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Chapter 2

Salmonellosis in Animals

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Additional information is available at the end of the chapter

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Abstract

Salmonella has long been recognized as an important zoonotic pathogen of economic importance in animals and humans. The prevalent reservoir of *Salmonella* is the intestinal tract of a wide range of domestic and wild animals which may conclude in a diversity of foodstuffs of both animal and plant origin becoming infected with faecal organisms either directly or indirectly. In spite of mounting concerns about other pathogens in recent years, *Salmonella* remains among the leading causes of food-borne disease throughout the world. Lots of both domestic and wild animals are infected by *Salmonella* spp., mostly harboring the bacteria in their gastrointestinal tracts with no obvious signs of illness. Therefore, *Salmonella* are usually present in faeces excreted by healthy animals and many times pollute raw foods of animal origin through faecal contact during production and slaughter. The organism may also be transmitted through direct contact with infected animals or humans or faecal contaminated environments. Infected food handlers may also act as a source of contamination for foodstuffs. Because of increasing antibiotic resistance of organism and companion animals, animals are important source of *Salmonella* infection for human. The organism can be monitored and precautions should be taken regularly by new technological methods.

Keywords: salmonellosis, animals, zoonosis

1. Introduction

Salmonella enterica subspecies *enterica* can be separated into more than 2400 antigenically different serovars and the pathogenicity of most of these serovars is unspecified. The greater number of incidents of salmonellosis in humans and domestic animals originated from relatively few serovars and these can be separated into three groups on the basis of host prevalence. Host-specific serovars are the first group. These typically result in systemic disease in a small number of phylogenetically connected species. For example, *S. enterica* serovar

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Abortus ovis, serovar Paratyphi and serovar Pullorum are almost exclusively associated with systemic disease in sheep, fowl and humans, respectively. Host-restricted strains are the second group. These are mainly connected with one or two closely related host species but may also unusually result with disease in other hosts. For instance, *S. enterica* serovar Choleraesuis and serovar Dublin are generally associated with severe systemic disease in pigs and ruminants, respectively [1]. Nevertheless, these serovars are possibly efficient of infecting other animal species and humans. The third group comprises of the extensive *S. enterica* serovars, such as Infantis and Enteritidis that usually induce gastroenteritis to a large extent of unrelated host species. Obviously the nature and rigidity of *Salmonella* infections in different animal species varies hugely and is affected by many factors including the *Salmonella* serovar, dose, age, strain virulence, host animal species, immune status of the host and the geographical region [2].

Salmonella enterica subsp. *enterica* remains a main cause of infection and disease in human and animals worldwide. Much of the public health and economic problem originated from diseases or infected animals carriage. In Europe, animal salmonellosis as a cause of human infection became increasingly important as agricultural production started to intensify after World War II. In the 1950s, the rapid intensification of the poultry industry in numerous countries was supported by importation of dried fish meal from South America which comprised many *Salmonella* serovars. So, non-typhoidal salmonellosis is one of the leading causes of acute bacterial gastroenteritis in the USA, responsible for an estimated 1.4 million cases of illness annually. Widespread commercial distribution of contaminated foods can sometimes involve huge numbers of consumers in *Salmonella* outbreaks. For example, a 1994 S. Enteritidis outbreak associated with ice cream in the USA affected 224,000 people. *Salmonella* outbreaks can particularly have severe consequences for highly vulnerable populations in facilities such as day care centres and nursing homes [3, 4].

Although the genus *Salmonella* consists of more than 2400 serovars, most human cases of salmonellosis in the USA are caused by 5–8 serovars. United States (US) Centers for Disease Control and Prevention (CDC) reported that approximately 60% of human cases were caused by *Salmonella enterica* ser Enteritidis (24.7%), S. ser Typhimurium (23.5%), *S* ser Newport (6.2%) and *S* ser Heidelberg (5.1%). These same four serovars represented 46.4% of the isolates from nonhuman sources that year. Also serotypes are changing with time, for example, CDC reported that many of *Salmonella* serotypes decreased in incidence compared with 2012, infections caused by serotype 4, [5],12;I:- continued to rise [5].

Salmonella ser Enteritidis infections are mostly seen with fresh shell eggs and egg products, in which the bacteria contaminate the interior essences of the egg through transovarial infection. *Salmonella* ser Enteritidis infects the ova or oviduct of the hen's reproductive tract, which causes contamination of the albumen, vitelline membrane and possibly the yolk. Internal contamination of the egg's content performs egg-sanitizing practices, which focus on decreasing pathogen contamination on the eggshell surface, ineffective.

Salmonella Typhimurium definitive phage type DT104 appeared in the early 1990s as the dominant type of *Salmonella* spp. Most isolates have chromosomally encoded resistance to

five antimicrobials, specifically sulfonamides, chloramphenicol, ampicillin, streptomycin and tetracycline (R-type ACSSuT). There is sign that some penta-resistant DT104 strains are also evolving resistance to quinolones and trimethoprim [6]. Evidence in Europe indicates that the emergence of DT104 in cattle was the harbinger to its spread to other animals used for food production [2].

Although DT104 is currently the dominant penta-resistant clone of *S* ser Typhimurium, many other phage types (DT29, DT204, DT193 and DT204C) of this serovar have also be seen with multi-drug resistance. Understanding the causes that influence the emergence of these prevalent serovars of *Salmonella* spp. and the factors leading to the distribution and persistence of *Salmonella* spp. in animals is beneficial for the occurrence of effective intervention strategies to decrease human exposure to *salmonellae* [7].

Forms of livestock production and movement are varying as the world is changing. Advanced wages in the West conclude in increased production and importation of poultry meat and processed products from countries in South America and Asia. An improved standard of living in many countries is attended by increased meat ingestion, chiefly pork and poultry but also beef and dairy yields. Regulation of meat production in many countries is improving but there are presently large problems of antibiotic resistance which is enhancing a global problem. Poor control and hygiene conclude in the transmission of many microorganisms of which *Salmonella* is just one. Other changes connected with increasing living standards in world contain the increasing importance of companion animals in people's lives which are adequately recognized as sources of infection. Correlated to global changes in trade and human populations, improvements in technology have allowed us to obtain an unprecedented understanding of the biology of *Salmonella* [7].

However, many aspects of *Salmonella* biology and infection biology remain tantalizingly unresolved after the last 10 years of research, and more than 50 years after Professor Buxton's book [8] acted, such that the *Salmonella* should stay the centre of worldwide investigation activity for many more years. In many details the study of this organism is now a global project. Shrinking investigation budgets in the West have been changed with increasing concern in those countries with increasing budgets and where a value of the animal and public health *Salmonella* problem is increasing [7].

2. Infection in animals

Salmonella infections occur in lizards, snakes and turtles (including tortoises), in birds such as parrots, canaries, finches and pigeons and in mammals such as dogs and cats. They are less common in small caged animals. In dogs, cats and reptiles, infection may be unapparent and *salmonellae* can be found in the faeces of normal animals. These organisms can live happily in the intestine of some animals. They are called carrier animals. *Salmonella* infections most often cause enteritis and diarrhoea. The bacteria can also invade the body to cause septicaemia. This invasion results in fever that commonly accompanies the enteritis caused

by *Salmonella* infection. Affected animals are lethargic, do not eat and have diarrhoea. The diarrhoea is often not distinguishable from that caused by other microbes. The diarrhoea may be profuse and normally house-trained dogs and cats may become incontinent and foul the house unintentionally. In birds, the illness can be less apparent and may only be seen as pasting of the vent.

Very young, old or immunosuppressed animals or birds may be severely affected by the dehydration accompanying the diarrhoea, develop septicaemia or even die. Survivors may have diarrhoea for a time, but most go on to recover completely. Any recovering animal may be a carrier for a varying length of time. The organism can live in the gut lining in small numbers and within local lymph nodes, particularly in the lymphoid areas such as the caecum of birds. Persistence inside the animal can lead to reappearance of infection if the animal develops a different disease [9].

3. *Salmonella* infections in the domestic fowl

Four diseases induced by *Salmonella* are significant in poultry; pullorum disease caused by *Salmonella enterica* serovar Pullorum, fowl typhoid (FT) caused by *S*. Gallinarum, paratyphoid caused by several serovars and subspecies of *Salmonella* most particularly *S*. Typhimurium, *S*. Enteritidis, S. Infantis to name a few and arizonosis caused by *S. enterica* subsp. *arizonae* [7]. The poultry's specific *S. enterica* serovars Gallinarum and Pullorum have mostly been eradicated from the industries of Europe and North America. Nevertheless, in parts of the world with less developed industries, and especially in systems with poor biosecurity, these serovars still represent larger threats to bird health and welfare. Even though chickens are the normal hosts of *S. enterica* serovars Gallinarum and Pullorum, natural outbreaks induced by these serovars have been explained in turkeys, guinea fowl and other several species. There are many sources of infection in poultry containing vertical transmission, contaminated feed and the environment. Asymptomatic excreting of *Salmonella* from the intestine causes the contamination of eggs concluding in vertical transmission. As soon as after hatching, oral intake by the chicks results in very high numbers of *Salmonella* in the gut and great shedding in the faeces. This causes rapid horizontal spread around the hatchery [2].

Domestic fowl compose one of the largest reservoirs of *Salmonella* and is significant as a risk to public health through consumption of polluted eggs and meat. Arizonosis caused by *S. enterica* subsp. *arizonae* is an egg-transmitted infection mainly of young turkey poultries that still happens sporadically in commercial flocks and which may as well infect and unusually induce disease in chickens or other species of birds. Reptiles can be a reservoir of *S. arizonae* for birds and for man. The bacteria to place in the ovary and oviduct of breeder turkeys and the poults hatched from infected breeders develop disease. The disease is described by diarrhoea with pasting of faeces in the vent, huddling near the heat source, anorexia and boosted mortality sometimes accessing 50% [10].

4. *Salmonella* infection in poultry

Poultry products are frequently identified as important sources of *salmonellae* that cause human illness. An estimated 182,060 Americans became infected with S. Enteritidis during 2000 after consuming contaminated eggs [11]. Approximately 80,010 of S. Enteritidis outbreaks occurring in the USA between 1985 and 1999 with an identified food source were attributed to eggs [12]. Eating contaminated chicken has also been identified as a significant risk factor for S. Enteritidis infection [13]. Illustrating the importance of poultry as a reservoir for the transmission of *salmonellae* to humans, many of the serotypes that are most prevalent in humans (such as S. Typhimurium and S. Enteritidis) are also found common in poultry [4].

The ability of *Salmonella* to cause disease in poultry is closely related to the infecting serovar and the age and genetic background of the bird. Fowl typhoid (FT) is a disease caused by *S. enterica* serovar Gallinarum that is usually transmitted by the oro-faecal route and mainly affects adult birds [2]. The first described outbreak of FT was characterized by high mortality, especially during the first 2 months of the outbreak [7]. The pullorum disease (PD) is caused by *S. enterica* serovar Pullorum, is egg transmitted and occurs primarily in the first few days of life, high numbers of dead-in-shell chicks are seen (white bacillary diarrhoea). The ability of serovars other than Gallinarum and Pullorum to cause disease is relatively poorly understood [2].

Poultry may be infected with a wide variety of *Salmonella* serovars with the infection largely confined to the gastrointestinal tract with faecal excretion [7]. *S. enterica* serovar Typhimurium is primarily known for producing clinical salmonellosis in very young birds. Mortality rates vary enormously, from less than 10% to more than 80% in severe outbreaks. Resistance to infection develops rapidly over the first 72 hours of life and has been attributed to maturation of macrophages and the development of a commensal flora in the gut leading to competitive exclusion of *Salmonella* [7]. Strains of *S. enterica* serovar Enteritidis are also highly virulent for young chicks [14]. *S. enterica* serovar Enteritidis, and in particular strains of phage type 4 (PT4) can also cause asymptomatic and chronic infections in older birds including commercial layers and broiler breeders [15–17]. Epidemiological data demonstrate a clear association between food poisoning caused by serovar Enteritidis PT4 and the consumption of undercooked eggs [18]. The extent to which egg contamination occurs before or after egg formation is unclear [2].

Many *S. enterica* serovars have been associated with food poisoning in humans, however the potential for such serovars to infect poultry has been little studied in controlled experiments. A chick isolate of *S. enterica* serovar Kedougou colonized the gut, but did not intrude on the mucosa of tentatively infected day old chicks [19]. Likewise, strains of serovars Heidelberg, Senftenberg, Infantis, Montevideo and Menston all expeditiously colonized the intestines of youth birds, but were less invasive than a strain of serovar Typhimurium [20]. Lately, the virulence of various different serovars of *Salmonella* was evaluated in day old specific pathogen-free chicks. The host-specific serovar Pullorum affirmed to be the most virulent, pursued by the omnipresent serovars Typhimurium and Enteritidis. Three out of the four strains of serovar Heidelberg made low levels of mortality, whereas birds infected with isolates of Kentucky, Hadar and Montevideo all lived. Nevertheless, these latter serovars all colonized

the intestines expeditiously and caused a reduction in body weight, showing that subclinical *Salmonella* infections can even be harmful to bird health, welfare and productivity [21]. The reasons why such serovars are clearly much less virulent in chicks, yet retain the ability to induce human food poisoning are not seen [2].

5. *Salmonella* infections in cattle

Salmonella infections are an important cause of mortality and morbidity in cattle and subclinically infected cattle are frequently found. Cattle thus constitute an important reservoir for human infections. There have been numerous reviews over the years [22] increasingly reporting about multi-drug resistant strains [23] as well as the importance of *Salmonella* for the food industry. Interestingly, despite decades of research into salmonellosis, the disease and its public health consequences are not really resolved [7]. Salmonellosis occurs worldwide in cattle and has been associated primarily with serovars Dublin and Typhimurium. Other serovars are sporadically associated with bovine infections [2]. During the period 1968–1974, Sojka *et al*. [1] recorded the isolation of 101 different *Salmonella* serovars, usually at a low prevalence, detected annually in cattle [7]. Salmonellosis reached a peak in the British cattle industry in the 1960s with over 4000 incidents in 1969 [1, 2]. In the USA, 48% of the 730 *Salmonella*, other than S. Dublin and S. Typhimurium, isolated from cattle were represented by 7 serovars [24]. In the UK, in 2009, there was 10 *Salmonella* reports of non-GB origin reported from cattle, these included *S*. Typhimurium DT104, *S*. Mbandaka, *S*. Anatum and *S*. Dublin, clearly showing that importation of new strains remains a constant risk [7].

In the recent times, there has been a sharp reduction in the number of *Salmonella* outbreaks and over the last 5 years there have been only 400–500 cases annually, with similar numbers of events caused by *S. enterica* serovar Typhimurium and serovar Dublin in adult cattle and calves. *S. enterica* serovar Dublin and serovar Typhimurium are endemic in northern Europe, despite the divisions of these serovars vary. The origin of most outbreaks of salmonellosis in cattle is possibly faecal to oral contact. Infected cattle may excrete up to 108 CFU *Salmonella*/g of faeces and pollution of the environment in the nearness of other animals is a potent source of infection. Subclinical discharge of *Salmonella* aggravates the problem of pollution. Cattle that discharge an active *Salmonella* infection but show no clinical symptoms (often convalescing animals) are known as "active carriers". These may spread *Salmonella* constantly in quantity greater than 105 cfu/g of faeces and thus can be determined by routine bacteriological examination. Active carriage is commonly the sequel to clinical enteritis or systemic infection, and infected animals may excrete *Salmonella* for years or as well for life. "Passive carriers" are immunized animals that swallow *Salmonella* with feed and subsequently pass them in their faeces with no active infection of the intestines. Hence, when eliminated from a dirty environment these animals will stop excreting *Salmonella*. "Latent carriers", *Salmonella* remains subclinically in the tissues but is just randomly excreted in faeces [2]. Excretion may be initiated by stress, for example, at parturition. Understanding the biology of this true "carrier state" is likely to be key to ultimately controlling this important pathogen in cattle and may also provide insight into, for example, the asymptomatic carriage of *S. enterica* serovar Typhi by humans [7].

The spread of *S. enterica* serovar Dublin to reproductive tissues is not well understood and may originate either from a systemic infection or possibly from faecal contamination of the vagina. Adult survivors of *S. enterica* serovar Dublin infections often become latent carriers, a state which may last for life. The outcome of infection with other serovars seldom results in the latent carrier state although active excretion may continue for years. The reasons for this remain unclear [2].

6. *Salmonella* infections in pigs

The organism now known as *Salmonella enterica* serovar Choleraesuis was first isolated from pigs by [25], when they considered it to be the cause of swine fever (hog cholera). The ability of *Salmonella* to cause disease in pigs depends on numerous factors including the infecting serovar and the age of the pig. Regional variation in salmonellosis incidence is loosely correlated to pig density, husbandry practices and co-mingling of pigs [7]. The serovars of *Salmonella* associated with clinical disease in pigs can be divided into two groups: the host-restricted serovars typified by *S*. Choleraesuis and the ubiquitous serovars typified by S. Typhimurium. Then the existence of *S*. Choleraesuis has diminished dramatically and it is now only isolated sporadically. In contrast, this serovar stays a major threat to the pig industry in the USA. The fall of serovar Choleraesuis in the UK was not linked with any specific intervention measure. It was later understood that a diversity of antigenically distinct *S. enterica* serovars can be isolated from pigs, some of which are of zoonotic as they transferred through the food chain and farm environment to humans, where they typically cause acute but self-limiting gastroenteritis [8]. *S*. Typhimurium is the most usual serovar isolated from pigs both in Europe and in the USA. Likewise, S. Derby has a strong linked with pigs on both sides of the Atlantic Ocean, and for the past 20 years it has been the second most predominant serovar in pigs in the UK. Oral ingestion is thought to be an important route of infection as *Salmonella* are shed in high numbers in the faeces of clinically infected pigs.

Consistent results are only received applying a lower portion if the gastric pH is first neutralized with antacids [26]. This showed that the low pH of the stomach is a productive barrier to infection by *Salmonella*. Aspiration of infected material into the upper respiratory tract is another possible route of infection. Pneumonia is a general feature of S. Choleraesuis infections in pigs [27] and several works have shown that pigs can be experimentally infected by intranasal inoculation. Pigs infected with S. Choleraesuis via the intranasal route improve more severe clinical signals than those infected via the oral route [28]. Together these observations indicate that the tonsils and lungs are likely to be significant sites of invasion. Clinical salmonellosis in pigs is standardly of two forms; septicaemia caused by host limited *S. enterica* serovars such as Choleraesuis, and enterocolitis originated by broad host limit serovars such as Enteritidis. Unsurprisingly, weaned pigs that are intensively reared are most often influenced by *Salmonella* infections. Like other host-specific serovars, S. Choleraesuis has the capacity to induce disease in both young and older animals, whereas S. Typhimurium typically lead to disease in pigs aged between 6 and 12 weeks, but seldom in adult animals. In older animal, subclinical infections with S. Typhimurium are frequent, leading to high transmission rates if active carrier animals are not detected. S. Choleraesuis typically cause septicemic forms of infection. S. Typhimurium typically causes enterocolitis [2].

A year-long work during 2006–2007 determined *Salmonella* in the ileocaecal lymph node of 21.2% of pigs at slaughter in the UK, with S. Typhimurium by far the most dominant serovar. This correlated to a usual across Member States of the European Union of 10.3% [29]. European Community-wide it is estimated that 10–20% of human non-typhoidal salmonellosis may be linked to pigs [30]. In the USA, the most common serovars isolated from pigs during the National Animal Health Monitoring Survey in both 2000 and 2006 were Typhimurium, Derby, Agona, Typhimurium-Copenhagen and Heidelberg, three of which were also in the top five serotypes isolated from humans in the same period [31]. The number of investigation of some other serovars has developed during the last 20 years, but it is not understood whether this is the result of better monitoring or whether it indicates increased disease or environmental prevalence. It is evident that the problem of *Salmonella* in pigs is not limited geographically, and this is valuable considering the range of global trade in pork as personal countries are no longer isolated from world events [7].

7. *Salmonella* infections in sheep

In most countries of the world with a large sheep population, including the UK, Australia, New Zealand and the USA, sheep salmonellosis is apparently rare and does not represent a relevant economic issue. Disease distribution and prevalence of infections due to ubiquitous serovars is typically seasonal and associated with animal movement and shipping [32, 33]. Exposition to prolonged environmental stress, including cold, poor nutrition and concurrent diseases, might be important to activate latent infection and *Salmonella* shedding in faeces [33].

Serovar Abortus ovis strains, being host restricted to ovines, are expected to be introduced into a flock by an infected sheep and transmitted by the faecal-oral route [34]. There is no convincing proof of bacterial spread by water, feed or other host's faeces. Therefore, precaution has to be taken when transferring animals from a flock with history of infection into non-infected ones. Particularly, while many authors have published faecal shedding of culturable infectious bacteria up to 3 months following abortion [35], S. Abortus ovis DNA has also been detected in faeces up to 12 months from abortion [36], suggesting that sheep may be long-term asymptomatic carriers. Experimental infection studies have demonstrated that sheep may become infected by the conjunctival and vaginal routes [34, 35], but their significance in natural transmission has not been evaluated. Due to serovars Dublin, Abortus ovis and others induce pneumonia in young lambs, infection of grazing animals because of the nasal path might also be possible and respiratory secretion may distribute the infection to other individuals. High bacterial load in aborted foetuses and discharged placenta, elimination of bacteria with vaginal emissions following abortion and by scouring lambs are the main source of transmission throughout a flock during the lambing season [36].

Examination of slaughter-age healthy sheep and identification of *Salmonella* species have been often reported in the past few years, due to public health concerns of these serovars entering the human food chain [37]. Ovine salmonellosis might be an important zoonotic reservoir for human infection and a number of studies have reported food-borne transmission to humans [30–40].

8. *Salmonella* infections in horses

By the 1950s, *Salmonella enterica* serovar Abortus equi had disappeared from the USA following widespread use of bacterin and other control measures. The non-host adapted serovar S. Typhimurium was first recognized as a cause of colitis in 1919 [41] and has since dominated globally as a cause of equine salmonellosis. Antibiotic usage in combination with stressors associated with hospitalization has proved to be potent influences in increasing susceptibility of the horse to invasion by *Salmonella* spp. and in selection of resistant strains. Anorexia, antimicrobial administration, intestinal surgery and marked changes in diet increase the susceptibility of horses to *Salmonella* challenge [42].

Salmonella Abortus equi, the cause of equine paratyphoid, is the sole *Salmonella* host adapted for equids. A notable feature of the epidemiology of equine salmonellosis in the USA has been the rise and fall in incidence of infection by specific serovars. This may result in growing of herd immunity and/or reduction of virulence of the specific serovar. The latter may be conducting by the choosing pressure of antibody as herd immunity progresses. Topical spikes in the rate of isolation of particular serovars is often correlated with nosocomial outbreaks in local veterinary hospitals where in there is improved transmission. Control methods including closure of affected facilities will decrease the number of new cases finally providing to disappearance of the epidemic serovar.

The widespread dispersion of *Salmonella* spp. in wild and domestic animals and their environment is an important barrier to the persistence of a *Salmonella*-free horse population on a farm or following admission to a veterinary hospital. The origin of infection is often not understand in the first stages of an outbreak and so primary control efforts must be focused on rigid isolation of clinically problematical animals with diarrhoea or colic or those known to be shedding *Salmonella* spp. control measures on farms differ in some significant considerations from what are needed in a hospital environment [7].

9. *Salmonella* infections in dogs and cats

Carriage of *Salmonella* in dogs and cats may be asymptomatic, with intermittent shedding. Disease occurs intermittently, and ranges from mild to severe gastroenteritis, with occasional occurrence of abortion, systemic spread or septicaemia [43]. Recovered animals may shed *Salmonella* for several weeks, and chronic carriage with periods of recrudescence is possible. The challenges joined with making a diagnosis of bacterial associated diarrhoea in the lack of objective advices for faecal testing and the fact that identical isolation ratio have been found for presumed bacterial entero pathogens in some populations of animals with and without diarrhoea [44]. Both selective and non-selective serovars have potential for zoonotic spread, and may also be important in the emergence of antimicrobial resistance in the bacterial population [45]. Most of the infections were clinically silent, but mild diarrhoea without fever developed in only nine dogs from one kennel. Latest studies have demonstrated dogs eaten raw meat diets can go on to shed the organism in the faeces for a while time. Twenty-eight research dogs were entered to detect the prevalence of *Salmonella* shedding after ingestion of

a *Salmonella*-contaminated commercial raw food diet meal [46]. Cats have also been detected to carry *Salmonella*. Studies of the prevalence of *Salmonella* shedding in normal, asymptomatic cats have identified a prevalence typically of between 0.8 and 2.1% in cats [47, 48]. The epidemiology, prevalence, clinical signs, diagnosis and pathological findings and sources of salmonellosis in 100 cats in Scotland and England during 1955–2007 were reported [49]. Of the 49 isolates, 28 (57%) were from kittens less than 6 months of age. From the point of their function in the transmission of salmonellosis, cats were discovered to be the most abundant ecological section (125 of all samples positive) in a 2-year investigation of the circulation of *Salmonella* on 12 pig production units in the USA [50]. In addition, the presence of cats on the farm was identified as a significant risk factor for outbreaks of clinical salmonellosis on Dutch dairy farms [51]. Tauni and Osterlund [52] reported an outbreak of S. Typhimurium in cats and humans connected with infection in wild birds in Sweden in 1999. A total of 62 ill cats were investigated. Altogether were anorectic and lethargic, 31% had diarrhoea and 57% were vomiting. It was thought similar that salmonellosis was passed on from cats to humans, but there were just a few such cases. These studies indicate that *Salmonella* shedding is comparatively sporadic in cats and that clinical signals such as diarrhoea are not trusted predictors of whether a cat is potently shedding enteric organisms. Nevertheless, when infection does happen, cats may take part in a significant role in the transmission of the organism. That is, the prevalence of *Salmonella* spp. in healthy dogs and cats is very similar to the prevalence in diarrhoeic dogs and cats while the prevalence in stray or kennelled dogs and cats is often higher. The prevalence of *Salmonella* infection in kennelled or stray cats and dogs is often excessive. Most events of salmonellosis in dogs and cats are subclinical. Following contact to *Salmonella*, the organism is usually discharged by the host's immune system. Nevertheless, in a small rate of cases the organism may continue leading to the formation of a transmitter state. A small percentage of cases of human salmonellosis are related to contact with infected dogs and cats.

10. *Salmonella* infections in exotic pets

Reptiles are known to release *Salmonella* frequently [53] and reptile-associated salmonellosis has been recognized as an emerging zoonosis. From the epidemiological point of view [54] and in addition to an earlier recommendation ('Reptile-Associated Salmonellosis', RAS, [55] we suggest to call this particular type of epidemic 'Reptile-Exotic-Pet-Associated Salmonellosis' (REPAS). The primary statement for this proposal is that past several years the approach of trading reptiles has changed substantially and this will likely continue in the future. The particular risk of *Salmonella* dissemination from reptiles to humans is not due to European wild species but, as outcome of this study also demonstrate, at present is mainly due to 'exotic' imported reptile species. Moreover, following new investigations *Salmonella* shedding is higher in reptiles kept in captivity in comparison to wild reptiles [53, 56] and 'pet' reptiles are apparently in closer contact to humans. These arguments justify the inclusion of 'exotic pet' into the term describing the problem. The risk to human health connected with the reptile pet market has been highlighted recently [57] and the exact definition of the problem using REPAS might be significant to contribute the problem in education and support the European Commission to contribute suggestions to harmonize animal welfare and public health [7].

Each year infections are also obtained through direct or indirect animal contact in homes, farm environments, veterinary clinics, zoological gardens, or other public, professional or private settings. Clinically infected animals may propagate a higher prevalence of shedding than seemingly healthy animals, but both can exhibit *Salmonella* over long periods of time. Also, environmental contamination and indirect dissemination through contaminated food and water may complex control efforts. The public health risk varies by mammals, birds and reptile species, age group, husbandry practice and health status [58]. A study from Canada conducted between 1994 and 1996 illustrated the potential problem of reptile-associated salmonellosis for the first time. In 2011, a 13-month-old child from Austria passed away on the transport to the hospital with vomiting and diarrhoea. A multi-state outbreak in the USA in 2008 was associated with pet turtle exposure. In nearly half of the 135 cases, children ≤5 years were affected. This outbreak was the third turtle-associated outbreak since 2006 [59].

11. *Salmonella* detection

Diagnosis is based on the identification of the *Salmonella* either from faeces or from tissues collected aseptically at necropsy, environmental samples or rectal swabs, feedstuffs and food products; prior or current infection of animals by some serovars may as well be detected serologically. If reproductive organs are infected, abortion or conceptus occurs, it is essential to culture vaginal swabs, placenta, foetal stomach contents and embryonated eggs. Organism may be identified using a diversity of techniques that may include pre-enrichment to resuscitate sublethally damaged *salmonellae*, enrichment media that comprise inhibitory substances to inhibit competing organisms, and selective agars to differentiate *salmonellae* from other enterobacteria. Various biochemical, serological and molecular tests can be used to the pure culture to allow for a reliable verification of an isolated strain. Organism has antigens named somatic (O), flagellar (H) and virulence (Vi), which may be identified by special typing sera, and the serovar may be assignated by reference to the antigenic formulae in the Kauffman-White scheme. Many laboratories may require to send isolates to a reference laboratory to ensure the full serological identity and to verify the phage type and genotype of the strain, where suitable [60].

Serological tests should be carried on a statistically representative sample of the population, but results are not at all times signifier of active infection. In the laboratory, the tube agglutination test is the procedure of choice for export and diagnostic plans for samples from all species of farm animals. Enzyme-linked immunosorbent assays are usable for some serovars and may be used for serological diagnosis and observation, especially in pigs and poultry. Vaccination may risk the diagnostic worth of serological tests [60].

Since some of the common serovars such as *S*. Enteritidis and *S*. Infantis not only solely induce human infections but are also important livestock colonizers, the *Salmonella* subclassification needs more discriminative methods than serotyping. During the past 50 years, phage typing gets a very worthful device for epidemiological aims. The scheme for *S*. Typhimurium developed by Felix in 1956 (England) played a big role in many outbreak investigations and the

S. Enteritidis scheme from Ward [61] and Lalko/Laszlo [62] has been invaluable in the investigation of egg- and poultry-associated outbreaks that have been accomplished worldwide from the 1980s till today [7].

In 1929, White developed a typing scheme consisted on this antigenic chancing, which was afterwards changed by Kauffmann. This investigation allowed the separation of *Salmonella* into serovars. In 1934, the first Kauffmann-White scheme comprising 44 serovars was reported by Kauffmann and the *Salmonella* Subcommittee [63].

Phage typing supplies a worthful epidemiological work for greater sub-distinction of different serovars and is of exceptional importance in outbreak research. At the NRC, this method has been accomplished for serovars Enteritidis, Typhimurium and some others. Moreover, molecular techniques such as ribotyping (for *S*. Enteritidis) and pulsed field gel electrophoresis (PFGE) (for S. Typhimurium and others) are utilized to presumed outbreak isolates [7].

Whole of the methods; the gold standard diagnostic method for *Salmonella* is culture.

- Culture.

The culture techniques and media that may result best in a specific diagnostic condition subject to a variety of factors, including the *Salmonella* serovar, type and source of specimens, practice of the microbiologist, animal species of origin, availability of selective enrichment and selective plating media. *Salmonella* determination by bacteriological methods generally requires 5–11 days, and samples with low numbers of *Salmonella* cells, generally seen in subclinically infected chickens, may give false-negative results. The increasing application of external quality assurance programmes has led to larger use of international standard methods, such as ISO 6579:2002; [64] while this has not been validated for faecal and environmental samples and was intended for foodstuffs and feeding stuffs. Latest years a standard method for determination of *Salmonella* from primary animal production has been developed and assessed, and an ISO method (ISO 6579:2002 Annex D) has now been accepted (ISO, 2002). The core of the standard method is pre-enrichment in buffered peptone water, enrichment on modified semi-solid Rappaport-Vassiliadis (MSRV) and isolation on xylose-lysine-deoxycholate (XLD) and an additional plate medium of choice. This method has also been demonstrated to be greatly effective for animal feed and meat products, and is simpler and less costly than the full ISO method [61].

- Immunological and nucleic acid recognition methods.

Numerous alternative *Salmonella* detection methods have not been fully validated for faecal and environmental samples, although progress has been made [65, 66] and are more suited for analysis of human foodstuffs where inhibitors of the PCR reactions are not so problematic even though there is a role for quick methods in test and release of batches of *Salmonella*-free animal feedstuffs. The quick methods are generally more costly than conventional culture, but can be economically convenient for screening materials where a low prevalence of transmission is expected or where materials, such as feedstuffs, are held pending a negative test. An enrichment/IMS method associated with ELISA or PCR can identify most transmission within 24 hours but faecal and environmental samples can be problematic for quick methods. At present none of the quick methods has been proved to be acceptable for direct detection of *Salmonella*

so non-selective or selective enrichment stages are necessary [67]. Standardly, this introduces more actions and operator time in the detection procedure. For DNA-based methods, inhibition of the PCR reaction by components of the test sample substance, particularly in the case of faeces, is problematic and needs appropriate DNA extraction techniques and controls to determine inhibition, which may reduce the sensitivity of the test in some cases [65]. Quick isolation methodologies may also be linked with sophisticated detection systems, such as biosensors [68]. There are many variations and developments in rapid methods for *Salmonella* detection, but none has been shown to satisfactorily replace culture in all circumstances [60].

Salmonella enterica subspecies *enterica* is an interesting pathogen varying in its pathogenesis and virulence in different animal species. Some serovars have a broad host range and typically cause subclinical intestinal infections and/or acute enteritis. In contrast, host-restricted and host-specific serovars have narrower host ranges and associated infections tend to be of the more severe systemic form. By targeting the intestines and/or reproductive tracts of animals, *Salmonella* are disseminated between animals in high numbers concluding in maximum levels of disease and transmission. High costs are met annually by public health services and farming industries in monitoring and trying to control *Salmonella*. Knowledge of the pathogenesis of *Salmonella* infections in divergent animal species would support to discover measures to hinder the spread of these pathogens between animals. The mechanisms of pathogenicity of a *S. enterica* serovar have been mainly studied in rodent models of infection. However, the behaviour of these microorganisms in one particular animal species is not necessarily predictive of its behaviour in another host species. Therefore, the application of modern molecular genetics to strains of defined virulence, together with infection studies in natural target animal species will enable a more comprehensive understanding of the determinants *Salmonella* serovar host-specificity and of the biology of these pathogens in individual animal species.

S. Enteritidis, S. Infantis, S. Typhimurium and lots of serovars are most commonly connected with human illness. Human S. Enteritidis cases are most frequently related with the consumption of contaminated eggs and poultry meat, while S. Typhimurium cases are mostly associated with the consumption of contaminated poultry, pig and bovine meat. In animals, subclinical infections are common. *Salmonella* may easily spread between animals in a herd or flock without detection and animals may become intermittent or persistent carriers. All animal and human perform the below precautions to prevent from companion animals and other food-associated Salmonellosis. Clean and disinfect utensils such as food dishes, feed foods that are more likely to be free from *Salmonella* such as processed foods, for example, those that are tinned, packaged or bagged. If you are buying a pet ensure that it is healthy first, keep dogs away from carrion, animal faeces and prevent them from drinking suspected contaminated water as far as possible, consider any case of diarrhoea as a potential source of infection for other animals, make sure that diarrhoea is treated properly, always disinfect after cleaning up diarrhoea, consider all diarrhoeas in your pet as potentially infective, dispose of diarrhoea safely, wrapped and double polythene bagged into a bin, washed down the lavatory, burned or buried in a safe place after disinfection, disinfect the contaminated area, wash your hands after handling your pet at all times, do not allow infected pets to come into contact with young children, old people or those already ill and keep infected dogs away from food preparation area.

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