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## THE FOOD POSIONING

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### ABSTRACT :

The Food poisoning is a common disease condition that usually results from eating food contaminated with infectious microorganisms (viral, bacterial, parasitic or fungal) or toxins secreted during the different stages of food processing, production or preservation. Most cases of food poisoning are considered moderate and their symptoms resolve spontaneously within several days without treatment, while severe and chronic cases require hospitalization. Food poisoning can usually be prevented by keeping hands clean and preparing food in proper, hygienic and clean ways. The majority of reported cases of foodborne illness are individual or sporadic. The origin of this sporadic infection often cannot be determined. For example, the majority of foodborne disease outbreaks (58%) in the USA originate in commercial food establishments ( Food net 2004 data). An outbreak is defined as when two or more people are exposed to the same disease after eating food from a common food supply. There is often more than one reason for an outbreak or outbreak, for example, food may be left at room temperature for many hours, allowing the growth of bacteria that accumulate through inadequate cooking of the food, resulting in failure to eliminate or kill Bacteria levels are very dangerous .

**Keywords :** Food poisoning , Accidental poisoning , Deliberate poisoning , Foodborne diseases , Staphylococcal food poisoning (SFP) ,

### INTRODUCTION :

Food poisoning is a set of symptoms resulting from eating food contaminated with bacteria, or toxins produced by these organisms, and food poisoning results from eating food contaminated with various types of viruses, germs, parasites and toxic chemicals such as poisoning caused by eating mushrooms, and it is said that food poisoning may An outbreak if it happened that symptoms of the disease appeared in more than two people, and laboratory studies have shown that the ingested food is the direct cause by planting the bacteria that cause poisoning, and food poisoning caused by bacteria is the main cause in more than 80% of food poisoning cases. Trusted? Foodborne illness, or foodborne disease, colloquially referred to as food poisoning, represents every illness caused by eating contaminated food.

There are two types of food poisoning cases: the case of botulism poisoning and the case of botulism poisoning. Food infections indicate the presence of bacteria or other microbes that cause infection in the body after eating. The possibility of food contamination has increased in our contemporary times due to the accelerated

globalization in food production and trade. Some foodborne disease outbreaks that were once contained within a small community may now occur across larger communities or on global dimensions. This made food and food safety authorities around the world acknowledge that the issue of food safety should not be dealt with at the local level, but also through the establishment of closer relationships and links between those authorities at the global level [4,5].

This is necessary to exchange regulatory information on food safety issues as well as facilitate access to data and information in the event of exposure to food safety emergencies. It is also difficult to estimate the global incidence of foodborne diseases, but it was reported that in 2000 About 2.1 million people died from diarrhoeal diseases, many of these diseases are attributed to the contamination of drinking water and food, in addition to that diarrhoea is a major cause of malnutrition in children and young people.

It has been reported that about 30% of the total population in the industrialized countries themselves suffer from foodborne illness infection annually. The rate of infection with food-borne diseases is estimated at 76 million cases annually, of which about 325,000 cases are treated in hospitals, in addition to about 5,000 deaths [6]. However, developing countries in particular suffer worse from the risks of exposure to foodborne diseases due to the wide spread of diseases, including those caused by parasites.

Here, we note that food-borne diseases cause serious and severe harm to society. In 1994, an outbreak of salmonella infection caused by contaminated ice cream occurred in the United States of America, resulting in the infection of 224,000 individuals. In 1998, an outbreak of hepatitis A occurred in China, which was caused by eating contaminated mollusks, resulting in an infection of 300,000 individuals. As a result, food contamination creates widespread social and economic pressure on communities exposed to outbreaks or disease outbreaks [7]. The medical cost and production losses of diseases caused by pathogens in the United States of America in 1997 were estimated at 35 billion US dollars. The outbreak of cholera in Peru in 1991 led to a loss of 500 million US dollars in losses in fish and fish product exports during that year.

As for body toxicity, it refers to the ingestion and digestion of toxins in foods, including exotoxins, which express protein bio-toxins made by some organisms from plants and animals such as castor seed toxin. The toxin is no longer present or unable to cause infection. Despite that commonly used term, food poisoning, most cases are caused by a variety of pathogenic bacteria, viruses or parasites, all of which contaminate food, rather than chemical or natural toxins. Scientists have limited the main types of bacteria that cause food poisoning to twelve types

1. Clostridium perfringens
2. Staphylococcus aureus
3. Vibrio species : v. cholerae : v. parahaemolyticus
4. Bacillus cereus
5. Salmonella.
6. Clostridium botulinum.
7. Shigella
8. Toxigenic E. coli
9. Campylobacter
10. Yersinia
11. Listeria
12. Aeromonas

Poisoning is the harmful effect that occurs when a toxic substance is swallowed or inhaled or upon contact with the skin, eyes, or mucous membranes, such as those in the mouth or nose. Possible toxicants include over-the-counter and prescription drugs, prohibited drugs, gases, chemicals, vitamins, food, mushrooms, plants, and animal poisons. Some toxins cause no harm, while others can cause severe damage or death. The diagnosis is based on

symptoms, information from the poisoned person and those present, and sometimes from urine and blood tests. Therefore, medicines should always be kept in their original containers, out of reach of children.

Treatment involves supporting the person and preventing more venom from being absorbed and, sometimes, an increased excretion of the venom. More than two million people suffer from some form of poisoning in the United States each year [9]. Prescription or over the - counter and prohibited drugs are a common source of serious poisoning-related poisoning and death (see acetaminophen poisoning; see also aspirin poisoning). Other common toxins include gases (such as carbon monoxide), household products (see caustic poisoning), agricultural products, plants, heavy metals (such as iron and lead), vitamins, animal poison, and foods (particularly certain types of mushrooms and fish). However, ingesting almost any substance in very large quantities can be toxic.

## **ACCIDENTAL POSIONING :**

Accidental poisoning is a leading cause of nonfatal injuries in the home, particularly affecting young children and the elderly. Children's natural curiosity and tendency to explore make them especially vulnerable, while the elderly may face risks due to medication confusion. It's crucial to store medications and hazardous substances securely to prevent accidental ingestion.

Food safety is paramount in preventing foodborne illnesses. Food preparation establishments play a vital role by sourcing meat from approved suppliers and maintaining proper storage temperatures to inhibit bacterial growth. Adhering to sanitation standards and ensuring workers practice good hygiene, such as thorough handwashing after using the restroom, are essential preventive measures. Additionally, protecting food from exposure to insects and heat, using gloves during food handling, and discarding spoiled items daily contribute to food safety. It's important to avoid mixing old and fresh foods, especially if the former have changed in colour , taste, or smell. A strong sense of responsibility towards consumers should guide these practices, prioritizing their well-being over purely financial considerations.

Implementing these measures can significantly reduce the risk of poisoning and foodborne illnesses, ensuring a safer environment for all.

## **DELIBERATE POISONING :**

Poisoning may be a deliberate attempt to kill or commit suicide. Most adults who attempt suicide by poisoning use more than one drug and also drink alcohol. Poisoning can be used to weaken a person (for example, to rape or rob a person). In rare cases, parents with a mental disorder poison their children to make them ill, thereby gaining medical attention (a disorder called Factitious Disorder). Foodborne diseases often result from poor handling, improper preparation and processing, and poor food storage. Noting that good hygiene practices before, during and after food processing reduce the chances of exposure to foodborne disease .

A common awareness trend among the public health is the regular hand washing is one of the most effective defences against the spread of foodborne diseases. This movement to control and control food to ensure that it does not cause exposure to foodborne diseases is known as food safety. This makes the underlying cause of exposure to foodborne diseases a wide variety of toxins affecting the environment. For more information on chemical-borne diseases, see Food contamination. Foodborne diseases can also be caused by pesticides or medicines in foods, in addition to natural toxic substances, including poisonous mushrooms or reef fish.

## **CAUSATIVE AGENTS OF FOOD POISONING :**

### **Bacterial toxic poisoning**

Bacterial toxins are virulence elements that manipulate host cell function and take over the control of vital processes of living organisms to Favor microbial infection.

- Clostridium botulinum
- Clostridium perfringens
- Staphylococcus aureus
- Bacillus cereus

## **Mycotoxin poisoning**

Mycotoxins are metabolites of fungi (Molds), which cause both animal and human disease. Mycotoxins are toxic products of fungi, not mushrooms. The best known mycotoxins are aflatoxins, ergot derivatives, ochratoxin A, fumonisin, and deoxynivalenol.

## **Phytotoxin poisoning :**

Phytotoxins are toxins produced by plants. Phytotoxin poisoning occurs as a result of the intake of non-edible plants, to the consumption of insufficiently prepared plants or to plant toxins resistant to the food preparation process. Phytotoxin includes alkaloids, terpenes, glycosides, tannins, and phenols.

## **Chemical food poisoning :**

Chemical food poisoning occurs owing to a pre-contamination of plants or animals by heavy metals (lead, cadmium, mercury) which are consumed by humans. A wide range of clinical manifestations is seen depending on the chemical substance involved in poisoning. These toxic chemical products may be added directly during the food preparation process as have been seen in some cases .

## **Types of bacterial toxin :**

A bacterial toxin is a macromolecule mainly of protein origin, which can cause toxic damage in a specific organ of the host . Toxins can be divided in endotoxins and exotoxins :

**Endotoxins or lipopolysaccharides [LPS] :** These are the components of the outer membrane of the Gram-negative bacteria; they are considered the most important antigen of the bacteria; they are released into the medium after different processes such as lysis and cell division. This endotoxin is capable of causing endotoxic shock and tissue damage .

LPS are formed by three regions ,

**Lipids A** is a glycolipid formed by a disaccharide (glucosamine) bounds to fatty acids, that are usually capric, lauric, myristic, palmitic, and stearic acids, which are inserted in the outer membrane of the bacterium.

The nucleus a heteropolysaccharide derived from hexoses and heptoses.

The O chain is a repeating unit polymer of 1–8 glycosidic residues; this polymer is highly variable among bacterial species and genus.

In addition to the pyrogenicity of the endotoxin, an important role has been attributed to the adherence mechanism of the bacteria to the host cell; since in previous studies, it has been observed that when LPS is modified or not expressed, the adherence observed is modified or inhibited.

**Exotoxins :** These are the macromolecules of protein origin, which are produced and later released to the medium by the microorganism. Depending on their mechanism of action, exotoxins are divided as follows:

**Toxins type I :** These toxins modify the host's cells without internalizing in the cells; for example, the superantigens produced by Staphylococcus aureus and Streptococcus pyogenes

**Toxins type II :** Within this group there are hemolysins and phospholipases; this group of toxins is characterized by pore formation and/or destroying the membranes of the host cells.

With this virulence factor, the pathogen can invade the host cell

**Toxins type III :** These toxins are known as A/B due to their binary structure. Fraction B has the function of binding to the receptor of the cell and fraction A is the unit that possesses enzymatic activity, which, depending on the toxin and its mechanism of action, will be the damage to the cells .

| Toxins type                 | Definition                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                |
|-----------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Enterotoxin                 | It produces a net secretion in ligated intestinal segments without histological evidence of intestinal lesion or damage to nonerythrocytic cells in in vitro tests. It stimulates the increase in the short circuit current (isc) and the potential difference (pd) in the using chamber without evidence of intestinal damage; this result involves the secretion of (active) electrogenic anions. Additionally, a toxin can impair electrically neutral nacl absorption, which also results in a net Secretion of ions. |
| Cytoskeleton altering toxin | It alters the cellular form and has been frequently shown to be caused by the f-actin rearrangement. The toxin can cause limited cell damage but is not lethal, and it may or may not be associated with the evidence of net secretion in in vivo or in vitro disease models in intestinal Epithelial cells.                                                                                                                                                                                                              |
| Cytotoxin                   | It causes cell or tissue damage, usually ending with cell death. The toxin may or may not be associated with net secretion in in vivo or in vitro disease models in intestinal epithelial cells.                                                                                                                                                                                                                                                                                                                          |
| Neurotoxins                 | It involves the release of one or more neurotransmitters from the enteric nervous system. It alters the activity of smooth muscle in the intestine.                                                                                                                                                                                                                                                                                                                                                                       |

### Foodborne diseases :

In food products, we can find different types of toxins such as, bacterial, fungal (mycotoxins), algae or plant toxins, as well as metals, toxic chemicals (zinc, copper, and pesticides), and physical contaminants that can cause diseases in people who eat them; all of these can cause the well-known —foodborne diseases.

Foodborne diseases can be classified into two groups : **poisoning and infection**.

**Poisoning** is caused by the intake of chemical or biological toxins; or toxins produced by pathogens, the latter can be found in food, even if the bacterium is not there.

**Infection** is caused by the intake of food containing viable pathogens. Furthermore, a toxic infection (toxico infection), formerly known as a toxin-mediated infection, is caused by eating food with bacteria that grow and produce a toxin inside the body

To meet the ideal conditions, microorganisms in food grow and produce toxins. By ingesting contaminated food, toxins are absorbed through the intestinal epithelial lining, and it causes local tissue damage. In some cases, toxins can reach organs such as the kidney or the liver, the central nervous system or the peripheral nervous system, where they can cause some damage

The most common clinical symptoms of foodborne diseases are diarrhoea , vomit, abdominal cramps, headaches, nausea, pain, fever, vomit, diarrhoea with mucus and blood (dysentery), and rectal tenesmus. Some of the microorganisms causing foodborne diseases, either from poisoning, intoxication or toxico infection are described in

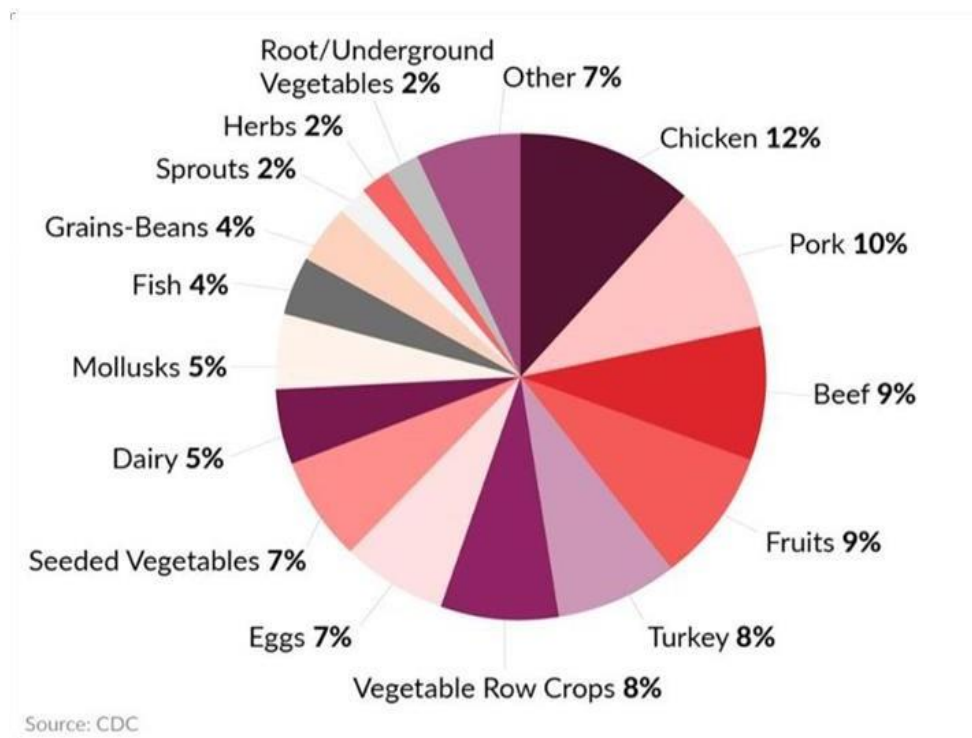


Fig . The following table shows common causes of foodborne illnesses, symptoms and common sources food product involved.

| Bacteria                                                                     | Disease/ medical complication                                                                                                                 | Food products involved                                                                      |
|------------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------|
| Salmonella enterica serovar Typhi and Salmonella enterica serovar Para typhi | Typhoid and paratyphoid fever.                                                                                                                | Undercooked pork, beef and poultry, contaminated eggs, and milk.                            |
| Salmonella spp.                                                              | Salmonellosis (Salmonella Typhimurium, Salmonella Enteritidis).                                                                               | Undercooked poultry, cauliflowers, and tomatoes.                                            |
| Vibrio vulnificus                                                            | Septicaemia in people with underlying diseases or people who are taking immunosuppressive drugs or steroids.                                  | Seafood, usually oysters.                                                                   |
| Mycobacterium Bovis                                                          | Cervical lymphadenopathy, intestinal lesions, chronic cutaneous tuberculosis.                                                                 | Contaminated milk.                                                                          |
| Mycobacterium avium, subspecies paratuberculosis                             | Crohn's disease.                                                                                                                              | Pasteurized milk.                                                                           |
| Listeria monocytogenes                                                       | Meningitis, encephalitis, sepsis in pregnant women, intrauterine or cervical infection that can lead to miscarriage or birth of a dead child. | Raw beef, pork, poultry, vegetables and milk, cheese, ice cream, smoked fish, and raw fish. |

**Pathogens that cause infection :**

| Bacteria              | Disease/medical complication                             | Food product involved                                                                    |
|-----------------------|----------------------------------------------------------|------------------------------------------------------------------------------------------|
| Clostridium botulinum | Paralysis of arms, legs, trunk, and respiratory muscles. | Mixture of oil and non-acid garlic, potatoes cooked at high temperatures, and stews      |
| Bacillus cereus       | Fried rice syndrome.                                     | Rice cooked at high temperatures, sauces, soups, and puddings.                           |
| Staphylococcus aureus | Toxic shock syndrome.                                    | Meat and meat products cooked at high temperatures, poultry, and salads with mayonnaise. |

**Pathogens that cause intoxication :**

| Bacteria                     | Disease/medical complication                                                                                                                          | Food product involved                                                                     |
|------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------|
| Escherichia coli O157:H7     | Haemorrhagic colitis, Haemolytic uremic syndrome in children.                                                                                         | Hamburgers, non -pasteurized milk, contaminated water, spinach, and lettuce.              |
| Shigella spp.                | Haemolytic Uremic Syndrome.                                                                                                                           | Salads, lettuce, raw vegetables, and milk.                                                |
| Aeromonas spp.               | Meningitis, peritonitis, myocarditis, haemolytic uremic syndrome, necrotizing fasciitis in wounds                                                     | Meat (beef, sheep, pork and chicken), vegetables, eggs, fish, seafood, and prepared food. |
| Cronobacter sakazakii        | Permanent neurological or developmental deficits; death                                                                                               | Powdered infant formula.                                                                  |
| Vibrio parahaemolyticus      | Gastroenteritis, septicaemia and wound infection. Severe infections in immunocompromised people                                                       | Raw or undercooked seafood, usually oysters.                                              |
| Clostridium perfringens      | Clostridial necrotizing enteritis.                                                                                                                    | Meat juice, stews, cooked beans, meat cooked at high temperatures                         |
| Campylobacter spp.           | Campylobacteriosis, arthritis, meningitis, Campylobacter jejuni can cause Guillain- Barré Syndrome.                                                   | Cheese made with raw milk and chicken meat.                                               |
| Yersinia enterocolitica      | Yersiniosis, enterocolitis, pseudo appendicitis, mesenteric lymphadenitis, infections in wounds, joints and the urinary tract, and Reiter's syndrome. | Non pasteurized milk, tofu, non chlorinated water, undercooked meat, oysters and fish.    |
| Vibrio cholerae serogroup O1 | Cholera.                                                                                                                                              | Contaminated water and raw seafood                                                        |

**Staphylococcal food poisoning :**

Staphylococcal food poisoning (SFP) occurs when foods contaminated with Staphylococcus aureus bacteria produce toxins that cause illness when ingested. Common sources of contamination include food handlers carrying the bacteria in their noses or on their hands, leading to contamination through direct contact or respiratory droplets. Additionally, S. aureus can be present in food animals, particularly dairy cattle, sheep, and goats affected by subclinical mastitis, potentially contaminating milk .

Foods commonly associated with SFP include meat and meat products, poultry and egg products, milk and dairy products, salads, bakery items (especially cream-filled pastries and cakes), sandwich fillings, and salted products like ham. This is due to *S. aureus*'s ability to grow at relatively low water activity levels .

Preventing SFP involves proper food handling practices :

- **Hand Hygiene** : Food handlers should wash their hands thoroughly before preparing food.
- **Food Storage** : Keep perishable foods refrigerated to inhibit bacterial growth.
- **Avoid Cross-Contamination** : Use separate utensils and surfaces for raw and cooked foods.
- **Health Monitoring**: Individuals with skin infections should refrain from preparing food until healed.

Despite preventive measures, SFP remains common, with its true incidence likely underestimated due to factors like misdiagnosis and unreported minor outbreaks. The disease poses social and economic challenges, leading to lost workdays, increased healthcare costs, and financial losses in the food industry.

### Staphylococcus aureus enterotoxins :

The *S. aureus* enterotoxins (SEs) are potent gastrointestinal exotoxins synthesized by *S. aureus* throughout the logarithmic phase of growth or during the transition from the exponential to the stationary phase. They are active in high nanogram to low microgram quantities, and are resistant to conditions like heat, treatment, low pH that easily destroy the bacteria that produce them, and to proteolytic enzymes, hence retaining their activity in the digestive tract after ingestion.

Table 1. General properties of SEs and SEIs and genomic location of the encoding genes

| Toxin       | Molecular Mass (kDa) | Emetic Activity | Crystal Structure Solved | Gene         | Accessory genetic element                                                                                                                     |
|-------------|----------------------|-----------------|--------------------------|--------------|-----------------------------------------------------------------------------------------------------------------------------------------------|
| SEA         | 27.1                 | yes             | yes                      | <i>sea</i>   | $\Phi$ Sa3ms, $\Phi$ Sa3mw, $\Phi$ 252B, $\Phi$ NM3, $\Phi$ Mu50a                                                                             |
| SEB         | 28.4                 | yes             | yes                      | <i>seb</i>   | pZA10, SaPI3                                                                                                                                  |
| SEC         | 27.5–27.6            | yes             | yes                      | <i>sec</i>   | SaPI <sub>n</sub> 1, SaPI <sub>m</sub> 1, SaPI <sub>m</sub> 2, SaPI <sub>bo</sub> v1                                                          |
| SED         | 26.9                 | yes             | yes                      | <i>sed</i>   | pIB485-like                                                                                                                                   |
| SEE         | 26.4                 | yes             | no                       | <i>see</i>   | $\Phi$ Sa <sup>b</sup>                                                                                                                        |
| SEG         | 27.0                 | yes             | yes                      | <i>seg</i>   | <i>egc</i> 1 (vSa $\beta$ I); <i>egc</i> 2 (vSa $\beta$ III); <i>egc</i> 3; <i>egc</i> 4                                                      |
| SEH         | 25.1                 | yes             | yes                      | <i>seh</i>   | MGE <sub>m</sub> 2/mssa476 <i>seh</i> /Δ <i>seo</i>                                                                                           |
| SEI         | 24.9                 | weak            | yes                      | <i>sei</i>   | <i>egc</i> 1 (vSa $\beta$ I); <i>egc</i> 2 (vSa $\beta$ III) ); <i>egc</i> 3                                                                  |
| SEJ         | 28.5                 | nd              | no                       | <i>selj</i>  | pIB485-like; pF5                                                                                                                              |
| SEK         | 26.0                 | nd              | yes                      | <i>selk</i>  | $\Phi$ Sa3ms, $\Phi$ Sa3mw, SaPI1, SaPI3, SaPI <sub>bo</sub> v1, SaPI5                                                                        |
| SEL         | 26.0                 | no <sup>a</sup> | no                       | <i>sell</i>  | SaPI <sub>n</sub> 1, SaPI <sub>m</sub> 1, SaPI <sub>m</sub> 2, SaPI <sub>bo</sub> v1                                                          |
| SE/M        | 24.8                 | nd              | no                       | <i>selm</i>  | <i>egc</i> 1 (vSa $\beta$ I); <i>egc</i> 2 (vSa $\beta$ III)                                                                                  |
| SE/N        | 26.1                 | nd              | no                       | <i>seln</i>  | <i>egc</i> 1 (vSa $\beta$ I); <i>egc</i> 2 (vSa $\beta$ III); <i>egc</i> 3; <i>egc</i> 4                                                      |
| SE/O        | 26.7                 | nd              | no                       | <i>selo</i>  | <i>egc</i> 1 (vSa $\beta$ I); <i>egc</i> 2 (vSa $\beta$ III); <i>egc</i> 3; <i>egc</i> 4; MGE <sub>m</sub> 2/mssa476 <i>seh</i> /Δ <i>seo</i> |
| SE/P        | 27.0                 | nd <sup>a</sup> | no                       | <i>selp</i>  | $\Phi$ N315, $\Phi$ Mu3A                                                                                                                      |
| SE/Q        | 25.0                 | no              | no                       | <i>selq</i>  | $\Phi$ Sa3ms, $\Phi$ Sa3mw, SaPI1, SaPI3, SaPI5                                                                                               |
| SER         | 27.0                 | yes             | no                       | <i>ser</i>   | pIB485-like; pF5                                                                                                                              |
| SES         | 26.2                 | yes             | no                       | <i>ses</i>   | pF5                                                                                                                                           |
| SET         | 22.6                 | weak            | no                       | <i>set</i>   | pF5                                                                                                                                           |
| SE/U        | 27.1                 | nd              | no                       | <i>selu</i>  | <i>egc</i> 2 (vSa $\beta$ III); <i>egc</i> 3                                                                                                  |
| SE/U2 (SEW) | nd                   | nd              | no                       | <i>selu2</i> | <i>egc</i> 4                                                                                                                                  |
| SE/V        | nd                   | nd              | no                       | <i>selv</i>  | <i>egc</i> 4                                                                                                                                  |

### Structure :

SEs and SEIs constitute a family of structurally related exoproteins that range in size from ~22 to 28 kDa (Table 1). Based on amino acid sequence comparisons, they have been distributed into four or five groups (Table 2), depending on the inclusion or not of SEH within group 1 [21,29,40,49]. The recently described SET is most related to a putative exotoxin from an *S. aureus* isolate involved in bovine mastitis, and to streptococcal pyrogenic toxin type K (SpeK). TSST-1 (Toxin Shock Syndrome Toxin), which is functionally a superantigen with no emetic

activity, is more distant to SEs and SEIs than to SSLs (staphylococcal superantigen-like proteins) [50]. The SSLs, first identified by screening staphylococcal genomes using two conserved amino acid motifs placed in the N-terminal and C-terminal domains of SAgS, are not mitogenic to T cells and do not bind MHC class II, although they display a wide array of activities targeting key elements of the innate and specific immunity, such as neutrophils, complement factor C5, and IgA.

Table.2 : Grouping of SEs and SEIs based on amino acid sequence comparisons. Modified from Larkin et al. [21]. Enterotoxins encoded by the *egc* cluster are shown in bold. SEH (in parenthesis) has been placed within Group 1 or Group 5, depending on the author

| Group     | SEs and SEIs                                                      |
|-----------|-------------------------------------------------------------------|
| Group 1   | SEA, SED, SEE, (SEH), SE/J, <b>SE/N</b> , <b>SE/O</b> , SE/P, SES |
| Group 2   | SEB, SEC, <b>SEG</b> , SER, <b>SE/U</b> , <b>SE/U2</b>            |
| Group 3   | <b>SEI</b> , SE/K, SE/L, <b>SE/M</b> , SE/Q, <b>SE/V</b>          |
| Group 4   | SET                                                               |
| (Group 5) | (SEH)                                                             |

The three-dimensional structures of TSST-1 and several SEs and SEIs have been solved by crystallography (Table 1). The structures are remarkably conserved, although they interact differently with MHC class II molecules, and show different TCR specificity [70]. They are compact ellipsoidal proteins with two unequal domains separated by a shallow groove. The larger C-terminal domain is a  $\beta$ -grasp fold consisting of four- to five-strand  $\beta$ -sheet that packs against a highly conserved  $\alpha$ -helix. The smaller N-terminal domain consists of a mixed  $\beta$ -barrel with Greek-key topology, similar to the OB (oligosaccharide/oligonucleotide binding)-fold [72] also found in many other bacterial toxins (SSLs, streptococcal superantigens, nucleases and toxins of the AB5 family, including cholera and pertussis toxins, and verotoxin) [29,50]. The two domains are stabilized by close packing and by a section of the N-terminus that extends over the top of the C-terminal domain. The N-terminal extension contributes substantially to the TCR-binding site, located in the cleft between the two protein domains, while the MHC class II binding site is in the OB-fold [29,50]. The top of the N-terminal domain usually contains a highly flexible disulfide loop, which has been implicated with emetic activity.

### Mode of action :

*Staphylococcus aureus* produces various enterotoxins (SEs) that can cause vomiting (emesis). Identifying specific amino acids and regions within these toxins responsible for emesis has been challenging, yielding limited and sometimes conflicting results. For example, TSST-1, SEIL, and SEIQ do not induce vomiting, while SEI has weak emetic activity. Notably, these non-emetic toxins lack the disulfide loop found in the N-terminal domain of other SEs. This suggests that while the loop may stabilize a conformation important for emesis, it is not strictly necessary for this activity.

Modifying certain histidine residues in SEA and SEB toxins by carboxymethylation eliminates their ability to cause diarrhoea but preserves their super antigenic properties, which activate T-cells. This indicates that emesis and superantigenicity are distinct functions of these proteins. However, there is often a strong correlation between the two activities; mutations that reduce superantigen activity typically also diminish emetic activity.

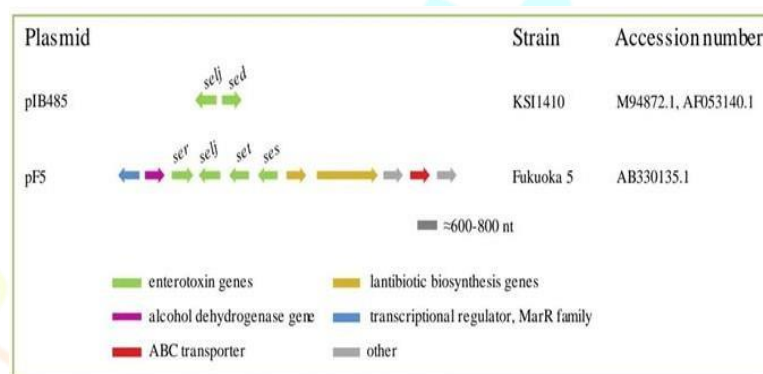
Unlike many bacterial toxins, specific cells and receptors in the digestive system linked to SE-induced vomiting have not been clearly identified. It is proposed that SEs stimulate the vagus nerve in the abdominal area, which then sends signals to the brain's vomiting centre. Supporting this, receptors on vagal afferent neurons are crucial for SEA-induced vomiting. Additionally, capsaicin—a compound from chili peppers that depletes certain sensory nerve fibres—reduces SE effects in mammals.

SEs can penetrate the gut lining, triggering local and systemic immune responses. This leads to the release of inflammatory mediators like histamine, leukotrienes, and substance P, which contribute to vomiting. Blocking

histamine receptors and calcium channels can alleviate this response. Activation of the local immune system by SEs may also cause gastrointestinal damage, with the stomach and upper small intestine being most affected. The diarrhoea sometimes seen with SE intoxication might result from inhibited water and electrolyte absorption in the small intestine.

It has been suggested that the enterotoxin activity of SEs facilitates their transport across cell barriers, allowing them to enter the bloodstream. This enables interaction with antigen-presenting cells and T-cells, leading to super antigenic effects. Thus, when SEs circulate following ingestion or spread from an infection site, they can have widespread effects on the host.

The genes encoding SEs and related toxins are located on various mobile genetic elements, such as plasmids, prophages, and pathogenicity islands. These elements can spread among *S. aureus* strains, influencing their virulence and contributing to the evolution of this pathogen. Plasmids, in particular, are efficient vehicles for transferring resistance and virulence genes through horizontal gene transfer.



## Symptoms :

Symptoms vary depending on what is causing the illness. They may begin within a few hours or a few weeks depending on the cause.

## Common symptoms are:

- Upset stomach.
- Vomiting.
- Diarrhoea.
- Diarrhoea with bloody stools.
- Stomach pain and cramps.
- Fever.
- Headache.

Less often food poisoning affects the nervous and can cause severe disease . Symptoms may include:

- Blurred or double vision
- Headache
- Loss of movement in limbs
- Problems with swallowing
- Weakness
- Changes in sound of the voice

## Treatment of food poisoning :

Treatment of food poisoning is divided into ;

- A. Symptomatic treatment (medications)
- B. Supportive therapy (Oral Rehydration Therapy)
- C. Symptomatic treatment

### A. Anti-diarrhoea :

**Absorbents** (e.g. attapulgite, aluminium hydroxide) : Attapulgite is an oral, non-absorbed medication that is used in the management of diarrhoea. Its mechanism is by adsorbing (binding) large numbers of bacteria and toxins and reducing the diarrhoea. Attapulgite lessens the number of bowel movements, improves the consistency of loose or watery stools, and relieves the cramping which is associated with food poisoning [24].

**Antisecretory agents** (e.g. bismuth subsalicylate) : Bismuth adsorbs extra water in the bowel during diarrhoea. It also adsorbs toxins and forms a protective coating for the intestinal mucosa

**Antiperistaltics** (e.g. loperamide): Loperamide is useful in the treatment of diarrhoea and irritable bowel syndrome because it inhibits intestinal secretion and peristalsis, thereby reducing the intestinal transit time and permitting more fluids and electrolytes to be absorbed; at rest, it improves anal sphincter tone, hence, it has minimal effect on the pain associated with irritable bowel syndrome .

### B. Anti-emetic : promethazine, metoclopramide

Promethazine is a first-generation antihistamine. It is an H1-receptor antagonist with antiemetic and sedative properties.

Metoclopramide is a dopamine antagonist used to treat the symptoms of slow stomach emptying (gastroparesis). It increases the movement of the upper gastrointestinal tract without increasing secretion. It relieves symptoms such as nausea, vomiting, heartburn, a feeling of fullness after meals, and loss of appetite secretion. It relieves symptoms such as nausea, vomiting, heartburn, a feeling of fullness after meals, and loss of appetite.

Antibiotics: (e.g. tetracycline, ciprofloxacin, norfloxacin, co-trimoxazole, doxycycline, rifaximin).

Ciprofloxacin (a fluoroquinolone) is a broad-spectrum antibiotic which is active against both Gram-positive and Gram-negative bacteria. It inhibits bacterial cell division by inhibiting DNA gyrase, and a type II topoisomerase, topoisomerase IV, necessary to separate bacterial DNA.

### C. Supportive therapy :

Oral rehydration therapy (ORT) is a form fluid replacement useful in the prevention and treatment of dehydration, particularly in diarrhoea. It involves drinking water with modest amounts of sugar and salts, specifically sodium and potassium and this is to make up the salt water deficits and to replenish lost nutrients. The risk of death associated with diarrhoea has been estimated to decrease by about 93% with use of oral rehydration solution and recommended home fluids.



## Diagnosis :

A diagnosis is based on a physical exam and a review of things that may be causing vomiting, diarrhoea or other symptoms. Questions from your health care provider will cover:

### Your symptoms :

food or drinks you've had recently ,

- symptoms in people who ate with you
- recent changes in the drug you take

### Recent travel :

Your health care provider will examine you to rule out other causes of illness and check for signs of dehydration.

### Your provider may order tests including:

Stool sample tests to name the bacteria, viruses, parasites or toxins.

Blood tests to name a cause of illness, rule out other conditions or identify complications.

When one person or a family gets food poisoning, it's hard to know what food was contaminated. The time from eating the contaminated food to the time of sickness can be hours or days. During that time, you may have had one or several more meals. This makes it difficult to say what food made you sick.

In a large outbreak, public health officials may be able to find the common food all of the people shared.

## Prevention :

Treatment for food poisoning depends on how severe your symptoms are and what caused the illness. In most cases, drug treatment isn't necessary.

### Treatment may include the following :

Fluid replacement. Fluids and electrolytes maintain the balance of fluids in your body. Electrolytes include minerals such as sodium, potassium and calcium. After vomiting or diarrhoea, it's important to replace fluids to prevent dehydration. Severe dehydration may require going to the hospital. You may need fluids and electrolytes delivered directly into the bloodstream.

### Antibiotics :-

If the illness is caused by bacteria, you may be prescribed an antibiotic. Antibiotics are generally for people with severe disease or with a higher risk of complications.

**Antiparasitic :-**

Drugs that target parasites, called antiparasitic , are usually prescribed for parasitic infections.

**Probiotics :-**

Your care provider may recommend probiotics. These are treatments that replace healthy bacteria in the digestive system.

**Lifestyle and home remedies :**

For most people, symptoms improve without treatment within 48 hours. To help keep yourself more comfortable and prevent dehydration while you recover, try the following :

**Let your stomach settle :**

Eat after your stomach is settled and you are hungry again.

**Replace fluids :**

Replace fluids with water, sports drinks, juice with added water or broths. Children or people at risk for serious illness should drink rehydration fluids (Pedialyte, Enfalyte , others). Talk to your doctor before giving rehydration fluids to infants.

**Ease back into eating :**

Gradually begin to eat bland, low-fat, easy-to-digest foods, such as soda crackers, toast, gelatine, bananas and rice. Stop eating if you feel sick to your stomach again.

Avoid certain foods and substances until you're feeling better. These include dairy products, caffeine, alcohol, nicotine, and fatty or highly seasoned foods.

**Rest :**

Rest to recover from illness and dehydration.

**How are foodborne illness prevented :**

Foodborne illnesses can be prevented by properly storing, cooking, cleaning, and handling foods.

- Raw and cooked perishable foods—foods that can spoil—should be refrigerated or frozen promptly. If perishable foods stand at room temperature for more than 2 hours, they may not be safe to eat. Refrigerators should be set at 40 degrees or lower and freezers should be set at 0 degrees.
- Foods should be cooked long enough and at a high enough temperature to kill the harmful bacteria that cause illnesses . A meat thermometer should be used to ensure foods are cooked to the appropriate internal temperature:
  - 145 degrees for roasts, steaks, and chops of beef, veal, pork, and lamb, followed by 3 minutes of rest time after the meat is removed from the heat source
  - 160 degrees for ground beef, veal, pork, and lamb
  - 165 degrees for poultry
- Cold foods should be kept cold and hot foods should be kept hot.
- Fruits and vegetables should be washed under running water just before eating, cutting, or cooking. A produce brush can be used under running water to clean fruits and vegetables with firm skin.
- Raw meat, poultry, seafood, and their juices should be kept away from other foods.

- People should wash their hands for at least 20 seconds with warm, soapy water before and after handling raw meat, poultry, fish, shellfish, produce, or eggs. People should also wash their hands after using the bathroom, changing diapers, or touching animals.
- Utensils and surfaces should be washed with hot, soapy water before and after they are used to prepare food. Diluted bleach—1 teaspoon of bleach to 1 quart of hot water—can also be used to sanitize utensils and surfaces.

## Complications :

In most healthy adults, complications are uncommon. They can include the following. Dehydration

The most common complication is dehydration. This a severe loss of water and salts and minerals. Both vomiting and diarrhoea can cause dehydration.

Most healthy adults can drink enough fluids to prevent dehydration. Children, older adults, and people with weakened immune systems or other illnesses may not be able to replace the fluids they've lost. They are more likely to become dehydrated.

People who become dehydrated may need to get fluids directly into the bloodstream at the hospital. Severe dehydration can cause organ damage, other severe disease and death if not treated.

### Complications of systemic disease

Some contaminants can cause more widespread disease in the body, also called systemic disease or infection. This is more common in people who are older, have weakened immune systems or other medical conditions. Systemic infections from foodborne bacteria may cause:

- **Blood clots in the kidneys :** E. coli can result in blood clots that block the kidneys' filtering system. This condition, called haemolytic uremic syndrome, results in the sudden failure of the kidneys to filter waste from the blood. Less often, other bacteria or viruses may cause this condition.
- **Bacteria in the bloodstream :** Bacteria in the blood can cause disease in the blood itself or spread disease to other parts of the body.
- **Meningitis :** Meningitis is inflammation that may damage the membranes and fluid surrounding the brain and spinal cord.
- **Sepsis :** Sepsis is an overreaction of the immune system to systemic disease that damages the body's own tissues

### Pregnancy complication

Illness from the listeria bacteria during pregnancy can result in:

- Miscarriage or stillbirth
- Sepsis in the newborn
- Meningitis in the newborn

## Rare complications :

Rare complications include conditions that may develop after food poisoning, including:

- **Arthritis :** Arthritis is swelling, tenderness or pain in joints.
- **Irritable bowel syndrome :** Irritable bowel syndrome in a lifelong condition of the intestines that causes pain, cramping and irregular bowel movements .
- **Guillain-Barre syndrome :** Guillain-Barre syndrome is an immune system attack on nerves that can result in tingling, numbness and loss of muscle control .

- **Breathing difficulties** : Rarely, botulism can damage nerves that control the muscles involved in breathing.

## CONCLUSIONS :

Food poisoning, also known as foodborne illness, is an illness caused by eating contaminated food. Infectious germs, including bacteria, viruses, and parasites, or their toxins, are the most common cause of food poisoning. Infectious germs and their toxins can contaminate food at any stage of processing or production. Contamination can also occur in the home if food is prepared or cooked incorrectly. Symptoms of food poisoning, which can begin within hours of eating contaminated food, often include nausea, vomiting or diarrhoea. Often, food poisoning is mild and goes away without treatment. But some patients need to go to hospital.

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