WORLD JOURNAL OF ADVANCE HEALTHCARE RESEARCH

ISSN: 2457-0400
Volume: 2.
Issue: 1.
Page N. 42-52
Year: 2018

Review Article www.wjahr.com

ZOONOTIC IMPORTANCE OF FOOD BORNE SALMONELLOSIS: A REVIEW

Dr. Shibabaw Bejano* and Abay Fiseha

Department of Veterinary science, Assosa University, P.O.Box 18, Asosa, Ethiopia.

Received date: 13 November 2017 **Revised date:** 04 December 2017 **Accepted date:** 25 December 2017

Corresponding author: Dr. Shibabaw Bejano
Department of Veterinary science, Assosa University, P.O.Box 18, Asosa, Ethiopia.
Email ID: bejanoshibabaw@yahoo.com,

ABSTRACT

Salmonellosis is a disease which ranges in man and animals from severe enteric fever, through severe enteritis (with complications) to mild food poisoning. The disease has a worldwide distribution. Salmonellosis is caused by bacteria of the genus *Salmonella*, of which over 2500 types (serotypes) have been identified. The disease is characterized by host specificity. Some types cause disease only, or primarily in one animal species, whilst others are ubiquitous and cause disease in many species. The disease in animals in the UK is most severe in cattle and particularly calves, and can result in high morbidity and mortality. The principal serotypes involved are *S. dublin* and *S. typhimurium*. The former is endemic in dairy herds, particularly in the west of the UK, and may be maintained in herds by 'carriers'. The disease is also frequently mild or characterized by carriage of salmonellas in the absence of disease. The disease is controlled in cattle by vaccination and the application of strict hygiene. Some serotypes, and particularly *S. typhimurium*, have developed resistance to a number of antibiotics and multiple antibiotic resistance may be transferred between strains.

KEYWORDS: Salmonellosis, Salmonella nomenclature, Zoonotic importance, Transmission, Risk groups.

1. INTRODUCTION

Salmonellosis is an infection with bacteria called *Salmonella*. Salmonella infections are very common and an important public health problem in many parts of the world. There are many different types of *Salmonella* but, with the exception of the few which cause typhoid or paratyphoid fever, the illness they cause is similar. *Salmonella* are widespread in cows, poultry, eggs, pigs, pets and wild animals. It is one of the most common foodborne diseases. It has long been said that, in 1885, pioneering American veterinary scientist, Daniel E. Salmon, discovered the first strain of *Salmonella*. Actually, Theobald Smith, research-assistant to Dr. Salmon, discovered the first strain of *Salmonella*–*Salmonella* cholerae suis. But, being the person in charge, Dr. Salmon received credit for the discovery. In any case, today the number of known strains of the bacteria totals over two thousand (Miller and Pegues, 2005; Behravesh *et al*., 2008).

Most persons infected with *Salmonella* develop diarrhea, fever, and abdominal cramps 12 to 72 hours after infection. The illness usually lasts 4 to 7 days, and most persons recover without treatment. However, in some persons, the diarrhea may be so severe that the patient needs to be hospitalized. In these patients, the *Salmonella* infection may spread from the intestines to the blood stream, and then to other body sites and can cause death unless the person is treated promptly with antibiotics. The elderly, infants, and those with impaired immune systems are more likely to have a severe illness (CDC, 2010).

Every year approximately 40,000 cases of salmonellosis are reported in the U.S. Many milder cases are not diagnosed or reported, so the actual number of infections may be 30 or more times greater. Salmonellosis is more common in the summer than in the winter (CDC, 2010).

Salmonella can cause enteric and multisystem disease in humans and animals. Human salmonellosis is most commonly associated with foodborne infection, but transmission of *Salmonella* from pets can occur. The risk is greatest with reptiles, but zoonotic transmission involving various other animal species has been documented. An infection with *Salmonella* bacteria usually affects the gastrointestinal system (the stomach and intestines) in humans. In more severe cases, *Salmonella* can spread to the blood, the bones, or even to

the fluid around the brain, but these types of infection are less common (Stevens *et al.*, 1989).

Salmonella bacteria are often found in the feces (poop) of some animals, particularly reptiles. Iguanas, for example, carry *Salmonella marina*. People who have these animals as pets are at more risk of getting salmonellosis because the bacteria from a reptile's feces can get on its skin. Then, when people handle the reptiles, they get the bacteria on their hands (CDC, 2009).

OBJECTIVE

- To review the zoonotic importance of salmonellosis

2. ETIOLOGY

The genus *Salmonella* belongs to the family *Enterobacteriaceae*. It is made up of gram-negative, motile (with a few exceptions), facultative anaerobic bacteria. Salmonellae grow between 8°C and 45°C and at a pH of 4 to 8. They do not survive at temperatures higher than 70°C. Pasteurization at 71.1°C for 15 seconds is sufficient to destroy salmonellae in milk. These bacteria can resist dehydration for a very long time, both in feces and in foods for human and animal consumption. In addition, they can survive for several months in brine with 20% salinity, particularly in products with a high protein or fat content, such as salted sausages; they also resist smoking. It has been indicated that they can survive for a long time in soil and water (WHO Expert Committee on Salmonellosis Control, 1988).

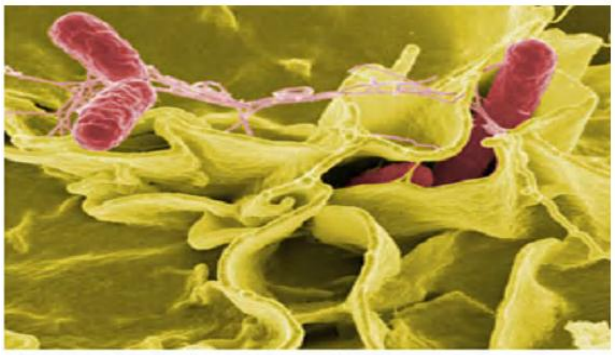
Salmonella nomenclature is complex, since different scientists use different systems to refer and communicate about this genus. Some individuals prefer Kauffman's initial 'one serovar-one species' concept, while others favor schemes based on clinical presentation, biochemical characteristics or genetic relatedness (Brenner *et al*., 2000).

Currently, there are three recognized species: *S. enterica*, *S. bongori* and *S. subterranean*, with six main subspecies: *enterica* (I), *salamae* (II), *arizonae* (IIIa), *diarizonae* (IIIb), *houtenae* (IV), and *indica* (VI). Historically, serotype (V) was *bongori*, which is now considered its own species (Achtman *et al.*, 2012).

The serovar (i.e. serotype) is a classification of *Salmonella* into subspecies based on antigens that the organism presents. It is based on the Kauffman-White classification scheme that differentiates serological varieties from each other. Serotypes are usually put into subspecies groups after the genus and species, with the serovars/serotypes capitalized but not italicized: an example is *Salmonella enterica* serovar Typhimurium (Porwollik, 2011; CDC, 2011).

From clinical perspective, the serovars of *Salmonella* which are pathogenic for humans are traditionally divided into two groups. The typhoidal group included the serovars which cause enteric fever (Typhi, Paratyphi A, Paratyphi B, Paratyphi C and Sendi). The second group usually referred to as the non-typhoidal *Salmonella* (NTS) contains all remaining serovars of subspecies I (WHO, 2003).

The genus is further divided, serologically, into over 2,500 serovars (or serotypes), these being distinguished according to their possession of different antigens (0, H, or Vi). Most of the recognised serovars belong to the species *S. enterica*, and only 20 are included in S. *bongori.* Nearly 1,500 serovars, including almost all of those important in foodborne disease, belong to a single subspecies, *S. enterica* subsp. *enterica.* The serotype names have traditionally been used as if they denote species (e.g. S. Enteritidis, S. Typhimurium), but an example of the correct nomenclature under the currently accepted classification would be *S. enterica* subsp. *enterica* serovar Enteritidis**.** For convenience this can be abbreviated to *S.* Enteritidis, and this form will be continued here. Further differentiation of *Salmonella* isolates can be done by biochemical characteristics and by' phage-typing ' (Burros, 2006).



Source: Ryan and Ray (2004)

Figure 1: Color enhanced scanning electron micrograph showing *Salmonella* Typhimurium (red) invading cultured human cells.

Except for serotypes *S*. Typhi and *S*. Paratyphi A, and *S*. Paratyphi C, which are strictly human and whose only reservoir is man, all serotypes can be considered zoonotic or potentially zoonotic. Salmonellae have several virulence factors that contribute to causing diarrhea, bacteremia, and septicemia. These factors include the lipopolysaccharide of the outer wall, pili, flagella, cytotoxin, and enterotoxin (Murray, 1986).

3. Geographic Distribution/Epidemiology

3.1 Typhoidal salmonellosis

Typhoid fever is a global problem and its real impact is difficult to estimate because the clinical picture is similar with those of many other febrile infections, a recent study estimated that globally there are more than 22

million cases of typhoid fever each year with more than 200,000 deaths (WHO, 2003).

S. Typhi have high host specificity for humans and most often, acquisition of the organisms occurs by ingestion of food or water contaminated with human excreta and associated with poor sanitation and hygiene (Miller *et al*., 2000).

The disease is endemic in many developing countries, particularly in Asia, Africa, Latin America and Caribbean regions. These countries share several characteristics including rapid population growth, increased urbanization, inadequate human waste treatment, limited water supply, and over-burdened health care systems. In developed countries, typhoid fever is predominantly related to traveling to the previously mentioned regions (Crump *et al*., 2004).

The epidemiology of paratyphoid fever is less well described than typhoid fever. Nevertheless, estimates suggest that as much as 25% of enteric fevers may be caused by *S.* Paratyphi A (Crump *et al*., 2004). Furthermore, *S.* Paratyphi A is on the rise in South Asia (Ochiai *et al*., 2005) and may cause as severe infection as *S.* Typhi. The disease patterns associated with *S.* Paratyphi A in developing countries are probably similar to that of *S.* Typhi (Maskey *et al*., 2006).

3.2. Non-typhoidal salmonellosis

Unlike *S.* Typhi and *S.* Paratyphi, whose only reservoir is humans; non-typhoid salmonellosis is acquired from multiple animal reservoirs. The main mode of transmission is from food products contaminated with animal products or wastes, most commonly eggs and poultry products. Data concerning NTS serovars is notoriously difficult to obtain, as most patients have a mild and usually self-limiting illness, rather than systemic infections, so do not need to consult the health services (Miller *et al*., 2000).

In developing countries NTS is also an important cause of invasive disease, particularly in tropical regions of Africa, where *Salmonella enterica* serovar Typhimurium (*S.* Typhimurium) and *Salmonella enterica* serovar Enteritidis (*S.* Enteritidis) are consistently the most common causes of childhood bacteraemia, as well as important causes of meningitis, septic arthritis and pneumonia (Kariuki *et al*., 2006).

According to the 2002 WHO Global *Salmonella* Surveillance report on the distribution of *Salmonella* serotypes from 2000 to 2002, during the 3-year period, *Salmonella enterica* serovar Enteritidis was by far the most common serotype reported from human isolates globally (Galanis *et al*., 2006).

In 2002, it accounted for 65% of all isolates, followed by *S.* Typhimurium at 12% and *S.* Newport at 4%. Among nonhuman isolates, *S.* Typhimurium was the most commonly reported serotype in all 3 years, accounting for 17% of isolates in 2002. It was followed by *S.* Heidelberg (11%) and *S.* Enteritidis (9%). In Africa in 2002, *S.* Enteritidis and *S.* Typhimurium were each reported from approximately one fourth of isolates from humans (Galanis *et al*., 2006).

Nontyphoidal *Salmonella* has a particularly profound impact in populations of high HIV/AIDS prevalence, since mortality in those with co-infections has been reported to be high. However, very little is known regarding the source and transmission of NTS in developing countries. It is likely that animal-human transmission via the food chain is less responsible than is human-human transmission, with contaminated water an important vehicle in communities with poor hygiene, sanitation and overcrowding (Graham, 2002; Kariuki *et al*., 2006). A major and worrying development that has global significance is the increasing and often rapid emergence of multidrug resistant (MDR) strains for all the above serovars (Graham, 2002).

Studies from tropical Africa have consistently shown that NTS bacteremia is more common during the rainy season. This may be the result of a seasonal increase of intestinal salmonellosis which has been reported from other (non-malarious) tropical regions. It may also reflect the seasonal pattern of diseases such as malnutrition and malaria, which may confer an increased risk of *Salmonella* infection (Graham, 2002).

4. PATHOGENESIS

An infectious process can only begin after living salmonellae (not only their toxins) reach the gastrointestinal tract. Some of the microorganisms are killed in the stomach, while the surviving salmonellae enter the small intestine and multiply in tissues (localized form). The local response to the endotoxins is enteritis and gastrointestinal disorder. In the generalized form of the disease, salmonellae pass through the lymphatic system of the intestine into the blood of the patients (typhoid form) and are carried to various organs (liver, spleen, kidneys) to form secondary foci (septic form). Endotoxins first act on affected organs' vascular and nervous systems, manifested by increased permeability and decreased tone of the vessels, upset thermal regulation, vomiting and diarrhea. In severe forms of the disease, enough liquid and electrolytes are lost to upset the body's water-salt metabolism, to decrease the circulating blood volume and arterial pressure, and to cause hypovolemic shock. Septic shock also may develop. Shock of mixed character (with signs of both hypovolemic and septic shock) is more common in severe salmonellosis. Oliguria and azotemia develop in severe cases as a result of renal (kidney) involvement due to hypoxia and bacteremia (Dworkin *et al.*, 2001).

5. CLINICAL PRESENTATIONS

Salmonella infections can have a broad range of illness, from no symptoms to severe illness. The most common clinical presentation is acute gastroenteritis. Symptoms include diarrhea and abdominal cramps, often accompanied by fever of 100°F to 102°F (38°C to 39°C).Other symptoms may include bloody diarrhea, vomiting, headache and body aches (Miller and Pegues, 2005 ; American Academy of Pediatrics, 2006). The incubation period, or the time from ingestion of the bacteria until the symptoms start, is generally 6 to 72 hours; however, there is evidence that in some situations the incubation can be longer than 10 days. People with salmonellosis usually recover without treatment within 3 to 7 days. Nonetheless, the bacteria will continue to be present in the intestinal tract and stool for weeks after recovery of symptoms—on average, 1 month in adults and longer in children (Behravesh *et al*., 2008).

S. Typhi and Paratyphi generally cause a bacteremic illness—*Salmonella* found in the blood—of long duration. This illness is called enteric, typhoid, or paratyphoid fever. Symptoms start gradually, and include fever, headache, malaise, lethargy, and abdominal pain. In children, it can present as a non-specific fever. The incubation period for *S.* Typhi is usually 8 to 14 days, but it can range from 3 to 60 days. For *S.* Paratyphi infections, the incubation period is similar to that of non-typhoidal *Salmonella*, 1 to 10 days (Miller, *S.* and Pegues, 2005; Behravesh *et al*., 2008).

6. SOURCES OF INFECTION AND MODE OF TRANSMISSIONS

The type of *Salmonella* usually associated with infections in humans, nontyphoidal *Salmonella*, is usually contracted from sources such as: Poultry, pork, and beef, if the meat is prepared incorrectly or is infected with the bacteria after preparation. Infected eggs, egg products, and milk when not prepared, handled, or refrigerated properly. Reptiles such as turtles, lizards, and snakes, which may carry the bacteria in their intestines. Tainted fruits and vegetables can also act as sources (Patrick, 2004).

6.1 Humans

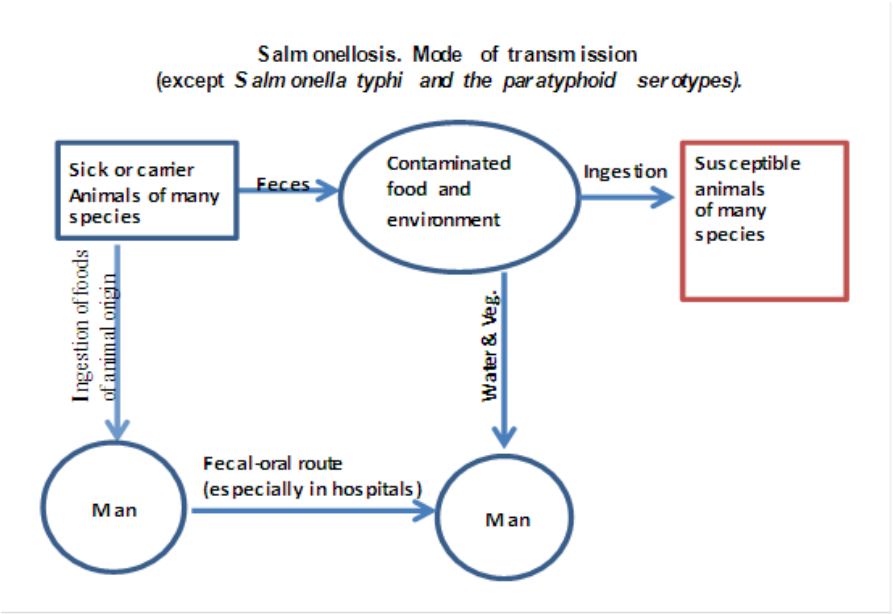
Contamination of food by infected food handlers is unusual; it is only likely to occur when a handler has diarrhoea and contaminated hands come in direct contact with food that is not subsequently cooked. Human transmission via the faecal-oral route can occur, especially among patients in hospitals and institutions. In these cases, poor personal hygiene has a vital role in the transmission (Hargrett *et al.*, 1998).

6.2 Animals and environment

Food animals can become infected via direct contact with other infected or symptomless animals, via contaminated feed or water, via their environment, or via wild birds or rodent pests. *Salmonella* may also infect common pets such as cats and dogs, and thus these animals may act as a source of the food contamination. Reptiles such as terrapins are also common carriers of *Salmonella.* In addition, manufacturing, catering, or domestic environments can become contaminated with *Salmonella* and act as a source of the organism, for example, mops and cloths, refrigerators, slicing machines and other inadequately cleaned and sanitized equipment can harbor *Salmonella* (Advisory Committee on the Microbiological Safety of Food, 1996).

6.3 Foods

The main source of *Salmonella* for man is food from infected food animals. Thus, meat, poultry, eggs, or raw milk can become contaminated with intestinal faecal material from infected livestock. Eggs can be contaminated on the shell with S. Enteritidis, as well as other salmonellas, as a result of either faecal carriage of the chicken or contamination from the environment. Under certain conditions, shell organisms are able to penetrate into egg contents. More importantly, however, egg contents can be contaminated with **S.** Enteritidis as a result of systemic infection of the laying hen, which results in infection of the reproductive tissues, particularly the oviduct. In 1993, it was estimated that in eggs sampled from retail outlets, 1 in 880 were positive for *Salmonella*. An unpublished report suggested that eggs sampled in 1996 showed a contamination rate of approximately 1 in 700**.** However, evidence collected by the egg industry more recently suggests that the contamination rate of eggs produced in the UK has fallen significantly since then, as a result of improvements in hygiene, and the large-scale vaccination of laying flocks. Consumption of contaminated foods that are consumed raw or not properly cooked can lead to food poisoning. Cross-contamination of other food materials that are not subsequently cooked - during processing, via chopping boards and other equipment used during food preparation – can lead to further incidents of food poisoning. The prevalence of *Salmonella* in foods is variable. In 2001, a UK study reported that average of 5.8% of poultry carcasses were contaminated with *Salmonella*. In Spain, a study of the prevalence in chicken legs carried out in 1999 showed that 35.8% of samples were contaminated (Humphrey, 1995), and in the same year, a survey of *Salmonella* in broilers in the US gave a contamination rate of 11%. The prevalence in beef and lamb carcases is reported to be less than 1%. Vegetables, h i t and herbshpices can be contaminated with *Salmonella,* and levels reported are 1.9-8%, 15.4% and 6.7-13.8%, respectively. Data collected for seafood in the US over an eight-year period during the 1990s showed that the overall prevalence of *Salmonella* was 7.2% for imported products, and 1.3% for domestically produced seafood (Dominguez *et al*., 2002).

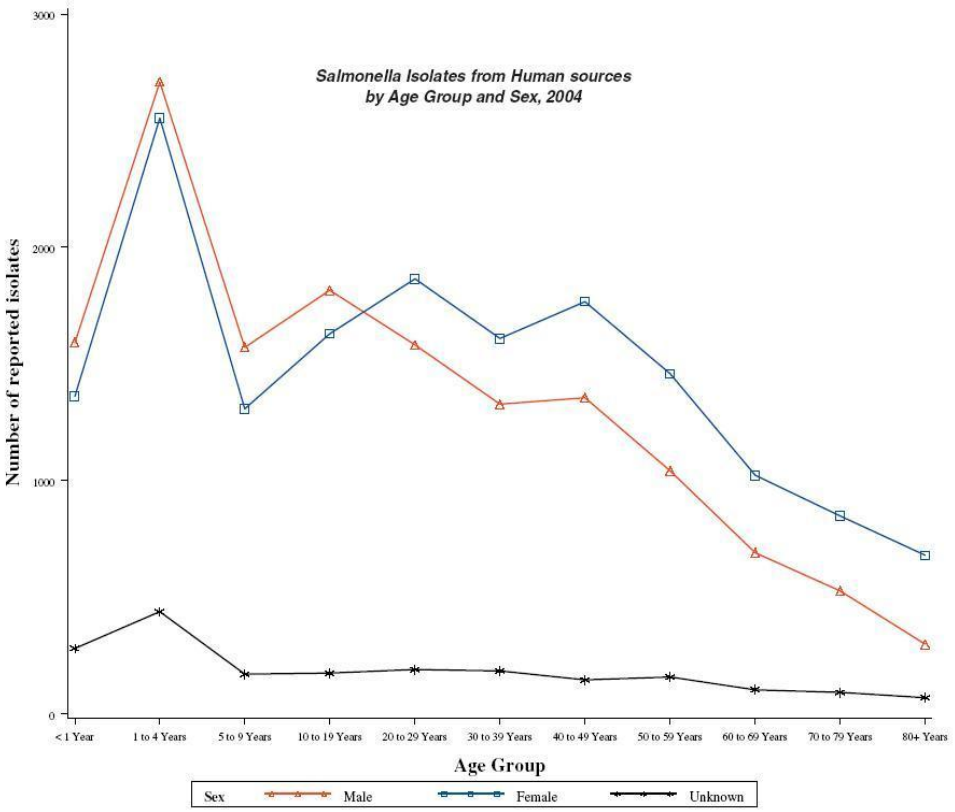


Source: Acha and Szyfres (2001)

Figure 2: Mode of transmission of Salmonellosis.

7. RISK GROUPS

Risk factors for salmonellosis include people who take cancer drugs and antacids or stomach acid suppression medication, young children, extremes of age, and those with weakened immune systems (such as people with HIV and those with sickle cell anemia). In these higher risk groups, *Salmonella* is more likely to invade beyond the gastrointestinal tract and cause bacteremia (bacteria in the bloodstream). From there the bacteria can spread deeper into the body and cause more serious diseases, like meningitis (Crum-Cianflone, 2008). Approximately 95% of cases of human salmonellosis are associated with the consumption of contaminated products such as meat, poultry, eggs, milk, seafood, and fresh produce (Foley and Lynne, 2008).



Source: Acha and Szyfres (2001)

Figure 3: Salmonella isolates from Human source by age group and sex, 2004.

8. DIAGNOSIS

Diagnosis of salmonellosis presents several difficulties. In developing countries, salmonellosis is frequently diagnosed solely on clinical grounds. However, isolation of the causative organism is necessary for a definitive diagnosis, for performing antimicrobial susceptibility testing, and for further characterization (WHO, 2003).

8.1 Culture

Various enrichment and selective media are used to isolate salmonellae from different clinical specimens. Non-typhoidal *Salmonella* gastroenteritis is commonly diagnosed from stool culture. In cases where there is concern about bacteremia, blood culture is indicated (WHO, 2003).

S. Typhi and other typhoidal *Salmonella* are frequently isolated from blood during the first weeks of illness and usually positive stool cultures occur during the second and third weeks of disease (Cheesbrough, 2000). *S.* Typhi can also be isolated from bone marrow, rose spots and, infrequently, from urine cultures (Khan *et al*., 1998). Occasionally *Salmonella* may be cultured from other samples such as joint aspirates, cerebrospinal fluid or endocarditic heart valves. Specialist environmental laboratories may look for *Salmonella* in food or water samples, either as routine or in outbreak situations. The best recovery of *Salmonella* species from fecal samples can be achieved by the use of direct plating and inoculating on standard enrichment broths. Many selective agar plates are available for *Salmonella.* Most laboratories use one medium with low selectivity, such as MacConkey, Deoxycholate agar (DCA) or Cystine Lactose Electrolyte-deficient (CLED) agar, and one with higher selectivity, such as Xylose Lysin Deoxycholate agar (XLD). *Salmonella* enrichment broths (e.g. selenite broth and tetrathionat broth) may help to recover low numbers of organisms (Cheesbrough, 2000).

8.2 Biochemical tests

Salmonella species are motile (with a few exceptions), facultative anaerobic, produce acid from glucose usually with the production of gas, and are oxidase negative. Most produce hydrogen sulphide except *Salmonella* Paratyphi A and *Salmonella* Typhi, which is a weak producer. Most *Salmonella* species don't ferment lactose. However, approximately 1% of the organism is able to ferment this sugar and thus may not be detected by clinical laboratories that use MacConkey agar and most laboratories use XLD agar or similar selection media to detect lactose fermenter *Salmonella* isolates (Miller *et al*., 2000). Urease production and indole production are negative, and most NTS produce citrate and hydrogen sulfide (Cheessbrough, 2000).

8.3 Serogrouping / serotyping

Salmonellae can be characterized by their somatic (O) and flagellar (H) antigens, the latter existing in some serotypes of phases 1 and 2. Some salmonellae also have an envelope antigen called Vi (virulence). The O antigen is usually determined by means of slide agglutination test with group-specific antiserum followed by agglutination with factor antiserum. H antigen is usually determined by means of the tube agglutination test. Partial serotyping is often sufficient for diagnosing purpose. Although serotyping seems convenient and easy to perform, there is a delay of three days or more to generate result. Complete serotyping is limited to specialist laboratories, because it requires highly trained personnel, and tube agglutination and phase-conversion plates are labor intensive. A wide range of antisera is required; production of antisera to rare antigens is expensive. A further limitation is that 5-8% of isolates are only partially typed or untyped. These include mucoid strains, in which the capsular polysaccharides block the exposure of O antigens. Non motile isolates cannot be fully typed to serovar level. Prolonged sub-culturing may theoretically affect the antigenic properties of a strain (Kim *et al*., 2006).

8.4 Serology

The Widal test is used for serological diagnosis of typhoid fever and measures agglutinating antibody levels against O and H antigens. The levels are measured by using double dilutions of sera. Usually, O antibodies appear on days 6-8 and H antibodies on days 10-12 after the onset of the disease (WHO, 2003). The test has only moderate sensitivity and specificity. It can be negative in up to 30% of culture proven cases of typhoid fever. This may be because of prior antibiotic therapy that has blunted the antibody response. A study conducted in Vietnam on the evaluation of Widal test, showed that the antibody responses to both antigens were highly variable among individuals infected with serotype Typhi, and elevated antibody titers were also detected in a high proportion of serum samples from healthy subjects from the community (House *et al*., 2001).

In a study done to evaluate the significance of Widal test on 242 typhoid suspected patients, the Widal qualitative test was positive in 92 (38%), 10 (43.5%), and 55 (31.3%) in typhoid suspected patients, febrile non-typhoidal patients, and healthy blood donors, respectively. It is therefore important to determine the antibody level in normal population in a particular locality in order to determine a threshold above which the antibody titer is significant. This is important, if as usually happens, a single acute sample is available for testing. If paired sera are available, a fourfold rise in the antibody titer between acute sera and convalescent is diagnostic. Despite these limitations, this test could be of use for the diagnosis of typhoid fever in patients who have clinical typhoid fever but are culture negative or in regions where bacterial culturing facilities are not available (Awol, 2004).

8.5 Molecular technique

The rapid detection of microbial pathogens is critical since people's lives may depend on it. Thus, there is a need for more reliable and faster methods. *Salmonella*

cultures take 4–7 days for isolation and identification, a problem for diagnosis and treatment. In addition, sensitivity of cultures can be affected by antibiotic treatment, inadequate sampling, variations of bacteraemia and a small number of viable organisms in faeces (Jordan *et al*., 2009).

DNA probes and polymerase chain reaction (PCR) protocols have been developed to detect *S*. Typhi directly from the blood (Haque *et al*., 1999). PCR has proven an invaluable tool for detection and it should be implemented to obtain a rapid yes/no answer on-site. It is possible, using molecular methods, to identify and distinguish between different *Salmonella* serovars within 4 h if a whole cell PCR is performed or 7 h if genomic DNA is to be extracted first. With gene specific PCR, it is also possible to specifically detect a pathogenic organism from a mixed bacterial culture (Chaudhry *et al*., 1997).

It was also proven that RFLP analysis of 16S rRNA PCR amplicons could be used as a first step fingerprint in the molecular based approach for distinction between different *Salmonella* serovars. With Multiplex PCR, multiple gene products can be amplified in a single PCR reaction and in this study it is clearly shown as a rapid method to distinguish between *Salmonella* Typhimurium and *Salmonella* Typhi, taking 4 h to make that distinction (Prakash *et al*., 2005). Haque and his colloquies (1999) tried to compare polymerase chain reaction-based technique with blood culture and Widal test during the first week of illness of 82 suspected cases of typhoid and found that the respective figures of positivity for PCR, blood culture and Widal test were 71.95%, 34.1%, and 36.5%.

A study in Jordan on *Salmonella* isolates from poultry and meat products showed that out of 212 total samples, *Salmonella* was detected in 185 samples (87%) by PCR technique, while 172 (81%) samples were detected *Salmonella* positive by conventional microbiological methods (Malkawi and Gharaibeh, 2003). PCR assay proved to be an effective method for *Salmonella* detection in meat and poultry products with less time-consuming procedure. Using PCR, *Salmonella* spp. detection could be achieved within 24–36 h compared to 3–8 days for the conventional microbiological methods (Malkawi and Gharaibeh, 2003).

Pradyot Prakash *et al.* compared nested PCR using *H1-d* primers, which is specific for *Salmonella enterica* serovar Typhi, with blood culture and the single-tube Widal test and found out that nested PCR can be used as a gold standard to determine the cut off titer of the Widal test for diagnosis of typhoid fever (Prakash *et al.*, 2005).

9. TREATMENT

Treatment is restricted to management of clinical salmonellosis. There is no indication to treat healthy carriers. There is no evidence that antimicrobials are effective for eradication of *Salmonella* colonization, and there is evidence that colonization is transient. Antimicrobial treatment could also increase the risk of antimicrobial resistance and antimicrobial - associated diarrhea, and is not recommended. Attempts made by turtle breeders to produce "*Salmonella* - free" turtles through the use of antimicrobials have just resulted in production of turtles carrying antimicrobial – resistant *Salmonella* (CDC, 2010).

Management of salmonellosis depends on the nature and severity of disease. In mild cases, close observation for dehydration and other consequences of diarrhea and anorexia may be adequate. Fluid therapy may be indicated in more severe or prolonged cases, with possible need for adjunctive therapy such as nutritional support and colloids (Sirinavin and Garner, 2000).

Antimicrobial use in salmonellosis is controversial. There is no evidence that antimicrobials are effective against enteric salmonellae or that they decrease the severity or duration of diarrhea. Antimicrobials, if used, are directed against systemic manifestations of salmonellosis to prevent or treat bacteremia and secondary infections such as arthritis, meningitis, and pneumonia, which are rare, especially in immune competent adult animals. Antimicrobials are not typically recommended in dogs and cats with uncomplicated gastroenteritis but may be indicated in animals with severe disease, as well as young and old animals, immunosuppressed animals, or animals with significant co morbidities. Drug choice should be based on *in vitro* susceptibility testing (CDC, 2010).

10. PREVENTION AND CONTROL

Theoretically, it is possible to eliminate salmonellae that cause enteric fever since the bacteria survive only in human hosts and are spread by contaminated food and water. The control and near elimination of typhoid fever in developed countries has been achieved largely because of improved sanitation, surveillance, contact tracing and successful therapy; this is also supported with vaccination. In developing countries reducing the number of cases in the general population requires the provision of safe drinking water, effective sewage disposal and hygienic food preparation (Mastroeni, 2006). In areas where the epidemic is high, mass immunization has been used successfully. Currently three vaccine alternatives are available: 1) a heat–killed, phenol extracted, whole cell vaccine, 2) Ty21a, an attenuated *S.* Typhi vaccine, 3) Vi vaccine, consisting of purified Vi polysaccharide from the bacterial capsule (Cammie and Miller, 2000). In developed countries, most cases are the result of travel to endemic areas. Travelers in such areas need to take particular care with water and food (Parry *et al.*, 2002).

Non-typhoidal *S. enterica* infections are a major public health problem world-wide and reduction of these diseases presents a serious and challenging problem.

These diseases have several animal reservoirs. In additions, the fact that a large number of different *S. enterica* serovars cause gastroenteritis in humans probably makes vaccines very difficult to realize and/or use commercially (Strugnell and Wijburg, 2006). The incidence of nontyphoidal salmonellosis continues to rise along with rates of emergence of antibiotic resistant strains and increased centralization of food production. Thus, it is important to monitor every step of food production, from handling of raw products to preparation of finished foods. In particular, with the increasing prevalence of *S.* Enteritidis in egg-laying hens, it is recommended that pasteurized eggs should be substituted for bulk-pooled eggs. The prudent use of antimicrobial agents in both humans and animals is necessary to minimize the further emergence of antibiotic resistant strains (Cammie and Miller, 2000).

11. THE ECONOMIC IMPACT OF SALMONELLA INFECTIONS

Salmonella causes billions of dollars in medical costs and lost productivity every year. The USDA Economic Research Service (ERS) published its first comprehensive cost estimates for sixteen foodborne bacterial pathogens in 1989. Five years later, it was estimated that the medical costs and productivity losses that *Salmonella* infections caused each year ran from $1.188 billion to over $11.588 billion, based on an estimate of 1.92 million cases and between 960-1,920 deaths (Council for Agriculture, Science and Technology / CAST, 1994).

In 1996, ERS updated the cost-estimates for six bacterial pathogens, including *Salmonella*. ERS continued to use cost-of-illness (COI) methodology for nonfatal illnesses, but adopted two different health valuation methodologies for premature deaths: the individualized human capital approach and the willingness-to-pay (WTP) approach. This report concluded as follows**:** We assumed that each of the 800-4,000 salmonellosis cases who die prematurely because of their illness incurred the same amount of medical costs as a salmonellosis patient who was hospitalized and survived ($9,087). We estimated medical costs for those who die from salmonellosis to range between $7.3 million and $36.3 million annually (Buzby et al., 2009).

ERS updated the cost-estimates for four pathogens (*Campylobacter*, *Salmonella*, *E. coli* O157:H7, and *Listeria monocytogenes*) again in 2000. The 2000 estimates were based on newly released estimates of annual foodborne illnesses by the CDC, and put the total cost in the United States for these four pathogens at $6.5 billion a year. More recently, in 2007, it was estimated that the annual costs of all foodborne disease in the United States was $1.4 trillion (Buzby et al., 2009).

12. STATUS OF SALMONELLOSIS IN ETHIOPIA

Salmonellosis caused by non-typhoidal *Salmonella* is particularly common in children of developing countries. The research conducted by Faris and Kaba in 1999, indicated that the local practice of sanitation was far from satisfactory and that the personal hygiene status of house mothers responsible for food preparation and child rearing was poor (Faris and Kaba, 1999). Mache (2002) reported that of all his *Salmonella* isolates, 78 % (46/59) belonged to non-Typhi serogroups, which in turn indicates that these serogroups are responsible for the majority of diarrhoea in children. In many developing countries, non-Typhi serogroups have become serious pathogens especially among immuno-suppressed adults, particularly those with HIV/AIDS (Arthur *et al*., 2001).

In a study conducted in Jimma Hospital, South West Ethiopia, from March to July 2000, a total of 59 *Salmonella* strains were isolated from 384 pediatric outpatients with diarrhoeal illness (Mache, 2002). Of these, Serogroup A comprised 5 (8.5%) isolates, B 17 (28.8%), C 13 (22%), D 8 (13.6%, other than *S.* Typhi), E 3 (5.1%) and *S.* Typhi 13 (22%). The most frequently isolated serogroup was B (28.8%) while the least frequent was group E (5.1%). The increased isolation rate *Salmonella* in Jimma diarrhoeal pediatric outpatients, may indicate the poor sanitary condition of the town and endemicity of the isolates in the area (Mache, 2002).

Other study was conducted from Feburary to July 2001 in Jimma Hospital, South West Ethiopia (Awole *et al*., 2002). The aim of study was to isolate and determine the magnitude of potential bacterial pathogens in the stool of HIV-positive and negative patients and their antimicrobial resistance profile. A total of 372 patients (192 HIV positive and 180 HIV-negative) were selected and stool specimens were collected and 63 cultured. Among 99 HIV-positive patients with diarrhoea, a total of 8 (8.1%) *Salmonella* strains were isolated. This result is in agreement with studies conducted in Rwanda (11%) and Argentina (5%) (Clerinx *et al*., 1995; Olmos *et al*., 1996) but much lower than the previous study conducted in Ethiopia (55.1%) (Wolday and Erge, 1998).

Another recent study in 2000-2001, compared the prevalence of *Yersinia enterocolitica* isolates with other commonly encountered enteropathogens causing diarrhea among patients in Addis Ababa. The study showed that among stool samples of 205 patients 3 (1.5%) were positive for *Y. enterocolitica,* 22 (10.7%) for *Salmonella* and 12 (5.8%) for *Shigella* (Andualem and Geyid, 2003). The frequency of isolation of *Salmonella* in their study (10.7%) was much higher than the isolation rate in all age groups (Asrat *et al*., 1999) or from adult outpatients (4.5%) (Ashenafi, 1983). From this previous data, the isolation rate of both *S.* Typhi and NTS appears to be decreasing from time to time. This does not mean that *Salmonella* infection in the country is

decreasing rather the reverse seems true (Andualem and Geyid, 2003).

13. CONCLUSION AND RECOMMENDATIONS

Salmonellosis is an infection with bacteria called *Salmonella*. Most persons infected with *Salmonella* develop diarrhea, fever, and abdominal cramps 12 to 72 hours after infection. The illness usually lasts 4 to 7 days, and most persons recover without treatment. However, in some persons, the diarrhea may be so severe that the patient needs to be hospitalized. The elderly, infants, and those with impaired immune systems are more likely to have a severe illness. It is a major public health problem worldwide. Infants and young children are at the highest risk. It also contributes to negative economic impacts due to the cost of surveillance investigation, treatment and prevention of illness. Most commonly spread by unwashed hands, cross-contamination, and infected animals.

People who have salmonellosis should not prepare food or pour water for others until their diarrhea has resolved. Many health departments require that restaurant workers with *Salmonella* infection have a stool test showing that they are no longer carrying the *Salmonella* bacterium before they return to work.

People should wash their hands after contact with animal feces. Because reptiles are particularly likely to have *Salmonella*, and it can contaminate their skin, everyone should immediately wash their hands after handling reptiles. Reptiles (including turtles) are not appropriate pets for small children and should not be in the same house as an infant.

Based on the above conclusion, the following recommendations are forwarded:

- Protection of prepared foods against contamination. Maintain sanitary kitchens and protection of foods against rodent and insect contamination
- Health education to caterers, food handlers, house wives about the severity of disease, importance of thorough cooking of food and personal hygiene (washing of hands before eating and after defecation).
- Further and systematic study especially in developing countries is necessary
- Laboratory facility should be ensured to examine the suspected food stuffs
- Continuous disease surveillance and monitoring system should be ensured

14. REFERENCES

1. Acha, P. N. and Szyfres, B.: Zoonoses and communicable diseases common to man and animals. Third Edition, Pan American Health Organization. Washington D.C, U.S.A, 2001; 238 & 241.
2. Achtman, M., Wain, J.,Weill, F. O. X., Nair, S., Zhou, Z., Sangal, V., Krauland, M. G., Hale, J. L., Harbottle, H., Uesbeck, A., Dougan, G., Harrison, L. H., Brisse, S.: S. Enterica MLST Study Group: "Multilocus Sequence Typing as a Replacement for Serotyping in Salmonella enterica". In Bessen, Debra E. *PLoS Pathogens,* 2012; 8(6): e1002776. doi:10.1371/journal.ppat.1002776. PMC 3380943. PMID 22737074.
3. Advisory Committee on the Microbiological Safety of Food. Report on poultry meat. Annex D: survey of *Salmonella* contamination in UK produced raw chicken on retail sale. Ed. Advisory Committee on the Microbiological Safety of Food. London. *HMSO*, 1996; 110-30.
4. American Academy of Pediatrics, "*Salmonella* infections," RED BOOK.: Report of the Committee on Infectious Diseases, edited by L. K. Pickering, 2006; 581–584.
5. Andualem, B. and Geyid, A., The prevalence of *Yersinia enterocolitica* isolates in comparison to those of the commonly encountered enteropathogens causing diarrhoea among Ethiopian patients in Addis Ababa. *Ethiop Med J*, 2003; 41: 257-66.
6. Arthur, G., Nduba, V. N., Kariuki, S. M., Kimari, J., Bhatt, S. M. and Gilks, C. F., Trends in bloodstream infections among human immunodeficiency virus-infected adults admitted to a hospital in Nairobi, Kenya, during the last decade. *Clin Infect Dis,* 2001; 33: 248-56.
7. Ashenafi, M., The prevalence of *Salmonella*, *Shigella* and *Yersinia enterocolitica* in adult diarrhoea out-patients in some hospital of Addis Ababa.: *M.Sc thesis School of graduate studies*, Addis Ababa, 1983.
8. Asrat, D., Hathaway, A. and Ekwall, E., Studies on enteric campylobacteriosis in Tikur Anbessa and Ethio-Swedish children's hospital, Addis Ababa, Ethiopia. *Ethiop Med J*, 1999; 37: 71-84.
9. Awole, M., Evaluation of the significance of Widal test in the diagnosis of Typhoid fever in Tikur Anbessa and Jimma University Hospital and Awolia Health Center, Ethiopia, MSc thesis: *Department of Microbiology, Immunology and Parasitology*, Addis Ababa University, 2004.
10. Behravesh, C.B. and Heymann, D., "Salmonellosis," in Control of Communicable Diseases Manual, 19th Edition, published by American Public Health Association, 2008; 535-540.
11. Brenner, F. W., Villar, R. G., Angulo, F. J., Tauxe, R. and Swaminathan, B., Salmonella nomenclature. *J Clin Microbiol*, 2000; 38: 2465-7.
12. Burros, M. (March 8, 2006): "More Salmonella Is Reported in Chickens". The New York Times. Retrieved 2007-05-13.
13. Buzby, Jean, Roberts, and Tonya., "The Economics of Enteric Infections: Human Foodborne Disease Costs, Gastroenterology, May 2009; 136(6): 18, 51-62.
14. Cammie, F. and Miller, S., Salmonellosis. In E. Brauneald, A. Faucl, D. La[ser, S. Hauser, D. Longo, and J. Jameson (Eds): *Principle of internal*

medicine. 15th,ed, Churchil Living Stone, UK, 2000; 970-975.
15. Centers for Disease Control and Prevention (2010): *Salmonella*. Available online: http://www.cdc.gov/nczved/divisions/dfbmd/diseases/*salmonella*.
16. Centers for Disease Control and Prevention: "Reptile-Associated Salmonellosis --- Selected States, 1998--2002". 2011.
17. Chaudhry, R., Laxmi, B. V., Nisar, N., Ray, K. and Kumar, D., Standardisation of polymerase chain reaction for the detection of Salmonella typhi in typhoid fever. *J Clin Pathol*, 1997; 50: 437-9.
18. Cheesbrough, M., *District laboratory practice in tropical countries, 3rd ed.* Cambridge University press. United Kingdom, 2000.
19. Clerinx, J., Bogaerts, J., Taelman, H., Habyarimana, J. B., Nyirabareja, A., Ngendahayo, P. and Van de Perre, P., Chronic diarrhea among adults in Kigali, Rwanda: association with bacterial enteropathogens, rectocolonic inflammation, and human immunodeficiency virus infection. *Clin Infect Dis*, 1995; 21: 1282-4.
20. Communicable Disease and Control 2005 (2007): *Salmonella* Surveillance: Annual Summary: http://www.cdc.gov/ncidod/dbmd/phlisdata/salmtab/2005/Salmonella Introduction 2005.pdf.
21. Council for Agriculture, Science and Technology (CAST): "Foodborne Pathogens: Risks and Consequences: Task Force Report No.122," download at http://www.castscience.org/publications/index.cfm/foodborne_pathogens_risks_and_consequences?show=product&productID=2852, Sept. 1994; 1-87.
22. Crum-Cianflone, N.F., Salmonellosis and the gastrointestinal tract: more than just peanut butter. *Curr Gastroenterol Rep*., 2008; 10: 424-31.
23. Crump, J. A., Luby, S. P. and Mintz, E. D., The global burden of typhoid fever. *Bull World Health Organ*, 2004; 82: 346-53.
24. Dominguez, C., Gomez, I. and Zumalacarregui, J., Prevalence of *Salmonella* and *Campylobacter* in retail chicken meat in Spain. *International Journal of Food Microbiology*, January 30, 2002; 72(1-2): 165-8.
25. Dworkin, M.S., Shoemaker, P.C., Goldoft, M.J. and Kobayashi, J.M., "Reactive arthritis and Reiter's syndrome following an outbreak of gastroenteritis caused by Salmonella enteritidis". *Clin Infect Dis*, 2001; 33(7): 1010–14. doi:10.1086/322644. PMID 11528573.
26. Faris, K. and Kaba, M., Hygienic behavior and environmental conditions in Jimma town, Southwestern Ethiopia. *Ethiop J Health Sci*, 1999; 13: 77-86.
27. Foley, S.L. and Lynne, A.M., Food animal-associated Salmonella challenges: pathogenicity and antimicrobial resistance. *J Anim Sci*., 2008; 86: E173-87.
28. Galanis, E., Lo Fo Wong, D. M., Patrick, M. E., Binsztein, N., Cieslik, A., Chalermchikit, T., Aidara-Kane, A., Ellis, A., Angulo, F. J. and Wegener, H. C., Web-based surveillance and global Salmonella distribution, 2000-2002. *Emerg Infect Dis*, 2006; 12: 381-8.
29. Graham, S. M., Salmonellosis in children in developing and developed countries and populations. *Curr Opin Infect Dis*, 2002; 15: 507-12.
30. Haque, A., Ahmed, J. and Qureshi, J. A., Early detection of typhoid by polymerase chain reaction. *Ann Saudi Med*, 1999; 19: 337-40.
31. Hargrett-Bean, N.T., Pavia, A.T. and Tauxe, R.V., Tauxe. *Salmonella* isolates from humans in the United States, 1984.1986. *MMWR Morb Mortal Wkly Rep*, 1998; 37(Suppl 2): 25-31.
32. House, D., Wain, J., Ho, V. A., Diep, T. S., Chinh, N. T., Bay, P. V., Vinh, H., Duc, M., Parry, C. M., Dougan, G., White, N. J., Hien, T. T. and Farrar, J. J., Serology of typhoid fever in an area of endemicity and its relevance to diagnosis. *J Clin Microbiol*, 2001; 39: 1002-7.
33. Humphrey, T.J., Public health aspects of contamination of eggs and egg products with salmonellas. London. SCI, *SCI Lecture Papers* No.*0065*, 1995.
34. Jordan, R., van Heerden, E., Hugo, C. and Piater, L., Using current molecular techniques for rapid differentiation of *Salmonella* Typhi and *Salmonella* Typhimurium. *African Journal of Biotechnology*, 2009; 8: 1815-1818.
35. Kariuki, S., Revathi, G., Kariuki, N., Kiiru, J., Mwituria, J. and Hart, C. A., Characterisation of community acquired non-typhoidal Salmonella from bacteraemia and diarrhoeal infections in children admitted to hospital in Nairobi, Kenya. *BMC Microbiol*, 2006; 6: 101.
36. Khan, M., Coovadia, Y. M., Connoly, C. and Sturm, A. W., The early diagnosis of typhoid fever prior to the Widal test and bacteriological culture results. *Acta Trop*, 1998; 69: 165-73.
37. Kim, S., Frye, J. G., Fedorka-Cray, P. J., Gautom, R. and Boyle, D. S., Multiplex PCR-based method for identification of common clinical serotypes of Salmonella enterica subsp. enterica. *J Clin Microbiol*, 2006; 44: 3608-15.
38. Mache, A., *Salmonella* serogroup and their antibiotic resistance patterns isolated from diarrhoeal stools of pediatric out patients in Jimma Hospital and Jimma Health Center, South West Ethiopia. *Ethiop J Health Sci*, 2002; 37: 37-45.
39. Malkawi, H. I. and Gharaibeh, R., Multiplex PCR for the direct detection of Salmonella enterica from chicken, lamb and beef food products. *J Basic Microbiol*, 2003; 43: 328-36.
40. Maskey, A. P., Day, J. N., Phung, Q. T., Thwaites, G. E., Campbell, J. I., Zimmerman, M., Farrar, J. J. and Basnyat, B., Salmonella enterica serovar Paratyphi A and S. enterica serovar Typhi cause

indistinguishable clinical syndromes in Kathmandu, Nepal. *Clin Infect Dis*, 2006; 42: 1247-53.
41. Mastroeni, P., Mechanisms of immunity in *Salmonella* infection. In P. Mastroeni, and D. Maskell (Eds): *Salmonella infections: Clinical, Immunological and Molecular aspects*, Cambridge University press, Cambridge, 2006; 207-254.
42. Miller, S. and Pegues, D., "*Salmonella* Species, Including *Salmonella* Typhi," in Mandell, Douglas, and Bennett's PRINCIPLES AND PRACTICE OF INFECTIOUS DISEASES, Sixth Edition., 2005; 220: 2636-650.
43. Miller, S., Hohmann, E. and Pegues, D., *Salmonella* species, including *Salmonella typhi*. In G. Mandell, B. JE, and R. Dolin (Eds): *principles and Practice of infectious disease*, Chirchil Livingstone, New York, 2000; 2013-2033.
44. Murray, M.J., *Salmonella*: Virulence factors and enteric salmonellosis. *J Am Vet Med Assoc.*, 1986; 189: 145-147.
45. Ochiai, R. L., Wang, X., von Seidlein, L., Yang, J., Bhutta, Z. A., Bhattacharya, S. K., Agtini, M., Deen, J. L., Wain, J., Kim, D. R., Ali, M., Acosta, C. J., Jodar, L. and Clemens, J. D., Salmonella paratyphi A rates, Asia. *Emerg Infect Dis*, 2005; 11: 1764-6.
46. Olmos, M., Molina, C., Piskorz, E., Tawil, J., Magnanini, F., Casiro, A., Cahn, P., Concetti, H., Janeiro, M., Guelfand, L. and Kaufman, S., [Diarrhea and AIDS: more complex diagnostic techniques; better therapeutic results]. *Acta Gastroenterol Latinoam*, 1996; 26: 91-100.
47. Parry, C. M., Hien, T. T., Dougan, G., White, N. J. and Farrar, J. J., Typhoid fever. *N Engl J Med*, 2002; 347: 1770-82.
48. Patrick, M.E., "*Salmonella* Enteritidis infections, United States, 1985–1999," EMERGING INFECTIOUS DISEASES, Jan. 2004; 10(1). available online at http://wwwnc.cdc.gov/eid/article/10/1/02-0572_article.htm.
49. Porwollik, S., *Salmonella: From Genome to Function*. Caister Academic Press, 2011; ISBN 978-1-904455-73-8.
50. Prakash, P., Mishra, O. P., Singh, A. K., Gulati, A. K. and Nath, G., Evaluation of nested PCR in diagnosis of typhoid fever. *J Clin Microbiol*, 2005; 43: 431-2.
51. Ryan, K.J. and Ray, C.G., *Sherris Medical Microbiology* (4th ed.). McGraw Hill, 2004; 362–8. ISBN 0-8385-8529-9.
52. Sirinavin, S. and Garner, P., Antibiotics for treating *Salmonella* gut infections. *Cochrane Database Syst Rev*, 2000; CD001167.
53. Stevens, A., Joseph, C. and Bruce, J., A large outbreak of *Salmonella enteritidis* phage type 4 associated with eggs from overseas. *Epidemiol Infect*, 1989; 103: 425-433.
54. Strugnell, R. and Wijburg, O. L. C., Newer vaccines against typhoid fever and gastrointestinal Salmonellosis. In P. Mastroeni and D. Maskell (Eds): *Salmonella infections: Clinical, Immunological and Molecular Aspects*, Cambridge, 2006; 323-370.
55. WHO (2003): Background document: the diagnosis and treatment and prevention of typhoid., 1-33.
56. WHO Expert Committee on Salmonellosis Control, *Salmonellosis Control, The Role of Animal and Product Hygiene. Report of a WHO Expert Committee*. Geneva: WHO, (Technical Report Series 774), 1988.
57. Wolday, D. and Erge, W., Antimicrobial sensitivity pattern of *Salmonella*: comparison of isolates from HIV-infected and HIV-uninfected patients. *Trop Doct*, 1998; 28: 139-41.