

# Animal Reservoirs of Shiga Toxin-Producing *Escherichia coli*

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**ABSTRACT** Shiga toxin-producing *Escherichia coli* (STEC) strains have been detected in a wide diversity of mammals, birds, fish, and several insects. Carriage by most animals is asymptomatic, thus allowing for dissemination of the bacterium in the environment without detection. Replication of the organism may occur in the gastrointestinal tract of some animals, notably ruminants. Carriage may also be passive or transient, without significant amplification of bacterial numbers while in the animal host. Animals may be classified as reservoir species, spillover hosts, or dead-end hosts. This classification is based on the animal's ability to (i) transmit STEC to other animal species and (ii) maintain STEC infection in the absence of continuous exposure. Animal reservoirs are able to maintain STEC infections in the absence of continuous STEC exposure and transmit infection to other species. Spillover hosts, although capable of transmitting STEC to other animals, are unable to maintain infection in the absence of repeated exposure. The large diversity of reservoir and spillover host species and the survival of the organism in environmental niches result in complex pathways of transmission that are difficult to interrupt.

*Escherichia coli* strains that carry Shiga toxin genes are commonly isolated from the gastrointestinal tract of a wide variety of animal species (Table 1). Intestinal carriage of most Shiga toxin-producing *E. coli* (STEC) strains by domestic and wild animals has little clinical relevance to either the animal hosts or humans. Most animals lack receptors for Shiga toxin, and in humans, the presence of additional virulence factors, in addition to the *stx* gene, is associated with disease outcomes (1–3). However, animals may harbor STEC strains that are pathogenic to humans. This article focuses on the role of animals as reservoirs for infection or as spillover hosts. Within the animal, these bacteria may be resident or transient in the gastrointestinal tract. Determining

whether STEC is resident in flora or transient is not possible during cross-sectional observational epidemiological studies when only one sample is collected from an animal and there is no serial testing. Even under experimental conditions it is difficult to determine if repeated isolation from the feces over time is a result of replication of the organism in the animal or repeated exposure.

Animals capable of maintaining STEC carriage in the absence of continuous exposure or those that frequently are reexposed to STEC from environmental sources can serve as potential sources of interspecies and intraspecies infection. Cattle are regarded as the natural reservoir of STEC (1), but other ruminant species such as sheep, goats, and deer may also act as reservoirs. Animals may also be categorized as spillover hosts. Similar to reservoirs, these animals are susceptible to colonization and may transmit disease; however, once they are no longer exposed to a source of STEC, they do not maintain this colonization. This inability to maintain STEC colonization in the absence of exposure is the critical factor that differentiates these animals from reservoirs. Epidemiological evidence indicates that birds, swine, dogs, and horses may be spillover hosts. Dead-end hosts, as the name suggests, are incapable of transmitting STEC

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**TABLE 1** Animal hosts of Shiga toxin-producing *E. coli*

Common Name	Scientific Name	Reference
Cattle	<i>Bos taurus</i>	<a href="#">1</a> , <a href="#">7</a> , <a href="#">8</a> , <a href="#">10</a> , <a href="#">19</a> , <a href="#">21–23</a> , <a href="#">27</a> , <a href="#">29–33</a>
Goats	<i>Capra aegagrus hircus</i>	<a href="#">34</a> , <a href="#">39</a> , <a href="#">40</a> , <a href="#">43</a> , <a href="#">44</a> , <a href="#">48</a> , <a href="#">49</a> , <a href="#">53</a>
Sheep	<i>Ovis aries</i>	<a href="#">1</a> , <a href="#">35</a> , <a href="#">39</a> , <a href="#">43–47</a>
Water buffalo	<i>Bubalus bubalis</i>	<a href="#">53</a> , <a href="#">54</a> , <a href="#">61</a>
White-tailed deer	<i>Odocoileus virginianus</i>	<a href="#">62–64</a> , <a href="#">67–71</a>
Bison	<i>Bison bison</i>	<a href="#">74–77</a>
Elk	<i>Cervus canadensis</i>	<a href="#">72</a> , <a href="#">73</a> , <a href="#">80</a>
Llamas	<i>Lama glama</i>	<a href="#">191</a>
Alpaca	<i>Lama pacos</i>	<a href="#">83</a> , <a href="#">192</a>
Yak	<i>Bos grunniens</i>	<a href="#">83</a>
Eland	<i>Taurotragus oryx</i>	<a href="#">83</a>
Antelope	<i>Antilope cervicapra</i>	<a href="#">83</a>
Mountain goat	<i>Oreamnos americanus</i>	<a href="#">84</a>
Guanaco	<i>Lama guanicoe</i>	<a href="#">79</a>
Horses	<i>Equus ferus caballus</i>	<a href="#">85–88</a> , <a href="#">91</a>
Donkey	<i>Equus africanus asinus</i>	<a href="#">84</a> , <a href="#">89</a> , <a href="#">90</a>
Domestic swine	<i>Sus domesticus</i>	<a href="#">1</a> , <a href="#">92</a> , <a href="#">94–96</a> , <a href="#">101</a> , <a href="#">102</a>
Feral swine	<i>Sus scrofa</i>	<a href="#">103–105</a>
Chicken	<i>Gallus gallus domesticus</i>	<a href="#">92</a> , <a href="#">94</a> , <a href="#">125</a> , <a href="#">126</a>
Turkeys	<i>Meleagris gallopavo</i>	<a href="#">92</a> , <a href="#">126</a>
Pigeon	<i>Columba livia</i>	<a href="#">111</a> , <a href="#">116</a>
Starling	<i>Sturnus vulgaris</i>	<a href="#">110</a> , <a href="#">112–114</a>
Geese	<i>Branta canadensis</i>	<a href="#">107</a> , <a href="#">119</a>
Turtle dove	<i>Streptopelia turtur</i>	<a href="#">112</a>
Barn swallow	<i>Hirundo rustica</i>	<a href="#">112</a>
Dogs	<i>Canis lupus familiaris</i>	<a href="#">39</a> , <a href="#">163</a> , <a href="#">165</a>
Cats	<i>Felis catus</i>	<a href="#">166</a> , <a href="#">170</a> , <a href="#">171</a>
Coyote	<i>Canis latrans</i>	<a href="#">84</a>
Fox	<i>Vulpes vulpes</i>	<a href="#">84</a>
Rabbit	<i>Oryctolagus cuniculus</i>	<a href="#">143</a> , <a href="#">144</a>
Raccoon	<i>Procyon lotor</i>	<a href="#">152</a>
Fish and shellfish		<a href="#">129–132</a>
Norway rats	<i>Rattus norvegicus</i>	<a href="#">108</a> , <a href="#">137</a> , <a href="#">138</a>
Ground hog	<i>Marmota monax</i>	<a href="#">84</a>
Patagonian cavy	<i>Dolichotis patagonus</i>	<a href="#">83</a>
Frogs		<a href="#">193</a>
Ferrets <sup>a</sup>	<i>Mustela putorius furo</i>	<a href="#">172</a>
Mice <sup>a</sup>	<i>Mus spp.</i>	<a href="#">114</a> , <a href="#">142</a> , <a href="#">180</a>

<sup>a</sup>Experimental infections only.

naturally to other animals. In the absence of evidence that aquatic species such as finfish and shellfish transmit the organism to other animals, they may act as dead-end hosts for STEC, only transmitting STEC when they are consumed ([4–6](#)).

The factors governing the prevalence and number of bacteria present in the digestive tract of animals are poorly understood, even for the best-studied species, bovines. The prevalence and magnitude of STEC infection

in animals is dependent on a complex interaction of external and internal conditions: the frequency and dose of exposure, the host's susceptibility to infection, and the duration of shedding. Moreover, these factors may vary considerably among species and even among the same species as a function of age, immunity, housing, diet, climate, and sanitation.

## ANIMAL SPECIES OF IMPORTANCE IN THE EPIDEMIOLOGY OF STEC

### Cattle

Cattle are recognized as a primary reservoir for STEC strains, especially the serogroup O157 ([1](#)). Like humans, cattle are exposed to STEC through contaminated food and water or by exposure to the feces of other animals shedding the organism. The infectious dose in cattle is estimated to be as low as 300 CFU ([7](#)). STEC colonization in cattle is usually asymptomatic due to the absence of vascular receptors for Shiga toxins ([8](#)). The absence of these globotriaosylceramide-3 (Gb3) vascular receptors, especially in the intestinal vasculature, means Shiga toxins cannot be endocytosed and transported to other organs that may be sensitive to Shiga toxins ([9](#)). The terminal part of the large intestine, the recto-anal junction (RAJ), is the main site of STEC colonization in cattle ([10](#)). The increased production of factors associated with environmental survival among cattle isolates of STEC O157, compared to human-origin isolates, combined with the low infectious dose, may provide a selection bias for these organisms to recolonize cattle and maintain the organism in the bovine population ([11](#)). Improper feed storage facilities or poorly designed feeding troughs can result in feed being contaminated with the feces of wild or domestic animals.

Livestock drinking water contamination can occur at its source or at the farm. Surface water and groundwater sources may be contaminated from effluent runoff from farms and urban areas. Leaching from pastures may also result in groundwater contamination ([12](#), [13](#)). At the farm, improperly designed water troughs can be contaminated by animal feces. LeJeune et al. ([14](#)) showed that 1.3% of 473 water troughs sampled in three U.S. states were positive for STEC O157. STEC has also been demonstrated to persist for more than 4 months in contaminated water troughs ([15](#)).

Other management practices may also affect the incidence of STEC in animal populations. Flushing alleyways with water increased the incidence of STEC in animals compared to other manure removal strategies

(16). Animals housed on sawdust were also found to have a higher incidence of STEC than animals housed on sand-based bedding (17). Movement of animals to and from farms also increases the risk of STEC transmission: Animals carried to animal exhibitions have a greater likelihood of contracting STEC than animals not carried to shows (18). These animals, on returning to the farm, can then shed STEC, thus exposing other animals to infection or colonization.

In the United States, STEC O157 is found on almost all cattle farms, with the organism being shed intermittently by most animals (19). STEC is shed mainly through the feces of colonized animals; however, Shiga toxin genes have been detected from *E. coli* strains isolated from the milk of mastitic cows (20). Although a rare occurrence, milk from these animals can be a potential source of STEC infection to nursing calves, animals fed waste milk, and the human population. Most milk-borne STEC cases are, however, due to postmilking contamination and the subsequent consumption of these products without pasteurization.

The prevalence of STEC in cattle populations is highly variable, with peaking and dropping at unpredictable times. At any specific time, the global prevalence of STEC O157 in cattle has been reported to range from 0 to 71% (21), and the herd infection rate has been reported to be up to 100% in some studies (22). In the United States, the herd prevalence of STEC may range between 10 and 20% (23). The global prevalence of STEC O157 has been reported to range from 0.2 to 48.8% in dairy cattle and 0.2 to 27.8% in beef animals whereas the global prevalence of non-O157 STEC may range from 0.4 to 74% in dairy cattle and 2.1 to 70.1% for beef animals, respectively, as reported in two independent studies (24, 25).

Colonized cattle can shed STEC O157 at levels as high as  $1.1 \times 10^5$  CFU/g feces (26) and for as long as 10 weeks (27). The average duration of STEC O157 carriage is 30 days; however, in rare cases animals may be colonized for up to 1 year (28). Animals excreting greater than  $10^4$  CFU/g feces are termed “super-shedding animals” (29, 30). Longitudinal studies, however, indicate super-shedding represents a phase or stage of colonization of all cattle and can be typically observed in a small fraction of animals in a population at any given time (31). Nevertheless, it is not debatable that animals excreting these high levels of STEC are responsible for the majority of environmental contamination (30, 32). Calves tend to shed STEC at the lowest levels before weaning; however, the highest shedding is exhibited in the period immediately post weaning (33). The shedding

of STEC also tends to be higher in the warmer months, with peak prevalence being in summer and early fall with a drastic decrease in prevalence during the winter months (19).

### Small Ruminants

Small ruminants, particularly sheep and goats, are important reservoirs of STEC O157 (34). Considerable research has focused on the role of sheep in the epidemiology of STEC infections; however, there is limited published research on the role of goats (34). Although cattle have been identified as the major reservoir for STEC in the United States, small ruminants play a greater role in the epidemiology of STEC infections in other countries. For example, sheep have been identified as the host of significance in Australia (1) and have also been recognized as an important reservoir of STEC O26 in Norway (35). In addition to STEC serogroups O157 and O26, sheep have been cited as reservoirs for more than 100 other serotypes of STEC, including O115, O128, and O130 (34, 35).

Transmission of STEC to small ruminants occurs through the same pathway as in cattle. The site of STEC colonization, however, may be different. Unlike cattle, tropism for RAJ has not been described for all small ruminants (34). Following exposure to STEC O157, in some studies few attachment and effacement lesions were visible on the intestinal mucosa and the entire distal intestine, including the cecum, colon, and rectum, was colonized, not only the RAJ (34, 36). However, in mature sheep given a single oral dose of a human clinical isolate of STEC O157:H7, analysis of digesta and intestinal mucosa showed colonization occurs exclusively at the RAJ mucosa (37; CJ Hovde, University of Idaho, personal communication). STEC O157 shedding patterns between rectally and orally inoculated sheep were found to be similar, thus indicating that STEC O157 may be able to effectively colonize the terminal rectum (38). However, inefficient attachment of large numbers of STEC to the RAJ may account for reduced shedding periods compared to those in cattle.

Similar to cattle, small ruminants tend to be asymptomatic shedders of STEC. This trait was demonstrated when the screening of “healthy animals” in Berlin reported that 66.6% of the sheep and 56.1% of the goats tested were found to be STEC carriers (39). Similar results were obtained in Spain where 47% of healthy goats tested positive for shedding STEC (40). The asymptomatic feature of STEC carriage is possibly also due to the lack of Shiga toxin vascular receptors in small ruminants.

Many direct-contact human infections are attributed to contact with sheep and goats at petting zoos and open farms (41, 42). One study investigating the prevalence of zoonotic agents on small city farms in southern Germany found that 100% of the sheep and 89.3% of the goats tested positive for STEC (43). Small ruminants, especially goats, generally exhibit inquisitive behavior and thus may have greater contact with humans, increasing the potential for transmission to humans (34). Human infections have also been linked to the consumption of unpasteurized milk and cheese made from contaminated goat or sheep milk (40, 44).

Sheep are the primary reservoir for STEC in Australia, with the serotype of importance being O26; however, the risk of human infection was deemed insignificant due to low prevalence rates (1, 45). Although the within-herd prevalence was low, previous research reported that 90% of Australian sheep farms had animals testing positive for STEC (46). In Norway, however, the risk of human infection from sheep was much more significant since almost 50% of the sheep O26 isolates had multiple-locus variable number tandem repeat analysis profiles similar to that found in human clinical cases (35). The importance of sheep in STEC epidemiology was also demonstrated by Oporto et al. (47), who reported that greater than 50% of sheep herds in Spain had animals shedding non-O157 STEC compared to 20.7% in dairy cattle and 46% in beef cattle.

In the United States, Jacob et al. reported that 11.1% of goat fecal samples collected at slaughter had STEC O157 and 14.5% had at least one non-O157 STEC serotype (48, 49). The STEC O157 flock prevalence in Spain was reported to be 8.7% and individual prevalence, 7.8% (47). A similarly low STEC O157 prevalence of 5.8% was also reported in Scotland (50). Low STEC O157 prevalences were also reported in the United Kingdom and Holland, with the prevalence being 0.1% and 4.0%, respectively (51, 52). Lesser developed countries have also reported the presence of STEC in their small ruminant population. In Vietnam, 100% of the goat farms surveyed had animals shedding STEC, and the within-herd shedding was dramatically higher than that reported elsewhere, with up to 65% of animals shedding STEC (53). In Bangladesh, almost 10% of the small ruminants being slaughtered tested positive for STEC O157 (54). As evidenced by past outbreaks, STEC in animals from lesser developed countries can potentially be a serious threat to food safety, since in those countries there may not be strict hygienic slaughter practices; thus contaminated meat could easily enter the food chain (55, 56).

The shedding of STEC in small ruminants has been demonstrated to be age and season dependent. Younger animals tend to have a lower prevalence of STEC than older animals do (40, 57–59). A longitudinal study spanning 6 months in the United States demonstrated a peak in STEC prevalence during summer (60). This trend was also observed in Italy, where animals screened during the warmer months of the year had a higher prevalence of STEC O157 (58).

## Other Ruminants

### Water buffalo (*Bubalus bubalis*)

In addition to cattle, sheep, and goats, other ruminant species have also been identified as shedders of STEC. Water buffalo (*Bubalus bubalis*) has been identified as an important reservoir of STEC O157 in many countries (61). The water buffalo is reared in many countries because of its ability to serve a dual purpose, as both a milk and meat producer. Buffaloes are also able to thrive much better on poor quality forages than the *Bos taurus* species, thus making them suitable for subsistence farming. There are large commercial meat and milk water buffalo herds in Asia and South America, while in Europe water buffalo is primarily reared for milk production. In Bangladesh, STEC colonies were isolated from 38% of the buffaloes sampled before slaughter. Almost half of these isolates were identified as being O157 (54). Galiero et al. reported an almost a similar prevalence in Italy, with 14.5% of the animals shedding O157 (61). In Vietnam, 27% of the buffaloes screened were found to be positive for STEC. Serotyping of the isolates, however, revealed that none of the isolates were O157 (53).

### Deer

There are an estimated 30 million white-tailed deer in the United States (194). The role of white-tailed deer (*Odocoileus virginianus*) as a potential reservoir for STEC was first reported in 1999 when almost 2.4% of deer sampled tested positive (62). The presence of STEC O157:H7 in deer feces was later confirmed by Renter et al. (68), who found the STEC prevalence in Nebraska white-tailed deer to be 0.25%. Similarly, low STEC prevalences of 0.2% were reported in hunter-harvested captive deer in Louisiana (63) and 3.3% in farm-raised deer in Ohio (64). Other species of deer, including red deer (*Cervus elaphus*), fallow deer (*Dama dama*), and roe deer (*Capreolus capreolus*), have also been identified as capable of shedding STEC serotypes (65, 66). Almost 50% of Pennsylvanian white-tailed deer fecal samples screened tested positive for *stx* genes; however, only 8%



possessed the *eae* gene, which is necessary for colonization of the human intestine (67).

Feral deer are known to share pastures with cattle and can also be found in close proximity to many dairy farms. The close association between deer and livestock implies that deer can serve to maintain and disseminate STEC between and within cattle herds (68, 69).

Human STEC O157 infections were first associated with venison in 1997 when six persons became ill as a result of consuming jerky made from venison (70). Since then, there have been numerous other cases associated with venison, with one of the most recent published reports being an outbreak of non-O157 STEC among high school students that was associated with consumption of venison they had killed and processed. Two STEC serotypes, O103:H2 and O145:NM, were isolated from the samples analyzed; however, the O145:NM serotype was found to be Shiga toxin negative (71).

### Elk (*Cervus canadensis*)

Similar to deer, elk have also been associated with numerous food-borne disease outbreaks (72). Gilbreath et al. (80) reported that over 22% of wild Idaho elk screened were positive for STEC. A slightly lower prevalence (7%) of *stx* genes was detected in fecal pellets collected from elk in Colorado (73). In this study none of the animals were found to shed serogroup O157, but serogroups O103 and O146 were detected. Interestingly in both studies, the incidence of STEC in the elk feces was found to be higher than in mule deer, which shared the same grazing grounds.

### Bison (*Bison bison*)

Another potentially important animal reservoir of STEC is the American bison (*Bison bison*). This potential is supported by the fact that both cattle and bison share similar RAJ morphological characteristics (74). In the United States consumption of bison meat has increased, thus increasing the risk of transmission from bison to humans (75). This risk was exemplified in 2010 when a multistate outbreak of STEC O157:H7 was associated with bison meat consumption. The prevalence of STEC O157:H7 in bison has been reported to be as high as 42% (76). STEC O157 has also been isolated from the carcass of slaughtered bison at a prevalence of 1.13% (77). Non-O157 STEC serotypes including O45, O103, O111, O113, O121, and O145 have also been isolated from bison carcasses; however, none of these isolates possessed *stx* genes (78).

STEC O157 and non-O157 STEC strains have been isolated from other captive and wild nondomesticated

ruminant species. They include llamas, moose, alpacas, antelopes, and yaks (79–84). These animals can transmit STEC to humans directly by contact at petting zoos or indirectly through fecal deposition in water sources, vegetable fields, or recreational areas or on meat. Further research is required to determine the role these animals may have in the epidemiology of human STEC infections.

### Equine

Published data on the epidemiology of STEC carriage in horses are limited. There are also no published case reports describing the clinical features of STEC infection in horses. The available published data on the prevalence of STEC in horses (85–88) and donkeys (84, 89, 90) indicate that they are not major reservoirs of STEC and may instead be spillover hosts. Only one of 400 horse fecal samples screened in Germany was positive for STEC. The serotype isolated was O113:H21 (88). A similarly low prevalence of STEC has also been detected in the equine population in the United States. Only one of 242 horse fecal samples from Ohio tested positive for STEC O157:H7. Interestingly, this case shared housing accommodations with a goat that also shed STEC O157. The isolates from both animals had indistinguishable multiple-locus variable number tandem repeat analysis patterns (87). A similarly low prevalence has been reported by Hancock et al., who reported that 1% of horses sampled (n = 90) tested positive for O157:H7 (85). Screening of fecal samples from horses located in the Sacramento Valley revealed a slightly higher prevalence than that recorded in Ohio. Four of 156 samples (2.6%) tested positive for the Shiga toxin 2 gene (86). Notably, as was seen in Ohio, all the positive horses were also housed on farms containing ruminants. Despite the low STEC prevalence in horses, there are reported human clinical cases associated with infection from horse contact (91), and one must be aware of this potential source of infection.

### Swine

Swine can be colonized with various serotypes of STEC, including O157; however, the risk of causing human disease is low (12, 92, 93). The prevalence of STEC O157:H7 in domestic swine has been reported to range from 0 up to 10%, with the prevalence in the United States usually being less than 2% (12, 92, 94, 95). As in humans, STEC O157:H7 can be highly pathogenic in pigs (92). Unlike ruminants, pigs possess *stx*-sensitive vascular receptors, and edema disease can develop post intestinal colonization with STEC strains producing

Stx2e (8, 96). Stx2e is the most frequent subtype of Stx2 found in porcine feces (97). The receptor affinity of Stx2e is different from Stx1a and Stx2a, since the primary receptor targets are not Gb3 receptors but rather globotetraosylceramide (Gb4) receptors (98, 99). Recently weaned pigs are most susceptible to edema disease, and the clinical signs include subcutaneous and submucosal edema, ataxia, incoordination, stupor, and recumbency (98, 100). While morbidity in the herd may be low, the case fatality rate for edema disease is high, and surviving pigs may have neurological deficits.

Though a relatively low prevalence of STEC O157:H7 has been reported, swine have been shown to harbor and shed STEC for up to 2 months post infection (101). Non-O157 STEC serotypes have also been isolated from pigs; however, many of these isolates lack the virulence factors required to cause human disease (1). Despite a low prevalence of pathogenic STEC serotypes, the potential for human infection from swine exists. This risk is exemplified by a recent Canadian outbreak of STEC O157:H7 associated with consumption of pork, with infected persons having the identical STEC O157:H7 isolate to that found in the pork meal served (102)

### Feral Swine

Feral swine is another wildlife species that has been associated with STEC disease in the human population. There are approximately 5 million feral swine in the United States, and they can be found in more than 35 states (195). These animals are highly adaptable to varying environmental conditions and can serve as a vector for disease between livestock farms and as a source of contamination of vegetable production fields.

In the United States, feral swine was first identified as a reservoir for STEC O157:H7 in 2007 in California (103). In that study, STEC O157:H7 was isolated from 14.9% of the swine specimens tested, and these isolates were found to be indistinguishable from STEC O157:H7 isolates obtained from an outbreak in the human population associated with the consumption of spinach. Interestingly, all cattle, feral swine, and environmental samples from the region where the spinach was cultivated had the same STEC isolate O157 (103). STEC was also detected in feral swine from Sweden, Switzerland, and Spain. Approximately 9% of the tonsil samples screened (n = 153) in Switzerland were positive for STEC O157, but none of the corresponding fecal samples were positive (104). A similar prevalence of 8% was reported for Spanish feral swine fecal samples (105). The isolates were serotyped, and 3.3% of the animals were identified

as shedding STEC O157:H7 and 5.2% of the animals as shedding non-O157 STEC.

The identification of STEC from feral swine samples indicates that they can play a role in the epidemiology of STEC infections. As such, their ability to potentially contaminate vegetable production fields and serve as vectors for STEC transmission between livestock must be recognized, and measures employed to mitigate this risk

### Birds

Birds are capable of harboring many bacterial organisms in their gastrointestinal tract and are capable of acting as spillover hosts for STEC. Wild birds were first identified as a potential source of STEC infection in 1997 (106). Since then, STEC has been isolated from starlings, pigeons, sparrows, and other avian species (106, 107). Many species of wild birds can be found in close proximity to livestock operations and waste disposal landfill sites. These birds are attracted to farms since they can easily obtain a food source from animal feed. Nielsen et al. identified that 2% of the wild bird fecal samples collected in close proximity to farms contained *stx* genes (108). Similar results were also obtained in England where 1.5% of wild bird samples had the *stx1* gene, 7.9% the *stx2* gene, and 4.9% the *eae* gene (109). Similarly, low prevalence rates of STEC O157:H7 in the starling (*Sturnus vulgaris*) population in Ohio and other wild bird species in Scotland and Japan have also been reported (110–112). Though the STEC prevalence levels are reportedly low, the potential of these birds to transmit STEC to other birds and contaminate the environment is of serious risk. Studies have shown that once colonized, a starling may shed STEC O157 at levels greater than 100 CFU/g of feces for up to 13 days post colonization (113).

The migratory pattern of birds and the fact they can traverse long distances in a single day mean they can serve as a mode of transmission of STEC between and within farms. This was demonstrated by Williams et al. (114), who reported that starlings and cattle on different farms had molecularly indistinguishable subtypes of STEC O157:H7, thus confirming that starlings were able to transmit STEC to different farms. Bolstering the role of starlings in STEC epidemiology is the fact that the number of starlings per milking dairy cow was also found to be significantly associated with the presence of STEC O157:H7 in bovine fecal pats (115).

Migratory birds can also interact with peridomestic birds such as pigeons and thus propagate the transmission of STEC. Pigeons and finches have been identified as

two species that can potentially serve as a source of human infection since these birds inhabit buildings, parks, and other recreational areas and are in close association with the human population (111, 116). Fecal depositions by these birds in areas frequented by humans increase the exposure potential to STEC.

Water fowl, including geese and ducks, are identified as a source of surface water and pasture contamination (107, 117, 118) and implicated as the source of numerous food-borne pathogens (119–122). One goose is reportedly capable of producing up to 5 pounds of feces per day, and this can result in mass contamination, since these birds are usually found in flocks (123). These birds are also able to travel large distances per day and can disperse pathogens over a wide area. Geese are known to forage within vegetable fields and also inhabit ponds and other surface water sources used for irrigation (124). The birds can thus contaminate produce when they defecate within vegetable fields and irrigation water sources.

STEC carriage has also been reported in domestic poultry. The prevalence of STEC O157:H7 in domestic chicken is relatively low, ranging from 0 to 1.5%, depending on the geographic location sampled (92, 94, 125, 126). Interestingly, the prevalence in turkeys was higher than that in chickens, with up to 7.5% of fecal samples testing positive (92, 126). Experimentally colonized chickens have been reported to harbor and shed STEC O157:H7 in their feces for periods in excess of 11 months (127). Pet birds such as canaries (*Serinus canaria domestica*) have also been reported to be capable of harboring and shedding STEC (128).

That both wild and domestic birds are able to harbor, transmit, and shed STEC is of serious concern since they potentially are a major risk to human health and disease, and as such, precautions should be taken to limit human or animal exposure to the excrement from these birds.

### Fish and Shellfish

Fish and shellfish can be exposed to STEC when their aquatic environment becomes contaminated with mammalian fecal matter. These species do not act as reservoirs of infection or spillover hosts but rather dead-end hosts. Fish residing in close proximity or downstream of animal livestock facilities have also been found to be contaminated with STEC (129). Shellfish, due to their filter feeding ability, pose a significant risk to human infection since they can concentrate and retain pathogens (4, 130). Numerous studies have reported the recovery of both O157 and non-O157 STEC from the carcass of fish and shellfish offered for sale (131–135).

The detection of STEC in these carcasses highlights the potential for human STEC infection through the consumption of undercooked or raw fish and shellfish.

### Rodents

Rodents have also been identified as being capable of harboring STEC within their gastrointestinal flora (108, 136–138). STEC O157 and non-O157 STEC have been recovered from *Rattus* spp. living in urban areas and on farms (137, 138). Cizek et al. demonstrated that Norway rats (*Rattus norvegicus*) were capable of shedding STEC O157:H7 for up to 11 days post exposure to high doses of STEC ( $10^9$  CFU) and 5 days post exposure to lower doses of STEC ( $10^5$  CFU) (139). This shedding ability indicates that while rats may not be long-term reservoirs of STEC, they are certainly capable of transmitting STEC between and within farms. This transmission potential was highlighted by Nielsen et al. (108), who found that STEC recovered from Norwegian rat fecal pellets was identical to that shed by cattle on the same farm. Contaminated rat feces may also be capable of harboring STEC O157:H7 for up to 9 months post inoculation, thus increasing the risk of transmission to other animals (139). Although rodents, especially rats, are not regarded as having a major role in the epidemiology of STEC (69, 85, 140), their potential to harbor and transmit STEC exists. Unlike for rats, there is little published information describing the role of mice in the epidemiology of STEC. Mice have, however, been used as animal models to study STEC infection in humans (141, 142).

### Rabbits

Rabbits have been identified as potential vehicles for the transmission of both O157 and non-O157 STEC (143–145). Rabbits have also been used as a possible animal model to study STEC infection in humans, since they demonstrate enteric and renal lesions when challenged with STEC (146). Globally, consumption of rabbit meat is increasing; over 1 million tons of rabbit meat are consumed annually (147), thus increasing the potential for food-borne infection. Rabbits are also popular at petting zoos, and more than 6 million rabbits are kept as pets in United States (148). Similar to dogs and cats, their close association with humans may lead to the exchange of microbiota between species and thus STEC transmission. Wild rabbits are able to traverse long distances and may inhabit both urban and agricultural areas and potentially serve as vectors for the transmission of STEC from farm environments to the human population (144).

## Raccoons

Raccoons are of particular interest since they can reside in a wide range of habitats, including agricultural, forested, and urban areas. Raccoons have been identified as a reservoir for numerous pathogens, including *Salmonella*, *Leptospira*, and *Campylobacter* species (149–151); however, there is only one report of STEC being isolated from raccoon feces. This animal had been residing within the hay barn of a dairy farm (152) and thus may be a spillover host. Despite an extensive literature search, no other reports of STEC raccoon colonization could be found (153, 154).

## Insects

Insects can be important vectors in the transmission and dissemination of STEC in the environment. STEC O157:H7 has been recovered from houseflies (*Musca domestica*), dump flies (*Hydrotaea aenescens*), and dung beetles (*Catharsius molossus*) residing on animal farms and at animal fairs (155–158). Houseflies, in addition to being a mechanical vector, may also be involved in bioenhanced transmission (159). Kobayabashi et al. (159) suggested this additional role because STEC O15H:H7 could be detected within the alimentary tract of inoculated houseflies for at least 3 days post inoculation. The ability of houseflies to transmit STEC O157:H7 to animals was demonstrated by Ahmad et al. (160), who exposed naïve calves to houseflies inoculated with STEC; within 24 hours, STEC could be recovered from the feces of all eight calves in the experiment (160). Houseflies have also been demonstrated to transfer STEC onto the surface of vegetable produce (161). Houseflies are able to travel greater than 4 miles (162), and given their ability to transmit STEC, one has to be cognizant of the role they may play in the epidemiology of STEC infection.

## Pets

Pets, especially dogs and cats, are capable of shedding a diverse range of STEC serotypes in their feces (163–166). Interestingly, although both O157 and non-O157 STEC have been recovered from dogs, there are no published reports indicating that O157 STEC has ever been recovered from cat feces. Dogs and cats have historically had close interaction with humans, with exchange of microbiota resulting in the possible transmission of STEC between species. These animals can be asymptomatic shedders of STEC, as demonstrated by Beutin et al. (39), who reported that up to 12% of healthy dogs shed STEC in their feces. In addition to household dogs, farm dogs can be a vector for the

transmission of STEC. These dogs move freely among animals and humans, thus potentiating the spread of enterohemorrhagic *E. coli* (167). Dogs have also been reported to shed non-O157 STEC serotypes in their feces (163). Human infections due to canine exposure were also reported; one outbreak in Sweden resulted in 50 cases in humans after they attended a dog show (168). STEC has also been recovered from the feces of wild canids (169).

A highly virulent strain of STEC O146:NM has been isolated from the feces of an asymptomatic cat in Argentina (170). In Germany, there is also evidence of a cat and its owner shedding the same STEC O146:NM serotype (171). In this case, the source of the infection could not be determined, nor which animal was the index case.

## Animal Models

Animal models have been used to study the in vivo pathogenesis of STEC. Numerous animal models have been developed, including mice, rats, chickens, rabbits, cows, greyhounds, baboons, and macaques (141, 173–178). Although these models do not fully replicate all aspects of the STEC infection in humans, they provide valuable insight into intestinal colonization, STEC pathogenesis, immune response, and efficacy of possible treatment regimens (179, 180).

Compared to other animal models, the mouse models are preferred for in vivo STEC studies because of their small size, low cost, ease of care and maintenance, availability of numbers, and varying genetic backgrounds (<141). There are at least four mouse models, including streptomycin-treated, streptomycin and mitocycin C/ciprofloxacin-treated, intragastric-fed but not streptomycin-treated, and the malnourished mouse models (181). The two most popular models are, however, the streptomycin-treated mouse and axenic mice. The models have reduced or no gastrointestinal flora and are also susceptible to STEC or enterohemorrhagic *E. coli* colonization (141). The response of mouse models to STEC exposure is, however, dependent on the method of infection, the strain of STEC, and the type of mouse model used (181).

## Humans

Although animals are generally regarded as the main reservoir of STEC, humans can also be STEC reservoirs and may play a much larger role in the epidemiology of STEC infections than previously thought (182, 183). Asymptomatic infections in the human population can result in dissemination STEC and further propagation of



outbreaks (182). Given that food contamination is the source of almost 40% of STEC outbreaks and almost 30% are of unknown origin (184), it is possible that contamination by asymptomatic humans may be the source of many of these outbreaks.

Human STEC infections can present a wide spectrum of clinical signs, ranging from symptomatic infections to severe clinical syndromes such as hemorrhagic colitis and possibly hemolytic-uremic syndrome and thrombotic thrombocytopenic purpura in approximately 7% of the cases. Elderly persons, young children, and immunocompromised persons are at greatest risk (185).

Food and environmental contamination with STEC can occur as result of shedding by asymptomatic workers. Approximately 12% of dairy families in a Canadian study tested positive for O157 antibodies, and STEC isolates were recovered from 6% of the fecal samples, yet none of these positive cases could recall any clinical signs associated with STEC (186). Similarly, 1.1% of farm workers in Italy were found to be shedding STEC O157 asymptotically in their feces. Contamination of meat carcasses at the abattoir during slaughter is also a possibility. One study reported that 1.3% of abattoir workers sampled were actively shedding STEC in their feces (187). Asymptomatic children can also shed STEC serotypes for up to 30 days post detection, whereas adults in the recent German O104:H4 outbreak shed the organism for up to 13 weeks (182, 188).

Person-to-person or secondary transmission is important in propagation of outbreaks and can account for 15 to 20% of cases within outbreaks (184, 189). At particular risk of disease due to secondary transmission are children (1 to 6 years of age) due to close contact, their immature immune systems, reduced personal hygiene, and prolonged shedding time (184). An analysis of STEC outbreaks occurring between 1982 and 2006 as a result of person-to-person transmission showed that 45% of these outbreaks occurred due to transmission at home, 11% at nurseries, and 10% at recreational water sources (190).

## CONCLUSION

Most warm-blooded animals are capable of acting as reservoirs (symptomatic and asymptomatic), spillover hosts, or dead-end hosts of STEC. Animals are exposed to STEC by direct or indirect contact with the feces of a shedding animal. Cattle are recognized as the main reservoir of STEC, however, and other livestock species, including goats, sheep, bison, horses, pigs, and water buffalo, have been demonstrated to be capable of

harboring these organisms. Wild birds and animals pose a unique risk in their ability to travel long distances, increasing the dissemination of STEC in the environment and thus potentiating its spread. Domestic pets are also capable of harboring STEC, and thus serve as a source of contamination within the household. Given the demonstrated ability of STEC to colonize the gastrointestinal tract of a wide variety of animals, it is expected that numerous other unreported species may also be potential sources of contamination or transmission of STEC. Recognition of these potential novel animal sources of transmission and propagation of STEC is essential when conducting epidemiological investigations and developing proper risk mitigation strategies. Despite the widespread carriage of STEC by a variety of animal species, it is important to consider that the presence of the *stx* gene alone is not an indication of pathogenicity in the human host. Assessment of the complement of virulence factors present in STEC recovered from animal hosts is therefore important to develop risk models. The understanding of why certain pathoseroypes or strains of STEC have a predilection for different animal species may provide valuable insight in the design of interventions to control the organism in the live animals that have impacts on human health.

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