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Bacterial pathogens in wild birds: a review of the frequency and effects of infection

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ABSTRACT

The importance of wild birds as potential vectors of disease has received recent renewed empirical interest, especially regarding human health. Understanding the spread of bacterial pathogens in wild birds may serve as a useful model for examining the spread of other disease organisms, both amongst birds, and from birds to other taxa. Information regarding the normal gastrointestinal bacterial flora is limited for the majority of wild bird species, with the few well-studied examples concentrating on bacteria that are zoonotic and/or relate to avian species of commercial interest. However, most studies are limited by small sample sizes, the frequent absence of longitudinal data, and the constraints of using selective techniques to isolate specific pathogens. The pathogenic genera found in the gut are often those suspected to exist in the birds' habitat, and although correlations are made between bacterial pathogens in the avian gut and those found in their foraging grounds, little is known about the effect of the pathogen on the host, unless the causative organism is lethal. In this review, we provide an overview of the main bacterial pathogens isolated from birds (with particular emphasis on enteropathogenic bacteria) which have the potential to cause disease in both birds and humans, whilst drawing attention to the limitations of traditional detection methods and possible study biases. We consider factors likely to affect the susceptibility of birds to bacterial pathogens, including environmental exposure and heterogeneities within the host population, and present probable avenues of disease transmission amongst birds and from birds to other animal taxa. Our primary aim is to identify gaps in current knowledge and to propose areas for future study.

Key words: avian bacterial diseases, detection methods, enteropathogens, exposure, human waste, pathogen transmission, *Salmonella*, sewage, susceptibility, wild birds.

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I. INTRODUCTION

The emergence of new infectious diseases in wildlife (Alexander, 2003; Capua & Alexander, 2002; Rappole, Derrickson & Hubálek, 2000), and their potential threat as zoonoses (see Table 1 for glossary of terms), has increased general interest in birds as vectors of pathogens and their role in disease epidemiology. Birds are susceptible to many bacterial diseases common to humans and domestic animals (Broman, *et al.*, 2002; Kapperud & Rosef, 1983; Keymer, 1958; Wilson & MacDonald, 1967), and also to other potentially infectious microorganisms, including protozoa and viruses, such as the influenza A virus. Various wildfowl species serve as natural reservoirs for this virus, and have been the source of highly virulent strains that have caused a number of major pandemics over the last century (Capua & Alexander, 2002; Gauthier-Clerc, Lebarbenchon & Thomas, 2007). The virus can pass from infected birds to pigs, and on to humans (Trampuz *et al.*, 2004), though direct transfer from birds in close proximity to humans can also occur (Webster, 2004). Similarly, wild birds may act as natural reservoir hosts for West Nile virus, infecting mosquitoes, which in turn may infect other birds, horses and humans (Rappole & Hubalek, 2003), causing fatal encephalitis (Reed *et al.*, 2003).

Although viral transmission differs from bacterial transmission in a number of important ways (Anderson & May, 1992; Nelson, Williams & Graham, 2005; Swinton *et al.*, 2002), understanding the spread of avian bacterial pathogens may serve as a useful model for examining the spread of other disease organisms, both amongst birds and from birds to other taxa. Using bacterial pathogens as model organisms has the key advantage that they are often safer to work with than viral pathogens. Empirical studies documenting bacterial intestinal flora of wild birds are sparse; the majority have determined the prevalence of specific strains of bacteria that may present a potential health threat to humans or domestic animals (Daniels, Hutchings & Greig, 2003; Goodchild & Tucker, 1968; Johnston, MacLachlan & Hopkins, 1979; McClure, Eveland & Kase, 1957; Williams, Richards & Lewis, 1976). Studies of enterobacterial infections and carriage rates in wild birds have so far concentrated on those avian species most likely to acquire bacteria from human sources, especially gulls (*Larus* spp.), with salmonellae, campylobacters and *Escherichia coli* being the prevailing causative organisms of interest (Allos, 2001; Broman *et al.*, 2002; Casanovas *et al.*, 1995; Hurvell, 1973; Kapperud & Rosef, 1983; Keymer, 1958; Varslot, Resell & Fostad, 1996; Wilson & MacDonald, 1967). Avian feeding ecology appears to be a key determinant of enterobacterial acquisition (Cornelius, 1969; Fenlon, 1983; Williams *et al.* 1976), though different avian species seem to vary in susceptibility to enteropathogenic bacteria (Butterfield *et al.*, 1983; Fenlon, 1981; Fricker, 1984; Sixl *et al.*, 1997). Most data on the prevalence of enteropathogenic bacteria in passerines come from veterinary studies focussing on disease outbreaks resulting in high mortality (Faddoul, Fellows & Baird, 1966; Keymer, 1958; Kirkwood, Holmes & Macgregor, 1995). While these studies may give some indication of the frequency with which birds die from different infections, they provide little or no information on the bacterial source, or the prevalence of the pathogens in apparently healthy individuals. The role of birds as vectors of disease could be underestimated, as many individuals may asymptomatically harbour sub-lethal levels of potentially pathogenic bacterial species (Fenlon, 1981, 1983; Fricker, 1984). In contrast to wild birds, the gastrointestinal flora and the processes of disease transmission in commercially bred poultry have been extensively studied (Barnes, 1979; Basher *et al.*, 1984; Davies & Wray, 1996; Evans & Sayers, 2000), due to the zoonotic threat to the economic value of the industry. Whilst the types of pathogens that cause disease in poultry, their relative infectivity and the resulting symptoms in the birds, may help us to understand the dynamics of disease transmission in wild birds, it is difficult to extrapolate from the highly artificial conditions of the poultry house to free-living avian species. Though many of the bacterial enteropathogens that affect poultry have also been isolated from wild birds, relatively little is known about their effect on wild populations, with the exception of outbreaks of lethal diseases. Due to the general lack of interest in wild birds as zoonotic vectors of disease, combined with their relatively low commercial value, few studies have examined their normal gastrointestinal flora. However, bird feed manufacturers have increased the economic value of wild birds, and concurrently drawn attention to their potential as vectors of disease. Identification of the normal microfloral components of the avian gut is important if we are fully to understand the complexities of enterobacterial interactions within the bird, and to appreciate how inherent gut bacteria may influence the susceptibility of the host to pathogens acquired from the environment. Likewise, a sound understanding of non-enteropathogenic infections can provide insight into the transmission dynamics of other types of avian pathogen.

Table 1. Glossary of terms.

Term	Definition used herein
Abundance	The relative quantitative representation of bacterial species within the study organism or ecosystem
Acquired immunity	Immunity acquired by infection or vaccination as a result of the development of antibodies through exposure to an infective agent
Carriage rate	The frequency with which a microorganism is present in a population of (asymptotic) carriers, usually determined by the prevalence of culturable bacteria in faecal samples
Enzootic	A disease which is constantly present in an animal population, but which usually only affects a small number of individuals
Enteropathogenic	An entity which is capable of causing disease in the intestinal tract
Fomite	An inanimate object or substance that is capable of transmitting infectious organisms from one individual to another
Innate immunity	Immunity that is naturally present and is not due to prior sensitization to an antigen from, for example, an infection or vaccination. Since it is not stimulated by specific antigens, innate immunity is generally nonspecific
Intensity	The mean number of parasites per infected host
Molecular marker	A DNA sequence or gene that can be used to identify an organism, species, or strain associated with it
Pandemic	A disease that is prevalent throughout an entire country, continent, or the whole world
Prevalence	The proportion of the sample population that tests positive for a bacterial pathogen
Zoonoses	A disease of animals, such as rabies or psittacosis, that can be transmitted to humans

This review summarises our current knowledge of the bacterial pathogens carried by, and known to cause disease in, birds, with particular emphasis on enteropathogenic bacteria. We explore the factors that may affect their susceptibility to disease (summarised in Fig. 1), and investigate the mechanisms by which birds may act as vectors of pathogenic bacteria. We aim to identify gaps in our current knowledge and draw attention to areas that could be rewarding for further research.

II. BACTERIAL PATHOGENS FOUND IN BIRDS

(1) Enteropathogens

The majority of information regarding bacterial enteropathogens in wild birds stems from studies that have applied traditional microbiological techniques (Brittingham, Temple & Duncan, 1988; Kapperud & Rosef, 1983; Waldenström *et al.*, 2002). Quantitative data are lacking on the levels of enteropathogenic bacteria shed by wild birds, and the sparse literature documenting the faecal flora of wild birds has tended to focus on the prevalence of bacterial enteropathogens in a few, well-studied species, notably those most likely to impact upon human health. Table 2 provides an overview of the main bacterial enteropathogens isolated from wild birds. Methodological approaches and limitations are discussed in section VI.

A variety of *Salmonella* species have been found in both apparently healthy and obviously diseased wild birds. *Salmonella enterica* serotype Typhimurium (*S.* Typhimurium) is the serotype most commonly associated with wild birds, and has been found to cause disease in house sparrows *Passer domesticus*, brown-headed cowbirds *Molothrus ater*, white-throated sparrows *Zonotrichia albicollis* (Faddoul *et al.* 1966) and tufted ducks *Aythya fuligula* (Keymer, 1958). *S. pullorum* is known to reduce the probability of egg hatching and chick survival in ring-necked pheasants *Phasianus colchicus* (Pennycott & Duncan, 1999; Sharp & Laing, 1993). Domestic fowl commonly harbour *S. enterica* serovar Enteritidis (*S.* Enteritidis) without it causing discernible illness in the birds, though this bacterium causes food-borne outbreaks of salmonellosis in humans through the consumption of infected chicken eggs (Guard-Petter, 2001).

Clinical signs of birds sick from *Salmonella* infections include lethargy, fluffed-up plumage and a tendency to remain near feeding areas and, although they appear to have difficulties swallowing, they feed until shortly before death (Kirkwood *et al.*, 1995). *Post-mortem* examinations have shown birds to have poor body condition, crops full of undigested food, internal organs displaying lesions and nodules (Kirkwood *et al.*, 1995; Routh & Sleeman, 1995), and sometimes enlarged livers and spleens (Faddoul *et al.*, 1966). Salmonellae in humans can cause enteric fever (typhoid) resulting from bacterial invasion of the bloodstream, and acute gastroenteritis resulting from food-borne infection/intoxication (Finlay & Falkow, 1988).

Klebsiella species appear to be relatively common avian pathogens (Bangert *et al.*, 1988; Fudge, 2001; Hernandez *et al.*, 2003). *Enterobacter* species are well documented in birds but appear not to cause disease outbreaks (da Silva *et al.*, 2004; Glünder, 1989; Shane *et al.*, 1984). *Escherichia coli* has been isolated from a range of bird species, including apparently healthy passerines and waterfowl (Brittingham *et al.*, 1988; Damaré *et al.*, 1979; Foster *et al.*, 1998). However, avian pathogenic *E. coli* is known to cause extra-intestinal diseases in chickens, turkeys and other avian species (Dho-Moulin & Fairbrother, 1999), and *E. coli* producing cytolethal distending toxin have been isolated from dead wild finches (Foster *et al.*, 1998). As well as being a significant cause of human diarrhoea, *E. coli* can cause haemorrhagic colitis, haemolytic uraemic syndrome (Tarr, 1995) and thrombotic thrombocytopenic purpura in infected humans (Doyle, 1991).

Pseudomonas aeruginosa is a common avian pathogen (Brittingham *et al.*, 1988; Walker *et al.*, 2002) which

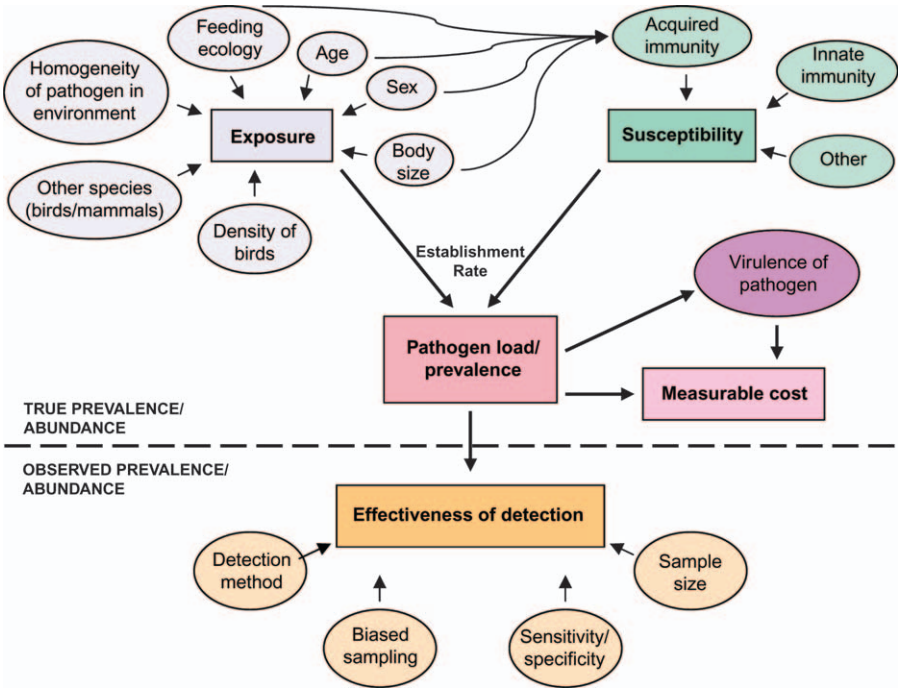

Fig. 1. A diagrammatic overview of the factors likely to influence the true prevalence/abundance of pathogens in birds, and the constraints that may affect observed prevalence/abundance data (see text for details). The true prevalence of pathogens in birds is likely to be affected by both exposure and susceptibility. Exposure to pathogens may be affected by a range of factors, including: life-history and demographic effects (e.g. age, sex, body size, *etc.*), population density, and the homogeneity of pathogens in the environment, and may also be dependent on contact with other species. Both acquired and innate immunity will affect the susceptibility of individuals to pathogens. The measurable fitness cost to the birds will be affected both by the abundance and virulence of the pathogens. The effectiveness of detecting the abundance of pathogens in birds is influenced by various methodological constraints (e.g. detection method; sensitivity, specificity and biases in sampling protocol deployed; sample sizes).

principally affects the upper respiratory tract, causing rhinitis, sinusitis and laryngitis (Bailey *et al.*, 2000; Gerlach, 1994; Momotani *et al.*, 1995). Infections are also associated with septicaemia and haemorrhagic enteritis in psittacines (Rich, 2003), corneal ulcers in captive cranes (Miller *et al.*, 1995) and mass mortality in free-living flamingos (Kock *et al.*, 1999). Different strains of *Streptococcus* and *Enterococcus* species have also been isolated from birds in association with septicemic disease (Devriese *et al.*, 1995, 1992, 1991; Droual *et al.*, 1997; Farrow & Collins, 1985). Staphylococcosis is a relatively common disease of domestic poultry, particularly turkeys, resulting in osteomyelitis, arthritis, tendonitis, and occasionally septicemia (Gross, 1978). Infections with *Staphylococcus aureus* are frequently secondary to impairment of the host defence mechanisms, or are due to a compromised immune system (Wobeser & Kost, 1992). Once inside the host organism, the bacterium can colonize a wide range of organs, and in birds dying from staphylococcosis, necrosis, vascular congestion and inflammation of internal organs have all been reported (Arp, Robinson & Jensen, 1983; Bergmann *et al.*, 1988; Cheville *et al.*, 1988). Wild birds are thought to be a significant reservoir for *Yersinia pseudotuberculosis*, and are also known to harbour *Y. enterocolitica* (Fukushima & Gomyoda, 1991; Fukushima, Gomyoda & Kaneko, 1991; Hubbert, 1972). If pathogenic species of *Yersinia* are common in wild bird populations, avian transmission of these bacteria to humans and other animals is possible (Niskanen *et al.*, 2003).

Various apparently healthy wild birds have been found to contain *Campylobacter fetus* subsp. *jejuni* (*C. jejuni*), suggesting that this organism may be a normal component of the intestinal flora of at least some bird species (Kapperud & Rosef, 1983). However, the presence of *Campylobacter* species appears to be influenced by feeding behaviour and differs considerably amongst ecological guilds of birds: the majority of insectivores and granivores rarely or never test positive for *Campylobacter* species (Waldenström *et al.*, 2002), whereas raptors, scavengers and most ground-foraging guilds, show high rates of carriage. *C. jejuni* alone has been isolated from feral pigeons *Columba livia*, blackbirds *Turdus merula*, starlings *Sturnus vulgaris*, house sparrows, dunlin *Calidris alpina* and various gulls (Smibert, 1969; Skirrow & Benjamin, 1980; Palmgren *et al.*, 1997; Craven *et al.*, 2000). Campylobacters can cause diarrhoea and vomiting in humans (Blaser, 1997; Gillespie *et al.*, 2002), with *C. jejuni* being the most common cause of bacterial gastroenteritis worldwide (Allos, 2005). Birds are ideal hosts for campylobacters, due to their relatively high body temperature (42°C), and human infections are most commonly associated with consumption of undercooked, contaminated poultry meat (Harris, Weiss & Nolan, 1986). The survival of *C. jejuni* in water and on ground surfaces (Blaser *et al.*,

Table 2. Bacterial enteropathogens isolated from waterfowl, gulls, shorebirds, pigeons, passerines and corvids. Sampling methods refers to the method by which bacteria were obtained from the bird. Probable source refers to the putative source of bacterial infection.

Bird species/group	Sampling method	Bacterial species and prevalence (% of birds infected)	Sample size	Location	Probable source	Reference
Anseriformes	Faecal sample	Salmonellae (4.2)	477	UK, London	–	Mitchell & Ridgewell (1971)
Anseriformes	Autopsy	*Campylobacter jejuni* (12.9)	31	USA, Louisiana	–	Yogasundram *et al.* (1989)
Canada goose (*Branta canadensis*) whistling swan (*Cygnus columbianus columbianus*)	Faecal sample	*E. coli*	–	USA	–	Damaré *et al.* (1979)
Migratory waterfowl	–	*C. jejuni* (35)	445	USA, Colorado	–	Luechtefeld *et al.* (1980)
Mute swan (*Cygnus olor*)	Autopsy	Salmonellae (71.9)	32	England, Staffordshire	Sewage	Clegg & Hunt (1975)
Black-headed gull (*Larus ridibundus*)	Cloacal swab	*C. jejuni* (36.2)	367	Sweden, Malmö	Refuse	Broman *et al.* (2002)
Black-headed gull	Cloacal swab	*C. jejuni* (27.9)	419	Sweden, Malmö	Refuse	Broman *et al.* (2002)
Black-headed gull, adults	Faecal sample/ cloacal swab	Salmonellae (4.2)	189	Czech Republic	–	Cižek *et al.* (1994)
Black-headed gull, young	Cloacal swab	Salmonellae (19.2)	740	Czech Republic	–	Cižek *et al.* (1994)
Black-headed gull	Faecal sample	Salmonellae (17.7)	1080	Scotland	Sewage	Fricker (1984)
Black-headed gull	Cloacal swab	Salmonellae (36.8)	171	Czech Republic	Polluted water	Literák *et al.* (1992)
Black-headed gull	Cloacal swab	Salmonellae (20.8)	96	Czech Republic	Polluted water	Literák *et al.* (1992)
Black-headed gull	Cloacal swab	*Salmonella enterica* ser. Typhimurium (4.9)	41	Sweden, Malmö	–	Palmgren *et al.* (1997)
Black-headed gull	Cloacal swab	*C. jejuni* (63) *S.* Typhimurium (51)	41	Czech Republic	–	Sixl *et al.* (1997)
Black-headed gull	Faecal sample	Salmonellae (12.3)	196	Germany	–	Wuthe (1973)
Black-headed gull Herring gull (*L. argentatus*) Common gull (*L. canus*)	Faecal sample/ cloacal swab	Campylobacters (62) Salmonellae (11)	207	Germany	–	Glünder *et al.* (1991)
Charidrii	Cloacal swabs/ cloacal lavage	Campylobacters (71.38)	311	Scotland	–	Fricker & Metcalfe (1984)
Gull (*Larus*) species	Faecal sample	Salmonellae (26.7)	60	Germany	Refuse	Edel *et al.* (1976)
Gull (*Larus*) species	Faecal sample	Salmonellae (23.7)	114	Germany	Refuse	Edel *et al.* (1978)
Gull (*Larus*) species	Faecal sample	Salmonellae (12.9)	1242	Scotland, Aberdeenshire	Sewage	Fenlon (1981)
Gull (*Larus*) species	Faecal sample	Salmonellae (55)	20	Scotland Aberdeenshire	Sewage	Fenlon (1983)
Gull (*Larus*) species	Faecal sample	Listerias (14.5)	275	Scotland, Aberdeenshire	Sewage	Fenlon (1985)
Gull (*Larus*) species	Cloacal swab	Salmonellae (10.7)	560	Scotland	–	Fricker *et al.* (1983)
Gull (*Larus*) species	Cloacal lavage	Salmonellae (7.8)	5888	Scotland	Refuse	Girdwood *et al.* (1985)
Gull (*Larus*) species	Cloacal swab	Campylobacters (23.3)	180	Norway	–	Kapperud & Rosef (1983)

Table 2. (*cont.*)

Bird species/group	Sampling method	Bacterial species and prevalence (% of birds infected)	Sample size	Location	Probable source	Reference
Gull (*Larus*) species	Autopsy	*S.* Typhimurium (6)	83	Scotland	–	Macdonald & Brown (1974)
Gull (*Larus*) species	Faecal sample	Campylobacters (13.7)	205	N. Ireland		Moore *et al.* (2002)
Gull, species unspecified	Autopsy	Campylobacters (33) Salmonellae (5.8)	103	Norway	Refuse	Willumsen & Hole (1987)
Herring gull	Cloacal swab	Salmonellae (17)	154	Scotland	–	Benton *et al.* (1983)
Herring gull	Cloacal swab	Salmonellae (2.1–8.4)	2786	England	–	Butterfield *et al.* (1983)
Herring gull	Faecal sample/ autopsy/ cloacal lavage	Salmonellae (9.6)	2021	Scotland, Clyde area	Refuse	Monaghan *et al.* (1985)
Herring gull	Faecal sample	Salmonellae (22.2)	514	Wales	Refuse	Williams *et al.* (1976)
Ring-billed gull (*L. delawarensis*)	Cloacal swab	Salmonellae (13) Campylobacters (4.3) *Listeria monocytogenes* (4.4)	264	Canada, Montreal	Refuse	Quessy & Messier (1992)
Scolopacidae, Charidriidae	Cloacal swab	Campylobacters (79.6)	382	Sweden	–	Waldenström *et al.* (2002)
Shorebirds (gulls and lapwing), corvids	Faecal sample	Vero cytotoxin-producing *Escherichia. coli* 0157 (1.9)	691	England	Refuse/ shoreline	Wallace *et al.* (1997)
Pigeon (*Columba livia*)	Crop sac samples	*Lactobacillus agilis* (80)	10	Belgium	–	Baele *et al.* (2001)
Pigeon	Cloacal swab	Campylobacters (26.2) Salmonellae (1.5)Yersiniae (0.2) Listeriae (0.2)	400	Spain, Barcelona	–	Casanovas *et al.* (1995)
Pigeon	Autopsy	*S.* Typhimurium (25.3)	178	USA, Massachusetts	–	Faddoul & Fellows (1965)
Pigeon	Cloacal swab	Campylobacters (4.2)	71	Norway	–	Kapperud & Rosef (1983)
Pigeon	Autopsy	Vibrios (20)	5	USA	–	Smibert (1969)
Columbiformes	Autopsy	*C. jejuni* (8.3)	12	USA, Louisiana	–	Yogasundram *et al.* (1989)
Passerines and woodpeckers	Cloacal swab	*E. coli* (1) *Pseudomonas* spp. (22) *Staphylococcus* spp. (15) *Streptococcus* spp. (18) *Yersinia* spp. (1)	387	USA	–	Brittingham *et al.* (1988)
Greenfinch (*Carduelis chloris*), house sparrow (*Passer domesticus*)	Autopsy of diseased, dead birds	*S.* Typhimurium (100)	28	England, Surrey	–	Cornelius (1969)
Finches	Autopsy	*E. coli* 086:K61 (93.5)	46	Scotland, Highlands	–	Foster *et al.* (1998)
Starling, house sparrow	Faecal sample/ cloacal swab	*C. jejuni* (10)	34	USA, Georgia	–	Craven *et al.* (2000)
Blackbird (*Turdus merula*), starling (*Sturnus vulgaris*)	Faecal sample	*C. jejuni* (3)	101	Sweden, Malmö	–	Palmgren *et al.* (1997)
Fringillidae	Autopsy	Salmonellae/ *E. coli* 086 (91.3)	103	Scotland (feeding station)	–	Pennycott (1998)
Brown-headed cowbird (*Molothrus ater*) house sparrow (*Parus domesticus*)	Necropsy/ bacteriological examination	*S.* Typhimurium (30)	187	USA, Massachusetts/ Rhode Island	–	Faddoul *et al.* (1966)

Assorted passerines	Pooled faecal sample	Salmonellae (45)	151	Scotland, S-W (feeding station)	–	Pennycott *et al.* (2002)
Assorted wild birds, mostly passerines	Faecal sample	Salmonellae (0.17)	599	England	–	Plant (1978)
Sparrow, blackbird, starling	Autopsy	Vibrios (52)	25	USA	–	Smibert (1969)
Various passerines	Cloacal swab, autopsy	Salmonellae (4.7)	277	England	–	Goodchild & Tucker (1968)
Carrion crow, blackbird, robin *Erithacus rubecula* starling, tufted duck	Necropsy	*Pasteurella aviseptica* (1)	513	UK	–	Keymer (1958)
Crow (*Corvus corone*)	Autopsy	Campylobacters (83.3) Salmonellae (8.3)	12	Norway	–	Willumsen & Hole (1987)
Hooded crow (*Corvus cornix*)	Cloacal swab	Campylobacters (89.8)	48	Norway	–	Kapperud & Rosef (1983)
Hooded crow	Cloacal swab	*C. jejuni* (89.6)	48	Norway, Oslo	–	Olsvik & Berdal (1983)
Rook (*Corvus frugilegus*)	Faecal sample	Listeria (46)	112	France	–	Bouttefroy *et al.* (1997)
Rook	Faecal sample/autopsy	Listeria (13)	123	Scotland	–	Fenlon (1985)
Raptors	Faecal sample	*Klebsiella* spp. (42.6)	47	USA, Washington State	–	Bangert *et al.* (1988)
Psittaciformes	Necropsy	*Enterococcus hirae* (8.7)	276	Belgium	–	Devriese *et al.* (1995)

1980) suggests that gulls and pigeons could be a possible source of human infection, especially amongst children, who are more suseptible to contact with these birds and/or their faeces through play in public open spaces (Hatch, 1996; Casanovas *et al.*, 1995).

Pasteurella aviseptica has been isolated from carrion crows *Corvus corone*, blackbirds, robins *Erithacus rubecula*, starlings and tufted ducks (Keymer, 1958), and *P. multocida* from various raptors (Morishita *et al.*, 1996). *P. multocida*, which causes fowl cholera, is known to be lethal to game birds such as ring-necked pheasants, partridges *Perdix perdix* and red grouse *Lagopus lagopus scoticus* (Jennings, 1954, 1955). When outbreaks of fowl cholera occur in wild game bird populations, high mortality ensues as a result of the birds' relatively gregarious nature and tendency to occur at high densities (Botzler, 1991) and, although fowl cholera is not usually a threat to human health, outbreaks may have severe economic implications for the game industry.

Listeria monocytogenes is ubiquitous in the environment (Beuchat, 1996; Fenlon, 1985) and grows at refrigeration temperature (approximately 4°C). It can therefore survive and multiply outside a host species, facilitating uptake by further hosts or vectors, such as wild birds (Fenlon, 1985). Listeriosis can cause gross abnormalities and histological lesions in the liver, heart, spleen and kidneys, as well as toe paralysis in newly hatched chicks (Basher *et al.*, 1984). Humans commonly ingest *Listeria* species through the consumption of raw and unprocessed food products. Listeriosis causes muscle ache, neck stiffness and convulsions, and can result in gastroenteritis, miscarriage (Altekruse, Cohen & Swerdlow, 1997), sepsis in immunocompromised patients, or meningitis in infants and patients with chronic diseases (Schlech, 2000).

Clostridium perfringens is another ubiquitous bacterium which is commonly associated with poultry houses and their surroundings (Craven *et al.*, 2001). It is often found in the intestinal tracts of healthy birds, and is the causative agent for outbreaks of both acute clinical disease and subclinical disease in broiler and turkey flocks (Engström *et al.*, 2003). The detection of *C. perfringens* in faeces from wild birds near broiler chicken houses suggests that wild birds that gain entry to poultry houses have the potential to transmit the pathogen to poultry (Craven *et al.*, 2000). *C. perfringens* is of concern to human health, as it can cause food borne disease by transmission through poultry products (Engström *et al.*, 2003). It is beyond the scope of this review to fully detail the symptoms and diseases resulting from bacterial infections that both birds and humans are susceptible to, though it is pertinent to note that both direct and indirect contact between birds and humans may have important implications for human health.

(2) Non-enteropathogens

(*a*) *Avian psittacosis*

Psittacosis, or avian chlamydiosis, is a zoonotic illness caused by *Chlamydia psittaci*; originally thought to occur only in psittacine birds, it is now known to affect a wide range of both avian and mammalian species, including humans

(Vanrompay *et al.*, 1995). Individual bird species may be infected by *C. psittaci* strains that differ in virulence; symptoms include respiratory infection, diarrhoea, polyuria and conjunctivitis (Vanrompay, Ducatelle & Haesebrouck, 1995). In poultry, avian chlamydiosis can either be asymptomatic, or may manifest itself as a disease of high morbidity and mortality, which can be economically devastating to poultry producers (Andersen & Vanrompay, 2000). Once within a flock, *C. psittaci* is primarily spread between birds by inhalation of desiccated droppings and secretions, both ocular and nasal, from infected birds, or through ingestion of contaminated faeces (Page, 1959; Takahashi, Takashima & Hashimoto, 1988). The infection may be transmitted to fledglings in the nest by parent birds that are shedding the organism (Burnet, 1935) and there is evidence of transmission through eggs (Vanrompay *et al.*, 1995). Additionally, blood-sucking ectoparasites have been shown to transfer the bacterium between birds, probably as mechanical rather than biological vectors (Shewen, 1980). The mechanism for introduction of avian chlamydiosis to poultry flocks is poorly understood, though wild birds are thought to play a major role in disease transmission (Andersen & Vanrompay, 2000). Free-living wild birds are important as reservoirs of *C. psittaci* (Brand, 1989), evidence of exposure to chlamydiae most frequently being reported in Charadriiformes, Passeriformes, and Anseriformes (Brand, 1989; Franson & Pearson, 1995). Both diseased birds and sub-clinically infected birds can shed chlamydiae and are therefore a potential threat to both human and animal health (Brand, 1989; Franson & Pearson, 1995; Roberts & Grimes, 1978; Wobeser & Brand, 1982). Since the same strains occur in both wild birds and domestic poultry flocks, wild birds may be a potential source of infection and should therefore be prevented from coming into contact with poultry (Grimes, 1978; Page, 1976).

The importance of poultry as a source of infection for humans became evident in the 1950s, when outbreaks occurred in humans due to contact with infected birds (Graber & Pomeroy, 1958; McCulloh, 1955; Meyer & Eddie, 1953), though most infections in humans are due to exposure to psittacine birds and pigeons (Andersen & Vanrompay, 2000). Psittacosis in humans is typically transmitted through inhalation of aerosolized bird excreta (Moroney *et al.*, 1998), and those most prone to the disease are usually owners of pet birds, or those exposed to birds by occupation, including pet shop employees, aviary workers, veterinarians, employees in poultry slaughtering and processing plants, farmers and zoo workers (Longbottom & Coulter, 2003).

(*b*) Mycoplasma gallisepticum

The bacterium *Mycoplasma gallisepticum* is frequently associated with respiratory tract disease, debilitation and carcass condemnation, as well as reduced egg production in domestic poultry (Bradbury, 2001; Jordan, Pattison & Alexander, 2001; Mohammed, Carpenter & Yamamoto, 1987). Historically, *M. gallisepticum* has not been considered a naturally occurring pathogen of wild birds, though incidents have been reported to occur in various species, such as wild turkeys *Meleagris gallopavo*, and captive reared ring-necked pheasants, chukar partridges *Alectoris chukar* and peafowl *Pavo cristatus* (Cookson & Shivaprasad, 1994; Fritz, Thomas & Yuill 1992). Serological surveys and experimental infections have suggested that house sparrows may act as transient carriers of the bacterium (Kleven & Fletcher, 1983).

An epidemic of conjunctivitis in eastern house finches *Carpodacus mexicanus* occurred in suburban Washington DC, USA, in 1994, since when the disease has become widespread throughout the eastern USA and Canada (Fischer *et al.*, 1997). Diagnostic testing confirmed the symptoms to be caused by a new strain of the non-zoonotic poultry pathogen *M. gallisepticum* (Ley, Berkhoff & McLaren, 1996; Luttrell *et al.*, 1996). By the end of 1995, the disease had spread, not only geographically, but also to other species such as the American goldfinch *Carduelis tristis*, purple finches *C. purpureus*, evening grosbeaks *Coccothraustes vespertinus* and pine grosbeaks *Pinicola enucleator* (Faustino *et al.*, 2004; Fischer *et al.*, 1997). The reason that house finches first became infected with *M. gallisepticum* remains unknown (Dhondt, Tessaglia & Slothower, 1998), as do the modes of transmission, though it is suspected that social and foraging behaviour at bird feeders and other sites of abundant food may facilitate transmission through direct contact (Dhondt *et al.*, 1998; Hartup, Mohammed, Kollias *et al.*, 1998). *M. gallisepticum* continues to spread across the United States and has been confirmed in the native western range of house finches (Faustino *et al.*, 2004). If feeders do play a significant role in the transmission of the disease, either as a fomite or by acting as a focal point for diseased birds unable to successfully secure natural food sources, appropriate strategies that modify bird feeding activities may help to decrease the spread of mycoplasmal conjunctivitis in wild populations (Hartup *et al.*, 1998).

(*c*) *Avian botulism*

Avian botulism is a neuroparalytic, often fatal, disease of birds that results from the ingestion of toxin produced by the bacterium *Clostridium botulinum*. There are seven distinct types of toxin, designated A-G (Hauschild & Dodds, 1993), of which almost all birds are susceptible to type C botulism, though waterfowl and shorebirds are most notably affected (Borland, Moryson & Smith, 1977; Brand *et al.*, 1988; Ortiz & Smith, 1994). Widespread outbreaks have resulted from birds eating toxin-laden maggots that have been feeding on the carcasses of other birds dead from botulism (Hauschild & Dodds, 1993). Among waterbirds, such as gulls, loons and grebes, outbreaks have been caused by type E toxin, probably as a result of ingestion of toxic fish (Hauschild & Dodds, 1993). Spores of *C. botulinum* are common in marsh soil and can persist there for years (Smith, Oliphant & White, 1982), and animals living in marsh areas ingest spores frequently (Reed & Rocke, 1992). Decaying animal material provides a suitable substrate for *C. botulinum* growth (Bell, Sciple & Hubert, 1955), vertebrate carcasses being of particular importance (Smith & Turner, 1987). When an animal containing *C. botulinum* spores dies, putrefaction, invasion of tissues by *C. botulinum* from the gut, and associated toxin production occurs (Notermans, Dufrenne &

Kovacki, 1980; Smith & Turner, 1987). The larvae of sarcophagus flies *Sarcophaga* spp., feeding on the carcasses, are not affected by the toxins and effectively act to concentrate the toxin (Duncan & Jensen, 1976). Birds that ingest these maggots may die of intoxication and their carcasses become substrates for the generation of further toxins and more maggots, thus perpetuating the cycle. Despite its microbiology being well understood, management of the disease still primarily consists of carcass collection during epizootics (Wobeser, 1997) rather than any form of preventative management.

(*d*) *Tick-borne bacterial pathogens*

Various human pathogenic microorganisms have been detected in ticks collected from migratory birds, including the causative agents of Lyme disease, rickettsiosis and human granulocytic ehrlichiosis (Alekseev *et al.*, 2001; Bjöersdorff *et al.*, 2001; Parola & Raoult, 2001). Rickettsioses are infectious diseases, and the implication of birds in their dissemination through tick dispersal seems highly likely (Fournier, Gouriet, Brouqui *et al.*, 2005; Fournier, Tissot-Dupont & Gallais, 2000). The involvement of birds in the ecology and epidemiology of ehrlichiosis, however, has yet to be established (Parola & Raoult, 2001).

Lyme disease is caused by three different species of spirochetes in the *Borrelia burgdorferi sensu lato* genogroup, and is transmitted to humans and other animals *via* Ixodid ticks (Barbour, 1998). Numerous studies have investigated the possible roles of wild birds in the perpetuation of enzootic cycles of the disease and the expansion of endemic ranges (Anderson *et al.*, 1986; Kurtenbach *et al.*, 1998; Olsen, Jaenson & Bergström, 1995). Of the tick species known to parasitize wild birds, *Ixodes* species are the most likely to carry *B. burgdorferi* in Europe and North America, and commonly infest a wide range of bird species (Anderson *et al.*, 1986; Anderson, Magnarelli & Stafford, 1990; Olsen *et al.*, 1995; Smith *et al.*, 1996).

The ticks attach themselves to their host for 24-48 h whilst acquiring a blood meal (Tsiodrasa *et al.*, 2008). This allows ample time for migrating birds to travel hundreds or even thousands of miles before the ticks finish feeding and drop off (Reed *et al.*, 2003), thereby depositing the infectious tick in a new geographical area. There is evidence of transhemispheric exchange of spirochete-infected ticks by seabirds, indicating the capacity for wild birds to carry infected ticks over long distances (Olsen *et al.*, 1995). Even if the relative ectoparasite load is small, the number of birds transporting tick vectors could contribute substantially to local tick populations (Ginsberg *et al.*, 2005), thereby affecting disease dynamics. Birds can also carry infections in their bloodstream, which can then be introduced to local populations of ticks at other sites (Humair, 2002; Richter *et al.*, 2000). Birds appear to play an important role not only in maintaining *B. burgdorferi* in areas where the pathogen is already established, but also by spreading the disease agent through migration, by spreading ticks both within and between continents (Ishiguro *et al.*, 2000; Scott *et al.*, 2001; Smith *et al.*, 1996).

(3) Drug-resistant bacteria in wild birds

Antibiotics are used in animals to control bacterial infections, with the result that resistance in both pathogenic bacteria and the endogenous flora of exposed individuals or populations occurs (Hinton, Al Chalaby & Allen, 1982; Howe, Linton & Osborne, 1976; Van den Bogaard, 1997). The use of antibiotics is deemed to be the most important factor promoting the emergence, selection and dissemination of antibiotic-resistant microorganisms in both veterinary and human medicine (Neu, 1992; Witte, 1998). In animals such as broilers and turkeys, antimicrobial agents are often continuously supplied as antimicrobial growth promoters, and this has resulted in increased antibiotic selection pressure for resistant bacteria, resulting in their faecal flora containing a relatively high proportion of resistant bacteria (Van den Bogaard & Stobberingh, 1999). Antimicrobial drug resistance is relatively commonplace in poultry, but has also been described in bacteria isolated from wild birds (Cole *et al.*, 2005; Middleton & Ambrose, 2005; Sjölund *et al.*, 2008). Arctic birds are known to contain multi-drug-resistant bacteria, indicating that migration behaviour may be responsible for the introduction and transfer of drug-resistant bacteria to geographically remote areas (Sjölund *et al.*, 2008). Canada geese *Branta canadensis* containing antibiotic-resistant *E. coli* use farmland for grazing, creating the opportunity for transfer of drug-resistant bacteria to cattle and other livestock (Cole *et al.*, 2005). Although wild animals do not naturally come into contact with antibiotics, they can become infected with resistant bacteria disseminated by wild birds, and act as reservoirs and vectors of resistant bacterial pathogens, encouraging new health problems in wildlife populations to emerge, as well as novel reservoirs of zoonotic disease to form (Cole *et al.*, 2005; Hudson *et al.*, 2000; Sayah *et al.*, 2005).

III. SUSCEPTIBILITY OF BIRDS TO BACTERIAL INFECTION

Birds are vulnerable to pathogenic infection at all stages of their life cycle, both before and after hatching. Although eggs present physical and chemical barriers that protect against microbial invasion (Board, 1966; Board & Fuller, 1974), bacteria can penetrate the eggshell and infect the contents (Cook *et al.*, 2003). Once hatched, nestlings are inoculated by microorganisms from the environment *via* food provided by parents, ingestion of adult saliva and from nest materials (Berger, Disko & Gwinner, 2003; Kyle & Kyle, 1993; Mills, Lombardo & Thorpe, 1999; Singleton & Harper, 1998). During the breeding season, almost any bacterial pathogen present in the gut may become sexually transmitted through the cloacal passage (Reiber, McInroy & Conner, 1995; Sheldon, 1993), which functions as a channel for both gamete transfer and excretion. Birds congregating at high-density communal roosts are potentially vulnerable to the spread of disease, both through direct contact and through the contamination of food and water sources by

diseased individuals (Rappole & Hubálek, 2006). Apparently healthy birds, carrying low numbers of potential pathogens, in addition to symptomatically infected birds, may act as a source of disease. In addition, heterogeneities within the host population, including age, sex and body size, may affect the susceptibility of some bird species to bacterial disease (Fig. 1).

Most disease outbreaks are recorded in winter (Faddoul *et al.*, 1966; Hurvell, 1973; Refsum *et al.*, 2002), possibly as a direct result of lowered immune function due to harsh weather conditions (Nelson & Demas, 1996). As local feeding densities increase, interactions between individual birds at food patches intensify, and stress associated with increased competition over reduced quantity and quality of natural food may occur. Feeding densities have been shown to influence aggressive interaction rates in both wild and domestic birds, and increased densities are often associated with higher levels of aggression (e.g. Metcalfe, 1989, but see Dawkins, Donnelly & Jones, 2003). Higher densities can also induce greater stress in birds (e.g. Nephew & Romero, 2003; Dickens, Nephew & Romero, 2006), which could result in reduced immune function by increasing corticosteroid levels (e.g. Saino *et al.*, 2003). Population turnover might further impact on density effects if dominance ranks need to be continually established or are absent (Banks, 1984; Cristal, 1995). The relationship between stress and population density in regard to bacterial disease epidemiology remains unclear. Further research is required to elucidate the relationships between density, individual interactions and susceptibility to disease.

(1) Sex differences

In a wide range of animal taxa, including birds, males and females differ in their physiology and behaviour and, consequently, they frequently differ in their exposure and susceptibility to pathogens (e.g. Poulin, 1996; Schalk & Forbes, 1997; Wedekind & Jakobsen, 1998; Moore & Wilson, 2002; Arnold *et al.*, 2003; Tschirren, Fitze & Richner, 2003; Wilson, Moore & Owens, 2003; Ferrari *et al.*, 2004; Seeman & Nahrung, 2004; Robb & Forbes, 2006). During the breeding season, for example, males and females of most, if not all, bird species are differentially exposed to bacterial pathogens, and suffer diverse pressures that may affect their susceptibility to infection (Fig.1). In the majority of bird species, males lack an intromittent copulatory organ (Briskie & Montgomerie, 1997), and sperm transfer occurs through very brief cloacal contact (Sheldon, 1993). Given the brevity of copulation, males should have a relatively small chance of contracting bacterial pathogens from contact with the female cloaca. Females, however, have prolonged exposure to the ejaculate once it enters the reproductive tract, and the ejaculate may serve to infect females with any pathogenic gastrointestinal bacteria it may have become contaminated with on passing through the male cloaca (Sheldon, 1993). Cloacal transmission of microbes during copulation has been documented in domestic fowl (Perek, Elian & Heller, 1969) and red-winged blackbirds *Agelaius phoeniceus* (Westneat & Rambo, 2000), and is also suggested to occur in tree swallows *Tachycineta bicolor* (Lombardo *et al.*, 1996). Such sex-specific asymmetry of exposure to bacterial transmission through copulation should render males less susceptible than females to sexually transmitted bacterial infections during the breeding season. In polygamous mating systems, individuals with more mating partners are expected to contract more infections (Thrall, Antonovics & Dobson, 2000); this applies both to females in polyandrous relationships, and to males in polygynous ones.

As well as differences in exposure, the sexes also differ in their physiological response to pathogens. Males commonly experience raised levels of the hormone testosterone during the breeding season (Wingfield *et al.*, 1990), which may increase their susceptibility to infection (Folstad & Karter, 1992; Grossman, 1989), though the exact immunosuppressive role of testosterone in birds is far from clear (Roberts, Buchanan & Evans, 2004). Though it has been demonstrated that male birds and mammals have significantly higher parasite prevalence than females overall (Poulin, 1996; Schalk & Forbes, 1997; Moore & Wilson 2002), analysis of data by parasite taxon showed that although male birds had higher parasite prevalence (percentage of hosts that are infected), female birds displayed a higher intensity (mean number of parasites per infected host) than males for certain parasites (Poulin, 1996). Interestingly, the sex bias in the ability to cope with infection was stronger in experimental studies than in field studies (Schalk & Forbes, 1997), suggesting that differences may lie in the host immune response rather than the infection process itself.

Not only is there potential sex-biased susceptibility to infection during the breeding season, but other factors may contribute throughout the life cycle. Competition between the sexes over food resources may lead to differences in exposure to food-borne bacteria. It has been observed that *Salmonella* spp. carriage by female herring gulls *Larus argentatus* (22% of 1433 birds examined) is more than double that of males (10%) in the non-breeding season, which may reflect differences in feeding ecology between the two sexes (Monaghan *et al.*, 1985). Male herring gulls tend to dominate food resources at refuse tips and, when competition is severe (e.g. in winter, or when the weather is harsh during the breeding season), males monopolise freshly dumped material, leaving the females to feed on older, more putrid food (Kihlman & Larsson, 1974; Monaghan, 1980; Spaans, 1971). During the summer, male herring gulls tend to make use of other food sources, for example those presented by the fishing industry, so females are more abundant on refuse tips and encounter less competition for resources (Monaghan *et al.*, 1985). In competitive feeding situations, individuals at the lower end of the pecking order are thus perhaps more likely to ingest infectious material than are the more dominant individuals, and these individuals may be more likely to be female. Alternatively, the proximate cause of these sex-specific patterns may stem from copulation behaviour during the breeding season. Further work is needed to quantify the sex differences in bacterial infections, and to identify the mechanisms underpinning those differences.

(2) Body size

Body size may influence susceptibility to pathogens if bacterial acquisition occurs predominantly through foraging, as larger individuals should eat more, and hence experience increased exposure to infected food (Arneberg, Skorping & Read, 1998; Moore & Wilson, 2002; Zuk & McKean, 1996). In mammals displaying sexual size dimorphism, there is good evidence for a positive relationship between host size and parasite load (Arneberg *et al.*, 1998; Moore & Wilson, 2002), and it might be reasonable to expect a similar pattern in birds. However, a comparative analysis of over 30 studies of blood parasitism rates in birds concluded that there was no evidence for a sexual size dimorphism effect on the prevalence or intensity of infections (McCurdy *et al.*, 1998). To our knowledge, there is a distinct lack of data regarding within-species correlations between body size and bacterial pathogen susceptibility in birds, which may be an area worth exploring.

(3) Age

Variation in age-related infection may be influenced by a range of factors, including parasite-induced host mortality, acquired immunity, age-related exposure and age-related predisposition to infection (Wilson *et al.*, 2002). Determining accurate age-related infection patterns in wild populations is difficult and they may also vary geographically (Gregory, 1992; Quinnel, Grafen & Woolhouse, 1995). Established age-related infection patterns come primarily from studies of humans, domesticated ruminants and laboratory animals (Anderson & May, 1992; Anderson & May, 1985; Crombie & Anderson, 1985); there remains a need for similar studies to characterise such patterns in birds, especially as age effects might interact with life-history factors that underpin population changes. Studies to date suggest that avian infection levels may vary amongst age groups (Butterfield *et al.*, 1983; Cicho, Sendecka & Gustafsson, 2003; Čižek *et al.*, 1994; Glünder *et al.*, 1991; Literák, Čižek & Honza, 1992; MacDonald & Brown, 1974; Sixl *et al.*, 1997), though current data are not available from rigorous longitudinal studies.

Accurately assessing the age of birds can be extremely difficult once adult plumage is attained, and little is known about the distribution of pathogenic prevalence across avian age groups. Although a few studies indicate that young or immature birds display higher bacterial carriage rates than mature birds (Butterfield *et al.*, 1983; Čižek *et al.*, 1994; Glünder *et al.*, 1991; Literák *et al.*, 1992; MacDonald & Brown, 1974; Sixl *et al.*, 1997), there are several potential explanations for the apparent susceptibility of younger birds to bacterial infection. Nestlings of species that breed in colonies (e.g. gulls) occur at high population density and, consequently, may have an increased likelihood of disease transmission until they fledge. Acquired immunity should act to reduce susceptibility to infection in older individuals, as populations subject to high rates of pathogen transmission should have higher peak levels of infection occurring in younger age classes (Scott & Smith, 1994). Few studies have clearly demonstrated acquired immunity in wildlife populations (Wilson *et al.*, 2002); rigorous longitudinal studies would be required to demonstrate that susceptibility of wild birds to pathogens is influenced by acquired immunity (Beal *et al.*, 2004). Colonisation of nestlings by environmental microbes begins soon after hatching (Lucas & Heeb, 2005) and bacterial diversity increases with nestling age (Mills *et al.*, 1999). Over time, faecal matter from nestlings will build up in the nests of some species, increasing exposure levels of nestlings to faecal bacteria. Adults and fledged young ought, therefore, to have lower exposure levels, as they are not reduced to spending all their time in what may be relatively highly contaminated areas. However, to our knowledge, no studies have yet tested this hypothesis.

It is evident that gulls pick up pathogenic bacteria from sewage and human refuse, though they appear, as adults, to have low susceptibility to infection from many of the pathogens in question. Immature gulls tend to feed at coastal, untreated sewage outfalls more often than adults and are thus exposed to higher levels of enteropathogens (Monaghan, 1980). Higher carriage rates would therefore be expected in younger individuals than in adults, although there is currently little evidence to support this idea. Despite older birds remaining apparently healthy on ingesting pathogenic bacteria, they may experience breeding losses by harbouring infectious organisms and transferring them from feathers and/or cloacae to the eggshell, which can then kill the embryo upon penetrating and multiplying inside the egg (Steiniger, 1970). Infected parent birds may also pass bacterial pathogens on to their nestlings through the food-provisioning process. Immunity developed in response to accumulated exposure to infections (Wilson *et al.*, 2002) is likely to affect susceptibility to pathogens. Young birds may be more susceptible to disease due to the lack of such acquired immunity. However, it is possible that the pathogen load is sufficiently low for an individual to remain apparently healthy without necessarily having acquired immunity (for a more detailed review of avian immune defence and life-history trade-offs, see Norris & Evans, 2000). Detection of a pathogen in an apparently healthy individual is not, in itself, sufficient to determine whether acquired immunity is preventing the individual from becoming ill. Complementing detection of pathogens with testing for antibodies would give a clearer picture of how age and acquired immunity affect susceptibility to pathogenic bacteria.

IV. EXPOSURE OF BIRDS TO ENTERIC BACTERIA

Feeding ecology appears to be the main factor influencing exposure of many wild bird species to enteric bacteria, and involves infection from disparate sources such as garden feeders, sewage, rubbish tips, carrion, drinking water and feed contaminated by the faeces of other animals (Cornelius, 1969; Fenlon, 1983; Williams *et al.*, 1976). For example, raptors are exposed to high levels of enteric bacteria and other potential pathogens from the intestines of the prey

they ingest, while species that scavenge on carrion have the added risk of contracting bacterial infection from carcasses, which may be an inherently rich source of pathogens. Waterfowl which feed solely on vegetable matter appear to have low enteropathogen prevalence, while enteropathogenic bacteria are frequently found in waterfowl that feed on animals or strain mud to obtain nutrition (Luechtefeld *et al.*, 1980). Ground-foraging species may ingest contaminated food in a variety of ways, for example, from food contaminated by bird droppings under feeders, or from eating filter-feeding molluscs living in sewage-contaminated habitats.

Gulls are notorious scavengers of sources prolific in bacteria, and studies have tended to concentrate on detecting and comparing types of bacteria found in both birds and their food. Often, distinct groups of bacteria have been targeted, resulting in a sound understanding of the correlation between recently consumed food and consumer prevalence of bacteria, but information on inherent bacterial presence in the birds remains lacking. This applies not only to studies of gulls, but also to those of other groups of birds that primarily scavenge on human waste. Many studies focus on birds foraging in environments likely to have high densities of pathogens, and it is important to bear in mind that variation in the spatial distribution of infective organisms will affect exposure rates of hosts to infection. If infectious agents are distributed unevenly in the environment, and hosts vary in their use of the environment, heterogeneities in host exposure rates are a likely result (Wilson *et al.*, 2002). An equally important factor to consider when studying exposure to bacterial pathogens is the virulence of the infective organism, since pathogens differ in their capacity to cause disease once ingested. Organisms with low virulence require the ingestion of high numbers for an infection to become established, whereas highly virulent organisms require much lower numbers (10-100 cells) to infect a host (Blaser & Newman, 1982; Tuttle *et al.*, 1999).

(1) Feeding stations

A variety of bacterial diseases are associated with passerine mortalities, including listeriosis, conjunctivitis, salmonellosis and staphylococcosis. Finch species and house sparrows are most frequently recorded with these infections (Fischer *et al.*, 1997), although a range of other species may also be affected (Borg, 1985; Prescott *et al.*, 1998). Passerine species suffering from the same bacterial diseases often share common symptoms, and *post-mortem* examinations can determine the causal agent. Sick birds habitually continue feeding until the time of death, and outbreaks of disease are primarily detected at feeding stations when carcasses start appearing. Consequently, garden-feeding passerines have been the focus of studies investigating the relationships amongst infectivity, feeding behaviour and population density (Brittingham & Temple, 1986; Hurvell, 1973; Kirkwood, 1998).

When disease outbreaks and mortalities occur at feeding stations, there is a limit to the extent to which the diseased birds can contaminate their surroundings. Once dead, the birds will not actively spread the causative organism, so any further direct infection will only be transmitted to scavenging animals. Healthy individuals that feed from the same food source may, however, be at risk of ingesting contaminated food, spreading the causative agent to others, and potentially dying from the pathogen themselves, though conclusive evidence for this is lacking. When large amounts of food are made available at feeding stations, a build-up of pathogens can occur around the feeders (MacDonald, Everett & Maule, 1968; Pennycott, 1998), and animals other than birds may also be attracted by the abundance of food. Rats and mice, both of which are recognised vectors of salmonellae and *E. coli* (Guard-Petter *et al.*, 1997; Henzler & Opitz, 1992; Hilton, Willis & Hickie, 2002), may contaminate the ground around feeding areas with infected faeces, which could affect ground-foraging birds. Although incidents of mice acting as carriers of salmonellae in poultry houses are well documented (Davies & Wray, 1995, 1996; Guard-Petter *et al.*, 1997), there appears to be little or no work investigating bacterial transmission from rodents to wild birds and, whilst logistically challenging, this may be an avenue worth exploring.

Most pathogens are assumed to be transmitted in a density-dependent manner (Anderson & May, 1992), such that as the degree of crowding increases, so too does the probability of pathogen transmission between infected and susceptible hosts (Fig. 1). Thus, the high concentration of birds at feeding stations could potentially increase their exposure to infection; birds carrying pathogens may contaminate both food and feeding surfaces, thereby facilitating the spread of disease to otherwise healthy individuals. Numerous studies have found an association between the intensity of provisioning at feeding sites and mortality in wild birds feeding at bird tables, caused by *S.* Typhimurium and *E. coli* (Cornelius, 1969; Kirkwood, 1998; MacDonald & Cornelius, 1969; Pennycott, 1998; Pennycott *et al.*, 2002; Wilson & MacDonald, 1967). Although the extent of these studies is somewhat limited, they do suggest that disease outbreaks are, in part, related to the intensity of provisioning by humans and the density of birds at feeding sites. It is possible, however, that multiple deaths occur in populations of non-provisioned birds, but that incidents are not recognised because the populations are not concentrated in areas where people are likely to find and report sick or dead birds. Lack of information on disease prevalence in non-provisioned populations makes it difficult to assess accurately the correlation between disease incidence and intensity of feeding.

(2) Sewage

Gulls (*Larus* spp.) are drawn to sewage outfalls as a source of food (Raven & Coulson, 2001), and have regularly been associated with the carriage of salmonellae (Edel, van Schothorst & Kampelmacher, 1976; Edel *et al.*, 1978; Fenlon, 1981; Fricker & Metcalfe, 1984; Williams *et al.*, 1976; Table 2). Studies investigating the range of *Salmonella* serotypes in faeces from gulls feeding on sewage sludge (Fricker & Metcalfe, 1984) or near sewage outfalls (Butterfield *et al.*, 1983; Fenlon, 1981) have found a close association between the two. The birds appear to pick up the bacteria through feeding, but tend not to excrete the

organism for long periods after ingestion, suggesting that gulls can carry infected material without becoming colonised. This could be due to the rapid passage time of food through the bird gut (Fenlon, 1981), combined with the possibility that the caecal contents of healthy adult birds may prevent *Salmonella* infection (Barnes, 1979). Salmonellae from the human population tend to appear in sewage (McCoy, 1979), resulting in similar ranges and frequencies of serotypes in the faeces of gulls feeding at sewage outfalls (Fenlon, 1981, 1983). Ingestion of *Listeria* species by gulls feeding at sewage works follows a pattern similar to that of *Salmonella* species (Fenlon, 1985). Thermophilic campylobacters are commonly associated with sewage effluent (Fricker & Park, 1989) and are probably also an important commensal component of the gull gut flora (Hatch, 1996). The optimum growth temperature of these bacteria coincides with the body temperature of birds (40–42°C), so higher rates may be excreted than are ingested, thereby amplifying the potential spread of the pathogen. If gulls feeding at sewage outfalls pick up pathogens from human sources, there is a danger that these may be reintroduced to the human population by contamination of, for example, water supplies, and facilitate the further spread of infection.

Most studies concerning transmission of bacterial pathogens from sewage to birds focus on gulls, though a few encompass other shoreline-foraging species that consume filter-feeding invertebrates, such as bivalve molluscs. When sewage effluents are present in the habitat of the filter feeder, some pathogens may become concentrated in the organism, and it may thereby act as an infection source to predators. Both shoreline-foraging birds feeding on invertebrates (Waldenström *et al.*, 2002) and various wading birds, including oystercatchers *Haematopus ostralegus*, which feed mainly on bivalve molluscs (Fricker & Metcalfe, 1984), have been shown to have high carriage rates of *Campylobacter* species (Table 2). This may be a reflection of contamination of their food sources by sewage (Fricker & Metcalfe, 1984). By contrast, a study of predominantly wild passerines, feeding at a sewage treatment works, showed no association between bacteria found in birds and those present in the sewage (Plant, 1978). Despite six serotypes of *Salmonella* appearing in sewage samples, only one bird of the 599 tested, a dunnock *Prunella modularis*, was actively excreting the bacterium (Table 2). No gulls were included in the study, and the lack of active *Salmonella* excretors in the samples may be indicative of different adaptations of the passerine and gull guts to the introduction of salmonellae.

(3) Human refuse

Landfill sites provide scavengers with a ready supply of household refuse, and attract large flocks of birds (Bowes, Lack & Fletcher, 1984; Sol, Arcos & Senar, 1995). Gulls scavenging on rubbish tips often roost on nearby fields, pastures and reservoirs, (Benton *et al.*, 1983; Monaghan *et al.*, 1985), and wash in local water bodies, and bacteria ingested at feeding sites may enter other food chains once excreted by the birds. A range of bacterial pathogens, including Vero cytotoxin-producing *E. coli* 0157, salmonellae, listerias and campylobacters have been isolated from the faeces or cloacae of gulls (*Larus* spp.), lapwings *Vanellus vanellus* and corvids feeding at refuse sites (Edel, 1976, 1978; Quessy & Messier, 1992; Wallace, Cheasty & Jones, 1997; Willumsen & Hole, 1987). Comparative studies of gull feeding habitats have isolated higher rates of *Campylobacter* and *Salmonella* species from gulls feeding at rubbish dumps than from coastal- and island-dwelling birds (Glünder *et al.*, 1991; Williams *et al.*, 1976). This suggests that landfill sites may be an important source of avian pathogens: they contain materials such as soiled nappies and decaying food, items expected to be an attractive source of nutrition to birds, and certain to contain high numbers of bacteria. Correlations between the incidence of different *Salmonella* serotypes in gulls and in the local human population reflect environmental contamination of the gulls' feeding sites. The variety and high prevalence of bacterial serotypes in gulls and other scavenging species may be related to their choice of feeding grounds, the presence of multiple serotypes being consistent with food acquired from numerous sources. Although birds may introduce pathogens to the human population, humans likewise introduce infected material to birds, often through taking poor measures to decontaminate and contain waste sufficiently.

Species scavenging in urban habitats may be a likely source of human infection, as they are often found in public spaces where children play and are susceptible to contact with the birds and/or their excrement (Casanovas *et al.*, 1995). Feral pigeons and crows (Corvidae) scavenge on litter and rubbish from waste containers, and harbour pathogens such as salmonellae, campylobacters, enterococci and streptococci (Table 2; Baele, Devriese & Haesebrouck, 2001; Bouttefroy, Lemaître & Rousset, 1997; Casanovas *et al.*, 1995; Kapperud & Rosef, 1983; Müller, 1965; Shetty *et al.*, 1990), all of which can cause disease in humans. A variety of bacterial pathogens have been isolated from starling *Sturnus vulgaris* faeces, and there is circumstantial evidence that they can transmit a number of diseases to humans and other animals, including the fungal disease histoplasmosis (Berger *et al.*, 2003; Bullough, 1942; Craven *et al.*, 2000; D'Alessio *et al.*, 1965; Palmgren *et al.*, 1997; Smibert, 1969). Starlings, which often congregate in large, city-based communal roosts, can cause concentrated faecal contamination of public areas (Feare, 1984; Grimes *et al.*, 1979), and as such, may pose a health threat to humans.

(4) Domestic animals

The role of birds as vectors of disease transmission to domestic livestock has been attributed to environmental contamination of, amongst others, water supplies (Johnston *et al.*, 1979; Jones, Smith & Watson, 1978), pastureland (Coulson *et al.*, 1983; Williams *et al.*, 1976) and feed (Fenlon, 1985) by avian faeces. Rooks, carrion crows and gulls have tested positive for various *Campylobacter* and *Listeria* species (Bouttefroy *et al.*, 1997; Fenlon, 1985; Hatch, 1996; Kapperud & Rosef, 1983; Willumsen & Hole, 1987) and there is a concern that grazing cattle may be exposed to pathogens deposited by these birds, as they commonly frequent fields where livestock graze (Coulson *et al.*, 1983; Williams *et al.*, 1976). Incidents of disease transmission, from

one species to another, through environmental contamination of grazing pastures, have been documented in mammals. Badgers *Meles meles* have been blamed by some authors for the infection of cattle with the tuberculosis-causing bacterium *Mycobacterium bovis* (Clifton-Hadley *et al.*, 1995; Delahay, Cheeseman & Clifton-Hadley, 2001; Máirtín *et al.*, 1998), whereas Yellowstone bison *Bison bison* are perceived as a threat to the local cattle industry as vectors of *Brucella abortus* (Baskin, 1998). However, it was infected cattle that originally introduced *B. abortus* to the bison (Dobson & Meagher, 1996), and the evidence for transmission of tuberculosis from badgers to cattle is contentious, as transmission could equally be from cattle to badgers (Cleaveland *et al.*, 2002; Woodroffe *et al.*, 2006). Birds have been implicated in the faecal contamination of silage with *Listeria* species (Fenlon, 1985), although speculations that birds are the source of the pathogen are not supported by the evidence. Alternatively, it could be argued that birds foraging on silage may acquire the pathogen from the feed, as *Listeria* appears to be commonly associated with silage, especially when anaerobic conditions are not maintained (Grønstøl, 1979). Both *Salmonella* and *Campylobacter* species have been isolated from pig slurry (Watabe *et al.*, 2003), while *Campylobacter* species have been isolated from bovine slurry, dung and cattle bedding, all sources which may be scavenged by wild birds (Stanley & Jones, 2003). Despite these potential sources of pathogens being accessible to wild birds, enabling the spread of pathogens from domestic animals, *via* birds, to other hosts, there is little evidence supporting this route of transmission. It has also been suggested that wild birds may introduce bacterial pathogens to poultry houses, and act as vectors between them (Craven *et al.*, 2000; Goodchild & Tucker, 1968). Whether wild birds actually introduce infectious agents, or simply have the potential to transfer already existing disease organisms, remains to be determined.

V. AVIAN FAECAL POLLUTION OF WATER

Birds not only acquire pathogens from the environment, but also return them *via* excretion, potentially facilitating the dissemination of pathogenic organisms to both humans and other animals, especially through water. Livestock on many farms rely on rivers, streams and other untreated water sources for at least part of their drinking water (Reilly, 1981), and wild birds roosting in large numbers on or near water may contribute to its contamination and the spread of disease to other animals. Since many of the pathogens found in bird faeces originate from human sewage, it should follow that humans are highly susceptible to infection when the bacteria re-enter the human food chain through drinking water supplies and bathing water.

(1) Drinking water

Waterfowl and gulls tend to congregate in large numbers and roost on reservoirs, especially during winter (Bowes *et al.*, 1984), and are therefore a possible source of contamination for drinking water supplies. Geese are notorious for their ability to leave abundant amounts of faecal matter whilst grazing and roosting. They are predominantly vegetarian, however, and there are no data to suggest that geese are an important source of salmonellae, though they have been shown to shed large quantities of enterobacteria and campylobacters (Alderisio & DeLuca, 1999; Varslot *et al.*, 1996); both pathogens that can cause disease in humans.

Gulls are capable of carrying bacterial pathogens whilst remaining apparently unaffected (Butterfield *et al.*, 1983; Monaghan *et al.*, 1985), and move between feeding grounds and roosts (Fenlon, 1983), carrying bacteria with them, often over considerable distances (Coulson, Butterfield & Thomas, 1983; Wuthe, 1973). Birds roosting in large numbers on reservoirs have been reported to carry bacterial pathogens and contaminate domestic water supplies (Alderisio & DeLuca, 1999; Mitchell & Ridgewell, 1971), though some argue that the level of pathogens harboured by birds is too low to present a major health hazard to humans (Girdwood *et al.*, 1985). It has been suggested that wild birds could mediate environmental contamination of surface waters with campylobacters, which might represent a risk to public health in places where the water is consumed untreated or is used for recreational purposes (Moore *et al.*, 2002; Obiri-Danso, Paul & Jones, 2001). High isolation rates of *C. jejuni* in free-living and migratory waterfowl indicate that Anseriformes (ducks and geese) can serve as important carriers of *Campylobacter* infection (Table 2; Luechtefeld *et al.*, 1980; Pacha *et al.*, 1988; Yogasundram, Shane & Harrington, 1989), which may be of public health importance through the contamination of water, or when these birds are used as food (Luechtefeld *et al.*, 1980). Indeed, incidents of water-borne outbreaks of *C. jejuni* infections in humans have been attributed to the contamination of untreated drinking water by the droppings of pink-footed geese *Anser brachyrhynchus* (Varslot *et al.*, 1996). Although it has been demonstrated that contamination of reservoirs with faecal coliforms and salmonellae may be significantly reduced when gulls are discouraged from roosting (Benton *et al.*, 1983), indicating that the birds are a probable source of the pathogens, it has been argued that gulls may not actually be an important factor in the aetiology of human salmonellosis because they do not shed the bacteria for long periods after ingestion (Girdwood *et al.*, 1985). A better understanding of the rates and duration of bacterial shedding in birds would provide insight into the relative importance of birds as pathogenic vectors.

(2) Bathing water

It has been argued that shorebirds may contaminate bathing waters (Jones, 2001; Obiri-Danso *et al.*, 2001), although the evidence is limited. Campylobacters in intertidal sediments are generally absent in warm summer months, while they are consistently present in colder winter months (Jones, Betaieb & Telford, 1990; Teunis *et al.*, 1997). The bacteria are unable to multiply in water; survival is short and is both temperature and UV-B dependent (Blaser *et al.*, 1980; Davies & Evison, 1991; Rollins & Colwell,

1986), and the presence of campylobacters in sediment is indicative of recent faecal contamination (Obiri-Danso & Jones, 1999*b*). *C. lari* from bird faeces is better able to survive in sea water than *C. jejuni*, which is found in sewage effluent, and is also mainly deposited on or near the shore where bathers might encounter it, rather than further offshore, as is the case for most sewage effluent (Obiri-Danso *et al.*, 2001). It has been suggested that the presence of *C. lari* and urease-positive thermophilic campylobacters, in the absence of *C. jejuni*, is due to the presence of birds rather than contamination from sewage (Obiri-Danso & Jones, 2000). Campylobacters were reported in greater numbers in winter, concomitant with large flocks of migratory birds (Obiri-Danso & Jones, 2000), though no avian faecal samples were tested for the presence of the bacteria. It could equally be that higher numbers of campylobacters found in winter are due to longer survival rates facilitated by reduced temperatures and lower UV-B levels, and that the increased presence of birds is coincidental. Regardless of the source of the campylobacters, it appears doubtful that the levels of contamination are sufficient to pose a threat to human health, since recreational use of beaches diminishes during winter. It is also noteworthy that *C. jejuni* is the species that causes human disease, whereas *C. lari*, the dominant species isolated from coastal bathing waters and birds, rarely causes disease in humans (Hatch, 1996).

Shorebirds have often been cited as a direct source of *E. coli* (Fogarty *et al.*, 2003; Jones & Smith, 2004; Levesque *et al.*, 2000; Whitman & Nevers, 2003), salmonellae (Whitman & Nevers, 2003) and other faecal coliforms (Jones & Obiri-Danso, 1999; Standridge *et al.*, 1979) found in both sand and marine water. Conversely, fresh-water bathing sites are mainly at risk from contamination by wildfowl, which have been shown to be substantive reservoirs of pathogenic bacteria for rivers (Obiri-Danso & Jones, 1999*a*; Yogasundram *et al.*, 1989). Beach visitors spend the majority of their time in contact with sand, and hence any associated contamination of the shoreline. Bacteria persist longer in sand than in water because they adhere to sediment particles, which provide a stable, non-starvation environment (Howell, Coyne & Cornelius, 1996; Whitman & Nevers, 2003). Thus, sediment may serve as a reservoir for pathogenic organisms (Obiri-Danso & Jones, 2000), especially to children who may be exposed to infection whilst digging and playing in wet sand. Recreational use of the water in general disturbs the sediment, releasing faecal coliforms into the overlying water (Obiri-Danso & Jones, 1999*b*), increasing the risk of contact for humans.

VI. METHODS FOR DETECTING AVIAN ENTERIC BACTERIA

There is a current lack of quantitative data on the amounts of enteropathogenic bacteria shed by wild birds, as much of the information is restricted to the presence or absence of specific pathogens (i.e. pathogen prevalence). Since selection of specific bacteria is a limiting factor in traditional microbiological methods, studies may be biased towards testing for the types of bacterial pathogens suspected to be present, resulting in others being overlooked (Fig. 1). This makes it extremely difficult to tell whether the birds are important zoonotic reservoirs, and are major health risks and contributors to pollution, or whether they are simply passive carriers of the bacteria and relatively unimportant in this regard.

Most studies investigating the presence of bacterial enteropathogens in wild birds have applied traditional microbiological techniques, which use selective agars and broths, to isolate target species from faecal matter or cloacal swabs (Brittingham *et al.*, 1988; Kapperud & Rosef, 1983; Waldenström *et al.*, 2002). These methods are not only highly time consuming and labour intensive, but are also associated with various problems in regard to culturing bacteria from faeces, as it contains a mixed population of numerous different bacterial types; a number of authors has stated that only a minority of bacterial species visualised by direct microscopic counting methods can be cultivated (Amann, Wolfgang & Schleifer, 1995; Head, Saunders & Pickup, 1998; Vaughan *et al.*, 2000). Reasons for this anomaly include a lack of knowledge concerning growth requirements, the necessity for strictly anoxic growth conditions for some species, selectivity of media used, and the intricacies of reproducing the interactions of bacteria with other microorganisms and host cells in their natural environment. Thus, characterising microbial communities exclusively on the basis of culturable bacteria may be misleading, especially for environmental bacteria that have not been extensively studied.

The inability to culture the majority of bacteria has led to the development of various culture-independent methodologies, particularly those relating to small subunit (SSU) ribosomal RNA (rRNA) typing, for studying complex microbial ecosystems such as that of the mammalian intestinal tract (Tannock, 1999). Several fingerprinting techniques utilising SSUs have been developed to monitor bacterial community shifts and to compare bacterial communities. These techniques include denaturing gradient gel electrophoresis (DGGE), temperature gradient gel electrophoresis (TGGE), single strand conformation polymorphism (SSCP) and terminal-restriction fragment length polymorphism (T-RFLP) analysis. To date, these fingerprinting techniques have been used successfully to characterise and monitor a variety of gastrointestinal bacterial communities, including those in insects (Hongoh, Ohkuma & Kudo, 2003; Mohr & Tebbe, 2006), rodents (Deplancke *et al.*, 2000; Inoue & Ushida, 2003; McCracken *et al.*, 2001; Walter *et al.*, 2000), dogs (Simpson *et al.*, 2002; Suchodolski, *et al.*, 2004), pigs (Konstantinov *et al.*, 2003; Simpson *et al.*, 2000; Simpson *et al.*, 1999), cattle (Kocherginskaya, Aminov & White, 2001), poultry (Chambers *et al.*, 2001; Gong *et al.*, 2002; Van der Wielen *et al.*, 2002; Zhu *et al.*, 2002) and humans (Seksik *et al.*, 2003; Tannock *et al.*, 2000; Zoetendal, Akkermans & de Vos, 1998). As a great deal of the work on the shedding of enteropathogens by wild birds is restricted to relatively basic phenotypic analysis, the need remains to implement these contemporary molecular methods to complement and expand current data. Extraction of rRNA

from avian faeces for examination of bacterial presence is relatively rapid, sensitive and specific, is more reliable and reproducible than traditional culturing methods (Chambers *et al.*, 2001), and enables the detection of pathogens that are present even in relatively low concentrations (Murray *et al.*, 1996; Muyzer, de Waal & Uitterlinden, 1993). The alternating, interspecific, variable and highly conserved regions of the SSUs enables the detection, identification and enumeration of microbial species (Muyzer & Smalla, 1998; Vaughan *et al.*, 2000). Though exploring microbial populations with molecular techniques encompasses its own set of biases and limitations (Muyzer & Smalla, 1998), a wider range of organisms can be scrutinised than when using culture-dependent methods. Although culture-independent techniques are readily accessible, and have been used to study microbial communities in both mammals and poultry (see above), there is a paucity of similar studies in wild birds. The most likely explanations for this are the high costs associated with using modern molecular techniques coupled with commercially unimportant bird species.

The use and further development of molecular techniques, which enable a greater degree of species detection when studying complex bacterial communities, should facilitate a better understanding of the normal intestinal flora of wild birds. In so doing, it should be possible to determine more effectively how microbes affect avian health, life-history trade-offs and perhaps even the dynamics of bacterial transmission (both pathogenic and non-pathogenic) amongst birds, and between birds and other taxa (Fig. 1).

VII. CONCLUSIONS

(1) In light of the emergence of new infectious diseases in wildlife, it is currently of great interest to address the implications of birds as potential vectors of pathogenic bacteria. Though diverse studies have examined the bacterial enteropathogens found in wild birds, there remains a paucity of quantitative data regarding the amounts of avian enteropathogenic bacteria in apparently healthy populations. This renders it difficult to determine whether wild birds pose a major health hazard and contribute significantly to pollution, or whether they are relatively unimportant in this regard. Use of culture-independent methods for studying avian microbial communities could prove invaluable for expanding our current knowledge, and facilitate a better understanding of the complexities and interactions of the genera inherently present in the avian gut, and with those acquired from the environment.

(2) Various factors appear to bias our current knowledge of pathogen prevalence in wild birds, including:

(*a*) Selective isolation of pathogens of interest, thereby potentially excluding the detection of other equally important infectious organisms;

(*b*) Concentrating prevalence studies on provisioned populations of wild birds, thus excluding non-provisioned birds from the potential host population;

(*c*) Concentrating studies and reports on birds that have died from disease, excluding apparently healthy individuals that may be carrying the pathogens;

(*d*) Studies with small sample sizes, which may result in abnormally high or low prevalences due to sampling biases.

Future studies should aim to avoid these factors, and, as far as possible, exclude as many variables as possible that are likely to bias results.

(3) Although there is evidence of wild birds acquiring pathogens through foraging on contaminated material, there appears to be little or no evidence that once infected, the birds in turn transmit the causative agent to other birds through contamination of feeding areas. This has implications for the relative importance of feeding stations as foci for disease transmission, and merits further exploration in light of the increase in the amount of provisioning of wild birds.

(4) Birds are vulnerable to pathogenic infection at all stages of life, and although heterogeneities within host populations may affect the susceptibility of birds to bacterial disease, more rigorous, longitudinal studies are required to determine the relative importance of host intrinsic effects on susceptibility to bacterial infection.

(5) Because wild birds may be vectors of disease, it is important to understand the true source of the infectious organisms. By identifying the extent of pathogenic contamination of the environment through human activity, measures may be taken to reduce the scope for acquisition, and thus spread of disease, by birds.

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