Education of food handlers in matters of personal hygiene, food handling, utensils, dish washing, and insect and rodent control is the best means of promoting food hygiene. Many of the food handlers have little educational background. Certain aspects of personal hygiene are therefore required to be continually impressed upon them: (a) *Hands* : The hands should be clean at all times. Hands should be scrubbed and washed with soap immediately after visiting a lavatory and as often as necessary at other times. Finger nails should be kept trimmed and free from dirt. (b) *Hair* : Head coverings should be provided, particularly in the case of females to prevent loose hair entering the food-stuffs. (c) *Overalls* : Clean white overalls should be worn by all food handlers. (d) *Habits* : Coughing and sneezing in the vicinity of food, licking the fingers before picking up an article of food, smoking on food premises are to be avoided.

FOODBORNE DISEASES

The term "foodborne 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." With the increase in urbanization, industrialization, tourism and mass catering systems, foodborne diseases are on the increase throughout the world. Foodborne diseases may be classified as :

A. Foodborne intoxications

1. Due to naturally occurring toxins in some foods *(142)* :
   a. Lathyrism (beta oxalyl amino-alanine)
   b. Endemic ascitis (Pyrrolizidine alkaloids)
2. Due to toxins produced by certain bacteria *(134,143)* :
   a. Botulism
   b. Staphyloccus poisons
3. Due to toxins produced by some fungi *(144,145)* :
   a. Aflatoxin
   b. Ergot
   c. Fusarium toxins
4. Foodborne chemical poisoning (146,147) :
   a. Heavy metals, e.g., mercury (usually in fish), cadmium (in certain shellfish) and lead (in canned food)
   b. Oils, petroleum derivatives and solvents (e.g., Trycresyn phosphate or TCP)
   c. Migrant chemicals from package materials
   d. Asbestos
   e. Pesticide residues (DDT, BHC)

B. Foodborne infections

Group	Examples of illness in each group
(1) Bacterial diseases	Typhoid fever, Paratyphoid fever, Salmonellosis, Staphylococcal intoxication, *Cl.perfringens* illness, Botulism, B.cereus Food Poisoning, *E.coli* diarrhoea, Non-cholera vibrio illness, V.parahaemolyticus infection, Streptococcal infection, Shigellosis Brucellosis
(2) Viral diseases	Viral hepatitis, Gastroenteritis
(3) Parasites	Taeniasis Hydatidosis, Trichinosis, Ascariasis, Amoebiasis, Oxyuriasis

FOOD TOXICANTS

1. Neurolathyrism

The cause of neurolathyrism is a toxin, Beta oxalyl amino alanine (BOAA) which is found in the seeds of the pulse, *L. sativus* (Khesari dhal). Neurolathyrism is a public health problem in certain parts of the country where this pulse is eaten (see page 467).

2. Aflatoxins *(158, 159)*

Aflatoxins are a group of mycotoxins produced by certain fungi, *Aspergillus flavus* and *A parasiticus*. These fungi infest foodgrains such as groundnut, maize, parboiled rice, sorghum, wheat, rice, cotton seed and tapioca under conditions of improper storage, and produce aflatoxins of which B_1 and G_1 are the most potent hepatotoxins, in addition to being carcinogenic. The most important factors affecting the formation of the toxin are moisture and temperature. Moisture levels above 16 per cent and temperatures ranging from 110 to 37°C favour toxin formation. Aflatoxicosis is quite a public health problem in India. The latest report (1975) of 400 cases of aflatoxin poisoning including 100 deaths from Banswada and Panchmahal districts of Rajasthan and Gujarat respectively highlight the problem in India. Aflatoxin B_1 has also been detected in samples of breast milk and urine collected from children suffering from infantile cirrhosis. Attempts are also being made to relate aflatoxin with human liver cirrhosis.

Control and Preventive Measures : A crucial factor in the prevention of fungal contamination of foodgrains is to ensure their proper storage after drying. Moisture content should be kept below 10 per cent. If the food is contaminated, it must not be consumed. It is also essential to educate the local population on the health hazards of consuming contaminated foodgrains.

3. Ergot *(160, 161)*

Unlike Aspergillus, ergot is not a storage fungus, but a field fungus. Foodgrains such as bajra, rye, sorghum, and wheat have a tendency to get infested during the flowering stages by the ergot fungus, *Claviceps fusiformis*. Fungus grows as a blackish mass and the seeds become black and irregular and are harvested along with food grains. Consumption of ergot infested grain leads to ergotism. Sporadic outbreaks of ergot poisoning in human population have been reported from time to time in areas where bajra is consumed as a staple. The symptoms are acute but rarely fatal and include nausea, repeated vomiting, giddiness and drowsiness extending sometimes for periods up to 24 to 48 hours after the ingestion of ergoty grain. In chronic cases, painful cramps in limbs and peripheral gangrene due to vasoconstriction of capillaries have been reported. However, the long-term effects of consuming small amounts of the toxin are not known. A disquieting feature is that the recently introduced high-yielding varieties of bajra are more susceptible to infestation. Ergot-infested grains can be easily removed by floating them in 20 per cent salt water. They can also be removed by hand-picking or air floatation. The upper safe limit for the ergot alkaloids has been estimated to be 0.05 mg per 100 grams of the food material.

4. Epidemic Dropsy *(162, 163)*

From time to time, outbreaks of "epidemic dropsy" are reported in India. The cause of epidemic dropsy was not known until 1926, when Sarkar ascribed it to the contamination of mustard oil with argemone oil. Lal and Roy (1937) and Chopra et.al., (1939) gave experimental proof of the cause of epidemic dropsy. Mukherji et.al., (1941) isolated a toxic alkaloid, *sanguinarine* from argemone oil and found out its chemical formula. This toxic substance interferes with the oxidation of pyruvic acid which accumulates in the blood.

The symptoms of epidemic dropsy consist of sudden, non-inflammatory, bilateral swelling of legs, often associated with diarrhoea. Dyspnoea, cardiac failure and death may follow.

Some patients may develop glaucoma. The disease may occur at all ages except breast-fed infants. The mortality varies from 5-50 per cent.

The contamination of mustard or other oils with argemone oil may be accidental or deliberate. Seeds of *Argemone mexicana* (prickly poppy) closely resemble mustard seeds. The plant grows wild in India. It has prickly leaves and bright yellow flowers. Crops of mustard are gathered during March, and during this period, the seeds of argemone also mature and are likely to be harvested along with mustard seeds. Sometimes unscrupulous dealers mix argemone oil with mustard or other oils.

Argemone oil is orange in colour with an acrid odour. The following tests may be applied for the detection of argemone oil : (1) *Nitric acid test* : A simple test is to add nitric acid to the sample of oil in a test tube. The tube is shaken and the development of a brown to orange-red colour shows the presence of argemone oil. The nitric acid test is positive only when the level of argemone oil is about 0.25 per cent *(167)*. (2) *Paper Chromatography test* : This is the most sensitive test yet devised. It can detect argemone oil up to 0.0001 per cent in all edible oils and fats.

The accidental contamination of mustard seeds can be prevented at the source by removing the argemone weeds growing among oil-seed crops. Unscrupulous dealers may be dealt with by the strict enforcement of the Prevention of Food Adulteration Act.

5. Endemic Ascites *(164, 165)*

In Kusmi Block of Sarguja district in Madhya Pradesh, during 1973 and again during 1976, an outbreak of rapidly developing ascites and jaundice was reported among the Nagesia tribals. Both the sexes and all the age groups, except infants, were affected. The overall mortality was 40 per cent.

Studies conducted by the National Institute of Nutrition, Hyderabad showed that the local population subsist on the millet *Panicum miliare* (known locally as Gondhli) which gets contaminated with weed seeds of Crotalaria (locally known as *Jhunjhunia*). On chemical analysis, *Jhunjhunia seeds* were found to contain *pyrrolizidine alkaloids* which are *hepatotoxins*.

The preventive measures comprise educating the people in the affected areas about the disease, deweeding of the Jhunjhunia plants which grow along with the staple, and simple sieving of the millet at the household level to remove the seeds of *Jhunjhunia* which are considerably smaller than those of the millet.

6. Fusarium toxins *(166)*

Fusarium species of field fungi are known to contaminate food crops and pose health hazards to livestock and man. The problem of fusarium contamination of sorghum is believed to be on the increase. Rice is also known to be a good substrate for fusarium. Work is now in progress at the National Institute of Nutrition to isolate, and identify the toxic metabolites produced by *fusarium incamatum*.

Food additives are defined which are added intentionall quantity, to improve its appear properties *(148)*. This definit adjuncts which may result in components of packing materia food *(149)*.

Food additives may be c Additives of the **first categor** saffron, turmeric), flavouring sweeteners (e.g., saccharin), sodium benzoate), acidity im acetic acid), etc *(150)*. These safe for human consumptic **category** are, strictly spea through packing, processi (insecticides) or other en Uncontrolled or indiscriminate health hazards among con preservatives such as nitrite production of toxic substanc been implicated in cancer aeti

The use of food additiv regulations throughout the (viz. the Prevention of Food Products Order) govern the additives *(151)*. Any food tha not permitted is considered to limit exceeds then also the fo nature and quantity of the ac the label to be affixed to extraneous colouring matter food, the words "Artificially label. At the international le programme on food standarc WHO Codex Alimentarius C Protection of the health of co Commission. The ultimate ef an important problem of pub concern to the public and the

Food fortification

Fortification of food is at reinforcing the usual d additional supplies to pr disorders. WHO *(1)* has def process whereby nutrients a small quantities) to maintain of a group, a community, or a

Programmes of demonstr of food or water are : fluorid dental caries; iodization of s endemic goitre, and food fo with vitamins A and D. developed for the twin fortific

In order to qualify as suit

erally causes local reactions and also very frequently malaise, headache, and ubside within 36 hours. To has been advised that the the afternoon or evening. s may be administered to s. Women should not be

es should be stored in a ould not be frozen. Under cy is retained for a period of

oid are on the scene *(19)*. *S.typhi*, Ty 21a developed sts, are being evaluated as in Chile and Egypt have a in more than 500,000 f a liquid formulation gave 3 years. Clinical trials with ve only recently begun, but has been safe and highly

: Based on the results of is registered in more than oral is an enteric-coated ontains not less than 10^9 *Salmonella typhi* strain completely devoid of ble change in cell-wall munization of adults and e capsule is administered e, one hour before a meal r. Protection commences e, and lasts for at least doses) is recommended,

n congenital or acquired treatment with ugs, acute febrile illness

it. Med.J., 3 : 329-331
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eport of the Director General
ed.Med. Sc. (INDIA) 24 (4)
n of India 1995 and 1996, Delhi..
Epidemiology and Clinical
es., 55 : 219
o. 78
Feb. 1984)
l, 2 : 56
plement No. 1 56 : 45
WHO, 32 : 29-36
p.Med & Hyg., 74 (5) 553
mmunization, The Indian
69-474

FOOD POISONING

Food poisoning is an acute gastro-enteritis caused by ingestion of food or drink contaminated with either living bacteria or their toxins or inorganic chemical substances and poisons derived from plants and animals. The condition is characterised by (a) history of ingestion of a common food (b) attack of many persons at the same time and (c) similarity of signs and symptoms in the majority of cases.

Types of Food Poisoning

Food poisoning may be of two types : non-bacterial and bacterial. *(a) Non-bacterial :* Caused by chemicals such as arsenic, certain plant and sea foods. In recent years, there has been a growing concern about contamination of food by chemicals, e.g., fertilisers, pesticides, cadmium, mercury. *(b) Bacterial :* Caused by the ingestion of foods contaminated by living bacteria or their toxins. The conventional classification of bacterial food poisoning into toxic and infective types is becoming increasingly blurred with the knowledge that in some types, both multiplication and toxin production are involved *(1)*. Bacterial food poisoning may be of the following types :

Salmonella Food Poisoning

An extremely common form of food poisoning. Five reasons have been given for its increase in recent years : (a) an increase in communal feeding (b) increase in international trade in human food (c) a higher incidence of salmonellosis in farm animals (d) widespread use of house-hold detergents interfering with sewage treatment, and (e) wide distribution of "prepared foods" *(2)*.

(a) AGENT(S) : The species most often incriminated in human outbreaks are *S. typhimurium, S. cholera-suis* and *S. enteritidis,* besides many others. (b) SOURCE : Salmonellosis is primarily a disease of animals. Man gets the infection from *farm animals* and *poultry* - through contaminated meat, milk and milk products, sausages, custards, egg and egg products. Rats and mice are another source; they are often heavily infected and contaminate foodstuffs by their urine and faeces. Temporary human carriers can also contribute to the problem. (c) INCUBATION PERIOD : 12 to 24 hours commonly (d) MECHANISM OF FOOD POISONING : The causative organisms, on ingestion, multiply in the intestine and give rise to acute enteritis and colitis. The onset is generally sudden with chills, fever, nausea, vomiting, and a profuse watery diarrhoea which usually lasts 2-3 days. Mortality is about 1 per cent. A convalescent carrier state lasting for several weeks may occur *(1)*.

Salmonellosis is described in detail separately.

Staphylococcal Food Poisoning

It is about as common as salmonella food poisoning. (a) AGENT : Enterotoxins of certain strains of coagulase-positive *Staphylococcus aureus*. (At least 5 different enterotoxins have been identified, and a sixth may exist *(3)*. Toxins can be formed at optimum temperatures of 35 deg. to 37 deg. C. These toxins are relatively heat stable and resist boiling for 30 minutes or more (b) SOURCE : Staphylococci are ubiquitous in nature, and are found on the skin and in the nose and throat of men and animals. They are a common agent of boils and pyogenic infections of man and animals. Cows suffering from mastitis have been responsible for outbreaks of food poisoning involving milk and milk products. The foods involved are salads, custards, milk and milk products which get contaminated by staphylococci. (c) INCUBATION PERIOD : 1-6 hours. The incubation period is short because of "preformed" toxin. (d) MECHANISM OF FOOD POISONING : Food poisoning results from ingestion of toxins preformed in the food in which bacteria have grown ("intradietetic" toxins).

Since the toxin is heat-resistant, it can remain in food after the organisms have died. The toxins act directly on the intestine and CNS. The illness becomes manifest by the sudden onset of vomiting, abdominal cramps and diarrhoea. In severe cases, blood and mucus may appear. Unlike salmonella food poisoning, staphylococcal food poisoning rarely causes fever. Death is uncommon.

Botulism

Most serious but rare. It kills two-thirds of its victims. (a) AGENT : Exotoxin of *Clostridium botulinum* generally Type A, B or E. (b) SOURCE : The organism is widely distributed in soil, dust and the intestinal tract of animals and enters food as spores. The foods most frequently responsible for botulism are home preserved foods such as home-canned vegetables, smoked or pickled fish, home-made cheese and similar low acid foods *(4)*. In fact, botulism derives its name from the Latin word for sausage (botulus). (c) INCUBATION PERIOD : 12 to 36 hours. (d) MECHANISM OF FOOD POISONING : The toxin is preformed in food ("intradietetic") under suitable anaerobic conditions. It acts on the parasympathetic nervous system. Botulism differs from other forms of food poisoning in that the gastrointestinal symptoms are very slight. The prominent symptoms are dysphagia, diplopia, ptosis, dysarthria, blurring of vision, muscle weakness and even quadriplegia. Fever is generally absent, and consciousness is retained. The condition is frequently fatal, death occurring 4-8 days later due to respiratory or cardiac failure. Since the toxin is thermolabile, the heating of food which may be subjected to 100 deg. C for a few minutes before use will make it quite safe for consumption.

Botulism occurring in infants is called "infant botulism". It is due to infection of the gut by *Cl. botulinum* with subsequent in vivo production of toxin *(5)*.

Antitoxin is of considerable value in the prophylaxis of botulism. When a case of botulism has occurred, antitoxin should be given to all individuals partaking of the food. The dose varies from 50,000 to 100,000 units IV *(6)*. The antitoxin will be of no avail if the toxin is already fixed to the nervous tissue. Guanidine hydrochloride given orally in doses of 15 to 40 mg/kg of body weight has been shown to reverse the neuromuscular block of botulism. When combined with good medical and nursing care, the drug can be a useful adjunct in the treatment of botulism *(7)*. Active immunization with botulinum toxoid to prevent botulism is also available *(8)*.

Cl. perfringens Food Poisoning

(a) AGENT : *Cl. perfringens (welchii)*. (b) SOURCE : The organism has been found in faeces of humans and animals, and in soil, water and air. The majority of outbreaks have been associated with the ingestion of meat, meat dishes and poultry. The usual story is that the food has been prepared and cooked 24 hours or more before consumption, and allowed to cool slowly at room temperature and then heated immediately prior to serving. (c) INCUBATION PERIOD : 6 to 24 hours, with a peak from 10 to 14 hours (d) MECHANISM OF FOOD POISONING : The spores are able to survive cooking, and if the cooked meat and poultry are not cooled enough, they will germinate. The organisms multiply between 30 deg. and 50 deg. C and produce a variety of toxins, e.g., alpha toxin, theta toxin, etc. Prevention consists either by cooking food just prior to its consumption or, if it has to be stored, by rapid and adequate cooling *(10)*. (e) CLINICAL SYMPTOMS : The most common symptoms are diarrhoea, abdominal cramps and little or no fever, occurring 8 to 24 hours after consumption of the food. Nausea and vomiting are rare. Illness is usually of short duration, usually 1 day or less. Recovery is rapid and no deaths have been reported.

B. cereus Food Poisoning

Bacillus cereus is an aerobic, spore-bearing, motile, gram positive rod. It is ubiquitous in soil, and in raw, dried and processed foods. The spores can survive cooking and germinate and multiply rapidly when the food is held at favourable temperatures. *B. cereus* has been recognised as a cause of food poisoning, with increasing frequency in recent years.

Recent work has shown that *B. cereus* produces at least 2 distinct enterotoxins, causing 2 distinct forms of food poisoning. One, the *emetic form* with a short incubation period (1-6 hours) characterised by predominantly upper gastro-intestinal tract symptoms, rather like staphylococcal food poisoning. The other, the *diarrhoeal* form, with a longer incubation period (12-24 hours) characterised by predominantly lower intestinal tract symptoms like *Cl perfringens* food poisoning (diarrhoea, abdominal pain, nausea with little or no vomiting and no fever. Recovery within 24 hours is usual). The toxins are preformed and stable.

Diagnosis can be confirmed by isolation of 10^5 or more B. cereus organisms per gram of epidemiologically incriminated food. Treatment is symptomatic.

Differential diagnosis

Food poisoning may be mistaken for cholera, acute bacillary dysentery and chemical (arsenic) poisoning. The differentiating points between cholera and food poisoning are given in Table 1.

TABLE 1

Differential diagnosis of cholera and Food Poisoning

		Cholera	Food poisoning
1.	Epidemiology	Occurs often in epidemic form associated with other cases in the neighbourhood	Often a single group of persons who shared a common meal
		Secondary cases occur	No secondary cases
2.	Incubation	From a few hours up to 5 days	1 to 24 hours
3.	Onset	With purging	With vomiting
4.	Nausea and retching	None	Present
5.	Vomiting	Projectile, effortless, watery and continuous	Often single, severe vomit, mucus and blood streaked
6.	Stools	Copious rice watery, inoffensive	Frequent, may contain mucus and blood, offensive
7.	Tenesmus	None	Yes
8.	Abdominal tenderness	None	Yes
9.	Dehydration	Very marked	Distinct
10.	Muscular cramps	Constant and severe	Less constant
11.	Surface temperature	Subnormal	Often upto 100-102 deg.F.
12.	Headache	None	Often
13.	Urine	Suppressed	Seldom suppressed
14.	Blood	Leucocytosis	Normal

INVESTIGATION OF FOOD POISONING

(a) Secure complete list of people involved and their history : All the people who have shared part of the food should be interviewed. They may be supplied questionnaires concerning the foods eaten during the previous 2 days, and place of consumption; time of onset of symptoms; symptoms of illness (e.g., nausea, vomiting, diarrhoea, abdominal pain, headache, fever, prostration, etc.) in order of occurrence; personal data such as age, sex, residence, occupation, and any other helpful information. Questionnaires may be administered to kitchen employees and those working in the dining halls. *(b) Laboratory investigations* : An important part of the investigation. The object is not only to incriminate the causative agent from stool, vomit or remnants of food by inoculating into appropriate media, but also to determine the total number of bacteria and the relative numbers of each kind involved. This will give a better indication of the organism involved. Stool samples of the kitchen employees and food handlers should also be investigated. The samples should be examined aerobically and anaerobically. Phage typing of the organisms should be done to complete the laboratory investigation. *(c) Animal experiments* : It may be necessary to feed rhesus monkeys with the remnants of food. Protection tests are useful in the case of botulism; in this, a saline filtrate of food-stuff is injected subcutaneously into mice protected with antitoxic sera, keeping suitable controls. *(d) Blood for antibodies* : This is useful for retrospective diagnosis. *(e) Environmental study* : This includes inspection of the eating place(s), kitchen(s), and questioning of food handlers regarding food preparation. *(f) Data analysis* : The data should be analysed according to the descriptive methods of time, place and person distribution. Food-specific attack rates should be calculated. A case control study may be undertaken to establish the epidemiologic association between, illness and the intake of a particular food.

Prevention and control :

(a) FOOD SANITATION : *(i) Meat inspection* : The food animals must be free from infection. This can be ensured by their examination by veterinary staff, both before and after slaughter. *(ii) Personal hygiene* : A high standard of personal hygiene among individuals engaged in the handling, preparation and cooking of food is needed. *(iii) Food handlers* : Those suffering from infected wounds, boils, diarrhoea, dysentery, throat infection, etc should be excluded from food handling. The medical inspection of food handlers is required in many countries; this is of limited value in the detection of carriers, although it will remove some sources of infection *(12)*. *(iv) Food handling techniques* : The handling of ready-to-eat foods with bare hands should be reduced to a minimum. Time between preparation and consumption of food should be kept short. The importance of rapid cooling and cold storage must be stressed. Milk, milk products and egg products should be pasteurised. Food must be thoroughly cooked. The heat must penetrate the centre of the food leaving thereby no cool spots. Most food poisoning organisms are killed at temperatures over 60 deg. C. *(v) Sanitary improvements* : Sanitization of all work surfaces, utensils and equipments must be ensured. Food premises should be kept free from rats, mice, flies and dust. *(vi) Health education* : Food handlers should be educated in matters of clean habits and personal hygiene, such as frequent and thorough hand washing.

(b) REFRIGERATION : In the prevention of bacterial food poisoning, emphasis must be placed on proper temperature control. Food should not be left in warm pantries; a few germs can multiply to millions by the next morning. Foods not eaten immediately should be kept in cold storage to prevent bacterial multiplication and toxin production. "Cook and eat the same day" is a golden rule. When foods are held between 10 deg. C (50 deg. F) and 49 deg. C (120 deg.F) they are in the danger zone for bacterial growth. Cold is bacteriostatic at temperature below 4 deg. C (40 deg.F), and refrigeration temperature should not exceed this level.

SURVEILLANCE : Food samples must be obtained from the food establishments periodically and subjected to laboratory analysis if they were unsatisfactory. Continuing surveillance is necessary to avoid outbreaks of food-borne diseases.

References

1. Mandal, B.K. (1981). *Medicine International,* 2 : 56
2. Beveridge, W.I.B. (1967). in *Health of Mankind,* 100th Symposium, Ciba Foundation, Churchill, London
3. Werner, S.B. (1980). in *Maxcy-Rosenau : Public Health and Preventive Medicine,* 11th ed, John M. Last (ed), Appleton-Century Crofts, New York
4. WHO (1976). *Tech. Rep. Ser.,* No.598
5. Arnon, S.S. et al (1977). *JAMA,* 237 : 1946-1951
6. Christie, A.B. (1980). *Infectious Diseases : Epidemiology and Clinical Practice,* 3rd ed., Churchill Livingstone
7. Ryan, D.W. et al (1971). *JAMA,* 216 : 513
8. Stuart, P.F. et al (1970). *Cand. J. Pub. Health,* 61 (6) 509
9. Nakamura, M. et al (1970). *Annual Review of Microbiology,* 24 : 359-367
10. Bailey, J. (1977). *Guide to Hygiene and Sanitation in Aviation,* WHO, Geneva.
11. Terranova, W. et al (1978). *N. Eng. J. Med.,* 298 : 143
12. WHO (1980). *WHO Chronicle,* 34 (2) 83.

AMOEBIASIS

The term "amoebiasis" has been defined by WHO *(1)* as the condition of harbouring the protozoan parasite *Entamoeba histolytica* with or without clinical manifestations. The symptomatic disease occurs in less than 10 per cent of infected individuals *(2)*. The symptomatic group has been further subdivided into intestinal and extraintestinal amoebiasis. Only a small percentage of those having intestinal infection will develop invasive amoebiasis. The intestinal disease varies from mild abdominal discomfort and diarrhoea to acute fulminating dysentery. Extraintestinal amoebiasis includes involvement of liver (liver abscess), lungs, brain, spleen, skin, etc. Amoebiasis is a potentially lethal disease. It carries substantial morbidity and mortality.

Problem statement

WORLD : Amoebiasis is a common infection of the human gastro-intestinal tract. It has a world-wide distribution. It is a major health problem in the whole of China, South East and West Asia and Latin America, especially Mexico. Globally it is estimated that, in 1997, 45 million people carried *E. histolytica* in their intestinal tract and approximately one-tenth of infected people, suffered from invasive amoebiasis. It is probable that invasive amoebiasis, accounted for about 70,000 deaths in the world *(3)*. Prevalence rates vary from as low as 2 per cent to 60 per cent or more in areas devoid of sanitation *(4)*. In areas of high prevalence, amoebiasis occurs in endemic forms as a result of high levels of transmission and constant reinfection. Epidemic water-borne infections can occur if there is heavy contamination of drinking water supply.

INDIA : It is generally agreed that amoebiasis affects about 15 per cent of the Indian population *(5)*. Amoebiasis has been reported throughout India : the prevalence rate is about 15% ranging from 3.6 to 47.4 per cent in different areas *(4)*. The reported variations in prevalence are attributed to variations in clinical diagnostic criteria *(6)* and to technical difficulties in establishing a correct diagnosis and lack of sampling criteria *(7)*.

Table 23.5. Food Borne Disease Caused by Some Pathogenic Organisms

	Microorganism	Foods commonly involved	Ill-effects and diseases
Bacterial	Bacillus cereus	Cereal products	Nausea, vomiting abdominal pain
	Cl. Botulinus toxins	Defectively processed meat and fish and honey	Botulism (muscular paralysis, death due to respiratory failure)
	Cl. perfringens (Welchii)	Defectively processed precooked meat	Nausea, Abdominal pain and diarrhoea
	Salmonella	Defectively processed meat, fish and egg products, Raw vegetables grown on sewage	Salmonellosis (vomiting, diarrhoea and fever)
	Shigella sonnei	Foods kept exposed for sale in unhygienic surroundings	Bacillary dysentery
	Staphylococcus aureus	,,	Increased salivation, vomiting, abdominal pain and diarrhoea
	Streptococcus pyogenes	,,	Scarlet fever, septic sore throat
Fungal	Aspergillus flavus (Aflatoxin)	Corn and groundnut infected with Aspergillus flavus	Liver damage and cancer
	Claviceps purpurea (Ergot)	Rye and pearl millet infested with ergot	Ergotism (Burning sensation in extremities, peripheral gangrene)
	Fusarium sporotrichioides	Cereals and millets infected with fusarium Rice	Alimentary toxic aleukia
	Penicillium islandicum		Liver damage
Parasitic	Trichinella spiralis	Pork and pork products	Nausea, vomiting, diarrhoea, colic and muscular pains (Trichinosis)
	Ascaris lumbricoides	Raw vegetables grown on sewage farms	Ascariasis
	Entamoeba histolytica	,,	Amoebic dysentery
	Ancylostoma duodenale (Hookworms)	,,	Epigastric pain, loss of blood in anaemia

• **Mycotoxin** :- The term mycotoxin come from the greek word "mykes" meaning fungus & the latin word "toxicum", meaning poison or toxins. Mycotoxins are these substances produced by molds, like Aspergillus flavus, etc. in agricultural commodities either during pre or post harvest stages, which may be toxin or produce adverse effect in living organisms especially animals and human.

poisoning caused by ingestion of food containing mycotoxin is called mycotoxicosis. There are several types of mycotoxicosis like —

(Aflatoxin and Ergotism will be here)

Aflatoxins

Aflatoxin is a chemical substance toxic to the human body produced by the fungus Aspergillus, of the species flavous, parasiticus etc.

During 1974 aflatoxicosis disease was found to spread among tribles in western India.

Chemistry :- The two major metabolised of aflatoxins are known as B_1 & G_1, the former being blue in colour while the latter is green. These aflatoxins have a highly complicated structure. The other forms are B_2, G_2, M_1, M_2, P_1 etc

Toxicity :- Aflatoxin B_1 is the most toxic among all the aflatoxin & is harmful for various animals Aflatoxin may even be carcinogenic.

Food source :- Since the discovery of Aflatoxin several food products containing these product has been isolated. The most common are cereal, pulses, dairy product, fruit juices, pea nuts, corn etc.

Symptoms :- The most imp. characteristic feature of aflatoxicosis are jaundis, cirrhosis, and other liver diseases. Long term exposures to aflatoxins could lead to chronic hepatitis & primary liver cancer.

Prevention :- Aflatoxins are quite stable in many food & are fairly resistent to degradation. The complet elimination of human & animal food, while desirable is extreamly unlikly as the climatic conditions determine the occurance of aflatoxin during preharvest, harvest & post harvest stages.

prevention measures include drying the agricultural commodities well keeping the commodities dry & decontaminated. Detoxification using ammonia is also useful.

• Ergotism

Ergot is a disease of cereal crops like wheat, millet, sorghum, Ergot of rye is a plant disease caused by the fungus claviceps purpurea. The infected flower, instead of developing into a normal grain develops to a dark black mass often refered to as Ergot. The ergot grain get harvested along with the good grains & contamination spreads.

This disease was common in Europe in the middle of age. In India it is caused by the consumption of bajra.

Symptoms! In India ergotism is characterised by - nausea, vomiting, giddiness, prolong sleepiness convulsions, gangrin.

• Mycotoxin! - The term mycotoxin come from the greek word "mykes" meaning fungus & the latin word "toxicum", meaning poison or toxins. Mycotoxins are those substances produced by molds, like Aspergillus flavous, etc. in agricultural commodities either during pre or post harvest stages, which may be toxin or produce adverse effect in living organisms especially animals and human.

poisoning caused by ingestion of food containing mycotoxin is called mycotoxicosis. There are several types of mycotoxicosis like —

(Aflatoxin and Ergotism will be here.)

moldiness and ropiness usually termed mould ro

Food Spoilage	Food poisoning
1) Spoilage is the condition in which food is destroyed i.e, its quality is lowered.	1) Poisoning is a condition in which some toxin or harmful chemical develop in the food that may cause various toxic symptoms.
2) Spoilage may be due to any kind of adverse change in the food like bacterial growth, deterioration of food molecules etc.	2) Poisoning mainly result of presence bacterial itself chemical toxin etc.
3) Food spoilage is a process which can lead to poisoning. Food spoilage → Poison.	3) Food poisoning is the effect in the human body, i.e. seen due to consumption of deteriorate food.
4) Spoilage may be microbiological, chemical, physical biological.	4) Poisoning is specially due to microbiological cause only.

milk and Water