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A Review on Food-Borne Bacterial Pathogens

[1]Yadav Priyanka Mahendra and [2]Dr. Apurva Pathak

[1]Research Scholar, Department of Microbiology, Sri Satya Sai University of Technology and Medical Sciences, Sehore, M.P., India.
[2]Research guide, Department of Microbiology, Sri Satya Sai University of Technology and Medical Sciences, Sehore, M.P., India.

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ABSTRACT

Every year millions of people are affected and thousands of them die due to infections and intoxication as a result of foodborne outbreaks, which also cause billions of dollars' worth of damage, public health problems, and agricultural product loss. A considerable portion of these outbreaks is related to fresh produce and caused by foodborne pathogens on fresh produce and mycotoxins. Escherichia coli O104:H4 outbreak, occurred in Germany in 2011, has attracted a great attention on foodborne outbreaks caused by contaminated fresh produce, and especially the vulnerability and gaps in the early warning and notification networks in the surveillance systems in all around the world. In the frame of this paper, we reviewed the most common foodborne pathogens on fresh produce, traceback investigations of the outbreaks caused by these pathogens, and lastly international early warning and notification systems, including PulseNet International and Rapid Alert System for Food and Feed, aiming to detect foodborne outbreaks. In the frame of this paper we reviewed the most common foodborne pathogens.

1. Introduction

Foodborne pathogens are responsible for a wide range of illnesses, many of which have serious consequences for human health and the economy. The characteristics of the most common pathogenic bacteria (Bacillus cereus, Campylobacter jejuni, Clostridium botulinum, Clostridium perfringens, Cronobacter sakazakii, Escherichia coli, Listeria monocytogenes, Salmonella spp., Shigella spp., Staphylococcus aureus, Vibrio spp., Vibrio spp., Food safety control schemes relying on the traditional hazard-based approach have been shown to be ineffective, and leading experts and organisations are now recommending a risk-based food safety approach. In this sense, a food safety management framework should be structured to estimate the risks of food intake to human health, as well as to define, select, and implement prevention measures to monitor and reduce these risks. In addition, it is recommended that all people participating in the processing and use of foods participate in appropriate food safety education programmes.

Consumption of food infected by bacteria and/or their metabolites, fungi, pathogens, pesticides, or other agents is a frequent cause of foodborne illness (also known as food poisoning). Despite the fact that the United States' food system is among the best in the world, the federal government reports that 48 million cases of foodborne illness occur each year. This figure equates to one in every six Americans getting ill as a result of tainted food, resulting in 128,000 hospitalizations and 3,000 deaths.

People get sick by eating or drinking food or drinks that have been infected with bacteria, chemicals, or pollutants. Food poisoning signs and severity can be influenced by a number of causes, including a compromised immune system and advanced age. When the FDA becomes aware of an epidemic, the agency's Coordinated Outbreak Response and Evaluation (CORE) Network collaborates with state and local agencies as well as the Centers for Disease Control and Prevention to determine the source and eliminate further outbreaks.

Where required, the FDA partners with food suppliers to encourage voluntary recalls of potentially tainted products; however, the FDA Food Safety Modernization Act gives the agency mandatory recall authority (FSMA).

Bintsis T. (2017), foodborne pathogens are responsible for a wide range of diseases, all of which have significant health and economic consequences. The characteristics of the most common pathogenic bacteria (Bacillus cereus, Campylobacter jejuni, Clostridium botulinum, Clostridium perfringens, Cronobacter sakazakii, Esherichia coli, Listeria monocytogenes, Salmonella spp., Shigella spp., Staphylococccus aureus, Vibrio spp. and Yersinia enterocolitica), viruses (Hepatitis A and Noroviruses) and parasites (Cyclospora cayetanensis, Toxoplasma gondii and Trichinella spiralis), together with some important outbreaks, are reviewed., Food safety control schemes relying on the traditional hazard-based approach have been shown to be ineffective, and leading experts and organisations are now recommending a risk-based food safety approach. In this sense, a food safety management framework should be structured to estimate the risks of food intake to human health, as well as to define, pick, and incorporate prevention measures to monitor and reduce these risks. In addition, it is recommended that all people participating in the processing and use of foods participate in appropriate food safety education programmes.

2. MOST COMMON FOODBORNE PATHOGENS

Bacillus Cereus

B.cereus was originally isolated and described by Frankland and Frankland (1887). Anecdotal evidence of B. *cereus* food poisoning had existed in Europe since the turn of the last century. One of the earliest recorded episodes of B. *cereus* food poisoning was that of 1906 when Lubenau (1906) described an outbreak in a sanatorium m which 300 of 400

inmates and staff became ill with profuse diarrhoea, stomach cramps, and vomiting shortly after eating meatballs in the dinner. A large number of aerobic sporeforming *Bacillus* originally reported by him as *Bacillus peptonificans,* although most probably should be identified as a strain of *B. cereus,* was isolated from the remnants of the dish. Later Seitz (1913), Brekenfeld (1926,1929) and Trab and Wundram (1942) reported that Bacillus-contaminated foodstuffs stored for long periods at improper temperatures were able to cause illness when eaten. During the period 1936-1943,117 of 367 cases investigated by the Stockholm Board of Health were suspected of being caused by aerobic sporeformers (Plazikowski 1947). Confusion was there in the early nomenclatures during reporting of Bacillus-related food poisoning. This reflected the disorder in *Bacillus* taxonomy as well as contributed to the slow recogrution of pathogenicity among members of the genus *Bacillus* other than *B. anthrads.* Full credit goes to Smith *et al* (1952) and Gordon *et al* (1973) in bringing order to *Bacillus* taxonomy, and in providing a foundation of tests and interpretations on which rests our present understanding of this group of micro-organisms. However it was not until Hauge's remarkable experiments in the 1950s (Hauge 1950,1955) that B. *cereus* was established as a cause of food poisoning. He presented the first classic description of B. *cereus* gastroenteritis in his discussion of four Norwegian outbreaks involving 600 persons resulting from vanilla sauce prepared and stored at room temperature for one day before being served. Later Hauge consumed vanilla sauce containing 10^7-10^8 cells m^{-1}, and within 16 h he was suffering from profuse diarrhoea accompanied by cramping abdominal pain.

Cells of B. *cereus* are large (cell width, > 0.9 nm), gram-positive rods, and motile by means of peritrichous flagella. Single central to terminal, ellipsoid or cylindrical endospore without distention of the sporangium is present. B. *cereus* is able to metabolize glucose, fructose and trehalose, but not pentoses and many of the sugar alcohols. A small percentage of strains are urease-positive; the majority actively hydrolyzes starch, casein, and gelatin (Gordon *et al.* 1973; Granum 2007).

Growth and multiplication of vegetative *B. cereus* cells typically occur within 10-50°C, with the optimum between 28° and 30°C. However, psychrophilic variants identified in raw milk samples can grow at temperatures as low as 5 °C. An initial-stage spore germination has been demonstrated within wider limits, i.e., -1 °C, 30 °C and 59 °C being the minimum, optimum and maximum temperatures, respectively (Knaysi 1964). The range of pH permitting growth of *B. cereus* in laboratory media has been reported to be 4.9 to 9.3 when adjusted with mineral acids and alkalis (Fluer and Ezepchuk1970; Kimand Goepfert 1971). Minimum water activity (a_w for growth of *B.cereus* was reported to be 0.95, however in Cantonese style fried rice, the arranged from 0.912 to 0.961 (Kramer and Gilbert 1989).

One of the most important aspects of study is the heat resistance of B. *cereus* spores because spore is a factor of primary concern to the food and pharmaceutical industries (Kramer and Gilbert 1989). Since spore inactivation is the primary concern when processing appertized foods, far higher temperatures are used in the appertization process and in spore D-value calculation (decimal reduction time). The D-value is defined as the time it takes for the remaining population to decrease by one log cycle at a given temperature, and the z-value is defined as the temperature change that causes a 10-fold (1 log) change in D. (Moss and Adams, 1995).

D-values at 85°C, 90°C, 95°C and 100°C in phosphate buffer (pH 7.0) was reported to be 220 min, 71 min, 13 min and 8 min, respectively (Mol 1957). In contrast, D_{121}°C-values in soya bean oil and olive oil were 30 and 17.5 min, respectively, demonstrating that lipid material have a protective effect on the thermal resistance of *B.cereus* spores (Molin and Snygg1967). In low acid foods (>pH4.5),the D_{100}°C-value was found to be 5 min (Ingram 1969). A D^^-value of 2.7-3.1 min for B. *cereus* in skim milk was reported by Mikolajcik (1970). z-values ranging from 6.7 to 8.3°C were obtained in aqueous spore suspension by Gilbert *et al.* (1974). Straiiw (serotype H.l) producing emetic syndrome food poisoning are more heat resistant (D_{95}°C:22.4-36.2*min)* compared to routine isolates of *B.cereus*(random serotype) from samples of raw rice 1.5-6.0 min) (Parry and Gilbert 1980). D_{100}°C and D_{92}°C-values for *six* isolates in rice broth were 4.2-6.5 min and 16-36 min, respectively (Chung and Sun 1986). Rajkowski and Mikolajcik (1987) reported *D_{100}°C-values* of *B. cereus* ranging from 0.6 to 27.0 min in demineralized water. The heat resistance at 90 °C for spores of the 32 *B. cereus* strains isolated from fresh vegetables and refrigerated minimally processed foods ranged from 1.4 to 21.2 min. D^^^^and were 3.2-23.3,0.7-5.2 and 0.4-1.1 min, respectively, in strains isolated from Spanish raw rice (Sarrias *et al* 2002). Strains unable to hydrolyze starch were the most heat-resistant, with D_{90}°C-values higher than 10.8 min (Valero *et al.* 2002). Several studies have provided evidence of non-linear spore survivor curves associated with certain strains. Aging of spores affects the D-value (Collado *et d.* 2003). Banerjee and Sarkar (2004a) reported the *D_{100}°C-values* of *B. cereus* isolates from spices ranging from 3.5 to 5.9 min in glucose-supplemented brain-heart infusion broth.

Other factors that have been shown to exert a total inhibitory effect on the growth of *B.cereus* include 2 g sorbic acid rice filling of Karelian pastry (Raevuori 1976), 500µg benzoic acid ml^{-1} (pH 6.3) (Lueck1980) and 6-10mg garlic aqueous extract g^{-1} (Banerjee and Sarkar 2003). Application of nisin at the levels of 5mgl^{-1} has been shown to act as an effective preservative giving significant increase in shelf-life and providing protection against the growth of psychotropic *B.cereus* (Delves- Broughton *et al.* 1992). Addition of nisin to a batter of crumpets at levels of 3.75-6.26µg g^{-1} effectively prevented the growth to levels capable of causing food poisoning (Jenson *et al.* 1994).

The antibiotic susceptibility was tested for 66 isolates of *B.cereus* from rice in Taiwan against 12 different antibiotics. The isolates were 100% susceptible to chloramphenicol (30 µg $disc^{-1}$), erythromycin (15 µg $disc^{-1}$) and streptomycin (10 µg $disc^{-1}$), and 92.4% were sensitive to gentamicin (10 µg $disc^{-1}$). However, they were 100% resistant to penicillin G (10 units $disc^{-1}$) and polymyxin B (300 units $disc^{-1}$), 99% resistant to ampicillin (10 µg $disc^{-1}$) and carbenicillin (100 µg $disc^{-1}$) and 88% resistant to cephalothin (30 µg $disc^{-1}$) (Chung and Sun 1986). Shah *et al.* (19%) isolated *B.cereus* from about 300 samples of a variety of foods in which *B.cereus* was found in 20% spices. The antibiogram pattern of B. *cereus* was obtained with 50 isolates against nine antibiotics. Ail the isolates were resistant to ampicillin (10 µg $disc^{-1}$). A high resistance was

found against trimethoprim (5 µg disc^{-1}) (9 2 %), colistin (10 µg disc^{-1}) (86%) and rifampicin (5 µg disc^{-1} (92%). All the isolate were sensitive to chloramphenicol (30 µg disc^{-1}) and ciprofloxacin (5 µg disc^{-1}), and 88% sensitivity was seen against streptomycin (10 µg disc^{-1}) and vancomycin (30 µg disc^{-1}).

B. cereus produces several phospholipases, e.g. phospholipase C and egg yolk turbidity factor with preferences for different phospholipids, including phosphatidylcholine (~ 23 kD), phosphatidylinositol (~ 29-35 kD), and a sphingomyelinase (~ 29 kD) (Drobniewski 1993).

B. cereus causes two different types of food poisoning: (i) the diarrhoeal type, first recognized after a hospital outbreak associated with vanilla sauce in Oslo, Norway, in 1948 (Hauge 1955), and (ii) the emetic type, described about 20 years later after several outbreaks associated with fried rice in London (Mortimer and McCain 1974). The emetic toxin, which causes emesis (vomiting), is generated (preformed) by developing cells in food (Kramer and Gilbert 1989), while the diarrhoeal type of food poisoning is induced by a complex enterotoxin produced during B. cereus vegetative development in the small intestine (Kramer and Gilbert 1989). (Granum 1994). The food used with both cases of foot-borne disease has normally been heat-treated, and the food poisoning is caused by remaining spores. Cerulide is the name given to an emetic toxin that has a ring structure made up of three repeats of four amino acids or oxy acids and has a molecular weight of 1.2 kD. Cerulide is heat tolerant (90 minutes at 121°C), pH resistant, and proteolysis resistant, but it is not antigenic. At least three related enterotoxins, namely haemolysin (Hbl), non-haemolytic enterotoxin (Nhe), and CytK, induce diarrhoea. Two of the enterotoxins, one haemolytic and the other n-haemolytic, are multicomponent, while the third (CytK) is a single antigen (Granum 2007). Proteinaceous ingredients, fruits, sauces, and puddings are often associated with the so-called 'diarrhoeal syndrome.' Before the onset of stomach pain, profuse watery diarrhoea, rectal tenesmus, and mild nausea that rarely ends in vomiting, the condition has an incubation time of 8-16 hours (av. 10-12 hours). Symptoms typically go away in 12 to 24 hours. The 'emetic syndrome', on the other hand, is almost exclusively associated with farinaceous foods, especially cooked rice, and is characterised by a rapid onset (1–5 hours) of nausea, vomiting, and malaise, which may be accompanied by diarrhoea lasting 6–24 hours (Kramer and Gilbert 1989).

B. cereus is not a competitive microbe, but it grows well after being cooked and cooled (to 48 °C). In the absence of competitive flora, heat treatment allows spore germination, and B. cereus grows well, with a generation period as short as 12 minutes under ideal conditions (Borge et al. 2001). The infective dosage of B. cereus that is needed to cause illness varies. It was discovered that a B. cereus count of 4.5-9.0logcfu in foods caused enteritis. Furthermore, one of the epidemiological conditions for implicating B. cereus in food poisoning outbreaks is the presence of B. cereus (>104cfu g-1) in food (Concon 1988). A population of > 105 B.cereus cells g-1 is expected for a food poisoning outbreak, according to Johnson (1984).

Holding foods at a temperature where spores do not germinate and cells do not expand is recommended for preventing B. cereus gastroenteritis, as are good sanitary precautions during handling to avoid post-preparation infection, and uniform reheating of a suspected food to above 75 °C before serving (Ray 2001)

Clostridium Petfringens

The first description of C. *perfringens,* formerly known as C. *welchii,* was given by Welch and Nuttal (1892). However, it was not until 1940 that Knox and MacDonald in England confirmed C. *perfringens* as a cause of food poisoning. In addition to enteritis, C.*perfringens* is responsible for necrotizing tissue infections. Historically, the organism is best known for its association with gas gangrene (Adams and Moss 1995).

C. *perfringens* is a typical gram-positive, rod shaped (1 µm x 3-9 µm), anaerobic, sporeforming (oval in shape, subterminal in position) bacterium that is encapsulated and non-motile. Though catalase- negative, it survives and occasionally grows in the presence of oxygen (Labbe 1989). Growth of C. *perfringens* occurs at a temperature of 2-50 °C, although it is very slow below 20 °C. The most important characteristic of C. *perfringens* relative to food safety is the organism's ability to grow optimally at elevated temperatures ranging between 43 and 45 °C. However, Willardsen *et al.* (1978) found that one strain (NCTC 8238) has a shorter generation time of 7.1 min at 41 °C than at 45 °C. Minimum pH for growth is 5.0, whereas 6.0-7.5 is found to be optimum. Minimum for growth is 0.95-0.97. Tompkin *et al.* (1974) found an inhibitory effect of sorbic acid on C. *perfringens.* Control of C *perfringens* was achieved in Italian sausage by incorporating 5 ng nisin (Caserio *et al.* 1979). Growth of most strains of C. *perfringens* is prevented by sodium chloride at a level of 70-80 g although some inhibition occurs at a level of 50-60 g kgr' (Roberts and Derrick 1978).

Food poisoning due to C. *perfringens* is usually self-limiting. Non-febrile illness characterized by nausea, abdominal pain, diarrhoea and, less commonly, vomiting usually occurs 8-24 h after the ingestion of food. The minimum required ingested dose of C. *perfringens* has been variously estimated at 10M0« cfu g-\ However, the median count of the pathogen in foods implicated in outbreaks in UK was 7 X10®g"^. In otherwise healthy individuals, medical treatment is not usually required and recovery is complete within 1-2 d, although occasional fatalities occur in the very old or debilitated persons (Adams and Moss 1995; CDC1985; Shandera *et al.* 1983).

C. *perfringens* is classified into five types, designated A through E, based on the production an expression of four (a, p, e and i) 'typing' toxins (McClane and Rood 2001). Type A strains are involved in foodborne intoxication. The enterotoxin associated with the food-borne disease is a heat- labile protein. During sporulation in the intestine, the cells generate and release this intracellular protein. Some studies claim that, in addition to the intestine, sporulation and enterotoxin formation at certain levels can occur in certain foods (Garvam 1987; Labbe 1988). In the most common cause of food poisoning, the enterotoxin has been found to be the main virulence factor. Stark and Duncan (1971) were the first to prove that the enterotoxin was responsible for all clinically relevant properties. Human volunteer experiments (Skelkvale and Uemura 1977) backed up the hypothesis, and gene deletion studies proved conclusively that the results are attributable solely to enterotoxin development (Brynestad and Granum 2002).

In the United States, perfringens is a frequent source of food poisoning outbreaks (CDC1985). In the 1960s and 1970s, there was a man named C. Perfringens are found in more than

7% of all food-borne infections, accounting for more than 10% of all cases. In the 1980s, the rate of outbreaks fell to about 3% of overall outbreaks, causing about 5% of all incidents. The majority of the outbreaks occurred in cafeterias, hotels, classrooms, and banquet halls (Bean and Griffin 1990). Between 1980 and 1990, outbreaks in England and Wales ranged from 46 to 69 per year, with an average of 8 percent to 1624 cases; in Scotland, outbreaks ranged from 5 to 11 per year, with an average of 75 to 364 cases (Adams and Moss 1995).

The creation of C. Temperature, pH, and a. perfringens in food are affected by a number of environmental factors. The ability of C. contributes in part to the heat-resistance property of spores. By allowing this bacterium to live in undercooked foods, perfringens can cause food poisoning. Food poisoning extract spores are normally much more heat tolerant than C. difficile spores. perfringens isolates obtained from other sources; food poisoning isolate spores' excessive heat tolerance can lead to food-borne virulence. It's also worth noting that foods that haven't been fully cooked may not just fail to destroy C. perfringens spores in foods, but it also encourages the growth of C. by inducing spore germination in S. perfringens class A food poisoning (McClane 2007). According to Roberts (1968), heat-resistant strains' spores had D90°C values of 15-145 minutes and z-values of 9-16°C, while heat-sensitive strains' spores had D90°C values of just 3-5 minutes and z-values of 6-8 °C. Furthermore, heat-resistant strains' spores needed heat activation at 78-80 °C, while heat-sensitive strains expanded without heat activation in up to 50% of cases (Crowther and Baird-Parker 1984). The D-values of C.perfringens spores display a significant inter-strain variation, ranging from 0.31 to > 38 minutes. In water as the heating medium, the D-values of NCTC 8798 spores at 95 °C, 105 °C, and 120 °C were found to be 52.7 min, 2.5 min, and 0.01 min, respectively. The D-values for strain NCTC 10240 were 15.5 minutes and 0.2 minutes at 90 and 100 degrees Celsius, respectively, while the values for ATCC 3624 were 27.5 minutes and 0.22 minutes, respectively (Labbe 1989). Heat resistance to spores in phosphate buffer is lower than in water, but it is higher in cooked meat than in water (Sutton 1966). Apart from genetic differences between strains, sporulating medium and heating medium, as well as contamination of vegetative cells and sporangia, all contribute to the heterogeneity in D-values (Labbe 1989). C. elegans' antibiotic resistance Meat-derived perfringens strains are identical to clinical isolates, indicating that meat is not a typical host of resistant or multi-resistant strains. Porcine isolates from farms where antibiotics are widely used, on the other hand, are often immune to several antibiotics, one of which is transferable (Rood et al. 1978). C. has used penicillin G as the first line of therapy for gas gangrene. perfringens is a kind of perfringens. In most cases of human food poisoning, antibiotics are not used. Antibiotic-associated diarrhoea caused by C. difficile has been reported recently. There are perfringens.

The lowest a_w supporting growth of C. *perfringens* is 0.93 when other growth conditions are near-optimal. Although C. *perfringens* is an anaerobe, it does not require an extremely reduced environment to grow. Provided the environmental E_h is suitably low for initiating growth, C. *perfringens* can produce reducing molecules such as ferredoxin to modify the of its environment and create favourable growth conditions. In fact, the of many common foods such as raw meats and gravies is often adequate to support the growth of C. *perfringens* which is pH sensitive, with optimal growth being at 6-7. It grows slowly, if at all, at pH values of < 5 and > 8.3 (Labbe 1989).

C. *perfringens* is widely distributed throughout the natural environment, including soil, food dust and the intestinal tract of humans and domestic animals (Labbe 1989). However, it is now understood that < 5% of global C. *perfringens* isolates carry the enterotoxin gene *(cpe)* necessary for causing C. *perfringens* type A food poisoning (McClane 2007).

In virtually all outbreaks the principal cause is failure to refrigerate properly previously cooked foods, especially when perspired in large portions. Rapid and uniform cooling of foods is therefore imperative. Gravies, broths and large pieces of meat should be cooled to < 10°C within 2-3 h. Cooked, chilled foods should be related to minimal internal temperature of 75°C immediately before serving to destroy vegetative cells. Cooked meat should be kept above 60 °C or below 10°C. As most people harbour C. *perfringens* in their intestinal tract, preventing carriers from handling food is rather impossible. Similarly, the organism is present in a wide variety of foods. So, education of the food handlers remains a critical aspect of C. *perfringens-related* food poisoning control (Labbe 1989).

Staphylococcus Aureus

Ogston (1881) was the first to identify staphylococci as a pyogenic infection in humans. Food poisoning caused by Staphylococcus aureus is one of the most common causes of gastroenteritis worldwide. It is caused by eating food infected with Staphylococcus and ingesting one or more preformed staphylococcal enterotoxins. Members of the genus Staphylococcus, mostly S. aureus, are the etymological agents. aureus is a type of bacteria. Intoxication is a form of food poisoning that does not require inflammation and growth of bacteria in the host. Staphylococcal toxin, the first true enterotoxin to be identified, is not completely eliminated even after 30 minutes at 100°C (Seo and Bohach 2007).

S. Aureus cells are gram-positive spheres with a diameter of 0.5-1.5 m. Cell division in more than one plane produces irregular clumps that resemble grape bunches. They will ferment glucose and are catalase-positive, oxidase-negative, facultative anaerobic, and catalase-negative. Most strains develop coagulase, thermonuclease, and hemolysin after fermenting mannitol. The cells are destroyed in 12 minutes at 66°C and 15 seconds at 72°C. The ideal temperature for growth is between 7 and 48 °C, with accelerated growth occurring between 20 and 37 °C. At pH 6.0-7.0, growth is optimum, with minimum and maximum limits of 4.0 and 9.8-10.0, respectively. It grows rapidly in media containing 50-70 g kg-1 sodium chloride, and some strains can expand in up to 200 g 100 kg-1 sodium chloride (Adams and Moss 1995). According to Lahellec et al. (1981), the development of S. 10 g sorbate F suppressed aureus in a brain heart infusion (pH 5.0), but the organism expanded in the presence of 50 g sorbate P at pH 7.0. Inhibition of S. cerevisiae was reported by Tompkin et al. (1974). sorbic acid in fried uncured sausage kills E. coli. Nisin is toxic to the bacteria (Thomas et al. 2000). It shrinks to aw 0.83, with a generation time of 300 minutes (Adams and Moss 1995).

S. aureus is wide spread, but occurs most frequently on the skin of higher primates. In humans, it is particularly

associated with the nasal tract where it is found in 20-50% of healthy individuals. It may be isolated from faeces and a variety of other environmental sites, including dirt, sea and fresh water, plant surfaces, pollen, and the atmosphere (Adams and Moss 1995).

Between 1983 and 1987, staphylococci were responsible for 7.8% (47) of the 600 bacterial food poisoning outbreaks reported in the United States. For the same time frame, the same statistic for England and Wales was 1.9 percent (54) out of a total of 2815 outbreaks. In the United Kingdom, cases of staphylococcal food poisoning peaked in the 1950s at 150 per year, but have since dropped to 10-20 per year (Adams and Moss 1995).

Toxins that are produced in foods have a brief incubation time. S. A, B, C, C, C, C, D, and E are the names of the seven protein exotoxins produced by S. aureus. They have a molecular mass of 27.1, 28.366, 27.496, 27.531, 27.438, 26.360, and 26.425 kD. Toxins are single-chain polypeptides with a single disulfide loop at the centre of the molecule. They are immune to gut proteases and heat stable, inactivated only by sustained boiling due to their compact form. Food poisoning outbreaks are most often linked to toxin types A and D, either alone or in combination. In the United Kingdom, type A is accounted for 52% of outbreaks, type D for 6%, and type E for 2%. Types A and D account for 19% of the total, while types C and D account for 9%. Individual susceptibility varies, although it's been estimated that in outbreaks, just 1 ng of pure toxin is required to cause symptoms (Adan\s and Moss 1995). But for clinical isolates like certain community-acquired methicillin-resistant S. aureus, there are no other methicillin-resistant S. aureus strains. Most staphylococci are susceptible to p-lactams, tetracyclines, macrolides, lincosamides, novobiocin, and chloramphenicol, but immune to polymyxin and polyene. aureus (MRSA) strains subjected to antimicrobial treatment, most staphylococci are sensitive to p-lactams, tetracyclines, macrolides, lincosamides (Stefani and Varaldo 2003).

Humans are the primary source for staphylococci that cause disease in humans. The anterior nares are the most common site of colonisation in humans, while S. Some places where aureus can be found include the skin and the perineum. S. aethiopicus (A. aethiopicus) is aureus can spread between humans and from humans to food through direct contact, indirect contact via skin fragments, or respiratory tract droplet nuclei (Seo and Bohach 2007).

The majority of staphylococcal food poisonings nowadays can be traced back to humans who contaminate food during cooking. S. aureus may also be spread by food handlers who are carriers. Contaminated food processing machines, such as meat grinders, scissors, storage utensils, chopping boards, and saw blades, may also inject aureus into food. The following factors were found to be the most often associated with food poisoning in a study of 700 food-borne disease outbreaks: I insufficient refrigeration; (ii) food storage well in advance; (iii) poor personal hygiene, such as not washing hands and equipment properly; (iv) inadequate cooking or heating of food; and (v) repeated use of warming plates when preparing meals (Bryan 1976).

The aim would be to reduce the initial load of S. aureus to reduce the occurrence of staphylococcal food poisoning. proper selection of raw materials and ingredients, sanitation of food conditions, and proper personal hygiene among food handlers will also help to reduce the presence of aureus in food. Heat treatment of the materials should be used wherever possible to ensure that live cells are killed. Contamination after preparation and temperature violence can be avoided (Ray 2001).

Enterobacteriaceae

ENTEROBACTERIACEAE is a family of bacteria. The family Enterobacteriaceae gets its name from the Latin word enterobacterium, which means "intestinal bacteria." Escherichia coli is the family's type species (Brenner 1984). The Enterobacteriaceae family comprises a diverse group of biochemically and genetically linked bacteria with a wide variety of ecology, pathogenic ability, and host range. The species are gram-negative, facultative anaerobic straight rods with a diameter of 0.3-1.5 urn that ferment glucose to produce acid and, in certain cases, steam. They lack oxidase and, with the exception of Erwinia chrysanthemi, all have a different antigen. With a few exceptions, such as Shigella dysenteriae, all members of the Enterobacteriaceae family are catalase-positive. They are not spore-fonning, acid-fast, or halophilic, but they do tolerate bile salts (Brenner 1984).

The Enterobacteriaceae family is found all over the world. They can be present in soil, water, fruits, herbs, nuts, flowering plants and trees, as well as in the intestines of humans and animals (Breimer 1984). They're linked to a wide range of human diseases, including more than 70% of urinary tract infections and almost 50% of septicemia cases. Salmonella and Shigella were thought to be the only gastrointestinal food or waterborne bacteria before the 1940s. Enteropathogenic E. coli serotypes were discovered in the late 1940s. coli is discovered to be gastrointestinal parasites, inducing diarrhoea and vomiting in babies and then adults.

Traditionally, the community (known as coliforms) has been used as a faecal emission measure. These species can ferment lactose in the presence of bile at 37 degrees Celsius. This classification contains not only the majority of Escherichia coli strains, but also organisms like Citrobacter and Enterobacter that are not exclusively faecal. The faecal coliforms are a more restricted category of species that can expand and produce gas at 44-45 degrees Celsius in suitable selective media (ICMSF1978). One criticism of using coliforms and faecal coliforms is that when lactose-negative species predominate, their absence can give the impression of protection. Salmonella and Shigella are among the lactose-negative species, as are enteropathogenic strains of £. E. coli, such as 0 1 2 4. As a result, tests for the entire Enterobacteriaceae family are becoming more common. The Enterobacteriaceae family contains many more genera with non-faecal origins than the coliforms, such as plant-associated Erwinia and Serratia members. As a result, Enterobacteriaceae counts are used more broadly as a hygienic quality measure rather than a faecal pollution indicator, and therefore tell more about overall microbiological quality than potential health risks faced by the substance (Adams and Moss 1995).

Some important food-borne pathogens of this group, namely *E. coli, Salmonella* and *Shigella* are reviewed here.

Escherichia Coli

Even though E. While E. coli was first isolated from children's faeces and identified by Theodor Escherich in 1885,

it wasn't until the early 1940s that it was recognised as a cause of gastroenteritis in babies. E. coli's widespread appearance in faeces, easy culturability, inherently nonpathogenic nature, and water survival characteristics contributed to its acceptance. coli as a faecal infection measure and the prevalence of enteric pathogens like Salmonella typhi in water This practise has been extended to foods, where greater caution is expected when interpreting the importance of potential outcomes (Adams and Moss 1995).

Until 1982, diarrhoea-causing strains were divided into three categories: E. coli enteropathogenic Enteroinvasive E. coli (EPEC), enteroinvasive E. E. coli (EIEC) and enterotoxigenic E. coli (EIEC). However, E coli serotype 0157:H7, an enterohaemorrhagic type (EHEC), has been identified as a cause of many outbreaks since 1982. Enteroadherent E. is the fifth kind. E. coli has been linked to a number of outbreaks based on epidemiological evidence. Its function in diarrhoea is being researched (Adams and Moss 1995).

E. coli cells coli are small, non-spiral curved rods that are gram-negative, catalase-positive, oxidase-negative, fermentative, and catalase-positive. E. coli is a normal mesophile, growing at temperatures ranging from 7 to 50°C with an optimum around 37°C, though some ETEC strains have been recorded to grow at temperatures as low as 4°C. It has a low heat resistance (D-value at 60°C on the order of 0.1 min) and can be kept frozen or refrigerated for long periods of time. For development, a pH of near-neutral is ideal. Under otherwise ideal conditions, growth at pH 4.4 is feasible (Adams and Moss 1995).

Growth must be at least 0.95. On E. coli, 7.5 g sorbic acid (pH 5) had an impact. Doell confirmed the presence of E. coli (1962). Lueck cited minimum inhibitory concentrations of 50-100g sorbic acid ml-1 (pH 5.2-5.6) and 50-1200g benzoic acid ml-1 (pH 5.2-S.6) as minimum inhibitory concentrations (1980).

E. coli symptoms Mild to moderate diarrhoea will accompany E. coli gastroenteritis; in the most severe cases, vomiting, prostration, and shock can occur. Not all has symptoms, but those who do shed the microbes in their faeces after they heal (Doyle and Padhye 1989). EPEC strains often cause child diarrhoea, which is associated with a high mortality rate in many tropical and developed countries. ETEC strains cause diarrhoea in travellers. Adults must consume 106-109 viable cells ml-1 for signs to appear within 24-72 hours (Ray 2001).

Twenty E. coli isolates from humans and cattle were studied. Eighty percent of E. coli strains were immune to at least one antibiotic. Streptomycin resistance was 70%, sulfonamide resistance was 65%, tetracycline resistance was 50%, ampicllin resistance was 25%, sulfonamide/trimethoprim resistance was 20%, chloramphenicol resistance was 10%, cephalothin resistance was 8%, and gentamicin resistance was 5%. (Farina et al. 1996). In certain E. coli strains, antibiotic tolerance to tetracycline, chloramphenicol, streptomycin, and sulfonamide is at an 80 percent stage. Teuber identified coli strains from mastitis infections in cows (1999).

The most effective factor in preventing pathogenic E. coli gastroenteritis in humans. coli is working to improve sanitation of water sources, as well as sewage treatment and disposal. Personal hygiene should be maintained, and food should be reheated before intake (Ray 2001).

Anas A et al.(2016), described Camel milk has the capacity to inhibit the development of a wide variety of foodborne pathogens in addition to its nutritious and medicinal properties, but there is no research about how these pathogens behave in products like yoghurt made from camel milk. The aim of this research was to see how Listeria monocytogenes and Escherichia coli O157:H7 behaved during the manufacturing and storing of camel yoghurt. Camel milk inoculated with Listeria monocytogenes and Escherichia coli O157:H7 was fermented for 5 hours at 43°C using freeze-dried lactic acid bacteria (LAB) starter cultures (Streptococcus thermophilus and Lactobacillus bulgaricus) and preserved for 14 days at 4 or 10°C. Camel milk inoculated with L. monocytogenes and E. coli O157:H7 was also prepared without the starter culture. In the presence of LAB, the numbers of L. monocytogenes and E. coli O157:H7 increased by 0.3 and 1.6 log cfu/mL, respectively, during fermentation, while in the absence of LAB, they increased by 0.3 and 2.7 log cfu/mL. In camel milk without LAB, the amount of L. monocytogenes increased 0.8 to 1.2 log cfu/mL after 14 days of storage at 4 or 10°C, but in the presence of LAB, the number of L. monocytogenes decreased 1.2 to 1.7 log cfu/mL after 14 days. Furthermore, E. coli O157:H7 numbers in camel milk were decreased by 3.4 to 3.5 log cfu/mL in the absence of LAB, but E. coli O157:H7 was not found by 7 days in camel yoghurt made with LAB and processed at any temperature (6.3 log cfu/mL reduction). Despite the presence of elevated levels of natural antimicrobials in camel milk, L. monocytogenes was able to withstand these compounds in camel yoghurt kept at refrigerator temperatures. As a result, proper precautions should be taken when making yoghurt from camel milk to reduce the risk of postprocess infection by this and other foodborne pathogens.

As per Yukyung Choi et. al(2017), the serotyping and genotyping properties of Escherichia coli strains isolated from kimchi and various raw vegetables used in kimchi preparation were investigated in this research. A predictive microbiological model was also used to assess the kinetic activity of E. coli strains in kimchi during fermentation. The study found that enterohemorrhagic E. coli (O6:H34) was isolated from napa cabbage (3.3 percent; 1/30), and eight normal colonies isolated from kimchi (15 percent; 6/40) were enteropathogenic E. coli (H8, H8, H12, H34, H30, O20:H39, H39, and H12). The strains' genetic similarities did not reveal any similar genetic correlations. On the other hand, using a predictive Baranyi model (primary model) and a polynomial equation (secondary model), followed by validation by calculating root mean square error (RMSE), the kinetic behaviour of E. coli strains in kimchi during fermentation revealed that pathogenic E. coli cell counts increased (with RMSE of 0.280 in growth curve) in the early stage of fermentation and decreased (wi As a result, this discovery revealed that pathogenic E. coli isolated from kimchi and associated vegetables proliferated at the start of fermentation and then decreased. As a result, the findings of this study show that eating enough fermented kimchi will help avoid foodborne illness caused by pathogenic E. coli.

Accrding to Yukyung Choi et. al.(2018), the survival of *Escherichia coli* and *Salmonella* strains during diced white radish kimchi fermentation was studied. Kimchi batches inoculated with the pathogens were fermented at 4, 15, and 25°C for 42 to 384 h. Cell counts of *E.*

coli and *Salmonella* were enumerated on *E. coli*–coliform count plates and xylose lysine deoxycholate agar, respectively. Baranyi (primary model) and polynomial (secondary model) models, validated by root mean square error, were used to describe the kinetic behavior of the pathogens. In the primary model, both the death phase shoulder (*E. coli*: 208.18 to 8.25 h, 4 to 25°C; *Salmonella*: 79.91 to 0.97 h, 4 to 25°C) and bacterial cell counts (log CFU per gram per hour) decreased with increasing temperature ($P < 0.05$) (death rate: *E. coli*: −0.02 to −0.09, 4 to 25°C; *Salmonella*: −0.01 to −0.10, 4 to 25°C), the results being equally significant in the secondary model. The root mean square error (0.480 to 0.485) showed that the model performance was good. The fermentation temperature and time are the critical factors that control pathogenic *E. coli* and *Salmonella* in kimchi.

Salmonella

The majority of salmonellae are considered human pathogens, though their characteristics and duration of illness vary. Since typhoid fever is the most serious salmonella infection, it was the first to be accurately defined. Eberth was the first to discover the typhoid bacillus (1880). The paratyphoid bacilli, which cause paratyphoid fever, were first isolated by Achard and Bensaud (1896), and Schottmiiller reported that they were culturally and serologically distinct from the typhoid bacilli (1901). Lignires named the genus Salmonella after D.E. Salmon, who died in 1900. Salmon, an American veterinary pathologist who first isolated Salmonella cholerae-suis (now known as Salmonella enterica serovar Choleraesuis) from hogs suffering from hog cholera (Salmon and Smith 1885). The species' taxonomic nomenclature differs significantly from that of other genera. The genus Salmonella currently has only one species, S. typhimurium, based on DNA-DNA hybridization. There are seven subspecies of enterica. To explain the biochemical and serological diversity within the Salmonella community, several classification schemes are commonly used (Chikami et al. 1985). The system began with 100 serotypes in 1941, and the number has since increased to about 2000. The most recent suggestion to add more taxonomic rigour is to use the non-italicized serovar name after the species name, resulting in S. S. typhimurium is the new name for typhimurium. enterica subsp. enterica subsp. enterica subsp. enter Typhimurium enterica serovar (Adams and Moss 1995).

Sahnonellae are gram-negative, nonsporulating, facultative anaerobic rods that are catalase-positive, oxidase-negative, and motile with peritrichous flagella (typically 0.5 m 1-3 m). When they emerge in glucose-containing media, they produce gas (D'Aoust 1989). They ferment dulcitol but not lactose, use citrate as a carbon source, produce hydrogen sulphide, decarboxylate lysine and ornithine, but do not produce indole, and are urease negative.

Salmonella spp. are bacteria that cause food poisoning. D'Aoust and Maurer (2007) described resilient microorganisms as those that can quickly adapt to severe environmental conditions. Temperatures ranging from 5 to 47 °C are optimal for development, with 35-37 °C being the optimum. Salmonellae are heat-sensitive bacteria that are easily killed by pasteurisation (Adams and Moss 1995). The ideal pH for development is between 6.5 and 7.5, with growth possible at pH values varying from 4.5 to 9.5 and gradual death at higher pH values (Bryan et al.1979). Salmonella development is commonly inhibited in the presence of 30-40g sodium chloride1-1. A higher temperature encouraged initiation of growth in a medium with reduced salinity; high sodium chloride concentrations prolonged the latency time and slowed growth. D'Aoust (1989) found that foods with a value of 0.93 do not help the development of salmonellae. A analysis of 23 Salmonella strains developed at 10-30 °C in the presence of 20-80 g sodium chloride 1-1 found similar findings (Alford and Palumbo 1 percent 9). Doell confirmed that 7.5 g sorbic acid (pH 5.0) inactivated Salmonella (1962). S was inactivated by 3 g sorbic acid P (pH 5.0), according to Park and Marih (1972). typhimurium in vitamin broth at 37°C for 12 hours.

Salmonella has long been known for its ability to harbour antimicrobial resistance (Besser et al. 1997; Breuil et al. 2000; CDC1997; Cohen and Tauxe 1986). Bajaj et al. (2003) found very high resistance to penicillin (.9%), vancomycin (83.3%), erythromycin (81.8%), and rifampicin (80.3%) in 66 poultry egg isolates, moderate resistance to trimethoprim (42.4%), chloramphenicol (28.7%), and streptomycin (24.2%), and low resistance to gentamycin (9.0 percent). Another research observed high tolerance to sulphonamides (75.8%) and nitrofurantoin in 91 Salmonella enteritidis isolates from broiler carcasses, fruit, human, and poultry-related samples from the south of Brazil (52.8 percent). Tetracycline (15.4 percent), streptomycin (7.7%), nalidixic acid (7.7%), gentamicin (5.5 percent), norfloxacin (3.3 percent), trimethoprim (3.3 percent), cafalotin (2.2 percent), ampicillin (1.1 percent), and chloramphenicol (1.1 percent) also had lower amounts of tolerance (1.1 percent). In an analysis of 502 Salmonella isolates from food samples conducted by the US Food and Drug Administration (FDA), 247 (49.2%) were immune to one or more antimicrobials, with 170 (68.8%) resistant to one antimicrobial agent, 33 (13.4%) to two, 25 (10.1%) to three, 7 (2.8%) to four, 8 (3.2%) to five, and 2 (0.8%) each to six and seven antimicrobials (Kiessling et al. 2002). Some Salmonella isolates tested positive for tetracycline, gentamicin, sulphamethoxazole, streptomycin, ampicillin, chloramphenicol, and karwmycin in two turkey processing plants in the United States (Olah et al. 2004). Other studies have published similar results (Manie et al. 1998; Threlfall et al. 1997). According to Mayrhofer et al. (2004), Salmonella has the greatest tolerance to nalidixic acid (42 percent), led by tetracycline (33 percent), streptomycin (27 percent), ampidllin, chloramphenicol (17 percent), and ciprofloxacin (17 percent) (9.6 percent). 25 (48.0%) of 52 Salmonella isolates from raw chilled market poultry meats were immune to one antibiotic, 5 (9.6%) were resistant to two, 4 (7.7%) were resistant to three, 6 (11.5%) were resistant to four antibiotics, and 5 (9.6%) were resistant to five antibiotics. Two of the isolates (3.8 percent) were immune to up to nine of the antibiotics tested. Fifty-one isolates (98%) were novobiocin resistant, 18 (34.6%) were streptomycin resistant, and 14 each (26.9%) were tetracycline and oxytetracycline resistant (Oain and Chen 2006). Another research in the United States found that 23 (11%) of 208 Salmonella isolates recovered from imported foods were immune to at least one antimicrobial, and 7 (3.4%) were resistant to three or more antimicrobials. Tetracycline resistance was the most common (9%), followed by sulphamethoxazole (5%), streptomycin (4%), nalidixic acid (3%), and trimethoprim/sulphamethoxazole (3%) resistance (2

percent). Cotrimoxazole resistance was found in all of the Salmonella isolates from betel leaves, and 97 percent of the isolates were resistant to chloramphenicol, imipenem, ciprofloxacin, ceftriaxone, and neomycin. Multidrug resistance (against 5-18 antibiotics) was widespread, with luOidixic acid (65.8%), cephalothin (68.4%), cefoperazone (57.9%), sulphamethizole (52.6%), furazolldone (65.8%), kanamydn (68.4%), doxycycline (50.0%), and cefotaxime (44.7%) were the most common (Singh et al. 2006). An person must eat approximately K-IO* cells to contract food-borne salmonellosis. However, certain virulent strains can induce disease even though only a few cells are consumed. Symptoms occur 8-42 hours after ingestion of the pathogen. Abdominal cramps, diarrhoea, nausea, vomiting, chills, fever, and prostration are the most common symptoms. It is particularly dangerous to the sick, infants, and the elderly (O'Aoust 1989).

Salmonellae has been one of the most common sources of food poisoning around the world. In 1889, the annual prevalence of salmonellosis in Europe was about 50 per 100,000 people. Salmonellosis is on the rise in the United States; between 1969 and 1976, the total number of food-borne infections was about 37 a year, while between 1983 and 1987, it was over 68. A significant number of Salmonella outbreaks have been linked to animal-based foods such as beef, chicken, turkey, pork, poultry, milk, and items derived from them. Salmonellae have also been found in a variety of plant foods (due to the use of waste as fertiliser or the washing of goods in contaminated water), as well as sea food, fin fish, and crustaceans (Bean and Griffin 1990; Hatha and Lakshmanaperumalsamy 1997). Salmonella has been linked to a number of food-borne illness outbreaks, with poultry meat being a particularly common source of this pathogen (Bryan and Doyle 1995; Cloak et al. 1999).

Salmonella infections are more common in newborns, babies, the elderly, and people with compromised immune systems than in healthy adults (ty Aoust 1989). Recent literature shows that a human infectious dosage may be as low as 1-10 cells (D'Aoust et al. 1985; Kapperud et al. 1990). Nausea, vomiting, diarrhoea, fever, and headache are the most common acute symptoms. Since birds, mosquitoes, and tainted food handlers can all contaminate foods directly or indirectly, there are several possible food vehicles for Salmonella.Meat, milk, poultry, and eggs are the main carriers of Salmonella; they may be undercooked, causing the bacteria to thrive, or they may cross-contaminate other foods that are eaten raw.

Controlling Salmonella contamination requires proper cooking of foods (minimum to pasteurisation temperature and duration, such as 71.7 for 15 seconds or equivalent) and timely cooling. Cross-contamination of foods should be avoided using cutting boards, tools, utensils, and paws. Strong personal hygiene and proper sanitation in the food system will help to minimise the occurrence. Refrigerated meals should be reheated thoroughly before serving (D'Aoust 1989). The discovery that a single SalmonellaceU can be contagious highlights the need for more stringent food quality assurance programmes (D'Aoust et al. 1985; Adams and Moss 1995).

Shigella

Shiga discovered the genus Shigella as the source of bacillary dysentery (1898). The genus comprises four species, each of which is serologically classified based on its O-antigens. S.flexneri (group B), S. dysenteriae (group A), S.flexneri (group C), S.flexneri (group D), S.flexneri (group E S. boydii (group C) and S. boydii the sonnei (group D). Their only hosts are humans and a few primates. The species are spread either directly by faecal-oral paths or indirectly by food and water polluted by faeces. S. In tropical areas, dysenteriae has caused extreme bacillary dysentery epidemics, but it is now uncommon in Europe and the United States, where S. The term sonnei is more often used. S. The disease caused by S. sonnei is the mildest, whereas that caused by S. S.flexneri and S.boydii are of moderate magnitude (Adams and Moss 1995).

Shigellae belong to the Enterobacteriaceae family. They are Gram-negative rods that are non-motile, non-sporeforming, catalase-positive (except Shiga's bacillus, S. dysenteriae serotype 1), oxidase-negative, and facultative anaerobes. They contain acid, but not gas, as they consume glucose. The strains can grow at temperatures ranging from 7 to 46 degrees Celsius, with a maximum temperature of 37 degrees Celsius (Adams and Moss 1995). Under various physical and chemical strains, the cells will live for days. Refrigeration, melting, 50g sodium chloride1-1, and pH4.5 are among them. Pasteurization destroys them. Shigella can live for up to 120 days in water. Many food additives, however, such as mayonnaise, vinegar, salt, and sodium benzoate, may act as inhibitors. When processed at the growth temperature level, the strains will multiply in a variety of foods (Smith 1987).

Shigella is more difficult to isolate from foods than from other sources. The physiological status of shigellae in food is a contributing factor in the pathogen's complete recovery. In suspected food samples, Shigella can be present in low numbers or in a compromised physiological condition. In these circumstances, special enrichment procedures are needed for Shigella identification to be effective (Andrews 1989). On a global scale, food-borne shigellosis is a neglected field of research (Lampel and Maurelli 2007).

Abdominal pain, diarrhoea frequently combined with vomit, mucus, and pus, fever, chills, and headache are signs caused by both the invasiveness of epitiielial mucosa and the enterotoxin. Kids are more vulnerable to the condition than adults in general (Smith 1987). The infective dosage is extremely low, ranging from 10-1000 cells per human. Symptoms appear within 12 hours to 7 days of consuming infected food. The symptoms of a moderate infection last around 5 to 6 days, but in extreme cases, the symptoms can last up to 3 weeks (Ray 2001).

Antibiotic treatment is said to shorten the duration of infection and the duration of the shigellae carrier condition. Antibiotics have, however, culminated in the selection of resistant strains and their susceptibility determinants. Physical hygiene and wellness education for food handlers are the strongest prevention steps for food-borne illness. Any foodborne and waterborne outbreaks of shigellosis may be avoided with proper water treatment (chlorination) and sanitary waste disposal (Doyle et al. 1985).

According Falguni Debnath et. al.(2018), in November 2016, a household in Pakapol Village, South 24 Parganas District, West Bengal, India, experienced a foodborne acute gastroenteritis outbreak caused by Shigella sonnei infection after consuming foods at a housewarming session. The epidemiological and microbiological observations of this outbreak are presented here. On November 23, 2016, thirty-

four people attended the party and shared lunch. From the time food was consumed until the onset of acute gastroenteritis, the median incubation period was 18.5 hours (interquartile range, 16.5–22 hours). Overall, 73 percent (25/34) of the attacks warranted hospitalisation, with 76 percent (19/25) requiring hospitalisation. With a 100% recovery rate, all age ranges were affected. Tomato salad (risk ratio, 4.14; 95 percent confidence interval, 1.21–14.13) was found to be substantially correlated with sickness. S. sonnei was found in 8 of the 12 stool specimens examined (67 percent; 8/12). Nalidixic acid, norfloxacin, ciprofloxacin, ofloxacin, and erythromycin were totally resistant, while tetracycline, doxycycline, streptomycin, and trimethoprim/sulfamethoxazole were partly resistant. The latest epidemic strains of S. sonnei is clonally linked to the locally circulating strains in Kolkata, according to a pulsed-field gel electrophoresis study.

Occurrence Of Pathogeiiic Bacteria In Food

Pathogenic bacteria in food Despite its undeniable nutritional benefit, food has a long history of being linked to disease transmission. Food hygiene regulations can be found in a variety of early texts, including the Old Testament, Confucius' books, Hinduism, and Islam. Those early authors had only a hazy understanding of the real causes of food-borne illness, and many of their recommendations had only a minor impact on the disease's occurrence (Adams and Moss 1995). 'Food-borne illness is perhaps the most common health issue in the contemporary world and an important source of decreased economic growth,' considering our improved understanding. The WHO defines food-borne disease as "an infectious or radioactive disease caused by, or believed to be caused by, the ingestion of food and water."

Food-borne gastrointestinal disorders can be classified into three groups: (a) those caused by the ingestion of viable pathogenic microorganisms or their preformed toxins in food; (b) those caused by the ingestion of pathogenic algae, parasites, and their preformed toxic by food; and (c) those caused by factors other than viable pathogens or their toxins, such as natural toxic chemicals (Adams and Moss 1995).

Food-borne bacterial infections are on the rise due to rapidly changing urban lifestyles. The number of women working outside to support their families is on the rise. As a result, dining outdoors has become something of a must for metropolis residents. Many people choose to eat outdoors because it is more convenient to get RTE food at work or close to home (Kakar and Udipi 2002). The many steps involved in the processing and delivery of these foods leave plenty of space for pathogenic and spoilage microorganisms to infect them. Bacillus species are the most widely found pathogens since the bulk of these foods are cereal or pulse-based and require limited heat treatments. In a test of eight consumer idli samples, none were found to be tainted with B. cereus, while other Bacillus species were present (Varadaraj et al.1992). B. gathered 25 samples of commonly cooked pulses from local markets and various household populations. cereus was used in 32% of the samples (Shah et al. 1996). In an analysis involving 10 samples of soya bean wadi and 20 samples of pulses collected from various retail shops and food establishments in and around Bareilly, Uttar Pradesh, B. Cereus was found in 20% of all samples, regardless of type (Meena et a l 2000). Earlier studies of neutral-to-alkaline legume-fermented foods, such as tempe, showed the existence of high amounts of potentially harmful microorganisms such as B.cereus, S.aureus, and S.aureus. Enterobacteriaceae and Staph aureus (Samson et al. 1987). The presence of B. in dawadawa is important. Antai and Ibrahim both noticed cereus (1986). Five of the fifteen kinema consumer samples analysed contained > l0-4 cfu of B. cereus, as well as two samples containing more than 105 cfu of E. coli. E. coli was discovered (Nout et a l 1998). The presence of B was stated by Han et al (2001). cereus, as well as a low number of C. perfringens in an analysis of 23 sufu samples.

While there is little literature on the prevalence and eventual epidemic of food-borne bacterial pathogens in conventional legume-based fermented foods, there is plenty of evidence on other foods that food-borne diseases of microbial origin have become the top food protection issue among consumers and regulatory agencies in recent years. This pattern can be seen in both developed and emerging countries (Garvani 1987).

Te Giffel et al (1996) recorded B. in a food survey conducted in the Netherlands. cereus was found in 48 percent of the 200 food samples sampled, with levels of contamination ranging from 102 to 106 cfu g-1 (or ml-1). B. Cereus was the cause of 33% of all food poisoning cases (excluding virus) in Norway from 1988 to 1993, 47% in Iceland from 1985 to 1992, 22% in Finland in 1992, 8.5 percent in the Netherlands in 1991, and 5% in Denmark from 1990 to 1992. (Granum and Lund 1997; Schmidt 1995). Other nations, such as England and Wales (0.7%), Japan (0.8%), the United States (1.3%), and Canada (2.2%), have previously recorded even lower figures (Kramer and Gilbert 1989). Between 1983 and 1987, there were a total of 479 food-borne disease outbreaks a year in the United States, affecting 18,336 people. However, just 38 percent of the outbreaks affecting 10,908 people were confirmed on average. Bacterial infections were responsible for about 66% of the outbreaks, accounting for 92 percent of the cases and 96 percent of the casualties (Bean et a l 1990). Many pathogenic bacteria are found in raw materials of animal and plant origin, many are present in food cultures, many develop very effectively in various foods, and many are not destroyed by the conditions used to process various foods (Ray 2001).

Despite the fact that multiple pathogenic bacterial species and viruses have been linked to food-borne disease outbreaks, these have happened more often than others. From 1983 to 1987, the number of outbreaks and fatalities associated with the two most common pathogens associated with food-borne intoxication was higher for C. S. botulinum has a much larger average number of cases than B. botulinum. aureus is a type of bacteria. Food-borne infections caused by Salmonella spp. caused the most outbreaks, cases, and casualties of the enteric pathogens that cause infection. The number of cases and outbreaks of toxicoinfections is higher in C. B.cerms is more resistant to perfringens than B.cerms. The most common of the three forms of food-borne illnesses. Salmonella was linked to the most infections, affecting the most number of people and resulting in the greatest number of deaths (Bean et a l 1990).

Anand and Singh (1987) tested 102 samples of baby foods for enterotoxigenic staphylococci. They found a broad range of occurrence, with an average log count of 3.5 g-*. In 57.8

percent of the samples, staphylococci were found. Thirty percent of the staphylococci isolates developed enterotoxin. In a study of 606 rice products, Chyan et al (1989) discovered coli types and E.coli in 31% and 9%, respectively. The isolation rates of B.cereus in daily instant cereals and cereal-mix were 26 percent and 38 percent, respectively, in the study by Fang et al (1997). Coliforms were included in 2.7 percent of daily instant cereal products and 7.4 percent of cereal-mix products, respectively. In their analysis of the microbiological content of fried RTE foods sold in Mumbai, Kakar and Udipi (1998) discovered that 11 percent of samosa and batatawada samples and 45 percent of patra samples had staphylococcal counts of more than 3 logs. S. coagulase-positive In three samosa samples and two patra samples, aureus was found. In none of the samples, Sulnionella, Shigella, or Yersinia enterocolitica were found.

Fang et al. (1999) examined the microbiological content of 320 vegetarian food samples purchased from Chinese markets. The prevalence of E. coli is on the rise. coli and coliforms were present in 28% and 33% of these vegetarian food items, respectively, while S.aureus and B.aureus were found in 18% and 3% of the samples, respectively. cereus, and so forth. The highest identification rate was found in soya bean samples (74.5 percent).

Mukhopadhyay et al. (2002) investigated the microbiological consistency of sliced papaya (Carica papaya) sold on the street in Kolkata. The overall aerobic plate count varied from 3.3 to 6.52 log cfu g-1 in their analysis, with an average of 5.96 log cfu g-1. Coliforms were used in 70% of the samples, with an average load of 13.5. In 48 percent of the samples that tested positive for coliforms, E. coli was found. In one sample each, Salmonella and Vibrio cholerae were found, as well as low levels of coagulase-positive S. In 17% of the samples, the bacteria Staph aureus was included.

Altug and Bayrak (2003) studied the microbiological quality of caviar from Russia and Iran. The relevant figures that were detected among 68 samples are as follows; standard plate count varied from 10^3-10^6 cfu g^{-1} coliforms varied from 10^1-10^4 cfu g^{-1} yeasts varied from $<10^1$ to $6x10^5$ cfu g^{-1}, *E.coli* varied from $<10^1$-10^2 cfu g^{-1} Only in one sample S. *aureus* was detected as $5x10^2$ cfu g^{-1}.

Hanashiro *et al.* (2005) studied the occurrence of some foodborne bacterial pathogens in 40 popular street foods available in a restricted area in Sao Paulo of Brazil and concluded that 35% of the samples were unsuitable for consumption according to the microbiological criteria. Mankee *et al.* (2005) studied the microbiological quality of 196 samples of 'bara', 'channa', condiments/spices and RTE 'doubles' sold in Trinidad. *E. coli* was detected in 0%, 7.1%, 49% and 34.2%, respectively, of the samples. Staphylococci were isolated from 53.1%, 36.2%, 65.8% and 62.8%, respectively, of the samples, and *B. cereus* was recovered from 11.2%, 43.4%, 51% and 44.9%, respectively, of the samples.

3. BEHAVIOUR OF FOOD-BORNE BACTERIAL PATHOGENS DURING FERMENTATION

Bacteria get acid-stressed at low pH levels, but they will tolerate this stress if they've already been subjected to a less intense acid shock and have developed an acid tolerance response. Pickling and fermentation are two traditional methods of food preservation that use acidification (Browne and Dowds 2002). B. cereus grew to more than 107 cfu m-1 in the mageu foundation, which was not fermented by lactic acid bacteria, and it was suggested that B. cereus could grow to potentially harmful levels in maize porridge on its own but was reduced to smaller numbers within 24 hours. After 24 hours of fermentation, B. cereus inhibition was reduced from 106 cfu ml-1 to 102 cfu ml-1 in the mageu base inoculated with both the starter culture and B. cereus (Byaruhanga et al. 1999; Svanberg et al. 1992).

Guven and Benlikaya (2005) discovered that after 72 hours of fermentation, the B. cereus count fell to 1 log cfu ml-1 in the boza base inoculated with both the starter culture of lactic acid bacteria and B. cereus and in the control boza base to which no starter culture was applied. They found a pH of 4.9 after 12 hours of fermentation in all batches of boza fermentation in which the amount of B. cereus decreased dramatically, and this result is very well associated with Goepfert and Kim (1975) and Hancioglu et al (1999). B. cereus developed rapidly to 8-9 cfu g-1 tempe produced from non-acidified soya beans, according to Nout et al. (1987), and E. coli 0157:H7 was not inhibited and lasted a 32-hour fermentation at pH 3.7, according to Hancioglu et al. (1999). S. typhimunum and S. aureus, on the other hand, were inhibited after 12 hours of fermentation at pH 4.5. Wong and Chen studied the fate of B. (xreus added into the lactic acid fermentation at various levels (1988). B. cereus did not affect the development of lactic acid bacteria, although when cells were inserted at the start of lactic acid bacteria growth, B. cereus rose steadily to around 106 cfu ml-1 from an initial amount of 104 cfu ml-1. However, it inactivated slowly after 24 hours and quickly after 72 hours of lactic acid bacterial development.

S. aureus was able to thrive in freshly begun soya bean soaks (final pH 4.7) during tempe production, but it died in soaks with accelerated souring (final pH 4.0). The highest levels of enterotoxin were found after 48 hours of fermentation (Nout et al. 1988). S. aureus increased 1.5-log in Italian style dry sausage formed with 105 cfu g-1 Lactobacillus sp. and 2.5-log in fermentation with 104-106 lactic acid bacteria g-1 under the same fermentation conditions, according to Metaxopoulos et al. (1981). Turantas (1991) discovered that beginning on the sixth day, the count of S. aureus in sucuks with starter culture fell below the measurable level.

Antony et al. investigated the inhibitory activity of finger millet (Eleusine coracana) flour fermented for different time periods on S. typhimurium and E. coli after extended incubation (0-48 h) (1998). The fate of E. coli 0157:H7 in fermented dry sausage was investigated by Glass et al. (1992). They used 4.8xl04 E.coli 0157:H7 g-1 to inoculate a commercial sausage batter, fermented top H4.8, and dried until the moisture-protein ratio was 1.9:1. After that, the sausage chubs were vacuum sealed and held at 4°C for two months. The species survived but did not thrive during fermentation, dr5dng, or subsequent preservation at 4°C, and by the end of storage, they had decreased by around 2-log cfu g-1. Tsegaye et al. tested the survival of E. coli 0157; H7 during the fermentation of Datta and Awaze, typical lactic acid fermented Ethiopian condiments (2004). The research strains were not recovered after 24 hours of fermentation when fermenting Datta or Awaze were initially inoculated at a low inoculum level (103 cfu g-1). However, at a higher initial inoculum level (103 cfug-1), the test strain counts

in Datta at day 7 were around 1.5-log units lower than the initial inoculum level. After 7 days of fermenting Awaze, all of the research strains were finally gone. At this time, the pH of fermenting green and red Datta was reduced from 5.2 to 4.4, and the pH of Awaze was reduced from 4.9 to 3.8. Tsegaye and Ashenafi (2005) investigated the path of E. coli 0157:H7 during the preparation and storing of standard Ethiopian dairy products Ergo and Ayib. They discovered that when milk was inoculated with both lactic acid bacteria and E. coli 0157:H7 and fermented for 72 hours, the pathogen increased for the first 24 hours before the pH reached 3.5, but then reduced until the pH reached 3.9. The pathogen grew slowly in the absence of lactic add bacteria.

Tetteh et al. (2004) investigated how fortification with cowpea affected the longevity and development of acid-adapted and unadapted Shigella flexneri in a conventional fermented Ghanaian weaning meal. S.flexneri remained viable but did not rise in porridge made from fermented com dough with a pH of 4.07 and held at 10 or 30°C for 24 hours.

4. CONCLUSION

The large number of studies gathered in this paper reaffirms the significance of foodborne pathogens as a global problem and offers strong, up-to-date scientific advice. The importance of rapid identification of foodborne pathogens using responsive culture-independent methods and emerging technology such as WGS or other biomarker assay research has been highlighted. Foodborne pathogens' development and diffusion, as well as their food vehicles and how infection can occur in the food supply chain, are both factors to consider during outbreak investigations.

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