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Salmonellosis: A Review

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Abstract

Salmonellosis is one of the significant causes of economic loss in farm animals because of the cost of clinical disease which include deaths, diagnosis and treatment of clinical cases, diagnosis laboratory costs, the cost of cleaning and disinfection, and the cost of control and prevention. The loss incurred by the livestock producers includes feed efficiency, and reduced weight gains or deaths because of Salmonellosis. Human foodborne *Salmonellosis* constitutes a major health problem in many countries. Moreover the costs associated with *Salmonellosis* could be considerable. Financial costs are not only associated with investigation, treatment and prevent ion of human illness but may affect the chain of production. Poultry products have always topped the incidence of *Salmonellosis* in many developing countries including India, Egypt, Brazil and Zimbabwe and is the most seriously perceived food risks in chicken meat, even in the developed countries. *Salmonella* Enteritidis is transmitted to the human food supply through eggs of hens that appear healthy. Contamination with *Salmonella* in poultry products can occur at multiple steps along the food chain, which includes production, processing, distribution, retail marketing, handling and preparation.

Keywords: Antimicrobial, Environmental, *Salmonella*

Introduction

Salmonellosis is one of the major food borne diseases in the world and it is estimated that 93.8 million cases of gastroenteritis due to *Salmonella* species occur globally each year, with 155,000 deaths. Ethiopia has about 56.87 million chickens comprising mainly indigenous chickens, with the majority (95%) kept in low-input low-out-put village chicken production systems (Duguma, 2009; CSA, 2015). Food safety remains a critical issue with outbreaks of food borne illness resulting in substantial costs to individuals, the food industry and the economy. Despite advances in food science and technology, food borne diseases remain one of the major public health and economic problems all over the world (Legnani *et al*., 2004). The risk of food borne illness has increased markedly over the last 20 years, with nearly a quarter of the population at higher risk for illness (CDC, 2003; 2004).

Salmonella enterica sero var Enteritidis is considered the major cause of human *Salmonellosis* outbreaks in the United States and Europe (Collard *et al*., 2008 and Gould *et al*.,2013), linked mainly to consumption of contaminated poultry products, including eggs (Braden 2006 and Much *et al*., 2009). Live poultry are considered the main reservoir for *Salmonellae*; the microorganism is present in the intestinal tract, skin, and feathers of living birds. Bacterial contamination of poultry carcasses and cuts are a result of improper hygienic measures, improper cooking, and abuse of temperature. The dissemination of infection throughout plants during processing occurs in the evisceration, cooling, packaging, and transport stages (Zhang *et al*., 2013). Based on food standards, the presence of *Salmonellae* in foodstuff makes food unsafe for human consumption. The most serious form of *Salmonellosis* for humans is typhoid fever, which is still a major problem in developing countries, mainly due to the lack of sanitation and hygiene standards (Dougan *et al*., 2011). The disease is transmitted mainly via foodstuff and water contaminated with the pathogen, and it affects more than 90 million people worldwide yearly, with variable morbidity and mortality rates. In poultry, the severity of the infection depends on many factors, including the strain of *Salmonella*, the standard of hygiene, age of the bird, route of infection, and immune status of the bird (Chao *et al*., 2007).

Although primarily intestinal bacteria, *Salmonella* are widespread in the environment and commonly found in farm effluents, human sewage, and in any material subject to fecal contamination. *Salmonellosis* has been recognized in all countries but appears to be most prevalent in areas of intensive animal husbandry (Wray and Davies, 2003).

Although most of the infections in humans cause mild gastroenteritis, life-threatening systemic infections are common especially among high risk categories (Raufu, 2013). Invasive non typhoidal *Salmonella* commonly cause infection among infants, children, elderly and immunocompromised individuals worldwide and especially in African countries, where these diseases are driven in part by co-infection with malaria or human immunodeficiency virus (HIV) (AoTT , 2015). Sources and modes of transmission of non typhoidal*Salmonella* are still poorly understood in Africa due to the lack of coordinated national epidemiological surveillance systems (Kagambèga, 2013).

In food-producing animals and especially in poultry, *Salmonella* is one of the leading causes of infection, and this has a direct impact on the global marketing of the respective food-producing animals and animal-derived food products (Magwedere, 2015). In early life, *S*. Pullorum causes extremely high mortality of both broilers and commercial laying birds. Similarly, older birds succumb heavily to other serovars of *Salmonella* and it is assumed that *Salmonella* infections of this category of birds are mainly due to *S*. Gallinarum (Mamman, 2014).In addition to these host adapted serovars causing systemic disease, poultry harbor in their gastrointestinal tracts zoonotic serovars with no apparent signs of illness. Hence, these *Salmonella* serovars can be present in feaces excreted by healthy animals and may be transferred to raw foods of animal origin through contamination during slaughtering and processing (Sanchez, 2002). Generally, *Salmonella* in food producing animals, including poultry, manifests as long periods of latent carriage with occasional faecal shedding, which is the leading source of contamination of feed, water and environment (Magwedere, 2015)

Foods of animal origin, especially poultry and poultry products, are often involved in sporadic cases and outbreaks of human *Salmonellosis* (Sanchez-Vargas *et al.,* 2011). Prior to this Saif (2008) also quoted that poultry and poultry products are a common food borne illness vector and consistently among the leading animal sources of *Salmonella* that enter the human food supply. He also added that humans encountered this problem by consuming raw or undercooked food especially of poultry and egg products.

The increased global population coupled with mass production of animal and human food and the rapid international trade in livestock and food products has worsened the problem (Molla, 2003). Food animals harbor a wide range of *Salmonella* serotypes and act as source of contamination, which is of paramount epidemiological importance in non-typhoid human salmonellosis. Contamination of meat by *Salmonella* may occur at abattoirs from the excretion of carrier animals, contaminated abattoir equipment, floors and personnel and the pathogen can gain access to meat at any stage during slaughtering operations. Cross-contamination of carcasses and meat products could continue during subsequent handling, processing, preparation and distribution (Adesiyun and Oni, 1989).

The commonest serotypes causing disease in humans are *Salmonella* Enteritidis and *Salmonella* Typhimurium (Moussa *et al.,* 2011). Detection of *Salmonella* in live animals, food of animal origin and the environment is essential to generate data which can be used in designing the effective and affordable control and prevention measures. Studying resistant bacteria has become an important worldwide sanitary problem, so that, *Salmonella* species was studied due to its zoonotic importance. *Salmonella* spp. resistance percentage was probably due to the use of antibiotics in animal farms (Usera *et al*., 2002).

The routine practice of using antimicrobial agents to livestock to prevent and treat disease is an important factor in the emergence of antibiotic resistant bacteria that are subsequently transferred to humans through the food chain (Tollefson, 1997; Witte, 1998). Most infections caused by antimicrobial resistant *Salmonella* are acquired by contaminated foods of animal origin indicated that the use of antibiotics for growth promotion is banned in European Union (EU) but permitted in USA and Canada and much of the rest of the world. Studies from different countries reveal that *Salmonella* serotypes isolated from foods of animal origin have multidrug resistance profiles (Holt *et al*., 2007).

Significance of the problem

Insight towards detection of the possible sources of *Salmonella*, for effective control and prevention of the disease, as well as prevention of antimicrobial resistant serotypes, intensive investigation along farm management found a significant contribution for poultry industry. Moreover, investigation along poultry farm with consumers' perception on poultry associated food borne pathogen under such multi factorial structured study has paramount in reducing or prevention of poultry related food bone *Salmonellosis* in human.

Review Of Literature

Salmonellosis is an infectious disease of humans and all animal species worldwide. Infection of animals with various serotypes of *Salmonellae* can result in serious clinical disease particularly in young, elder and stressed animals and always constitutes the vast reservoir for human infections. Clinically, Salmonellosis can be manifested by one of three major syndromes: per-acute septicemia, acute or chronic enteritis (Radostits *et al*., 1994).

Historical Consideration of *Salmonella*

The *Salmonella* bacterium was first described by Theobald Smith (1859-1934) and then in 1885, two American veterinarians, Salmon and Smith isolated the bacterium causing hog cholera from infected pigs (Salmon and Smith, 1886). The name *Salmonella* was subsequently adopted in honor of Dr. Salmon. Over the decades following the pioneering work of Salmon and Smith, many other *Salmonella* were isolated from both animals and humans (Widal, 1896; Getenet, 2008). The antigenic classification or serotyping of *Salmonella* used today is a result of years of study of antibody interactions with bacterial surface antigens by Kauffman and White in the 1920s to 1940s (Kauffmann, 1950). According to this Kauffmann-White scheme, each *Salmonella* serotype is recognized by its possession of a particular lipopolysaccharide (LPS) or O antigen and a flagellar or H antigen. This led to the description of more than 2500 serotypes at present (Brenner *et al*.,2000).

General Characteristics of Salmonella

The morphology of *Salmonellae* is similar to that of other enterobacteria. They are Gram-negative bacilli which are non-acid fast and non-sporing. These organisms usually occur as short rods, measuring 2-4 micrometer in length and 0.5 micrometer in diameter. Occasionally, they develop into longer pleomorphic forms or very short coccobacilli after prolonged culture on laboratory media. With the exception of *S*. Pullorum and *S*. Gallinarum, all strains normally possess peritrichous flagella and are actively motile. Occasionally non motile variants may be encountered in any serotype as a result of the occurrence of dysfunctional flagella. Many serotypes are also known to develop fimbriae (Old, 1990; Bhatia and Ichhpujani, 1994). Like other members of the family Enterobacteriaceae, they produce acid on glucose fermentation; reduce nitrates to nitrite, and donot produce cytochrome oxidase and all but S. typhi produce gas (H2S) on sugar fermentation (Fuaci and Jameson, 2005 and Getenet, 2008). *Salmonella* are non- capsulated except *S*. Typhi, *S*. Paratyphi C and some strain of S. Dublin (Getenet, 2008).

Salmonella make up a large genus of gram-negative bacilli within the family Enterobacteriaceae and it constitute a genus of more than 2300 serotypes that are highly adapted for growth in both humans and animals and that cause a wide spectrum of disease. The growth of S. typhi and S. paratyphi is restricted to human hosts, in whom these organisms cause enteric (typhoid) fever. The remainder of *Salmonella* serotypes, referred to as non-typhoid*Salmonella*, can colonize the gastrointestinal tracts of a broad range of animals, including mammals, reptiles, birds, and insects. More than 200 of these serotypes are pathogenic to humans, in whom they often cause gastroenteritis and can also be associated with localized infections and/or bacteremia (Fuaci and Jameson, 2005).

Most *Salmonella* serotypes grow at temperature range of 5 to 47°C with optimum temperature of 35 to 37°C but some can grow at temperature as low as 2 to 4°C or as high as 54°C . They are sensitive to heat and often killed at temperature of 70°C or above. *Salmonella* grow in a pH range of 4 to 9 with the optimum between 6.5 and 7.5. They require high water activity (aw) between 0.99 and 0.94 (pure water aw=1.0) yet can survive at water activity less than 0.2 such as in dried foods. Complete inhibition of growth occurs at temperatures less than 7°C, pH less than 3.8 or water activity less than 0.94 (Pui*et al*., 2011).

Classification and Nomenclature

The genus consists of two species: the first is *S. enterica* which is divided into six Subspecies: *S. enterica* subsp. *enterica*, *S. enterica* subsp. *salamae*, *S. enterica* subsp. *arizonae, S. enterica* subsp. *diarizonae*, *S. enterica* subsp. *houtenae* and *S. enterica* subsp. *indica*; and the second is *S. bongori* (formerly called *S. enterica* subsp. *bongori*) (WHO, 2003). *Salmonella enteric* subspecies I is mainly isolated from warm-blooded animals and accounts for more than 99% of clinical isolates whereas remaining subspecies and *S. bongori* are mainly isolated from cold-blooded animals and account for less than 1% of clinical isolates. As an example, the Kauffmann species *Salmonella* Typhimurium is now designated as *Salmonella enterica* subspecies I serotype Typhimurium. Under the modern nomenclature system, the subspecies information is often omitted and culture is called *S. enterica* serotype Typhimurium and in subsequent appearance, it is written as *S*. Typhimurium. This system of nomenclature is used nowadays to bring uniformity in reporting (Andrews and Baumler, 2005 and Parry, 2006).

Based on their association with human and animal hosts *Salmonella* can be classified into three main groups (Radostits*et al*., 1994). The first group comprises *Salmonella* Typhi and Paratyphi A and C, which infect only man and are spread either directly or indirectly (via food and water) from person to person (WHO, 1988; Radostits*et al*., 1994). The second group includes serovars that are host adapted for particular species of vertebrates, example *S*. Gallinarum in poultry, *S*. Dublin in cattle, *S*. Abortusequi in horse, *S*. Abortusovis in sheep and *S*. Choleraesuis and *S*. Typhisuis in swine. Some of these are also pathogenic for man (especially *S*. Dublin and *S*. Choleraesuis). The third group contains the majority of other *Salmonella* serovars with no particular host preference that infect both animals and man. Among this third reservoir of serovars are principal agents of *Salmonellosis* that occurs today (Jay, 2000).

Epidemiology of Salmonella

Although primarily intestinal bacteria, *Salmonella* are widespread in the environment and commonly found in farm effluents, human sewage, and in any material subject to fecal contamination. *Salmonellosis* has been recognized in all countries but appears to be most prevalent in areas of intensive animal husbandry, especially poultry and swine production. Approximately 2500 different *Salmonella*serovars have been described, and the number increases annually as new serovars are recognized (Wray and Davies, 2003).

The epidemiology of *Salmonellosis* is complex largely because there are more than 2,500 distinct serotypes (serovars) with different reservoirs and diverse geographic incidences. Changes in food consumption, production, and distribution have led to an increasing frequency of multistate outbreaks associated with fresh produced and processed foods (Rounds *et al*., 2010).

According to the WHO Global Salm-Surv, during 2000-2002, *S*. Enteritidis was by far the most common serotype reported from humans globally. In 2002, it accounted for 65% of all isolates, followed by *S*. Typhimurium at (12%) and *S*. Newport at (4%). Among non- human isolates, *S*. Typhimurium was the most commonly reported serotype in all the three years, accounting for (17%) of isolates in 2002 followed by *S*. Heidelberg (11%) and *S*. Enteritidis (9%). *Salmonella* Enteritidis, *S*. Typhimurium and *S*. Typhi were ranked among the fifteen most common human serotypes in all regions of the world throughout the three year study period. *Salmonella* Agona, *S*. Infantis, *S*. Montevideo, *S*. Saintpaul, *S*. Hadar, *S*. Mbandaka, *S*. Newport, *S*. Thompson, *S*. Heidelberg and *S*. Virchow were also widespread. In Africa in 2002, *S*. Enteritidis and *S*. Typhimurium were each reported from approximately one fourth of isolates from humans (Galanis *et al*., 2006 and Swaminathan *et al*., 2006).

Pathogenesis

Transmission of *Salmonellae* is usually by the fecal oral route but infection via mucous membranes of the conjunctivae or upper respiratory tract is suspected. *Salmonellae* are often pathogenic to humans or animals when acquired by oral route. *Salmonellae* need to colonize the distal small intestine and the colon is a necessary first step in the pathogenesis of enteric *Salmonellosis*. Indigenous fusiform bacteria that lie in the mucous layer infesting the epithelium of the large intestine normally inhibit growth of *Salmonella* by producing volatile organic acids. The normal flora also blocks access to attachment sites needed by the pathogens. Factors, which disrupt the normal colonic flora, such as antibiotic therapy, diet, and water deprivation, greatly increase the host's susceptibility to enteric and septicemic Salmonellosis. Reduced peristalsis also enhances colonization by *Salmonella* because it allows temporary overgrowth to occur, especially in the small intestine. Peristalsis is stimulated by an active indigenous microflora, suppression of which increases the host's susceptibility to colonization (Venter *et al.,* 1994; Gillepsie and Timoney, 1981).

Following an adhesion-dependent attachment of *Salmonellae* to luminal epithelial cells, the invasive pathogen is internalized within an epithelial cell by a receptor-mediated endocytotic process. Cytotoxin localized in the bacterial cell wall suggestively may facilitate *Salmonella* entry into the epithelial layer. Cytoplasmic translocation of the infected endosome to the basal epithelial membrane culminates in the release of *Salmonellae* in the lamina propria. During this invasive process, *Salmonella* secretes a heat-labile enterotoxin that precipitates a net efflux of water and electrolytes into the intestinal lumen. *Salmonella*enterocolitis, which brings profuse loss of intestinal fluids, is the result of enterotoxin-mediated activation of adenylcyclase localized in the cytoplasmic membrane of host epithelial cells (D'Aoust, 1991). The activation of adenylcyclase may be due to the effects of prostaglandins induced by the inflammatory response to the invading *Salmonella*. However, some strains of S. Typhimurium are known to produce enterotoxin-like substances. Inflammatory enteritis quickly develops and is characterized by extensive neutrophil invasion of villous cores with acute ileitis and colitis. Neutrophils are also shed in the stool, and their presence has diagnostic value (Venter *et* al., 1994; Gillepsie and Timoney, 1981).

The pathogenesis of the septicemic phase of Salmonellosis appears to be related to the effects of endotoxin released from bacterial cells (Wray and Davies, 2000; Gillepsie and Timoney, 1981). In *Salmonella*,endotoxic activity resides in the lipopolysaccharide of the cell wall. The lipopolysaccharide is composed of an O– specific chain, a core oligosaccharide common to all *Salmonella*, and a lipid A component. The latter is the part of the lipopolysaccharide molecule that contains endotoxin activity. The effect of endotoxin on the host includes fever, mucosal hemorrhages, leucopenia followed by leukocytosis, thrombocytopenia, and depletion of liver glycogen with prolonged hypoglycemia and shock. The shock effect may be severe and irreversible and may lead to death (Gillepsie and Timoney, 1981).

Salmonella Infections in poultry

Salmonella have a wide variety of domestic and wild animal hosts. The infection may or may not be clinically apparent. In the subclinical form, the animal may have a latent infection and harbor the pathogen in its lymph nodes, or it may be a carrier and eliminate the agent in its fecal material briefly, intermittently, or persistently. In domestic animals, there are several well-known clinical enteritis due to species-adapted serotypes, such as *S*. pullorum or *S*. abortusequi. Other clinically apparent or in apparent infections are caused by serotypes with multiple hosts (PAHO, 2001).

Two serotypes, *S*. Gullorum and *S*. Pallinarum, are adapted to domestic fowl. They are not very pathogenic for man, although cases of *Salmonellosis* caused by these serotypes have been described in children. Many other serotypes are frequently isolated from domestic poultry; for that reason, these animals are considered one of the principal reservoirs of *Salmonellae*. Pullorum disease, caused by serotype *S*. pullorum, and fowl typhoid, caused by *S*. gallinarum, produce serious economic losses on poultry farms if not adequately controlled. Both diseases are distributed worldwide and give rise to outbreaks with high morbidity and mortality. Pullorum disease appears during the first 2 weeks of life and causes high mortality. The agent is transmitted vertically as well as horizontally. Carrier birds lay infected eggs that contaminate incubators and hatcheries. Fowl typhoid occurs mainly in adult birds and is transmitted by the fecal matter of carrier fowl. On an affected poultry farm, recuperating birds and apparently healthy birds are reservoirs of infection. *Salmonella* un-adapted to fowl also infect them frequently. Nearly all the serotypes that attack man infect fowl as well. Some of these serotypes are isolated from healthy birds. The infection in adult birds is generally asymptomatic, but during the first few weeks of life, its clinical picture is similar to pullorum disease (loss of appetite, nervous symptoms, and blockage of the cloaca with diarrheal fecal matter). The highest mortality occurs during the first two weeks of life. Most losses occur between six and ten days after hatching (PAHO, 2001).

Salmonellosis in Humans

Salmonella infections in humans can range from a self-limited gastroenteritis usually associated with non-typhoidal*Salmonella* (NTS) to typhoidal fever with complications such as a fatal intestinal perforation (OIÉ, 2000). Non-typhoidal*Salmonella* is one of the principal causes of food poisoning worldwide with an estimated annual incidence of 1.3 billion cases and 3 million deaths each year (Torpdahl*et al*., 2007). Outbreaks of *Salmonellosis* have been reported for decades, but within the past 25 years the disease has increased in incidence in many continents. The disease appears to be most prevalent in areas of intensive animal husbandry (OIÉ, 2000).
The incubation period in people is variable but is usually between 12 and 36 hours. The typical presenting symptom is diarrhea but this may be accompanied by nausea and abdominal pain, although vomiting is not usual. There may also be a headache and fever. While the infection is normally self-limiting and does not require antibiotic treatment, occasionally, with more invasive *Salmonella* such as *S*. Virchow, bacteremia can occur. The infection is rarely fatal in people (Gracey *et al.*, 1999).

Salmonellosis is most commonly caused by *S*. Typhimurium or *S.* Enteritidis. Secondly, *S*. *enterica* subsp. *typhi* and *S.enterica* subsp. *paratyphi* are the causes of typhoid fever or paratyphoid fever, respectively. *Salmonella* can replicate both inside the vacuoles of host cells and in the external environment. *Salmonella* are the second most common pathogens isolated from humans with gastroenteric disease in developed countries (Buncic, 2006).

*Salmonella*Typhimurium and *S*. Enteritidis occur in the gastro intestinal tract of animals, including livestock. The disease is self-limiting, but can be severe in young, elderly or otherwise IC (immunecompromised) people. *Salmonella* invade epithelial cells in the ileum and proliferate in the lamina propria and profuse, watery diarrhoea results. Some isolates produce a heat-labile enterotoxin, which initiates diarrhoea. Sequelae include post-enteritis reactive arthritis and reiter's syndrome and systemic infection can result. Individuals can develop carrier status of up to 6 months in duration. The infectious dose varies, from only a few CFU to >105 CFU, so growth of the pathogen in foods has not been a factor in all cases of foodborne Salmonellosis, but appears to have been in some. Foods known to have been vehicles of *Salmonellosis* include poultry, eggs, and meat, milk, chocolate, coconut and frog legs. However, any faecally contaminated food can be implicated (Buncic, 2006).

*Salmonella*Typhi and *S. enterica* subsp. *paratyphi* cause the systemic diseases typhoid fever and paratyphoid fever, respectively. These pathogens occur in human faeces, and are spread via human faeces to the environment and to foods. Person-to-person transmission is common. The disease symptoms of typhoid and paratyphoid fevers are dissimilar to those of enteric Salmonellosis. *Salmonella* penetrate the intestinal epithelium, possibly proliferating in macrophages and polymorphs, pass into mesenteric lymph nodes, liver or spleen then cause septicemia. Peritonitis and subsequent death can occur. Ulceration of the ileum can occur if organisms multiply in the bile of the gall bladder and cause re-infection. Any food could be a vehicle of infection if contaminated with human faeces. Foods known to have been vehicles of typhoid fever include raw milk, shellfish and meat. However, typhoid fever is predominantly spread by water contaminated with human faeces (Buncic, 2006).

Economic Importance of *Salmonellosis*

Salmonellosis, a common human intestinal disorder primarily caused by *Salmonella* contaminated meat and poultry is estimated to cost nations billions of dollars annually thereby draining funds that could have been used for development (WHO, 1988; Radostits *et al.*, 1994).

Salmonellosis is one of the significant causes of economic loss in farm animals because of the cost of clinical disease which include deaths, diagnosis and treatment of clinical cases, diagnosis laboratory costs, the cost of cleaning and disinfection, and the cost of control and prevention. The loss incurred by the livestock producers includes feed efficiency, and reduced weight gains or deaths because of Salmonellosis (Radostits *et al*., 1994). Human foodborne *Salmonellosis* constitutes a major health problem in many countries. Moreover the costs associated with *Salmonellosis* could be considerable (Persons and Jendteg, 1992). Financial costs are not only associated with investigation, treatment and prevent ion of human illness but may affect the chain of production. Poultry products have always topped the incidence of *Salmonellosis* in many developing countries including India, Egypt, Brazil and Zimbabwe (Henson, 2003) and is the most seriously perceived food risks in chicken meat, even in the developed countries (Yeung, 2001). *S.* Enteritidis is transmitted to the human food supply through eggs of hens that appear healthy (Porwollik*et al.,* 2005). Contamination with *Salmonella* in poultry products can occur at multiple steps along the food chain, which includes production, processing, distribution, retail marketing, handling and preparation (Cui *et al.,* 2005).

Antimicrobial Resistance and *Salmonella*

Antimicrobial resistant *Salmonella* are increasing due to the use of antimicrobial agents in food animals (Zewdu and Cornelius, 2009) at sub-therapeutic level or prophylactic doses which may promote on-farm selection of antimicrobial resistant strains and markedly increase the human health risks associated with consumption of contaminated meat products (Molla *et al.*, 2003; Molla *et al.*, 2006; Zewdu and Cornelius, 2009). Cattle have been implicated as a source of human infection with antimicrobial resistant *Salmonella* through direct contact with livestock and through the isolation of antimicrobial resistant *Salmonella* from raw milk, cheddar cheese, and hamburger meat traced to dairy farms. Antimicrobial use in animal production systems has long been suspected to be a cause of the emergence and dissemination of antimicrobial resistant *Salmonella* (Alexander *et al.*, 2009).

Prevention and Control of Salmonellosis

Prevention of salmonellosis by the implementation of hygiene measures is difficult and use of antibiotics may give rise to the emergence of resistance problems. Reducing *Salmonella* prevalence requires a multi-hurdle approach at all stages of breeding, hatching, grow-out, transportation and processing. Attenuated DNA recombinant live *Salmonella* vaccines, combined with comprehensive control strategy in animals, feed and animal food stuffs will help to reduce Salmonellosis. Additional measures to control secondary contamination could be prevention of contamination by cleaning and disinfection, hygiene of personnel and proper processing (Sinell, 1995). Growth of micro-organisms in meat and poultry products can be controlled by maintaining a cold chain at 10^{o}C, especially for *Salmonella* during transport and storage (Coleman *et al.,* 2003).

Vaccination does not offer complete protection of the birds against infection. An effective way to control *Salmonella* in eggs is by preventing the vertical spread of *S.* Enteritidis between different generations of birds. The principle is a top down approach, eliminating *Salmonella* from the top of the production pyramid and downwards by test and slaughter (Hans and Dean, 2006).

Conclusion and Recommendation

Salmonellosis is one of the significant causes of economic loss in farm animals because of the cost of clinical disease which include deaths, diagnosis and treatment of clinical cases, diagnosis laboratory costs, the cost of cleaning and disinfection, and the cost of control and prevention. Moreover the costs associated with *Salmonellosis* could be considerable. Financial costs are not only associated with investigation, treatment and prevent ion of human illness but may affect the chain of production. Prevention of salmonellosis by the implementation of hygiene measures is difficult and use of antibiotics may give rise to the emergence of resistance problems. Reducing *Salmonella* prevalence requires a multi-hurdle approach at all stages of breeding, hatching, grow-out, transportation and processing. Therefore additional measures to control secondary contamination could be prevention of contamination by cleaning and disinfection, hygiene of personnel and proper processing.

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