

## REVIEW ON PUBLIC HEALTH IMPORTANCE OF BOVINE SALMONELLOSIS

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**SUMMARY:** Salmonellosis is the most common food-borne and zoonotic bacterial disease in the world. Salmonella is a significant pathogen for food-producing animals and these animals are the primary source of salmonellosis. The pathogen is spread by trade in animals and non-heated animal food products. Sporadic disease is more common than outbreaks. Salmonellae are able to breach the intestinal barrier via phagocytosis and trafficking by CD18-positive immune cells, which may be a mechanism key to typhoidal Salmonella infection. Microscopically, Hemorrhage, oedema, necrosis and leucocytic infiltration (mainly macrophages) are seen in the Mucosae of intestinal wall. The economic loss associated in human salmonellosis is due to investigation, treatment and prevention of illness. Diagnosis of salmonellosis depends on clinical signs and isolation of the pathogen from feces, blood, or tissues of affected animals. Salmonellosis affect animal of all stages but, the elderly, infants, and those with impaired immune systems are more likely to develop severe illness and show prominent clinical sign. The most common presentation includes fever, nausea, and diarrhea, cramping and sometimes vomiting. Antimicrobial therapy (or treatment with antibiotics) is not recommended for uncomplicated gastroenteritis. In contrast, antibiotics are recommended for persons at increased risk of invasive disease, including infants younger than 3 months of age. To prevent Salmonella one should use caution when handling and storing raw meat. Salmonella enteritidis and Salmonella typhimurium are the most important serotypes for salmonellosis transmitted from animals to human.

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### 1. INTRODUCTION

Salmonellosis is a disease caused by the bacteria salmonella. The genus Salmonella was named after Daniel E. Salmon who first reported the isolation of Salmonella from a pig in 1885 and named the organism Bacterium Choleraesuis (currently known as Salmonella enterica serovar Choleraesuis) (Rao, 2004). Salmonella species are Gram-negative, non-spore forming rod-shaped bacteria and are members of the family Enterobacteriaceae. Salmonella spp. are also classed as facultative anaerobic organisms as they do not require oxygen for growth (Jay *et al.*, 2003). The genus Salmonella is divided into two species: *S. enterica* (comprising six subspecies) and *S. bongori*. Over 99% of human Salmonella spp. infections are caused by *S. enterica subsp. Enterica*. (Bell and Kyriakides, 2002). The bacteria can have a combination of three antigens: the O antigen, H antigen, and VI antigen. The O antigen is located in the cell wall of the bacterium, and each salmonella bacillus may possess 2 or more O antigens on its surface. Also, the H antigen is a flagellar antigen that can be destroyed by heat and enables the motility of the Salmonella bacterium. The last antigen is known as the VI antigen because this

antigen is related to the virulence of the bacterium. As a capsular antigen, its presence enhances the virulence of the bacterium that has it of all the subspecies of Salmonella, only two, Salmonella enterica serovar (*S. Typhi* and *S. choleraesuis*), have the VI antigen (CDC, 2007).

Domestic and wild animals are the natural reservoir for all Salmonella serovars except for *Typhi* and *Paratyphi*, thus highlighting the zoonotic potential of this pathogen. Salmonella can be found in the gastrointestinal tract of a wide range of species, often without evidence of clinical disease: cattle, horses, pigs, dogs, cats, rodents, poultry, wild birds, reptiles, amphibians, and fish (Sanchez *et al.*, 2002). Some Salmonella serovars are host-adapted to individual animal species and may differ vastly in the severity of the disease they cause; others such as *S. Typhimurium* have a broad host range, with an ability to infect a wide range of animals, including humans (Jay *et al.*, 2003). Salmonella is a specialized invader of the gastrointestinal system that can cause a variety of painful conditions of varying severity. Salmonella causes gastroenteritis and typhoid fever and is one of the major foodborne pathogens of

significant public health concern (Fluit, 2005). Salmonella is considered as one of the most wide spread foodborne zoonosis in industrialized as well as developing countries even though the incidences seems to vary. Salmonellosis is a disease caused by many serotypes of Salmonella and characterized clinically by one or more of the three major syndromes; septicemia, acute and chronic enteritis (Davison *et al.*, 2005). There are more than 2500 different salmonella serotypes and all are considered potentially pathogenic to human (Popoff *et al.*, 2003). In human Salmonella is one of the most common causes of bacterial gastroenteritis. Death from Salmonella food poisoning estimated 1,000 deaths each year in USA (Mead *et al.*, 1999). Infection of humans and cattle with Salmonella enterica serotype *Typhimurium* results in an enteric disease characterized by diarrhea that is associated with massive infiltration of neutrophils in the mucosa (Santos *et al.*, 2001).

The economic loss associated in human salmonellosis is due to investigation, treatment and prevention of illness. The disease is usually spread by direct and indirect fecal–oral transmission and is a recognized as occupational risk among veterinary, animal care, and food processing personnel. Salmonella bacteria typically live in animal and human intestines and are shed through feces. Humans become infected most frequently through contaminated water or food. Typically, people with salmonella infection have no symptoms. Others develop diarrhea, fever and abdominal cramps within eight to 72 hours. Most healthy people recover within a few days without specific treatment (Hohmann, 2013).

Therefore, the objective of this review paper is:

- To highlight the general characteristics of salmonella spp.
- To indicate the overall course of bovine salmonellosis and
- To review the possible public health importance of bovine salmonellosis.

## 2. BOVINE SALMONELLOSIS

### 2.1 Definition and etiology

Salmonellosis is caused by the genus salmonella and an important zoonotic disease of world-wide significance. Salmonellosis is one of the major zoonotic diseases all over the world with annual estimates of 22 million cases and 200,000 deaths due to typhoid fever (Crump *et al.*, 2004) and 93.8 million cases of gastroenteritis and 155 000 deaths due to non-typhoidal Salmonellae (NTS)

(Majowicz *et al.*, 2010). In resource-poor settings of Africa, enteric fever is a public health concern with an incidence of 10-100/100,000 cases per year. Of the NTS, *S. Typhimurium* and *S. Enteritidis* are common (Gordon *et al.*, 2008). And in 2002 each accounted for approximately 25% of the human isolates (Galanis *et al.*, 2006).

Salmonella species are facultative intracellular pathogens. Taxonomically, the genus Salmonella which belongs to the Enterobacteriaceae family is divided into two species: *Salmonella bongori* and *Salmonella enterica*. Using the Kauffmann White Scheme, members of this genus are classified into more than 2500 serotypes based on differences between the O (cell wall) and H (flagella) antigens. Salmonella serotypes such as *S. enterica Typhimurium* and *S. Enteritidis* can infect humans as well as several animal species, others such as *S. Cholerae suis*, *S. Dublin*, and *S. Typhi* are restricted to fewer host species, primarily infecting pigs, cattle and humans, respectively (Kingsley & Baumler 2000; Parry *et al.*, 2002). In particular, food animals with subclinical infection constitute a vast reservoir for disease in humans (Davison, 2005).

### 2.2. Epidemiology

#### 2.2.1 Transmission

Transmission is a complex process involving components of both the pathogen and the host. Salmonella spp. are transmitted by the faecal-oral route by either consumption of contaminated food or water, person-to-person contact, or from direct contact with infected animals (Jay *et al.*, 2003). The epidemiology of Salmonella is complex which often makes control of disease difficult. However, the sources and transmission routes of Salmonella in developing countries are poorly understood due to the lack of coordinated national epidemiological surveillance systems (Acha and Szyfres, 2001).

In general, the primary sources of salmonellosis are considered to be food-producing animals such as cattle, poultry and swine (Thorns, 2000). The pathogens are mainly disseminated by trade in animals and uncooked animal food products (Gillespie *et al.*, 2000). Trade in contaminated animal feed products has also significantly contributed to the spread of *Salmonella*, and several large outbreaks in humans have been traced back to contaminated animal feed (Crump *et al.*, 2002). The process of removing the gastrointestinal tract during slaughtering of food animals is regarded as one of the most important sources of carcass and organ contamination with Salmonella at abattoirs

(Stopforth *et al.*, 2006). Epidemiological pattern of prevalence of infection and incidences of disease differ greatly between geographical areas depending on climate, population density, land use, farming practice, food harvesting and processing technologies and consumer habits. In addition, the biology of serovar differs so widely that *Salmonella* infection or *Salmonella* contamination are inevitably complex (Radostits *et al.*, 2007).

The majority of *Salmonella* isolations in the UK are associated with clinical disease as there is no routine surveillance for *Salmonella*. However, it has been found in other countries that many dairy farms may harbor *Salmonella*, either in the environment (up to 93% of herds tested) or as sub-clinical cases where shedding is from the cows themselves (10% of cows), thus, the farm environment should be viewed as an important potential reservoir of infection. Members of the genus *Salmonella* are often referred to as universal pathogens because of their ubiquitous presence in nature. *Salmonella* are recovered from almost all vertebrates and some insects. *Salmonella* infections in wild fauna, such as rodents, are usually secondary to the infection of farm animals, even though infection cycles may continue independently of any continuous input of *Salmonella* bacteria from farm animals. *Salmonella* are found in the intestinal tract of wild and domesticated animals and humans. Some serotypes of *Salmonella*, such as *S. Typhi* and *S. Paratyphi* are only found in humans (Behravesh *et al.*, 2008).

### 2.2.2. Species affected

There are two types of *Salmonella* bacteria which commonly affect cattle in the UK, these being *Salmonella Dublin* and *Salmonella Typhimurium*. In recent years, *S. Dublin* has become the most common while the incidence of *S. Typhimurium* has declined since the late 1990's. However, the true prevalence is unknown because the majority of *Salmonella* is found in animals with clinical disease and there is a lack of routine monitoring. The disease can affect all species of domestic animals; young animals and pregnant animals are the most susceptible. Some *Salmonella* serotypes can infect a broad range of domestic animals including poultry, sheep, cattle and pigs and cause symptoms of varying severity ranging from mild gastro-enteritis to death. Some of these serotypes, such as *S. Typhimurium* and *S. Enteritidis*, can infect humans. Other serotypes are host specific, infecting a single species and generally causing severe, typhoid-like symptoms sometimes

leading to death. *Salmonellae* have evolved into a diverse genus of *Enterobacteriaceae*; some members being adapted to specific hosts with others having a broad host range (Radostits *et al.*, 2007).

### 2.2.3. Incidence of salmonellosis

In the United States during 2008-2009, a multistate outbreak of *Salmonella Typhimurium* - linked to peanut butter resulted in more than 700 cases of illness (CDC, 2009). In 2009, over 40,000 cases of *Salmonella* (13.6 cases per 100,000 persons) were reported to the Centers for Disease Control and Prevention (CDC) by public health laboratories, representing a decrease of approximately 15% from the previous year, but a 4.2% increase since 1996 (CDC, 2008). Overall, the incidence of *Salmonella* in the United States has not significantly changed since 1996 (Miller and Pegues, 2005). *Salmonella* can be grouped into more than 2,400 serotypes. The two most common serotypes in the U.S. are *Salmonella Typhimurium* and *Salmonella Enteritidis*. *Salmonella Typhi*, the serotype that causes typhoid fever, is uncommon in the U.S. But, globally, typhoid fever continues to be a significant problem, with an estimated 12-33 million cases occurring annually (Miller and Pegues, 2005).

*Salmonellosis* is the second most common reported bacterial cause of infectious intestinal disease in the UK. It is estimated that over 60-80% of cases occur sporadically, though large outbreaks are not uncommon. *Salmonellae* spread readily by means of food, from zoonotic hosts and directly from person to person. The progressive intensification and mechanization of production, and globalization of distribution of our food supply, has meant that outbreaks of *Salmonella* can be very extensive, and their sources, deeply embedded (Hawker *et al.*, 2005).

### 2.2.5. Risk factor

Introducing animals that have been mixed with animals of unknown origin, especially in stressful environments such as saleyards, introducing animals to the main herd before there has been time for any sick animals to be identified, contact between susceptible animals and effluent on the home property or from neighbors, mixing aged animals with other animals or transporting them in dirty trucks, rodent infestations (Radostits *et al.*, 2007).

## 2.3. Pathogenesis

Salmonellae are able to breach the intestinal barrier via phagocytosis and trafficking by CD18-positive immune cells, which may be a mechanism key to typhoidal Salmonella infection. This is thought to be a more stealthy way of passing the intestinal barrier, and may, therefore, contribute to the fact that lower numbers of typhoidal Salmonella are required for infection than non typhoidal Salmonella. Salmonella cells are able to enter macrophages via macro pinocytosis. Typhoidal serovars can use this to achieve dissemination throughout the body via the mononuclear phagocyte system, a network of connective tissue that contains immune cells, and surrounds tissue associated with the immune system throughout the body. Pathogenic salmonellae ingested in food survive passage through the gastric acid barrier and invade the mucosa of the small and large intestine and produce toxins (Haraga *et al.*, 2008).

Host and bacterial factors underlying Salmonella-induced enteritis (Layton & Galyov, 2007). As with invasion, structural components of T3SS-1 are required for induction of intestinal secretory and inflammatory responses in ligated ileal loops calves (Watson *et al.*, 1998) and pigs (Boyen *et al.*, 2006b; Paulin *et al.*, 2007). However, it should be noted that SPI-1 mutants can still elicit inflammatory responses when incubated in ileal anastomoses for a longer duration (Coombes *et al.*, 2005a), indicating that SPI-1-independent mechanisms exist. The extent to which invasion per se is required for enteritis, as opposed to the T3SS-1-mediated injection of effectors by luminal bacteria, remains unclear. Salmonella spp. possesses a number of structural and physiological virulence factors, enabling them to cause acute and chronic disease in humans. The virulence of Salmonella spp. varies with the length and structure of the O side chains of lipopolysaccharide molecules at the surface of the bacterial cell. Resistance of Salmonella spp. to the lytic action of complement (part of the immune response) is directly related to the length of the O side chain (Jay *et al.*, 2003)

Other important virulence factors include the presence and type of fimbriae, which is related to the ability of Salmonella spp. to attach to host epithelium cells, as well as the expression of genes responsible for invasion into cells (Jones, 2005). As *Salmonella typhimurium* invades the mucosal lining, the host inflammatory response is activated. Salmonella typhimurium uses the type III secretion system to invade the epithelial cells in the intestines. To initiate disease in the gastrointestinal tract,

salmonellae must adapt to the hypoxic, acidic, and alkaline environments that they encounter en route from the stomach to the small intestine. *Salmonella* spp. produce a heat labile enterotoxin, resulting in the loss of intestinal fluids (Causing diarrhea). This enterotoxin is closely related functionally, immunologically and genetically to the toxin of *Vibrio cholera* and the heat labile toxin of pathogenic *Escherichia coli*. Most *Salmonella* strains also produce heat labile cytotoxin which may cause damage to the intestinal mucosal surface and results in general enteric symptoms and inflammation. Infection with non-typhoidal *Salmonella* is generally limited to a localized intestinal event. However, the presence of virulence plasmids has been associated with non-typhoidal *Salmonella* spp. surviving in phagocytes and spreading from the small intestine to the spleen and liver (Jay *et al.*, 2003).

#### 2.2.4. Occurrence

Sporadic disease is more common than outbreaks. But widespread outbreaks in the community, restaurants, health care institutions and nursing homes have been reported (WHO, 2005). Only a small proportion of cases are recognized clinically. The incidence of nontyphoidal *Salmonella* infection is highest during the rainy season in the tropical climates and during the warmer months in temperate climates, coinciding with the peak in foodborne outbreaks (Heymann, 2008).

#### 2.4. Clinical sign

In humans, salmonellosis varies from a self-limiting gastroenteritis to septicemia. Whether the organism remains in the intestine or disseminates depends on host factors as well as the virulence of the strain. Asymptomatic infections can also be seen. The incubation period ranges from 6 to 72 hours (usually 12-36 hours), and is dependent on size of the inoculum and host factors (WHO, 2005). Salmonellosis cannot be reliably distinguished clinically from other enteric bacterial infections. The most common presentation includes fever, nausea, and diarrhea, cramping and sometimes vomiting. Stools are usually non-bloody. Other symptoms include chills, myalgia, and headache. The disease is usually self-limiting with recovery within three to seven days. Hospitalization may be required (22% of cases) in cases with severe diarrhoea and dehydration. On average, patients may carry Salmonella in their intestinal tract for around four weeks after acute infections. This period may be as long as seven weeks for infants and children. In

about 0.5% of salmonellosis cases, the patient becomes a chronic carrier. The elderly, infants, and those with impaired immune systems are more likely to develop severe illness (Jones *et al.*, 2008).

Clinically, Salmonella infection in cattle is typically manifested as watery or bloody diarrhea, and often associated with fever, depression, anorexia, dehydration and endotoxemia. On the other hand Salmonella spp. can be localized into the gallbladder of asymptomatic ruminants (Van den Bogaard and Stobberingh, 2000). There are three major forms of salmonellosis in animals: enteritis, septicemia and abortion, but all combinations of the three forms have been observed in susceptible animals. Enteritis can have acute, sub-acute or chronic forms. Acute enteritis is the common form in adult animals, but can also occur in calves under 1 week of age. In affected animals, there is fever (40.5-41.5°C) initially, followed by severe diarrhoea, sometimes dysentery and often tenesmus. Subacute enteritis can occur in adult sheep and cattle. The main clinical signs include fever (39-40°C), soft faeces and dehydration. A high incidence of abortion is common in cows and ewes with subacute enteritis. Chronic enteritis is common in adult cattle and pigs. Affected animals have persistent diarrhoea, intermittent fever and are emaciated. Salmonella infections, also known as salmonellosis, come in a variety of forms, each with different sets of symptoms and severities. The most common, gastroenteritis, is associated with at least 150 serotypes and is characterized by nausea, vomiting, abdominal cramping, and diarrhea. Enteric fevers such as Typhoid include fever, anorexia, lethargy, and constipation. These are more severe than gastroenteritis, and may result in death, however, enteric are typically associated most often with humans. (Van den Bogaard and Stobberingh, 2000).

### 2.5. Pathology

The main lesions are: Septicemia and enterocolitis, Presence of mucoenteritis in ileum and colon. The mucosae of the intestine is hyperemic or hemorrhagic. Enlarged mesenteric lymph nodes and hemorrhagic necrotic enteritis are also seen in the infected animals. The mucosae are covered with red yellow or grey exudate and even ulcers are formed in the intestines. Microscopically, Hemorrhage, oedema, necrosis and leucocytic infiltration (mainly macrophages) are seen in the mucosae of intestinal wall. In the livers, there are foci of necrosis and formation of paratyphoid nodules which consist of aggregates of reticuloendothelial cells like histiocytes

or macrophages etc. The Kupffer cells are prominent and leucocytes are present in the sinusoids. There is reticuloendothelial hyperplasia in lymph nodes and spleen. Haemorrhage and necrosis are seen in mesenteric lymph nodes. Septicaemia in the animals is marked by petechiae, ecchymoses on the pleura, peritoneum, endocardium, kidneys and meninges. Microscopically, there is fibrinoid necrosis of the vessel walls and hyaline material is deposited in the glomerular capillaries and small vessels of the dermis. Petechial hemorrhages in kidneys, congestion and hepatisation of lungs, skin discoloration (e.g., as seen in pigs), hemorrhagic enteritis, putrid odors of the intestinal contents, diphtheritic pseudo membrane, enlarged mesenteric lymph nodes and thickened wall of gall-bladder are some important changes in salmonellosis (Singh, 2008).

### 2.6. Diagnosis and Differential Diagnosis

Salmonella infection is diagnosed through testing of a stool sample. Salmonella can sometimes also be isolated from blood, urine or tissue samples. (Tauxe, 1997) in cases of bacteremia or invasive illness, the bacteria can also be detected in the blood, urine, or on rare occasions in tissues. To provide scientifically and statistically valid results the specimens collected must be appropriate for the intended purpose, and adequate in quality, volume, and number for the proposed testing (Miller and Pegues, 2005).

The clinical sign and finding at postmortem examination are not unique to salmonellosis although a tentative diagnosis may be made; but, it should be differentiated from colibacillosis, and cryptosporidium. They should confirm either in diseased animal or at necropsy by isolation of organisms in their feces and determination of viable counts. Fecal samples rather than swabs should be taken and these should obviously be obtained before administration of antibiotics. It may be also possible to isolate organism from oral secretion and by blood culture although these are less reliable than feces culture and must be taken with care to avoid contamination. Animal that died of salmonellosis usually have large number of Salmonella distributed throughout their tissue and sample of spleen, liver, hepatic, mediastinal and bronchial lymph nodes may yield count in excretion of 10<sup>6</sup> organisms/gram. Similar concentration may be present in the wall and content of the ileum, cecum, colon and associated lymph nodes. Sample should be taken from internal organs in order to distinguish animal that have died of enteritis without septicemia (Jones *et al.*, 2008)

## Rapid detection methods

**Detection of antibodies to salmonella by enzyme immuno assay (EIA):** The detection of antibodies to Salmonella by EIA offers a sensitive and cost-effective method for mass screening of animal flocks/herds for indications of a past/present Salmonella infection. The limitation of the method is that the immune response of the individual animal is not elicited before 1-2 weeks after infection takes place. A number of commercial kits are available for testing poultry, cattle and pigs. (ArunK, 2008).

**Nucleic acid-based assays:** Real-time quantitative polymerase chain reaction using PCR (Q-PCR), reverse transcriptase PCR (RTPCR), and nucleic acid sequence-based amplification (NASBA) have been used for detection of Salmonella from various food matrices. Salmonella enterica was detected at 1 cfu ml<sup>-1</sup> after a culture enrichment of 8-12 h in the TaqMan-based Q-PCR using *invA* gene as target. (ArunK, 2008).

### 2.7. Treatment

Salmonella infections usually resolve in 3 to 7 days, and many times require no treatment. Persons with severe diarrhea may require rehydration, often with intravenous fluids. Antimicrobial therapy (or treatment with antibiotics) is not recommended for uncomplicated gastroenteritis. In contrast, antibiotics are recommended for persons at increased risk of invasive disease, including infants younger than 3 months of age (Miller and Pegues, 2005). In situations in which antibiotics are needed, trimethoprim/sulfamethoxazole, ampicillin, or amoxicillin, are the best choices. Ceftriaxone, cefotaxime, or fluoroquinolones are effective options for antimicrobial-resistant strains, although fluoroquinolones are not approved for persons less than 18 years of age. For persons with an infection in a specific organ or tissue (invasive disease), treatment with an expanded-spectrum cephalosporin is recommended, until it is known if the bacteria is susceptible to one of the more commonly used antibiotics listed above. For these rare situations, treatment with antibiotics for 4 weeks is generally recommended (Behraves, *et al.*, 2008).

In animal treatment supportive treatment with intravenous fluid is necessary for patients that have anorexia, depression, significant dehydration. Individual patient may be treated aggressively following acid base and electrolyte assessment. Oral fluid and electrolyte may be somewhat helpful and

much cheaper than IV fluid for cattle demand to be mildly or moderately dehydrated. The effectiveness of oral fluid may be somewhat compromised by malabsorption and maldigestion in salmonellosis patient but still should be considered useful. Cattle that are willing to drink can have specific electrolyte (NaCl, KCl) added to drinking water to help correcting electrolyte (Rebhun, 1995). The implementation of broad prophylactic strategies that are efficacious for all Salmonellae may be required in order to overcome the diversity of Salmonella serovars present on farms, and the potential for different serovars to possess different virulence factors (Mohler *et al.*, 2009). Early treatment is essential for septicemic salmonellosis but there is controversy regarding the use of antimicrobial agent for intestinal salmonellosis. Oral antibiotic may alter the intestinal micro flora and interfere with competitive antagonism and prolong shading of the organism. There is also a concern that antibiotic resistance strain of Salmonella selected by oral antibiotic may subsequently infect human. Antibiotic such as ampicillin or cephalosporin lead to lyses of bacteria with release of endotoxin. NSAID may be used to reduce the effect of endotoxemia (Davison, 2005).

### 2.8. Prevention and Control

There is no vaccine to prevent Salmonella typhimurium. Salmonella typhimurium can be traced to many dairy, poultry, and meat products. However, the most common sources where Salmonella is found are eggs and chickens. Salmonella can also be acquired through water and foods that have been contaminated with feces. To prevent Salmonella one should use caution when handling and storing raw meat. Animals with diarrhea should be isolated and the area disinfected. Meat and eggs should be adequately cooked and proper food handling hygiene should be used. Always wash hands after touching or working with animals. Make sure to keep the raw chicken separated from all other meats. Also, preparation tops, cutting boards, and hands should be thoroughly washed before handling or preparing other meats to prevent cross contamination. One should also use caution when cooking eggs and meat to ensure they are cooked thoroughly and to a safe internal temperature for human consumption. Additionally, water should come from regulated sources or be filtered to make sure it is free of Salmonella bacteria. Reptiles (especially small turtles) and birds are notorious for carrying Salmonella on their skin. Children, the elderly, and immunocompromised individuals should stay away

from these animals to protect themselves from contracting the Salmonella bacterium. (CDC, 2009).

## 2.9. Economic importance

Salmonella infections in dairy calves have many impacts on animal and human health that are considered as a major worldwide problem. Substantial economic losses were manifested through mortality and poor growth of infected animals as well as the potential of zoonotic transmission (Smith *et al.*, 2004). Members of the genus Salmonella remain a potential threat to human and animal health. Salmonella outbreaks in humans often follow consumption of contaminated animal products. Contamination usually results from infection of animals used in food production, or from contamination of the carcasses of infected or carrier animals. Pigs and poultry are mostly responsible for these outbreaks, while cattle and sheep are less incriminated. Bovine salmonellosis represents a major problem in view of the economic and health consequences. Severe financial losses result from the death of young animals, decreased milk and meat production, expensive testing and control programs, and the reduced value of contaminated products (Joseph *et al.*, 1998).

Acute salmonellosis in adults is manifested by fever, dullness, loss of appetite and reduced milk yield, while abortion in pregnant animals is often caused by *S. Dublin*. Severe dysentery may also occur and mortality may be as high as 70% if the condition is not treated. Salmonella infections in cattle originate from various sources, such as importation or local purchase of infected animals, contamination of feed and water, and cross-infection from other domestic or free-living animals. Carrier animals shed salmonellae in their faeces, thereby contaminating calf pens, yards, calving pens and milk sheds. Approximately 40% of all agricultural output on Earth is associated with raising livestock, and approximately 20% of the world's population (1.3 billion people) was employed in this sector as of 2006. In the U.S., there are currently greater than 75,000 operators just in Dairy, and more than 5,000 food animal vets employed by the industry (Cummings *et al.*, 2012).

## 3. PUBLIC HEALTH IMPORTANCE

Salmonellosis is a common zoonotic and foodborne disease of humans. *Salmonellae* cause disease in both humans and animals. Salmonella serovars can be divided into two main groups typhoidal and

nontyphoidal Salmonella. Nontyphoidal serovars are more common, and usually cause self-limiting gastrointestinal disease. They can infect a range of animals, and are zoonotic, meaning they can be transferred between humans and other animals. Typhoidal serovars include Salmonella Typhi and Salmonella Paratyphi A, which are adapted to humans and do not occur in other animals the serovar *S. Typhi* and most *S. Paratyphi* strains (A, B and C), which cause serious systemic infections in humans, are specific human pathogens. And these pathogens have no animal reservoir. The remaining serovars, usually known as the 'zoonotic *Salmonella* spp.', which cause so-called non-typhoidal salmonellosis in humans and sometimes also in animals (WHO, 2001).

A group of more frequently isolated serovars, such as *S. Typhimurium*, *S. Enteritidis*, *S. Hadar* and *S. Infantis* (among others), readily affect both humans and animals. *Salmonella enteritidis* and *Salmonella typhimurium* are the most important serotypes for salmonellosis transmitted from animals to human. In food animals, these serovars manifest themselves clinically through per-acute Septicaemia, acute enteritis or chronic enteritis. In the subclinical form of the disease, the animal may either have a latent infection or become a temporary or persistent carrier (Quinn *et al.*, 2002). Any serovar is considered capable of causing gastrointestinal illness of varying severity in humans. In a global survey covering the years 1990 and 1995, *S. Enteritidis* and *S. Typhimurium* were the two most frequently isolated serovars among human isolates (Herikstad *et al.*, 2002). These serovars were also the most frequently found in human outbreaks of salmonellosis in Europe in the period 1993 to 1998, being responsible for 77.1% of the recorded outbreaks and occurring in a ratio of approximately 3:1 (WHO, 2001).

The specific agents of salmonellosis in humans (*Salmonella Typhi*, *Paratyphi*, and *Sendai*) are the agents of typhoid and paratyphoid fevers. Worldwide, the human deaths caused by Typhoid fever are estimated at 600,000 per year (Hu and Kopecko, 2003). In Africa, non-typhoidal Salmonella has consistently been reported as a leading cause of bacteremia among immunocompromised people, infants and newborns (Bryce *et al.*, 2005). In the United States, Salmonella infection is estimated to occur in more than 1.4 million individuals each year. *Salmonella* spp are one of the major bacterial causes of foodborne gastroenteritis. The CDC report approximately

40,000 confirmed cases of salmonellosis annually. Many infections are due to ingestion of contaminated

food. People acquire Salmonella from undercooked contaminated meat, infected eggs, or unpasteurized milk products. If hands are not washed after direct contact with infected feces, then accidental ingestion of bacteria can occur (Andrew *et al.*, 2008). Humans may develop diarrhea, abdominal cramping, and fever, which can be very severe.

#### 4. CONCLUSION AND RECOMMENDATION

Salmonellosis is the most common food-borne and zoonotic bacterial disease in the world. Members of the genus Salmonella are major enteric pathogens of human beings and animals. The increase of the human population and food production increases the potential for dissemination of these ubiquitous microorganisms. The high incidence of human nontyphoid salmonellosis demonstrates the potential harm of the presence of Salmonella in food and contact animals to people. Treatment of salmonellosis by antibiotics may not be successful or the bacteria may develop resistance for the antibiotics and there is no effective vaccination until now. In most food producing animals, Salmonella infection in cattle is mostly subclinical, although clinical disease such as enteritis, Septicaemia or abortion can occur. Subclinical infection can be of variable duration including a carrier state and can play an important role in the spread of Salmonella within and between herds and pose a public health risk. This means the owner of the animal cannot look for any symptom in the case of subclinical salmonellosis. This in turn poses great economic and public problems. Finally, implementing basic and applied research to the agent that cause foodborne salmonellosis will be a crucial point for new approaches to prevent and control the disease.

Based on the above conclusion the following recommendation are forwarded:

- Washing hands with soap after handling animals
  - All meat should be cooked to appropriate internal temperatures.
  - Researches should be done on the vaccination since it is not efficient enough
  - During laboratory examination Specimens must be collected using appropriate biosafety and containment measures in order to prevent contamination of the environment, animal handlers,
- The control of Salmonella at pre-harvest level should be focus on preventive action because there is no 'silver bullet' through which the level of Salmonella contamination can be simply reduced.
  - Collaboration between government agencies, professional organizations and special interest groups.

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#### 5. REFERENCES

1. Acha, P.N. and Szyfres, B., 2001. Salmonellosis: Zoonosis and communicable diseases common to man and animals. Volume I: *Bacterioses and Mycoses*. 3rd ed. Washington, DC: pan American health organization. Pp.233-246.
2. Andrews, R. W., Blowey, H., Boyd. And Eddy, R.G. 2008. Bovine Medicine: Diseases and Husbandry of Cattle. 2<sup>nd</sup> ed. John Wiley & Sons.
3. Behravesh, C.B., 2008. Salmonellosis, in control of communicable diseases manual. 19th ed. American public health association, Pp. 535-540.
4. Bell, C. and Kyriakides, A., 2002. Salmonella - a practical approach to the organism and its control in foods. Blackwell science, oxford.
5. Boyen, F., Pasmans, F., Van Immerseel, F., Morgan, E., Adriaensen, C., Hernalsteens, J.-P., Decostere, A., Ducatelle, R. and Haesebrouck, F., 2006b. Salmonella typhimurium spi-1 genes promote intestinal but not tonsillar colonization in pigs. *Microbes infect*, 8: 2899–2907.
6. Bryce, J., Boschi-Pinto, C., Shibuya, K. and Back, R.E., 2005. Who estimates of the causes of death in children? *Lancet*, 365, Pp. 1147-1152.
7. CDC, 2008 Preliminary food net data on the incidence of infection with pathogens transmitted commonly through food—10 states, 2008, *morbidity and mortality weekly report*, 58: 333-337.

8. CDC. 2009: Multistate outbreak of salmonella infections associated with peanut butter and peanut butter-containing products--United States, 2008-2009. *Mmwr morb mortal wkly rep*, 58(4):85 - 90.
9. Crump, J., Luby, S., and Mintz, E.D., 2004. The global burden of typhoid fever. *Bull world health organ*: 82, Pp. 346-353.pubmed central.
10. Crump, J.A., Griffin, P.M. and Angulo, F.J., 2002. Bacterial contamination of animal feed and its relationship to human foodborne illness. *Clin. Infect. dis.*, 35 (7), 859-865. Epub.5 September 2002.
11. Davison, S., 2005. Salmonellosis. In: Merck veterinary manual. 10th edition. Edited by Cynthia, M. Kahn. Merck and Co.J (Inc. white house station, n.j u.s.a.
12. Fluit, A., 2005. Towards more virulent and antibiotic-resistant salmonella? *Fems immunol med microbiol*43: 1-11.
13. Galanis, E., Wong, D.M., Patrick, M.E., Binsztein, N., Cieslik, A., Chalermchikit, T., Aidara-Kane, A., Ellis, A., Angulo, F.J. and Wegener, H.C.,2006. Web-based surveillance and global salmonella distribution, 2000–2002. *Emerg infect dis*.12: 381-388. 10.3201/eid1205.050854.
14. Gillespie, I.A., O'Brien, S.J., Adak, G.K., Ward, L.R. and Smith, H.R., 2000. Foodborne general outbreaks of salmonella enteritidis phage type 4 infection, England and wales, 1992–2002: where are the risks?*Epidemiol infect*. 133:Pp. 795-801.
15. Gordon, M., Graham, S., Walsh, A., Wilson, L., Phiri, A., Molyneux, E., Zijlstra, E., Heyderman, R., Hart, C.A. and Molyneux, M.E.,2008. Epidemics of invasive salmonella enterica serovar enteritidis and S. enterica serovar typhimurium infection associated with multidrug resistance among adults and children in Malawi. *Clin infect dis.*, 46, Pp. 963-969. 10.1086/529146
17. Haraga, Andrea; Maikke, B. Ohlson, Samuel I. Miller., 2008. Salmonellae interplay with host cells. *Nature reviews microbiology*. 6, Pp. 53–66. Doi: 10.1038/nrmicro1788
18. Hawker, J., Begg, N., Blair, I., Reintjes, R., Weinberg, J., 2005. Communicable disease control handbook, Blackwell.
19. Heymann, D.L., 2008. Salmonellosis. In: Control of communicable diseases manual. 19th ed. American public health association, Washington, Pp.534-540.
20. Hohmann, E., 2013. Approach to the patient with nontyphoidal salmonella in a stool culture. <http://www.uptodate.com/home/>. Accessed Dec. 9.
21. Hu L., Kopecko, D., 2003. Typhoid Salmonella. In: Miliotis N., Bier j. (eds.), international Society for Microbiology.
22. Jay, L., Davos, D., Dundas, M., Frankish, E. and Lightfoot, D., 2003. Salmonella. Ch. 8 in: hocking
23. Jones, B. 2005. Salmonella invasion gene regulation: a story of environmental awareness.
24. Jones, T., Ingram, L. and Cieslak, P.,2008. Salmonellosis outcomes differ substantially by serotype. *J infect dis*. Jul 1; 198(1):Pp. 109-114.
25. Kingsley, R. and Bäumlner, A.,2000. Host adaptation and the emergence of infectious disease: the salmonella paradigm. *Molecular microbiology* 36 (5), Pp.1006.
26. Layton, A. and Galyov, E., 2007. Salmonella-induced enteritis: molecular pathogenesis and therapeutic implications. *Exp. rev. mol. med*. 9, Pp. 1–11.
27. Majowicz, S., Musto, J., Scallan, E., Angulo, F., Kirk, M., O'Brien, S., Jones, T., A, Hoekstra, R., 2010. The global burden of Nontyphoidal salmonella gastroenteritis. *Clin infect dis*. 50: Pp. 882-889. 10.1086/650733.
28. Mead, P., Slutsker, L., Dietz, V., Mccaig, L., Bresee, J. 1999. Food-related illness and death in the United States. *Emerge infect dis*5, Pp. 607-625.
29. Miller, S. and Pegues, D., 2005. “Salmonella species, including salmonella Typhi,” in Mandell, Douglas, and Bennett’s principles and practice of infectious diseases, 6th ed, chap. 220, Pp. 2636-2650.
30. Parry, CM, Hien, T., Dougan, G., White, N.J., 2002. Farrar JJ. Typhoid fever. TheNew England
31. Paulin, S., M., Watson, P. R., Benmore A. R., Stevens M. P., Jones P. W., Villarreal-Ramos, B., Wallis T. S., 2002. Analysis of salmonella enterica serotype-host specificity in calves: a virulence of S. enterica serotype Gallinarum correlates with bacterial dissemination from mesenteric lymph nodes and persistence in vivo. *Infect. immun*. 70, Pp. 6788–6797.
32. Pegues, DA., Miller, S., 2010Salmonella Species, including Salmonella Typhi. In: Mandell GL, Bennett JE, Dolin R (Eds). Mandell, Douglas and Bennett’s Principles and Practices of Infectious Diseases, 7th ed. Elsevier, Philadelphia, Pp. 2887–2903.

33. Popoff, M., Bockemühl, J., Gheesling, L., 2003. Supplement 2001 (no. 45) to the Kauffmann-white scheme. *Res microbiol* 154: Pp. 173-174.
34. Radostitis, O., Gay, C., Hinchliff, K., Constable, P., 2007. *Veterinary medicine: a text book of the disease of cattle, horses, sheep, pigs, and goats*. 10th ed. Elsevier Ltd: 325-326.
35. Rao, P., 2004. *Essential of microbiology*. Satish Kumar Jain for Cbs publishers and distributors, New Delhi, India: Pp. 146-148.
36. Santos, R., Zhang, S., Tsolis, R., Kingsley, R., Adams, L., Baumler, J., 2001. Animal models of salmonella infections: enteritis versus typhoid fever. *Microbes infect*; 3: Pp. 1335-44.
37. Smith, K. E., Stenzel, S. A., Bender, J. B., wagstrom, E., Soderlund, D., Leano, F. T. 2004.
38. Stopforth, J.D., Lopes, M., Shultz, J., Miksch, R., Samadpour, M., 2006. Location of bung bagging during beef slaughter influences the potential for spreading pathogen contamination on beef carcasses. *J food Prot.* 69: Pp. 1452-1455.
39. Tauxe, R., 1997. "Emerging foodborne diseases: an evolving public health challenge." *emerging infectious diseases*, 3, no. 4, Pp. 425-34 at <http://www.ncbi.nlm.nih.gov/pmc/articles/pmc2640074/pdf/9366593.pdf>
40. Thorns, C.J., 2000. Bacterial food-borne zoonosis. *Rev Sci tech.* 19: Pp. 226-239.
41. VAN den Bogaard, A.E., Stobberingh, E., 2000. Epidemiology of resistance to antibiotics. Links between animals and humans. *Int J antimicrob agents.* (4): Pp. 327-335.
42. Watson, P. R., Gautier, A. V., Paulin, S. M., 2000a. Salmonella enterica serovars typhimurium and Dublin can lyse macrophages by a mechanism distinct from apoptosis. *Infect. immun.* 68, Pp. 3744-3747.
43. World health organization. Drug-resistant salmonella fact sheet number 139. 2005; <http://www.who.int/mediacentre/factsheets/fs139/en/>. Accessed 2011-09-19.
44. Quinn P.J., Markey B.K., Carter M.E., Donnelly W.J. & Leonard F.C., 2002. *Veterinary microbiology and microbial disease*. Blackwell Science, Oxford.
45. Herikstad H., Motarjemi Y. & Tauxe R.V., 2002. Salmonella surveillance: a global survey of public health serotyping. *Epidemiol. Infect.* 129(1), Pp. 1-8.
46. World Health Organization (WHO). 2001. WHO Surveillance Programme for control of foodborne infections and intoxications in Europe, 7th Report 1993-1998 (K. Schmidt & C. Tirado, Eds). Federal Institute for Health Protection of Consumers and Veterinary Medicine, Berlin.
47. Rebhun C.W., 1995. *Disease of Dairy cattle*: 1st Edition. Awa Verly Company.
48. Mohler, V.L., Izzo, M.M., House, J.K., 2009. Salmonella in calves. *Vet Clin North Am Food Anim Pract* 25, Pp. 37-54.
49. Jones, P.J., Weston, P.R., Swail, T., 2007. Salmonellosis In: *Bovine medicine, diseases and husbandry of cattle*. Edited by Andrew, A.H. 2nd ed: Blackwell
50. ArunK, B., 2008. Mechanisms and Pathogenesis In: *Foodborne Microbial Pathogens*: pp. 201-215.

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