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Salmonella in Wild Animals: A Public Health Concern

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Abstract

Wildlife can be a reservoir of infectious agents for humans and domestic and wild animals. In this regard, widespread *Salmonella* spp. in wildlife is a problem for public and environmental health. Currently, more than 2500 serovars of *Salmonella* spp. are widely distributed among humans, animals, and the environment. This ubiquity favors the bidirectional transmission of the pathogen between wild and domestic animals. Moreover, when farmed animals acquire *Salmonella* spp. from wildlife, the likelihood of humans becoming infected increases. The risk is higher in forest environments impacted by human activities or when animals are removed from their natural habitat. Consequently, human contact with wild animals in captivity increases the risk of salmonellosis outbreaks. These animals are often carriers of *Salmonella* spp. strains multiresistant to antibiotics, which makes it difficult to treat and control the disease. Therefore, prevention and control measures of this pathogen must include both the pathogen-host relationship and the environment, with a surveillance system for emerging and re-emerging diseases from wildlife.

Keywords: wild fauna, *Salmonella* asymptomatic carrier, salmonellosis, anthropized forest environment, wild animals in captivity

1. Introduction

Natural environments have been altered by the destruction of forests and habitats to expand habitable zones for humans. These changes expose humans and animals to infectious agents that were restricted to certain species and geographical areas. Furthermore, these changes cause an epidemiological, sanitary, and environmental rearrangement of diseases, especially those with zoonotic profiles, as in the case of salmonellosis [1].

Salmonella spp. is a bacterium with pathogenic characteristics often associated with food infections and outbreaks, with serious public health implications. Infections can affect people, livestock such as cattle, pigs, sheep, poultry, pets, and even wild animals. Concerning wild animals, the characteristic ubiquity of the bacterium also favors cross-contamination to domestic animals, especially in areas for livestock close to forests.

Epidemiologically, one of the main characteristics of *Salmonella* is its condition as a latent carrier [2]. Latency corresponds to a state in which the individual does not present clinical symptomatology, but continues eliminating the agent intermittently in the

feces. Thus, these asymptomatic latent carriers become natural reservoirs and, consequently, maintainers of the pathogen both in the food chain and in the environment.

Naturally, wild animals can be asymptomatic carriers of *Salmonella* spp., with the bacterium remaining in equilibrium with the intestinal microbiota. When these animals are kept away from their natural habitat, the resulting stress compromises their immune system and destabilizes the microbiota, leading to increased elimination of the pathogen in feces. Therefore, wild animals kept in captivity tend to have a higher prevalence of *Salmonella* spp. than free-living animals, possibly leading to outbreaks of salmonellosis in humans due to cross-contamination by serotypes of *Salmonella* spp. This scenario is even worse when the serotype involved is multidrug-resistant to antibiotics.

The maintenance of wild animals in captivity is a major public health concern, especially in the case of reptiles. We conducted a study with fecal samples of 30 tegu lizards born in captivity that were asymptomatic latent carriers of *Salmonella* spp., with nine serotypes with resistance to at least two antibiotics being isolated [3]. In another study using 31 snakes kept in captivity, 58% tested positive for *Salmonella* spp. and seven serotypes were isolated [4]. Some of the animals, both among the tegus and the snakes, tested positive for more than one serotype with different resistance profiles. In preserved forest areas, the prevalence of *Salmonella* spp. in wild animals is usually lower. Our research team sampled 518 free-living wild animals in forest fragments (388 mammals, 114 birds, and 16 reptiles) from 2015 to 2021 in four mesoregions of Bahia (north-central Bahia, south-central Bahia, Metropolitan Salvador, and south Bahia), Brazil, and observed that only three mammals (unpublished data) and one bird [5] tested positive for *Salmonella* spp.

Notably, the manifestation of salmonellosis is associated with factors inherent to the etiological agent, the host, and the environment. The correlation between the three will determine the impacts on biosecurity and persistence of the bacterium in ecosystems, food, and carriers. The prevention and control of this pathogen demand interdisciplinary and international cooperation based on shared data to ensure a more effective approach to outbreaks.

2. General characteristics of the genus *Salmonella*

Salmonella is a genus of pathogenic bacteria named by Lignières in 1900, after the veterinarian pathologist and microbiologist Daniel Elmer Salmon, who isolated the agent and associated it with a disease for the first time [6]. These bacteria are part of the Enterobacteriaceae family and are morphologically composed of non-spore-forming, Gram-negative, facultatively anaerobic, rod-shaped bacteria, with optimum growth temperature between 35°C and 37°C [7].

Currently, *Salmonella* spp. is divided into two species: *Salmonella enterica* and *Salmonella bongori*. The first species is divided into six subspecies with a Roman numeral, as follows: *enterica* (serogroup I), *salamae* (serogroup II), *arizonae* (serogroup IIIa), *diarizonae* (serogroup IIIb), *houtenae* (serogroup IV), and *indica* (serogroup VI) [8, 9].

Salmonella bongori (serogroup V) has 23 serotypes and *S. enterica* has more than 2500 serotypes (*S. enterica* subsp. *enterica* serogroup I = 1547, *S. enterica* subsp. *salamae* serogroup II = 513, *S. enterica* subsp. *arizonae* serogroup IIIA = 100, *S. enterica* subsp. *diarizonae* serogroup IIIb = 341, *S. enterica* subsp. *houtenae* serogroup IV = 73, and *S. enterica* subsp. *indica* serogroup VI = 13 [10]. This characterization of species and subspecies into serotypes is based on the model proposed by Kauffman-White from differences observed in flagellar (H), capsular (K), and somatic (O) antigens [11].

Species	Salmonella enterica						Salmonella bongori
Subspecies	enterica	salamae	arizonae	diarizonae	houtenae	indica	
Dulcitol	+	+	—	—	—	*	+
Malonate	—	+	+	+	—	—	—
Gelatinase	—	+	+	+	+	—	+
Sorbitol	+	+	+	+	+	—	+
Galacturonate	—	+	—	+	+	+	+
Salicin	—	—	—	—	+	—	—

*Variable according to serovar.

Table 1.
Biochemical characteristics of *Salmonella* species and subspecies.

The species and subspecies of *Salmonella* also have distinguishing biochemical characteristics (**Table 1**). These bacteria are catalase-positive and oxidase-negative and can form hydrogen sulfide through the enzyme cysteine desulphydrase, which promotes sulfur reduction. Moreover, they can reduce nitrite to nitrate and use citrate as an energy source. In contrast, they do not produce indole or hydrolyze urea [6].

Salmonella is a bacterium of worldwide geographical distribution and, therefore, many animal species, including wild animals, can act as a reservoir of its various serovars [12]. Wild and domestic animals and humans can be affected by any of the more than 2500 different serovars [13]. *S. enterica* subsp. *enterica* determines infections mainly in warm-blooded animals [11], chiefly mammals [14], and is associated with most of the world’s foodborne diseases [11]. Nevertheless, different serovars of this subspecies have been isolated from exotic reptile kept as pets, as we will report throughout this chapter. The other subspecies of *S. enterica* are uncommon for humans and are usually found in cold-blooded animals and environmental samples [14]. Similarly, *S. bongori* is more common in cold-blooded animals, especially reptiles, and in the environment [6], but can also infect humans [15].

Salmonella habitat, based on the host’s specificity and clinical manifestations, can be characterized as follows: a. *highly adapted to humans*, corresponding to serotypes *S. Typhi*, and *S. Paratyphi A*, *B*, and *C*; b. *highly adapted to animals*, responsible for paratyphoid fever in animals, consisting of *S. Dublin* (cattle), *S. Choleraesuis* and *S. Typhisuis* (pigs), *S. Abortusequi* (equines), and *S. Pullorum* and *S. Gallinarum* (birds); and c. *zoonotic Salmonella*, which affect humans and domestic and wild animals indistinctly and are involved in food poisoning and gastroenteritis. This third group is more representative of public health due to its high morbidity and mortality rates [6, 9].

Notably, *Salmonella* spp. can survive in the environment, mainly in organic matter, and can continue infecting for 280 days in soils used for cultivation, 120 days in pastures, 30 days in bovine feces, and 28 days in bird fecal matter [6, 16]. Moreover, it adheres to the surface of plant roots and survives for long period underground [17]. This occurs because these bacteria, which inhabit the intestinal tract of humans and animals, are eliminated in the feces and can then contaminate both water and soil. Furthermore, in aquatic ecosystems, *Salmonella* can adhere to sediments [18] and survive in high densities in these systems and water after 56 days [19]. In this regard, sediments provide a protective layer for enteric bacteria from a nutrient reserve and prevent stress from the aquatic environment [20].

3. Salmonella spp. in wild animals

The increased demand for wild animals raised as domestic animals has become a public health and environmental concern due to the spreading of pathogens [21].

Class	Year	Animal	Pet (P) Free (F)	Salmonella serovar	Cases: numbers of Illnesses (I); Hospitalizations (H); Deaths (D)			Ref.
					I	H	D	
Reptiles	2021	Turtle	P	S. Typhimurium	87	32	01	[22]
	2020	Bearded Dragon	P	S. Muenster	18	11	00	[22]
	2020	Turtle	P	S. Typhimurium	35	11	00	[22]
	2019	Turtle	P	S. Oranienburg	26	08	00	[22]
	2017	Turtles	P	S. Agbeni	76	30	00	[22]
	2015	Crested Geckos	P	S. Muenchen	22	03	00	[22]
	2015	Turtle	P	S. Sandiego S. Poona	133	38	00	[22]
	2014	Crested Dragon	P	S. Cotham	166	61	00	[22]
	2014	Snake	P	S. Enteritidis	1*	1	00	[23]
	2012– 2013	Turtle	P	S. Sandiego S. Pomona S. Poona	473	78	00	[22]
	2009	Bearded dragon	P	Salmonella enterica subsp. houtenae 6,7:z4,z24:–	19	NI	00	[24]
	2009	Bearded dragon	P	S. Rubislaw	01	01	00	[25]
	2009	Bearded dragon	P	S. Apapa	01	01	00	[26]
	2009	Bearded dragon	P	S. Pomona	01	NI	NI	[27]
	2008	Turtle	P	S. Abony 4,5: b: enx	01	01	00	[28]
	2008	Snake	P	S. enterica subesp. Arizonae 41: z4, z23: –	03	01	00	[29]
	2007	Terrapin	P	S. Pomona	01	NI	NI	[30]
	2006	Bearded dragon	P	S. Apapa	03	NI	NI	[26]
	2005– 2008	Snakes	P	S. Paratyphi B biovar Java 4,5,12: b: 1,2 S. Morehead 30: i: 1,5 S. enterica subesp. Diarizonae 47: –: –	03	NI	NI	[29]
	2005	Turtle	P	S. Braenderup 6,7: e, h: e, n, z15	06	00	00	[29]
	2005	Turtle	P	S. Paratyphi B	01	NI	00	[31]
	2003	Turtle	P	S. Enteritidis	01	01	00	[32]
	2003	Snake	P	S. enterica subsp. arizonae	01	01	01 **	[33]
	2000	Water dragon	P	S. Rubislaw	02	01	01 ***	[34]
	2000	Iguana	P	Salmonella bongori sorovar 44: Z23	01	01	00	[35]
	2000	Iguana	P	S. Poona	01	NI	00	[36]

Class	Year	Animal	Pet (P) Free (F)	Salmonella serovar	Cases: numbers of Illnesses (I); Hospitalizations (H); Deaths (D)			Ref.
					I	H	D	
Amphibians	2011	Frog	P	S. Typhimurium	241	72	00	[22]
	2009	African dwarf frog	P	S. Typhimurium	85	00	00	[37]
	2001	Frog and toad	NI	S. Javiana	55	09	00	[38]
Small Mammals	2020	Hedgehog	P	S. Typhimurium	49	11	00	[22]
	2019	Hedgehog	P	S. Typhimurium	54	08	00	[22]
	2018	Guinea Pig	P	S. Enteritidis	09	01	00	[22]
	2014	Frozen Feeder Rodents	****	S. Typhimurium	41	06	00	[22]
	2012	Hedgehog	P	S. Typhimurium	26	08	01	[22]
	2010	Frozen Feeder Rodents	****	S. entérica subsp. enterica 4,[5],12:i:-	34	01	00	[22]
	2008– 2009	Feeder mice	****	S. Typhimurium DT191	12	NI	00	[39]
	2005– 2006	Frozen Feeder Rodents	****	S. Typhimurium	04	00	00	[40]
	2003– 2004	Rodent	P	S. Typhimurium	28	06	00	[41]
	2000	Hedgehog	F	S. Typhimurium	37	00	00	[42]
Wild Birds	2021	Wild Songbird	P / F	Salmonella spp.	29	14	00	[22]
	2001	Owl	NI	S. Typhimurium	40	04	00	[43]

NI: not informed.
*4-day-old neonate developed Salmonella meningitis.
**3-month-old child with microcephaly.
***3-week-old baby developed Salmonella meningitis and died.
****Used to feed pet reptiles.

Table 2.
Salmonellosis outbreaks in humans associated with wild animals (2000–2021).

Cases of salmonellosis in humans caused by contact with wild animals kept away from their natural habitat have been reported (**Table 2**). These animals are often the carriers of not only *Salmonella* strains, but of other pathogens, for which there are not always effective control measures [44].

As shown in **Table 2**, among wild animals in captivity, reptiles cause most outbreaks of salmonellosis in humans [45, 46]. Salmonellosis in reptiles usually occurs asymptotically [47]. The animals shed the bacterium intermittently and the elimination of the pathogen may increase due to stress factors [48]. Moreover, it is difficult to diagnose even in the presence of clinical signs [47]. However, human infections arising from human-reptile interaction can lead to clinical conditions ranging from mild to severe enteric infections, hospitalizations, and even deaths, especially in children, the elderly, and people with comorbidities [45].

Human contamination by *Salmonella* spp. from reptiles can be direct or indirect through secretions and excretions [49]. In a study conducted in southwest England between 2010 and 2013, 27.4% (48/175) of children under the age of five who had some contact with reptiles tested positive for *Salmonella* spp. and hospital admission

rates totaled 50% for children under 1 year of age [50]. In another study conducted between 2008 and 2009 in New Zealand with 378 cloacal swabs of 24 different exotic reptile species kept as pets, 11.4% tested positive for *Salmonella enterica* subsp. *Enterica*, with emphasis on the serovars Onderstepoort (30.2%), Thompson (20.9%), Potsdam (14%), Wangata (14%), Infantis (11.6%), and Eastbourne (2.3%), which can also cause infectious conditions in humans [51].

The participation of free-living wild reptiles in the epidemiology of *Salmonella* should also be stressed. In a park in Poland, 16 free-living road-killed snakes were analyzed and 87.5% were positive for *Salmonella* spp. [52]. Briones et al. [53] analyzed free-living wild reptiles in preserved areas in Spain and found that 41.4% tested positive for *Salmonella enterica*, with 27 serotypes identified, 37.5% of which were associated with salmonellosis in humans. Regarding the group of affected animals, snakes and lizards are more prevalent than chelonians [51, 54].

A high prevalence of *Salmonella* spp. with serotype diversity is also found in amphibians. In a study conducted in Indiana County, Pennsylvania (USA), Chambers and Hulse [55] collected 92 free-living amphibians and found that 39.1% tested positive (23 salamanders and 13 frogs), with isolated serotypes Muenchen, Enteritidis, Typhimurium, Senftenberg, and Montevideo. The prevalence of *Salmonella* in amphibians was also examined in 58 *Bufo marinus* of the West Indies and 41% tested positive to five serotypes, especially *Salmonella enterica* subsp. *Enterica* serovar Javiana (33%) and *S. Rubislaw* (33%) [56]. In Thailand, eight serotypes of *Salmonella* spp. were identified (Hvitittingfoss, Newport, Thompson, Stanley, Wandsworth, Panama, Muenchen, and subsp. *diarizonae* ser. 50:k:z) in 69.07% of the amphibians sampled in three different habitats - rural areas, protected areas, and urban areas. Of these serotypes, the first six have already been isolated in people in Thailand. Surprisingly, the animals coming from urban areas were negative [57]. The prevalence of *Salmonella* in amphibians regarding habitat remains unclear, although a possible cause is an environmental contamination by sewage [58]. This scenario is a public health concern because these amphibians can spread *Salmonella* spp. from the aquatic environment.

Outbreaks of salmonellosis in humans associated with contact with wild birds have been reported [59–61]. In 2000, an outbreak was reported in New Zealand caused by *S. Typhimurium* DT160, which led to the death of wild birds in rural areas, mainly sparrows, and enteric infections in humans [62]. In 2001, New Zealand reported an outbreak of human salmonellosis by *S. Typhimurium* DT160 related to contact with dead wild birds [63]. In 2001, two outbreaks were reported in the United States with at least 40 people contaminated with *S. Typhimurium* from the dissection of owls in two primary schools [43].

Between 1995 and 2003, Pennycott et al. [64] sampled 779 free-living wild birds in Great Britain and identified that the most prevalent serotype was *S. Typhimurium*. In Norway, *S. Typhimurium* variant O: 4,12 was identified in 96% of the isolates in a sample of 470 wild birds of 26 different species [44]. Despite the acute and chronic infection caused by *Salmonella*, in wild birds, it is asymptomatic [65]. During migration, the immune system can be affected by stress, as in the case of hunger, which may lead to a greater release of the pathogen by feces, contributing to even greater environmental contamination.

Regarding wild mammals, some species such as African pygmy, ferrets, hedgehogs, prairie dogs, primates, and sugar gliders are raised as pets [66], which can cause salmonellosis infections and outbreaks from direct human contact with carrier animals or indirectly due to access to or living in the same contaminated environments

as these animals [49]. Two human outbreaks in Norway, caused by *S. Typhimurium* 4.5, 12:i: 1.2 associated with hedgehogs, were reported from August to October 1996 and from July to November 2000, with 28 confirmed cases and 37 confirmed cases, respectively. In both cases, hedgehogs were the only common source, with positivity rates of 39% and 41%, respectively for the outbreaks of 1996 and 2000 [42].

Free-living wild mammals can also be asymptomatic carriers of *Salmonella* spp.; however, the prevalence is usually lower than when these animals are bred in captivity. From 2002 to 2010, 2713 animals were sampled in Italy, a total of 1612 mammals (1222 canids, 221 mustelids, 100 rodents, 69 ungulates), resulting in 7.25% animals positive for *Salmonella* spp. (63 canids, 25 mustelids, 5 ungulates, 24 birds), with emphasis on the *Typhimurium* serotype [67].

Notably, urbanization causes the spread of zoonotic agents due to new ecological interactions [68], from changes in eating habits to changes in migration routes [69]. When wild animals have access to urban spaces or modified environmental areas, they also come into contact with waste produced by humans, such as garbage and sewage. Moreover, these spaces are a food source for these animals [70]. Due to ineffective waste management, contaminated environments can be the source of numerous pathogens and favor the spread of antimicrobial resistance genes [71].

4. Antimicrobial resistance in wild environments

Antibiotic resistance is a health threat for humans, animals, and the environment [72]. Regarding microorganisms, this resistance initially occurred in the absence of anthropogenic factors and without the clinical application of antibiotics [73]. Thus, this resistance can develop naturally from the ecological evolution of microorganisms, such as gene mutation, due to environmental pressure [74]. However, human factors have contributed to greater antimicrobial resistance with a direct impact on ecosystems [75].

The anthropization of forest areas favors the contact of wildlife with domestic animals and humans [76]. In this regard, resistance can be acquired through the consumption of water or food and can also occur through direct contact with human waste and sewage [77]. Another factor that favors the spread of resistant microorganisms is the displacement capacity of the carrier [78]. However, although wildlife has not had direct access to antibiotics, natural habitats altered by demographic expansion can enhance the sharing of resistance across different ecological niches [79]. According to Jechalke et al. [80], free-living wild animals that have not been exposed to antibiotics exhibit high drug resistance rates due to environmental contamination. Gilliver et al. [81] identified a marked prevalence of antibiotic-resistant wild rodents that were not exposed to antimicrobials.

Residues from antibiotics applied in human and veterinary medicine enable the spread of resistant agents to wild species through environmental contamination, especially among those that share the same habitat [82, 83]. Therefore, antimicrobial resistance can be greater in forest areas close to rural properties due to the inappropriate use of antibiotics to prevent and control diseases or due to their use as animal performance enhancers [84]. These conditions increase contamination of the environment, water resources, the food chain, and, finally, human and animal health. Sub-doses of antibiotics may select multiresistant plasmids [85]. It should be noted that resistance plasmids are highly associated with cases of resistance to beta-lactam antibiotics in gram-negative bacteria from extended-spectrum β -lactamases (ESBLs) [86].

Antimicrobial residues that accumulate in sediments can determine changes in the microbiome of soils in aquatic and terrestrial environments [87]. These effects are intensified by erosion, surface runoff, and displacement of soil minerals [88]. When these elements reach the springs or are used for irrigation, or when the sediment is used as decomposed organic matter for agriculture, cyclic, rotational maintenance of this contamination occurs in the environment [89].

5. Conclusion

The alteration of forest areas through anthropic actions favors increases the spread of infectious agents since it enables a pathogen to leave its ecosystem and natural hosts and adapt to other environments and reservoirs. These new interactions create different environmental, epidemiological, and sanitary patterns, especially in emerging and neglected zoonoses, and hinder control and eradication, as in the case of salmonellosis. Wild animals raised as pets or illegally kept in captivity also increase the prevalence of salmonellosis cases in humans mainly caused by exotic serotypes of *Salmonella*, due to direct contact with the bacterial strains in these animals.

Since *Salmonella* spp. can also be transmitted by wild animals, prevention and control measures should include sanitary-environmental factors and an international health inspection system for emerging and re-emerging diseases originating from wild fauna. These measures would enable a better understanding of the epidemiology and pathogenesis of infections and reduce economic and health costs with diagnosis and medications.

Acknowledgements

The authors thank Universidade Estadual de Santa Cruz for the financial support.

Conflict of interest

The authors declare no conflict of interest.

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