Chapter 1
Overview: Foodborne Pathogens in Wildlife Populations

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Abstract Numerous bacterial and parasitic pathogens may be transmitted through food and included in that group are zoonotic pathogens that not only proliferate within domesticated animals but may also be resident within wildlife. As a result of wildlife being a pathogen reservoir and the ability of this animal group to easily intrude on farms, wildlife contributes to the maintenance of infections on domestic farms as well as serves as an environmental source of fresh produce contamination. To discern the degree to which wildlife represents a food safety risk, this overview first summarizes those documented incidents in which contaminated wildlife has been directly or indirectly associated with human illness. It continues with providing a set of tables that document the results of studies directed at assessing the prevalence of bacterial, parasitic, and viral pathogen contamination in mammals, birds, and amphibians and reptiles. To understand the strengths and limitations of those surveillance studies, discussion is included that describes how sample source, cultivation conditions, sample size and number, and specificity of the detection method may impact the data collected. Discussion on factors that contribute to pathogen transmission to wildlife are also presented and include the physiological state of the animal, behavioral features of the animal that contribute to intra- and interspecies interactions, seasonal effects on transmission, and management practices applied to wildlife or domestic animals. The overview concludes with a section directed at discussing other drawbacks to pathogen contamination of wildlife and includes contamination of water sources and wildlife serving as a reservoir for antibiotic resistance and emerging pathogens.

Keywords *Campylobacter* • *Cryptosporidium* • *Escherichia coli* • Foodborne pathogens • Foodborne disease outbreak • *Listeria monocytogenes* • Prevalence • *Salmonella* • *Trichinella* • Wildlife • Zoonosis

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Introduction

Foodborne pathogen contamination of edible horticultural crops, often consumed raw or minimally processed (e.g., fruits, nuts, and vegetables), has over the past few decades been increasingly linked to foodborne illnesses, outbreaks, and recalls (Batz et al. 2012; Berger et al. 2010). Although there are over 250 pathogens and toxins that can be transmitted by food, 31 are classified as major foodborne pathogens (Scallan et al. 2011), and included among that group are zoonotic pathogens or pathogens that affect multiple animal species. Moreover, those bacterial and parasitic pathogens that contribute to the greatest proportion of illnesses and outbreaks in humans (*Campylobacter jejuni*, nontyphoidal *Salmonella enterica*, Shiga toxin-producing *Escherichia coli*, *Listeria monocytogenes*, *Cryptosporidium*) are largely attributed to their proliferation within domesticated animals. However, wild animals may also serve as a reservoir of zoonotic pathogens affecting humans and domesticated animals. It has been reported that 26 % of human pathogens infect both domestic and wild animals (Cleaveland et al. 2001) and, therefore, there is concern that wildlife contributes to the maintenance of infections on domestic animal farms (Liebana et al. 2003). Given the ease with which wild animals may intrude and defecate in produce fields, this group of animals has also raised concern that they are a likely environmental source for contamination of fresh produce (Jay-Russell 2013; Langholz and Jay-Russell 2013).

Another food safety risk from infection of wild animals by human pathogens is the consumption of their meat when the animal is killed and not properly dressed and cooked. Moreover, another potential consequence of pathogen contamination of wildlife is their potential to serve as a reservoir for emerging diseases. For example, approximately 75 % of all diseases, including zoonoses which have emerged in the last few decades, are of wildlife origin (Jones et al. 2008). Based on the concerns associated with foodborne pathogens in wildlife populations, this chapter will provide an overview of this subject and recount some of the incidents in which contaminated wildlife has been directly or indirectly associated with human illness, summarize some of the data collected on the prevalence of foodborne pathogens in wildlife, briefly address factors that affect prevalence levels in wildlife, and finally touch on other drawbacks to pathogen contamination of wildlife that adversely affect humans. The material presented in this chapter is not intended to be comprehensive but to provide a basic understanding of the subject on which subsequent chapters will expand.

Illnesses/Outbreaks Attributed to Contamination of Wildlife

Direct Association: Consumption of Contaminated Meat

Prior to the domestication of animals, wild animals served as the major source of protein for humans. Today, this proportion has decreased dramatically, but consumption of wild game and reptile meat continues to occur by groups that value

Table 1.1 Examples of reports documenting links between human illnesses/outbreaks and consumption of pathogen-contaminated wildlife

Source	Pathogen	Evidence for linkage between wildlife and human illness	Reference
Deer jerky	*E. coli* O157:H7	PFGE patterns of isolates from the patients, jerky, and source deer were identical	Keene et al. (1997)
Undercooked venison		PFGE pattern of the uncooked venison isolate was indistinguishable from the pattern of the clinical isolate	Rabatsky-Ehr et al. (2002)
Deer sausage		PFGE patterns of isolates from deer sausage and patients were identical	Ahn et al. (2009)
Undercooked venison	*E. coli* O103:H2 and O145:NM	PFGE patterns of isolates from patients and venison were indistinguishable	Rounds et al. (2012)
Uncooked liver from wild boar	Hepatitis E	Two patients eating the liver contracted the illness but none of the liver remained for analysis of pathogen contamination	Matsuda et al. (2003)
Raw deer meat		DNA sequence from leftover frozen deer meat was 99.7–100 % identical to the viruses recovered from the four human patients	Tei et al. (2003)
Wild boar meat		Genotype 3 hepatitis E virus RNA was detected in both patient serum and wild boar meat	Li et al. (2005)
Wild boar meat	*Trichinella spiralis*	47 Thai patients became ill after eating wild boar meat. Encysted *Trichinella* larvae were identified in implicated meat	Marva et al. (2005)

PFGE pulsed-field gel electrophoresis

these animals for subsistence or sport hunting. As a result, there are multiple reports whereby consumption of contaminated meat has been directly linked to human illness (Table 1.1). Additional incidents of infections associated with consumption of reptile meat have also been tabulated in the review of Magnino et al. (2009).

In some cases, zoonotic pathogens (e.g., *Brucella* spp., *Trichinella* spp.) have been controlled in domestic livestock herds in developed countries, but continue to circulate in wild animal populations and cause human infections via consumption of mishandled or undercooked game meat. For example, swine *Trichinella* infection has been virtually eliminated in US swine raised in confinement, but human cases are still reported due to transmission via feral swine, bear, and other wild game meat. Additionally, concerns have been raised regarding the potential for infected wild animals to spread the parasite to domestic swine raised outdoors for "pastured pork," a growing niche market (Burke et al. 2008).

An important point to acknowledge, however, is that with many of these foodborne pathogens, the potential for causing illness and the severity of disease will depend on the strain. For example, genotypes 1 and 2 of hepatitis E virus are

restricted to humans and associated with epidemics in developing countries, whereas typically sporadic cases are associated with the zoonotic genotypes 3 and 4 (Meng 2011). As another example, most human illnesses are caused by only three of the serotypes (1/2a, 1/2b, and 4b) of *L. monocytogenes* (Jay-Russell 2013).

Indirect Association: Contamination of Produce Fields

Attention to wild animals serving as a vector for pathogen contamination of produce fields arose following the highly publicized 2006 *Escherichia coli* O157:H7 outbreak associated with ready-to-eat packaged baby spinach that was traced to one field in the central California coast (Jay et al. 2007). In that outbreak, the outbreak strain was isolated from both domestic cattle and feral swine sharing rangeland adjacent to the implicated spinach field. Moreover, evidence of intrusion by the feral swine, including tracks, rooting, or feces in crop fields and adjacent vineyards, was documented.

Other cases that have implicated wild animals as potential sources of contamination of field crops and subsequent infection of humans have been documented. In Finland in 2004, schoolchildren became ill after eating a carrot–white cabbage mixture, with *Yersinia pseudotuberculosis* identified as the likely cause for illness (Kangas et al. 2008). Traceback of the carrots to the processor and farms growing the carrots revealed the presence of this bacterium in one environmental sample from the carrot-peeling processing line and from a pooled sample of common shrew intestines collected from one of the farms. In Alaska, 63 cases of laboratory-confirmed *C. jejuni* infections that occurred in 2008 were associated with the consumption of raw shelled peas (Gardner et al. 2011). Pulsed-field gel electrophoresis (PFGE) patterns of clinical isolates, and pea and Sandhill crane fecal samples taken from the implicated pea farm located near a crane stopover and breeding site, were indistinguishable. Finally, in Oregon in 2011, 14 cases of laboratory-confirmed *E. coli* O157:H7 infections were associated with consuming strawberries purchased at roadside stands or farmers' markets (Laidler et al. 2013). A single farm was identified as the source of the contaminated strawberries, and environmental samples containing visible deer pellets that were collected at the farm were indistinguishable from the outbreak pattern by PFGE.

Prevalence of Foodborne Pathogens in Wildlife

Over the years, numerous studies have been conducted to address the prevalence of bacterial, parasitic, and viral pathogens in wildlife. Initial studies were focused primarily on assessing the degree of contamination resident within an animal's population solely, whereas studies more recently have focused on understanding the factors that contribute to the prevalence in wildlife. For this review, nearly 90 % of the data items included in Tables 1.2, 1.3, and 1.4, covering prevalence of foodborne pathogens in various groups of animals, were obtained from studies conducted in the past

10 years. Results from earlier studies were included to provide an example of a particular animal type or pathogen that may not have been addressed in a recent study. However, there is the possibility that data from older studies are no longer relevant if conditions under which they occurred no longer exist. Additional examples of the prevalence of foodborne pathogens in wildlife have been reviewed in other publications (Simpson 2002; Meerburg and Kijlstra 2007; Benskin et al. 2009; Ferens and Hovde 2011; Langholz and Jay-Russell 2013). In those reviews as well as the data presented in Tables 1.2, 1.3, and 1.4, one observation that is pervasive is the wide range of frequency that foodborne pathogens are detected in wildlife. In the following section, to understand the limitations and strengths of different studies, factors that contribute to pathogen detection in wildlife are discussed.

Factors Affecting Prevalence Levels in Wildlife

Methodology Used for Surveillance

Sample source. One of the common types of samples collected to assess the prevalence of foodborne enteric bacterial pathogens in wildlife is fecal pellets. The assumption in collecting this type sample is that the enteric pathogen either survives in or colonizes the gut of the animal and then is shed with the feces. Studies based on this type of sample, however, may be underestimating the prevalence due to a number of shortcomings. First and foremost is the possibility that contaminated wild animals may only intermittently shed the pathogen as has been reported for pigeons (De Herdt and Devriese 2000). Negative results may also occur when delays in collection of the fecal pellets occur but would vary with the pathogen as they have different degrees of susceptibility to desiccation. Moreover, in collecting fecal waste, there is the assumption that it represents the population at large and that may not be the case, especially if the animals become sick upon infection. Additionally, even a trained biologist may not be able to identify the source of the fecal material on the ground, which may require another method such as wildlife trail cameras or the use of genetic markers to accurately identify feces from different animals. Cloaca or rectum swabs are therefore more accurate in assessing whether carriage of the pathogen by the animal is occurring, but these require capture of the animals. Only on rare occasions are the animal's extremities sampled (Burt et al. 2012) to determine if the animal is serving as a pathogen vector.

Cultivation bias. To detect low levels of bacterial pathogens in a matrix such as feces or food, it is common practice to enrich the sample in a culture broth to increase their numbers and then qualitatively detect their presence using either selective media for colony isolation or a polymerase chain reaction (PCR) assay to screen for the pathogen's DNA. Critical to this approach is the assumption that viable pathogen cells will multiply under the enrichment conditions in the allotted time frame. Unfortunately, it has been observed that *Salmonella* strains vary in their ability to grow in enrichment cultures containing bovine feces (Singer et al. 2009), with strains of serogroups C2 and E more likely to dominate in enrichment culture

Table 1.2 Prevalence of zoonotic pathogens in mammals

Pathogen	Sample description with % prevalence of total number of samples tested in parentheses	Reference
Campylobacter jejuni	Rectal samples from *hares* (4.3 % of 23) in woodlands	Rosef et al. (1983)
	Rectal samples from *rodents* including wood mouse and bank vole (0.0 % of 44) in woodlands	
	Colon, intestinal, or muscle samples from *rodents* including brown rat, house mouse, and yellow-necked mouse (4.5 % of 201) on nine pig farms and five chicken farms	Backhans et al. (2013)
Campylobacter spp.	Fecal samples from *deer* (19.5 % of 113) in Oldman River watershed, Alberta, Canada	Jokinen et al. (2011)
	Fecal samples from *red deer* (0.0 % of 295), *wild boar* (65.5 % of 287), and *other ungulates* including fallow deer and mouflon (0.0 % of 9) in south-central Spain	Díaz-Sánchez et al. (2013)
	Rectal swabs of *wild boars* (43.8 % of 121) and *Sika deer* (0.0 % of 128) in Japan	Sasaki et al. (2013)
Clostridium difficile	Fecal samples from *feral swine* (4.4 % of 161) in North Carolina	Thakur et al. (2011)
	Paws, tail, and snout from *house mouse* (66.0 % of 53) on pig farm	Burt et al. (2012)
	Colons from *rats* (13.1 % of 724) in inner-city neighborhood of Vancouver, Canada	Himsworth et al. (2014)
Enteropathogenic *E. coli* (EPEC), Shiga toxin-producing *E. coli*, enterohemorrhagic *E. coli*	Fecal samples of *roe deer* (17.3 % of 52) and *red deer* (13.6 % of 81) in Belgium	Bardiau et al. (2010)
Shiga toxin-producing *E. coli* (STEC)	Fecal pellets from *rabbits* (20.6 % of 97) during summer on 16 dairy and beef farms	Schaife et al. (2006)
	Tonsil samples from *wild boars* (9.1 % of 153) from Geneva, Switzerland	Wacheck et al. (2010)
	Fecal samples from *red deer* (33.7 % of 264), *wild boar* (3.6 % of 301), and *other ungulates*, including fallow deer and mouflon (33.3 % of 9) in south-central Spain	Díaz-Sánchez et al. (2013)
	Fecal and rectal swabs from *roe deer* (52.5 % of 179), *wild boars* (8.4 % of 262), and *foxes* (1.9 % of 260) from northwest Spain	Mora et al. (2012)
	Fecal samples of *ungulates* (19.4 % of 160) in Idaho	Gilbreath et al. (2009)
	Fecal samples from *roe deer* (73.3 % of 30) and *red deer* (70.0 % of 30) from a national park in Germany	Eggert et al. (2013)

(continued)

Table 1.2 (continued)

Pathogen	Sample description with % prevalence of total number of samples tested in parentheses	Reference
E. coli O157:H7	Colon stool samples from *rodents* including brown rat, wood mice, and house mouse (21.0 % of 19) on beef cattle farm	Čížek et al. (1999)
	Fecal samples from *white-tailed deer* (0.2 % of 1608) in Nebraska	Renter et al. (2001)
	Rectroanal mucosal swabs from *roe deer* (0.0 % of 20), *red deer* (1.5 % of 206), *fallow deer* (0.0 % of 6), and *mouflon* (0.0 % of 11) during hunting season in southwestern Spain	García-Sánchez et al. (2007)
	Fecal samples from *wild boars* (3.3 % of 212) in southwest Spain	Sánchez et al. (2010)
	Buccal swabs, colonic feces, rectal-anal swabs, and tonsils from *feral swine* (40.0 % of 30) on a cattle ranch in California	Jay-Russell et al. (2012)
	Fecal samples from *rodents* (0.2 % of 1043) in 13 agricultural systems (nine produce farms, three cow-calf rangeland operations, and one beef feedlot)	Kilonzo et al. (2013)
E. coli O157:H7, sorbitol-fermenting	Fecal samples from *red deer* (1.1 % of 264) during hunting season in south-central Spain	Díaz et al. (2011)
Non-O157 STEC	Fecal samples from *wild boars* (5.2 % of 212) in southwest Spain	Sánchez et al. (2010)
	Fecal samples in *ruminants*, including red deer, roe deer, fallow deer, and mouflon (23.9 % of 243) in southwest Spain	Sánchez et al. (2009)
	Rectal swabs from several types of *rodents* (4.8 % of 145) in city parks in Buenos Aires, Argentina	Blanco Crivelli et al. (2012)
Atypical EPEC	Fecal samples from *coyotes* (4.9 % of 103) in leafy greens production region at U.S.–Mexico border	Jay-Russell et al. (2014)
Listeria monocytogenes	Tonsil samples from *wild boars* (17.0 % of 153) from Geneva, Switzerland	Wacheck et al. (2010)
Mycobacterium bovis	Tissue from *ferrets* (17.9 % of 548) and *stoats* (1.6 % of 62) in New Zealand	Ragg et al. (1995)
	Tissue rom *hedgehogs* (5.8 % of 69) in tuberculosis-endemic areas of New Zealand	Lugton et al. (1995)

(continued)

Table 1.2 (continued)

Pathogen	Sample description with % prevalence of total number of samples tested in parentheses	Reference
Salmonella spp.	Intestinal samples from *mice* (5.1 % of 175) on six swine farms	Barber et al. (2002)
	Tonsil samples from *wild boars* (12.4 % of 153) from Geneva, Switzerland	Wacheck et al. (2010)
	Fecal samples from *coyotes* (32.0 % of 103) in leafy greens production region at U.S.–Mexico border	Jay-Russell et al. (2014)
	Fecal swabs from *raccoons* in rural (7.8 % of 28), forested (8.7 % of 332), and suburban (5.7 % of 278) areas of western Pennsylvania	Compton et al. (2008)
	Fecal samples from *deer* (0.0 % of 113) in Oldman River watershed, Alberta, Canada	Jokinen et al. (2011)
	Fecal samples from *white-tailed deer* (1.0 % of 500) in southeastern Nebraska	Renter et al. (2006)
	Fecal samples from *rodents* including rats, mice, and voles on 13 production-infected (5.2 % of 135) and non-infected (0.0 % of 68) farms (five pig and eight cattle) and surrounding areas without production animals (0.0 % of 22)	Skov et al. (2008)
	Fecal or cloacal swabs from *Diprotodontia*, including koala, wombats, and possums (1.7 % of 291)	Parsons et al. (2010)
	Fecal samples from *coyotes* (5.0 % of 40), *deer* (1.9 % of 104), *elk* (2.6 % of 39), *wild pigs* (2.4 % of 41), *rabbits* (0.0 % of 57), *raccoons* (0.0 % of 2), and *skunks* (30.7 % of 13) in major produce region of California	Gorski et al. (2011)
	Fecal samples from *feral swine* (5.0 % of 161) in North Carolina	Thakur et al. (2011)
	Fecal and lymph node samples from *wild boars* (41.1 % of 543) at 93 locations in Australia	Cowled et al. (2012)
	Colon, intestinal, or muscle samples from *rodents* including brown rat, house mouse, and yellow-necked mouse (0.0 % of 184) on nine pig farms and five chicken farms	Backhans et al. (2013)
	Fecal samples from *red deer* (0.3 % of 295), *wild boar* (1.2 % of 333), and *other ungulates*, including fallow deer and mouflon (0.0 % of 9) in south-central Spain	Díaz-Sánchez et al. (2013)
	Rectal or cloacal swabs of *deer* (0.0 % of 73) from the Eastern Shore of Virginia	Gruszynski et al. (2013)
	Fecal samples from *rodents* (2.9 % of 1043) in 13 agricultural systems (nine produce farms, three cow-calf rangeland operations, and one beef feedlot)	Kilonzo et al. (2013)
	Intestines, spleens, and livers from *rodents* including rats and mice (10.2 % of 88) on 13 pig farms	Andrés-Barranco et al. (2014)

(continued)

Table 1.2 (continued)

Pathogen	Sample description with % prevalence of total number of samples tested in parentheses	Reference
S. Enteritidis	Liver, spleen, and intestines of four types of *rodents* on *Salmonella*-infected (75.3 % of 483) and non-infected (0.0 % of 232) poultry farms	Henzler and Optiz (1992)
Yersinia enterocolitica	Tonsil samples from *wild boars* (34.6 % of 153) from Geneva, Switzerland	Wacheck et al. (2010)
	Rodents including mice and rats on pig (8.2 % of 110) or chicken (0.0 % of 55) farm	Backhans et al. (2011)
	Colon, intestinal, or muscle samples from *rodents* including brown rat, house mouse, and yellow-necked mouse (4.8 % of 189) on nine pig farms and five chicken farms	Backhans et al. (2013)
Hepatitis E	Sera from *brown rats* (76.8 % of 108) in inner city of Baltimore, Maryland, urban and rural regions of Hawaii, and New Orleans, Louisiana	Kabrane-Lazizi et al. (1999)
	Sera were immunologically assayed for RNA from *wild boar* (12.1 % of 1029), *red deer* (5.3 % of 38), and *roe deer* (0.0 % of 8) in The Netherlands	Rutjes et al. (2010)
	Sera were immunologically assayed from *Yezo deer* (34.8 % of 520) in Hokkaido, Japan	Tomiyama et al. (2009)
	Sera were immunologically assayed from *white-tailed deer* (62.7 % of 142) in Northern Mexico	Medrano et al. (2012)
Nipah virus	Sera from *large flying foxes* (32.8 % of 253) and *small flying foxes* (11.1 % of 117) in Malaysia	Rahman et al. (2013)
	Urine from *Lyle's flying foxes* (1.8 % of 2696) from seven colonies in central Thailand	Wacharapluesadee et al. (2010)
Cryptosporidium spp.	Scats from *wombats* (0.0 % of 55) on stream banks in riparian corridors in Australia	Borchard et al. (2010)
	Fecal samples from *coyotes* (22.2 % of 18), *mountain lions and bobcats* (0.0 % of 11), and *opossums* (25.0 % of 68) in the Monterrey Bay region of California	Oates et al. (2012)
	Colon, intestinal, or muscle samples from *rodents* including brown rat, house mouse, and yellow-necked mouse (11.0 % of 155) on nine pig farms and five chicken farms	Backhans et al. (2013)
	Fecal samples from 11 types of *rodents* (26.0 % of 285) in 13 agricultural systems (nine produce farms, three cow-calf rangeland operations, and one beef feedlot)	Kilonzo et al. (2013)

(continued)

Table 1.2 (continued)

Pathogen	Sample description with % prevalence of total number of samples tested in parentheses	Reference
Giardia spp.	Scats from *wombats* (0.0 % of 55) on stream banks in riparian corridors in Australia	Borchard et al. (2010)
	Fecal samples from *coyotes* (38.9 % of 18), *mountain lions and bobcats* (18.2 % of 11), and *opossums* (14.7 % of 68) in the Monterrey Bay region of California	Oates et al. (2012)
	Colon, intestinal, or muscle samples from *rodents* including brown rat, house mouse, and yellow-necked mouse (13.5 % of 155) on nine pig farms and five chicken farms	Backhans et al. (2013)
	Fecal samples from 11 types of *rodents* (24.2 % of 285) in 13 agricultural systems (nine produce farms, three cow-calf rangeland operations, and one beef feedlot)	Kilonzo et al. (2013)
Toxoplasma gondii	Brain and heart tissue from six types of *rodents* (11.9 % of 101) on three organic pig farms	Kijlstra et al. (2008)
	Sera were immunologically analyzed from *roe deer* (52.0 % of 73) and *red deer* (0.0 % of 7) in Belgium	De Craeye et al. (2011)
	Brain samples from *red foxes* (18.8 % of 304), *roe deer* (5.0 % of 20), and *red deer* (0.0 % of 13) in Belgium	De Craeye et al. (2011)
	Colon, intestinal, or muscle samples from *rodents* including brown rat, house mouse, and yellow-necked mouse (0.0 % of 147) on nine pig farms and five chicken farms	Backhans et al. (2013)
	Sera were immunologically analyzed from *roe deer* (46.4 % of 222) in an agro-system in France	Candela et al. (2014)
Trichinella spp.	Colon, intestinal, or muscle samples from *rodents* including brown rat, house mouse, and yellow-necked mouse (0.0 % of 160) on nine pig farms and five chicken farms	Backhans et al. (2013)

mixtures than strains of serogroups B or C1 (Gorski 2012). To circumvent this limitation due to culture bias, it was recommended that analysis of environmental samples includes multiple enrichment protocols (Gorski 2012); however, there still remains the possibility that the *Salmonella* strain would not be detected if it was incapable of outcompeting indigenous fecal bacteria and growing to sufficient numbers for detection through traditional protocols.

Another complication in the detection of pathogens can occur when using cultural cultivation conditions prior to PCR as was reported for a study of wild mule deer and elk in Idaho (Gilbreath et al. 2009). In this case, loss of the hybridizable *stx* genotype occurred in up to 80 % of subcultured isolates of Shiga toxin-producing *E. coli* (STEC). The question therefore remains as to whether the instability of these genes would have occurred under field conditions and, hence, the risk of human illness associated with these organisms compared to stable STEC isolates.

Table 1.3 Prevalence of zoonotic pathogens in birds

Pathogen	Sample description with % prevalence of total number of samples tested in parentheses	Reference
Campylobacter spp.	Fecal samples from *gulls* (13.7 % of 205) from three coastal locations in Ireland	Moore et al. (2002)
	Cloacal swabs from *yellow-legged gull chicks* (10.4 % of 182) in northeast Spain	Ramos et al. (2010)
	Fecal samples from *gulls* (33.3 % of 3), *geese* (26.2 % of 80), and *ducks* (42.1 % of 38) in Oldman River watershed, Alberta, Canada	Jokinen et al. (2011)
	Fecal samples from *black swan* (45.0 % of 80), *Canada geese* (40.0 % of 80), *duck* (29.0 % of 80), and *gulls* (59.0 % of 80) from New Zealand	Moriarty et al. (2011)
	Oropharyngeal and cloacal swabs from *common tern chicks* (0.6 % of 179) during the breeding season in New Jersey	Rivera et al. (2012)
	Fecal samples from *European starlings* (50.4 % of 113) on dairy cattle farm in NE, Ohio	Sanad et al. (2013)
	Cloacal samples from *griffon vultures* (1.0 % of 97) in eastern Spain	Marin et al. (2014)
Campylobacter jejuni	Fecal samples or cloacal swabs from *graylag geese* (0.0 % of 219), *rock pigeons* (3.0 % of 200), and *mallards* (20.0 % of 5) in Norway	Lillehaug et al. (2005)
	Cloacal samples from feral *pigeons* (69.1 % of 94) in public parks and gardens in Madrid, Spain	Vázquez et al. (2010)
	Fecal samples from *California gulls* (1.2 % of 159) in southern California	Lu et al. (2011)
	Cloacal swab samples from urban *pigeons* (48.3 % of 1800) in coastal area of southern Italy	Gargiulo et al. (2014)
	Fecal and cloacal samples from ten species of *wild birds* (8.1 % of 781) in New Jersey, Delaware, and Pennsylvania	Keller and Shriver (2014)
	Fecal samples from 15 species of *wild birds* (7.4 % of 446) from two ruminant farm sites in Virginia and Maryland	Pao et al. (2014)
	Fecal or cloacal samples from *American crows* (55.1 % of 127) in California	Weis et al. (2014)
Clostridium difficile	Tissue from dead *house sparrows* (65.7 % of 35) on a commercial pig farm in The Netherlands	Burt et al. (2012)
Enteropathogenic *E. coli*	Fecal samples from *ducks* (54.0 % of 50) from a poultry farm in India and cloacal samples from domestic *pigeons* (6.0 % of 100) from seven fanciers in India	Farooq et al. (2009)
	Cloacal or fecal swabs from 15 *avian host sources* (15.3 % of 412) from five locations in British Columbia, Canada	Chandran and Mazumder (2014)
Shiga toxin-producing *E. coli*	Fecal samples from *wild birds*, comprised of 24 species (1.6 % of 244) from cattle and pig farms in Denmark	Nielsen et al. (2004)
	Fecal samples from *ducks* (0.0 % of 50) from a poultry farm in India and cloacal samples from domestic *pigeons* (9.0 % of 100) from seven fanciers in India	Farooq et al. (2009)
	Cloacal or fecal swabs from 15 *avian host sources* (22.6 % of 412) from five locations in British Columbia, Canada	Chandran and Mazumder (2014)

(continued)

Table 1.3 (continued)

Pathogen	Sample description with % prevalence of total number of samples tested in parentheses	Reference
E. coli O157:H7	Fecal samples from *gulls* (0.0 % of 3), *geese* (1.2 % of 80), and *ducks* (2.6 % of 38) in Oldman River watershed, Alberta, Canada	Jokinen et al. (2011)
	Fecal or intestinal contents from *European starlings* (1.2 % of 430) from 150 dairy farms in northern Ohio	Williams et al. (2011)
	Cloacal swab samples from *urban pigeons* (7.8 % of 1800) in coastal area of southern Italy	Gargiulo et al. (2014)
Salmonella spp.	Fecal samples from *birds* (7.9 % of 38) from six swine production facilities in Illinois	Barber et al. (2002)
	Fecal samples or cloacal swabs from *graylag geese* (0.5 % of 219), *rock pigeons* (0.0 % of 200), and *mallards* (0.0 % of 5) in Norway	Lillehaug et al. (2005)
	Fecal samples from *barn swallows* (0.0 % of 500+) in northern, central, and southern Sweden	Haemig et al. (2008)
	Cloacal swabs from *birds* (55 species) at or near *Salmonella*-infected (1.5 % of 185) and non-infected (0.0 % of 1004) cattle and pig farms in Denmark and surrounding areas without production animals (0.0 % of 278)	Skov et al. (2008)
	Fecal or cloacal swabs of *birds* (0.0 % of 689) in Australia	Parsons et al. (2010)
	Cloacal swabs from *yellow-legged gull chicks* (17.0 % of 182) in northeast Spain	Ramos et al. (2010)
	Gastrointestinal tract samples from *European starlings* (2.5 % of 81) in three cattle-concentrated animal feeding operations	Carlson et al. (2011)
	Fecal samples from *birds* (6.6 % of 105) in major produce region of California	Gorski et al. (2011)
	Fecal samples from *gulls* (66.7 % of 3), *geese* (10.0 % of 80), and *ducks* (7.9 % of 38) in Oldman River watershed, Alberta, Canada	Jokinen et al. (2011)
	Fecal samples from *black swan* (0.0 % of 80), *Canada geese* (0.0 % of 80), *duck* (0.0 % of 80), and *gulls* (0.0 % of 80) from New Zealand	Moriarty et al. (2011)
	Oropharyngeal and cloacal swabs from *common tern chicks* (0.6 % of 179) during the breeding season in New Jersey	Rivera et al. (2012)
	Rectal or cloacal, or carapace swabs of *geese* (0.0 % of 7) and *gulls* (29.8 % of 47) from the Eastern shore of Virginia	Gruszynski et al. (2013)
	Cloacal samples from *griffon vultures* (52.6 % of 97) in eastern Spain	Marin et al. (2014)
	Fecal samples from 47 different species of *birds* (4.0 % of 672) on 41 pig farms in Northeast Spain	Andrés-Barranco et al. (2014)
	Cecal samples from *migratory birds*, including brown-headed cowbirds, common grackles, and cattle egrets (14.9 % of 376) during fall migration in Texas	Callaway et al. (2014)
	Fecal swabs from *waterfowl*, including Franklin's gull, kelp gull, grey gull, and Andean goose (6.1 % of 758) from eight sites in five Chilean regions	Fresno et al. (2013)
	Fecal samples from *gulls* (17.2 % of 360) from three landfill sites and on the Eastern shore of Virginia	Gruszynski et al. (2014)
	Fecal samples from 15 species of *wild birds* (0.2 % of 446) from two ruminant farm sites in Virginia and Maryland	Pao et al. (2014)

Table 1.3 (continued)

Pathogen	Sample description with % prevalence of total number of samples tested in parentheses	Reference
Salmonella Oranienburg	Fecal samples from *wild turkeys* (22.5 % of 71) from a home garden fertilized with raw horse manure	Jay-Russell et al. (2013)
S. Typhimurium	Cloacal swab samples from urban *pigeons* (0.9 % of 1800) in coastal area of southern Italy	Gargiulo et al. (2014)
Cryptosporidium spp.	Fecal samples from *gulls* (0.0 % of 205) from three coastal locations in Ireland	Moore et al. (2002)
	Fecal samples from *Canada geese* (23.4 % of 209) from ten sites in Ohio and Illinois	Zhou et al. (2004)
	Fecal samples from *black swan* (2.5 % of 80), *Canada geese* (5.0 % of 80), *duck* (1.3 % of 80), and *gulls* (0.0 % of 80) from New Zealand	Moriarty et al. (2011)
	Fecal samples from *gulls* (0.0 % of 145) in the Monterrey Bay region of California	Oates et al. (2012)
	Fecal samples from *common tern chicks* (72.2 % of 54) during breeding season in New Jersey	Rivera et al. (2012)
Giardia	Fecal samples from *gulls* (2.1 % of 145) in the Monterrey Bay region of California	Oates et al. (2012)

Sample size and number. Depending on the wild animal and its typical fecal mass, the prevalence of zoonotic enteric pathogens may be underestimated. This situation may occur when fecal amounts per assay are less than 0.10 g and pathogen shedding intensity is low. Under these conditions, there occurred an artificial downward bias for the prevalence by well over 50 % (Atwill et al. 2012). Such a situation would explain why double sampling improved the detection of methicillin-resistant *Staphylococcus aureus* carriage in 4 different types of wild animals in Spain (Concepción Porrero et al. 2013).

Surveillance of wildlife in many studies has been conducted with samples obtained by trapping the animals or collecting samples from hunters. Although such sampling is assumed to be representative of a population, Hoye et al. (2010) suggested that it likely involved selection bias, making it difficult to develop statistically valid estimates of pathogen prevalence. Hence, to enhance the design and interpretation of wildlife surveys, these investigators also provided estimates of the number of animals that should be sampled to achieve the study's objective (establishing absence of infection or an estimate of pathogen prevalence).

Specificity of detection method. A number of methods for detecting enteric foodborne pathogens in wildlife have been used and vary in their specificity relative to the organism present. Culture-based assays, for example, are often only capable of specifying the bacteria growing on a specific agar by its genus (i.e., *Campylobacter* spp., *Salmonella* spp., etc.), hence it is not possible to know if the pathogen is pathogenic. More recent studies that are conducted often employ advanced assays to characterize the phenotypic and genotypic properties of organisms isolated from wildlife so that these isolates can be compared to isolates associated with human illness. Another purpose for molecular characterization of wildlife isolates is for comparison to isolates obtained from domestic animals or to isolates obtained over

Table 1.4 Prevalence of zoonotic pathogens in amphibians and reptiles

Pathogen	Sample description with % prevalence of total number of samples tested in parentheses	Reference
Campylobacter spp.	Cloacal samples from *European pond turtle* (0.0 % of 83) and *red-eared terrapin* (0.0 % of 117) in 11 natural ponds in eastern Spain	Marin et al. (2013)
	Fecal samples from *bullfrogs* (0.0 % of 164) from Belgium and The Netherlands	Martel et al. (2013)
Mycoplasma spp.	Oral and cloacal swabs from *tortoises* (36.7 % of 30) in Italy	Lecis et al. (2011)
Salmonella spp.	Cloacal swabs of free-ranging *alligators* (2.8 % of 71) in southeast Texas and south Louisiana	Scott and Foster (1997)
	Fecal samples from *reptiles* (41.5 % of 94) and *amphibians* (0.0 % of 72) in Spain	Briones et al. (2004)
	Fecal or cloacal swabs of *frogs* (0.0 % of 106), *lizards* (10.7 % of 298), *crocodiles* (3.0 % of 33), *snakes* (0.0 % of 48), and *turtles* (0.0 % of 64) in Australia	Parsons et al. (2010)
	Oral and cloacal swabs from *tortoises* (10.0 % of 30) in Italy	Lecis et al. (2011)
	Cloacal swabs and cecal contents from *cane toads* (41.4 % of 58) in Grenada	Drake et al. (2013)
	Cloacal and ventral swabs as well as washes from *frogs* (1.2 % of 331), *lizards* (9.0 % of 59), *newts* (0.0 % of 5), *salamanders* (0.0 % of 6), *snakes* (59.0 % of 39), and *toads* (5.0 % of 20) in a produce-growing region of the California Central Coast	Gorski et al. (2013)
	Cloacal swab samples from *caimans* (13.9 % of 21), *turtles* (21.2 % of 32), *green iguanas* (15.2 % of 23), *other lizards* (25.2 % of 38), and *snakes* (24.5 % of 37) from French Guiana	Gay et al. (2014)
	Fecal samples from *bullfrogs* (0.0 % of 164) from Belgium and The Netherlands	Martel et al. (2013)
	Cloacal swabs from *wild green iguanas* (57.4 % of 47) in Grenada	Sylvester et al. (2014)

extended periods of time. Hence, isolates obtained from different animals, but having similar or identical molecular profiles, can be evidence that transmission between the two groups occurred (Williams et al. 2011), whereas isolates with similar profiles obtained at different time points can be evidence that they are persistent in the environment (Gorski et al. 2011). Detection of viruses and parasites in animals, however, often rely on immunological assays to detect antibodies in the serum that have been expressed when the pathogen invades the animal's system. The drawback to immunological assays, however, is that immunity may extend for periods long after the pathogen is eliminated from the animal. Examples of studies that have employed serotyping and molecular characterization of isolates recovered from wildlife as a means to measure their potential to serve as a reservoir of infection for humans or animals are listed in Table 1.5.

Table 1.5 Selected studies employing serotyping or molecular characterization of wildlife isolates to assess their contribution as reservoirs of foodborne pathogens to humans or domesticated animals

Pathogen	Observations and overall conclusions of study	Reference
Campylobacter spp.	Although culture-based and genus-specific PCR-detection assays attributed 45 % positive samples from gulls as *Campylobacter* spp., sequence analysis revealed that most were novel *Campylobacter* spp. and would not likely present a major public health risk. A smaller proportion of the sequences (2–5 %) were closely related to pathogenic species (*C. jejuni* and *C. lari*) at a 99 % or greater sequence identity	Lu et al. (2011)
Campylobacter jejuni	Serotype distribution of isolates collected from multiple species of wildlife was significantly different from known distributions in broilers and humans. Macrorestriction profiles of two serotypes (O:12 and O:38) isolated from wildlife and other sources indicated propagation in a wide group of animal species but were not detected in humans or broilers. The typing methods used revealed that wildlife is of limited importance as a reservoir of infection	Petersen et al. (2001)
	Despite this pathogen being genotypically diverse and host restricted, PFGE analysis revealed that there were several shared genotypes between dairy cattle and starling isolates recovered from the same farm. Moreover, multilocus sequence typing (MLST) analysis revealed that cattle and starlings also shared many clonal complexes. Overall, these results highlighted that starlings serve as potential reservoirs for *C. jejuni*	Sanad et al. (2013)
Escherichia coli O157:H7	PFGE patterns of *E. coli* O157:H7 isolates recovered from a wild boar and from a human patient with diarrhea living in the same geographic area were indistinguishable	Sánchez et al. (2010)
	Isolates obtained from fecal samples of dairy cattle and European starlings collected on different farms were subjected to multiple-locus variable-nucleotide tandem repeat analysis (MLVA). Indistinguishable subtypes between starlings and cattle on different farms were found, supporting the hypothesis that these birds contributed to the transmission of *E. coli* O157:H7 between dairy farms	Williams et al. (2011)
Salmonella spp.	22 different serotypes of *Salmonella* were isolated from wild amphibians and reptiles. Many of the isolates were of subtypes IIIa and IIIb, which are less frequently associated with human infections than subtype I	Gorski et al. (2013)
	Twenty-two serotypes of *Salmonella* were isolated from gull fecal samples, with the most common serovars (Infantis [21 %], Typhimurium [12.9 %] and Newport [11.3 %]) also recognized by CDC's FoodNet as being in the top ten laboratory-confirmed *Salmonella* serotypes (CDC 2013). PFGE patterns from gull isolates that were clustered in time and space are evidence that gulls are colonized by *Salmonella* in the environment for short periods of time, but during those times, would be capable of contaminating nearby tomato fields	Gruszynski et al. (2014)
Toxoplasma gondii	Although seroprevalence of deer for *T. gondii* was 45.2 %, this pathogen was only found in 3.0 % of deer brain tissue when analyzed by PCR. In contrast, PCR detected this pathogen in 18.8 % of red fox brain tissues. Genotyping of the fox samples revealed that all but one was type II, which is the predominant genotype among patients who acquired toxoplasmic infection in Europe (Ajzenberg et al. 2009)	De Craeye et al. (2011)

Host Attributes That Impact Contamination by Pathogens

Physiological state of host. Prevalence of foodborne pathogens within a wildlife population is often not uniform but is influenced by the physiological state of the individuals. One phenotypic variable that differentiates a population into distinct groups is age, with younger animals being more susceptible to infection than adults. As examples, carriage of *Clostridium difficile* was more common in younger urban Norway rats than in their adult counterparts (Himsworth et al. 2014), and *Campylobacter*'s prevalence in feral swine (Jay-Russell et al. 2012) and in Canada geese (Keller and Shriver 2014) was greater in younger versus older animals. Similarly, when prevalence in both domestic and wild animals was investigated using multiple logistic regression models, it was determined that young animals were approximately twice as likely to shed *Cryptosporidium* and *Giardia* in their feces than adults (Oates et al. 2012). In these cases, establishment of the pathogen in the young animal's gut maybe due to the presence of an immature gut microbiota which, when mature, would in older animals outcompete the pathogen and prevent colonization. This relationship, however, has not been observed in all cases. For example, the percentage of carriers of pathogenic *E.coli* (EPEC, EHEC, and STEC) by wild cervids (red and roe deer) did not differ between adult and juvenile/sub-adult animals (Bardiau et al. 2010), whereas in pteropid bats, a greater number of adults were seropositive for Nipah virus than juveniles or pups (Rahman et al. 2013).

Another characteristic of individuals within an animal population that is associated with different degrees of pathogen prevalence is the sex of the animal. In the case of feral swine sampled in Geneva, Switzerland, 71 % of females carried one or more foodborne pathogens compared to 53 % of males (Wacheck et al. 2010). Similarly, more feral swine females were positive for *Campylobacter* than were males in a study conducted in California (Jay-Russell et al. 2012). In contrast, in another study conducted in California that included both domestic and wild animals, but no feral swine, males were 1.2 times more likely to be *Giardia* spp.-positive than were females (Oates et al. 2012). Sex, however, was not a notable variable for prevalence of pathogenic *E. coli* in roe and red deer (Bardiau et al. 2010), nor was it associated with the seroprevalence of Nipah virus in bats (Rahman et al. 2013). A higher rate of seropositivity to Nipah virus was observed in nursing bats which was attributed to the increased stress that they have experienced in reproductive and nursing activities, which in turn likely increased their risk for infection (Rahman et al. 2013). Pathogen prevalence differences between sexes may also be attributed to behavioral differences that occur between the sexes. For example, in a wild pig population in Australia, transmission of *Salmonella* was more common between males than females and was attributed to the previous observations that adult male pigs have larger home ranges than females, and were more often found associating in small male groups in the study area (Cowled et al. 2012).

In general, susceptibility of animals to infection by foodborne pathogens increases with diminished health or increased stress, both of which compromise the immune system. For example, when wild animals are sampled during the hunting

season and would be under increased stress, there is a greater likelihood that the pathogen would be present if the animal was recently exposed to the pathogen. Hence, studies that rely on this method of collection may be measuring prevalence that would not be typical throughout the year. Good health and decreased stress, however, do not always translate into reduced pathogen prevalence in wildlife. In the wild pig population in Australia, better conditioned (fatter) pigs were associated with an increased probability of infection (Cowled et al. 2012). To explain this statistic, the authors suggested that the better body conditions of these pigs actually enabled them to travel farther and forage more effectively and widely for food, and in turn be exposed to more pathogens.

Another factor that affects the efficacy of an animal's immune system to combat colonization by foodborne pathogens is the exposure dose. For example, European starlings transiently excreted *E. coli* O157:H7 following a low-dose inoculation, but when exposed to a population greater than 5.5 log, shedding occurred in 50 % of the birds for more than 3 days (Kauffman and LeJeune 2011). Similarly, exposures to high pathogen dosages have resulted in both rats and pigeons fecally shedding *E. coli* O157:H7 for longer periods of time than if exposed to smaller dosages (Čížek et al. 2000).

Although immunity to pathogens is usually considered beneficial from an individual standpoint, when the animal population is only partially immune and exposed to a new source of the pathogen, the pathogen may actually survive within the population for longer periods of time and increase the risk of spread to non-infected animals. Such a situation has been proposed as the scenario leading to the outbreak of Nipah virus in Malaysia during 1998–1999 (Epstein et al. 2006). More specifically, it was hypothesized that Nipah virus-infected fruit bats were attracted to fruit trees surrounding a large intensive pig farm and led to an initial infection that died out quickly. In the subsequent year, reintroduction of the virus into a partially immune population resulted in prolonged circulation on the farm, and when these infected pigs were sold from the affected farm and transported to other areas where there was a high density of smaller intensive pig farms and a high human density, a large outbreak occurred in humans, stimulating an investigation.

Hosts' behavioral features that contribute to intra- and interspecies interactions and pathogen transmission. Contamination of wildlife by foodborne pathogens requires that the wild animal first be exposed to a pathogen source which is often related to the animal's behavior patterns and food choices. Once pathogen transmission to the wild animal has occurred, that animal may then serve as a vehicle for intra- or inter-species transmission to other non-infected animals, but the extent to which that occurs will be dependent on the animal's behavioral patterns and whether the infection is self-limiting or not. Multiple examples illustrating the relationship between behavioral attributes and the observed or perceived potential for pathogen transmission between wildlife are presented in Table 1.6.

In general, pathogenic *E.coli* is found in many wild animals at a low prevalence due to limited intra-species interactions (Nielsen et al. 2004). Moreover, when wild animals are contaminated, the animal has likely been living close to domestic animals whose infection is perpetuated by their high-density living conditions (Díaz-

Table 1.6 Selected examples of behavioral attributes of wildlife that affected their intra- and interspecies interactions and the degree of foodborne pathogen transmission

Examples	Reference
Salmonella was detected in wild birds on pig and cattle farms carrying *Salmonella*-positive production animals and only during the periods when *Salmonella* was detected in the production animals. Presence of *Salmonella* in wild birds significantly correlated to their migration pattern and food preference. More specifically, partially migratory or short-to-medium distance migratory birds were at a higher risk of contracting *Salmonella* infections than non-migrating (resident) birds. Birds feeding on insects and invertebrates in the summer were at a higher risk of infection compared to birds feeding on seeds and grains. A slightly higher risk of *Salmonella* detection occurred for birds foraging on the ground in comparison to aerial foraging or foraging in the vegetation	Skov et al. (2008)
Campylobacter occurrence in gull chicks was directly related to their degree of refuse consumption, whereas *Salmonella* prevalence did not reflect any dietary relationship. Gulls are attracted to garbage dumps, untreated sewage, and manure where pathogens may be consumed or physically attached to the birds, allowing the animals to spread the pathogen to other resting areas, including agricultural fields	Ramos et al. (2010)
In ten concentrated animal feeding operations (CAFOs), *Salmonella* contamination of cattle feed and water troughs occurred where starlings also frequented. Probability of contamination of cattle feed by *Salmonella* increased as the number of starlings in feed troughs increased. *Salmonella* contamination in water troughs increased asymptotically as the numbers of starlings on CAFOs increased. Although carriage of *Salmonella* was documented in starlings, the serotype did not match the serotypes in the water and feed samples, suggesting that fecal material adhering to their feet and feathers was the likely mode of dissemination of the pathogen	Carlson et al. (2011)
Toxoplasma gondii was detected in red foxes at a greater frequency than in deer. Foxes are carnivores and are considered opportunistic feeders. Due to these feeding habits, foxes are more likely to have greater exposure to both parasites' tissue cysts and oocysts compared to deer, which largely feed on grass, leaves, young shoots, and berries	De Craeye et al. (2011)
Feral swine were observed to be infected with *Campylobacter* strains that were more often associated with cattle. Feral swine were observed sharing pastures, food, and water with cattle. Rooting in cow pats and wallowing in riparian areas of the pastures where cattle waste was deposited likely contributed to the *Campylobacter* contamination of feral swine	Jay-Russell et al. (2012)
Pathogen clustering occurred in deer mice. Frequent licking of extremities and close contact between deer mice may facilitate fecal–oral transmission and maintenance of infection within a close-knit population	Kilonzo et al. (2013)
Seroprevalence for *Toxoplasma gondii* was less in juvenile females of roe deer than in juvenile males. It was speculated that young males differed from young females by being more exploratory that increased their likelihood of consumption of vegetation on contaminated soil	Candela et al. (2014)

The highest prevalence of *C. jejuni* was in ruddy turnstones (a shorebird that undergoes long-distance complete migrations). The second highest prevalence occurred in Red Knots and Semipalmated Sandpipers, both of which undergo long-distance migrations. These birds often intermingle on stopover sites, providing opportunities for bacterial exchange. In comparison, lower prevalences were in the Snow Geese, another migratory bird, and in resident Canada Geese	Keller and Shriver (2014)
It was determined statistically that starling roosts were spatially associated with an increased prevalence of *E. coli* O157:H7 infection in dairy cattle, a greater diversity of distinguishable MLVA types, and a higher number of isolates with MLVA types from starling-bovine clades versus bovine-only clades. These data were supported by the behavioral patterns of starlings in which starlings tend to fly directly from their night roosts to preferred dairies in the morning; however, on the return flights, the birds often stop at farms along the flight routes	Swirski et al. (2014)
Campylobacter jejuni were detected in high frequency in American crows. This bird forages in a variety of settings, including dumps, animal feedlots, pastures, and urban areas, and therefore has the potential to transfer pathogens from waste sites to other uncontaminated areas	Weis et al. (2014)

Sánchez et al. 2013). Hence, when conditions of low stocking density were encountered in rangeland beef cattle, it was speculated that those conditions were responsible for the lack of interspecies transmission of *Cryptosporidium* from infected cattle to susceptible rodents (Kilonzo et al. 2013).

In cases when pathogen prevalence is low in a wildlife population, the contribution of these animals to persistence on source farms and to transmission between farms is minimal compared to animals, such as red deer and feral swine, whose gut is colonized by *E. coli* O157:H7 (Díaz-Sánchez et al. 2013) and *Campylobacter* (Jay-Russell et al. 2012), respectively. In either case, however, infected wild animals that are extremely mobile amplify the likelihood of transmission by disseminating the pathogen through uncontrolled routes. An example is migratory birds that often associate with cattle (brown-headed cowbirds, common grackles, and cattle egrets); such birds become infected with *Salmonella* and *E. coli* O157:H7 while migrating (Callaway et al. 2014). In other situations, wildlife behavior has become less of an issue as expansion of agriculture into wildlife habitats has created new opportunities for spillover of pathogens from domestic animals to wildlife or vice versa (Jones et al. 2013). A good example would be the emergence of Nipah virus in Malaysia where intensification of the pig industry combined with fruit production occurred in an area already populated by Nipah virus-infected fruit bats (Epstein et al. 2006).

Climatic (seasonal) impacton pathogen prevalence in wildlife. Many surveys on pathogen prevalence in wildlife have been conducted by collecting samples at different times of the year or over multiple years to determine if fluxes in prevalence occur in response to climatic or environmental changes. Through knowledge of seasonal preferences, pathogen transmission dynamics may be better understood and could assist in defining effective interventions for disease management.

One study addressing a seasonal preference in disease transmission of Nipah virus was conducted in Central Thailand, with differences observed between the Malaysian and Bangladesh strains (Wacharapluesadee et al. 2010). The Bangladesh strain was almost exclusively detected during April to June, whereas the Malaysian strain was found dispersed during December to June; however, the cause for these differences could not be determined. In another study, seasonal shedding patterns were observed in wild rodents, with fewer rodents trapped during the spring and summer months shedding *Cryptosporidium* oocysts than rodents trapped during autumn (Kilonzo et al. 2013). In this case, higher prevalences in autumn may have been linked to the breeding cycles of the animals, since most of the animals give birth during the warmer months of the year and begin to disperse in autumn (Ziegler et al. 2007). Behavior may have also contributed to the seasonal prevalence patterns observed in Canada geese, with prevalence of pathogenic *E.coli* being positively correlated with prevailing warmer seasonal temperatures, being higher in the spring and summer and lower during the fall and winter (Kullas et al. 2002). In this case, it was hypothesized that during the fall and winter, the daily movement patterns of the birds largely occurred on dry upland harvested grain fields located outside of town and away from habitats contaminated with mammalian sources of *E. coli*. In contrast, during the spring and summer, the birds did not move far from their nests

during breeding and these areas consisted of small water impoundments and littoral zones that easily become fouled. A similar scenario was offered as an explanation for the dominance of *Salmonella*-positive birds or rodents detected in the winter compared to the summer in that wildlife moved closer to farms in winter in search of food and shelter (Skov et al. 2008).

Long-term climatic fluxes may also impact on the degree of pathogen prevalence in wildlife over successive years. Moreover, the impact of this variable may be more evident in wildlife where population turnover is greater. For example, the sharp declines in *T. gondii* seropositive roe deer from one year to another were partly explained by the replacement of seropositive individuals with new seronegative ones (Candela et al. 2014).

Management practices applied to wildlife or domestic animals that influence pathogen prevalence in wildlife. To minimize the transmission of enteric pathogens from wildlife to production sites (both animal and produce), efforts to control access of wildlife into those sites have been studied but with variable results. Furthermore, the type of management will vary depending upon whether the wild animal species is an invasive species causing environmental and economic damage or if it is considered an endangered or threatened species and is protected. When animals such as starlings that are not protected are targeted, as they are in the United States and Australia, management focuses on lethal control that includes chemical toxicants and shooting. Even in these situations, however, these methods are not always foolproof as evident in an Australian study that found a chemical toxicant to be ineffective because of poor bait acceptance (Bentz et al. 2007). When the target wild animal for control is a species of conservation concern, managing that animal's activities becomes more complex. Examples of non-lethal management techniques include non-lethal chemical repellants (Glahn et al. 1989), exclusionary devices (Khan et al. 2012), and frightening devices (Berge et al. 2007). As an example of the effectiveness of exclusionary devices, bats contacted date palm sap at a 2 % frequency when the food source was protected by a bamboo, *dhoincha*, jute stick, or polythene skirt compared to a frequency of 83 % when the date palm sap was not protected by a skirt (Khan et al. 2012). Enhanced efficacy of any of these management tools, however, generally requires that they be used in tandem or switched on a regular basis (Berge et al. 2007). In addition, direct management tools are not always the most effective means by which to reduce prevalence or sharing of pathogens between domestic and wild animals. For example, Mentaberre et al. (2013) determined in Northeastern Spain that cattle removal was more efficient than the culling of wild boar by hunting or trapping as a means of reducing the prevalence of shared serotypes of *Salmonella*.

In many cases, management of the pathogen prevalence of wildlife requires a systems approach. For example, to understand potential inter-species transmission pathways among wild and domestic ungulates in Kenya, *E.coli* collected from feces were genetically compared (VanderWaal et al. 2014). Under the assumption that when two individuals shared the same genetic subtype of this organism, they were part of the same transmission chain, the zebra was identified as an animal bridging distinct transmission networks. Therefore, these investigators hypothesized that interventions targeted at the zebra would diminish transmission among discrete networks.

Other Drawbacks to Pathogen Contamination of Wildlife That Impact Humans

Contamination of Water Sources

Although the focus of this chapter to this point has been primarily aimed at the contribution of wildlife to the direct spread of enteric foodborne pathogens to either animal production facilities or produce fields, wildlife may also indirectly impact these sites through contamination of water sources that would subsequently be used in agricultural production. In fact, the number of studies addressing this latter route of contamination is much greater than those addressing the direct routes for pathogen transmission. Support for this route of contamination stems first from the results of studies revealing that enteric foodborne pathogens are in surface and irrigation waters. For example, a study of ten irrigation ponds in Georgia revealed that nine of the ponds were contaminated with *C. jejuni* at some point during the year, with an overall prevalence of 19.3 % (Gu et al. 2013b), *E. coli O157* was found in all ponds occasionally, but mainly in summer and fall (Gu et al. 2013a), and *Salmonella* was found in 39 % of pond samples (Aminabadi et al. 2013). The presence of pathogens in surface water is not unique to Georgia as *Salmonella* was also detected in 7.1 % of surface waters in a major produce region of California (Gorski et al. 2011) and in 94 % of surface irrigation water sources in New York (Jones et al. 2014). In a later study of water and sediment from leafy green produce farms and streams on the Central California coast, *Salmonella* was detected in 6.2 % of water and 4.3 % of sediment samples, and *E. coli* O157 was detected in 13.8 % of water and 1.7 % of sediment samples (Benjamin et al. 2013). In all these cases, it is presumed that contamination of these water sources by wildlife could occur either directly or through storm runoff of adjacent contaminated lands.

An additional line of evidence for pathogen contamination of waterways and irrigation ponds by wildlife is through two avenues of exploration. First, studies have documented either directly or indirectly (through global positioning collars) that wildlife accesses water sources and engages in behavior that would lead to contamination of the water (Hampton et al. 2006; Cooper et al. 2010). In the second approach, pathogen isolates obtained from water and wildlife fecal samples have been compared by serotyping and molecular subtyping to determine their similarity. Similar serotypes of *Salmonella* have been isolated from both water and wildlife samples (Gorski et al. 2011; Jokinen et al. 2011; Aminabadi et al. 2013). Molecular typing by restriction fragment length polymorphism of *C. jejuni* isolates from water has revealed clustering with duck and geese isolates (Jokinen et al. 2011). Not all studies, however, have established a relationship between pathogen isolates in water and wildlife. For example, Gorski et al. (2013) detected *Salmonella* in both wildlife and associated water samples; however, the PFGE of the isolates did not match. In another study, *Campylobacter* was detected in shorebird excreta but was not found in the water samples collected from locations presumed to be impacted by these

birds (Ryu et al 2014). These latter results suggest that large numbers of animals may be needed to impact the water quality especially if the animal has low resident populations of a given pathogen. Large resident pathogen populations in a waterway, however, are not always indicative of a large public health risk. Many pathogen parasite lineages, such as *Cryptosporidium* and *Giardia*, are host specific and not zoonotic to the human population at large. However, such parasites could still cause opportunistic infections in animals and humans. Hence, the significance of wildlife contamination of water sources with foodborne pathogens must be determined on a case-by-case basis.

Reservoir for Antibiotic Resistance

Over the past two decades, a growing concern has arisen regarding antimicrobial resistance in pathogenic and commensal bacteria. These concerns extend to wildlife as antibiotic-resistant pathogenic bacteria have been associated with wildlife (Bardiau et al. 2010; Drake et al. 2013; Fresno et al. 2013; Gorski et al. 2013; Sasaki et al. 2013; Sylvester et al. 2014) and is evidence of transmission from environmental sources or animals that have been exposed to antibiotic therapy. Unfortunately, the presence of antibiotic-resistant pathogens in wildlife only serves to perpetuate this human health problem, especially as limited options are available to effectively control these animal populations.

Reservoir for Emerging Pathogens

Pathogens, including those of foodborne origin, have an extremely high evolutionary potential given their large populations, high genetic variation, and short generation times. Given that approximately 75 % of emerged diseases, including zoonoses, were of wildlife origin (Jones et al. 2013), efforts are being intensified to focus on these animals as the driving forces (i.e., climate change, agricultural expansion, urbanization, and habitat destruction) continue to have an impact. The emergence of Nipah virus, for example, revealed the interplay between several of those driving forces (Daszak et al. 2013; Hayman et al. 2013). Another example for which wildlife has appeared to play a role in the evolution of foodborne pathogens is with enterohemorrhagic *E. coli* (EHEC) O157 (Jenke et al. 2012). By sequencing O157:H7/H- isolates, it was determined that deer occupied an intermediate position between O55:H7 and both sorbitol-fermenting (SF) and non-SF O157 branches. Based upon a study of Díaz et al. (2011), it also appeared that free-ranging red deer has been a possible reservoir of Stx-negative derivatives of SF O157:H7.

Summary

Additional research and monitoring of foodborne pathogen carriage by wildlife is needed to better elucidate transmission cycles, temporal–spatial fluctuations, and emerging strains with the goal of reducing potential risks to public health. Given the large number of wildlife species as well as the large number of foodborne pathogens, this task may be daunting. To date, numerous studies have already been conducted to investigate the prevalence of foodborne pathogens in many of the wildlife species that could interface with humans or their agricultural activities. To ensure that available resources applied to future surveillance have the greatest impact, prioritizing the pathogens and wildlife as to their need for surveillance using risk assessment systems, such as the Wildtool described by Tavernier et al. (2011), should be considered.

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