Review article

Bacteriology  Study of Shigella Species, and the Effect Some Ecological and Chemical factors.

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ABSTRACT

Shigella organisms are gram-negative rods that belongs to the family Enterobacteriace. This genus consists of four species, S. dysenteriae, S. flexneri, S. boydii and S. sonnei. These are often referred to as subgroups A, B, C and D respectively. genus was divided into four groups (designated A, B, C, and D) on the basis of their capacity to ferment sugars and on their O-antigen serotypes. These groups are: Group A: S. dysenteriae comprised 15 serotypes. Group B: S. flexneri comprised 6 serotypes. Group C: S. boydii comprised 18 serotypes. Group D: S. sonnei comprised a single serotype. The four species can be differentiated from each other by the fermentation of sugars, sugar alcohols, production of indole, and the synthesis of ornithine decarboxylase or arginine dehydrase. Shigella can enter into the host colonic cell, the vacuole that forms from the fusion of the cell membrane around the phagocytized Shigella and multiplication. Intra- and intercellular spread of the bacterium. The death of the host cell and ulceration of the mucosa. Resultant inflammatory response. Diarrheal is the leading worldwide cause of death among children. According to World Health Organization estimates that 5 million deaths occur annually from diarrheal diseases, and Shigella are responsible for 10% of these mortalities. The Shiga have three types of toxic activities, Neurotoxic activity; Enterotoxic and Cytotoxic activity. Shiga toxin clearly causes fluid secretion when placed in the small bowel lumen of rabbits and results in inflammatory enteritis in this model. Dyssentery involves bloody diarrhea, but the passage of bloody mucoid stools is accompanied by severe abdominal and rectal pain, cramps and fever. While abdominal pain and diarrhea are experienced by nearly all patients with shigellosis, fever occurs in about one-third and gross blood occurs in about 40% of cases.

INTRODUCTION

Shigella spp. are gram-negative, short (1-3µm) non-motile, non-pigmented, non-encapsulated, non-spore forming, facultatively anaerobic rods. An important biochemical characteristic that distinguishes these bacteria from other enterics is the ability to ferment lactose, unlike other members in the Enterobacteriaceae group, Shigella are non-lactose fermenting on MacConkey agar or deoxycholate citrate agar after a period of incubation of 24 hrs [1]. However, some strains of S. sonnei may ferment lactose slowly or utilize citric acid as a sole carbon source. They do not produce H2S, except for S. flexneri serotype 6 and S. boydii serotypes 13 and 14, and do not produce gas from glucose. Shigella spp. are inhibited by potassium cyanide and do not synthesize lysine decarboxylase or hydroxylase arginine, they are oxidase-negative, ornithine decarboxylase-negative (S. sonnei is positive) [2]. Actually, there is a continuum of biotypes and bioserotypes between typical Shigella and Escherichia spp., and the so-called enteroinvasive E. coli (EIEC) strains are responsible for a disease similar to shigellosis. E. coli and the four groups of shigellae are so closely related that they constitute a single spp., and the decision to maintain Shigella and E.coli as separate entities was made only in the interest of epidemiology and clinical medicine [3]. Mentioned that enteroinvasive E. coli (EIEC) has pathogenic and biochemical properties similar to those of Shigella spp. this similarity poses a problem in distinguishing these pathogens. For example, EIEC is non-motile and unable to ferment lactose. Some serotypes of EIEC also have O antigens identical to those of Shigella. Shigella grows less profusely on artificial media than coliform bacteria and other members of the family Enterobacteriaceae. They are less active in their utilization of carbohydrates than E. coli and do not form visible gas from carbohydrates (except for certain biotypes of S. flexneri 6). Urease, phenylalanine deaminase and hydrogen sulphide are not produced. The Voges-proskauer test is negative, and methyl red reaction positive. Sodium malonate is not utilized, gelatin is not liquefied and growth does not occur in Simmon's....
citrate agar or in potassium cyanide medium [4]. Shigella spp. have a minimum temperature for growth of 6.1°C, and a maximum of 47°C. Shigella does not need to grow in food to cause illness, as the very low infective dose means that the presence of the organism in food is sufficient to cause infection. Shigella spp. survive at frozen and chill temperature, although the time of survival depends on the type of food environment as well as temperature. At room temperature S. sonnei rapidly increased in numbers in shredded cabbage stored in vacuum/modified-atmosphere (30% N2, 70% CO2) packaging, and Shigella numbers remained static when stored under similar conditions at chilled temperature [5]. The reported pH range allowing growth of Shigella spp. is 4.6-9.3, although actual values will depend on acid type. Shigella spp. are gradually inactivated at pH values lower than 4.0, but the organism can survive for some time in acid conditions. Shigella spp. can grow at water activities down to 0.96 (maximum salt conc. 5.2% NaCl). The organism dies out slowly at low water activities. Even at high NaCl concentration (10%) some strains can survive for 4 days [5].

2.1. Distribution in nature and source of isolation

Humans and higher primates are the main reservoir for Shigella spp. Individuals recovering from infection can continue to shed the pathogen for weeks after the symptoms have ceased and the organism can survive for sometime in feces. Shigella is not normally found in a free living in the environment and only present in the food as a result of fecal contamination [5]. Sewage-contaminated water can be a source of Shigella contamination. Although it is commonly thought that water; rather than food, is the more important vehicle for Shigella, public health data suggests that the reverse may be the case. Food can become contaminated from soiled hands, contaminated water, the use of night soil as manure and flies that have been feeding on human feces. Foods that require a lot of handling during preparation and are not subsequently cooked, such as salads and sandwiches, are at particular risk of contamination from infected food handlers [5]. Public health data suggests that foodborne Shigella infections are more common than waterborne infections. Shigella have been isolated from various foods including potato, tuna and other salads, milk, cheese, butter, chicken, fish and seafood and from water [4]. Shigella are transmitted by contact via hands soiled with feces or food and by flies. They can be cultured from fingers several hours after experimental inoculation. Following inadequate drinking-water treatment or the seepage of sewage through the earth, water can become a vehicle for the transmission of Shigella [4].

2.2. Epidemiology:

Diarrheal diseases are the leading worldwide cause of death among children. The World Health Organization estimates that 5 million deaths occur annually from diarrheal diseases [8], and Shigella are responsible for 10% of these mortalities. Epidemic dysentery, usually prolonged and large, caused by S. dysenteriae 1 (the only Shigella spp. to produce Shiga toxins), is a recurrent problem in many of the poor areas of the world, notably in Africa, Central America and parts of Asia. Many of these outbreaks are caused by multiple antibiotic resistant strains; the fatality rate of these infections can be as high as 20%. Furthermore, antimicrobial resistance appears to develop more quickly with S. dysenteriae 1 than in other Shigella spp. [9].

Shigella spp. are predominantly transmitted by fecal-oral route through person to person contact, contaminated food and water. Flies have also been identified as a transmission vector from contaminated fecal waste. Shigella infections can also be transmitted by oral anal contact in male homosexuals [10]. Shigella generally is considered to have a narrow distribution in nature, inhabiting essentially the intestinal tract of humans, as well as captive primates in which shigellosis naturally occurs. There is no evidence, however, that the disease naturally occurs, particularly in the wild in those monkeys, without prior contact with humans. Although shigellae are difficult, if not impossible, to grow from environmental samples, they are consistently present, particularly in sewage, and accidental contamination of water supplies by sewage influxes is regularly followed by an outbreak of shigellosis. Most cases following person-to-person transmission. Outbreaks are usually a response to food and/or water contamination [10]. The disease is endemic throughout the world, although 99% of the cases occur in developing world. Shigellosis is a disease that affects the poorest populations of the planet. It is estimated that there are 164.7 million cases of bacillary dysentery, annually, of which 163.2 million are in developed countries and 1.5 million in industrialized countries. Approximately 1.1 million people die from shigellosis each year, 61% of these are children under 5 years old [11]. Although distributed throughout the world, the prevalence of bacillary dysentery differs from place to place. Epidemics of shigellosis always have occurred among humans who gather in poor hygienic conditions: armies during military campaigns, pilgrimages, and refugee camps. In spite of the numerous epidemiological studies available, there is still a great need for accurate and frequently updated evaluation of the disease burden [10]. Due to the low infectious dose of Shigella the dissemination of the bacteria from person to person can be extremely swift and can be responsible for the high secondary attack rate when introduced within environs such as crowded and institutionalized populations. Children age 1 to 6 are most susceptible to infections due to Shigella. This phenomenon is compounded in poor developing nations because of the high numbers of malnourished children. These children face an increase in the attack rates and also greater mortality [12].

Figure(1): A; Shigella spp on Mac agar. B; Shigella spp. on XLD agar. C; Shigella spp in Microscopically examination[6].


2.2. Pathogenicity

Shigella has invasive properties that enable them to penetrate epithelial tissue in the bowel, while the toxin is also significant in pathogenesis [4]. The infectious dose is very small. Volunteer studies have shown that ingestion of as few as 100-200 viable organisms in milk is able to cause disease. Shigella is able to survive the low acidity of the stomach by upregulating the acid resistant genes. Shigellosis is characterized by a severe inflammatory response at the colonic mucosa and destruction of colonic epithelial cells [13]. Pathogenesis can be divided into five stages, entry of the bacterium into the host colonic cell. Lysis of phagosomes (the vacuole that forms from the fusion of the cell membrane around the phagocytized Shigella) and bacterial multiplication. Intra- and intercellular spread of the bacterium. The death of the host cell and ulceration of the mucosa. Resultant inflammatory response [13].

2.2.1. Mechanism of pathogenicity:

Shigella spp. possess several key properties that are responsible for their virulence. The ingested microbes must survive in the acidic environment of the stomach, which is the first significant host defensive barrier encountered by the bacteria. To produce clinical symptoms, Shigella must attach to and invade the epithelial cells of the colon, multiply and disseminate intracellularly through adjacent colonic epithelial cells, and cause abscesses and ulcerations of the intestinal lining leading to the bloody mucoid stools characteristic of dysentery. Bacterial invasion and replication also lead to an intense inflammatory response that serves both the host and the pathogen [13][14]. Shigella spp. have a preference for M cells of the colon; these are specialized epithelial cells associated with mucosal lymphoid tissue. After adherence to and uptake into colonic M cells, shigellae are engulfed by phagosomes and approximately 1.5 hrs later lyse the M cells vacuoles. The pathogen multiplies and spreads intracellularly from the basolateral side into the submucosa of the colon. Further events are: (1) the interaction with host immune effector cells, (2) apoptotic lysis of macrophages, and (3) cytokine release and infiltration of polymorphonuclear leukocytes (Lampel et al., 2000). Essential virulence attributes of Shigella are the abilities to enter into and disseminate within epithelial cells, as well as the ability to induce apoptosis in macrophages. The cellular biology and genetic studies of entry and dissemination have been performed mainly with S. flexneri but most conclusions derived from these studies probably also apply to other Shigella spp. and to EIEC [16].

2.2.2. Shiga toxin:

The name Shiga toxin is derived from a toxic activity originally discovered in Shiga's bacillus, S. dysenteriae. Credit for the discovery of Shiga toxin is generally accorded to Conradi, who described many of its properties in 1903. This activity was known as Shiga neurotoxin because when injected parenterally into mice or rabbits it resulted in limb paralysis followed by death of the animal. It has been realized that the Shiga family of toxins are in fact a major cause of disease in many developed countries [17]. The nomenclature for the Shiga toxin family has become confusing. In 1972, a toxin causing fluid secretions by rabbit small bowel was identified in S. dysenteriae 1 and named Shigella enterotoxin. This toxin was subsequently proved to be identical to the originally described Shiga neurotoxin. Following the discovery of that E. coli cytoxins were active on vero cells they were referred to as Verotoxins. This name is still used by many workers in the field who identify Verotoxin-producing E. coli as VTEC. However, when it became apparent in the early 1980s that these newly described E. coli toxins were very similar to Shiga toxin and were neutralized by antisera to Shiga toxin, other workers referred to them as Shiga-like toxins. By 1996, when the common mechanism of action and cellular binding site was proven, an international group of investigators decided to designate this group of biologically homogenous toxins simply as Shiga toxin (Stx), irrespective of their bacterial origin. The gene designation (Stx) for Shiga toxin from S. dysenteriae 1 was already well established and the new nomenclature therefore maintained the stx gene designation for the E. coli derived toxins [17]. The toxins are divided into two main groups, based on antigenic differences. Shiga toxin (stx) from S. dysenteriae 1 and Shiga toxin 1 (stx1) form one group, and the Shiga toxin 2 (stx2) family form the other group. As a result, Shiga toxin from S. dysenteriae and stx1 from E. coli are virtually identical, whereas Shiga toxin 2 differs significantly and is made up of a number of subfamilies [17]. Shiga toxin is a heat-labile protein and acts as enterotoxin and neurotoxin. The Shiga toxin shows three types of toxic activities:

1. Neurotoxic activity: This activity is demonstrable by paralysis and death of experimental animals following injection with the toxin. Although called neurotoxic, the primary site of its action is not the neural tissue but is the blood vessels, mainly of the central nervous system.

2. Enterotoxic activity: these toxins are enterotoxins which induce fluid accumulation in ligated rabbit ileal loop.

3. Cytotoxic activity: this is demonstrated by cytotoxicity of toxin of vero, HeLa, and some selected endothelial cells such as human renal vascular endothelial cells (Parija, 2009).

Shiga toxin clearly causes fluid secretion when placed in the small bowel lumen of rabbits and results in inflammatory enteritis in this model. Although it is cytotoxic to human colonic epithelial cells and thus can mimic colonic manifestations of clinical shigellosis, the interpretation is complicated because shigellae are invasive and multiply within epithelial cells [17].

2.2.3. Symptoms and Characteristics of the Disease:

Shigella dysentery, or bacillary dysentery, is characterized by the sudden onset of abdominal cramps, diarrhea, and fever after 1-4 days incubation. Mucus and sometimes blood appear in the feces. Bacteraemia occurs, occasionally in the compromised host, with a high fatality rate. The triad of symptoms — fever, abdominal pain and watery diarrhea — are not enough to implicate Shigella as the etiological agent, because other organisms, e.g. pathogenic E. coli, Salmonella, and Campylobacter, cause similar symptoms. If the illness progresses to the colonic phase (usually within 1-3 days), in
which the scanty stool becomes bloody and mucoid, specific diagnosis can be suspected. The severe (colonic) phase is characterized by waves of intense cramps, frequent bowel movements producing only scanty quantities of blood and mucus and a acute pain with each motion [4].

Shigellosis is differentiated from diseases caused by most other foodborne pathogens by at least two important characteristics:

(i) The production of bloody diarrhea or dysentery.

(ii) The low infectious dose.

Dysentery involves bloody diarrhea, but the passage of bloody mucoid stools is accompanied by severe abdominal and rectal pain, cramps and fever. While abdominal pain and diarrhea are experienced by nearly all patients with shigellosis, fever occurs in about one-third and gross blood occurs in about 40% of cases. The clinical features of shigellosis range from a mild watery diarrhea to severe dysentery. The dysentery stage caused by Shigella spp. may or may not be preceded by watery diarrhea. During the dysentery stage, there's extensive bacterial colonization of the colon and invasion of the cells of the colon. As the infection progresses, dead cells of the mucosal surface slough off. This leads to the presence of blood, pus and mucus in the stool [2]. The incubation period is usually 12-50 hrs; onset is rapid and accompanied by fever and severe abdominal pain. Symptoms usually last 3 to 4 days, but can persist for 14 days or longer. In healthy adults, death is rare, but Shigella dysentery is a major cause of death among infants in countries where hygiene is poor. Infections with S. dysenteriae almost always develop full and severe symptoms of dysentery and may be associated with many complications like leukaemoid reaction and haemolytic-uremic syndrome. It is known to produce proctored epidemics and pandemics and is usually multi-drug resistant. Similar symptoms, although often less severe, can also be associated with S. boydii and S. flexneri. Most adult infections by these spp., however, and virtually all by S. sonnei do not progress beyond relatively mild, non-bloody diarrhea. Symptoms may differ in young children and be of greater severity, possibly involving extraintestinal symptoms, including convulsions, headaches and delirium [18] [19]. The only natural hosts of Shigella are humans and monkeys; infections are localized in the colon and restricted to the outermost layer of the intestinal wall, where they elicit a strong inflammatory reaction [3].

2.4. Control of Shigella by some ecological and chemical factors.

2.4.1 Effect of temperature on Shigella growth:

The growth temperature is an important factor in controlling virulence. Virulent strains of Shigella spp. are invasive when grown at 37°C but non-invasive when grown at 30°C. This strategy ensures that the organism conserves energy by synthesizing virulence products only when the bacterium is in the host [2]. A chromosomal gene partly responsible for the temperature regulation of virulence gene expression is virR (hns). At 30°C, Shigella spp. are not pathogenic; on shifting the growth temperature to 37°C, the organism become virulent. Inactivation of virR by transposon mutagenesis yields constitutive expression of the invasive phenotype at both 30 and 37°C [20]. It was found that shigellae are killed at a temperature of 55°C within 1 hr (21).

2.4.2. The effect of pH on Shigella growth:

The reported pH range allowing growth of Shigella spp. is 4.8-9.3, although actual values will depend on acid type, Shigella spp. are gradually inactivated at pH values below 4.0, but the organism can survive for some time in acid conditions. Survival of Shigella in fruit juices and fresh fruits depend upon their pH, the type of strain and the incubation temperature. Fresh orange juice has been linked to a S. flexneri outbreak in South Africa, and Shigella spp. survived for up to 14 days in tomato and apple juice stored at 7°C. [23]. Lampel reported that under laboratory conditions, S. sonnei and S. flexneri grow in culture media with nearly the same pH values, between 4.5 and 9.3 [20]. In media with a pH of 4, no growth is observed and survival of the bacteria declines. With brain heart infusion medium, growth for S. sonnei and S. flexneri was observed at a minimum pH of 4.50 and 4.75, respectively [22]. Laboratory studies revealed that S. sonnei can survive on shredded cabbage at 0-6°C for 3 days without decrease in number but soon died at 24°C because of the pH drop of the cabbage due to the fast outgrowth of the spoilage microorganisms at this high temperature [23] [24].

2.5. The effect of inorganic and organic food preservatives.

2.5.1. Sodium chloride:

Common salt, sodium chloride, was undoubtedly the first antimicrobial substance to be used in foods. One can be confident that in early civilizations it was regarded as a preservative rather than for flavouring. Salting is the traditional method of preserving meat, often in combination with smoking and drying. Modern technology has provided more rapid methods of getting the salt into the meat, but the essentials have remained unchanged for centuries. Solutions containing 15-25% salt are used to bring water activity, aw, down to about 0.96. This has the effect of retarding the growth of most microorganisms, including the majority of those responsible for meat spoilage [25]. When high concentrations of salt are added to foods for the purpose of preservation, foodborne microbes undergo plasmolysis (shrinkage), as well as inhibition or death of microbial cells [26].

2.5.2. The effect of organic acids:

The use of organic food preservatives is among the oldest methods of microbial control in food preservation that have been used to control microbes in food. Most of these preservatives do not necessarily kill microbes but control them by inhibiting their growth, they are bacteriostatic rather than bacteriocidal [2]. Although they are all found naturally in nature, acids used as food preservatives are usually made chemically. These are effective mostly in foods having low pH preferably less than 5.5 [27]. The antimicrobial activity of a particular organic acid is attributed to the
reduction in pH as well as activities of the undissociated form of the molecule. These activities can exert deleterious effect on bacterial cell function in a synergetic manner. The drop in the pH of the medium forces cells to tolerate acidification of the cytoplasm or expand energy reversing this effect, while the undissociated acid, being soluble in lipids, can diffuse passively across the cell membrane and interfere with normal metabolism [28]. Studies concerning the mechanism of action of antimicrobials the most effects are those that occur at concentrations similar to those that reduce the rate of growth and approach the MICs. In order to understand the extent to which these effects are the primary cause of inhibition or lethality, it is necessary to consider the relative concentrations necessary for these effects [29].

2.5.2.1. Acetic acid:

Acetic acid (C2H4O2, m.w. 60.05) is produced in foods such as pickles by fermenting organisms, and it is a component of mayonnaise. It has a pungent odor, and is miscible with water and ethanol. Although it is known to depress pH, it is antimicrobial by other poorly understood mechanisms [26]. Acetic acid is one of the most important organic acids with broad use in the food industry, as acidifying additive and/or preservative. Although most of the market demand for acetic acid is satisfied by chemical synthesis, all of the acetic acid used in the food industry must be of biological origin and is produced using acetogenic bacteria [30]. Acetic acid is a weak acid, forming a dynamic equilibrium in aqueous solution between undissociated acetic acid molecule and acetate anions. The undissociated acid predominates at low pH and appears solely responsible for the antimicrobial activity. Undissociated acetic acid is a small, unmolecule that is able to dissolve in the hydrophobic lipid plasma membranes of microbes, and thus rapidly pass by diffusion into the cytoplasm. Once in the cytoplasm, acetic acid dissociates rapidly into acetate ions and protons, causing a severe drop in the pH of the cytoplasm, and inhibiting or killing the microbe Stratford M. Food and beverage spoilage yeasts [31].

2.5.2.2. Citric acid:

Citric acid is a popular acidulant and, due to its flexibility, its use as standard in virtually every preserved food. In fact citric acid is one of the most commonly used organic acids in the food as well as pharmaceutical and chemical industries [32]. Citric acid has antimicrobial properties due to its acidulation and chelating metal ions that catalyses oxidation. By chelating or binding metal ions, the substrate for bacterial growth is diminished in the food, thus influencing growth [33]. Citric acid is present in a variety of fruits and their juices in concentrations dose to 1%, although 4% has been reported in blackcurrants. As an antimicrobial agent, citric acid is poorly effective and required at high concentration for activity. It was found that 0.3% citric acid affect Salmonellae, 0.35% affect Enterobacteriaceae and 0.5% suppress the growth of some molds in bread. The most probable primary action by acetic acid is as an acidulant, lowering the pH of the cellular medium [33].

Lactic acid is a colorless or yellowish liquid that consists of a mixture of lactic acid (C3H6O3) and lactic anhydride (C6H10O5). It is hygroscopic and miscible with water and ethanol. It is produced naturally in many fermented foods such as yogurt and sauerkraut [26]. Lactic acid and lactates are used in a number of foods to improve stability and the inhibitory effects of lactic acids on pathogens and spoilage organisms in meat products can be observed, even in neutral pH. The inhibitory effect of lactate is usually ascribed to the undissociated acid that is membrane permeable and may compromise pH homeostasis of the cytoplasm. A decrease in water activity as a mechanism of action was considered insufficient for inhibition as was acidification of the cytoplasm. Lactate at neutral pH is a low affinity chelator of metal ions. Since the concentrations of lactic acid used are high, it is possible that the removal of metal ions, particularly Fe+3, may contribute to the antimicrobial action of lactate at neutral pH [34].

2.6. Antibiotic resistance of Shigella spp.

Among the bacterial cause of dysentery, Shigella spp. continue to be the most important, with a high infectivity rate and the development of antibiotic resistance. The prevalence of Shigella spp. varies over time and in different geographical areas. Antibiotic treatment is usually indicated for individuals with moderate to severe symptoms of shigellosis. Most Shigella infections are treated empirically, and therefore an understanding of resistance patterns is important for management. Empirical treatment has been compromised in large part by emerging resistance and inadequate surveillance to monitor trends [31]. Quinolones such as norfloxacin, ciprofloxacin, ofloxacin and feroxacin have emerged as drugs of choice for the treatment of various bacterial enteric infections, including shigellosis. Controlled trials have shown that quinolones in varying regimens, from a single dose to 5 days of treatment, significantly reduce the intensity and severity of traveller’s diarrhea as well as shigellosis. Quinolone resistance is presently uncommon among shigellae, but it is inevitable that resistance will develop from increased usage of these agents [35]. A recent study analyzed antibiotic susceptibility of Shigella isolates from eight Asian countries, the highest resistance rate was found for trimethoprim/sulfamethoxazole (81%), followed by tetracycline (74%) and ampicillin (53%). An Indian study of antibiotic resistance pattern of 166 shigellae strains isolated from stool samples of pediatric patients showed that all strains were susceptible to norfloxacin, but more than 90% strains were resistant to tetracycline and trimethoprim/sulfamethoxazole and 67% strains were resistant to ampicillin. Resistance to amoxicillin, chloramphenicol and nalidixic acid was found in 55, 46 and 29% strains, respectively. In contrast to neighbouring countries, low percentages of resistance were found to nalidixic acid norfloxacin (3-5%) and no resistance was found to ciprofloxacin, indicating that nalidixic acid with its low cost and safety in children could be recommended for the treatment of shigellosis [36].
3. Conclusion

Study Shigella spp. that cause dysentery and comparing their resistance to some growth factors, sodium chloride, organic food preservatives and some widely used antibiotics for this hazard bacterium which only little studies about it has done, it was observed that eight Shigella spp. The toxins are divided into two main groups, based on antigenic differences. Shiga toxin (stx) from S. dysenteriae 1 and Shiga toxin 1 (stx1) form one group, and the Shiga toxin 2 (stx2) family form the other group. Shigella are killed at a temperature of 55°C within 1 hr and died at 24°C and high concentrations of salt.

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