World Applied Sciences Journal 38 (1): 01-07, 2020
ISSN 1818-4952

DOI: 10.5829/idosi.wasj.2020.01.07

Prevalence of Bovine Salmonellosis in Ethiopia: A Review

[1]*Temesgen Zekarias and* [2]*Teferi Mandado*

[1]Ethiopian Institute of Agricultural Research, Addis Ababa, Ethiopia
[2]Livestock and Fishery Development Department, Dawro Zone, Ethiopia

Abstract: Salmonellosis is caused by pathogenic *Salmonella* Species. The feco-oral route is the most important mode of transmission of Salmonellosis in animals. Epidemiological pattern, prevalence and incidences of disease differ greatly between geographical areas. This is affected by pathogens themselves, industrialization, urbanization and change of lifestyles, knowledge, belief and practices of food handlers and consumers, demographic changes, international travel and migration, international trade in food, animal feed and poverty and lack of safe food preparation facilities. Some studies conducted in Ethiopia on prevalence of *Salmonella* provided that there were different levels of prevalence of the disease in different parts of the country. Thus the objective was to review the prevalence and current distribution of bovine salmonellosis in Ethiopia. This paper also highlighted the taxonomy of the bacteria, the epidemiology and the susceptibility of the hosts, mode of transmission, pathogenesis, clinical manifestations, diagnosis, treatment and control of the disease in Ethiopia with conclusion and recommendations.

Key words: Control · Epidemiology · Foodborne · *Salmonella* Species · Treatment

INTRODUCTION

Salmonellosis is a common intestinal illness manifested clinically in animals [1] and humans [2] as acute and chronic enteritis, an acute septicemic disease or as subclinical infections [3]. *Salmonella* are common in cattle. They are often of concern due to the disease of cattle and the potential to infect human that come in contact with cattle or consume dairy and meat products [4]. 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, especially of poultry or pigs and dairy cattle reared in confinement [5]. There are pandemics of *S. Enteritidis* and *Typhimurium* DT 104 which resulted in enacting regulations in many countries to control the prevalence of salmonellosis in farm animals in order to prevent foodborne infection [6]. The clinically normal carrier animal is a serious problem in all host species. Foods of animal origin, particularly meat, poultry and in some instances, unpasteurized egg products are considered to be the primary sources of human Salmonellosis [3, 6, 7]. Most of these food products, for example; beef, mutton and poultry, are contaminated during slaughter and processing, from the gut contents of healthy excreting animals. In the same way, all food that is produced or processed in a contaminated environment may become contaminated with *Salmonella* and be responsible for outbreaks or separate cases of disease as a result of faults in transport, storage or preparation [8]. Therefore, the objectives of this paper were to review the general information concerning the bacteria, the current prevalence and distribution of bovine salmonellosis to give a general recommendations based on the shortcomings in Ethiopia.

Taxonomy of the Bacteria: The genus *Salmonella* obtained its name from the American veterinarian Daniel Elmer Salmon, who first isolated *Salmonella* enteric serotype Cholerae Suis from pigs in 1885 [9]. *Salmonella* are facultative anaerobic, Gram-negative rods belonging to the family *Enterobacteriaceae*. Although members of this genus are motile by peritrichous flagella, non-flagellated variants, such as *Salmonella pullorum* and *Salmonella gallinarum* and non-motile strains resulting from dysfunctional flagella do occur [8]. The naming system for *Salmonella,* which

Corresponding Author: Temesgen Zekarias, Ethiopian Institute of Agricultural Research, Addis Ababa, Ethiopia P.O. Box: 2003, Tel: +251912844964.

has been in use, so far combines several nomenclatural systems that inconsistently divide the genus into species, subspecies, subgenera, group, subgroups and serotypes (serovars) and thus causes confusion [10]. For convenience, the species (enterica) designation is frequently eliminated, leaving *Salmonella Typhimurium* [11]. The genus name is italicized and for named serotypes, to emphasize that they are not separate species, the serotype name is not italicized and the first letter is capitalized [10]. Except for serotypes *S. Typhi, S. Paratyphi* A and *S. Paratyphi* C, which are strictly human and whose only reservoir is man, all serotypes could be considered zoonotic or potentially zoonotic [3].

Epidemiology of Salmonellosis: The epidemiology of *Salmonella* is complex which often makes control of disease difficult. Epidemiological pattern of prevalence of infection and incidences of disease differ greatly between geographical area 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 [5].

Although *Salmonella* are primarily intestinal bacteria, they 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 [6]. Sibhat *et al*. [12] found the serovars Newport, Anatum and Eastbourne to be the most prevalent in Ethiopia. The rate of infection in domestic animal has been estimated from 1-3%. Epidemiological surveillance of animals including bird is very important since source of large majority of non-typhoidal salmonellosis cases are of food animal origin [13].

Susceptibility of the Hosts: All animals are at increased risk of developing disease if their normal flora is disrupted (stress, antibiotics). These circumstances render animals susceptible to exogenous exposure or activation of silent infections. Young animals are more susceptible to Salmonellosis than older ones. Poor sanitation, overcrowding, unfavorable weather, stress of hospitalization and surgery, parturition, parasitism, transportation and concurrent viral infections are all factors which predispose animals to clinical Salmonellosis. Many animals suffer unapparent infections during their lifetimes. This is especially true of swine [14] and poultry fed rations that contain *Salmonella.* 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 [5].

Mode of Transmission: The feco-oral route is the most important mode of transmission of *Salmonella* in animals. Infection in cattle may also occur via other routes, including the respiratory tract, by inhalation of aerosol [15]. However, the cycle of infection may be more complex in some animal populations; in poultry, for example, the primary source of infection may be contaminated feed and subsequent spread may occur via the feco-oral route or from egg to chick in the hatchery. Meat and bone meal, fish meal and soya bean meal have all been shown to be frequently and heavily contaminated [3].

Salmonella multiply optimally at a temperature of 35 to 37°C, pH about 6.5-7.5 and water activity between 0.94-0.84 [16]. They are able to multiply in the environment with low level or no oxygen [17]. The bacteria are sensitive to heat and will not survive at temperature above 70°C; so it is sensitive to pasteurization, but resistant to drying even for years. Especially in dried feces, dust and other dry materials such as feed and certain food [5].

Carrier and Sources of Infection: Because *Salmonella* are facultative intracellular organisms that survive in the phagocyte of some macrophages, they can evade the bactericidal effects of antibody and complement. Thus, persistence of infection in animals and in the environment is an important epidemiological feature of Salmonellosis [1]. Although *Salmonella* may survive for long periods in the environment [15], it is the carrier state that provides the major source of infection for animals and humans. Certain stress factors have been shown to promote excretion of *Salmonella* by carriers and to lead to activation or reactivation of disease in carrier animals [3]. Transportation of animals, overcrowding and administration of corticosteroids, parturition and concurrent viral and protozoan infections have all been shown to increase susceptibility of animals to disease [14]. Intensification of husbandry in all species is recognized as a factor contributing significantly to an increase in the new infection rate [1]. The carrier domestic (including poultry) and wild animals, turtles and other pets shed *Salmonella*.

Pathogenesis of the Disease: The outcome of infection with *Salmonella* depends essentially on three factors: the infective dose, predisposing factor influencing the host and the level of immunity [18]. In cattle the common route of infection is ingestion of the bacteria through contaminated feed and water. It may disseminate to the rest body part of the host via the lymph fluid or blood and usually also lead to fecal excretion of bacteria. *Salmonella* are normally inhibited by the high concentrations of volatile fatty acids and the normal pH below 7 in the rumen [19]. The bacteria adhere to and invade intestinal cells in the mucosa mainly associated with the Peyer's patches in the terminal jejunum and ileum through the columnar enterocytes and specialized microfold enterocytes (M cells). Once the bacteria have crossed the intestinal epithelium they enter macrophages in the underlying lymphoid tissue from where they are drained to the local lymph nodes, which are important barriers for further dissemination. If this barrier is overcome, the bacteria reach the reticuloendothelial tissue containing organs while surviving and replicating inside the macrophages [20].

Colonization of the distal small intestine and the colon is a necessary first step in the pathogenesis of enteric salmonellosis. 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 [18].

Following an adhesion-dependent attachment of *Salmonella* to luminal epithelial cells, the invasive pathogen is internalized within an epithelial cell by a receptor-mediated endocytosis process. Cytotoxin localized in the bacterial cell wall suggestively may facilitate *Salmonella* entry into the epithelial layer. During this invasive process, *Salmonella* secretes a heat-labile enterotoxin that precipitates a net efflux of water and electrolytes into the intestinal lumen [21]. The activation of adenyl cyclase 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. Neutrophils are also shed in the stool and their presence has diagnostic value [18]. The shock effect may be severe and irreversible and may lead to death.

Clinical Signs: Salmonellosis is manifested in animals in three major forms: enteritis, septicemia and abortion. However, in an outbreak, or even in a single animal, any combination of the three may be observed. Fever, inappetence and depression are commonly observed in acutely ill animals. Enteritis caused by *Salmonella* results in the passage of foul-smelling, watery feces, which may contain fibrin, mucus and sometimes blood. When the enteric disease is severe, death may result from dehydration, electrolyte loss and acid-base imbalance [14].

The disease manifestations depend on the virulence of different *Salmonella* serovars; the number of *Salmonella* ingested and host immunity. Many *Salmonella* infections are opportunistic infection in compromised hosts. The majority of *Salmonella* infections in a herd over time are subclinical; the clinical infections are only the tip of the iceberg, even during outbreaks of clinical disease [15]. Adult cattle generally contract either acute or subacute enteric salmonellosis and pregnant animal may abort during the early stage of acute enteric disease. Severely affected animals show fever, depression, inappetence and drop in milk yield. These sign are followed by diarrhea which is fowl smelling, the faeces being mucoid and usually containing a clot of blood and shred of necrotic intestinal mucosa. Abortion may either precede the onset of dysentery or follow it within two or four weeks. Alternatively abortion may occur in cows that show no sign of illness, septicemia and/or placentitis being the case of death of fetus. Retention of the placenta occurs in approximately 70% of cases that abort but subsequent fertility is not usually affected [18].

Septicemic form often leads to abortion. *Salmonella Dublin* has been associated with outbreaks of abortion in cattle and several other serovars adapted to animal hosts have a particular association with abortion. The signs are not pathognomonic and in many cases, especially in poultry and pigs, *Salmonella* infections may be unapparent [4]. Clinical sign vary but typically the enteric form of diseases predominate which is characterized by pyrexia, dullness and anorexia, followed by diarrhea that may contain fibrin and mucus. The feces may become blood stained and "stringy" due to the presences of necrotic intestinal mucosa. Calves that recover from infection do not typically remain carrier [22].

Diagnosis: Diagnosis is based on the isolation of the organism either from tissues collected aseptically at necropsy or from feces, milk, blood, rectal swabs or

environmental samples [4]. When infection of the reproductive organs or conceptus occurs, it is necessary to culture fetal stomach contents, placenta and vaginal swabs and, in the case of poultry, egg contents. However, salmonellosis is particularly difficult to determine in clinically normal carrier animals [23].

The clinical sign and finding at postmortem examination are not unique to salmonellosis although a tentative diagnosis may be made. 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 106 organisms/gram. Similar concentration may be present in the wall and content of the ileum, cecum and colon and associated lymph nodes [24].

Salmonella may be isolated by a variety of techniques, which may include pre- enrichment in non-selective medium to resuscitate sub-lethally damaged *Salmonella*, enrichment media that contain inhibitory substances to suppress non-*Salmonella* organisms and selective plating agars to differentiate *Salmonella* from other Enterobacteriaceae [4]. A number of serological tests, such as the serum agglutination test [15] and indirect enzyme linked immunosorbent assays have been developed for diagnosis of *Salmonella.* Serological tests are useful for the identification of infected herds but are inadequate for the identification of persistently infected animals [4].

Treatment and Control

Treatment: Treatment of the systemic form includes nursing care and appropriate antimicrobial therapy as determined by retrospectively acquired susceptibility data. Treatment options may be compromised due to acquisition of resistance (R) plasmids encoding resistance to multiple antibiotics [25].

In animal treatment supportive treatment with intravenous fluid is necessary for patients that have anorexia, depression, significant dehydration. Oral fluid and electrolyte may be somewhat helpful and much cheaper than IV fluid for cattle demand to be mildly or moderately dehydrated. Cattle that are willing to drink can have specific electrolyte (NaCl, KCl) added to drinking water to help correcting electrolyte [26]. The implementation of broad prophylactic strategies that are efficacious for all *Salmonella* 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 [27].

Control: Condition that contribute to an increasing incidence of epidemic salmonellosis include large herd size, more intensive and crowded husbandry and the trend of free-stall barn with loose housing, which contribute to the fecal contamination of the entire premise. When salmonellosis has been confirmed in a herd, the following control measures should be considered; isolate obviously affected animals to one group if possible, treat severely affected animals, affected animals institute measure to minimize public health concern like (no raw milk should be consumed) physically clean the environment and disinfect the premise following resolution of the outbreak or crises period. Prevention is best accompanied by maintaining a cross herd and culturing new feed additives and components before using the entire ration [24]. It is generally agreed that supportive therapy and good nursing are important. These include oral or parenteral rehydration, correction of electrolyte balance and stabilization of acid base equilibrium [18].

The control of *Salmonella* in meat animals and derived products is a most challenging task because of the complexity and interdependence of various aspects of animal husbandry, slaughtering and food processing. Because of the complexity of *Salmonella* virulence factors, little progress has been made in converting the available knowledge in to therapeutics. Good Agricultural Practices (GAP), Good Manufacturing Practices (GMP), Hazard Analysis Critical Control Point (HACCP), system appropriate food handling and adequate water treatment remain the best preventive measures for most *Salmonella* infection, although the typhoid vaccines are effective against *S. typhi* in humans and vaccines for several other serovars have shown promise in food animals [28, 11].

Prevalence of Salmonellosis in Animal Products in Ethiopia: Some studies conducted in Ethiopia on the prevalence of *Salmonella* provided the following data in different sources of meat samples. For instance, 2.8 and 9.8% in apparently healthy slaughtered sheep and goats respectively [29]; 7.1% in apparently healthy slaughtered cattle [30]. 16.2% in camel [5], 21.1% in chicken carcass and giblets [31]. The study conducted to determine the prevalence and antimicrobial resistance

pattern of *Salmonella* isolates from lactating cows and in contact humans in dairy farms of Addis Ababa determined the overall prevalence of 10.7% of cows and 13.6% of the humans shaded *Salmonella* [32].

Lactating cows could be potential sources of *Salmonella* infection for individuals' working in dairy farms and for the community at large. Addis *et al*. [32] reported a prevalence of 7.1% from apparently healthy slaughtered cattle. The disease can be also controlled by vaccination of cattle [33]. The overall prevalence of *Salmonella* was 0.85% for beef samples derived from meat cuts, meat fluid and carcass swabs. The prevalence of *Salmonella* in carcass swabs was 2.67%, from that of meat cuts was 0.50% and meat fluid was 0.43% [34]. A cross sectional study determined overall prevalence of 23.6% from bulk milk of dairy farms in Debre Zeit which was undertaken from December, 2013 to April, 2014 in supermarket, large and small holders. The occurrence of *Salmonella* in large and small scale farm was 20.4 and 27.3% respectively [35].

According to the study conducted by Radostitis *et al*. [5], out of the total 300 meat samples (minced beef meat, mutton and pork) examined in Addis Ababa, Ethiopia; 14.7% were positive for *Salmonella* serotypes. This study indicated that, *Salmonella* was detected in 14.4% of minced beef, 14.1% of mutton and 16.4% of pork samples. Their results also showed that 66.7% of the samples collected from retail supermarkets comprising 58.3% of minced beef, 41.2% of mutton and 54.5% of pork samples were *Salmonella* positive. The isolation of *Salmonella* with an overall prevalence of 14.7% from minced beef, mutton and pork indicated the wide spread occurrence and distribution of *Salmonella* in meat samples obtained from retail supermarkets in Addis Ababa. The prevalence of *Salmonella* varied among the sampling sites/ supermarkets and sample type from 0 to 40%. Samples examined from supermarkets 24, 32, 4, 19 and 1 with the prevalence of 40, 22.7, 15, 18.2 and 16.5%, respectively, were the most contaminated [36].

In Southern Ethiopia, Hawassa, a prevalence of 9% was reported for *Salmonella* from raw beef samples of butchers' shops [37]. According to a study conducted to overview the Microbiological Quality of Milk Produced in Urban and Peri-Urban Farms in Central Ethiopia and its Public Health Impact to help implement quality standards and determine the public health significance of milk borne pathogens, a pilot study was performed to establish baseline data on the microbiological quality of milk throughout central Ethiopia. Fresh bovine milk samples were taken and combined bulk tanks in Selale, Asela, Akaki and Debre Zeit was determined and showed that 0% prevalence of *Salmonella* in both pooled and bulk tank samples [38]. A total of 1.6% horses were positive for *Salmonella* by either primary bacterial culture and additional horses 0.5% were positive for *Salmonella* by PCR. The combined prevalence of *Salmonella* fecal shedding from among all the horses in this study was 2.1%. The results of this study suggest that the prevalence of fecal shedding of *Salmonella* among racehorses in Louisiana is low [39]. Out of 300 cattle carcasses samples collected, 23(7.6%) showed positive results for *Salmonella* species. Of these positive carcass samples, 6(6%) were from the hind limb; 10(10%) from the abdomen and 7(7%) were from the neck region [38].

Relatively higher prevalence of *Salmonella* was detected in the abdomen than the neck and hind limb. Out of 23 *Salmonella* isolates 11(47.8%); 9(39.1%) and 3(13.1%) were *Salmonella* group A; *Salmonella* arizonae and *Salmonella typhi* respectively [38]. The other studies on milk samples collected from lactating dairy cows at Sebeta, Ethiopia [40] and Addis Ababa [32] reported prevalence estimates of 16 and 28.6% respectively. According to Shilangale *et al*. [34] and Barrow *et al*. [22] the prevalence estimate of *Salmonella* in milk of lactating dairy cows and fecal samples from beef, dairy and veal calf of cattle were 2.1 and 14.4% in Addis Ababa, Ethiopia and Australia respectively.

CONCLUSIONS AND RECOMMENDATIONS

Salmonellosis is a common intestinal illness caused by numerous *Salmonella* serovars. The feco-oral route is the most important mode of transmission of *Salmonella* in animals. Due to the different levels of prevalence of disease in both animals and human in the country, higher attention and collaboration are needed from veterinary and public health professionals for control and prevention. Based on the above conclusion the following recommendations are forwarded: Collaboration is needed among government, professional organizations and interest groups on control and prevention of the disease. Encouraging judicious use of antimicrobial drugs in veterinary and public sectors is crucial.

REFERENCES

1. Radostits, O.M., D.C. Blood and C.C. Gay, 1994. Diseases caused by Salmonella species. In: Veterinary Medicine, a Text Book of the Diseases of Cattle, Sheep, Pigs, Goats, Horses. 8th ed., Baillie Tindall, London. pp: 730-745.

2. Hohmann, E.L., 2001. Non typhoidal salmonellosis. Clin.Infec. Dis., 32: 253-269.
3. Acha, P.N. and B. Szyfres, 2001. Zoonosis and communicable disease common to man and animals, 3rd ed., pp: 233-245.
4. OIE, 2000. Salmonellosis. In: Manual Standards for Diagnostic Test and Vaccines, 4th ed. France, Paris. pp: 1-18.
5. Radostitis, O.M., C.C. Gay, K.W. Hinchliff and P.D. Constable, 2007. Veterinary Medicine: A text book of the disease of cattle, horses, sheep, pigs and goats. 10th ed. Elsevier Ltd. pp: 325-326.
6. Wray, C. and R.H. Davies, 2000. Salmonella Infections in Cattle. In: Wray, C. and A. Wray (Eds.). Salmonella in Domestic Animals. New York, CABI Publishing, pp: 169-190.
7. Nielsen, B., D. Baggrsen, F. Bager, J. Haugegaal and P. Lind, 1995. The serological response to Salmonella serovars Typhimurium and Infantis in experimentally infected pigs. The time course followed with an indirect anti-LP ELISA and bacteriological examinations. Vet. Microbiol, 47: 205-218.
8. D'Aoust, J.Y., 1997. Salmonella Species. In: Doyle, M.P., L.R. Beuchat and T.J. Montville (Ed). Food Microbiology Fundamentals and Frontiers, ASM Press, Washington D.C., pp: 129-158.
9. Rabsch, W., C. Altier, H. Tschape and A.J. Baumler, 2003. Foodborne Salmonella infection. In: Torrence, M.E. and Isaacson, R.E. (eds). Microbial Food Safety in Animal Agriculture. Current Topics.1st ed. USA, Blackwell Publishing, pp: 97-108.
10. Brenner, F.W., R.G. Villar, F.J. Angulo, R. Tauxe and B. Swaminathan, 2000. Salmonella nomenclature, guest commentary. J. Clin. Microbiol., 38: 2465-2467.
11. Institute of Food Technologist (IFT), 2003. IFT Export Report on Emerging Microbiological Food Safety Issues. Implications for Control in the 21st Century, S. Lowry/ univ. ulster/ stone, pp: 14-21.
12. Sibhat, B., B. Molla, A. Zerihun, A. Muckle and L. Cole, 2011. Salmonella Serovars and Antimicrobial Resistance Profiles in Beef Cattle, Slaughterhouse Personnel and Slaughterhouse Environment in Ethiopia. Zoonosis Public Health, 58: 102-109.
13. Oliveira, S.D., L.R. Santos, D.M.T. Schuch, A.B. Silva, C.T.P. Salle and C.W. Canal, 2002. Detection and identification of Salmonella s from poultry-related samples by PCR. Vet. Microbiol., 87: 25-35.
14. Clarke, R.C. and C.L. Gyles, 1993. Salmonella. In: Pathogenesis of Bacterial Infections in Animals. 2nd ed. Eds., Gyles C.L and C.O. Thoen. Ames, IA: Iowa State University, pp: 133-153.
15. Gay, J., 2003. Bovine Herd Salmonellosis. Washington State University, College of Veterinary Medicine. Field Disease Investigation Unit., pp: 1-11.
16. Johnson, T.J., Y.M. Wannemuehler, S.J. Johnson, C.M. Logue, D.G. White, C. Doetkott and L.K. Nolan, 2007. Plasmid replicon typing of commensal and pathogenic Escherichia coli isolates. Appl Environ Microbiol, 73: 1976-1983.
17. European Commission, 2000. An opinion of the scientific committee on veterinary measure relating to public health on foodborne zoonosis. Health and consumer protection directorate general, Directorate B, Scientific health opinions, Unit B3- Managements of Scientific Committees II, pp: 24-27.
18. Venter, B.J., J.G. Myburgh and M.L. Van der Walt, 1994. Bovine Salmonellosis. In: Coetzer, J.A.W., Thomson, G.R., Tustin, R.C. and Kriek, N.P.J. (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Oxford University Press, Cape Town, pp: 1104-1112.
19. Morisse, J.P. and J.P. Cotte, 1994. Evaluation of some risk factors in bovine salmonellosis. Vet. Res., 25: 185-191.
20. Scherer, C.A. and S.I. Miller, 2001. Molecular Pathogenesis of Salmonella. In: Groisman, E.A. (Ed). Principles of Bacterial Pathogenesis. Academic Press, New York, pp: 265-333.
21. D'Aoust, J.Y., 1991a. Pathogenicity of foodborne Salmonella. Int. J. Food Microbiol., 12: 17-40.
22. Barrow, P.A., M.A. Jones and N. Thomson, 2010. Pathogenesis of bacterial infection in animals, 4th eds. Edited by Gyles, C.L., Songer, G. Theon, C.O: Blackwell Publishing, pp: 233.
23. Mohr, J. and G. Pollex, 1998. Agglutinating monoclonal antibodies in diagnosis of salmonellosis. Biotest Bulletin, 6: 75-83.
24. Jones, P.J., P.R. Weston and T. Swail, 2007. Salmonellosis In: Bovine medicine, diseases and husbandry of cattle. Edited by Andrew, A.H. 2nd Edition: Blackwell Publishing, pp: 215-230.
25. Hirsh, D.C., 1999. Salmonella. In: Hirsh, D.C. and Zee, Y.C. (ed.) Veterinary Microbiology. 1st ed., Blackwell Science Inc., pp: 75-79.
26. Rebhun, C.W., 1995. Disease of Dairy cattle: 1st Edition. Awa Verly Company.
27. Mohler, V.L., M.M. Izzo and J.K. House, 2009. Salmonella in calves. Vet Clin North Am Food Anim. Pract., 25: 37-54.
28. Seifert, H.S.H., 1996. Tropical Animal Health, 2nd ed. Kluwer Academic Publishers, pp: 368-371.

29. Woldemariam, E., M. Bayleyegn, D. Alemayehu and A. Muckle, 2005. Prevalence and distribution of Salmonella in apparently healthy slaughtered sheep and goats in Debre Zeit, Ethiopia. Small Ruminant Research, 58: 19-24.
30. Alemayehu, D., A. Muckle and B. Molla, 2003. Prevalence and antimicrobial resistance pattern of Salmonella isolates from apparently healthy slaughtered cattle in Ethiopia. Trop. Anim. Health Prod., 35: 309-319.
31. Molla, B. and A. Mesfin, 2003. A survey of Salmonella contamination in chicken carcass and giblets in central Ethiopia. Revue Méd. Vét., 154: 264-270.
32. Addis, Z., N. Kebede, Z. Sisay, H. Alemayehu, A. Yirsawand and T. Kassa, 2011. Prevalence and antimicrobial resistance of Salmonella isolated from lactating cows and in contact humans in dairy farms of Addis Ababa: a cross sectional study, University of Gondar, College of Medicine and Health Science, Department of Medical Laboratory Science. BMC Infectious Diseases, 11: 1-10.
33. Kemal, J., 2014. A Review on the Public Health Importance of Bovine Salmonellosis. J. Veterinar. Sci. Technol., 5: 175.
34. Shilangale R.P., P.G. Kaaya and P.M. Chimwamurombe, 2015. Prevalence and Characterization of Salmonella Isolated from Beef in Namibia, European Journal of Nutrition & Food Safety, 5: 267-274.
35. Tesfa, M. and D. Assefa, 2016. Prevalence of Antimicrobial Resistant Salmonella Isolated from Bulk Milk of Dairy Cows in and around Debre Zeit, Ethiopia. World's Vet. J., 6: 110-116.
36. Endrias, Z., 2004. Prevalence, distribution and antimicrobial resistance profile of Salmonella isolated from food items and personnel in Addis Ababa. MSc Thesis, Faculty of Veterinary Medicine, Addis Ababa University, Debre Zeit, Ethiopia, pp: 14-36.
37. Mogessie, A., 1994. Microbial flora and incidence of some foodborne pathogens on fresh raw beef from butcher s shops in Hawassa, Ethiopia. Bull. Anim. Hlth. Prod. Afr., 42: 273-277.
38. Sophia, D., 2011. Microbiological Quality of Milk Produced in Urban and Peri-Urban Farms in Central Ethiopia and its Public Health Impact, MSc Thesis, The Ohio State University.
39. Anna, M.C., 2001. Characterizing Salmonella fecal shedding among racehorses in Louisiana, MSc Thesis, Graduate Faculty of the Louisiana State University and Agricultural and Mechanical College, pp: 1-12.
40. Abraham, A., K. Sudip, K. Rakshit and R. Anil, 2013. Genotypic and phenotypic characterization of antimicrobial resistance patterns of Salmonella strains isolated from raw milk in Sebeta, Ethiopia. International J. Adv. Research, 3: 193-196.