Chapter

Salmonellosis in Food and Companion Animals and Its Public Health Importance

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Abstract

Salmonellosis in animals is caused by typhoidal and non-typhoidal *Salmonella* organisms. Non-typhoidal salmonellosis is a zoonosis of major public health concern occasioning over 155, 000 mortalities yearly worldwide. The majority of the human infections are mainly acquired directly through consumption of contaminated foods of animal origin, particularly poultry, eggs and dairy products or consumption of contaminated fruits. Rodents and will birds are the main reservoirs of non-typhoidal salmonellosis. Salmonellosis has a great economic and health impact occasioned by the cost of surveillance, investigation, treatment, and prevention in both animals and humans. Non-typhoidal salmonellosis is further complicated by the wide host range and the emergence of multidrug resistant *Salmonella* strains due to intensification of livestock production and uncontrolled antimicrobial drug use. There is a need for more innovative prevention and control measures to safeguard losses in animals and human health. This chapter will discuss salmonellosis in food and companion animals, the public health importance, and the challenges facing its control.

Keywords: salmonellosis, animals, public health, control

1. Introduction

Salmonellosis is caused by bacterial species in the genus *Salmonella*, a member of the family *Enterobacteriaceae*, comprising about 63 genera. *Salmonella* has a wide host range, occurring in mammals, birds, reptiles, amphibians, fish and invertebrates. The genus has two taxonomic species, based on differences in their 16S rRNA sequence analysis, namely Salmonella enterica and *Salmonella bongori* [1, 2]. *S. enterica* has six subspecies namely subspecies *enterica*, *salamae*, *arizonae*, *diarizonae*, *houtenae*, and *indica* [3]. *S. enterica* subsp. *enterica* is the most common subspecies and predominantly infects warm-blooded animals. *S. bongori* species is usually found in cold-blooded animals and the environment but some are occasionally associated with human disease. *Salmonella* subspecies are further classified antigenically into serotypes, or serovars, of which there are currently close to 2700 [4]. Some *Salmonella* serovars are host adapted but the majority are not and can cause disease in a broad range of hosts.

In animals, host-adapted or typhoidal *Salmonella* serovars cause severe disease in the specific hosts, characterized by septicemia but generally pose no threat to other species including humans. The non-host -adapted serovars are generally carried asymptomatically in animals although in some cases they cause disease characterized by diarrhea. These serovars are zoonotic or potentially zoonotic. In humans, they cause non-typhoid salmonellosis (NTS) the 4th most important cause of gastroenteritis [1]. It is also one of the most important bacterial zoonotic diseases, estimated to cause, 155,000 deaths yearly worldwide [5]. Non-typhoidal salmonellosis is therefore not only a major public health concern worldwide but has great negative economic impact due to the cost of surveillance, investigation, treatment and prevention of illness [6]. It is transmitted to humans through the feco-oral route, mainly by consumption of contaminated raw or improperly cooked animal products. The major sources of infection are poultry, eggs and dairy products but contaminated fresh fruits and vegetables have also been recognized as vehicles of transmission [7, 8]. Other common sources of infection include pigs products and contact with companion animals, particularly dogs, cats and horses, as well as pet reptiles such as snakes and tortoises. Contamination of animal products with *Salmonella* can also negatively impact on food trade by limiting market access NTS organisms occur widely in the environment but the main reservoirs are rodents, reptiles and wild birds [9]. The wide host range further makes control of NTS challenging. Emergence of multidrug resistant *S. enterica* strains in animals due to misuse and over use of antimicrobial agents is an added complication. Since the majority of the human infections are acquired through the consumption of contaminated foods of animal origin, NTS from animals is likely to continue to be a threat to human health. This chapter will discuss salmonellosis in food and companion animals, the associated public health risks, the challenges facing its control and future research needs. The discussion material is derived from existing literature as well as personal experience. It is hoped the chapter will be found useful by students, researchers, practitioners and managers of animal and public health.

2. History of salmonellosis

Salmonellosis has been around for centuries. It has been determined, through recent technological development, that typhoid fever was implicated in a plague which wiped out a third of the population in the city Athens, around 430 B.C. [10]. The organism *Salmonella* is named after Daniel E. Salmon, an American veterinarian. It was named in his honor by his research assistant, Theobald Smith, who isolated the first known strain of *Salmonella* from a case of hog cholera, which he named *Salmonella choleraesuis,* in 1885 [11]. The first study of *Salmonella* in humans was conducted by Karl Joseph Eberth, a German pathologist and bacteriologist, when he described a bacillus that he suspected was the cause of typhoid. The findings were later confirmed by pathologist Georg Theodor August Gaffky and the organism name "Gaffky-Eberth bacillus", which today is known as *S. enterica* serovar Typhi [12].

Notable personalities thought to have died from *Salmonella* infection include a US president, William Henry Harrison [13] and one of the famous Wright brothers, Wilbur Wright. The importance of typhoid as a scourge in human health is exemplified by the story of "typhoid Mary "(Mary Mallon), the domestic worker who transmitted *Salmonella* to at least eight households [14].

3. Etiology of salmonellosis in animals

3.1 Classification and nomenclature

Salmonella genus is classified under the family *Enterobacteriaceae* comprising about 63 genera. Their natural habitat is the intestinal tract of animals, of which about 25, such *Shigella, Salmonella, Enterobacter, Klebsiella, Serratia, Proteus* as are clinically significant. Others, such as *Escherichia coli,* are considered part of the normal intestinal microbiota and cause disease only incidentally. Phylogenically, there are only two species in this genus, *S. enterica and bongori bongori* [1, 2], based on differences in their 16S rRNA sequence analysis. *S. enterica* has six subspecies (subspecies *enterica*, *salamae*, *arizonae*, *diarizonae*, *houtenae*, and *indica* [3].

3.2 Antigenic classification

Salmonella are further classified antigenically by the Kauffman and White classification system, which classifies the organism into serotypes on the basis of a common somatic (O), flagella (H) and capsular (K), antigens [15]. The (O) antigen is present in all serotypes and is a heat-stable component of the lipopolysaccharide (LPS) located in the outer cell membrane present in all Gram negative bacteria. The heat-labile H antigens are part of the flagella protein, flagellin, present in all motile *Salmonella* spp. Two different genes code for the flagella proteins and either or both may occur in a serovar. Only one gene is expressed at a time, hence a serovar may possess only one protein at a time and the cells are thus diphasic. The two proteins are designated as phase I and Phase II. Phase I antigens are specific to a serotype and confer serological identity whereas phase II antigens are non-specific [16]. The K antigens are heat-sensitive polysaccharides located in the bacterial capsule, which is rare among *Salmonella* serotypes. The human-restricted serovar Typhi and serovar Paratyphi C produce a variant of the K antigen, known as the virulence (Vi) antigen [17].

The antigenic structure of *Salmonella* is useful in identification of serovars. It is also a useful epidemiological tool in determining sources of infection and mode of spread [18]. In nomenclature of *Salmonella* serotypes, subspecies name is usually omitted. For instance, *S. enterica* subspecies *enterica* serotype *gallinarum* is shortened to *Salmonella* ser. *Gallinarum* or *Salmonella gallinarum* [1, 11]. There are about 2700 serotypes (serovars) so far identified [4], each having a unique combination of somatic O and flagella phase I and Phase II antigens. Over 50% of these serotypes belong to the *S. enterica* subspecies [11].

3.3 Cellular, cultural and biochemical characteristics

Salmonella species are Gram-negative non-spore forming large rods measuring 0.7–1.5 by 2.0–5.0 μm. They are motile by peritrichous flagellation with the exception of *S*. Gallinarum and *S*. Purollum. Capsulation in salmonella is limited to a few serovars such as *Salmonella typhi*. Salmonella are aerobic, facultatively anaerobic in gaseous requirements. Nutritional requirement is non-fastidious and they can be cultivated in simple media such as nutrient agar. The majority of *Salmonella* are lactose fermenters. Utilizing this characteristic, selective and differential media have been formulated for isolation, and identification. Such media include MacConkey agar, Salmonella-Shigella agar, brilliant green agar xylose lysine deoxycholate agar and Hektoen enteric [19].

Salmonella species have the ability of to utilize tetrathionate ($S_4O_6^{2-}$) as an alternative electron acceptor in anaerobic respiration. This confers the organism a selective growth advantage, a property that used for non-selective enrichment in cultures containing competitive bacteria [20]. Common to *Enterobacteria*, *Salmonella* are oxidase negative, catalase positive, nitrate positive and they metabolize glucose fermentative, often with gas production. Other biochemical properties used for identification of *Salmonella* include hydrogen sulfide production (except few serovars such as Salmonella paratyphi A, and *S. choleraesuis*), the ability to utilize citrate as a sole carbon source, decarboxylation of lysine and fermentation of dulcitol. *Salmonella* are negative for production of urease and indole, deanimation of phenylalanine or tryptophan and Voges–Proskauer reaction [21, 22].

3.4 Pathogenicity and virulence factors

Many virulence factors play a variety of roles in the pathogenesis of *Salmonella* infections. These factors enable the organism adhere to and colonize its host, invade host cell, survive and multiply in macrophages, secret toxins and evade or bypass host's defense mechanisms. The factors include capsule, flagella, fimbriae, adhesins, invasins, hemagglutinins, exotoxins and endotoxins [16]. The various virulence factors are encoded by gene clusters, referred to as *Salmonella* pathogenicity islands (SPIs), located in chromosomes, plasmids and transporons [23–25].

The polysaccharide capsular O and Vi antigens in no-typhoidal and typhoidal *Salmonella* are known to aid the organism evade host's defense by modifying the cell surface in order to inhibit host's cellular response [26, 27]. Flagella are possessed by majority of *Salmonella* serovars and are known to confer pathogenicity in addition to motility. Certain *Salmonella* serovars are able to evade or minimize the host immune response by antigenic variation of flagella antigens, from one phase to the other [24, 25]. Fimbriae are the most common adhesion factors in *Enterobacteria.* They facilitate adhesion of *Salmonella* not only to hosts' cells, thus enabling colonization, but also to surfaces and foods. They are also implicated in a variety of other roles such as biofilm formation [28], which serves to shield the organism from attack by host's defense systems.

Endotoxin or lipopolysaccharide (LPS) is located in the outer membrane of Gram negative bacteria. It is heat stable and is released only upon bacterial cell lysis. It plays a role in pathogenesis of *Salmonella* infection by evoking pyrexia, activating complement system and depressing lymphocyte function among others. Endotoxin also plays a part in septic shock that can occur in systemic infections [29, 30].

Exotoxins comprise of cytotoxins and the enterotoxins. Cytotoxins are associated with killing of the mammalian cells in vitro and probably play a role in none-secretory diarrhea [31]. There is limited information regarding the mode of action of enterotoxins of *Salmonella* but they are antigenically related to the cholera family of enterotoxins. They are associated with diarrhea disease, probably through stimulation of intestinal secretion [16].

Certain *Salmonella* strains are hemolytic, another important virulence factor mediated by HylE protein, a product of hylE gene, thought to play a role in the pathogenesis of systemic salmonellosis. The protein produces hemolysis in blood agar made from the blood of a range of animals, including humans, with certain blood types [16].

3.5 Host range

*Salmonell*a and salmonellosis occur worldwide in mammals, birds, reptiles, amphibians, fish and invertebrates. Majority of *Salmonella* are not host specific and can cause disease in a broad range of hosts but some are host restricted. *S. enterica* subsp. *enterica* is the most common and predominantly infects warm-blooded animals. In the subspecies *enterica*, serovars *typhi*, *paratyphi* and *hirschfeldii* are restricted to humans and cause typhoid and paratyphoid fever respectively. They have no significant animal or environment reservoirs. Serovars *pullorum* and *gallinarum* are restricted to poultry; *abortusovis* to sheep; *choleraesuis* to pigs; and *dublin t*o cattle [32, 33]. The rest of the serovars, referred to as non-typhoidal *Salmonella*, are zoonotic or potentially zoonotic, the most common being serovars *typhimurium* and *enteritidis* [1].

The other five *S. enterica* subspecies (*salamae, arizonae, diarizonae, houtenae* and *indica*) and *S. bongori* are usually found in cold-blooded animals and the environment but some are occasionally associated with human disease. All animal species are susceptible to *Salmonella* infection but clinical disease occurs more commonly in some and not others. Among domestic animals, poultry, cattle, pigs, poultry and horses show clinical disease but cats and dogs commonly do not [34, 35].

3.6 Isolation and identification

Isolation of *Salmonella* from samples with competing microbes involves an initial non-selective pre-enrichment followed by a selective enrichment. Selenite (SeO_3^{2-}) is inhibitory to coliforms and certain other microbial species such as fecal streptococci and is used for selective enrichment of *Salmonella spp* from both clinical and food samples. Selective enrichment is followed by plating onto selective agars, followed by biochemical and serological confirmation of suspect presumptive colonies [36]. Serogrouping by somatic and flagella antigens, can be achieved by using monovalent specific 'O', 'H' and 'Vi' antisera. Phage typing, immunomagnetic separation and ELISA-based assays are some of the screening methods developed to produce rapid results, especially from food and environmental samples [37]. Several PCR assays targeting various genes have also been developed for identification of *Salmonella.* These include the 16S rRNA, invA, agfA, viaB, hilA, sirA, ttr, bcfD and phoP genes, among others [38–40].

4. Salmonellosis in animals

*Salmonell*a has a wide host range that includes mammals, birds, reptiles, amphibians, fish and invertebrates. It is a major cause of morbidity and mortality in animals and also a major cause of economic loss in livestock. The main importance of non-typhoidal salmonellosis in animals is however its zoonosis, causing a major health, social and economic impact due to cost of surveillance, investigation and treatment. The major source of direct human infections is consumption of contaminated or infected foods of animal origin, particularly meat, eggs and dairy products, and direct contact with animals, particularly companion animals, mainly dogs, cats and horses. Rodents and wild birds are the main reservoirs of non-typhoidal salmonellosis for animals. This chapter wills therefore limit the discussion on salmonellosis to livestock food animals, companion animals, rodents and wild birds.

4.1 Salmonellosis in poultry

4.1.1 Etiology and transmission

Salmonellosis in poultry and other avian species is caused by serovars in the subspecies *enterica*. Two of the serovar, *S. pullorum* and *S. gallinarum* are avian host-specific and cause typhoidal salmonellosis, while other serovars cause non-typhoidal infections, the most important being S. *typhimurium* and Salmonella enteritidis [35, 41, 42] *S. enterica* subsp. *arizonae* is also recognized as a cause of paratyphoid but mainly in turkeys [43]. Although *S. pullorum* and *S. gallinarum* can infect a wide range of avian species, clinical signs are observed in a few, which include chickens, turkeys and wild birds such as quails and pheasants [42].

Transmission is horizontal via fecal-oral route and vertically via infected embrocated eggs. Transovarian infection in the egg results in subsequent infection in chicks or poults and is one of the most important modes of transmission of these two diseases. Some infected hens become asymptomatic carriers and continually transmit it to their progeny. This mode of transmission is particularly critical in hatcheries since it can result in widespread dissemination of the diseases. Transmission by cannibalism and through the respiratory tract has been reported. Humans constitute a big potential of disease introduction through mobility and duties. They can track infections on vehicles, footwear, clothing, hands and contaminated equipment. Mammals, particularly dogs, cats, rodents as well as insects can also act as mechanical transmitters [42, 44, 45].

Similar to typhoidal salmonellosis, non-typhoid salmonellosis in poultry is transmitted vertically or horizontally. *S. enteritidis* serovar has a particular preference for vertical transmission. Horizontal transmission occurs through fecal contamination of feed and drinking water and by penetration of microorganisms into the egg subsequent to fecal contamination. Infection can be introduced into a farm by humans through clothing, footwear, equipment and vehicles. Rodents and wild birds are a notable reservoir of paratyphoid *Salmonella*. They are attracted into poultry houses by left-over feed and contaminate the feed by fecal material [42, 46, 47]. Dogs and cats can also track *Salmonella* infections over long distances to contaminate farms.

4.1.2 Clinical signs

Both *S. gallinarum* and *S. pullorum* cause systemic disease but whereas the former affects birds of all ages, *S. pullorum* affects primarily young ones. Birds hatched from infected eggs may be found dead in the hatching trays. Young birds may die soon after hatching without any observable signs and most acute outbreaks occur in birds under three weeks of age. In mature birds, infection is manifested by decreased egg production, fertility, hatchability and by anorexia. Diarrhea, which is usually white or yellow, watery to mucoid, is common, with fecal pasting seen around the vent.

4.1.3 Post mortem lesions

Lesions from *S. gallinarum* and *S. pullorum* infections are characterized by septicemia, with inflammation of all internal organs, including intestines, and notably liver and spleen, which show classic gray granulomatous nodules [46]. Infected ovaries

may be misshapen and/or shrunken and follicles are often pedunculated, being attached to the ovary by fibrous stalks, while the abnormal ova may contain caseous material [45]. Impaction of oviducts, resulting in egg peritonitis, is also common [48]. Recently hatched chicks show signs of septicemia and omphalitis, a condition characterized by infected yolk sacs, often accompanied by unhealed navels. The yolk sacs usually contain creamy or greenish, caseous material [49–51].

In non- typhoidal salmonellosis, the highest morbidity and death rates are usually observed during the first 2 weeks after hatching. Infected adult birds are asymptomatic and do not present signs of the disease and main importance is human infection, through consumption of contaminated meat and eggs. In young birds, it may however cause enteritis with dissemination toward the spleen, lungs, liver, spleen, and kidneys [43]. An enlarged, friable liver, with necrotic foci, is common. Chicks infected transovariary will show signs and lesions similar to those in typhoidal salmonellosis.

4.1.4 Diagnosis, treatment and control

Diagnosis of salmonellosis in poultry is achieved through significant clinical signs, necropsy finding as well as isolation and identification of the organism. *S. gallinarum* and *S. pullorum* can be differentiated with biochemical, serological tests and PCR.

S. gallinarum and *S. pullorum* may survive for a long time, months or even years in the environment, which makes it difficult to eliminate them in infected poultry houses. Once a flock is infected, the amount of *Salmonella* can be reduced, but not completely eliminated and depopulation is usually the only option. Pullorum disease and fowl typhoid are notifiable disease in many countries under OIE guidelines. Both diseases can be controlled and eradicated by use of serological testing and elimination of positive birds but vaccines may be used to control the disease. The diseases have largely been eradicated from commercial poultry in developed countries. Various antibiotics can be used to treat clinical cases, but they do not eliminate the organisms from the flock. The two serovars are highly adapted to the host species, and therefore are of little public health significance [45, 51].

4.2 Salmonellosis in cattle

4.2.1 Etiology and transmission

Salmonellosis in cattle is caused mainly by *S. enterica* ser *dublin*. The serovar is adapted to cattle but can also cause infection in other species including human. It causes economic losses in cattle production and is also a threat to human health [52]. Other serovars can also infect cattle and indeed, majority of *Salmonella* isolated from cattle are the non–host specific [53]. *Salmonella* infection in cattle is most commonly acquired by ingestion of feed or water contaminated by fecal matter from other livestock, rodents and wild birds or by contaminated animal by-products. *Salmonella* are shed by clinically infected animals and contaminate feed, water, yards, and equipment. The bacterium is also shed in saliva, nasal secretions, urine and milk in cases of systemic illness. Aerosol transmission between animals is considered possible in closely confined production systems [53–55]. Probability of vertical transmission from a dam to fetus, with calves born already infected, has been proposed [56]. The outcome of infection is determined

by virulence of the serotype, dose of inoculum, degree of immunity and other stress factors.

4.2.2 Clinical signs

Salmonellosis in cattle affects all age groups causing both intestinal and systemic infection but is most severe in the young. Clinical presentations are highly variable and the differential diagnosis list is considerable [53]. Acute disease is characterized by fever, anorexia and diarrhea of varying degree. The feces may be foul smelling, and may contain varying amounts of blood, mucus, and shreds of intestinal lining. Lactation drops suddenly in dairy cows. Clinical signs may last up to a week and death is due to dehydration and toxemia. In newborn calves, the disease most commonly affects those that receive inadequate or no colostrum and signs may include central nervous system (CNS) signs or pneumonia, and death may occur in 1–2 days. Those calves that survive longer may develop complications such as polyarthritis, or gangrene of the extremities of limbs, ears and tail [57]. Pregnant cows may abort, either with or without other clinical signs [54, 57, 58]. Subacute disease is seen mainly in adult animals and signs may include mild fever, anorexia, diarrhea dehydration and weight loss. Chronic disease in manifested by low intermittent fever and anorexia. There may be watery diarrhea resulting in progressive dehydration and weight loss. The feces are usually normal or contain mucus or blood. Sick cows that recover may become carriers that shed *Salmonella* for varying periods of time and cause continuous new infections in the herd [59, 60].

4.2.3 Post mortem lesions

In animals that die peracutely due to septicemia, there may be no gross lesions other than extensive submucosal and subserosal petechial hemorrhages. In acute enteritis, seen mainly in calves, the small intestines typically shows a diffuse mucoid or mucohemorrhagic enteritis and the mesenteric lymph nodes are edematous, congested and greatly enlarged [58]. In adult cattle, chronic infection is characterized muco/necrotic enteritis, especially of the ileum, caecum and colon. The wall is thickened and covered with yellow-gray necrotic material overlying a red, granular surface. Characteristic "button" ulcers may be seen in the colon [61] and the mesenteric lymph nodes and spleen may be enlarged.

4.2.4 Diagnosis, treatment and control

Clinical signs of salmonellosis are indicative of infection but definitive diagnosis of infection involves isolation and identification of the organism. Response to antibiotic treatment is usually poor. Animals that recover from infection can remain carriers and shed bacteria intermittently or continuously for long, especially during stress periods such as transportation or calving. The carrier status can even progress to full blown clinical disease.

Control involves sourcing animals from disease-free herds in order to ensure a clean herd. New animals should be put on quarantine for at least 4 weeks. Continuous serological tests and fecal culture is recommended and positive animals culled. Control of rodents and wild birds, particularly in feeding troughs, is important. Routine disinfection of premises should be considered and aborting animals should be considered suspect, isolated, tested and culled. Vaccines are available as part of a prevention or control tool.

4.3 Salmonellosis in pigs

4.3.1 Etiology and transmission

Salmonellosis in swine occurs in form of two clinical disease entities, typhoidal and non-typhoidal. Typhoidal salmonellosis is mainly caused *S. choleraesuis.* This serovar is adapted to swine and do not commonly affect other animals including, humans. Non-typhoidal infections are caused by *S. typhimurium* and is the most commonly found serotype in pigs and a common source of food poisoning in humans Other serotypes that commonly infect pigs are *e*nteritidis, agona, d*erby, hadar* and h*eidelberg* [62, 63].

The main route of transmission is feco-oral, which is exacerbated by poor hygiene and overstocking [45]. Pigs start shedding the bacteria shortly after infection and can continue to shed up to 5 months after recovery from the illness. Feed ingredients of animal origin, are another important source of infection for pigs. Mechanical transmission can be effected by humans through tracking of infections on vehicles, footwear, clothing, hands and contaminated equipment [64]. *Salmonella* also localizes in the tonsils and can lead to nose-to-nose transmission [65, 66]. Piglets can also get infected by the sow through milk, although rarely [67]. In addition, transmission of non-typhoidal salmonellosis can occur indirectly through contamination of feed and water by infections carried in the intestinal tract of wild birds and rodents.

4.3.2 Clinical signs

S. choleraesuis infections may occur at any age, but are more frequent in growing pigs, between 8 weeks and 5 months old. Outbreaks are frequently associated with stress conditions such as overcrowding, transportation, weather, concurrent infectious diseases such as parasitism, and poor management [68]. The disease is manifested as an acute septicemia characterized by fever, depression and anorexia. Sudden death is quite common in the acute phase of the disease, with pigs showing signs of cyanosis on the extremities such as ears, nose and tail, due to septicemia [69]. Pigs that survive the acute phase will show signs of yellow diarrhea and coughing. The diarrhea is foul- smelling and may contain blood and mucus. The bacterium may cross the blood-brain barrier during the septicemia phase and cause meningitis and nervous signs may be observed, but rarely. Arthritis may also be observed subsequent to localization of the organism in joints. Sick pregnant sows may abort.

Morbidity in *S. choleraesuis* infection is usually low (less than 10%) but mortality is high. The organism may localize in the mesenteric lymph nodes and such subclinical carriers intermittently or continuously shed the organism in feces, particularly under stress conditions [70].

Clinical signs of *S. typhimurium* are not common in well managed commercial herds but can occur in stressed and immuno-compromised ones. The main symptoms are fever, anorexia, yellowish diarrhea, dehydration, prostration, and mortality [71]. Affected pigs may recover in a period of one week but re-infection is common within the next three to four weeks. Mortality is rare, but those animals that survive can remain carriers, and therefore a source of continuous infection, for up to five months after recovery.

4.3.3 Post mortem lesions

In pigs that die suddenly from *S. choleraesuis* infection, the most common lesion is skin cyanosis, particularly on the ears, feet and abdomen, accompanied by swelling of

the gallbladder, lymph nodes, spleen and liver. There may be necrotic foci in the liver, as well as icterus. Consolidative bacterial pneumonia will be observed in pigs that show coughing. In Pigs that show signs of diarrhea, intestinal lesions, mainly pseudomembranous inflammation of the ileum and button ulcers in the colon will be observed.

The most common macroscopic lesion in *S. typhimurium* infection in pigs is inflammation of the ileum, the caecum and colon. The inflammation is characterized the presence of yellowish necrotic pseudomembranes. Mesenteric lymph nodes may be inflamed and enlarged. Characteristic "button' ulcers may be observed in the colon [72]. Some cases of rectal strictures have been reported after clinical salmonellosis. In these cases, pigs cannot defecate and intestinal contents remain trapped in the intestines, creating severe distension.

4.3.4 Diagnosis, treatment, and control

Clinical signs and lesions found during necropsy can be indicative of salmonellosis but not diagnostic. A definitive diagnosis is achieved by isolation and identification of the organism from suitable samples such as lung, liver, spleen, kidney, or lymph nodes [73]. Isolation from the intestine or feces is often unsuccessful.

Clinical disease can be controlled my antimicrobial therapy early in the onset of the disease but this will not eliminate the pathogen. The prophylactic use of antimicrobial agents is also not recommended because of expense, and promotion of antimicrobial resistance. Vaccines are available for preventing infection but their efficacy is often disappointing [74]. However, good management and husbandry is the best method of preventing clinical disease. This involves, but is not limited to, proper cleaning and disinfection. All-in-all-out pig flow and rodent control should be part of management procedures.

4.4 Salmonellosis in companion animals

4.4.1 Salmonellosis in dogs and cats

4.4.1.1 Etiology and transmission

Numerous *Salmonella* serovars have been isolated from dogs and cats with *S. typhimurium* and *S. enteritidis* being the most common serovars. There are no host-adapted serovars identified in dogs or cats [75–77]. Most dogs and cats are asymptomatic carriers and prevalence of *Salmonella* in dogs is associated with raw feed diets and contaminated feed, due to indiscriminate feeding habits, including scavenging [76, 78]. Fecal shedding of *salmonellae* by dogs is also a possible source of infection for other dogs as well as humans [79]. Cats may get infection from eating birds and rodents [80].

4.4.1.2 Clinical signs

These are rare although some dogs and cats may manifest signs of septicemia, particularly in puppies and kittens or in adults stressed by debilitating concurrent diseases [76]. Acute gastroenteritis is the most common symptom. The signs include fever, anorexia, diarrhea and vomiting. The diarrhea may contain blood. Other syndromes may include pneumonia, pelvic limb paresis, or conjunctivitis. As enteritis progresses, abortion may occur in pregnant dogs and cats or they may give birth to

weak puppies or kittens. Recovered animals can continue to shed the pathogen in their feces and saliva due to localization of the organism in the lymph nodes.

4.4.1.3 Postmortem lesions

Description of post mortem lesions in dogs and cats is scarce but the most common is enterocolitis [81]. Other recorded lesions include liver necrosis [82], pyonephrosis [83], cholecystitis [84], hemorrhagic gastroenteritis [85] and pneumonia [57].

4.4.1.4 Diagnosis, treatment and control

Diagnosis is based on isolation of the organism in conjunction with significant clinical signs. A diagnosis is conclusive if the organism is isolated from a normally sterile site, such as blood or synovial fluid in a live animal or from tissues samples from postmortem examination. Isolation of *Salmonella* may not necessarily be a definitive diagnosis in healthy animals.

Treatment for a *Salmonella* infection is primarily supportive, to compensate for the fluid lost through vomiting and diarrhea. Depending on the extent of the infection, antibiotics may be required for septic cases to prevent shock. Control of fecal contamination is of primary importance. Dogs and cats should be fed uncontaminated and properly cooked food.

4.4.2 Salmonellosis in horses

4.4.2.1 Etiology and transmission

Salmonella abortusequi is an equine-adapted serovar and is associated with abortion in mares, neonatal septicemia, polyarthritis and testicular lesions in males [24, 25, 86]. Infections are common in Asia and African but rare in the rest of the world [87]. However, the most common serovar isolated from horses is S. *typhimurium* [88].

Salmonella abortusequi transmission is oral or venereal. Infection may result from ingestion of feed contaminated by uterine discharges from mares that have recently aborted or from carrier mares. Transmission from stallions to mare during mating is also thought to occur [89]. The infection may localize in the uterus and cause repeated abortion or infection of subsequent foals.

Transmission of *S. typhimurium* is primarily fecal-oral. Feed, water and environment are contaminated by organism excreted through feces of sick or carrier horses, birds and rodents. Acutely ill animals excrete large amounts of bacteria. Risk factors for development of disease include stress due to transportation, overcrowding, changes in feed, intense physical activity, deprivation of feed and water and surgical treatment. Antibiotic treatment has also been found to increase risk for symptomless carriers. Another source of infection is eating manure, especially in foals.

4.4.2.2 Clinical signs

Serovar *abortusequi* primarily affects the reproductive system. In mares, the main clinical sign is abortion, with no other evidence of illness. Abortion usually occurs at about the seventh or eighth month of pregnancy. Retained placenta and metritis are common sequel of abortion. Foals from infected mares may develop an acute septicemia soon after birth while those that survive longer may develop polyarthritis. Sign

of infection in the stallion include fever, swelling of the prepuce and scrotum, and arthritis. Epididymitis, orchitis and testicular atrophy are other abnormalities associated with infection [89] .

Equine salmonellosis caused by *S. tyhimurium* can be asymptomatic, but is commonly associated with fever and. Diarrhea that can progress to septicemia in young animals [90, 91]. Infected foals are more prone to clinical disease than adult horses. Diarrhea, often severe and watery, is the most common symptom. Other symptoms include fever, colic and poor condition. The infection is often self-limiting but some conditions may progress to septicemia, resulting in death. Septicemia leads to polyarthritis, and/or pneumonia. Laminitis is a possible complication of salmonellosis in horses, and is attributed to bacterial endotoxins.

4.4.2.3 Post mortem findings

Necropsy findings in cases of *S.* a*bortusequi* include placentitis manifested by edema, hemorrhages and areas of necrosis. Foals dying soon after birth will have nonspecific changes of acute septicemia. Polyarthritis is found in those dying at a later stage.

The main lesions in cases of *S. tyhimurium* infection in horses includes fibrinonecrotic or necrohemorrhagic enteritis, mainly in the large intestine (large colon and cecum) [90]. Other lesions reported are enterocolitis and meningoencephalomyelitis in foals [65].

4.4.2.4 Diagnosis, treatment and control

Salmonella a*bortusequi* can be isolated from the placenta, uterine discharges, aborted foals, and the joints of foals with polyarthritis. Serological diagnosis is possible since a high titer of anti- *Salmonella* agglutinins develop in mares about 2 weeks after abortion. *S. tyhimurium* may be isolated from fecal material but this is not reliable due to intermittent shedding of the bacteria.

Antimicrobial drugs recommended in the treatment of salmonellosis should also be effective against *S.* a*bortusequi* infection. However, antibiotics use may promote latent carrier state following recovery [92]. Isolation of infected mares and disposal of aborted material should be practiced to avoid spread of the infection and infected stallions should not be used for breeding. In areas where the disease is common, vaccination is also used as a control measure. The widespread use of vaccines is credited with the almost complete eradication of the disease in developed countries.

Antibiotic treatment of equine *S. tyhi*murium infection is not recommended, especially in cases of uncomplicated diarrhea, due to the risk of worsening symptoms, as a result of disruption of the normal intestinal microflora by the antibiotics. Instead, supportive treatment is recommended if necessary. A major problem in control is the long-term survival of the organism in the environment. Manure should be disposed of frequently and animals with diarrhea should be isolated. Rodents and wild birds control is advisable.

5. *Salmonella* from rodents and wild birds

"Typhimurium" comes from "murine" Latin for mouse, a rodent of the subfamily Murinae. Rodents and wild birds are the main reservoir for *Salmonella* in the environment. They carry the organism in their intestines, mostly asymptomatically,

which they transmit to food animals in the farm environment [16, 93]. Rodents are attracted to feed and shelter around livestock farms, particularly in intensive production systems [94, 95]. Apart from *Salmonella*, rodents are carriers of a variety of other diseases such as leptospirosis and plague [96]. The source for infection is rodents' droppings which contaminate feed and water but mice and rats can also carry disease-causing organisms on their feet and hair [97]. Chicken can also get infection from eating dead mice and rats [94].

Salmonellosis in wild birds can be asymptomatic or it can be a fatal disease [98]. Asymptomatic birds may disseminate *Salmonella* to susceptible individuals through fecal shedding, shared environments, and via direct contact [99]. Birds can also transmit *Salmonella* to food animals with their feet [100]. Wild birds are particularly hazardous since they can transmit infections over long distances through migration. The most frequent serovar isolated from wild birds is *S. typhimurium* [101].

6. Public health importance of salmonellosis in animals

Non-typhoidal salmonellosis is one of the four major global causes of diarrheal diseases in human, alongside *E. coli*, Cholera and *Campylobacter* [1]. It is also one of the most important bacterial zoonotic diseases, estimated to cause, 155,000 deaths yearly worldwide [5]. Non-typhoidal salmonellosis in humans is therefore not only are major public health concerns worldwide but great negative economic impacts due to the cost of surveillance, investigation, treatment and prevention of illness [6]. It is transmitted from animals by the fecal-oral route in several ways:

1. Direct contact with infected animals. *Salmonella* is an occupational hazard for those working or living with animals [102]

2. Consumption of contaminated raw or undercooked animal products

3. Consumption of foodstuff cross-contaminated by contaminated animal products

4. Consumption of foodstuffs such as vegetables contaminated my fecal material or untreated manure from infected animals

6.1 Transmission from poultry

Poultry meat and eggs are the most common vehicles of salmonellosis to humans [7, 8] and *S. enteritidis* is one of the most commonly identified serovars in association with human infection [103] .Contamination of poultry products can occur at multiple points in the production chain. This includes during rearing, live birds transportation, slaughter, dressing and packaging [104]. During slaughter, fecal contamination of carcasses can occur from gut contents. In retail outlets, including butcheries and supermarkets, poultry meat can get contaminated or cross contaminate other products [105–107]. Leaking poultry packages can contaminate ready-to-eat foodstuffs in supermarket refrigerators and in the kitchen, poultry meat can cross-contaminate other foodstuffs during meal preparation [108], particularly, foodstuffs that are eaten raw such as fruits and salads. Eggs are important sources of *Salmonella* for humans. Eggs become contaminated either by fecal contamination of the eggshell or through transovarian transmission from infected hens [109, 110], and this can lead to human

disease after consumption of the contaminated eggs. Another potential source of food contamination is poultry manure which can contaminate vegetables in the field [111].

6.2 Transmission from cattle, goats and sheep

Milk and dairy products are the second most important source of *Salmonella* infections for humans. Salmonellosis from dairy products is usually related to consumption of raw or inadequately pasteurized milk although *Salmonella* may contaminate dairy products after the pasteurization process. Milk may be contaminated by cow fecal material or manure during milking. The pathogen is shed in the feces of cows and can be present in or on the udders of cows and contaminate their milk. Unpasteurised milk and products made from it such as ice-cream, cheese, milk powder and infant formulae have been associated with *Salmonella* outbreaks [112, 113]. A variety of *Salmonella* serotypes have been isolated from these products. S. *dublin*, which is highly adapted to cattle as the primary host, has been associated with systemic form of salmonellosis in humans [52].

Goat meat, mutton, beef and beef products are recognized as important sources of human salmonellosis [114–116] Infections in most cases are associated with the consumption of raw meet, contaminated cooked meat or as a result of inadequate cooking. Organs and carcasses become contaminated with intestinal contents during slaughter and this is considered one of the important sources of infection [11]. Untreated manure can also contaminate vegetables at production stage [1, 117].

6.3 Transmission from pigs

Pork is ranked as the third most common source of human salmonellosis and *Salmonella* is the most common zoonotic pathogen affecting swine associated to human gastroenteritis [118, 119]. Many *Salmonella* serotypes are present in pigs, but the most commonly associated with foodborne illness in human is *Salmonella typhimurium*. One serotype, *S. choleraesuis* is adapted to swine as the primary host but also causes severe systemic illness in man [120], although it is not commonly isolated from pork. The most common cause of infection is eating improperly prepared or stored pork products that are contaminated with *Salmonella*.

6.4 Transmission from companion animals

Close contact between dogs and cats and their owners or those working with dogs can also be a potential source of *Salmonella* infections for humans [121, 122]. Organisms shed in the animal's feces can contaminate human food or hands. *Salmonella* shedding by dogs and cats has been incriminated in infections in humans living in the same household with the shedding pet, with children accounting for a high proportion of cases. Other persons that are particularly vulnerable are the aged and the immuno-compromised. Transmission of *Salmonella* from horses to humans in contact has also been documented [123].

7. Conclusion

Microorganism will always be with us [124], and in absence of effective control, salmonellosis in animals will continue to be a major economic and public health concern for several reasons:

1. Increased intensification of livestock production will enhance animal to animal and animal to human contact and facilitate transmission of *Salmonella* from animal to animal and from animal to human.

2. Challenges of biosecurity in intensive production systems.

3. Increased commercialization of animal food processing and marketing which will enhance food contamination and transmission to humans.

4. Zoonotic *Salmonella* serovars have a very wide host range and therefore difficult to control.

5. Emergence of multidrug resistant *S. enterica* strains in animals due to misuse and over use of antimicrobial agents.

Control of salmonellosis must therefore be addressed from these perspectives.

Salmonellosis in farms is spread by contact between animals, from the environment and from reservoirs, particularly rodents. Since the primary infection with *Salmonella* occurs at the farm level, on-farm control of *Salmonella* is critical in reducing transmission during production, thereby minimizing contamination of meet during slaughter and processing and therefore reducing food safety risks [125]. Design and implementation of innovative biosafety practices are needed. Although cleaning and disinfection are the main hygiene practices in livestock production, they are less effective in the presence of rodents. A central part of hygiene practice should therefore include rodent control. This should include design of farm structures so as to eliminate rodent breeding sites and to prevent entry of the pests into animal houses. It has been shown that even the smallest population of rodents on farms presents a hazard [94]. Innovative, safe and efficient methods of rodents control in farm structures, including use of natural predators such as barn owls, are need.

Vaccination is the most cost-effective method for prevention and control of animal diseases and the most widely used tool in veterinary medicine. It can play an important role in prevention of salmonellosis in food animals Although vaccines against *Salmonella* in various animal species are in use worldwide, their efficacy is limited probably due to the diversity and complexity of pathogenesis of *Salmonella* infections. There is need for research into more efficacious vaccines against *Salmonella.*

The close contact between companion animals and people constitutes a risk for transmission of salmonellosis particularly for children, the aged and the immunocompromised. Studies are required to determine the extent of human salmonellosis attributable to companion animals and to identify risk factors for transmission. Sensitization of animal owners, caretakers and animal and human medical practitioners on risks associated with companion animals is important.

The wide host range of NTS implies that the risk of infection for any host is high. Measures to prevent disease in animals and humans must therefore be directed at all *Salmonella* serovars. Surveillance systems designed to map the spread and identify sources of infection, particularly in humans will be of great value in control of infections.

Salmonella is a complex genus that has evolved intricate virulence and antimicrobial resistance mechanisms and uncontrolled and indiscriminate use of antibiotics has increased the isolation frequency of *Salmonella* serovars resistant to one or more antibiotics globally [6]. Non-therapeutic use of antibiotics in farms is a threat

to human and animal health since majority of the human infections are acquired through the consumption of contaminated foods of animal origin. It has been demonstrated that sub-therapeutic use of antibiotics in animals may even trigger the spread *Salmonella* infection throughout a herd [125]. Whereas the global movement toward barn of antibiotics use in animals is encouraging, one of the major causes of uncontrolled antibiotic use is the commercialization of manufacture, distribution and retail of antibiotics. A significant misuse of antibiotics in humans is therefore likely to continue in absence of stringent regulation supported by surveillance data. Ongoing research on methods of blocking development of antibiotic resistance in bacteria by preventing mutation, is encouraging.

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