Food Poisoning

A Threat to Humans

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Unit I

Introduction

Food poisoning includes ill effects caused by the ingestion of contaminated food by many ways apart from microbial agents. They may be

   i. Through the addition of proteins

  ii. Through eating of inherent poisonous substance such as certain mushrooms, fish and molluscs by mistake

 iii. Adulteration of food with poisonous substance such as *Argemone mexicana* in mustard producing epidemic dropsy

The term "food poisoning" is however restricted only to acute gastroenteritis due to bacterial pollution of food or drink. The term "food-borne" disease is defined as "**A disease, usually either infectious or toxic in nature, caused by agents that enter the body through the ingestion of food**". Food-borne diseases may be classified as

Food-Borne Intoxications

Food-borne intoxications are caused

1. Due to naturally occurring toxins in some foods, including

     i. Lathyrism (beta-oxalyl amino-alanine)

     ii. Endemic ascitis (Pyrrolizidine alkaloids)

2. Due to toxins produced by certain bacteria, including

     i. Botulism

     ii. Staphyloccal toxins

3. Due to toxins produced by some fungi, including

     i. Aflatoxin

     ii. Ergot

     iii. Fusarium toxins

4. Due to toxins produced by some algae, like

     i. Planktonic dinoflagellates

     ii. Diatoms

iii. Cyanobacteria

5. Due to food-borne chemical poisoning

Food-Borne Infections

Food-borne infections include

1. Bacterial infections such as

   i. Salmonellosis

   ii. Shigellosis

   iii. *E. coli* diarrhoea

   iv. Cholera

   v. Streptococcal infection

   vi. Brucellosis

   vii. Listeriosis

2. Viral infections such as

   i. Viral gastroenteritis

   ii. Hepatitis A

3. Parasitic infections such as

   i. Taeniasis

   ii. Trichinellosis

Bacterial Food Poisoning

Food-Borne Diseases and Food Poisoning

Food-borne disease is a disease caused by ingestion of food contaminated by any agent, chemical or biological. Food poisoning is an acute enteritis caused by the ingestion of food, characterized by diarrhea, vomiting, with or without fever and abdominal pains. Food poisoning is normally associated with the small and large intestine. Certain types of food poisoning are described as intoxications and others as infections

Intoxications

Intoxication involves food poisoning in which the organism grows in food and releases a toxin from the cells. When the toxin is ingested along with the food, it gives rise to the **Food Poisoning Syndrome**.

The presence of organism in the food is irrelevant to disease production. It is the toxin that gives rise to the disease. Bacterial toxins that produce intoxications are the exotoxin types of either enterotoxin (affecting the gut) as in staphylococcal intoxication or neurotoxin (affecting the nervous system) as in botulism.

Another category of intoxications are the mycotoxicoses (due to ingestion of mycotoxins) and the diseases caused by algal toxins (shell fish poisoning). Generally, intoxications have short incubation periods.

Infections

These involve food poisoning caused by the ingestion of live organisms. The organisms grow in the gastrointestinal tract to produce the disease. Most microbial food poisonings fall in this category. For example, salmonellosis caused by *Salmonella* sp. like *Salmonella typhi*. Enteritis associated with food poisoning infections is due to the production of exotoxins or endotoxins that act as enterotoxins.

In certain other types of food poisoning, as in the case of *Clostridium perfringens*, live cells need to be ingested for the disease to occur but the organism does not grow and reproduce in the gut. Vegetative cells sporulate after ingestion and enterotoxin released causing the disease symptoms. Since live cells are needed to be ingested to cause the food poisoning, it can be considered as food-borne infection.

Food Poisoning Bacteria

What are these microbes?

It is estimated that every year more than Eighty one million people are affected by food borne illness every year. Illnesses such as food poisoning are becoming more common as our lifestyles change – for one thing, we eat out more and more food is being prepared in advance. We have no accurate figures on how much food poisoning is the result of mishandling by the consumer, but it is thought to be between 12 to 20%.

Food safety is all about reducing the risk of becoming sick from eating foods. The principles of food safety are easy to apply in the home, when eating in the outdoors, at your local shop when buying groceries, and even at a restaurant.

Most food poisoning incidents are a result of mishandling food – keeping it at the wrong temperature, incorrect re-heating, and cross contamination. To help you handle your food safely, take a look at the following information.

Remember that nearly ALL foods need to be handled with care.

Unit II

Food Poisoning by *Salmonella* sp.

Salmonella is associated mainly with raw meats, poultry and dairy products. However, many other foods have been implicated in outbreaks caused by *Salmonella*, and these include mayonnaise, salads, milk, orange juice, sprouted seeds and dairy products, etc. It gets into other foods by cross contamination from contact with raw foods, utensils, equipment and hands.

Salmonellosis tends to be more prevalent in the warmer, and eating food that has been kept in the temperature danger zone for too long a time is often the cause of the illness. Numbers as low as less than 10 cells have been responsible for causing foodborne disease. Sensitive individuals, e.g. frail elderly, young children and **Immunocompromised** people, are much more likely to become ill after eating only a small number of cells. These people should never be given egg flips unless they have been made from pasteurised egg.

In many countries, Enteritidis is a major problem in eggs. Therefore, although consumption of lightly cooked eggs, eg. Runny poached or soft boiled eggs, is not recommended for sensitive individuals, most people can still consume such foods without contracting an infection.

It usually takes 8 to 72 hours for symptoms of the illness to occur, but can take up to a few weeks, so **it is not necessarily the last meal you ate that caused it**. *Salmonella* cause a 'gastro-flu-like' infection which in most cases lasts about 2 to 5 days.

However, in some people they can lead to chronic conditions such as **Reiter's Syndrome** or **Reactive Arthritis.** *Salmonella* usually needs to grow to reach high enough numbers to make healthy adults sick. However, in high fat foods, eg. peanut butter, potato crisps and chocolate and liquids which pass through the stomach quickly, eg. unpasteurised juices, the presence of only a few organisms can cause illness. It can also survive in fairly dry and acidic foods for some time.

Because *Salmonella* is found on a wide range of foods, we should assume that most animal foods are contaminated and handle them accordingly. Most outbreaks occur through cross contamination from raw to cooked food, and contaminated food remaining in the temperature danger zone for too long. If cooking or reheating is inadequate then the bacteria will survive.

Food Poisoning by *Campylobacter*

This is probably one of the most common causes of foodborne illness. It is present in the gut of a wide range of animals, especially birds. Outbreaks have been linked to the consumption of undercooked poultry, mince and sausages, unpasteurised milk, and cooked foods that have been contaminated by raw foods like meat and poultry. Pets may also be a source of infection. *Campylobacter* doesn't grow well in foods, rather its presence in food can result in an infection. It is a problem because quite low numbers, ie. 500 to 600 cells of the bacteria can cause illness.Symptoms can take 2 to 7 days to appear and are gastrointestinal, lasting for about 5 days. A fever may also be present. A small number of people are left with a chronic condition called **Guillain Barre Syndrome** which can last for several weeks or months.

Food Poisoning by *Listeria monocytogenes*

Listeriosis is a comparatively rare form of foodborne illness, but it can be a very serious disease in a small group of individuals. Those who are pregnant, immunocompromised, young children and the frail elderly are quite susceptible to food poisoning from *Listeria* and unfortunately, every year a few people die. It has also caused occasional outbreaks of mild gastroenteritis in healthy people.

The symptoms are usually described as 'flu-like', although vomiting and discoloured urine can occur. Miscarriage can result if a pregnant woman is infected, even if she doesn't show the symptoms. The time from infection to symptoms can be anywhere between 8 to 90 days. *Listeria* is widely found on foods and most raw foods are likely to be contaminated.

Listeria is easily killed by heat although cooked foods can easily become recontaminated through poor food handling. This is one of the few pathogens that can grow in the refrigerator, so ready to eat food should never be stored in the fridge too long. Although it can grow in the fridge, it will do so only very slowly so make sure your refrigerator is keeping your food at or less than 5°C.

Food Poisoning by *Escherichia coli*

Many strains of *E. coli* are harmless and are found naturally in the gut of humans and animals. Traditionally its presence in foods has been an indication of faecal contamination of food or water. However, particular strains are pathogenic and **Traveller's diarrhoea** and **Haemolytic uraemic syndrome** (HUS) are caused by *E. coli* strains.

Although pathogenic types are rare, in the last few years there have been several foodborne outbreaks from certain strains of *E. coli* worldwide. A wide variety of foods have been implicated including unpasteurised apple and orange juices, sprouted seeds, fruit, raw milk cheese, salads and meat and meat products especially undercooked minced meat patties in hamburgers.

E. coli is easily killed by heating so cooking food properly is a basic method of control. Water can also be a source of the bacteria.

Food Poisoning by *Staphylococcus aureus*

Staphylococcus aureus, also known as 'Golden staph', is important from both a medical and food perspective. About half of us carry this organism in our skin and nasal passages. If you have an infected cut or sore, it can contain large numbers of *Staphylococcus.* Keep any cuts or sores well covered if you are handling foods.

Animals and poultry also carry this bacteria on their bodies and all raw meat and poultry products should be handled as though they are contaminated. Raw milk can also be a source of this bacteria.

It likes to grow in salty and sweet foods like those containing custard, hams, frankfurters, salads, cream-filled bakery products etc. The important thing to remember is that *Staphylococcus* produces a heat stable toxin as it grows and it is the toxin that makes us sick. If it is allowed to grow in food the toxin will remain even if the food is cooked again. The toxin takes only a very short time to make us sick (1 to 6 hours) and causes nausea, vomiting, abdominal cramps and diarrhoea as the usual symptoms.

Food Poisoning by *Clostridium botulinum*

Clostridium botulinum causes botulism. First recognized as a food-borne disease in the late 1800 and since then it has been a major concern of food processors and consumers. Currently, four categories of human botulism are recognized.

i.   Food-borne botulism is caused by eating food contaminated with **Pre-formed botulinum neurotoxin. (BoNT)**

ii. Infant botulism is caused by ingestion of viable spores that germinate, colonize and produce neurotoxin in the intestinal tracts of infants under one year of age.

iii. Wound botulism results from infection of a wound with spores of *C. botulinum*, which grow and produce neurotoxin in the wound.

iv. Unclassified includes cases of unknown origin and adult cases which resemble **Infant botulism.**

Clostridium botulinum is one of the more well known foodborne disease microorganisms due to the severe nature of the illness. As *C. botulinum* grows in food it produces a neurotoxin. This causes symptoms after about 12- 36 hours after consumption, although this can vary. Early symptoms include nausea, diarrhoea and vomiting, but neurological symptoms follow. Infant botulism is commonly reported in some countries in children under 1 year and the source of the infection is usually unknown. Sources of infection are likely to be dust and soil.

C. botulinum forms spores when it is heated and when conditions become favourable the spores germinate. In the past it has been mainly associated with canned foods but it has also been recently associated with vegetables in oil and some other foods.

Commercial canneries follow strict time and temperature heating schedules that are capable of killing the spores. In home bottling such regimes are not possible, if we are bottling at home stick to the high acid fruits, eg. pears, apples and stone fruit. If we bottle tomatoes, mango, papaya, banana or any other tropical fruit we must add some citric acid. Vegetables can only be safely bottled if bottled in vinegar.

If we want to produce our own vegetables in oil or flavoured oils we can keep them refrigerated for up to 10 days. If we want to bottle them, we need to acidify the vegetables.

Classification

Gram-positive, anaerobic, rod-shaped, spore-forming bacterium. There are seven types of *C. botulinum*, A, B, C, D, E, F and G, based on the serological specificity of the neurotoxin produced. Food-borne botulism is associated with types A, B, E and very rarely F.

The species is also divided into four groups based on Physiological differences as follows.

Group I All type A strains and proteolytic strains of type B and F, produce neurotoxin. Optimal temperature for growth is 37°C with growth occurring between 10 & 48°C. Spores have a high heat resistance (D_{100} = 25 mins) To inhibit growth, the pH must be Below 4.6, salt concentration above 10% and the a_w below 0.94

Group II All type E strains and non-proteolytic strains of types B and E. They have a lower optimum growth temperature (30°C) and grows at temperature as low as 3.3°C. Spores have a D_{100} values of less than 0.1 minute. Strains are inhibited by a pH below 5.0, salt concentrations above 5% or a_w below 0.97

Group III All type of C and D strains.

Group IV Type G strains.

Survivability Characteristics

Temperature, pH, a_w, redox potential, added preservatives and the presence of other microorganisms are the major factors controlling growth of *C. botulinum* in foods.

1. **Low Temperature**: Refrigerated storage is used by present or inhibit the growth of *Clostridium botulinum*. The established lower limits are 10°C for group I and 3.3 for group II. Production of neurotoxin generally requires weeks at the lower temperature limits for group I and group II organisms. Survival of spores of group II in pasteurized, refrigerated products is of concern because of their ability to grow at refrigeration temperatures.

2. **Thermal inactivation**: Thermal processing is used to inactivate spores of *Clostridium botulinum* and is the most common method of producing shelf stable foods. Spores of types A and B are the most heat-resistant, having D_{121} values of between 0.1 and 0.2 min. These spores are of particular concern in the sterilization of cannned low-acid foods. The

canning industry has adopted a D value of 0.2 minutes at 121°C as a standard for calculating thermal processes. The Z value (the temperature change necessary to cause a 10-fold change in the D value) for the most resistant strains is approximately 10°C, which has also been adopted as a standard. Despite variations in D and Z values, the adoption of a 12 D process as the minimum thermoprocess applied to commercial canned, low-acid foods by the canning industry has ensured the production of safe products.

3. **pH**: The minimum pH allowing growth of *C. botulinum* group I is 4.6, for group II is 5.0. Substrate, temperature, nature of the acidulant agent, presence of preservatives, a_w, and Eh are the factors that influence the acid tolerance of C. botulinum. Acid-tolerant microbes such as yeasts and moulds may grow in acidic products and raise the pH in their immediate vicinity to a level that allows growth of *C. botulinum.*

4. **Salt and a_w:** The salt concentration in the aqueous phase, calle dthe brine cncentration is critical. The growth limiting brine concentrations are about 10% for group I and 5% for group II. The solute used to control a_w may influence these limits. Generally, NaCl, KCL, glucose and sucrose show similar effects, while glycerol allows growth at lower a_w.

5. **Atmosphere and Eh:** Modified Atmosphere Packaging (MAP) is being increasingly used to extend the shelf life and improve the quality of foods. MAP has been a concern because of creating conditions that might promote growth of *C. botulinum*. CO_2 is used in MAP to inhibit spoilage and pathogenic microbes, but CO_2 may stimulate *C. botulinum.* Levels of 15-30% for CO_2 does not inhibit the organism. While it is commonly assumed that *C. botulinum* cannot grow in foods exposed to oxygen, the redox potential (Eh) of most such foods is usually low enough to allow its growth since initial atmospheres containing 20%. O_2 does not delay neurotoxin production by *C. botulinum* in pork.

6. **Preservatives:** Nitrite has several functions in cured meat products, an important role is the inhibition of *C. botulinum*. Its effectiveness in the inhibition of the organism is dependent on complex interactions among pH, salt, heat treatment, time and temperature of storage and the composition of food. Nitrite is depleted from cured foods and the depletion rate is also dependent on product formulation, pH and time and temperature during processing and storage. A significant contribution of nitrite to the inhibition of *Clostridium botulinum* continues even when nitrite is no longer detectable. Nitrite reacts with many cellular constituents and appears to inhibit *C. botulinum* by more than one

mechanism, including reaction with essential iron-sulphur proteins to inhibit energy yielding systems in the cell. The reaction of nitrite, or nitric oxide, with secondary amines in meats to produce nitrosamines, some of which are carcinogenic has led to regulations limiting the amount of nitrite used. Sorbates, parabens, nisin, phenolic antioxidants, fumarates are also active against *C. botulinum.*

7. **Other Microorganisms:** The growth of other microorganisms in foods has a very significant effect on the growth of *C. botulinum.* Acid-tolerant yeasts and moulds may make the environment more favourable for growth of *C. botulinum*. Other microbes may inhibit *C. botulinum*, either by changing environment or by producing specific inhibitory substances or both. For example, lactic acid bacteria including *Lactobacillus, Pediococcus* and *Streptococcus* can inhibit growth of *C. botulinum* in meat products by reducing the pH and by the production of bacteriocins.

8. **Inactivation by Irradiation:** C. botulinum spores are the most rediation-resistant spores. Redappertization is designed to reduce the number of viable spores of the *C. botulinum* by 12 log cycles. Spores are more sensitive in the presence of O_2 or preservatives and at temperatures above 20°C.

Reservoirs

Research shows that spores of *C. botulinum* are commonly present in soils and sediments, but their numbers and types vary depending on the location. Food surveys have largely focused on fish (un-eviscerated salt-cured fish), meats and infant foods, primarily honey. The types most often associated with meats are A and B. These types may also be present in fruits and vegetables, particularly those in close contact with the soil. Different agricultural practices, such as the use of manure as fertilizer may affect the level of contamination. Products in which contamination has often been detected include asparagus, beans, cabbage, carrots, cherries, peaches and tomatoes. A product of particular concern because of the high number of spores found is cultivated mushrooms. The potential presence of spores in honey and other infant foods is problematic because in some infants, the spores can colonize the intestines, produce neurotoxin and cause infant botulism. Only a very low incidence of C. botulinum spores has been found in other foods, including dairy products, vacuum-packed products and ready-to-eat foods.

Other important reservoirs could be home-preserved vegetables or meats like ham, fermented sausages and canned products. Temperature abuse of home prepared foods continues to be an important cause of botulism.

Characteristics of the Disease

Food-borne botulism varies from a mild illness, which disregarded could be a serious disease that may be fatal within 24 hours. Symptoms typically appear 12-36 hrs after ingestion of neurotoxin. Earlier the symptoms appear, the more serious is the disease. The first symptoms are generally nausea and vomiting (type B and E), followed by neurological signs and symptoms including visual impairment (blurred or double vision, fixed and dilated pupils), difficulty in speaking and swallowing, dry mouth, throat and tongue infections, sore throat as seen frequently in type B strains, general fatigue and lack of muscle coordination, and respiratory impairment which are the main causes of death.

Other gastrointestinal symptoms may include abdominal pain, diarrhoea, or constipation.

Botulism may be confused with other illness, including other forms of food-borne poisoning, carbon monoxide poisoning. In botulism, the neurological signs appear first in the cranial nerve area (eyes, mouth and throat) and then descend.

The most common symptom of infant botulism is constipation. The infants usually show a generalized weakness and weak cry. Other symptoms may include feeding difficulty and poor sucking, lethargy, lack of facial expression, irritability and progressive "**Floppiness**". Respiration arrests occur frequently but are seldom fatal.

Initially, treatment of food-borne botulism tries to remove or inactive the neurotoxin by neutralization of circulating neurotoxin with antiserum or to use enema to remove residual neurotoxin from the bowel and gastric lavage or treatment with emetics. Subsequent treatment is mainly to counteract the paralysis of the respiratory muscles by artificial ventilation.

Infective Dose

Little is known concerning the minimum toxic dose of *C. botulinum* and its neurotoxins. There is no tolerance for the presence of neurotoxin or for conditions permitting growth of *C. botulinum*. The mouse LD_{50} for BoNT is approximately 0.1 ng/kg.

Pathogenicity

C. botulinum produces eight antigenically distinct toxins, designated types A, B, C, D, E, F and G. All the toxins except C_2 are neurotoxins. C_2 is an ADP ribosylating enzyme.

Laboratory Diagnosis

In view of the severity of the illness, rapid identification of the food source.

Food Poisoning by *Clostridium perfringens*

This is a less well known bacterium which causes severe stomach cramps and a mild form of diarrhoea that lasts only about 24 hours and therefore tends to go unreported. However, it is probably fairly common and can be fatal in the frail elderly or people who are already ill.

The symptoms begin in about 8 to 22 hours after the food is eaten. Large numbers of the bacteria have to be eaten before you get sick, but because the bacteria can grow very fast, the number can double every 18 minutes. It doesn't take long for large numbers to build up. The cause of the illness is a toxin that is produced when the bacteria forms spores in the gut. It's the presence of the toxin that makes us sick.

C. perfringens is widely found in soil and in intestinal tracts of humans. It is usually associated with food that has been allowed to stay warm for several hours. During cooking, which will kill most types of bacteria, *C. perfringens* turns into another form called a spore. A spore is like a seed, it stays dormant in the food until conditions are favourable, then like a plant seed it will germinate and grow. The spores of *C.perfringens* are very heat resistant and will withstand boiling for several hours.

It likes the sort of conditions you get in casseroles, stews, gravies, pie fillings and any other bulk cooked foods when they cool. In the nice warm conditions of cooling food, the spores germinate and grow. Whenever you cool food, make sure you cool it quickly by transferring it into a shallow container and refrigerating it when the steam stops rising. A large stockpot, even in a commercial fridge can take several days for the centre temperature in the pot to reach 5°C.

Food Poisoning by *Bacillus cereus*

Bacillus cereus causes two different types of food poisoning: the diarrhoeal type and the emetic type. The diarrhoeal type of food poisoning is caused by an enterotoxin produced during vegetative growth of *B. cereus* in the small intestine while emetic toxin is produced by cells growing in the food. *B. cereus* food poisoning is under reported, as both types of illness are relatively mild and usually lasts less than 24 hours.

Characteristic Features

B. cereus is a gram-positive, spore forming, motile, aerobic rod but it grows well anaerobically. The genus is divided into six different subgroups and *B. cereus* is classified in the Bacillus subtilis group. Four members of this group are closely related: *B. cereus, B. thuringiensis, B. anthracis* and *B. mycoides*. The variation among these four species is mainly due to genes on episomes rather than genes on the chromosome.

Bacillus sp. sporulate easily after 2-3 days on most media. It grows well on food that has been heat treated (48°C) which usually causes spore germination. The organism is unable to grow below 10°C.

Survivability characteristics

The organism can survive pasteurization through **Sporulation.** It cannot grow in milk and milk products stored at temperatures between 4°C and 8°C. but the **Psychrotrophic** stains that have developed can grow at temperatures as low as 4-6°C. Since it sporulates, it can survive pH changes (acidity) and it is problem in home-canned foods.

Spores

The spore of *B. cereus* is an important factor in food-borne illness as it is more hydrophobic than any other *Bacillus* sp. which enable it to adhere to several types of surfaces. Hence, it is difficult to remove from equipments during cleaning. *B. cereus* spores also possess appendages or pili that help in adhesion. These adherence properties not only enable spores to resist normal sanitation procedures, and thus contaminate food during processing., but also aid in binding to epithelial cells. Spore adhesion to epithelial cells followed by germination and

production of enterotoxin may explain the long incubation periods observed in some food-associated outbreaks.

Reservoirs

The organism is widespread being frequently isolated from soil and growing plants. From this natural environment it is easily spread to foods, especially those of plant origin. Through cross-contamination, it may then be spread to other foods such as meat products. The problems in milk and milk products are caused by *B. cereus* which is spread from soil and grass to the udders of the cow and into the raw milk.

Food-Borne Outbreaks

The number of outbreaks of *B. cereus* food poisoning is highly underestimated, the main reason being the relatively short duration of both types of diseases. The dominant type of illness caused by *B. cereus* differs from country to country. In Japan, the emetic type is reported about 10 times more frequently than the diarrhoeal type and in Europe and North America, the diarrhoeal type is more frequently reported. Confirmation of *B. cereus* as the cause of food-borne outbreak requires:

i. Isolation of strains of the same serotype from the suspected food and from faeces or vomitus of the patients.

ii. Isolation of significant numbers of *B. cereus* serotype known to cause food-borne illness from the suspected food or from the faeces or vomitus of affected individuals.

iii. Isolation of significant numbers of *B. cereus* from the suspected food and from the faeces or vomitus of the patients.

Characteristics of the Disease

In some cases, both the illness caused by *B. cereus* can be seen. Although there has been a debate about *B. cereus* food poisoning being an intoxicating type, the long incubation period (> 6 hours; average 12 hours) and studies revealing the degradation of the enterotoxin in the gut before reaching the ileum have made this fact unlikely. Infective doses range from 5×10^4 to 10^{11} cells per gram. But foods containing more than 10^4 cells/g may not be safe for consumption.

Pathogenicity

B. cereus toxins fall into 4 groups,

1. **The haemolysins** The first of these is the well defined cerolysin. These toxins are produced in an inactive, oxidized form with sulphydryl groups in disulphide bonds; reduction frees the sulphydryl groups to produce the active form. The binding site of the thiol activated cytolysins on the eukaryotic cell membrane is cholesterol and the result of binding is a characteristic morphological pitting and micropunching of cell membrane, visible through electron microscope, the result of which is loss of control of free ion exchange and intracellular K^+ ions. But net flow of ions and water into the cell causes swelling and the cell ruptures. After intravenous injection in mice, cereolysin is instantly lethal.

2. **The diarrhoeal enterotoxin** For many years cereolysin was thought to be the only toxin causing all the diarrhoeagenic episodes. It became clear in the 1970s that it was possible to distinguish and separate an entity, termed enterotoxin because of its ability to produce fluid accumulation in ligated rabbit ileal loops, from the phospho-lipolytic and haemolytic entities. It is regarded as a multi-component protein complex. In addition to eliciting fluid accumulation in ligated loops, it causes severe mucosal damage and these activities reflect in increased vascular permeability with marked necrosis in rabbit or guinea pig skin tests. The degree of production of this necrotic enterotoxin has also been shown to correlate roughly with the severity of infection in non-gastrointestinal *B. cereus* infections. This toxin is responsible for death in severe cases of *B. cereus* infection.

   B. cereus produces at least two different enterotoxins. Although several proteins may be involved in *B. cereus* food-borne illness, only one type of enterotoxin (B component of the haemolysin BL) is likely responsible for the major symptom. The haemolysin is made of three components: L1, L2 and B (the enterotoxin) and all the three must be present for full enterotoxin activity.

   Another toxin, enterotoxin T, is composed of a single protein. Much has not been unravelled about the T toxin.

3. **The emetic toxin** Highly stable compound which is formed at temperatures $< 40°C$ but survives $126°C$ for 1.5 hours, pH extremes and proteolytic enzymes. It may be associated

with sporulation or breakdown products from foods. It is named *cereulide* and it is thought to be enzymatically synthesized polypeptide.

4. The phospholipase C group of enzymes They are three in numbers, phosphatidylcholine hydrolase, phosphatidylinositol hydrolase and sphingomyelinase. They act on the phospholipases C and sphingomyelin of biological members. Under normal circumstances, they probably do not gain access to the phospholipids of cell membranes, but they may possibly act secondarily after the exposure of these substrates in the course of other pathological process.

Laboratory Diagnosis and Identification

Media: Polymyxin pyruvate egg yolk mannitol bromothymol blue agar (PEMBA) is most reliable. Another medium, phenol egg yolk polymyxin agar is also used for selective isolation of *B. cereus* from food samples.

Method: Serially diluted food samples can be enumerated (spread plate) using the selective media. After incubation (30°C for 24-48 hours) the specifically coloured colonies (dull peacock blue) confirms the presence of *B. cereus*. In addition, the lecithinase activity can also be detected. They are further confirmed by staining and subculturing onto blood agar for observing **β-haemolysis.**

Serially diluted food sample

↓

Enumerated using PEMBA

↓ Incubated at 30°C for 24-48 hours

Large colonies (3-7 mm diameter) and dull peacock blue confirm the presence of

B. cereus (showing zone of opacity indicating lecithinase activity)

↓

Gram-stained and subcultured onto blood agar, the organism being confirmed by the

β-haemolytic appearance

Table 2.1: Food Poisoning Bacteria

Sl. No.	Species	Infective dose (cells/g)	Source/reservoir	Typical foods	Symptoms (incubation time)
1.	B. cereus	~100,000	Soil, dust, sediments	Cooked rice, meat, vegetables	Vomiting, nausea, diarrhoea (1-16h)
2.	B. subtilis	~100,000	Soil, dust, vegetation	Cereal + dairy products	Vomiting, diarrhoea (1-14h)
3.	Campylobacter	~100	Farm animals, pets, man	Poultry, raw milk	Diarrhoea, fever, abdominal pain (2-5h)
4.	Cl. botulinum	Toxin is lethal in very low doses	Soil, mammals, birds, fish	Fish, meat, home-preserved vegetables	Double vision, dry mouth, respiratory difficulties (1-3 days)
5..	Cl. perfringens	~1,000,000	Soil, animals, man	Cooked meat	Diarrhoea, abdominal pain (8-18h)
6.	E. coli (VTEC)	Very high infectivity (e.g. 10s of cells)	Cattle, sheep	Undercooked meat cheese	Bloody diarrhoea, kidney and neurological complications (1-6days)
7.	Listeria	Relatively low infectivity	Wide environmental distribution	Cheese, raw milk, coleslaw	Mild flu, meningitis, (1-10wks)
8.	Salmonella	Risk varies	Animals, man	Meat, poultry, eggs, dairy produce, chocolate	Diarrhoea, fever, abdominal pain (12-24h)
9.	Staphylococcus aureus	100,000-1,000,000	Man	Ham, poultry/egg products, cream, cheese	Vomiting, diarrhoea (2-6 h)
10.	Yersinia	Relatively low infectivity	Water, domestic + wild animals	Milk, pork, poultry	Watery diarrhoea, abdominal pain, fever (3-7 days)

***Bacillus cereus* and other *Bacillus* species**

This pathogen can cause two types of foodborne illness — the diarrhoeal type and the emetic or vomiting type. Like *C. perfringens* the illness is a mild one, but unpleasant nevertheless.

The diarrheal type occurs within 8 to 16 hours of eating the food and lasts for about 24 hours. Foods involved vary from starchy vegetables, meat products, cereal foods, sauces, puddings and spices. A much shorter time is required for symptoms of the emetic type to appear, 0.5 to 5 hours. The most common food associated with the emetic type is rice. Cooked rice should always be cooled and stored in the refrigerator.

B. cereus can form heat resistant spores and a heat resistant toxin. If cooked food is allowed to cool slowly the spores can germinate. If growth occurs then the toxin can form under certain conditions. Reheating or lightly cooking the food will not destroy this toxin.

Although this bacteria can grow and produce toxin at refrigeration temperatures, it does so much more slowly than at room temperature. Precooked food should not be stored in the refrigerator for more than 2-3 days.

Food Poisoning by *Brucella* Sp.

Even though brucellosis is mainly classified under zoonoses (infection transmitted from animal to man), it can be referred as an example for food borne disease since it is also transmitted through milk and milk products. Brucellosis is also named as undulant fever, Malta fever or Mediterranean fever. It is caused by different species of the genus Brucella and characterized by intermittent or irregular febrile attacks, with profuse sweating, arthritis and an enlarged spleen.

Taxonomy

The genus Brucella can be divided into six species, four of which can infect man by direct or indirect contact with infected animals:

Brucella abortus Primary host cattle, but also transmissible to camels, deer, dogs, horses, sheep, goats, pigs and man.

Brucella melitensis Primary host goats and sheep, but can infect many other species, including man. This is most virulent in man, discovered by Bruce (army surgeon in Malta)

Brucella suis Primary host is dependent on the biovar, biovars 1, 2 and 3 infect pigs, biovar 4 infects reindeers and biovar 5 infects small rodents. All biovars can cause disease in man.

Brucella canis Primary host is the dog, man may occasionally be infected.

Brucella ovis Primary host is sheep, does not cause disease in man.

Brucella neotomae Infection limited to the desert wood rat.

The organism is a gram-negative, round or oval intracellular cocco-bacilli, about 0.4mm in diameter. Arranged singly, sometimes in pairs, short chains or small clusters. Does not produce capsules, spores or flagella.

Growth Characteristics

It requires multiple amino acids and vitamins for growth. The optimum pH range is 6.6-7.4 and temperature range is 20-40°C with an optimum at 37°C.

Survivability

The organism is killed after heating at 60°C for 10 minutes, therefore easily eliminated from milk by pasteurization by holder method and HTST. Brucellae can remain viable in refrigerated milk for 10 days and in cheese for up to 90 days. They may persist in meat for several weeks. The organism is moderately sensitive to acid and hence cannot survive in properly fermented milk products like cheese or yoghurt. It is also sensitive to disinfectants and antibiotics. Brucellosis is most prevalent under conditions of advanced domestication of animals in the absence of hygiene. The organism can travel long distances in milk and dust. It can survive for weeks, or months in favourable conditions of water, urine, faeces, damp soil and manure. Cells of Brucella are more resistant to lysozyme but are apparently destroyed by smoking. In raw milk, at room temperature, it dies out rapidly with the production of acid. Acid production seems to be the cause of its rapid death in butter and cheese.

Reservoirs

Main reservoirs of human infection are cattle, sheep, goats, swine, buffaloes, horses and dogs. The infected animals excrete the organism in the milk which is one of the important vehicles of food-borne brucellosis. Dairy products prepared from untreated raw milk like butter, cream, soured milk, meat and meat products can also carry the organism. Fresh raw vegetables can also carry infection if grown on soil containing manure from infected farms. Water contamined with the excreta of infected animals may also serve as a source of infection.

Characteristics of the Disease

It has a highly variable incubation period which usually lasts from 1-3 weeks, but may last for as long as 6 months or more. The disease can vary from an acute febrile disease to a chronic low grade ill defined disease, lasting for several days, months or occasionally years.

The acute phase is characterized by a sudden or insidious onset of illness with

Swinging **Pyrexia** especially at nights (40-41°C), rigors and sweating.

Anthralgia/Arthritis involving larger joints such as hip, knee, shoulder and ankle.

Low back pain.

Headache, insomnia.

Small firm **Splenomegaly** and **Hepatomegaly.**

Leucopenia with relative lymphocytosis.

The acute phase subsides within 2-3 weeks. The infection being intracellular, may persist giving rise to subacute or relapsing disease.

Pathogenicity

Upon ingestion by the humans, the organisms penetrate gastrointestinal mucosa and in direct contact, organisms enter through breaks in the skin or the conjunctiva. After penetration, the organisms spread via lymphatics through regional bodies nodes and thoracic duct to the bloodstream and get localized in spleen, bone marrow, liver, kidneys, endocardium and elsewhere. In animals, the organism stay confined to the mammary glands, genital organs and in pregnant uterus, foetal fluids and membranes. No exotoxins have been detected.

Food Poisoning by *Clostridium perfringens*

Clostridium perfringens actually causes two different human diseases that can be transmitted by food, i.e., *C. perfringens* type A Food poisoning and necrotic enteritis.

General charactersitics

C. perfringens is a gram-positive, rod-shaped, encapsulated non-motile bacterium variable size that is capable of causing a broad spectrum of human and veterinary diseases. the pathogenicity is largely derived from its prolific ability to express protein toxins, including at least two toxins, *C. perfringens* enterotoxin and b-toxin, that are active on the human gastrointestinal tract.

The vegetative cells of *C. perfringens* can double in less than 10 minutes, allowing the organism to multiply very rapidly in food and it forms spores that are highly resistant to environment conditions such as heat, radiation and dessication. *C. perfringens* is an anaerobic but can tolerate some exposure to air and compared with many other anaerobes, requires only relatively modest reductions in oxidation-reduction potential for growth.

Survival Characteristics

Temperature The heat resistance of *C. perfringens* spores contributes to the organism's ability to cause food poisoning by allowing it to survive to partially cooked foods. The hear resistance properties of *C. perfringens* spores depend on both environmental and genetic factors.

The medium in which a *C. perfringens* is heated clearly influences its heat resistance. In a relatively protective medium such as cooked meat medium, many spores will survive boiling for an hour or longer. The involvement of genetic factors in spore heat resistance has been established by observations indicating that spores made by different *C. perfringens* strains vary considerably in their heat resistance properties. Spores made by food vary considerably in their heat resistance properties. Spores made by food poisoning isolates are generally more heat-resistant than spores made by poisoning isolates are generally more heat-resistant than spores made by *C. perfringens* isolates from other sources. Partially cooked foods not only fail to rid spores but can actually facilitate the development of *C. perfringens* type A food poisoning, since

heating (70-80˚C for 20 minutes) is an excellent way to induce the germination of *C. perfringens* spores.

Water Activity: *C. perfringens* is less tolerant of low a_w environments than other common gram-positive food borne-pathogen like *Staphylococcus aureus*. The lowest a_w supporting vegetative growth of *C. perfringens* is reported to be 0.93-0.97, depending on the solute used to control the a_w of the medium.

Redox Potential: Relative to other anaerobes, *C. perfringens* does not require an extremely reduced environment for its growth. If the redox potential of the environment is low, *C. perfringens* will then modify the redox potential of its surrounding environment (by producing reducing molecules such as ferredoxin) to produce more optimal growth conditions. The redox potential of many common foods (like raw meats) is often low enough to permit the growth of *C. perfringens.*

pH: Growth of *C. perfringens* is also sensitive of pH extremes, with optimal growth occurring at pH 6-7 (pH that is commonly found in the meat and poultry products that usually serve as food vehicles for *C. perfringens* type A food poisoning and severe inhibition of growth occurring at pH of <5 and >8.

Chemicals: The effectiveness of curing agents on limiting *C. perfringens* growth in foods has not received much research attention since concentrations of curing salts needed to significantly inhibit survival of *C. perfringens* cells may exceed commercially acceptable levels. i.e., inhibition of *C. perfringens* growth may require at least 6-8% Nacl, 10,000ppm of $NaNO_3$, or 400ppm of $NaNO_2$.

1. The simultaneous coapplication of other preservation factors such as heating and non-neutral pH increases the sensitivity of *C. perfringens* to curing salts.
2. The simultaneous use of several curing agents often produces a synergistic inhibition of *C. perfringens* growth.
3. Foods may contain initial burdens of *C. perfringens* cells and spores lower than those used in laboratory studies evaluation the effectiveness of curing agents for inhibition *C. perfringens* growth.

The above-mentioned preservation agents and environmental factors play a major role in inhibiting the outgrowth of germinating of *C. perfringens* spores in foods.

Reservoirs for *C. perfringens* type A Food Poisoning

The organism is ubiquitous throughout the natural environment.

1. Soils (10^3-10^4 cfu/g)
2. Foods (50% of frozen foods)
3. Dust
4. Intestinal tracts of humans and domestic animals (human faeces contains about 10^3-10^6 cells per gram)

Surveys have suggested that <5% of all *C. perfringens* isolates actually carry the "*cpe*" gene, which is considered essential for producing *C. perfringens* type A food poisoning symptoms. Hence, it is suggestive to find out reservoirs for "*cpe*" positive *C. perfringens* which is the actual enterotoxigenic organism.

Food Poisoning by *Escherichia coli*

Escherichia coli strains are a common part of the normal facultative anaerobic microflora of the intestinal tracts of humans and warm-blooded animals. *E. coli* strains that cause disrrhoeal illness are categorized into specific groups based on virulence properties, mechanism of pathogenicity, clinical syndromes and distinct O:H serotypes. These categories include

Enteropathogenic *E. coli* strains (EPEC) EPEC can cause severe diarrhea. Humans are an important reservoir.

Enterotoxigenic *E. coli* strains (ETEC) ETEC cause infantile diarrhea in developing countries. They are also the agents most frequently responsible for **Traveller's diarrhea.** ETEC colonize the proximal small intestine by fimbrial colonization factors and produce a heat-labile or heat-stable enterotoxin that elicits fluid accumulation and a diarrhoeal response. Humans are the principal reservoir of ETEC.

Enteroinvasive *E. coli* strains (EIEC) ETEC cause non bloody diarrhea and dysentery similar to that caused by Shigella sp., by invading and multiplying within colonic epithelial cells. The principal site of bacterial localization is the colon, where EIEC invade and proliferate in epithelial cells, causing cell death. Humans are a major reservoir.

Enteroaggregative *E. coli* strains (EAggEC) Recently, they have been associated with persistant diarrhea in infants and children in several countries. They are different from the other types of pathogenic E. coli because of their ability to produce a characteristic pattern of aggregative adherence on HEp-2 cells. Considerably more epidemiologic information is needed to elucidate its significance as an agent of diarrhoeal disease.

Enterohaemorrhagic *E. coli* (EHEC) EHEC were first identified as human pathogens in 1982 when *E. coli* of serotype O157:H7 was associated with two outbreaks of haemorrhagic colitis. All EHEC produce factors cytotoxic to Vero cells. EHEC carries more importance because of its recent origin during 1980s with two food-borne outbreaks of haemorrhagic colitis. It is different from common strains of *E. coli* by their inability to grow well at temperatures of >44.5˚C, inability to ferment sorbitol within 24 hours, inability to produce -glucuronidase, possession of an attaching and effacing gene, carriage of a 60-M Da plasmid and expression of an uncommon 5000 – 8000 MW (outer membrane protein).

Survivability Characteristics

Acid Tolerance *E. coli* O157:H7 is uniquely tolerant to acidic environments thus making this an important food-borne pathogen associated with high-acid foods. It can survive fermentation, drying and storage of fermented sausage for up to 2 months at 4˚C.

Thermal Inactivation The pathogen has no unusual resistance to heat with D values at 60˚C for 45 seconds. The presence of fat increases the thermal tolerance of *E. coli* O157:H7. Pasteurization of milk is an effective treatment. Proper heating of foods of animal origin, i.e., heating foods to an internal temperature of at least 68.3˚C is an important critical control point to ensure inactivation of *E. coli* O157:H7.

Food Poisoning by *Shigella sp.*

Bacillary dysentery or Shigellosis is caused by members of the *Shigella* species. These are host-adapted organisms and infect only humans and other primates. With a low infections dose required to cause disease coupled with oral transmission via faecally contaminated food and water, it is not surprising that dysentery caused by *Shigella* sp. follows in the wake of many natural and man-made disasters.

Characteristics of the Disease

Disease caused by *Shigella* sp. is distinguished from disease caused by most of the other food-borne pathogens in at least two important aspects: Production of bloody diarrhea or dysentery and the low infectious dose that can cause clinical symptoms. The clinical picture of shigellosis ranges from a mild watery diarrhea to severe dysentery, the former usually preceding the latter. The dysentery stage of disease correlates with extensive bacterial colonization of the colonic mucosa. The bacteria invade the epithelial cells of the colon, spread from cell to cell, but penetrate only as far as the lamina propria. The incubation period for shigellosis is 1-7 days. Strains of *S. dysenteriae* type 1 cause the most severe disease, while S. sonnei produce the mildest.

An important aspect of *Shigella* pathogenesis is the extremely low infections dose (as low as 200 organisms). It is a **self-limiting disease** in normally healthy patients.

While improvements in sanitary and hygienic conditions can help contain secondary spread of Shigellosis, the single most effective means of preventing secondary transmission is hand washing.

Pathogenicity

Virulence Factors The clinical symptoms of Shigellosis can be directly attributed to the hallmarks of *Shigella* virulence: the ability to invade epithelial cells of the intestine, multiply intracellularly, and spread from cell to cell.

Along with the ability to colonize and cause disease, an intrinsic part of a bacterium's pathogenicity is its mechanism for regulating expression of the genes involved in virulence. Virulence in *Shigella* sp., is regulated by growth temperature. The non-invasive phenotype can regain virulence properties. Regulation of gene expression in response to environmental temperature is a useful bacterial strategy. By sensing the ambient temperature of the mammalian host to trigger gene expression, this strategy permits shigellae to economize energy that would be expanded on the synthesis of virulence products when the bacteria are outside the host. The system also permits the bacteria to coordinately regulate expression of multiple unlinked genes that are required for the full virulence phenotype.

While food-borne infections due to Shigella sp., may not be as frequent as those caused by other food-borne pathogens, they have the potential for explosive spread because of the extremely low infections dose needed to cause overt clinical disease.

Food Poisoning by *Vibrio* Species

There are over 20 species of *Vibrio* which are described including at least 12 capable if causing infection in humans. Of the 12, 8 have been shown to be directly food associated. One of the most consistent aspects of *vibrio* infections is a recent history of seafood consumption. Vibrios, which are generally the predominant bacterial genus in estuarine waters, are found associated with a great variety of seafoods.

The classical methods of identification are continued as for example, string test, growth on alkaline medium, oxidase test, etc. Molecular techniques to identify the presence of vibrios in foods are becoming more common and are proving to be a very powerful adjunct to more traditional taxonomic methods. The levels of most of the vibrios in both surface waters and shellfish show definite seasonal correlation, generally being greater during the warm weather months. Seasonality, is most notable for V. vulnificus and V. parahaemolyticus infections, whereas those of some vibrios such a V. fluvialis occur throughout the year. Unfortunately, because vibrios are part of the normal estuarine microflora and not a result of faecal contamination, vibrio infections will not likely be controlled through shellfish sanitation programs. It is thus essential that raw seafood be adequately refrigerated or iced to prevent significant bacterial growth.

Disease Symptoms and Infectious Dose

Majority of infections with *V. cholerae* O1 are mild or even asymptomatic. Profuse watery diarrhea with premonitory symptoms like anorexia, abdominal discomfort, and simple diarrhea are noted. Initially the stool is brown with feacal matter, but soon the diarrhea assumes a pale grey colour with an inoffensive, slightly fishy odour. Mucus in the stool imparts the characteristic "rice water" appearance. Vomiting is often present.

In most severe forms, termed "cholera gravis" the rate of diarrhea may quickly reach 500 to 1000 ml/hour, leading rapidly to tachycardia, hypotension and vascular collapse due to dehydration. Skin turgor is poor, giving the skin a doughy consistency with sunken eyes, hands and feet become wrinked, as after long immersion ("washerwomen's hands") leading to death. Other symptoms include abdominal cramps and fever with nausea and vomiting.

In healthy volunteers, doses of 10^{11} cfu of *V. cholerae* in buffered saline (pH 7.2) are required to consistently cause diarrhea.

Pathogenicity

Virulence Mechanisms Infections due to V. cholerae O1/0139 begins with the ingestion of food or water contaminated with the organism. After passage through the acid barrier of the stomach, vibrios colonize the epithelium of the small intestine by means of sone or more adherence factors, invasion into epithelial cells or the lamina propria does not occur. Production of CT disrupts ion transport by intestinal epithelial cells. The subsequent loss of water and electrolytes leads to severe diarrhea characteristic of cholera.

Food Poisoning by *Yersinia enterocolitica*

The genus Yersinia comprises 11 species. As members of the family Enterobacteriaceae, yersiniae are oxidase negative, gram-negative, rod-shaped facultative anaerobes which ferment glucose. The genus includes three primary pathogens of humans and several other species which may cause opportunistic infections. The three pathogenic species are *Y. pestis*, the causative agent of bubonic and pneumonic plague, *Y. pseudotuberculosis*, an intestinal pathogen of rodents which occasionally infects humans, and *Y. enterocolitica* a common intestinal pathogen of humans.

Y. enterocolitica first emerged as a human pathogen during the 1930s. It exhibits between 10 and 30% DNA homology with other genera in the Enterobacteriaceae. It is a highly heterogenous, being divisible into a large number of subgroups, chiefly according to biochemical activity and lipopolysaccharide O antigens. Most primary pathogenic strains of humans and domestic animals occur within biovars 1B, 2, 3, 4 and 5. *Y. enterocolitica* strains of biovar 1A are generally obtained from terrestrial and freshwater ecosystems and are often referred to as environmental strains. Not all isolates of *Y. enterocolitica* obtained from soil, water or unprocessed foods can be assigned to a biovar. These strains invariably lack the characteristic virulence determinants of the primary pathogenic yersiniae and may represent a novel non-pathogenic subtypes or even new *Yersinia* species.

Characteristics of Infection

Infection exhibits a wide range of clinical presentation and outcomes. Apart from self limiting diarrhea, yersiniosis may also give rise to a variety of suppurative and autoimmune complications which is underlined by factors such as age and immune status.

Acute Infection *Y. enterocolitia* enters the gastrointestinal tract after ingestion in contaminated food or water. The infective dose for humans is likely to exceed 104 cfu. Gastric acid appears to be a significant barrier to infection. Most symptomatic infections with *Y. enterocolitia* occur in children less than 5 years of age. It occurs as diarrhea, often accompanied by low grade fever and abdominal pain. The character of the diarrhea varies from watery to mucoid or bloody. The illness lasts for a few days to 3 weeks. Rarely, acute enteritis may progress to intestinal ulceration and perforation to ileocolic intussusceptions, toxic megacolon, or mesenteric vein thrombosis. Sore throat is a frequent accompaniment and may dominate the clinical picture of older patients.

In children, older than 5 years and adolescents, acute yersiniosis often occurs as a pseudo-appendicular syndrome. The usual features of this syndrome are abdominal pain and tenderness localized to the right lower quadrant. These symptoms are usually accompanied by fever, with little or no diarrhea. This closely resembles appendicitis.

Bacteraemia is a rare complication of infection except in patient who are immunocompromised or in an iron overloaded state. Infections with *Y. enterocolitia* are noteworthy for the large variety of immunological complications, including reactive arthiritis, erythema nodosum, iridocyclitis, glomerulonephritis, carditis and thyroiditis which follow acute infection. Of these, reactive arthritis is the most widely recognized. Arthritis (migratory) typically follows the onset of diarrhea or the **pseudo-appendicular syndrome** by 1 to 2 weeks. The joints most commonly involved are the knees, ankles, toes, tarsal joints, fingers, wrists and elbows.

Pathogenicity

Y. enterocolitia is an invasive pathogen which induces an inflammatory response in infected tissues. The distal ileum, in particular the gut-associated lymphoid tissue, bear the brunt of infection, although adjacent regions of the intestine and the mesenteric lymph nodes are also frequently involved.

Virulent strains proliferate in the gut-associated lymphoid tissue and the lamina propria, where they cause localized tissue destruction leading to the formation of microabscesses. They may also spread through lamina propria, to adjacent villi and *via* the lymph to more remote sites. Lesions occur chiefly within intestinal crypts. These lesions are microcolonies of bacteria surrounded by granulocyctic and mononuclear inflammatory cells. *Y. enterocolitia* may spread

via the lymph to the draining mesenteric lymph nodes where the organisms also produce microabscesses.

Food Poisoning by *Salmonella* sp.

Of the various food-borne diseases, salmonellosis is the most important one both in terms of the disease intensity, ease of spread and difficulty in prevention and control.

According to WHO, it represents 60-80% of all reported cases of food-borne diseases. The reasons for the increase in occurrence of salmonellosis are:

1. Increase in communal feeding
2. Increase in international trade in human food
3. Higher incidence of salmonellosis in farm animals
4. Widespread use of household detergents interfering with sewage treatment
5. Wide distribution of prepared foods
6. Widespread use of animal feeds containing antimicrobial drugs that favour drug-resistant salmonellae and their potential transmission to human beings.

It is important to learn the taxonomy and morphological features of the organism, growth characteristics, survival characteristics, reservoirs, food-borne outbreaks, disease symptoms, infectious dose, incubation period and mortality rate, pathogenicity and virulence factors, lab diagnosis.

Characteristics of the Disease

The illness presents with four symptoms as

1. Enteric fever (typhoid/paratyphoid)
2. Gastroenteritis
3. Septicaemia
4. Carrier state

Enteric fever has an incubation period of 7-28 days with diarrhea, fever, abdominal pain, headache and uncomplicated colitis. Treatment is with antibiotics and supportive therapy (fluid replacements).

Systemic infections by non-typhoidal organisms can also degenerate into systemic infections and precipitate chronic conditions with high levels of virulence like *S. dublin* and *S. cholerasuis.*

Salmonella-induced chronic conditions such as aseptic reactive arthritis, **Reiters syndrome** and ankylosing spondylitis are important since certain strains have the ability to infect mucosal surface with the help of outer membrane LPS and invasiveness.

Several theories have been put forth explaining the onset of chronic conditions; phagocytosis of salmonellae results in chemical alteration and slow phagocyte release of bacterial LPS. These altered LPS moieties are re-inserted into the cell membrane of monocytes and synoviocytes (associated with synovial fluids), thereby rendering these surface-altered host cells susceptible to lysis by antibody and complement, autoimmune response resulting in rheumatoid tissue damage caused by a localized inflammation of host tissue and release of cytokines and enzymes from antigen-specific activated phagocytes.

Infectious dose

Newborn infants, elders, immunocompromised individuals are susceptible to Salmonella infections. Infectious dose is food-dependent.

Sl. No.	Food	Microorganism	Quantity (cfu)
1.	Chocolate	*S. napoli*	$10^{-1} - 10^2$
		S. typhimurium	$< 10^1$
2.	Cheddar cheese	*S. typhimurium*	$10^6 - 10^1$
3.	Potato chips	*S. sinipaul*	$< 4.5 \times 10^1$

Generally, 1-10 cells can constitute a human infectious dose. Infectious dose also depends on food vehicles like high fat content cholcolates (cocoa butter), cheese (milk fat), meat (animal fat) since entrapment of salmonellae within the hydrophobic lipid micelles offers protection against the bactericidal action of gastric acidulants. Following bile-mediated dispersion of the lipid moieties in the duodenum, viable salmonellae would resume their course following attachment and colonization. Infectious dose is inversely related to the incubation

period. Generally the incubation period ranges between 5-72 hours/12-36 hours depending on the infectious dose and the immune status of the individual. The duration of the illness is 1-4 days (acute), 7-28 days (severe) and can be over 2 months with intermittent excretion (carrier state).

Food Poisoning by *Staphylococcus* sp.

Staphylococcal food poisoning (SFP) ranks as one of the most prevalent causes of gastroenteritis worldwide. It results from ingestion of one or more preformed staphylococcal enterotoxins in Staphylococcus-contaminated foods. The aetiological agents of SFP are members of the genus staphylococcus, predominantly *Staphylococcus aureus*. This form of food poisoning is considered as intoxication since it does not require growth of the organisms in the host.

The term 'staphylococci' informally describes a group of small, spherical, gram-positive bacteria. They are catalase positive chemo-organotrophs with a DNA composition of 30-40 mol % G + C content.

S. aureus can produce multiple toxins with similar molecular weights as well as similar biological and physico-chemical properties. Thus current classification is based on antigenicity. Initially, differentiation between the multiple antigenic forms of Staphyloccal enterotoxin (SE) was based on the observation that many food isolates produce one common antigenic type of toxin, tentatively designated the "F" toxin. Most other enterotoxigenic stains such as those from enteritis patients also produced the "F" toxin in addition to a second antigenic "E" toxin. The discovery of additional isolates that did not conform to this pattern prompted adoption of an improved nomenclature system. The SEs are sequentially assigned a letter of the alphabet in the order of their discovery. The "F" and "E" toxins were designated SEA and SEB respectively. Protein sequencing and recombinant DNA methods have resulted in the current detailed knowledge of the primary sequences of all of the classical SEs. More recent additions to the SE family include SEG and SHE. The SEC serological variant can be further divided into at least three subtypes SEC1, SEC2 and SEC3 based on minor differences in immunological reactivity.

Characteristics of the Disease

Staphyloccal food poisoning is a food-borne intoxication, and results from the ingestion of food in which *Staphylococcus aureus* has already grown to high numbers and produced

exotoxins. The short incubation period and symptoms experienced closely resemble those seen with many chemical intoxicants.

Symptoms typically develop within 6 hours after ingestion of contaminated food. The mean incubation rate is 4.4 hours although incubation periods as short as 1 hour have been reported. Death due to SFP is not common and treatment is minimal in most cases although administration of fluids is indicated when diarrhea and vomiting are severe. Effective doses of SE may be achieved when populations of *S. aureus* are greater than 10^5 organisms per gram of contaminated food.

A basal level of approximately 1 ng of SE per gram of contaminated food is sufficient to cause symptoms associated with SFP. Many factors contribute to the likelihood of developing symptoms and their severity. The most important include susceptibility of the individual to the toxin, the total amount of food ingested, the toxin type and the overall health of the affected person. Though SFP outbreaks attributed to ingesting of SEA are much more common, individuals exposed to SEB exhibit more severe symptoms (SEB is generally produced at higher levels than SEA).

Listeriosis

The genus *Listeria* belongs to the *Clostridium* sub-branch together with *Staphylococcus, Streptococcus, Lactobacillus, Brochothrix*. On the basis of DNA-DNA hybridization and 16S rRNA sequencing, the genus *Listeria* presently comprises six species divided into two lines of descent:

1. *Listeria monocytogenes* and the closely related species, *L. innocua, L. ivanovii, L.welshimeri* and *L. seeligeri*
2. *L. grayi*

Only *L. monocytogenes* and *L. ivanovii* are considered virulent. Only *L. monocytogenes* is considered as a public health concern. *L. monocytogenes* is a gram-positive, facultatively anaerobic, catalase-positive, oxidase-negative, non-sporeformer. Cells are coccoid to rod-shaped and motile with peritrichous flagella exhibiting a characteristic tumbling motility. There are 13 serovars which can cause disease, but 955 of human isolates belong to 3 serovars, 1/2a, 1/2b and 4b.

Listeriosis has emerged as one of the major food-borne diseases during the last decade. The emergence of listeriosis is the result of complex interactions between various factors reflecting changes in social patterns. These factors include

1. Medical progress and consequent demographic changes, such as the increasing proportion of immnocompromised people and the elderly.
2. Change in primary food production, food processing technology, expansion of the agro-food industry, and development of cold storage systems.
3. Changes in food habits (increase consumer demand for convenient food that has as fresh cooked taste, can be purchased ready-to-eat, refrigerated, or frozen, can be prepared rapidly and requires essentially little cooking before consumption).
4. Changes in handling and preparation practices.

Listeriosis is an atypical food-borne disease of major public health concern because of the severity and the non-enteric nature of the disease, a high mortality rate, a long incubation time and a predilection for individuals who have underlying conditions which lead to impairment of T-cell mediated immunity.

Listeria differs from other food-borne pathogen due to its ubiquity, its resistance to diverse environmental conditions such as low pH and high NaCl concentrations and its microaerobic and psychrotrophic characters.

Listeria is a major concern for the agro-food industry due to the following factors.

1. Various ways in which the bacterium can enter into food processing plants.
2. Its ability to survive for long periods of time in the environment and on foods and in food processing plants.
3. Its ability to grow at very low temperatures (2-4°C).
4. Its ability to survive in or on food for prolonged periods under adverse conditions.

Symptoms of the Disease

Pregnant women are most frequently affected who may be asymptomatic or characterized by a flu-like illness with fever, myalgia or headache. The foetus may be worst affected with foetal death and severe neonatal septicaemia and meningitis.

In non-pregnant adults, meningitis and meningo-encephalitis frequently occur in cases of listeriosis. Foetal infections, including endocarditis, pulmonary infection, septic arthritis, osteomyelitis and peritonitis hepatitis are common.

Infective Dose

The infective dose of L. monocytogenes depends on many factors including the immunological status of the host. Microbial characteristics are important risk factors for disease. Published data indicate that the numbers of L. monocytogenes in contaminated food responsible for epidemic and sporadic food-borne cases were more than 100 cfu/g. more epidemiological information is needed for an accurate assessment of the infective dose.

Pathogenicity

Virulence factors

During the last decade there have been major advances in studying and identifying the virulence factors of this highly invasive intracellular pathogen.

Once ingested, *L. monocytogenes* crosses the intestinal barrier and then internalized by resident macrophages, in which they can survive and replicate. They are subsequently transported via the blood to regional lymph nodes. When they reach the liver and the spleen, most listeriae are rapidly killed. Depending on the level of T-cell response induced in the first days following initial infection, further dissemination via the blood to the brain may subsequently occur. Hence, infection is not localized at the site of entry but involves entry and multiplication in a wide variety of cell types and tissues. The principal site of infection is the liver. Entry into epithelial cells has been studied by genetic approaches leading to the identification of internalin, a protein required for entry into epithelial cells.

Following invasion of the intestinal barrier, two types of cells, macrophages and hepatocytes are critical to infections. The internalization of the bacterium into the phagocytes is triggered by the bacterium and soon after the entry, bacteria and internalized in membrane bound vacuoles, which are lysed in less than 30 minutes. Intracellular bacteria are released to the cytosol and begin to multiply with a doubling time of about 1 hour. These intracytoplasmic

bacteria become progressively covered by a cloud of cell actin filaments which later rearranges into a polarized comet tail up to 40 μm in length. When bacteria reach the plasma membrane, they put out long protrusions, each with a bacterium at the tip. These protrusions are then internalized by a neighbouring cell, giving rise to a two membrane bound vacuole. After lysis of this vacuole, a new cycle of replication, movement, and spreading of the bacteria begins. The entire cycle is completed in 5 hours.

The strategy of direct cell-to-cell spread allows bacteria to disseminate within host tissues and induce the formation of infections foci while being sheltered from host defenses such as circulating antibodies.

Unit III

Viruses

Viruses are not living organisms but bits of reproductive material that attack human cells and hijack them. An infected cell then starts making more viruses until it can't make any more, breaking open and releasing the new viruses into the body to infect more cells. The most important viruses that cause foodborne disease are Hepatitis A, Norwalk virus, Norovirus and some of the Caliciviruses. Viruses don't grow in food, and one particle is enough to make you sick. Symptoms can be severe gastroenteritis or similar to the 'flu'. Generally the illness only lasts one or two days. The exception is Hepatitis A which can be a severe illness and last for many weeks.

Viral Gastroenteritis

Viral gastroenteritis is usually regarded as a mild self-limiting disease lasting 24-48 hours. However, people can feel debilitated for 2 to 3 weeks, which has considerable economic implications in terms of working days lost and impaired performance. Symptoms include, malaise, abdominal pain, pyrexia, diarrhea and/or vomiting. A range of symptoms occurring in an outbreak should alert investigations to the possibility of a viral cause. The viruses are usually transmitted by the faecal-oral route, but they are also present in vomitus. Onset of viral gastroenteritis may be sudden and can commence with projectile vomiting. Virus will be disseminated over a wide area in aerosol droplets, which is a particular hazard where food is being prepared and laid out. Although most transmission is directly from person to person, contaminated food and water can give rise to common-source outbreaks. The infective doses are not known, but the evidence form volunteer studies and the typically high attack rates observed in outbreaks suggest that they are very low. For instance, it has been estimated that NLVs have an infective dose of between 10 and 100 virus particles.

Norwalk-like Viruses

This group of viruses infects all age groups. There is a variable incubation period of 12-60 hours, which is thought to be dose-dependent. These viruses are responsible for both sporadic cases of gastroenteritis in the community and for outbreaks in schools, hospitals, old age homes, hotels and cruise ships.

Rotavirus

Rotaviruses mainly infect young children. It is estimated that they cuase one million deaths a year in children under 5 years of age, mostly in developing countries. In developed countries deaths are relatively rare, but rotavirus gastrotenteritis is the most frequent reason for admission of young children to hospital. Rotaviruses consistently account for around 80% of all gastroenteritis viruses. Food-borne and particularly water-borne spread are probably a significant route of transmission in developing countries, but in developed countries reports are rare.

Astrovirus

The astrovirus form a morphologically distinct group of viruses, been associated with illness in children, often under 1 year of age, although outbreaks have been reported in the elderly. The use of more sensitive molecular detection methods is required to assess the incidence and epidemiology of these viruses. Astroviruses have been seen in some adults following the consumption of shellfish or contaminated water, but these incidents appear to be comparatively rare.

Hepatitis

There are two forms of enterically transmitted hepatitis, hepatitis A and hepatitis E.

Hepatitis A

The most characteristic symptom of hepatitis A is jaundice, but milder symptoms of nausea and general malaise without jaundice are common. Patients may feel unwell for several weeks, but recovery is complete. Deaths are rare. Some infections, particularly in children, may be asymptomatic. Like viral gastroenteritis, transmission is by the faecal-oral route, but the primary site of viral replication is the liver. Virus excretion may commence up to a week before symptoms are apparent, making control difficult.

Hepatitis E

Hepatitis E has been associated with large water-borne outbreaks in some developing countries, notably in Asia. Africa and Central America. Food-borne transmission has been suggested, but not proved conclusively. Illness appears more severe than hepatitis A, particularly in pregnant women where a death rate of 17-33% has been observed. The primary source of infection appears to be contaminated water rather than person-to-person spread. Secondary person-to-person transmission is estimated at only 0.7-8%. With the worldwide distribution of foods, vigilance should be maintained.

What should We do, if We get sick?

What should we do when we have suspected food poisoning? Seek medical attention. Food poisoning can be particularly serious in young children, the elderly and people of all ages in poor health. Early medical attention is recommended, especially for these at risk groups.

It probably wasn't the last meal you ate, but if you have any food samples keep them for analysis. If you are feeling unwell visit your doctor. Some types of food poisoning are life threatening and early diagnosis can help avoid severe consequences.

Remember, food that makes us sick can still look fresh and tasty.

How to prevent these microorganisms making our self and our family sick

Sl. No.	Microorganism	Control Measures
1.	*Salmonella* spp. *Campylobacter* *E. coli* Viruses	• Keep hot food hot and cold food cold. Don't keep food in the temperature danger zone (i.e. at or below 5 and above 60°C) any longer than necessary • Reheat food to steaming hot before serving (at least 75°C) • Cook food properly, heat to at least 75°C • Keep raw and cooked food separate • Keep kitchen and utensils clean • Wash and dry your hands properly • Avoid handling food when you are ill
2.	*Listeria monocytogenes*	• Keep hot food hot and cold food cold. Don't keep food in the temperature danger zone (i.e. at or below 5 and above 60°C) any longer than necessary • Heat food to steaming hot before serving (above 75°C) • Cook food properly • Keep raw and cooked food separate • Keep kitchen and utensils clean • Store ready to eat foods in the fridge for only a short time

		• Avoid high risk foods
3.	*S. aureus*	• Keep hot food hot and cold food cold. Don't keep food in the temperature danger zone (i.e. at or below 5 and above 60°C) any longer than necessary • Keep raw and cooked food separate • Keep kitchen and utensils clean • Wash and dry your hands properly • Cover any cuts or sores when handling food
4.	*C. botulinum*	• Keep hot food hot and cold food cold. Don't keep food in the temperature danger zone (i.e. at or below 5 and above 60°C) any longer than necessary • Bottle only high acid fruits at home • Add citric acid to tomatoes, melons and tropical fruits • Bottle vegetables in vinegar, no less than half the liquid should be vinegar • Be careful when making vegetables in oil or flavoured oils to use only acidified or dried vegetables
5.	*C. perfringens* *B. cereus*	• Keep hot food hot and cold food cold. Don't keep food in the temperature danger zone (i.e. at or below 5 and above 60°C) any longer than necessary • Cool food quickly after cooking if you want to store it. • Reheating food to steaming hot (75°C) before eating, stir the food to make sure all parts are heated through

Food hygiene

Cooking. Foodborne bacteria are usually found on the surface of food and are destroyed by cooking. The main exception is *Clostridium perfringens*, whose spores survive high temperatures. Thorough cooking is particularly important for minced meat, as bacteria can be mixed throughout the meat.

Storage. Most foodborne bacteria can grow in food at temperatures between 10-50°C (fridges should be kept at ~4°C). Food poisoning is caused only if a person is infected by more than a certain number of bacteria – ranging from tens of bacteria for *E.coli* O157 to over 1million for *Clostridium perfringens*. Food lightly contaminated with *Clostridium perfringens* could, if not chilled appropriately, become toxic overnight.

Cross contamination. Bacteria can spread to 'clean' food from contaminated food via direct contact, or via surfaces and equipment. It is a particular problem with foods such as salads, which are not to be cooked before consumption.

Cleaning and hand washing. Removing harmful microorganisms from surfaces, equipment and hands stops them from spreading. A survey of catering staff carried out by the FSA in 2002 found that over a third did not wash their hands after visiting the lavatory while at work.

Animal surveillance data

The Veterinary Laboratories Agency (VLA), a Defra agency, collects and collates data from several sources.

• Government veterinary laboratories collate and analyse test results. Whether farmers or vets request tests in the first place is influenced by many factors including the level of awareness of a disease and its perceived importance, the value of the animal(s) affected and the general economic climate. A question therefore arises over the quality and consistency of the data.

• Defra commissions targeted surveillance, which gives a snapshot of the situation. For example, cattle, sheep and pigs at slaughterhouses were tested for *Campylobacter* and *Salmonella* over a 12 month period in 1999/2000. A similar survey is planned for 2003.

• Statutory surveillance collects continuous data for poultry breeding flocks, which are tested at regular defined intervals for Salmonella. The cost of this testing regime is borne by the farmers.

Data from other sources that is not reported to the VLA includes that collected by industry (for example as part of an initiative by the British Pig Executive to reduce the incidence of *Salmonella* in pigs) and by private vets.

Why did food poisoning increase?

The increase in food poisoning occurred in 1980s and 1990s. This could be partly accounted for, by greater reporting of food poisoning, linked to increased public awareness as well as advances in laboratory techniques enabling the micro-organisms to be identified more easily. However, it is widely agreed that there has been a genuine increase in food poisoning. It is likely that a combination of the factors below is responsible:

• **Changing social patterns**. The moves towards shopping less frequently and thus storing food for longer; the increasing use of pre-prepared dishes, which are not always stored or reheated appropriately; the trend towards eating out more often; and the increase in international travel.

• **Emergence of new diseases**. The first cases of E.coli O157 in England and Wales were reported in 1982.

• **Increasingly globalised food market**. The variation in standards of food safety between countries could allow micro-organisms to spread quickly across the globe.

Controlling *Salmonella* in eggs

An increase in Salmonella related food poisoning in the 1980s was associated with chickens and eggs. Counter measures introduced in 1989 included the slaughter of any flock that tested positive for the two most common strains of Salmonella - enteritidis and tymphimurium. Since 1993, only infected breeding flocks have been slaughtered. This prevents vertical transmission and to control subsequent infection in production flocks by horizontal transmission, the Government published voluntary Codes of Practice outlining good practice in biosecurity. These Codes have been adopted by industry through farm assurance schemes such as the Lion Quality mark for eggs, launched in 1998.

In addition, all Lion Quality eggs come from flocks that have been vaccinated against Salmonella enteritidis. The use of the vaccine has been credited by some as the driver behind the

reduction in Salmonella enteriditis infection. However, it alone cannot guarantee that eggs are not infected – in 2002 a Salmonella outbreak was traced to Lion branded eggs. Lion Quality eggs currently account for 75% of the UK retail egg market. Uptake has been slower in the catering sector.

Some ways of preventing food poisoning

_ Good personal hygiene; such as thoroughly washing and drying hands when handling food.

_ Avoid cross-contamination; such as keeping raw foods and ready-to-eat foods separate and using separate and clean utensils, containers and equipment.

_ Cook foods thoroughly; make sure foods such as meats and poultry are cooked until core temperature reaches 75°C.

_ Avoid the Temperature Danger Zone; keep chilled foods cold at 5°C or colder, and hot food hot at 60°C or hotter.

_ Avoid spoiled foods; foods past their use-by dates or food in damaged containers or packaging.

_ When in doubt, throw it out

_If working in a food business: Follow the business Food Safety Program.

_ Follow the advice given by the Food Safety Supervisor.

_ Be trained in safe food handling.

Unit IV

Mycotoxigenic Moulds as Agents of Food Poisoning

Mycotoxins brought the attention of the scientists in the early 1960s. *Aspergillus* was first described almost 300 years ago and is an important genus in foods. Most *Aspergillus* species occur in foods as spoilage or in biodeterioration. They are extremely common in stored commodities such as grains, nuts and spices. *Aspergillus* is a large genus containing more than 100 recognized species, most of which grow well as laboratory culture. There are a number of teleomorphic (ascosporic) genera which have *Aspergillus* conidial states (anamorphs), but the only two of real importance in foods are the xerophilic genus *Eurotium* and *Neosartorya* which produce heat-resistant ascospores and cause spoilage in heat-processed foods. Almost 50 species of *Aspergillus* have been identified as capable of producing toxic metabolites. Chief toxins produced by *Aspergillus* sp., are the aflatoxins, (*A. flavus, A. parasiticus, A. nomius*), Ochratoxin A (*A. ochraceus*), Sterigmatocystin (*A. versicolor*), Cyclopiazonic acid (*A. flavus, A. tamari*), Citrinin, Patulin and Penicillic acids.

Aspergillus flavus and *Aspergillus parasiticus*

The most important group of toxigenic aspergilli are the aflatoxigenic moulds. *A. flavus*, *A. partisiticus* and *A. nomius* are closely related but produce specific toxins. *A. flavus* produces aflatoxins and B_1 and B_2 and cyclopiazonic acid. Only certain strains are toxigenic. *A. parasiticus* produces aflatoxins B_1, B_2, G_1 and G_2 but not cyclopiazonic acid and almost all the strains are toxigenic. *A. nomius* also produces B and G aflatoxins but the potential toxigenicity of the organisms are not known.

Occurance of the Moulds

Apergillus flavus is widely distributed in nature, but *A. parasiticus* if less widely. *A. flavus* is the most common species in peanuts and the second most common in corn. Invasion takes place before harvest, not during storage. Peanuts are invaded while still in the ground. In corn, damage in developing kernels is caused by insects that allow entry of aflatoxigenic moulds. Cereals and spices are common substrates for *A. flavus* but aflatoxin production in these products is a result of poor drying, handling or storage.

Factors Affecting Growth and Toxin Production

Aspergillus flavus and A. parasiticus have similar growth patterns. Both require growth temperatures of 32-33°C and can grow at 10-42°C, Aflatoxins are produced at 12-40°C. Optimum water activity required for growth is near 0.99 and aflatoxins are generally produced in greater quantities at higher a_w values with lesser production apparently ceasing at or near a_w 0.85

Growth occurs at a pH ranging from 2 to 10.5 but aflatoxin production has been reported for *A. parasiticus* between pH 3 and 8 (opt. pH 6). Reduction of available oxygen by modified atmosphere packaging of foods with oxygen scavengers can inhibit aflatoxin formation by both the species.

Aflatoxins

Aflatoxins are difuranocoumarin derivatives. Aflatoxins B_1, B_2, G_1 and G_2 are produced in nature by the moulds. The letters B and G refer to the fluorescent colors (blue and green respectively) observed under UV light and the subscripts refer to their separation patterns on TLC plates.

Aflatoxins are both acutely and chronically toxic in animals and humans, producing acute liver damage, liver cirrhosis, tumour induction and teratogenesis. They also have immunosuppressive effects in combination with other mycotoxins, Immunosuppression can increase susceptibility to infectious disease. Outbreaks with consumption of rice have been reported with nearly 15% mortality. Another outbreak of hepatitis which affected 400 people, 100 of whom died, almost certainly was caused by aflatoxin that could have been from heavily infected corn that was consumed. The greatest direct impact of aflatoxins on human health is their potential to induce liver cancer where hepatitis B virus also has a role to play. Aflatoxins and hepatitis B virus are co-carcinigens and they increase the probability of human liver cancer. But high aflatoxin intakes are casually related to high incidences of cancer, even in the absence of hepatitis B.

Aflatoxin B_1 is metabolized by the microsomal mixed function oxidase system in the liver, leading to the formation of highly reactive intermediates, one of which is 2,3-epoxy aflatoxin B_1. Binding of these reactive intermediates to DNA results in disruption of transcription and abnormal cell proliferation, leading to mutagenesis or carcinogenesis. Aflatoxin also inhibits

oxygen uptake in the tissues by acting on the electron transport chain and inhibiting various enzymes resulting in decreased production of ATR.

Cyclopiazonic Acid

This is produced by *A. flavus, A. tamari* and *A. versicolor*. The toxin is an indole tetramic acid which can occur in naturally contaminated agricultural commodities and compounded animal feeds. It causes severe gastrointestinal and neurological disorders along with degenerative changes and necrosis in the digestive tract, liver, kidney and heart.

Toxin Detection

Aflatoxins can be detected by chemical or biological methods. In the chemical methods, samples are extracted with organic solvents such as chloroform or methanol in combination with small amounts of water. Extracts are further cleaned up by passage through a silica gel column. The extract if then concentrated, usually by evaporation under nitrogen and separated by TLC or HPLC. TLC is most often used, and aflatoxins are visualized under UV light and quantified by visual comparison with known concentrations of standards or by fluorimetry. The presence of fats, lipids, or pigments in extracts reduces the efficiency of the separation. Immunoassay techniques including enzyme-linked immunosorbent assays and dipstick tests for aflatoxin detection have been developed and commercially make available.

Food Poisoning by *Aspergillus ochraceus*

Aspergillus ochraceus is a widely distributed mould, particularly common on dried foods. There are three toxins, ochratoxin A, B and C. This organism also produces Penicillic acid, a mycotoxin of lower toxicity. Other reported metabolites are xanthomegnin and viomellein.

Aspergillus ochraceus is widely distributed in dried foods such as nuts, beans, dried fruits and dried fish. It is xerophile, capable of growth down to a_w 0.79 (opt. 9.9). It grows at temperatures of 8 to 37°C and within a wide pH range of 2.2 to 10.3. Ochratoxin A is produced at quite high temperatures between 25 and 30°C.

Isolation and Detection

This organism is best isolated on reduced aw media such as dichloran glucose agar. Colonies of *A. orchraceus* are deep ochre-brown to yellow-brown in colour with long stripes bearing radiate *Aspergillus* head. The vesicles are spherical bearing densely packed metulae and phialides with small, smooth pale brown conidia. Orhratoxins can be assayed by routine chromatographic methods like the TLC and HPLC with detection of green fluorescence under UV light at 333nm. Immunoassay techniques, including ELISA have also been developed and commercialized.

Aspergillus versicolor

This is the most important food spoilage and toxigenic species. It is the major producer of sterigmatocystin, a carcinogenic dihydrofuranoxanthone which is a precursor of the aflatoxins. Sterigmatocystin is also produced by *Aspergillus nidulans.*

Aspergillus versicolor is a xerophile, with a minimum a_w of 0.74 to 0.78 for growth. It is widely distributed in foods particularly stored cereals, cereal products, nuts, spices and dried meat products. The reported minimum temperature for growth is 9°C at aw 0.97 and the maximum temperature is 39°C at a_w 0.87. Natural occurrence of sterigmatocystin has been found in rice, wheat and barley. This toxin has low acute oral toxicity because it is relatively insoluble in water and gastric juices. But even low doses can cause tumor and pathological changes.

Isolation and Detection

This organism can be isolated in the low aw agar such as the dichloran glucose agar. Small grey-green colonies showing pinkish or reddish color in the mycelium and/or reverse and with mop-like heads are indicative of this organism.

Sterigmatocystin can be detected by TLC with chloroform-methanol as a solvent system. The toxin is visualized as an orange-red spot under UV light. A light yellow fluorescence develops after spraying with acetic acid. An ELISA method for detection of stegimatocystin has been reported.

Aspergillus fumigatus

It is best recognized as a human pathogen causing aspergillosis of the lung. It is a thermophile with a temperature range for growth of between 10 and 55˚C and an optimum between 40 and 42˚C. It is one of the least xerophilic of the common aspergilla with a minimum a_w of 0.85 for growth. It is best found in decaying vegetation, in which it causes spontaneous heating. It is isolated frequently from foods particularly stored commodities. This organism is capable of producing several toxins which affect the central nervous system, causing tremors. The toxin is called fumitremorgens A, B and C which are toxic cyclic dipeptides.

Aspergillus terreus

It occurs commonly in soil and foods particularly stored cereals and cereal products, beans, pulses and nuts. They produce a group of tremorgenic toxins known as territrems. These toxins do not contain nitrogen. Territrems are acutely toxic causing whole body tremors within 5 minutes and other neurological symptoms within 23-30 minutes, all of which subside within 1 hour. The toxin appears to act by blocking acetylcholinesterase activity.

The organism produces rapidly growing pale brown colonies, with *Aspergillus* heads bearing densely packed metulae and phialides with minute conidia borne in long columns. The territrems can be detected in chloroform extracts by TLC, exhibiting blue fluorescence under UV light.

Aspergillus clavatus

It is found in soil and decomposing plant materials and is easily recognizable by its large blue-green club-shaped heads. It is especially common in malting barley. The organism produces patulin, cytochalasins and the tremorgenic mycotoxins tryptoquivaline, tryptoquivalone and related compounds. A. clavatus is a recognized health hazard to workers in the malting industry. Inhalation of large numbers of highly allergenic spores can cause respiratory diseases such as bronchitis, emphyema or malt worker's lung, a serious occupational extrinsic allergic alveolitis.

Eurotium

Members of this genus are referred to as the *Aspergillus flancus*. All *Eurotium* species are xerphilic. They are important spoilage moulds in all types of stored commodities like stored grains, spices, nuts and animal feeds. The four most common species are *E. chevalieri, E. repens, E. rubrum* and *E. amstelodami*

Eurotium chevalieri and *E. amstelodami* have been reported to produce toxic alkaloid metabolites called echinulin and enoechinulins.

Penicillium as a Toxigenic Mould

The discovery of Penicillin in 1929 gave impetus to a search for other *Penicillium* metabolites with antibiotic properties and ultimately to the recognition of citrinin, patulin and griseofulvin as toxic antibiotics or mycotoxins. Retrospectively, over 120 metabolites from common moulds were demonstrably toxic to higher animals of which 42 were produced by one or more *Penicillium* species.

Penicillium is a large genus with 150 species. At least 50 species are of common occurrence. All common species grow and sporulate well on synthetic or semi-synthetic media and are usually readily recognizable to the genus level. Classification of the penicillia is based on microscopic morphology. The genus *Penicillium* is divided into subgenera based on the number and arrangement of phialides and metulae and rami on the main stalk cells. The majority of important toxigenic and food spoilage species are found in subgenus *Penicillium.* Growth of mould does not always mean production of toxin. The conditions under which toxins are produced are often narrower than the conditions for growth. Most toxins can be placed in two broad groups: those that affect liver and kidney function and those that are neurotoxins. The *Penicillium* toxins which affect liver or kidney function are asymptomatic. In contrast, toxicity of the neurotoxins is often characterized by sustained trembling.

Unit V

Algal Food Poisoning

Seafood toxin can be categorized according to their primary origin; toxins originating from toxic marine algae, principally dinoflagellates, which include ciguatoxins and those toxins associated with the major shellfish poisoning syndromes (diarrhoetic shellfish poisoning (DSP)); paralytic shellfish poisoning (PSP); amnesic shellfish poisoning (ASP); and neurotoxic shellfish poisoning (NSP); and contaminants produced by bacteria (tetdotoxins (TTXs)) associated with puffer fish poisoning or by bacterial decomposition (histamine associated with scombrotoxic fish poisoning). All these syndromes share a lack of organoleptic evidence of contamination in affected fish; most toxic seafoods looks, smell and taste quite normal. Adequate disease prevention requires widespread and efficient monitoring programmes employing accurate and rapid toxin detection methods and also an increased awareness by both the public and clinicians in order to effect swift diagnoses and supportive treatment, where necessary.

Toxic Syndromes Associated with Marine Algal Toxins

There are over 4000 species of marine algae (phytoplankton) and about 2% are known to produce toxins most of which are *Dinophyceae* sp. (dinoflagellates) with four more genera being responsible for the majority of toxic events, i.e., *Alexandrium* sp., *Gymnodinium* sp., *Dinophysis* sp., and *Pronocentrum* sp., A proportion of these toxic dinoflagellates have a red-brown pigmentation giving rise to the naming of algal blooms as"red-tides". However, not all toxic algae are colored and incidents of poisoning have occurred in the absence of red blooms. Many toxic species form resting cysts during adverse conditions but produce dense blooms, usually in the summer months, in response to favorable conditions of temperature, pH, salinity, light and the availability of nutrients.

Visible red tides may contain from 20,000 to 30,000 algal cells/ml of sea water; however, concentrations as low as 200 cells/ml may produce toxic shellfish. Algal toxicants are accumulated in the digestive gland of the shellfish, the shellfish themselves remaining unaffected by the toxins.

Both proliferating cells and resting cysts can contain toxins and act as sources of shellfish contamination. The cysts of some dinoflagellates are initially much more toxic than vegetative cells, they sink out of the water column and overwinter at the sediment-water interface. Resting

cysts are thought to contribute to shellfish toxicity in the absence of proliferating blooms and to the accumulation of toxins in scavenging crustacea and grazing fin-fish.

Paralytic Shellfish Poisoning

The world wide incidence of paralytic shellfish poisoning (PSP) has been estimated at 1600 cases per year with a possible 300 of these being fatal. Outbreaks have been occurring regularly throughout the world. Blooms of the associated dinoflagellates occur widely throughout Europe.

Time of onset of symptoms varies from 15 minutes to 10 hours, but usually occurs within 2 hours of consumption of the seafood, and is influenced by amount of toxin ingested, age and weight of the patient, nature of the contaminated food and any accompanying alcohol consumption. Dominant clinical features are neurological. Gastrointestinal symptoms are less common. In severe cases, death may occur within 2-25 hours. The toxins associated with PSP are a group of closely related compounds based on saxitoxin (STX), a compound comprising two fused **guanidium moieties**. PSP toxins are often found in toxic shellfish. The compounds are all water-soluble and heat-stable; a light 5 minutes cook will reduce toxicity by only 30% and increasing this to 20 minutes will only effect a 40% denaturation. All these toxins operate by blocking the sodium channel which traverses excitable cells comprising nerves and muscle fibres. Neurotransmission is facilitated by a Na^+ influx through the channel following depolarization.

Oral intake of 144-1660 µg per person leads to intoxication and may be fatal if the intake is 300-12,400 µg per person.

Diarrheic Shellfish Poisoning

Outbreaks of gastrointestinal illness associated with shellfish exposed to dinoflagellates were first reported in 1961 in the Netherlands followed by incidents in Japan and Europe, but now also include North and South America, Australia, Indonesia and New Zealand.

The predominant symptoms of DSP are diarrhea, nausea, vomiting and abdominal pain lasting up to 3-4 days with a typical onset period of 30 minutes to a few hours after consumption of the toxic seafood. Affected individuals rarely require hospitalization and any treatment prescribed is supportive of the gastrointestinal effects.

Dinophysis sp. and *Prorocentrum* sp. both produce the toxins associated with DSP. In Europe D. accuta and D. accuminata predominated, D. fortii is found occasionally, and D. norvegica is common in Norway. The DSP toxin family comprises at least 12 polyether carboxylic acids. Shellfish can create a series of derivatives of each of these toxins during their metabolic processes and these derivatives of each of these toxins during their metabolic processes and these derivatives all vary slightly in their toxicity. The toxins inhibit protein phosphates which are integral regulatory proteins in metabolism, membrane transport, secretion and cell division. These toxins are potent tumour promoters and are possibly mutagenic and immunotoxic.

Neurotoxic Shellfish Poisoning

Neurotoxic shellfish poisoning has occurred mainly in the USA and Mexico. These toxins are produced by *Gymnodinium* sp. and *Ptychodiscus breve* (also called *G. brave*)

Symptoms usually begin 1-3 hours after toxin ingestion and include paraesthesia (numbness/tingling) in the mouth progressing to the extremities, ataxia, gastrointestinal symptoms and the hot to cold temperature reversal phenomenon which affects the patients perceptions of touching hot and cold surfaces. Recovery normally occurs in 2-3 days and there is no specific treatment.

The responsible toxins are a family of nine brevetoxins which all act on excitable cells by opening sodium channels causing a sodium ion influx and the release of synaptic neurotransmitters. The toxins are also responsible for killing fishes. The dinoflagellates are easily lysed in surf, releasing toxins directly into the sea water which may cause dermatitis, conjunctivitis and respiratory problems in bathers.

Amnesic Shellfish Poisoning

Amnesic shellfish poisoning was first recognized in 1987 in Canada where 107 cases of human illness were associated with blue mussel consumption. *Pseudonitzschia pungens,* a marine algae has been responsible for this toxic syndrome.

The syndrome progresses from typical gastrointestinal symptoms (onset up to 24 hours after consumption if contaminated shellfish) to include a range of neurological effects such as autonomic dysfunction, seizures, focal difinits (onset up to 48 hours). Long-term sequelae include short-term memory loss. The causative agent is domoic acid, a potent neurotransmitter with receptor sites in the CNS. It mimics the natural neurotransmitter glutamate and will bind excessively to brain tissue causing necrosis. Recent work has shown that heterotrophic bacteria can enhance the production of domoic acid by *Pseudonitzschia pungens.*

Ciguatera Poisoning

Ciguatera Poisoning presents a health risk only in UK due to the importation of exotic fish. There are at least 400 species of tropical, reef feeding fish from oceans between latitudes 35°C on either side of the equator which are potentially ciguatoxic. Those most commonly associated with illness are groupers, was bass, snapper, barracuda, eels, parrot fish and mullet, all of which are regularly consumed in the UK. Globally ciguatera poisoning is the most common fish-borne poisoning syndrome with an estimated 50,000 cases a year.

Onset time varies between a few minutes to 30 minutes after consumption of the fish but most patients develop symptoms in less than 6 hours. The most often presented symptoms are tingling and numbness in the mouth, hands and feet and the cold to hot sensory effect (90%), dull aches and sharp shooting pains (85%) and gastrointestinal symptoms (45%). With no known specific antidote, self resolution usually takes about 2-5 days with some neurological effects persisting for weeks, even years in severe cases. Attacks are known to recur when stimulated by stress and hypersensitivity responses induced by foods containing substances which mimic ciguatoxins (e. g., fish, alcohol, nuts). Doses of 0.6 ng/kg body weight have been associated with human illness. Ciguatera poisoning has been shown to be sexually transmissible and toxins may be passed to foetuses in *utero* and to neonates in brest milk.

Dinoflagellates form the genera *Gambierdiscus* (specifically *G. toxicus*), *Ostrepsis, Prorocentrum, Amphidinium* and *Coolia* produce a family of ciguatera associated toxins.

Ciguatoxin, a polycyclic quaternary ammonium compound and scaritoxin are lipid-soluble toxins which can be modified as they move up the food chain to produce a range of toxins varying in toxicity and associated clinical features. These toxins facilitate neurotransmission by opening sodium channels in excitatory membranes by the competitive occupation of calcium receptor sites. A range of water-soluble toxins, based on maitotoxin, are found in small herbivorous fish, which play a more minor role in the syndrome and operate via the calcium channel. Maitotoxin has been found to be produced by *G. toxicus*; the fourth toxin group associated with this syndrome is the palytoxin family, like the ciguatoxins these are polycyclic ethers and have been associated with ciguatera poisoning following the consumption of contaminated mackerel. All the toxins are unaffected by typical cooking conditions.

Unit VI

Food Hygiene, Food Regulation and Standards

Introduction

The basic purpose of food laws and food regulatory agencies is to ensure that all foods reaching the consumer are safe, wholesome and are truthfully labeled. The production and marketing of wholesome foods should be important for everyone affiliated with the food industry.

Advantages of the Food Laws/Food Control Services

1. Protection of the consumer against health risks.
2. Reduction of food loss-well trained inspection machinery can provide guidance for the judicious use of pesticides and food additives which can prolong the shelf life of the food.
3. Prevention of gross food adulteration.
4. Control over contaminants and additives – the level of use of pesticides or contaminants (microbial or non-microbial) has to be maintained within the limits of no effect level.
5. Control over dumping of substandard foods. Substandard adulterated or contaminated foods mau be dumped in a country which does not have the requisite food laws.
6. Food control can basically contribute to the nutritional improvement of a country.
7. Improvement of foreign exchange – the exporting country can manufacture and label according to the requisite of Food laws of the importing country.

Food Hygiene

Food is a potential source of infection and is liable to contamination by microbes, at any point during its journey from the producer to the consumer. Food hygiene may be defined as the sanitary science which aims to produce food which is safe for the consumer and of good keeping quality. It covers a wide field and includes the rearing, feeding, marketing and slaughter of animals as well as the sanitation procedures designed to prevent bacteria of human origin reaching foodstuff. Food hygiene, in its widest sense, implies hygiene in the production, handling, distribution and serving.

WHO (1984) has defined Food Hygiene or Food Safety as all conditions and measures that are necessary during the production, processing, storage, distribution and preparation of food to ensure that it is safe, sound, wholesome and fit for human consumption. The primary aim of food

hygiene is to prevent food poisoning and other food-borne illness. The objective of food control has three aspects – economic, aesthetic and public health.

The laws and regulations are intended to protect the public against injury to health and fraud and deceit. The laws and regulations restrain the sale of foods which are decomposed, adulterated, improperly preserved or misbranded. They provide specifications for safe handling of such perishable and potentially dangerous foods such as milk or milk products and meats. Methods and procedures have been developed to minimize the danger of contamination of foods with poisonous substances or with pathogenic microbes.

Different branches of food hygiene include

1. Milk hygiene
2. Meat hygiene
3. Fish hygiene
4. Egg hygiene
5. Hygiene of vegetables and fruits
6. Food-handlers hygiene
7. Sanitation of eating place

Milk Hygiene

Milk is an efficient vehicle for a great variety of disease agents. Milk gets contaminated by various sources like udder, utensils, personel hygiene of the handlers, storage environment, water etc., This may lead to various milk-borne diseases that may affect the population. Requisites in the production of clean and safe milk are

1. Healthy and clean animal – Milk from a healthy udder contains only few microorganisms.
2. Sanitary conditions of the dairy farm i.e., the premises where the animal is housed and milked should be sanitary.
3. Containers and equipment should be sterile and kept covered.
4. Water supply should be bacteriologically safe.
5. Milk handlers must be free from communicable diseases and before milking they must wash their hands and arms. Milking machines should be used as far as possible.

6. Milk must be cooled immediately to 10°C after it is drawn to retard bacterial growth.

7. Milk should be properly pasteurized to increase shelf life.

Meat Hygiene

A number of diseases are transmitted through meat and meat-based foods since animal tissues are important vehicles for transmission of various protozoan diseases like Taeniasis, Trichinellosis and a number of bacterial infections.

Meat inspection is a very important process before being accepted or rejected. Two types of inspection, i. e., Ante-mortem and post-mortem inspection are being carried out.

Ante-mortem rejection: It is based on emaciation, exhaustion, pregnancy, sheep-pox, foot-rot actinomycosis, brucellosis, febrile conditions, diarrhea and other diseases.

Post-mortem rejection: It is based on *Cysticercus bovis*, liver fluke, abscesses, sarcocystis, hydatidosus, septicaemia, parasitic and nodular infection of liver and lungs, tuberculosis and *Cysticercus cellulosae*.

Good meat qualities: The meat should be firm and elastic to touch, not be pale pink or deep purple colour.

Slaughter house hygiene: Hygiene of slaughter house is of paramount importance to prevent the contamination of meat during the process of dressing. There is a Model Public Health Act (1955) in India, which standardizes on the location, structure, disposal of wastes, water supply, examination of animal, storage of meat, transportation of meat and miscellaneous other activities connected with meat processing.

Fish Hygiene

Fishes are also important agents for disease transmission since they are perishable foods. It is an intermediate host of tapeworm and consumption of spoilt fishes may lead to fish poisoning. Fresh fish is in a state of stiffness or rigor mortis with bright red gills, and clear and prominent

eyes. Fishes also get contaminated through various means during catching, transporting and processing. Prevention of contamination is of utmost importance at each level of processing for increasing the shelf life and quality of fish.

Egg hygiene

Majority of freshly laid eggs are sterile. Shells of eggs become contaminated by faecal matter from the hens or ducks. The microorganisms can penetrate the shell of the egg, cross the various chemical barriers to reach the yolk leading to spoilage of the egg. Eggs must be stored in a dry condition for preventing spoilage. Eggs can also be pasteurized to increase the shelf life.

Vegetable and Fruit Hygiene

Vegetable and fruits host many pathogens like bacterial, fungal, protozoan which can enter the plant materials during or after their harvest. Standards have been laid down for effective storage of the vegetable and fruits to prevent their spoilage and further disease transmission.

Hygiene for Food-Handlers

Food sanitation rests directly upon the state of personal hygiene and habits of the person working in food industries. They may transmit infections like diarrhea, dysenteries, typhoid, enteroviruses, viral hepatitis, protozoan cysts, eggs of helminthes, staphyloccal/streptococcal infections. Simple rules to be followed in food handling are given below:

Medical examination to be carried out at the time of employment, Persons with above diseases or communicable diseases (TB) are not to be employed.

Persons with wounds, otitis media, skin infections, etc., should not be permitted to handle food or utensils.

The day-to-day health appraisal of food-handlers is important. Those who are ill should be excluded from food handling.

Any illness which occurs n a food-handler's family, should be at once notified.

Education of food-handlers in matters of personal hygiene, food handling, utensil/dishwashing and insect or rodent control is the best mean of promoting food hygiene.

Personal hygiene to be promoted

1. Hands – scrubbed and washed with soap and water immediately after visiting lavatory and as often as necessary at other times. Nails to be kept trimmed and free from dirt.
2. Hair – to provide covering to the head.
3. Overalls – clean white overalls to be worn by all food-handlers.
4. Habits – coughing and sneezing in the vicinity of food, licking the fingers before picking up an article of foods, smoking on food premises are to be avoided.

Hygiene in Public Eating Places

The principles of food hygiene in public eating or drinking places are the same as in any other type of food handling activity. The complexity of the problem is mainly due to the ever increasing number of establishments which requires some supervision. At one time, public eating was restricted to special occasions, to the traveler, patient, boarding schools, etc., Due to increased urbanization, distance of working places, mobility and employment, many resort to dining in public eating places. The six minimum essentials for cleanliness and sanitation for public eating or drinking places are as follows

1. Avoid hand contact with food as far as possible.
2. Keep perishable food below 40°F or above 140°F.
3. Keep food protected from personal contact and contaminating insects, dust and animals.
4. Discard all food and food products which are not in good quality.
5. Clean and disinfect equipment that comes into direct contact with food.
6. Keep premises presentable and in a sanitary manner at all times.

Sanitation of eating establishments is a challenging problem in India. The Model Public Health Act, Govt. of India (1955) has suggested the following minimum standards for restaurants and eating places in India.

Location: Shall not be near any accumulation of filth or open drain, stable, manure pit and other sources of nuisances.

Floors: To be higher than the adjoining land, made with impervious material and easy to keep clean.

Rooms: Rooms where meals are served shall not be less than 100 sq. feet and shall provide accommodation for a maximum of 10 persons. Walls up to 3 feet should be smooth with rounded corners and should be impervious and easily washable. Lighting and ventilation should be ample with natural and artificial lighting systems along with a good circulation of air.

Kitchen: It should be with ample floor space, window opening, proper flooring and ventilation.

Storage of Cooked Food: Separate rooms to be provided for storing cooked foods. For long storage, control of temperature is necessary.

Furniture: Furniture should be reasonably strong and easy to keep clean and dry.

Disposal of refuse: Refuse to be collected in covered and impervious bins and disposed off twice a day.

Water Supply: It should be an independent source, adequate, continuous and safe.

Washing Facilities: Cleaning of utensils or crockery to be done in hot water and followed by disinfection.

Food Control Administration

This varies from country to country. The Central, State and local bodies are responsible to enact laws and implementations. There should be laws to control raw materials, processing of foods, preservation of methods and control of milk or meat hygiene, etc., Laws relating to food control in India are

1. The Prevention of Food Adulteration Act 1954, amended in 1976, 1986.
2. Prevention of Food Adulteration Rules, 1955.
3. India Penal code 1860 as amended.
4. Cantonment Act 1924.
5. The Bombay Municipal Corporation Act, 1888.
6. The Bombay Provincial Municipal Corporations Act 1949.
7. The Maharashtra Zilla Parishads and Panchayat Samiti's Act 1961.
8. The Maharashtra Municipalities Act, 1965.
9. Tamil Nadu Public Health Act 1939, and so on for each State.

Genesis of the PFA Act

Even prior to 1954, many of the states had their own food laws. "First law was followed in Bombay in 1899 to combat the evil of food adulteration. Until 1954, 22 out of the 28 states had their own food laws but they were not of the uniform pattern and hence uniform implementation was not possible. The Central Advisory Board of Health suggested the adoption of Central legislation. In October 1952, the Food Adulteration Bill was introduced in the Lok Sabha by the Health Minister and after approval of both the Houses, it was enacted in 1954 as the Prevention of Food Adulteration Act by the Indian Parliament which came into effect on 1st June, 1955. In 1972, the terms of the PFA Act were extended subsequently to Jammu and Kashmir.

Provisions of the Original PFA Act, 1954

1. Definition of Food
2. Definition of Food Adulteration or misbranding
3. Appointment of an Advisory committee called the Central Committee for Food standards (CCFC)
4. Establishments of the Central Food Laboratory, Calcutta, in 1955 and later in each state to give a final opinion in cases challenged in the court of laws.
5. Restriction on imports of adulterated or misbranded foods or other spoilt foods.
6. Power of the State Governments to appoint Public Analysts or Food Inspectors.
7. Procedure for Food Inspectors in drawing and dispatching sample of food to laboratory.
8. Powers to the Central Government for defining the standards of quality, control, over production, distribution, sales, packing, labeling, etc.,
9. Penal provisions provided a maximum imprisonment of one year or a minimum fine of Rs. 2000/- in the first instance and imprisonment of 2 years on the second offence and in the third instance, imprisonment up to 4 years.

Food standards

1. Codex Alimentarius is a collection of international food standards prepared by codex Alimentarius Commission (Organ of FAO/WHO food standards programme). Indian standards are based on this.

2. PFA standards based on PFA act 1954 "Central Committee of Food Standards" revised periodically to get minimum level of quality of food stuff attainable under Indian conditions.

3. AGMARK (Agricultural Marketing, HO, Faridabad) standards set by the Director of Marketing and Inspection, Government of India. The Agmark gives consumers the assurance of quality in accordance with standards laid down.

4. ISI standards, guarantees good quality in accordance with standard prescribed by Indian Standard Institution for the Commodity.

5. ASC specifications for Armed Forces.

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