UNIT 10 FOOD INTOXICATIONS

Structure

10.0 OBJECTIVES

After reading this unit, you will be able to:

- discuss the causal organisms responsible for food borne intoxications;
- explain the toxins production by mold and bacteria (*Clostridium botulinum* and *Staphylococcus aureus*);
- the microbial toxins produced, the foods associated in intoxication, symptoms of the disease, diagnosis, conditions necessary for outbreak and preventive measures required will also be discussed; and
- know the naturally present toxins in the food products will also be accounted for.

After reading this unit you will be able to distinguish between food borne intoxications caused by the various microbiological agents and their preventive measures.

10.1 INTRODUCTION

We have already studied about the food borne diseases and their classification. Food borne intoxications are basically food borne illness caused due to ingestion of toxin produced my microorganisms (mycotoxins, bacterial toxins). Natural toxins present in food may also result in food poisoning in humans.

Food poisoning is also caused by consuming old, used, residual, fermented, spoiled, contaminated, toxic and bacteria infested food.

10.2 NATURAL TOXINS

Some plants and animals originate food contain toxic substances. Some pulses and legumes contain a number of toxic substances such as protease inhibitors, lathyrogens, and flavism causing agents, cyanogens, haemaglutinins and paponins which are discussed below:

a) Trypsin inhibitor is a proteinous in nature. It suppresses the release of amino acid. It thus interferes with the normal growth of animals fed with such pulses.

b) Haemglutinins are also proteins. They impair the absorption system.

c) Cynogenic glycosides cause cyanide poisoning on hydrolysis of the glycoside by the enzyme α-glucosidase, hydrogen cyanide is liberated. A cyanides content of 10-20 mg per 10 gm of pulses is considered safe. Many legumes excepting limabean (*Phaseolus lunatus*) contains cyanide within these limits.

d) Saponins are glycosides of high molecular weight. This has been reported in soyabean, swordbean and jackbean. Toxic saponins cause nausea and vomiting and can be removed by soaking the beans prior to cooking.

e) Alkaloids are known to occur in the seeds of many legumes but they are relatively innocuous.

f) Some compounds present in pulses appear to bind iodine thus producing a state of iodine deficiency in the thyroid and eventually goitre.

g) Lathyrism is a disease that paralyses the lower limbs. The disease is associated with consumption of *kesari dal* regularly as high as 300g daily. In lathyrism, the toxic substances interfere with formation of normal collagen fibers in the connective tissue.

h) A hemolytic factor in *Vicia faba* causes flavism. It is caused by eating broadbeans or by inhaling pollen of its flowers. Flavism is hemolytic anemia. In several cases, death may occur within 24-48 hours of the onset of the attack.

i) Oxalic acid, a constituent of rhubarb, spinach and beet may cause oxalic poisoning in certain individuals.

j) Some poisonous substances may also be present in some cereals and vegetables e.g. protease inhibitor in cereals and potatoes, saponins in spinach and asparagus and goitrogens in rapeseed mustard, cabbage and related species. Goitrogens cause hypothyroidism and thyroid enlargement

k) Tissues of certain marine animals contain toxic substances, which cause adverse responses when eaten. Some algae like *Gymnodinium* and *Gonyaulax* are toxic. Heating does not destroy these substances.

l) Algal or Planktonic Fish Poisonings: Fish poisoning can result from the ingestion of fish or shellfish that have fed upon algae toxic to human beings. Paralytic shellfish poisoning is caused by ingestion of shellfish such as scallops, clams and mussels which have consumed toxic dinoflagellates. Symptoms appear within 10min after ingestion and include gastrointestinal distress, parasthesia of the lips and fingertips followed by ataxia, muscular uncoordination and ascending paralysis. Death may occur within 2 to 12 hours from cardiovascular collapse or respiratory failure. The human lethal dose of toxin is considered to be 3 to 4 mg.

Prevention: Soaking, heating or fermentation of pulses can reduce or eliminate most of the toxic factors in them. Heat causes denaturation of the proteins responsible for trypsin inhibition and haemagglutination and of the enzyme causing hydrolysis of cyanogenic glycosides. Fermentation also destroys toxic factors and yield more digestible products of high nutritive value.

10.3 MYCOTOXINS

Fungi are a very diverse group of organisms and have a significant impact on the production, spoilage and safety of food. Molds have not only served to synthesize antibiotics but also to produce some foods. Fermented foods such as some cheese, soy sauce, *miso, tempeh* and other oriental delicacies are prepared with the help of molds.

It is well documented that some molds produce toxic substances. Some fungi elaborate the toxin in large macroscopic fruiting bodies; for example, the toxin produced by certain species of *Amanita*, a poisonous mushroom. Other fungi always grow and sporulate as parasites on living host plants, and sometimes will do so only on a specific host. *Claviceps* is an example of this group of fungi and it produces mycotoxins. In contrast to fungi that are parasitic on living plants another group of fungi is saprophytic and causes destruction of dead plants and animal material. There is abundance of the spores of these molds in atmosphere and are found to inhabit stored grain and dried products and hence have been referred to as "storage fungi". These molds include *Cladosporium, Fusarium, Penicillium, Aspergillus* and *Alternaria*.

Mycotoxins are secondary metabolites produced by molds on foodstuffs that causes illness or death when ingested by man or animals. The primary metabolites are those that are essential for growth whereas secondary metabolites are formed during the end of the exponential growth phase and have no apparent significance to the producing organism relative to growth. The mycotoxins commonly encountered in food are around one million times less toxic than most lethal of the botulism toxin. But long term chronic toxicity is of special concern because several of the mold metabolites are carcinogenic and influence the immune response of a number of animal species. The syndrome resulting from ingestion of toxin in a mold contaminated food is referred to as mycotoxicosis.

At the beginning of the last century, two major mycotoxicosis caused considerable suffering and mortality. They were alimentary toxic aleukia (ATA) in Russia, caused by consumption of corn contaminated with T-2 toxin produced by *Fusarium sporotrichoides* and yellow rice disease in Japan, associated with *Penicillium islandicum*. More recently, outbreaks of aflatoxicosis caused by consumption of corn contaminated with *Aspergillus flavus* were reported from India involving approximately 1000 people of whom nearly 100 died.

Several very important mycotoxins such as the sporidesmins, slaframine and tremorgens are associated with animal feeds and forages which affect the quality of meat and other animal products. It is also seen that mycotoxins present in animal feed get into human foods because they pass through the food chain in either their original or metabolized form.

When we store foods under inappropriate conditions they are susceptible to mold growth. Many mycotoxigenic species are able to produce several mycotoxins. It is likely, therefore, that contaminated foods will contain a cocktail of toxins that can interact synergistically.

Some major mycotoxins found in foods

Mycotoxin	Major Foods	Common Producing Species
Aflatoxins	Corn, groundnuts, figs, tree nuts	*Aspergillus flavus, A. parasiticus*
Aflatoxin M_1	Milk, milk products	(secreted by cow after metabolism of Aflatoxin B_1
Deoxynivalenol	Cereals	*Fusarium graminearum, F. culmorum*
Fumonisin	Corn	*Fusarium moniliforme*
Ochratoxin	Corn, cereals, coffee beans	*Penicillium verrucosum, Aspergillus ochraceus*
Patulin	Apple juice	*Penicillium expansum*
Sterigmatocystin	Cereals, coffee, beans, cheese	*Aspergillus versicolor*
Zearalenone	Corn, barley, wheat	*Fusarium graminearum*

10.3.1 Aflatoxin

Aflatoxins are the most widely studied of all mycotoxins. Knowledge of their existence dates from 1960, when more than 100,000 turkey died in England after eating peanut meal imported from Africa and South America. From the poisonous feed were isolated *Aspergillus flavus* and a toxin produced by this organism that was designated aflatoxin (*Aspergillus flavus* toxin- A-fla-toxin). These compounds are highly substituted coumarins, and at least 18 closely related toxins are known. Aflatoxin B_1 is the most important of this large family of compounds and is produced by *Aspergillus flavus, A. parasiticus* and *A.nominus*. The toxicity of the six most potent aflatoxins decreases in the following order: $B_1>M_1>G_1>B_2>M_2\neq G_2$.

Occurrence: Aflatoxigenic molds can occur in warmer parts of the world and aflatoxicosis maybe produced in a wide range of tropical and subtropical food commodities such as figs, tree nuts and cereals. The most important crops are corn and groundnut, but it can also occur in temperate crops such as wheat. Although the production of aflatoxin initially was considered to be a problem in post harvest crops stored at inappropriate temperatures and water activities, it is now known that these compounds can be present in the field before harvest. *A.flavus* and *A. parasiticus* may infect healthy plants at a very early stage.

Biological effects: Aflatoxins are acute hepatotoxins and are known to be carcinogenic in some animal species as rat. Aflatoxin B_1 is acutely toxic to our species and is responsible for liver necrosis. The toxicological effect of the aflatoxins are influenced by their metabolism after intake into their body (Figure 10.1).

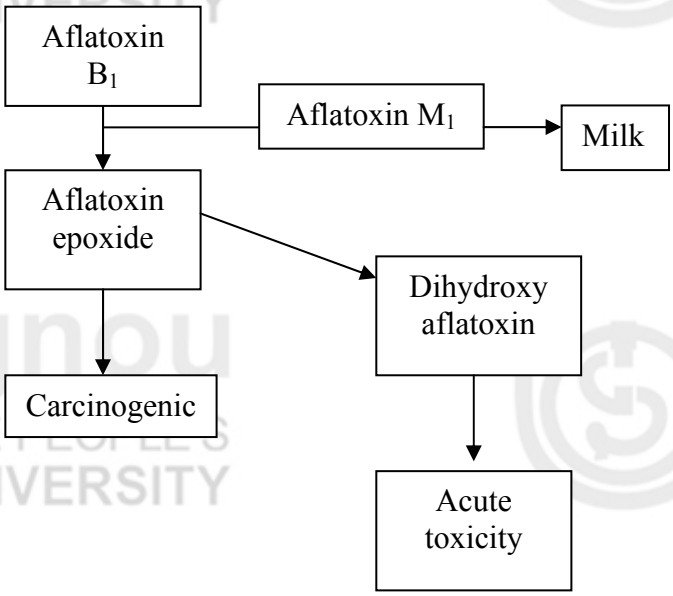

Figure 10.1: Representation of the metabolism of aflatoxin and its biological effects

When cows eat feed containing aflatoxin, aflatoxin M_1 and M_2 is excreted in the milk. Although M_1 and M_2 are less toxic than the parent compound B_1, M_1 retains its toxic and carcinogenic ability in many animals. The LD_{50} of AFB_1 for rats by the oral route is 1.2mg/kg and 1.5 to 2.0 mg/kg for AFG_1.

Control: Because aflatoxins are potentially widespread in occurrence and have an insidious combination of acute and chronic toxicity, it is prudent to control their presence in food. Many countries have legislation establishing maximum tolerance levels. Chemically treating the aflatoxin contaminated commodities like nuts maybe possible or to use technologically sophisticated equipment to sort and discard the contaminated units. It may also be possible to control the production of aflatoxin in the field by an integrated programme of agricultural management that may include plant breeding, improved irrigation and replacement of aflatoxigenic strains by non- aflatoxigenic strains of *A. flavus*.

Check Your Progress Exercise 1

Note: a) Use the space below for your answer.
b) Compare your answers with those given at the end of the unit.

1. What are mycotoxins? How are they harmful?

……

……

……

……

……

……

2. Give the biological effects of aflatoxin.

……

……

……

……

……

……

3. List the various natural toxins present in food.

……

……

……

……

……

……

10.3.2 Ochratoxin

Occurrence: Ochratoxin A is a phenylalanyl derivative of a substituted isocoumarin produced by *Penicillium verrucosum* in temperate climate and by several species of Aspergillus in warmer and tropical parts of the world. *Penicillium verrucosum* is especially associated with stored cereals although it has also been isolated from meat and fish, however the occurrence of ochratoxin A in meat products is usually due to transmission into muscle, kidney and blood in animal fed on contaminated animal feed such as barley. Ochratoxin may also be transferred from dietary intake into milk. *Aspergillus ochraceus* is common on coffee beans, spices, soybeans, groundnut, rice and corn. Ochratoxin is heat resistant and is not destroyed by roasting or autoclaving though the ochratoxin producing fungi are capable of growth and mycotoxin production at temperature below 10ºC.

Biological effects: It is associated with the chronic progressive kidney disease in humans known as Balkan endemic nephropathy. There is increased evidence that it can also be considered as a carcinogen with genotoxic property as well as a potent nephrotoxin. The availability of improved methods of analysis has demonstrated that ochratoxin is quite widespread in foods and its presence in human body fluids confirms that there is a significant exposure with the human population. Ochratoxin is immunosuppressive and inhibits protein biosynthesis. Ochratoxin A has been classified by the International Agency for Reseach on Cancer (IARC) as a possible human carcinogen. Low doses as 70μg/kg body weight can induce kidney tumors in male rats. Its oral LD_{50} in rats is 20 to 22 mg/kg, and it is both hepatotoxic and neprotoxic.

Control: Once ochratoxin A has been formed in a food, it is difficult to remove by most forms of food processing. Cooking with or without previous soaking removes a significant amount of ochratoxin from beans but does not lead to total destruction. Beans still contain 16% to 60% of the initial ochratoxin contamination and it seems probable that the material may have leached out rather than destroyed.

10.3.3 Patulin

It is a toxic and antibiotic metabolite produced by several species of *Penicillin, Aspergillus* and *Paeciliomyces* but the most important in the context of human food production is *P. expansum*, a soft rot pathogen of apple and pears. Patulin is an unsaturated lactose and is sensitive to sulphur dioxide and is unstable in alkali but stable in acid.

Occurrence: It is found in a range of foods based on fruits. Presence of patulin in fruit juice is a indication that the juice was extracted from poor quality fruit which is undesirable and should be avoided with good manufacturing practices. This mycotoxin has also been found in moldy bread, sausage, fruits (including bananas, pears, pineapples, grapes and peaches) and other products.

Biological activity: Patulin has an acute oral LD_{50} in rodents of about 30-50 mg/kg and has been shown to be teratogenic, immunotoxic and neurotoxic and to cause gastrointestinal disturbances in rats. Patulin is quite rapidly excreted from animals. It causes chromosomal aberrations in animal and plant cells and is a carcinogen.

Control: In apples molded by *Penicillium expansum*, most of the patulin is confined to the region of damaged tissue and simply removing the lesions reduces the toxin by 90%, but if 1cm around the lesion is also removed, no patulin is detectable in rest of the apple. Ascorbic acid has been reported to reduce levels of patulin. Although pasteurization (using high temperature, short time treatment of ten seconds at 90ºC) causes some reduction in patulin in fruit juices, it is only of the order of 20%, which is not sufficient to make a badly contaminated food product acceptable.

10.3.4 Alternaria Toxins

Several species of Alternaria (*A.citri, A. alternata, A.solani* and *A. tenuissima*) produce toxic substances that have been found in apples, tomatoes, blueberries and others. The toxins produced include alternariol, alternariol monomethyl ether, altenuene, tenuazonic acid and altertoxin-I.

10.3.5 Citrinin

This mycotoxin is produced by *Penicillin citrinum*, *P.viridicatum* and other fungi. It has been recovered from polished rice, moldy bread, country cured hams, wheat, oats, rye and other similar products. It is a known carcinogen.

10.3.6 Penicillic Acid

This mycotoxin has biological properties similar to patulin. It is produced by a large number of fungi, including many Penicillia as well as members of the *A.ochraceus*. One of the best producers is *P.cyclopium*, it has been found in corn, beans and other field crops. Its LD_{50} in mice by subcutaneous route is 100 to 300 mg/kg and it is a proved carcinogen.

10.3.7 Sterigmatocystin

These mycotoxins are structurally and biologically related to the aflatoxins, and like the latter, they cause hepatocarcinogenic activity in animals. Among the organisms that produce them are *Aspergillus versicolor*, *A. nidulans*, *A.rugulosus*. The LD_{50} for rats by intra-peritoneal injection is 60 to 65 mg/kg.

10.3.8 Fusarium Toxins

Another important genus of mycotoxin producers is *Fusarium,* many species of which produce members of the trichothecene family of mold metabolites like deoxynivalenol, neosolaniol and T-2 toxin etc.

Deoxynivalenol

Deoxynivalenol (DON) is a far more common, but much less toxic, trichothecene and is produced by species such as *F. graminearum* and *F. culmorum*. LD_{50} of DON is 70mg/kg. The trichothecenes are remarkably stable compounds, and DON will survive both dry milling and wet milling processes of corn. The baking of bread has relatively little effect on trichothecenes such as DON.

Zearalenone

It was first isolated as the agent responsible for vulvovaginitis in pigs, has very little acute toxicity, but there should be some concern about chronic exposure to a compound known to be estrogenic. It may be produced, together with DON and other trichothecenes, in a wide range of cereals including corn, barley and wheat.

Moniliformin

It was first obtained from a strain of *Fusarium moniliforme* isolated from southern leaf blight- damaged corn seed as a water soluble toxin. The LD_{50} for mice has been reported to be 20.9 mg/kg for females and 29.1 mg/kg for males. At toxic doses moniliformin causes rapid death without obvious overt cellular damage, although acute degenerative lesions in the myocardium are reported.

Fumonisins

The most recently characterized mycotoxins of any major significance in human health are the fumonisins produced by species of *Fusarium*, such as *F. moniliforme*. Like a number of mycotoxins, the fumonisins are relatively heat stable and would not be significantly destroyed by drying processes for corn or heat treatments used for the production of maize derivatives. Fumonisin B_1 is water-soluble is known to be responsible for equine encephalomalacia, porcine pulmonary edema syndrome and hepatic cancer in rats and maybe involved in

the epidemiology of esophageal carcinoma in humans in southern Africa and parts of China.

The range of regulatory limits for mycotoxins

Mycotoxin	Regulatory Limit (µg/kg)
Aflatoxins in foods	0-50
Aflatoxin M1in milk	0-0.5
Deoxynivalenol in wheat	1000-4000
Ochratoxin A in foods	1-300
Patulin in apple juice	20-50
T-2 Toxin	100
Zearalenone	30-1000

10.4 BOTULISM

Botulism (Latin *botulus*, sausage) is a neuro-paralytic disease caused by the ingestion of food containing the neurotoxin produced by *Clostridium botulinum*.

10.4.1 Occurrence

Clostridium botulinum is an anaerobic, Gram-positive, spore forming, rod that produces the potent neurotoxin. The organism and its spores are widely distributed in nature and occur in both cultivated and forest soils, bottom of streams, lakes and coastal waters and in the intestinal tracts of fish and mammals and in viscera of shellfish.

On the basis of the serological specificity of their toxins, seven types of *Clostridium botulinum* are recognized: A, B, C, D, E, F and G. Types A, B, E, F and G cause disease in humans; type C causes botulism in fowls, cattle, mink and other animals and type D is associated with forage poisoning of cattle. Being a saprophyte, the organism seldom grows or produces toxic in the live animal; it can do so only by growing in food. The toxins are simple heat labile proteins and can be destroyed if heated at 80ºC for 10 minutes or longer.

C.botulinum does not produce the fully toxic molecule; instead a progenitor toxin is activated to its full toxicity by enzymes. The progenitor toxin is hydrolyzed to the highly toxic derivative toxin and is carried to target nerves where it binds to the synapses of motor neurons and prevents the release of the neurotransmitter acetylcholine. As a consequence, muscles do not contract in response to motor neuron activity and flaccid paralysis results.

10.4.2 Types and Symptoms

Different types of botulism are recognized: adult, infant and wound. A very small amount (a few nano grams) of toxin can cause illness.

Adult Botulism

Symptoms of botulism may develop anywhere between 12 and 72 hours after the ingestion of toxin containing foods. Symptoms include nausea, vomiting, fatigue, dizziness and headache, dryness of skin, mouth and throat, constipation, lack of fever, paralysis of muscles, double vision and finally respiratory failure

and death. The duration of the illness is from 1 to 10 or more days depending upon host resistance and other factors.

Infant Botulism

In the adult form of botulism, preformed toxins are ingested; in infant botulism, viable botulinal spores are ingested and upon germination in the intestinal tract, toxin is synthesized. It is confined to infants under a year of age. High number of spores are found in the feces of infants during the acute phase of the disease. It appears that ingested endospores, which maybe present in honey or other baby foods, germinate in the infants intestine. *C.botulinum* then multiplies and produces the exotoxin. The infant becomes constipated, listless, generally weak and eats poorly. Death may result from respiratory failure.

Wound Botulism

It is the rarest form of botulism. The illness results when *C.botulinum* by itself or with other microorganisms infects a wound and produces toxins which reach other parts of the body via the bloodstream. Foods are not involved in this type of botulism.

10.4.3 Diagnosis

Although botulism can be diagnosed by clinical symptoms alone, differentiation from other diseases maybe difficult. The most direct and effective way to confirm the clinical diagnosis of botulism in the laboratory is to demonstrate the presence of toxin in the serum or feces of the patient or in the food which the patient consumed.

10.4.4 Foods Implicated in Botulism

The types of foods involved in botulism vary according to food preservation and eating habits in different regions. Any food that is conducive to outgrowth and toxin production, that when processed allows spore survival, and is not subsequently heated before consumption can be associated with botulism. Almost any type of food that is not very acidic (pH above 4.6) can support growth and toxin production by *C.botulinum.* Botulinal toxin has been demonstrated in a considerable variety of foods, such as canned corn, peppers, green beans, soups, asparagus, mushrooms, spinach, tuna fish ham, sausage and smoked and salted fish.

10.4.5 Conditions Necessary for Outbreak

The following conditions are necessary for an outbreak of botulism:

1. Presence of spores of *C.botulinum* of type A, B or E in foods being consumed or being processed in some other way
2. A food in which the spores can germinate and the clostridia can grow and produce toxin
3. Survival of the spores of the organism eg: because of inadequate heating in canning or inadequate processing otherwise
4. Environmental condition after processing that will permit germination of the spores and growth and toxin production by the organism
5. Insufficient cooking of the food to inactivate the toxin
6. Ingestion of the toxin-bearing food

10.4.6 Prevention and Control

The prevention and cure of botulism involves:

1. Strict adherence to safe food-processing practices by the food industry
2. Educating the public on safe home-preserving (canning) methods for foods
3. Not feeding honey to infants younger than 1 year of age
4. Not tasting any processed food having a questionable odor
5. Recommended treatment for botulism includes early administration of botulinal antitoxin and intensive supportive care (including mechanical breathing assistance).

Check Your Progress Exercise 2

Note: a) Use the space below for your answer.
b) Compare your answers with those given at the end of the unit.

1. What are the causative organism and the foods associated with botulinal food intoxication?

……

……

……

……

……

……

2. Differentiate between adult and infant botulism.

……

……

……

……

……

……

……

……

3. List down the various mycotoxins associated with food.

……

……

……

……

……

.....

.....

10.5 STAPHYLOCOCCAL FOOD POISONING

Staphylococcal food poisoning results from consumption of food containing enterotoxin produced by enterotoxigenic strains of *Staphylococcus aureus*. It is caused by ingestion of improperly stored or cooked food (particularly foods such as ham, processed meats, chicken salad, pastries and ice cream) in which *S.aureus* has grown.

S.aureus is a Gram-positive coccus, very resistant to heat, drying and radiation. If the bacteria are allowed to incubate in certain foods, they produce heat-stable enterotoxin that render the food dangerous. Six different enterotoxins have been identified and are designated as A, B, C, D, E and F.

10.5.1 Occurrence

Staphylococci exist in air, dust, sewage, water, milk and food or on food equipment, environmental surfaces, humans and animals. Humans and animals are the primary reservoirs. Staphylococci are present in the nasal passages and throats and on the hair and skin of 50 percent or more of healthy individuals. A wide range of foods maybe involved in Staphylococcal food poisoning including ham, turkey, chicken and chicken salad, baked products, especially filled pastries, table ready-meats (sausage etc.), precooked frozen foods and dairy products.

S.aureus cells are relatively more resistant than many gram negative food spoilage organisms. Human intoxication is caused by ingesting enterotoxins produced in food by strains of *S.aureus,* usually because the food has not been kept hot enough (60ºC, or above) or cold enough (7.2ºC, or below). In frozen foods they may survive at –10ºC. In general, survival of *S.aureus* is best in foods that contain high concentration of sugars, eggs and buffering component such as phosphates and protein. Salt concentration less than 9.5%, temperature more than 20ºC and a pH in the range 6-8 are favourable for growth and enterotoxin formation.

10.5.2 Symptoms

A toxin dose of less than one micro gram in contaminated food will produce symptoms of staphylococcal intoxication. This toxin level is reached when *S. aureus* populations exceed 100,000 per gram. Symptoms of staphylococcal food poisoning usually develop with 1-6 hours of ingestion of contaminated food. Typical symptoms include severe abdominal pain, diarrhoea, vomiting, sweating, headache, prostration, nausea and sometimes a fall in body temperature. The mortality rate of staphylococcal food poisoning is negligible among healthy individuals.

10.5.3 Diagnosis

Diagnosis is based on the symptoms or laboratory diagnosis of the bacteria from leftover foods and from the stool cultures of victims. Enterotoxin maybe detected in foods by animal toxicity tests.

10.5.4 Foods Incriminated

Foods that are frequently incriminated in staphylococcal food poisoning include meat and meat products, poultry and egg products, egg, tuna, chicken, potato and macaroni, bakery products like cream-filled pastries, cream pies, chocolate eclairs, sandwich fillings, milk and dairy products. Foods that require considerable handling during preparation and that are frequently involved in staphylococcal food poisoning.

10.5.5 Conditions Necessary for Outbreak

The following conditions are necessary for an outbreak of staphylococcal food poisoning:

1. The food must contain enterotoxin producing staphylococci.
2. The food must be a good culture medium for growth and toxin production by the staphylococci.
3. The temperature must be favourable for growth of the cocci and enough time must be allowed for production of enterotoxin.
4. The enterotoxin bearing food must be ingested.

10.5.6 Prevention and Control

Staphylococcal food poisoning can be prevented by:

1. Avoiding contamination of food with *S.aureus.*
2. Prevention of growth of staphylococci by adequate refrigeration of foods and adjustment of more acid pH.
3. Killing staphylococci in susceptible foods by heating rapidly to 65-70ºC for 12-15 minutes.
4. Good personnel hygiene- exudates from skin lesions (pimples, boils) and nasal discharges of food handlers are rich sources of staphylococci and should be avoided.
5. Prolonged storage at room temperature of filled pastries, meat, salads and similar products that receive only a minimal heat treatment should be avoided.

Check Your Progress Exercise 3

Note: a) Use the space below for your answer.
b) Compare your answers with those given at the end of the unit.

1. What are the conditions favouring the outbreak of Staphylococcal food poisoning?

……

……

……

……

……

……

2. Give the preventive and control strategies of Staphylococcal food poisoning.

……

……

……

……

……

……

……

……

10.6 LET US SUM UP

The unit deals with the food borne intoxications which are caused by ingestion of toxins produced from molds (*Aspergillus niger*, *Penicillium* sp.) or bacteria (*C.botulinum* and *S. aureus*). An effort has been made to point out the major conditions required for outbreak of the disease along with the diagnosis and the preventive measures required to prevent the outbreak of disease.

10.7 KEY WORDS

Food Borne Intoxication : It is the food borne disease caused due to ingestion of food containing the toxin.

Mycotoxins : These are secondary metabolites produced by filamentous fungi (molds) on food and feedstuffs that cause illness and death when ingested by man or animals.

Aflatoxin : This is the mycotoxin elaborated by *Aspergillus flavus*.

Ochratoxin : Mycotoxin produced by *Penicillium verrucosum*.

Patulin : Mycotoxin produced by species of *Penicillin, Aspergillus* and *Paeciliomyces*.

Citrinin : Mycotoxin produced by *Penicillin citrinum*.

Botulism : Food borne intoxication caused due to consumption of food containing toxin, produced by *Clostridium botulinum*.

Staphylococcal Food Poisoning : Food borne intoxication caused due to consumption of food containing toxin, produced by *Staphylococcus aureus.*

LD_{50} Dose : Lethal Dose of toxin at which minimum 50% of the population may get infected by the toxin.

Zearalenone : Mycotoxin produced by *Fusarium graminearum* and *F. tricinctum.*

10.8 ANSWERS TO CHECK YOUR PROGRESS EXERCISES

Check Your Progress Exercise 1

1.
   - Mycotoxins are toxins (secondary metabolites) produced by filamentous fungi on food and feedstuffs that cause illness when ingested
   - Cause hepatotoxicosis, carcinogenesis, liver cirrhosis etc.
2. Biological effects of aflatoxin include hepatotoxicosis, carcinogenesis and liver cirrhosis.
3. Natural toxins in foods: Trypsin inhibitor, haemaglutenins, cyanogenic glycosides, saponins, alkaloids, goitrogens .

Check Your Progress Exercise 2

1.
   - Botulism is caused by the bacteria *Clostridium botulinum*.
   - All low acid foods can support growth and toxin production by *Clostridium botulinum*.
   - Unheated foods are causative agents e.g.: canned foods (canned corn, peppers, green beans, soups), sausages, smoked fish etc.
2.
   - Adult Botulism is prevalent amongst adults whereas Infant Botulism is prevalent in infants of less than one year of age.
   - Caused due to ingestion of viable spores of *C. botulinum.*
   - Symptoms in adults: Nausea, vomiting, fatigue, dizziness, headache, dryness of skin and respiratory failure
   - Symptoms in infants: Constipation, weakness and loss of appetite and in severe cases death
3. Mycotoxins associated with foods are:
   - Aflatoxins (produced by *Aspergillus flavus*)
   - Ochratoxin (produced by *Penicillium verrucosum*)
   - Patulin (*P. expansum*)
   - Citrinin (*Penicillin citrinum*)
   - Alternaria toxins (*Alternaria* sp.)
   - Penicillic acid (*P.cyclopium*)
   - Sterigmatocystin (*Aspergillus versicolor*)
   - Fusarium toxins (*Fusarium* sp)

Check Your Progress Exercise 3

1. Conditions necessary for outbreak of Staphylococcal food poisoning:

- Presence of viable staphylococcal bacteria in the food
- Growth and toxin production in food
- Toxin containing food must be ingested into the body

2. To prevent and control Staphylococcal food poisoning:
   - Avoid contamination of foods
   - Kill organism by heating, refrigeration
   - Personnel hygiene
   - Adequate cooking
   - Proper storage

10.9 SOME USEFUL BOOKS

1. Defigueiredo, M.P. and Splittstoesser, D.F. (1976) Food Microbiology: Public Health and Spoilage Aspects, AVI Publishing Co. Inc., Westport, Connecticut. pp 492.
2. Jay, J. (1996) Modern Food Microbiology, CBS Publishers, New Delhi. pp701.