warm place. If the mushroom ingested id the Amanita phalloides, take the patient to the hospital immediately, but in the meantime even before he reaches the hospital give him, every half an hour, a teaspoon of salt dissolved in a glass of water.

11.4 TOXICOLOGY OF MARINE FOODS

Aquatic toxicology is the study of the effects of manufactured chemicals and other anthropogenic and natural materials and activities on aquatic organisms at various levels of organization, from subcellular through individual organisms to communities

and ecosystems. Aquatic toxicology is a multidisciplinary field which integrates toxicology, aquatic ecology and aquatic chemistry.

This field of study includes freshwater, marine water and sediment environments. Common tests include standardized acute and chronic toxicity tests lasting 24–96 hours (acute test) to 7 days or more (chronic tests). These tests measure endpoints such as survival, growth, reproduction, that are measured at each concentration in a gradient, along with a control test. Typically using selected organisms with ecologically relevant sensitivity to toxicants and a well-established literature background. Environmental toxicology has been and continues to be an important discipline (e.g., single-species testing for screening purposes). However, ecological toxicology (ecotoxicology – more realism in tests, test species and exposures) is required for predicting real world effects and for site-specific assessments. Ecotoxicology and ecology have shown similar developmental patterns over time; closer cooperation between ecologists and toxicologists would benefit both disciplines. Ecology can be incorporated into toxicology either extrinsically (separately, e.g., providing information on pre-selected test species) or intrinsically (e.g., as part of test species selection) – the latter is preferable. General guidelines for acute and chronic testing and criteria for species selection differ for ecotoxicology and environmental toxicology, and are outlined. An overall framework is proposed based on ecological risk assessment (ERA), for combining ecology and toxicology (environmental and ecological) for decision-making. Increased emphasis on ecotoxicology represents a shift from reductionist to holistic approaches.

11.5 MOLLUSKS

All land snails are hermaphrodites, producing both spermatozoa and ova. In other words, each individual is both male and female. Some freshwater snails, such as Apple Snails, and marine species, such as periwinkles, have separate sexes; they are male and female. Most snails can mate when they are around 1 year old. Prior to reproduction, most land snails perform a ritual courtship before mating. This may last anywhere between two and twelve hours. Prolific breeders, pulmonate land snails inseminate each other in pairs to internally fertilize their ova. Each brood may consist of up to 100 eggs.

Pulmonate land snails and slugs have a reproductive opening on one side of the body, near the front, through which the outer reproductive organs are extruded so that exchange of sperm can take place. After this, fertilization occurs and the eggs develop.

Garden snails bury their eggs in shallow topsoil primarily while the weather is warm and damp, usually 5 to 10 cm down, digging with their foot. Egg sizes differ between species, from a 3 mm diameter in the grove snail to a 6 cm diameter in the Giant African Land Snail. After 2 to 4 weeks of favorable weather, these eggs hatch and the young emerge. Snails may lay eggs as often as once a month. In bivalves, the sexes are usually separate, but some hermaphroditism is known. Bivalves practice external fertilization. External fertilization is a form of fertilization in which a zilo cell is united with an egg cell external to the body of the female. Thus, the fertilization is said to occur "externally". This is distinct from internal fertilization where the union of the egg and sperm occur inside the female after insemination through copulation.

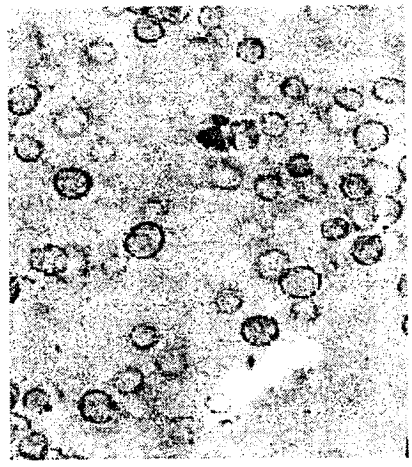
In sexual reproduction, there must be some way of getting the sperm to the egg. Since sperm are designed to be mobile in a watery environment, aquatic molluscs can make use of the water in which they live. Eggs and sperm are simultaneously shed into the water, and the sperm swim through the water to fertilize the egg in a process known as broadcast fertilization.

11.6 FISH AND MARINE ALGAE

The Sailfin/Algae Blenny is notable for its unique looks and winning personality. With a mottled tan color, oversized eyes, and typical body shape of the Blenniidae family, *Salarias fasciatus* adds visual appeal to any marine aquarium. But despite its tendency to grow to an impressive 5" in size in the home aquarium, the Sailfin/Algae Blenny remains peaceful, active and, therefore, fun to observe.

Also referred to as the Jewelled Rockskipper, Lawnmower, or Rock Blenny, the Sailfin/ Algae Blenny is native to the reefs across the Indo-Pacific. Like other Blennies, the Sailfin/Algae Blenny will perch on live rock, hide in caves, hop across the substrate, and graze onmicroalgae. Therefore, it requires a larger system with a variety of live rock and rockwork. The Sailfin/Algae Blenny is peaceful towards other fish unless the tank mate is similar in shape or appearance to the blenny. It is best to house the Sailfin/Algae Blenny singly unless kept in a larger aquarium and the two are a mated pair. It is known to nip at small-polyped stony coral and clam mantles.

The Sailfin/Algae Blenny does best in well-established aquariums with large amounts of natural algae to feed on. The diet can be supplemented with vegetable matter, spirulina, and herbivore preparations. Marine algae are among the most ancient members of the plant kingdom, and vital components of the ecosystem of marine life. These plants are abundant in coastal areas, usually anchoring themselves to a hard surface using specialized "holdfast" structures. These structures are not true roots, and there are also no true shoots, leaves, seeds, water-conducting tissues, or flowers. Species can range in size from very small (3 -10 microns) to very large (over 200 feet long) and some can grow more than 10 inches per day. The three best known types of marine algae are red algae (Rhodophyta), green algae (Chlorophyta and others) and brown algae (Phaeophyceae). In the Gulf of Mexico, marine algae play an irreplaceable role in maintaining marine life and habitats. In reef environments, some species of red algae "exceed corals in importance as reef building organisms." Hence, the term "tropical reef" is sometimes used instead of "coral reef" to reflect this diversity.



- Zooxanthellae -

Marine algae often live alongside sea gasses which have flattened leaves that allow easy attachment for the algae. These two types of plants combine to create diverse ecosystems. Marine animals

use these mats of algae and seagrass to rest, spawn, feed, and hide from predators. Examples of such animals are pipefish and snapper, shrimps and crabs,and tiny snails and clams.

This brown alga lends its name to the Sargasso Sea, a region in the North Atlantic Ocean between North America and Africa where large mats of *Sargassum* are concentrated. Some *Sargassum* species, like that in the Sargasso Sea, never anchor; other species attach to hard surfaces in shallow waters, near coral reefs and in mangrove thickets. Sargassum can be considered a habitat in addition to a species because fish and other animals use it as a refuge, as is true for other marine plants. There are actually several species of animals – crabs, shrimps, snails, and even a nudibranch – that live nowhere else. Since the oil spill, oily patches of *Sargassum* have been reported washing ashore on Gulf coastlines. The animals that take refuge in these plants will likely die from either the physical effects of the oil or from ingesting the oily residue that clings to the algae.

Some marine algae are necessary for coral growth. Many reef-building coral species have algal cells called zooxanthellae that live within its tissues in a symbiotic relationship. The zooxanthellae photosynthesize and provide supplemental energy to the coral. "Bleached" corals that have lost their zooxanthellae (through disease or stress) often die as a result. Algae can also be detrimental to coral growth. If marine algae are allowed to physically overgrow the coral, shading it from sunlight, the zooxanthellae can die or leave the coral, in turn putting the coral at risk. Overgrowth of algae can be caused by increased nutrients in the water or by decreased populations of animals (such as sea urchins) that normally graze upon the algae.

Algae are vitally important to marine and fresh-water ecosystems, and most species of algae are not harmful. Algal blooms can deplete the oxygen and block the sunlight that other organisms need to live, and some can produce toxins that are harmful to the health of the environment, plants, animals, and people. Harmful algal blooms have threatened beaches, drinking water sources, and even the boating venue for the 2008 Olympic Games in Beijing, China. Cyanobacteria (blue-green algae) and red tides are examples of algae that can bloom and produce toxins that may be harmful to human and animal health. HABs can occur in marine, estuarine, and fresh waters, and HABs appear to be increasing along the coastlines and in the surface waters of the United States, according to the National Oceanic and Atmospheric Administration (NOAA). HSB epidemiologists have led a number of studies to investigate the public health impacts of blue-green algae blooms and Florida red tide. The studies have demonstrated that there is the potential for exposure to potent HAB-related toxins during recreational and occupational activities on water bodies with ongoing blooms.

Although scientists do not yet understand fully how HABs affect human health, authorities in the United States and abroad are monitoring HABs and developing guidelines for HAB-related public health action. The U.S. Environmental Protection Agency (EPA) has added certain algae associated with HABs to its Drinking Water Contaminant Candidate List. This list identifies organisms and toxins that EPA believes are priorities for investigation. Many states regularly experience harmful algal blooms (HABs), and state public health departments are often are asked to provide guidance about HAB-associated human and animal illnesses. HSB subject matter experts help states to develop their public health responses to HAB events, including providing outreach and education materials and assessing exposure and the potential for health effects.

11.7 SUMMARY

To prevent mushroom poisoning, mushroom gatherers need to be very familiar with the mushrooms they intend to collect as well as with any similar-looking toxic species. In addition, edibility of mushrooms may depend on methods of preparation for cooking. Collectors also need to be well aware that edibility or toxicity of some species varies with geographic locationAlgal blooms occur in natural waters used for drinking and/or recreation when certain types of microscopic algae grow quickly in water, often in response to changes in levels of chemicals such as nitrogen and phosphorus from fertilizer, in the water.

11.8 SELF ASSESSMENT QUESTIONS

1.Explain marine toxicity importance in detail?

2.Describe Mollausk Toxicity?

3. Explain the types of Mahrooms toxicity inbrief?

11.9 REFERENCE BOOKS

1.Food toxicology part A.principles and concepts by jose M.concon marcelDekkher Inc, NEW YORK,1988.

2. Journal of Medical Microbiology

3. Current science.

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Lesson – 12

DERIVED FOOD TOXICANTS

12.0 Objective

After reading this chapter, you should be able to:

- Know the Derived Food Toxicants
- Understand the Formation of Toxicants

STRUCTURE

12.1.INTRODUCTION

Foods derived from sources not previously used as human foods must be evaluated for safety as they may contain toxins, contaminants and anti-nutritional factors. Conclusions from these assessments permit appropriate risk management measures to be taken. Toxicological testing is required for substances of unknown safety that may be introduced to the food supply. For foods that have no history of safe use, it may be difficult to identify individual components which are novel in the context of human consumption in the absence of a traditional counterpart.

Where it is not possible to identify novel components of the food, a case-by-case approach should be used to determine the appropriate toxicological tests to be carried out on the food. The history of the organism from which the food is derived as a source of toxins or antinutrients and a chemical analysis of its components will be considerations in determining requirements for toxicological testing. Depending on these determinations, conventional studies of toxicity, including chronic toxicity, developmental toxicity, genotoxicity or carcinogenicity, may need to be performed on the final food product or its components as appropriate.

A whole food presents some challenges due to the potential for inducing nutrition imbalances when the food is incorporated into the diet at high concentrations. In addition, toxicology studies on novel foods are used to reach a conclusion as to whether the food is safe to consume under expected consumption patterns, rather than to derive a quantitative limit such as an acceptable daily intake in the manner used for simple chemicals like food additives.

The toxicity of a substance is its ability to cause harmful effects. These effects can strike a single cell, a group of cells, an organ system, or the entire body. A toxic effect may be visible damage, or a decrease in performance or function measurable only by a test. All chemicals can cause harm. When only a very large amount of the chemical can cause damage, the chemical is considered to be practically non-toxic. When a tiny amount is harmful, the chemical is considered to be highly toxic. When Good Fat Turns Bad, provides a revolutionary new understanding of our current obesity epidemic by showing that it can be viewed as a form of cancer to be treated using the clinically proven pathway to change the expression of your genes using food as a drug to rid your body of toxic fat.

The first signs of toxic fat are the accumulation of excess body fat. In fact, obesity can be viewed as a form of "cancer" that is driven by toxic fat. However, only when this toxic fat begins to spread into the bloodstream does it begin to attack your other organs leading to early development of chronic disease. This is why some people can be overweight and be quite healthy, while others can be of normal weight and be quite sick. What ultimately determines your state of wellness is not your weight but the levels of toxic fat in your blood.

A key objective of the Toxic Substances Hydrology (Toxics) Program is to understand the physical, chemical, and biological processes that control contaminant transport in surface water. Many Toxics Program scientists participate in multidisciplinary investigations of the reactive and non-reactive transport of contaminants in rivers and streams. A part of many of these investigations is to develop simulation models to predict contaminant transport in streams. Environmental professionals can use the results of these investigations to design better restoration plans for rivers and streams impacted by contamination from many different sources. The information presented on this page cuts across lines drawn by individual individual investigations and projects so that information on contaminant transport surface-water can be presented in one place.
Natural toxins are chemicals that are naturally produced by living organisms. These toxins are not harmful to the organisms themselves but they may be toxic to other creatures, including humans, when eaten.

Some plants have the capacity to naturally produce compounds that are toxic to humans when consumed. For example, under certain conditions, microscopic algae (tiny plants) in the ocean can produce compounds that are toxic to humans but not to shellfish that eat this algae. When people eat shellfish that contain these toxins, illness can quickly follow. There is an active monitoring program in place to ensure that shellfish sold to Canadians do not contain these "shellfish toxins". This monitoring program is jointly administered by the Department of Fisheries and Oceans, the Canadian Food Inspection Agency, and Environment Canada.

12.2. FORMATION OF TOXICANTS:

Scientists recently uncovered a surprising and disturbing fact: Environmental toxins make you fat and can cause diabetes. (1) Inside the body, these chemicals monkey with our ability to balance blood sugar and metabolize cholesterol. Over time, the changes can lead to insulin resistance. This discovery should be headline news, but no one is talking about it. Diabetes and obesity — we've got to turn our attention to the heavy burden environmental toxins put on our bodies. Treatment of proteins with alkali may first described by Newberne and Younglead to several changes in biological and The induction of this renal lesion,chemical properties, such as decreased which to our knowledge has no counterdigestibilityand utilization of the protein, part in human renal disease and is desigracemizationand destruction of amino nated as nephrocytomegalia, was

attribacids, and formation of new amino acids, uted to nephrotoxic properties imparted toe.g., lysinoalanine (LAL) Rats fed the protein by alkali treatment, probablydiets containing 20% soybean proteinswhich had been drastically treated with Receivedfor publicationMarch9, 1976.
Toxigenesis, or the ability to produce toxins, is an underlying mechanism by which many bacterial pathogens produce disease. At a chemical level, there are two main types of bacterial toxins, lipopolysaccharides, which are associated with the cell wall of Gram-negative bacteria, and proteins, which are released from bacterial cells and may act at tissue sites removed from the site of bacterial growth. The cell-associated toxins are referred to as endotoxinsand the extracellular diffusible toxins are referred to as exotoxins.

Endotoxins are cell-associated substances that are structural components of bacteria. Most endotoxins are located in the cell envelope. In the context of this article, endotoxin refers specifically to the lipopolysaccharide (LPS) or lipooligosaccharide (LOS) located in the outer membrane of Gram-negative bacteria. Although structural components of cells, soluble endo toxins may be released from growing bacteria or from cells that are lysed as a result of effective host defense mechanisms or by the activities of certain antibiotics. Endotoxins generally act in the vicinity of bacterial growth or presence. Exotoxins are usually secreted by bacteria and act at a site removed from bacterial growth. However, in some cases, exotoxins are only released by lysis of the bacterial cell. Exotoxins are usually proteins, minimally polypeptides, that act enzymatically or through direct action with host cells and stimulate a variety of host responses. Most exotoxins act at tissue sites remote from the original point of bacterial invasion or growth. However, some bacterial exotoxins act at the site of pathogen colonization and may play a role in invasion.

12.3 OXIDATION AND RANCIDITY:

Most any food can technically become rancid. The term particularly applies to oils. Oils can be particularly susceptible to rancidity because their chemistry which makes them susceptible to oxygen damage. When food scientists talk about rancidity, they are often talking about a specific type of rancidity involving oxygen damage to foods, and this type of rancidity is called "oxidative rancidity." During the process of oxidative rancidity, oxygen molecules interact with the structure of the oil and damage its natural structure in a way that can change its odour, its taste, and its safety for consumption.

Oxidation of fats, generally known as rancidity, is caused by a biochemical reaction between fats and oxygen. In this process the long-chain fatty acids are degraded and short-chain compounds are formed. One of the reaction products is butyric acid, which causes the typical rancid taste. Acidification is the decomposition of fats, oils and other lipids by hydrolysis or oxidation, or both. Hydrolysis will split fatty acid chains away from the glycerol backbone in glycerides. These free fatty acids can then undergo further auto-oxidation. Oxidation primarily occurs with unsaturated fats by a free radical-mediated process. These chemical processes can generate highly reactive molecules in rancid foods and oils, which are responsible for producing unpleasant and noxious odours and flavours. These chemical processes may also destroy nutrients in food. Under some conditions, rancidity, and the destruction of vitamins, occurs very quickly. Fats and oils play an important role in the flavor, aroma, texture, and nutritional quality of foods, pet foods, and feeds. Fats and oils may be added during manufacturing or they may be inherent to the product or ingredient. The product may be pure oil or it may be part of a complex mixture with proteins, carbohydrates, minerals, and vitamins. The product may contain almost no fat or it may contain a considerable amount. Regardless of the source of fat, the amount of fat, or the product composition, predicting and monitoring fat and oil quality is an important component of developing and manufacturing high

quality products.As soon as a food, feed, or ingredient is manufactured, it begins to undergo a variety of chemical and physical changes. Oxidation of lipids is one common and frequently undesirable chemical change that may impact flavor, aroma, nutritional quality, and, in some cases, even the texture of a product. The chemicals produced from oxidation of lipids are responsible for rancid flavors and aromas. Vitamins and other nutrients may be partially or entirely destroyed by highly reactive intermediates in the lipid oxidation process. Oxidized fats can interact with proteins and carbohydrates causing changes in texture. Of course, not all lipid oxidation is undesirable. Enzymes, for example, promote oxidation of lipid membranes during ripening of fruit. For most products, though, predicting and understanding oxidation of lipids is necessary to minimize objectionable flavors and aromas arising from fat rancidity.Two Types of Rancidity Selecting an optimum test for lipid oxidation is difficult due to the complexity of the chemical processes involved. In fact, many of the oxidation pathways are not entirely understood. Two types of lipid oxidation cause the most concern. These are oxidative rancidity and hydrolytic rancidity.

Hydrolytic Rancidity: Hydrolytic rancidity results in the formation of free fatty acids and soaps (salts of free fatty acids) and is caused by either the reaction of lipid and water in the presence of a catalyst or by the action of lipase enzymes. Low levels of free fatty acids are not necessarily objectionable, particularly if they are sixteen or eighteen carbon fatty acids as commonly found in soybeans, corn or animal fat. However, for other fats like coconut oil or butter fat, low levels of shorter carbon chain fatty acids may be quite objectionable.

Antioxidants are often added to fat-containing foods in order to retard the development of rancidity due to oxidation. Natural anti-oxidants include flavonoids, polyphenols, ascorbic acid (vitamin C) and tocopherols (vitamin E). Synthetic antioxidants include butylated hydroxyanisole (BHA), butylated hydroxytoluene (BHT), propyl 3,4,5-trihydroxybenzoate also known as propyl gallate and ethoxyquin. The natural antioxidants tend to be short-lived, so synthetic antioxidants are used when a longer shelf life is preferred.

The effectiveness of water-soluble antioxidants is limited in preventing direct oxidation within fats, but is valuable in intercepting free radicals that travel through the watery parts of foods. A combination of water-soluble and fat-soluble antioxidants is ideal, usually in the ratio of fat to water.

In addition, rancidification can be decreased, but not completely eliminated, by storing fats and oils in a cool, dark place with little exposure to oxygen or free radicals, since heat and light accelerate the rate of reaction of fats with oxygen. (Oxidative rancidity or autooxidation is a chemical reaction with a low activation energy consequently the rate of reaction is not significantly reduced by cold storage).

Do not add fresh oil to vessels containing old oil. The old oil will trigger a reaction and the new oil will become rancid far more rapidly than if the oil was stored in a clean empty vessel. Avoid using vessels that are wet, this will also speed up the problems associated with oxidation, allow tanks to drain and dry adequately before use.

"It is an important further factor in the detoxification of the human body that substances can be stored in fatty tissues — these substances being the ones that are dissolved by oil, rather than water." "There are many other types of toxins that store in the fatty tissues. A few are excerpted below:

Some endocrine disrupting chemicals are persistent in the environment and bioaccumulate; they accumulate in the fatty tissue of organisms and increase in concentration as they move up through the food web. Because of their persistence and mobility, they accumulate in and harm species far from their original source.

POPs [persistent organic pollutants] accumulate exponentially in fatty tissue as they move up the food chain, such that concentrations can be 70,000 times the background levels in a top predator.

POPs are found in everything from paint to pesticides and remain in the environment for decades without breaking down. They are spread by winds and oceans, and have been found everywhere from Antarctica to remote areas of Canada. They accumulate in fatty tissue and have been blamed for disease and birth defects in humans and animals.Second, the toxics bioaccumulate in the fatty tissue of animals, and are difficult or impossible to metabolize or excrete. Even minute amounts may have a major effect on wildlife as the toxics build up to a dangerous level over the lifetime of the animal.

Bioaccumulating in the food chain occurs when, for example, plankton which has absorbed toxic chemicals from the water is eaten by fish, which, after storing the toxics in fatty tissues, are Until conventional medicine catches up, you've got to optimize your body's ability to rid itself of toxins. If your body's detoxification tools aren't up to snuff, waste will build up. Over time, the damage is similar to what happens when trash collectors go on strike and don't pick up the garbage off the streets. The waste piles high, making the neighborhood smell bad and creating a breeding ground for illness.

Don't let the word *detoxification* turn you off. You may think it sounds like a New Age idea or something from celebs in Hollywood on the heels of an alcohol or drug binge, but detoxification is a normal, everyday function. It's the body's way of breaking down and eliminating anything that doesn't belong. And, these days, there are a lot of things our bodies come into contact with that don't belong.

Because the birds are at the top of this food chain, they may over time accumulate levels of toxics which are thousands of times higher than those in their prey, which themselves were thousands of times higher than those in the plankton. Protein - Many people who go on weight loss programs greatly reduce their protein intake because of the association of protein and fat. This is a big mistake. Our muscle tissue is comprised of amino acids from protein. If you do not maintain adequate protein intake you will lose lean muscle tissue instead of body fat. When the diet is finished, lean muscle tissue will not be replaced by lean muscle tissue. It will be replaced by body fat. That is why we see people yo-yo dieting and getting larger and larger each time. Daily protein intake should be between 50 - 75% of body weight in grams of protein per day. As fat in the diet is decreased, the body gets less essential fatty acids which are necessary for many body functions and hormonal balance. Also the body will not release fat if there is a lack of essential fatty acids.

Obese, will have more toxins stored in your body. The preferential storage place is adipose tissue, the fat cells. If you are losing weight, and hopefully are doing weight lifting and aerobic exercise, then you're losing fat, which does mean that there are toxins circulating in your system. Even people who advocate Calorie Restriction do not recommend adopting the CR lifestyle too abruptly, for fear that losing a whole lot of weight quickly (and thus releasing a large the amount of toxins into the system all at once) might overwhelm the body.

The selection of plants for human consumption has been based on the safety of the whole plant *food*. If the plant was found to be toxic, it might still have been consumed if adequate processing eliminated or deactivated the toxic factor. During the development of toxicology, emphasis was put on isolation, characterization and quantification of the toxic principle and dose: effect relationships. This is still important. However, for inherent toxicants in plant foods not only does the toxic principle and its dose determine the toxicity, but other food factors can alter the bioavailability of the reactive

metabolite or give rise to interactions such as synergism and antagonism. Therefore, the toxic effect of the whole foodstuff has to be taken into account.5.3. *Toxicity characterization* Ideally the whole foodstuff should be tested in both animal and human subjects. In practice this will not always be possible.

Thus the amount of the whole foodstuff that can be consumed by experimental animals is constrained by the need to feed nutritionally balanced diets and the concentration of the toxicant in the food stuff may be too low to induce adverse effects in animals. In feeding trials with human subjects the possible consequences of the toxicity may prevent experimental testing .A solution to this problem is to organize the variables characterizing toxicity into a grid in which the irinter-relationships are displayed. The incorporation of appropriate data into this grid allows a quantified assessment to be made of the probable range of the toxic effect of the foodstuff on human subjects. The grid is formed from two strata; animal and human studies interacting with the degree of exposure and the magnitude of the effect. Each stratum consists of two parts, the pure substance and the whole foodstuff, described as the product. The evaluation of toxicity of a foodstuff progresses across the grid from left to right. The direction is from the most readily established data with the isolated toxicant in animals towards the determination of the toxicity of the whole foodstuff, the product, in humans. The pure substance and the foodstuff, in which it occurs, the product, are each evaluated in animals and humans so far as ethical and practical constraints permit. Progressing across the grid, the challenges are to establish a measure of exposure and to observed and quantify the toxic effect.

Many feeding trials have been reported in which GM foods like maize, potatoes, rice, soybeans and tomatoes have been fed to rats ormice for prolonged periods, and parameters such as body weight, feed consumption, blood chemistry, organ weights, histopathology etchave been measured. The food and feed under investigation were derived from GM plants with improved agronomic characteristics likeherbicide tolerance and/or insect resistance.

Direct quantification of the toxic effect of the food contained toxicant may not be possible, even in animals, and consequently markers of exposure and of effect are used. Biomarkers may be: 1. Exposure biomarkers, which relate the internal dose, or target organ dose, to the external dose and are specific for that chemical and animal model.2. *Effect biomarkers*, which relate the effect, or a surrogate end point, to the external dose, and may be either specific to the chemical or non-specific.3. *Susceptibility biomarkers*. which relate to the differences insusceptibility of exposed individuals, for example a genetic polymorphism. Such biomarkers are of greatest value in interpretation of epidemiological studies .The degree of uncertainty associated with the final evaluation of the toxicity of the product is least when the greatest number of the possible considerations in the grid have been taken into account. There are many risk assessment scenarios. For example, the toxic effect may have been established with the isolated toxicant in the animal studies, while it may not be possible to feed sufficient quantity of the product to achieve a toxic effect. Risk assessment is possible providing that exposure to the product has been defined and its effect monitored through biomarkers. The use of biomarkers of exposure makes it possible to predict the extent of any adverse toxic effect of the product.

The quality, safety and the health of feeds and foods as well as the concept of protecting local products (i.e. PDOs) are elements of basic importance both for consumers and for producers. Technological quality is a complex and multivariate property of animal-born raw materials which is affected by multiple interacting factors ranging from animal genotype to slaughtering procedures.Among the intra vitam factors influencing the nutritional and technological characteristics of food, the diet lipid component represents a major source of variability and it is tied to animal's fat

profile.It is important to tailor the feed formulation to match the quality traits requested by the food processors and consumers. With respect to local productions, it is worth noting that many of Italy's (and Mediterranean) registered products (PDOs and PGIs) are processed meats (salami, ham, sausages, etc.) and that a better knowledge of their curing aptitudes and properties could be an important element of protection and valorisation.

At the field scale, the biodegradation rate is usually estimated from analytical solutions to single species transport with first-order reactions, using measured data as input. Because many contaminants, e.g., chlorinated solvents, are degraded in a sequential pattern, with degradation products further reacting to produce new species, it is of great interest to quantify the transformation rate of every reaction. The conventional inverse solutions for identifying the transformation rates are limited to single species problems. In the present study, we propose a successive optimization approach to identify the biodegradation rate for each species by using a previously developed analytical solution to multi-species first-order reactive transport using data obtained at the field scale. By specifying a link between analytical solutions to sequentially reactive transport problems and optimization methods and assuming constant transport parameters (velocity, dispersivities, and retardation factors), the first-order transformation rates are optimized successively from parent species to its daughter species.

12.4. SUMMARY

The metabolic syndrome is the result of mitochondrial dysfunction, which in turn is caused by exposure to persistent organic pollutants.We live in an environment steeped in chemicals that our bodies were not designed to process. For a disturbing look at the chemicals that breach the boundaries of our bodies, look no further than the Centers for Disease Control and Prevention's National Report on Human Exposure to Environmental Chemicals . In the latest report, scientists at the CDC found that nearly every person they tested was packing a host of nasty chemicals, including flame retardants stored in fatty tissue and Bisphenol A, a hormone-like substance found in plastics, excreted in urine. Even babies are contaminated: The average newborn has 287 chemicals in her umbilical cord blood, 217 of which are neurotoxic (poisonous to nerves or nerve cells). The quality, safety and health of feeds and foods as well as the concept of protecting local products are of basic importance both for consumers and producers.

12.5. SELF ASSESSMENT QUESTIONS

1. Explain Food toxicants in detail?
2. Describe Rancidity and oxidation?

12.6 REFERENCE BOOKS

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అధ్యాపకుల, విద్యార్థుల సలహాలు, సూచనలు :

అధ్యాపకులు, విద్యార్థులు ఈ స్టడీ మెటీరియల్‌కు సంబంధించిన సలహాలు, సూచనలు, ముద్రణ దోషాలు తెలియపరచినచో, పునర్ముద్రణలో తగు చర్యలు తీసుకొనగలము. తెలియపరచవలసిన చిరునామా : **డిప్యూటీ డైరెక్టర్, దూరవిద్యా కేంద్రం, ఆచార్య నాగార్జున విశ్వవిద్యాలయం, నాగార్జున నగర్ - 522 510.**

Course	Year	Paper No. & Title
M.Sc.,	IInd Year	Food Microbiology and Texicology

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