Food borne diseases

Food borne diseases (FBD) are acute illnesses associated with the recent consumption of food.
The food involved is usually contaminated with a disease pathogen or toxicant.
Such food contains enough pathogens or toxicant necessary to make a person sick.

Human Gastrointestinal Disorders

The causes of foodborne gastrointestinal disorders can be broadly divided into three groups:

1. From consumption of food and water containing viable pathogenic microorganisms or their preformed toxins.
2. From ingestion of pathogenic algae, parasites, and their preformed toxins through food.
3. For reasons other than viable pathogens or their toxins.

Some of the factors included in Group 3 are:

1. Ingestion of toxins naturally present in many foods. This includes certain mushrooms, some fruits and vegetables, and some seafoods.
2. Toxins formed in some foods. Examples are some biological amines (e.g., histamine) that form in some fish, cheeses, and fermented meat products.
3. The presence of toxic chemicals in contaminated food and water, such as heavy metals and some pesticides.
4. Allergy to some normal components of a food. Some individuals are allergic to gluten in cereals and develop digestive disorders following consumption of food containing gluten.
5. Genetic inability to metabolize normal food components. The inability of some individuals to hydrolyze lactose in the small intestine, because of the lack of production of the enzyme lactase, results in digestive disorders (lactose intolerance).
6. Nutritional disorders such as rickets, which is caused by calcium deficiency.
7. Indigestion from overeating or other reasons.

Cost of Foodborne Diseases

Foodborne illnesses can be fatal as well as cause suffering, discomfort, and debilitation among the survivors. The economic losses from various factors, such as medical treatment, lawsuits, lost wages and productivity, loss of business, recall and destruction of products, and investigation of the outbreaks, can be very high. In the U.S., the annual cost of foodborne diseases is estimated to be more than $20 billion.

Most pathogens include (Campylobacter jejuni, Clostridium perfringens, Escherichia coli O157:H7, Listeria monocytogenes, Salmonella, Staphlococcus aureus, and Toxoplasma gondii)

Types of Microbial Foodborne Diseases

Foodborne diseases in humans result from the consumption of either food and water contaminated with viable pathogenic bacterial cells (or spores in the case of infant botulism) or food containing toxins produced by toxigenic bacteria and molds. On the basis of mode of illnesses, these can be arbitrarily divided into three groups: intoxication or poisoning, infection, and toxicoinfection.

Table 23.2 Confirmed Foodborne Disease Outbreaks, Cases, and Deaths by Etiological Agents from 1983 to 1987 in the U.S.

Etiological Agents[a]	Outbreaks		Cases		Deaths	
	No.	%	No.	%	No.	%
Bacterial	600	66.0	50,304	92.2	132	96.4
Viral	41	4.5	2,789	5.1	1	0.7
Parasitic[b]	36	4.0	203	0.4	1	0.7
Chemicals[c]	232	25.5	1,244	2.3	3	2.2
Total	909	100	54,540	100	137	100

[a]No incidence from mycotoxins was reported.
[b]Includes *Trichinella spiralis* and Giardia.
[c]Includes Ciguatoxin, scombrotoxin, mushrooms, heavy metals, and other chemicals.

1. Intoxication

Illness occurs as a consequence of ingesting a preformed bacterial or mold toxin because of its growth in a food. A toxin has to be present in the contaminated food. Once the microorganisms have grown and produced toxin in a food, there is no need of viable cells during consumption of the food for illness to occur. Staph food poisoning is an example.

2. Infection

Illness occurs as a result of the consumption of food and water contaminated with enteropathogenic bacteria or viruses. It is necessary that the cells of enteropathogenic bacteria and viruses remain alive in the food or water during consumption. Viable cells, even if present in small numbers, have the potential to establish and multiply in the digestive tract to cause the illness. Salmonellosis and hepatitis A are examples.

3. Toxicoinfection

Illness occurs from ingesting a large number of viable cells of some pathogenic bacteria through contaminated food and water. Generally, the bacterial cells either sporulate or die and release toxins to produce the symptoms. Clo. Perfringens gastroenteritis is an example.

Although many pathogenic bacterial species and viruses have been implicated with foodborne (and waterborne) disease outbreaks, some have occurred at a higher frequency than others.

Table 23.4 Predominant Bacterial and Viral Pathogens Associated with the Confirmed Foodborne Diseases from 1983 to 1987 in the U.S.

Bacteria and Viruses*	Outbreaks		Cases		Deaths	
	No.	%	No.	%	No.	%
Staphylococcus aureus	47	7.6	3,181	6.2	0	0
Clostridium botulinum	74	11.9	140	0.3	10	17.0
Salmonella	342	55.1	31,245	61.1	39	66.0
Shigella spp.	44	7.1	9,971	19.5	2	3.4
Escherichia coli	7	1.1	640	1.3	4	6.8
Campylobacter spp.	28	4.5	727	1.4	1	1.7
Clostridium perfringens	24	3.9	2,743	5.4	2	3.4
Bacillus cereus	16	2.6	261	0.5	0	0
Hepatitis A virus	29	4.7	1,067	2.0	1	1.7
Norwalk-like virus	10	1.5	1,164	2.3	0	0
Total	621	100	51,139	100	59	100

*Not included in the table are *Brucella* spp., *Streptococcus* spp., *Vibrio* spp., and several others, which combined were associated with 18 (2.8%) outbreaks. *Lis. monocytogenes* caused 3 outbreaks affecting 259 people, with 70 deaths. See Table 23.11 for current trends.

Human Factors in Foodborne Disease Symptoms

When a group of people consumes food contaminated with live cells of pathogens or their toxins, all the members might not develop disease symptoms. Among those who develop symptoms, all might not show either the same symptoms or the same severity of a symptom. This is probably due to the difference in resistance among individuals. One of the factors involved in developing symptoms from the consumption of a contaminated food is susceptibility of an individual to the contaminants. In general, infants and old, sick, and immunodeficient people are more susceptible than normal adults and healthy individuals. The chance of developing disease symptoms is directly related to the amount of a contaminated food consumed. This is related to the number of viable cells of a pathogen or the amount of a toxin consumed by an individual. The virulence of a pathogen or a toxin consumed through a food also determines the onset of a disease and severity of symptoms.

Sequence of Events in a Foodborne Disease

For a foodborne disease to occur, several events have to happen in sequence (Figure 23.1). Understanding these sequences is helpful to investigate the cause (source and means of transmission) of a foodborne disease. It also helps recognize how the sequence can be broken in order to stop a foodborne disease. Initially, there has to be a source of a pathogen. Next, the pathogen has to contaminate a food. Consumption of the food contaminated with a pathogenic virus can lead to viral infection.

For bacterial pathogens (and toxicogenic molds) the contaminated food has to support growth and be exposed for a certain period of time at a suitable temperature to enable the pathogens to grow.

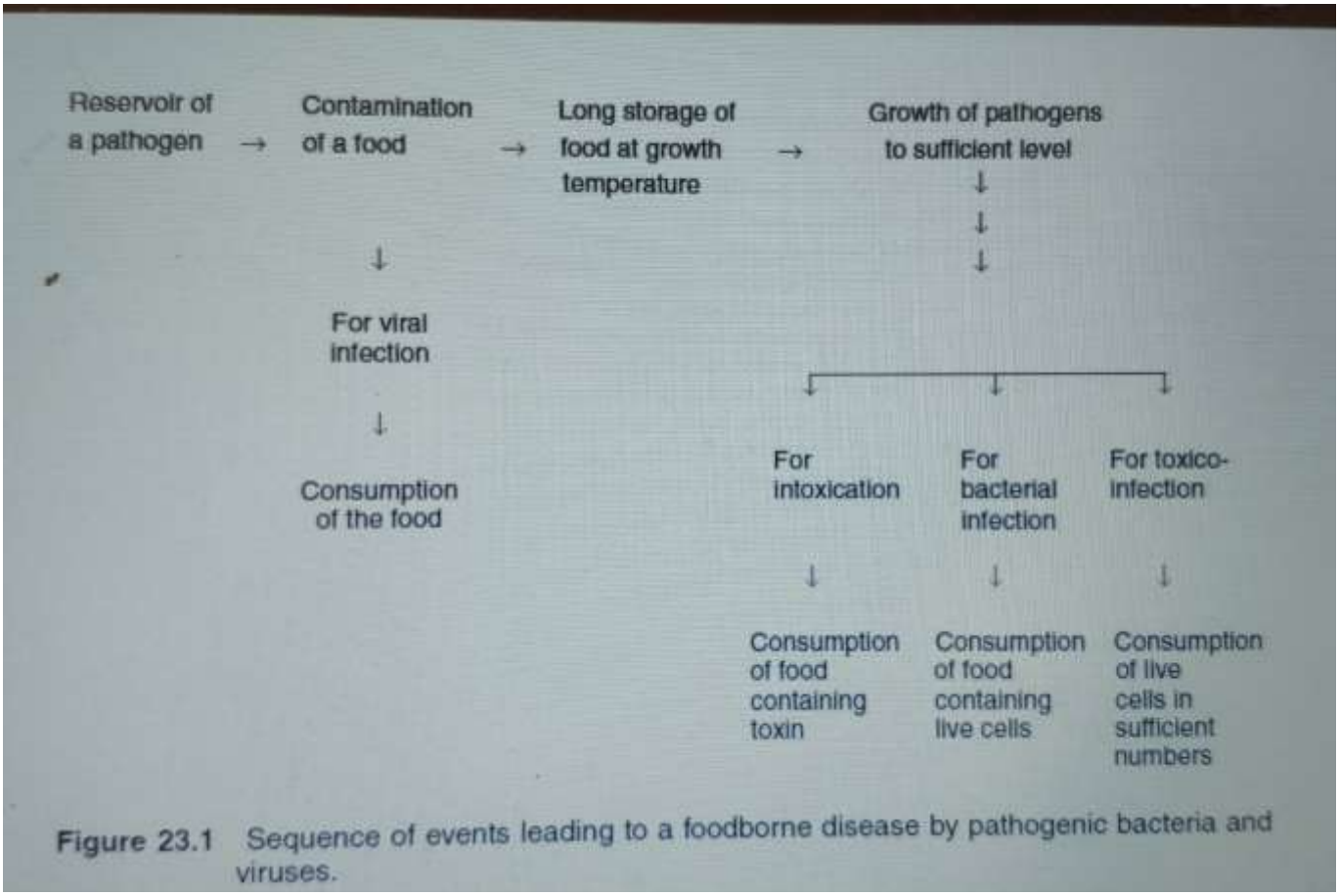

Figure 23.1 Sequence of events leading to a foodborne disease by pathogenic bacteria and viruses.

Foodborne intoxication or food poisoning of microbial origin occurs by ingesting a food containing a preformed toxin. Two of bacterial origin, staphylococcal intoxication and botulism, and mycotoxicosis of mold origin are briefly discussed in this chapter. Although Bacillus cereus can also form a heat-stable toxin and produce intoxication, this aspect is discussed under toxicoinfection. The discussions include relative importance of a disease, characteristics of the microorganisms involved,

predominant types of food, nature of the toxins, disease and the symptoms, preventative measures, and, for some, analysis of actual outbreak.

Some general characteristics of food poisoning are:

1. The toxin is produced by a pathogen while growing in a food.
2. A toxin can be heat labile or heat stable.
3. Ingestion of a food containing active toxin, not viable microbial cells, is necessary for poisoning (except for infant botulism, in which viable spores need to be ingested).
4. Symptoms generally occur quickly, as early as 30 min after ingestion.
5. Symptoms differ with type of toxin; enterotoxins produce gastric symptoms and neurotoxins produce neurological symptoms.
6. Febrile symptom is not present.

Staphylococcal Intoxication

Staphylococcal food poisoning (staphylococcal gastroenteritis; staphylococcal food poisoning; staph food poisoning), caused by toxins of Staphylococcus aureus, is considered one of the most frequently occurring foodborne diseases worldwide.

Habitat

Enterotoxin-producing Sta. aureus strains have generally been associated with staphylococcal food intoxication. Although strains of several other Staphylococcus species are known to be enterotoxin producers, their involvement in food poisoning is not fully known. Sta. aureus, along with many other staphylococci, are naturally present in the nose, throat, skin, and hair of healthy humans, animals, and birds. Sta. aureus can be present in infections, such as cuts in skin and abscesses in humans, animals, and birds, and cuts in hands and facial-erupted acne in humans. Food contamination generally occurs from these sources.

Nutritional Requirements for Growth

Staphylococci are typical of other Gram-positive bacteria in having a requirement for certain organic compounds in their nutrition. Amino acids are required as nitrogen sources, and thiamine and nicotinic acid are required among the B vitamins. When grown anaerobically, they appear to require uracil. In one minimal medium for aerobic growth and enterotoxin production, monosodium glutamate serves as C, N, and energy sources. This medium contains only three amino acids (arginine, cystine, and phenylalanine) and four vitamins (pantothenate, biotin, niacin, and thiamine), in addition to inorganic salts Arginine appears to be essential for enterotoxin B production.

Toxins and Toxin Production

Enterotoxigenic strains of Sta. aureus produce seven different enterotoxins: A, B, C1, C2, C3, D, and E (also designated as SEA, SEB, etc.). Thirteen staphylococcal enterotoxins (SEs) were identified.

They are serologically distinct heat-stable proteins and differ in toxicity. The staphylococcal enterotoxins are simple proteins with molecular weights between 26,000 and 30,000 dalton. The single polypeptide chains are cross-linked by a disulfide bridge to form a characteristic cystine loop. Since many of the amino acids in this loop are similar in each of the enterotoxin types, it is thought to be the toxic part of the molecule. The toxins vary in heat stability, SEB being more stable than SEA. Normal temperature and time used to process or cook foods do not destroy the potency of the toxins. Outbreaks from SEA are more frequent, probably because of its high potency.

Rate of toxin production by a strain is directly related to its rate of growth and cell concentrations. Optimum growth occurs ca. 37 to 40 C. Under optimum conditions of growth, toxins can be detected when a population has reached over a few million per gram or milliliter of food and generally in ca. 4 h. Some of the lowest environmental parameters of toxin production are 10 C, pH 5.0, or water activity 0.86. However, by combining two or more parameters, the lowest ranges can be adversely affected.

Disease and Symptoms

Staphylococcal toxins are enteric toxins and cause gastroenteritis. A healthy adult has to consume ca. 30 g or ml of a food containing 100 to 200 ng toxins produced by $10^{6–7}$ cells/g or /ml; infants and old and sick individuals need lesser amounts.

The symptoms occur within 2 to 4 h, with a range of 30 min to 8 h, and are directly related to the potency and amounts of toxin ingested and an individual's resistance. The disease lasts for ca. 1 to 2 d and is rarely fatal. The primary symptoms, from stimulation of the autonomic nervous system by the toxins, are salivation, nausea and vomiting, abdominal cramps, and diarrhea. Some secondary symptoms are sweating, chills, headache, and dehydration.

Food Association

Many foods have been implicated in staphylococcal foodborne outbreaks. In general, the bacterium grows in the food and produces toxins without adversely affecting the acceptance quality. Many protein-rich foods, foods that are handled extensively, foods in which associated bacteria grow poorly, and foods that have been temperature abused are associated with staphylococcal gastroenteritis. Some of the foods that have been more frequently implicated are ham, corned beef, salami, bacon, barbecued meat, salads, baking products containing cream, sauces, and cheeses.

Prevention (Reduction) of the Disease

The means of prevention of outbreaks of staphylococcus food poisoning include

(1) prevention of contamination of the food with the staphylococci,

(2) prevention of the growth of the staphylococci.

(3) killing staphylococci in foods.

Contamination of foods can be reduced by general methods of sanitation, by using ingredients free from the cocci, e.g., pasteurized rather than raw milk, and by keeping employees away from foods when these workers have staphylococcal infections in the form of colds, boils, carbuncles, etc.

Growth of the cocci can be prevented by adequate refrigeration of foods and, in some instances, by adjustment to a more acid pH. Also the addition of a bacteriostatic substance, such as serine or an antibiotic, has been suggested. Some foods may be pasteurized to kill the staphylococci before exposure of the foods to ordinary temperatures, e.g., pasteurization of custard filled puffs and eclairs for 30 min at 190.6 to 218.3 C oven temperature.

Identification Methods

To associate a food implicated in staphylococcal food poisoning, the food or vomit samples are analyzed for the presence of high levels of enterotoxigenic Staph. aureus cells and enterotoxins (see Appendix E). Enumeration technique in one or more selective differential agar media to determine the load of viable cells of Sta. aureus, followed by several biochemical tests, such as hemolysis, coagulase, thermonuclease reactions, or ability of a pure culture to produce enterotoxin, are performed to link the potential cause of the food poisoning outbreaks.

Enterotoxins from the food or vomit samples are extracted and tested, either by biological means or by serological means, to associate them with the outbreak. In the biological method, animals (e.g., cats, monkeys, or dogs) are given the enterotoxin preparation orally or it is injected intraperitoneally or intravenously. Vomiting symptoms by the test animals is a positive indication of the presence of staphylococcal enterotoxin.

In the serological methods, the enterotoxins are purified and examined by one of the several recommended immunological methods. Not only are these tests very sensitive, but they allow the identification of the types of enterotoxins involved in a food poisoning case.