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REVIEW

Wild birds and human pathogens in the context of ringing and migration

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Wild birds carry a diversity of micro-organisms that are pathogenic to humans, may be transmitted over long distances during migrations, and are potentially transmissible to people who handle and ring birds. High-profile diseases that are associated with carriage by birds include avian influenza, West Nile fever, and Lyme disease. Also potentially important is the existence of an avian reservoir of bacteria that are enteric human pathogens; for example species of *Campylobacter* and *Salmonella*, and toxin-producing strains of *Escherichia coli*. Wild birds have been implicated in the transfer of these enteric pathogens to people. The subject of wild birds as potential reservoirs of pathogens that may be transmitted to humans in the context of ringing and migration is reviewed and it is recommended that appropriate precautions to minimise risk should be taken during and subsequent to the handling of wild birds.

Wild birds are usually regarded as visible indicators of diverse and healthy environments. However, from a public-health perspective, this positive view is not always valid (Jones 2005). In some contexts wild birds are responsible for faecal pollution, for example waterfowl at amenity ponds (Abulreesh *et al* 2004, 2005), and they can carry a wide range of viral, bacterial, fungal and protozoan zoonotic agents (pathogens that may be transmitted to humans), either being themselves diseased or being seemingly healthy carriers, or the hosts of infected vectors (Hubálek 2004). Given their ability to fly freely and cover long distances during annual migrations, wild birds potentially play a role in the epidemiology of human-associated zoonoses.

The international news media have recently focused on examples of the actual and/or potential dissemination, by migrating birds, of diseases that infect humans, notably H5N1 avian influenza, West Nile virus and Lyme disease. Enteric pathogenic bacteria of humans, that can also be isolated from wild birds, are perhaps of less interest to the news media than are these higher-profile diseases; nevertheless, they are important in the context of transmission of potential human pathogens by migrating birds, including antibiotic resistant strains, and the handling of birds by ringers. This review considers the potential role of wild birds in the direct or indirect transmission of H5N1, West Nile virus and Lyme disease to humans and addresses the incidence of human-enteric bacterial pathogens in wild birds, their dissemination and public health relevance. It is our aim to provide this information within the context of bird ringing and the handling of wild birds. Diseases of wild birds caused by bacterial pathogens are not addressed.

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Avian influenza

Avian influenza is known to persist as a reservoir in wild birds, and although birds carrying a variety of virus subtypes may be asymptomatic (Webster *et al* 1992) these viruses can cause severe respiratory disease in a wide range of animals, including humans (Melville & Shortridge 2006, Woo *et al* 2006). In recent years an avian influenza virus subtype H5N1 has emerged in bird populations and although transmission to humans has been rare, outbreaks associated with close human–poultry interaction occurred in southern China in 2003, and in several other countries (Steensels *et al* 2006, Woo *et al* 2006) including Egypt and Turkey by 2006. Significantly, the H5N1 virus subtype has also been identified as the cause of substantial mortality amongst Bar-headed Goose *Anser indicus* and other migratory wildfowl at Qinghai Lake in western China. As this was originally described as being a very remote area without poultry markets to provide an obvious source of infection it was suggested that migratory birds flying into the area were expanding the range of the virus (Chen *et al* 2005). However, it has subsequently emerged that in the Qinghai area Bar-headed Geese are reared in captivity and there is a sufficient level of poultry keeping for farmed rather than wild birds to be the source of the outbreak (Butler 2006). Prior to, and at the time of, the Qinghai outbreak in 2005, H5N1 was restricted to southeast Asia, but during 2005 and 2006 the virus spread throughout Europe and into Africa.

A movement of H5N1 virus into Europe in spring 2006 by wild birds (especially Mute Swan *Cygnus olor*) has been recorded (Feare & Yasué 2006), but this was related to a short-distance movement of birds away from adverse weather conditions in the area of the Black and Caspian Seas rather than to migration. The chronology of the spread of the virus out of Asia (it was not coincident with migratory periods) and its geography (it did not follow known avian migration routes) suggests that although wild birds may be involved in small-scale movements of the virus they are unlikely to be the main vector for long-distance movement, or for the infection of new areas. Current evidence suggests that the virus is dispersed along human transport routes and thus commercial movements of domestic or farmed birds and bird products are likely to be the main agents spreading H5N1 virus (Gauthier-Clerc *et al* 2007).

West Nile virus

West Nile virus is a neuropathogen, widespread in wild birds in Europe and Asia, that has a bird–mosquito–bird life cycle and may be acquired by humans through mosquito (*Culex* spp.) bites. Although many people infected with West Nile virus remain asymptomatic, around 20% of cases lead to mild fever, headache, body aches and perhaps a rash on the body. Serious infections can result in meningitis/encephalitis and the disease can be fatal. During the late 1990s, localised fatal human epidemics in Romania, the Czech Republic, west Georgia (former Soviet Union) and in southern Russia highlighted the importance of this emerging zoonotic disease (Malkinson *et al* 2002). That the virus is carried through Europe by migrating birds has been confirmed by serological studies of a population of migrating White Stork *Ciconia ciconia*. Crucially, these birds, sampled in Eilat, Israel, during August, were juveniles, indicating that they were on their first southward migration and that they must therefore have been infected with the virus in the natal European/near-European area or along their trans-European migration route (Malkinson *et al* 2002).

The first outbreak amongst humans in the Americas was in 1999, in the New York area, and was accompanied by substantial mortality amongst wild birds, especially American Crow *Corvus brachyrhynchos*. There were also deaths amongst exotic zoo-kept birds, including Chilean Flamingo *Phoenicopterus chilensis* (Lanciotti *et al* 1999, Nash *et al* 2001). The outbreak may have originated from avian carriage by regular inter-hemisphere migration or, perhaps more likely, from vagrant West African birds displaced by tropical storms, or from (legal or illegal) importation of exotic birds (Rappole *et al* 2000). Rappole *et al* (2000) stress the potential for further dissemination throughout the Americas, from the original New York location, along migration routes elucidated on the basis of evidence from ringing returns; these authors suggest that European Starling *Sturnus vulgaris* migrating to the southeastern United States, Herring Gull *Larus (argentatus) smithsonianus* migrating to Florida and Central America, and Common Tern *Sterna hirundo* passing through sites throughout South America may be potential vectors of the disease.

Lyme disease

Lyme disease is acquired by humans through tick bites (*Ixodes* spp.) and has a diversity of symptoms including an initial rash followed by musculoskeletal and neurological problems. The causative agent is the spirochaete bacterium *Borrelia burgdorferi* (*sensu lato*) complex. The spirochaetes are transmitted through ticks that are parasitic on wild birds and mammals (Anderson *et al* 1986). Hilton (1991) noted ticks on 157 out of 17,018 individual birds from 16 species while ringing in South Carolina. He also observed that the incidence of tick infestation was sometimes markedly greater: thus 41 of 345 Purple Finches *Carpodacus purpureus* captured during January–March 1991 were infested. Many studies have retrieved *Borrelia*-positive ticks from migrating birds, for example in the US (Weisbrod & Johnson 1989, Nicholls & Callister 1996, Smith *et al* 1996), Canada (Scott *et al* 2001) and Japan (Ishiguro *et al* 2000). A study of a mixed woodland in southern England showed that many adults and children who used the woodland for recreation were bitten by infected ticks (Robertson *et al* 2000) – although a local clinic that removed ticks and followed up the patients reported no subsequent borreliosis. The identification of *Borrelia* genospecies by DNA fingerprinting implicated woodland birds as an important reservoir host.

Enteric pathogenic bacteria

Campylobacters

Thermotolerant campylobacters (*ie* bacteria that grow well at 35–37°C, but also tolerate and grow well at higher temperatures, *eg* 42°C) are possibly the leading worldwide cause of bacterial gastroenteritis in humans (Moore *et al* 2005). *Campylobacter jejuni*, *C. coli* and to some extent *C. lari* are the species most often encountered in water-borne and food-borne infections (Moore *et al* 2005, Lastovica 2006). The ecology of *Campylobacter* species, including the emerging multi-drug-resistant strains, involves their presence in an avian reservoir (Newell 2002, Lee & Newell 2006, Abulreesh *et al* 2006).

The incidence (*ie* the percentage of positive cloacal or faecal samples) of thermotolerant campylobacters in diverse wild birds from a wide range of geographical locations is summarised in Table 1. The occurrence of thermotolerant campylobacters in the gut of apparently healthy wild birds has frequently been reported (*eg* Waldenström *et al* 2002, studies cited in Table 1). Thus *Campylobacter* species colonising the gut of wild birds are regarded as commensals; an observation that might be related to the body temperature of wild birds (42°C) being the optimal growth temperature for thermotolerant campylobacters (Newell 2002, Waldenström *et al* 2002, Abulreesh *et al* 2006).

Salmonellae

Salmonella species are enteric pathogens of humans and animals, including birds (Murray 2000, Tizard 2004).The carriage of salmonellae, including the virulent multi-drug-resistant *S. typhimurium* definitive type 104 (DT104), by apparently healthy wild birds, has been widely reported (Table 1) but the incidence of the organism in birds tends to be low, often in only a few percent of samples (Palmgren *et al* 1997, 2006, Kirk *et al* 2002, Hernandez *et al* 2003, Reche *et al* 2003, Fallacara *et al* 2004). Indeed, when Hernandez *et al* (2003) sampled Palearctic birds migrating southwards and which were likely to have had no recent experience of areas with domestic animals, they found only one *Salmonella*-positive bird, a Mistle Thrush *Turdus viscivorus,* amongst 2,377 sampled from 110 species. Faecal samples from penguin colonies visited by tourists (233 samples from eight bird species) were all *Salmonella*- (and *Campylobacter*-) negative, suggesting that tourism has not yet introduced human-associated enteric pathogens to the Antarctic (Bonnedahl *et al* 2005). These studies suggest that wild birds may acquire salmonellae after exposure to human-contaminated environments, or after scavenging on refuse tips and sewage sludge, and that wild birds that live away from such environments are unlikely to harbour *Salmonella* (Murray 2000, Tizard 2004).

Toxin-producing *Escherichia coli*

Although *Escherichia coli* is part of the normal flora of the intestinal tract of vertebrates, nevertheless, virulent and sometimes lethal toxin-producing pathogenic strains do exist (Hunter 2003). Studies that have addressed the incidence of such strains in the intestinal tract of wild birds suggest that there is an avian reservoir (Hubálek 2004). The Vero cytotoxin-producing strain *E. coli* O157, that causes enterohaemorrhagic infections in humans (Hunter 2003), and other pathogenic serogroups have been recovered from wild birds (Wallace *et al* 1997, Kullas *et al* 2002, Wani *et al* 2004, Sonntag *et al* 2005, Ejidokun *et al* 2006, Foster *et al* 2006) (Table 1).

Antibiotic resistance

Antibiotic-resistant enteric bacteria have been isolated from birds. Waldenström *et al* (2005) visit the debate about the role of over-use of antibiotics in animal husbandry and in the treatment of disease in humans and raise the question of a potential role for birds in the transmission of antibiotic resistance into the environment from human-associated systems. They tested 137 isolates of *Campylobacter jejuni*, from 12 species of wild birds in Sweden, against 10 antimicrobial compounds and found a relatively low prevalence of resistance (*ie* lower than in isolates from humans or poultry). Nevertheless they did obtain a multiple-resistant (nalidixic acid and ciprofloxacin) isolate from a Long-eared Owl *Asio otus*. In a separate study two of 28 isolates of *Salmonella* from Black-headed Gulls *Larus ridibundus* exhibited multiple resistance; one to sulfisoxazole, ampicillin and trimethoprim, and one to sulfisoxazole, ampicillin, streptomycin and chloramphenicol (Palmgren *et al* 2006). Both isolates were from spring-migrating birds captured in Malmö, Sweden.

Other studies have addressed the incidence of non-pathogenic but emerging antibiotic-resistant strains of *Escherichia coli* and the role of an avian reservoir in their distribution in the environment. Cole *et al* (2005) showed an increased proportion of resistant *E. coli* isolates from Canada Goose *Branta canadensis* that had utilised swine-waste lagoons in Georgia, USA, while more than 95% of isolates from migrating Canada Geese in Maryland were resistant to at least one of penicillin G, ampicillin, cephalothin and sulfathiazole, and many isolates had multiple resistance (Middleton & Ambrose 2005). Dolejská *et al* (2007) took cloacal swabs from young Black-headed Gulls at three breeding colonies in the Czech Republic and from 75 of the 257 birds sampled they isolated *E. coli* showing resistance to tetracycline, cephalothin, streptomycin, sulphonamides and/or chloramphenicol. The relative incidences of resistance to each of these antibiotics were found to mirror the relative use of the compounds in human and veterinary medicine in the Czech Republic and the authors suggest that the birds are an important reservoir of antibiotic-resistant bacteria reflecting their presence in the gulls' food or water.

Public-health aspects of enteric bacterial pathogens of avian origin

Wild birds have been associated worldwide with outbreaks of water-borne and food-borne disease. In Florida, Common Grackles *Quiscalus quiscula* were implicated in the contamination of drinking-water settling tanks with *Campylobacter* (Sacks *et al* 1986). Similarly, two outbreaks of campylobacteriosis in Norway were attributed to water supplies contaminated by the faeces of Pink-footed Goose *Anser brachyrhynchus* (Varslot *et al* 1996). There is also strong evidence that wild birds such as Magpie *Pica pica* and Jackdaw *Corvus monedula* have contaminated milk with *Campylobacter* spp. by pecking through the aluminium-foil tops of bottles on doorsteps in England and Wales (Southern *et al* 1990, Hudson *et al* 1990, Palmer & McGuirk 1995).

Table 1. Selected examples of the incidence of the enteric pathogens, *Campylobacter* spp., *Salmonella* spp. and *Escherichia coli* in wild birds (detected in fresh faeces or cloacal swabs).

Bird species	Location	p:n (%)	Pathogen species or strain	Reference
CAMPYLOBACTER				
Black-headed Gull *Larus ridibundus*	Sweden	133:367 (36)	*C. jejuni*	Broman *et al* 2002
Waterfowl (Anatidae)	England	10:90 (11)	*C. jejuni*	Abulreesh 2005
	USA	190:450 (42)	*C. jejuni*	Fallacara *et al* 2004
	Taiwan	1,045:2,400 (44)	*C. jejuni, C. coli*	Tsai & Hsiang 2005
	Sweden	6:116 (5)	*C. jejuni, C. lari*	Waldenström *et al* 2006
Waders *Tringa totanus, Calidris alpina, Vanellus vanellus*	Sweden	14:129 (11)	*C. jejuni, C. lari*	Waldenström *et al 2006*
Jungle Crow *Corvus levaillantii*	Japan	7:11 (64)	*C. jejuni,* UPTC	Matsuda *et al* 2002
Feral Pigeon *Columba livia*	Spain	105:400 (26)	*C. jejuni*	Casanovas *et al* 1995
	Croatia	2:14 (14)	*C. jejuni*	Vlahović *et al* 2004
House Sparrow *Passer domesticus*	Chile	33:100 (33)	*C. jejuni, C. coli*	Fernández *et al* 1996
Starling *Sturnus vulgaris*	Sweden	1:31 (3)	ND	Palmgren *et al* 1997
Brown-eared Bulbul *Hypsipetes amaurotis*	Japan	4:36 (11)	*C. jejuni*	Ito *et al* 1988
Azure-winged Magpie *Cyanopica cyanus*	Japan	2:10 (20)	*C. jejuni*	Ito *et al* 1988
Blackbird *Turdus merula*	Sweden	2:32 (6)	ND	Palmgren *et al* 1997
	Sweden	41 isolates	*C. jejuni*	Waldenström *et al* 2005
Jackdaw *Corvus monedula*	Sweden	4:4 (100)	*C. jejuni*	Waldenström *et al* 2002
Dunlin *Calidris alpina*	Sweden	250:313 (80)	*C. jejuni, C. coli, C. lari*	Waldenström *et al* 2002
	Sweden	31 isolates	*C. jejuni*	Waldenström *et al* 2005
Song Thrush *Turdus philomelos*	Sweden	39 isolates	*C. jejuni*	Waldenström *et al* 2005
Long-eared Owl *Asio otus*	Sweden	8 isolates	*C. jejuni*	Waldenström *et al* 2005
Sparrowhawk *Accipiter nisus*	Sweden	1 isolate	*C. jejuni*	Waldenström *et al* 2005
SALMONELLA				
Black-headed Gull *Larus ridibundus*	Czech Republic	38:154 (25)	*S. typhimurium, S. enteritidis, S. panama, S. anatum*	Hubálek *et al* 1995
	Czech Republic	207:1,095 (19)	*S. typhimurium*	Čížek *et al* 2007
	Sweden	28:1,047 (3)	16 serotypes	Palmgren *et al* 2006
Waterfowl (Anatidae)	USA	8:450 (2)	*S. typhimurium*	Fallacara *et al* 2004
	Taiwan	91:2,000 (5)	ND	Tsai & Hsiang 2005
Feral Pigeon *Columba livia*	Japan	17:436 (4)	ND	Tanaka *et al* 2005
	Norway	3:72 (4)	*S. typhimurium*	Refsum *et al* 2002
	Croatia	2:14 (14)	*S. typhimurium*	Vlahović *et al 2004*
Coot *Fulica atra*	Czech Republic	1:3 (33)	*S. typhimurium*	Hubálek *et al* 1995
House Sparrow *Passer domesticus*	USA	14:451 (3)	*S. montevideo, S. meleagridis*	Kirk *et al* 2002
	Norway	7:31 (23)	*S. typhimurium*	Refsum *et al* 2002
Starling *Sturnus vulgaris*	USA	1:80 (1)	*S. typhimurium*	Kirk *et al* 2002
	Czech Republic	4 isolates	ND	Čížek *et al* 1994
Magpie *Pica pica*	Norway	1:40 (3)	*S. typhimurium*	Refsum *et al* 2002
Great Tit *Parus major*	Norway	6:87 (7)	*S. typhimurium*	Refsum *et al* 2002
	Czech Republic	1 isolate	ND	Čížek *et al* 1994
Brown-headed Cowbird *Molothrus ater*	USA	3:95 (3)	*S. meleagridis, S. muenster*	Kirk *et al* 2002
Rook *Corvus frugilegus*	Croatia	2:13 (15)	*S. typhimurium, S. enteritidis*	Vlahović *et al* 2004
Hooded Crow *Corvus cornix*	Norway	1:52 (2)	*S. paratyphi B*	Refsum *et al* 2002

Table 1 cont'd

Bird species	Location	p:n (%)	Pathogen species or strain	Reference
Long-eared Owl *Asio otus*	Spain	1:7 (14)	*S. typhimurium* DT104	Reche *et al* 2003
Lesser Kestrel *Falco naumanni*	Spain	3:59 (5)	*S. enteritidis*	Reche *et al* 2003
Buzzard *Buteo buteo*	Spain	1:17 (6)	*S. typhimurium* DT104	Reche *et al* 2003
TOXIN-PRODUCING *ESCHERICHIA COLI*				
Gulls *Larus canus, L. argentatus, L. ridibundus*	England	100:349 (29)	O157	Wallace *et al* 1997
Canada Goose *Branta canadensis*	USA	147:397 (37)	O8, O15, O18, O91, O159	Kullas *et al* 2002
Rook *Corvus frugilegus*	England	1:1(100)	O157	Ejidokun *et al* 2006
Feral Pigeon *Columba livia*	India	25:25 (100)	O9, O18, O157, O168, O77	Wani *et al* 2004
Garden birds	Scotland	1:231 (0.4)	O157	Foster *et al* 2006

p = number of positive samples, n = number of samples tested, (%) percentage of positive samples
ND = not determined
UPTC = urease-positive thermophilic *Campylobacter*

In New Zealand, isolates of *Salmonella typhimurium*, serotype DT160, from House Sparrow *Passer domesticus* and humans were indistinguishable (Alley *et al* 2002). Birds were believed to be a source of salmonellae to livestock (through birds foraging on feed) and also to domestic cats (through cats feeding on salmonellae-infected dead birds). Both livestock and cats were a source of *Salmonella* infection in the human community (Alley *et al* 2002). Furthermore, the incidence of DT160 salmonellosis in humans in New Zealand was linked to the handling of dead wild birds, mainly sparrows (Thornley *et al* 2003).

E. coli O157 has been isolated from faeces collected from a garden bird feeding station in southwest Scotland, albeit at a low level (Foster *et al* 2006). However, this does indicate that stringent hygiene for both birds and people is a priority during ringing exercises and during garden-bird feeding. Fatal bacterial infection of birds (*Salmonella typhimurium* DT40 and *E. coli* O86) at bird tables has been reported (Pennycott *et al* 1998). Although *E. coli* O157 has been isolated from wild birds (Table 1), evidence of transmission to humans from wild birds is not generally established, but it has been shown that contact with other animals and their products is important in the infection of humans. Significantly, a study in Gloucestershire, UK, showed that Rook *Corvus frugilegus* faeces were the source of *E. coli* O157 infection in two children and their mother; the father who worked as a forester had trapped and handled the birds and was the source of infection to his family (Ejidokun *et al* 2006).

Relevance to ringers

Licensed ringers will know that wild birds are a potential source of zoonotic infection and will be aware of the advice that is to be found on the web sites of licensing and related organisations; *eg* the British Trust for Ornithology (www.bto.org/ringing/diseases-from-birds.doc) and the (US) Ornithological Council (www.nmnh.si.edu/BIRDNET/). People who handle birds might also usefully consult the website of the UK Health Protection Agency (www.hpa.org.uk/topics/index.htm) which has a risk assessment that relates exposure to wild birds to the potential hazard of avian influenza.

All wild birds carry diverse micro-organisms and our review shows that many birds carry organisms that are human pathogens and that handling birds thus involves a risk to human health if good hygiene is not practised. Transmission from birds to humans may be direct (as is presumed to be the case for avian influenza and is known to be the case for enteric bacteria) or indirect (*ie* via ticks in the case of Lyme disease and mosquitoes in the case of West Nile virus). Ringing birds therefore carries risk as it brings individuals into close proximity with potentially infectious birds or the disease vectors associated with birds. It should also be remembered that human-to-human onward transmission of bacterial infection is also possible and that infection risks are higher for some categories of people, *eg* children, people who are elderly and those who are immuno-compromised because of HIV or cancer treatment, or as a result of organ transplant.

Precautions to minimise risk during and subsequent to the handling of birds, that are recommended by the web sites cited above, include:

- Avoiding or minimising contact with faeces.
- Avoidance of hand-to-face contact; *eg* licking of fingers or pencils, biting nails, smoking, eye rubbing or handling contact lenses.
- Use of wipes for removal of faeces from hands and clothing and for hand wiping, particularly when clean water for washing is not available.

- Hand washing with soap and water – which can be taken into the field.
- The bagging of used wipes for subsequent disposal, and of used bird bags and soiled clothing for machine washing with detergents and hot water.
- The restriction of cloacal sexing to very experienced ringers who should wear disposable gloves – and wash their hands thoroughly after removal of gloves.

It is apparent from the literature reviewed here that these and other recommended precautions should be taken seriously; more than lip-service should be accorded them.

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