Salmonellosis in Wild Birds

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Salmonella bacteria, especially *Salmonella enterica*, serotype Typhimurium, are commonly found in the intestine of wild birds. These organisms are maintained within bird populations by several mechanisms. The simplest of these mechanisms occurs in raptors since birds that eat other animals risk eating Salmonella-infected prey. Both wild and captive raptors may be temporary or permanent Salmonella carriers or even suffer from clinical Salmonellosis as a result of eating infected prey. A similar infection pathway affects scavenging or carrion eating birds such as vultures, crows, and, most importantly, gulls. For example, gulls are opportunistic scavengers who feed at sites where raw sewage is released. They appear to be relatively resistant to disease but may serve as effective carriers of Salmonella and thus are a source of infection for other animals. In other situations, birds exposed to a contaminated environment may become infected accidentally. This is the case with domestic pigeons and colonial waterbirds. The most significant outbreaks of wild bird Salmonellosis occur, however, in passerines. Thus, although only a few healthy passerines harbor Salmonella in their intestine, these birds often gather in very large numbers at bird feeders. The growth of the "bird feeding industry" has promoted this behavior. Garden bird feeders can become so contaminated with feces that Salmonella contamination may grow to significant levels. If this is accompanied by other stresses such as bad weather or a food shortage, large numbers of these birds may develop Salmonellosis and die. Finches, house sparrows, and cowbirds appear to be especially at risk. Phage and genetic typing suggests that these passerines carry strains of *S. enterica* Typhimurium that are specifically adapted to songbirds. These infected birds may transmit infection to humans, either directly as a result of handling, or more commonly, as a result of exposure to domestic cats infected by preying on sick and moribund birds.

Key words: Salmonellosis; Salmonella; birds

Salmonella live within the intestinal tract of vertebrates. As with many infectious agents, when present in small numbers and confined to their appropriate location, they are harmless. However, under conditions where the resistance of their host is impaired, or when changes occur in their microbial environment, they may increase in numbers significantly, invade their host, and cause disease or death. Their significance lies in the fact that they are found relatively commonly in both mammals and birds and that they may cause significant disease in birds and mammals, including humans. An example that most people are familiar with is Salmonellosis in poultry. Salmonellosis is an important cause of disease and death in poultry and contaminated poultry products pose a significant health hazard to human. Salmonella also occur in wild birds where they can cause disease and death, or even spread from their avian hosts to domestic mammals and man. Although well recognized as an avian disease for over a hundred years, Salmonellosis qualifies as an emerging disease because its prevalence in wild bird populations appears to have increased greatly over the past 40 years as a result of increased artificial feeding by humans. Salmonellosis in wild birds thus occurs most commonly in those on a carnivorous or omnivorous diet, those that feed on the ground or on food subject to fecal contamination, or those that live or feed in contaminated water.

Identification and Classification of Salmonella

The Salmonella are a diverse group of Gram-negative enterobacteria. Their classification has changed in recent years. Thus, although originally considered to consist of a large number of many different species, current opinion now considers the avian and mammalian salmonella to

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1055-937X/04/1302-0002$30.00

doi:10.1053/j.saep.2004.01.008

consist of a single species called *Salmonella enterica*. *S. enterica* is, however, divided into at least 2300 serotypes or serovars (serologic varieties). These serotypes are identified on the basis of their antigenic structure and classified by the Kauffmann-White scheme. Thus, a specific Salmonella is denoted by numbers that correspond to specific antigens on the organism's capsule, cell wall, and flagella. Some of these serotypes, such as *S. enterica*, serotype Typhi, cause a specific disease, typhoid fever, in a specific host, humans. Others, such as *S. enterica*, serotype Typhimurium are considered to be nonhost specific since they are capable of infecting an enormous diversity of species, including cattle, pigs, rodents. and birds. Because Typhimurium is by far the most prevalent serotype of *S. enterica*, epidemiologic investigations of outbreaks requires discrimination of subtypes within this serotype. This discrimination uses stable genetic markers to identify biological variants (biovars or biotypes). The most widely employed method of identifying these biovars is phage-typing. Thus, biovars of Typhimurium differ in their susceptibility to a panel of bacteriophages. By using an appropriate testing panel, it has proved possible to identify more than 300 different definitive phage types (DT) within the Typhimurium serotype by determining their susceptibility to a set of bacteriophages.[1] These phage types are stable and can be used to follow the spread of a specific biovar or determine the host preferences of individual Typhimurium biovars.

There are additional techniques that assist in investigating the epidemiology of Salmonella outbreaks and increase the precision by which biovars can be identified.[2] For example, biovars may differ in their ability to ferment selected carbohydrates or in their antibiotic-sensitivity patterns. Another commonly used technique is the use of plasmid profiles. In this technique, the plasmids present in different isolates can be separated by electrophoresis and compared.

One technique that has been proven to be most effective in identifying unique Salmonella biovars is pulsed-field gel electrophoresis (PFGE). This is a method used to separate fragments of DNA as long as several million bases by subjecting the gel to an electrical current alternately delivered from two angles in timed intervals. The technique minimizes the diffusion of these large molecules. PFGE is used to separate chromosomal fragments produced by enzymatic digestion of intact bacterial genomic DNA. To do this, bacterial chromosomal DNA is digested using restriction endonucleases that have relatively few restriction sites. This produces 10 to 20 DNA fragments ranging in size from 40 to 4000 kb that can be identified on electrophoresis and the genetic relationships of strains can be determined by this method.

When large numbers of isolates of Typhimurium are carefully typed, it is possible to show that certain biotypes are associated with disease in specific species. For example, Typhimurium biotypes isolated from pigeons commonly lack the O5 antigen and are often of phage types DT2 or DT 99. These pigeon isolates also are weak fermenters of rhamnose.[3] Likewise, many poultry and duck isolates commonly belong to phage types DT13 and DT14. These usually lack fimbriae and do not ferment either inositol or rhamnose (FIRN strains). As will be described in a later section, finch and sparrow isolates often belong to phage type DT40.

Routes of Salmonella Exposure

As enteric bacteria, Salmonella are found within the intestine. They spread to other individuals either because the intestine is eaten, for example by a predator, or alternatively, because they are shed in feces. Both types of spread, by carnivorism or by fecal contamination, are common among wild birds. Salmonella may be present in wild birds for two reasons. In one case, the organism is adapted to the host and establishes itself as part of the intestinal flora on a permanent basis. In this situation, typified by finch infections, one serotype tends to predominate. In a second situation, the Salmonella may be present in feces for a short time, as a result of environmental contamination. In this case, typified by gulls, no one serotype predominates reflecting the diverse array of organisms in the contaminated environment.

Salmonellosis in Specific Groups of Birds

Salmonella in Raptors

The simplest cycle of avian Salmonellosis is that which occurs in carnivorous or omnivorous wild birds that acquire Salmonella as a result of eating infected prey. For example, in a recent Spanish study,[4] it was found that 13/310 (4.19%) wild, free living raptors were positive for Salmonella. However, these birds were infected with many

different *Salm. enterica* serotypes including Enteritidis, Adelaide, Brandenburg, Newport, Typhimurium, Hadar, Saintpaul and Virchow. This pattern of multiple serotypes indicates that the infections were acquired from a wide variety of sources, as might be expected from eating infected prey. Avian carnivores consume both mammals and small bird,s so it is not clear just what is the relative importance of each. Salmonella serotype Amager has been isolated from peregrine falcons (*Falco peregrinus*) in Sweden.[5]

In North America, Kirkpatrick and Trexler-Myren examined raptors migrating through New Jersey and found only 2 out of 105 fecal samples positive for serotypes Enteritidis and Newport.[6] Both were from red-tailed hawks (*Buteo jamaicensis*). Nocturnal raptors such as owls may also acquire infection in this way. Thus, Kirkpatrick and Colvin,[7] also working in New Jersey, found 8/94 (8.5%) of barn owls (*Tyto alba*) to be Salmonella positive.

Captive raptors are also susceptible to Salmonellosis, especially when fed on live or freshly killed prey such as domestic pigeons.[8] Thus, captive peregrines and gyrfalcons (*Falco rusticolus*) have died as a result of septicemic Typhimurium infections. On necropsy, liver discoloration and enlargement was consistent and one bird had miliary abscesses in the liver consistent with infection entering via the portal route. Many of these captive birds also had pox or *Chlamydophila psittici* infection, and one was infected with a herpesvirus, raising the possibility that concomitant infection increases susceptibility to salmonellosis. In a large Spanish survey, 21/286 (7.36%) of captive raptors were Salmonella positive.[4] Most of these captive birds were infected with serotype Havana. An additional study showed that serotype Havana was also the predominant serotype isolated from raptors at a captive breeding center in Italy.[9] Additional biotyping and genetic typing of the Spanish Havana isolates suggested that they originated from a single source, most likely the poultry in the raptor's diet.[10]

It is to be expected that carrion-feeding birds such as vultures will also, from time to time, consume salmonella-infected carcases. Thus Winsor and coworkers[11] isolated Salmonella from 3 of 20 freshly killed turkey vultures (*Cathartes aura*) in Texas. Likewise, Asagi and coworkers isolated serotype Typhimurium var Copenhagen from 2/30 healthy crows (*C. corone* and *C. levaillantii*) in Japan.[12] These scavengers may also transmit the infection to other animals. For example, Watts and Wall investigated an outbreak of Salmonellosis in sheep in Australia where affected sheep had been isolated within one pasture for a very long time and no new animals had been introduced.[13] Serotype Typhimurium was, however, isolated from crows and magpies in the affected pasture and they surmised that the birds were the source of the infection. The weather had been hot and dry. Both birds and sheep had congregated at the limited sources of water. They suggested that the water supply had been fouled by bird feces In these studies it was shown that the birds readily picked up infection by feeding on infected sheep carcasses. Once infected, magpies and crows shed the Salmonella in their feces for up to 27 days.

Salmonella in Gulls

Gulls have increased greatly in numbers over the past 30 years in both North America and Europe.[14] This increase is largely attributed to the increased availability of food as a result of human activity. Gulls of many species are abundant in areas where food scraps are available, especially around landfills, other garbage dumps, and sewage outlets. It is not surprising therefore that gulls can pick up Salmonellae during the course of their scavenging activities. The gull-Salmonella pathway is clearly of importance in maintaining Salmonella in the environment.

Many different surveys of salmonellosis have been conducted in seagulls. For example Quessy and Messier tested ring-billed gulls (*L. delawarensis*) in Quebec and found that 8.7% of cloacal swabs contained Salmonella[15] These were a mixture of many different serotypes with only 1 of 23 being Typhimurium. The presence of so many serotypes is, as pointed out above, consistent with infections acquired from many different sources and tends to support environmental contamination as a source of infection. Snoeyenbos and coworkers examined cloacal swabs from herring gulls (*L. argentatus*) in Massachusetts and found 10/405 to be positive for Salmonellae.[16] The isolates belonged to nine different serotypes. They also examined gull eggs to determine if vertical transmission was a factor but only found Salmonella in 1 of 80 examined.

Many surveys of gulls have been performed in continental Europe, especially on the abundant and ubiquitous black-headed gull (*L. ridibundus*). For example, Selbitz in Germany, found that 42/852 (4.9%) were positive for Salmonella and that Typhimurium was the commonest serotype.[17] Three major surveys have been undertaken in the

Czech republic. Thus Hubalek and coworkers found a 24.7% infection rate involving predominantly Typhimurium followed by Enteritidis, Panama, and Anatum.[18] Cizek found 4.2% of adult *L. ridibundus* and 19.2% of fledglings to be infected.[19] Typhimurium DT 101 was the most common serotype. Literak and coworkers found 20% to 36% in juvenile gulls. On one occasion, multiple different serotypes were isolated while on another, only serotypes Typhimurium and Derby were found.[20] They did not detect salmonella in 79 eggs. Palmgren and her colleagues trapped 50 gulls in the center of Malmo, Sweden and found. 2/41 (4.9%) of adult black-headed gulls and 0/9 common gulls were positive for serotype Typhimurium.[21] Nielsen found two dead black-headed gulls positive for serotype Typhimurium at a Copenhagen cannery. He subsequently shot and examined 249 healthy gulls and isolated Typhimurium from 4 of them (1.6%).[22] As a result of gulls ingesting contaminated material, they also shed contaminated feces. Thus Muller found 78% of gull feces in the Hamburg sewage works to contain Salmonellae.[23] As might be expected, these reflected a very broad array of serotypes with Paratyphi B predominating.

British investigators have also studied Salmonellosis in gulls. For example, 22.2% of herring gull fecal samples in Wales were positive for Salmonella. These were a diverse mixture of many different serotypes.[24] Butterfield and coworkers studied Salmonella in herring gulls in the northeast of England.[25] They found that it increased in prevalence from 2.1% in 1975-76 to 8.4% in 1979. The serotypes isolated were very diverse with Typhimurium the most prevalent followed by Hadar and Heidelberg. They also noted that the proportion of positive birds was much higher in the first year birds (9.7%) than in four year birds (2%) and suggested that this was due to higher numbers of younger birds feeding along the coast. (It could also be a result of increased mortality among infected young birds so that positive birds are selectively eliminated. Salmonella-positive birds, however, appeared to be completely healthy). MacDonald and Brown found Salmonella in 6% (5/83) of gulls submitted for diagnosis in the United Kingdom.[26] These were from five different species and had been found dead or sick. They noted, however, that although these birds contained high numbers of bacteria, they had few obvious lesions of septicemia and speculated that the Salmonella bacteremia was a terminal event in gulls dying from other causes. As with Butterfield's study, most of these cases were in birds less than one year old. MacDonald and Brown also examined 70 healthy adult breeding gulls and found no Salmonella. A finding that supports the suggestion that Salmonella infections are primarily a problem in young gulls. More recently, Fenlon found Salmonella in 12.9% of 1242 fecal samples from gull flocks in Scotland.[27] He found that the number of positive samples was significantly higher (17-21%) near sewage outfalls. He isolated 27 different serotypes with the predominant serotypes being Muenchen, Heidelberg, San Diego and Derby. Given that the range and frequency of these serotypes was similar to that found in humans, Fenlon reasoned that sewage was a likely source of the infection in these birds. Fenlon also noted that the numbers of Salmonella in gull feces were relatively low (0.18-191 per gram). This was similar to the concentrations found in sewage and suggested that the gulls may simply be physical carriers. A similar result was obtained by Fricker who examined the secretion of salmonella from black-headed gulls feeding at a sewage treatment works.[28] Thus similar serotypes were found in gull feces and in the sewage sludge. He showed that gulls became infected after feeding on contaminated sludge. Interestingly, a rare serotype, Takoradi, appeared in the sludge for short periods and subsequently, appeared in gull feces for a few days. Fricker suggested therefore that the infection in gulls was likely short-lived (at least for this serotype). Despite the fact that healthy adult gulls appear to be asymptomatic carriers, occasional lethal Salmonella outbreaks can occur in gull colonies. One such outbreak occurred in ring-billed gulls in Quebec.[29]

Salmonellosis in gulls is not confined to the northern hemisphere. Thus Robinson and Daniel studied healthy gulls and other birds captured around a "meatworks" and a sewage works in Auckland as well as birds captured at a remote beach in New Zealand.[30] They found only two Salmonella-positive birds (2/43), both shags, (*Phalacrocorax* spp.) at the sewage works. However there were 8 positive gulls (8/44) found close to the meatworks. None of the gulls (0/24) from the remote beach were positive for Salmonella. The positive birds, two kelp gulls (*L dominicanus*), and six red-billed gulls (*L novaehollandiae*) likely contracted the infection as a result of scavenging near the meatworks. Salmonella has been isolated from birds such as gentoo penguins (*Pygoscelis papua*) and black-browed albatross (*Diomedea melanophrys*)

in the sub-Antarctic.[31] This may have derived from waste from scientific stations since multiple serotypes were isolated, a pattern that conforms to the sewage/scavenger cycle of gulls.

Because of their habit of congregating in large numbers at landfill sites and on agricultural land, gulls represent a potential source of Salmonella infection for humans and livestock. Many investigators have tried to prove a relationship between gulls and Salmonellosis in farm animals but with mixed results. For example, in one study, feces were cultured from gulls roosting near a cattle pasture. The unusual serotype Livingstone was isolated from both gulls and cattle and suggested that the gulls may have transmitted the infection to cattle. However, six other Salmonella serotypes were also isolated from gull feces on this farm but were not present in the cattle.[32] Coulson and coworkers have suggested that herring gulls may be a vector of Salmonellosis to sheep and cattle in the United Kingdom.[33] They based this on the remarkable overlap of gull breeding areas with the location of the Salmonella outbreaks as well as isolation of the same organism (serotype Montevideo) from both livestock and gulls. They also pointed out the coincidence of timing of cattle disease outbreaks with the movement of gulls from their wintering areas to their breeding areas as well as the use of agricultural land as feeding areas by herring gulls. The role of gulls as vectors of Salmonella is also supported by the occurrence of serotype Montevideo outbreaks in sheep on small isolated islands. Johnston and coworkers, investigated an outbreak of Salmonellosis in cattle in Scotland.[34] In this study, cattle grazing beside a lake suffered from Salmonellosis and the same serotypes were isolated from gull feces at the lakeside. The water for the cattle came from the lake. As in so many of these cases however cause and effect are difficult to disentangle. The gulls may well have acquired the infection from the cattle! Benton and coworkers described how a large gull roosting site contaminated a large water supply reservoir near Glasgow, Scotland, with both coliforms and Salmonella. Roosting was discouraged by broadcasting gull distress calls, and bacterial numbers in the water then dropped to safe levels.[35]

Salmonella in Waterfowl

Salmonellosis might well be expected to be a problem in waterfowl, especially when they congregate in large numbers during the winter months. Indeed, the recent growth in Canada goose numbers and their ability to foul waterside parks in North America has raised concerns that these might be a significant source of human pathogens such as Salmonella. However, waterfowl do not scavenge carrion in the way that gulls do, and there is no data to suggest that grazing geese are a significant source of Salmonella. On the other hand, waterfowl living in contaminated waters may become infected with Salmonella, especially if sewage contamination has occurred.[36] Mitchell and Ridgwell examined 477 duck fecal samples from a reservoir in London during the winters of 1969 and 1970.[37] They found that 20 (4.11%) contained Salmonella. The duck species involved included pochard (*Anas ferina*), Eurasian teal (*A. crecca*), and tufted duck (*Aythya fuligula*). There was little difference in the carrier rate between these species. The commonest serotype was Typhimurium. Salmonellosis has occasionally been a problem in domestic mallards (*A. platyrhynchos*). The serotypes of Typhimurium isolated from these birds appear to be different than those found in other wild birds. Thus the predominant mallard isolates in Germany between 1974 and 1996 have generally been DT8 and DT46.[38] DT46 tended to predominate in the 1980s while DT8 predominated in the 1990s, suggesting that there may have been two successive epidemics in these birds. Keymer reported an isolated case of disease due to serotype Typhimurium in a tufted duck.[39] Nielsen investigated an outbreak of salmonellosis in mallards raised for hunting and concluded that they acquired infection from wild birds, and possibly via vertical transmission.[22] Muller in Hamburg found 16% of duck feces contained Salmonella, with Typhimurium predominating.[23] Hubalek reported one isolate from a graylag goose and one from a coot.[18] Salmonellosis is not uncommon among commercial duck flocks. Indeed it has been recognized since 1920 and even was known by the colloquial name "keel." Price and coworkers reported 491 isolations from 7029 duck accessions from the commercial duck flocks on Long Island in 10 years[40] The predominant isolate (93%) was Typhimurium. Despite its name, serotype Anatum only accounted for 1% of cases. Lesions include necrotic foci in the liver, cheesy plugs in the caeca, and pale kidneys.

Salmonella in Pigeons

Salmonellosis is a common disease of domestic rock doves (*Columba livia*) where *S. enterica* causes "Avian Paratyphoid." This is generally a subclinical infection in adult doves but an acute lethal

disease in squabs. The only significant serotype involved is Typhimurium var Copenhagen.[41,42] The pathologic lesions found in diseased birds include swelling of the spleen, liver and kidneys. Necrotic foci may be found in the spleen, liver and lungs. The infection, once it gains access to a loft, is apparently maintained within the population by contaminated feces. Muller detected Salmonella in 30% of pigeon feces.[23] Faddoul found salmonella in 25% of submissions of domestic pigeons in Massachussetts.[43] Typhimurium, var Copenhagen was identified in 15/17 cases. These pigeon strains differ from other Typhimurium biotypes in that they do not ferment maltose whereas the chicken and turkey isolates obtained by Faddoul, were maltose fermenters. This suggested that there is a "pigeon type" of Typhimurium and that rock doves are not a source of infection for mammals or poultry.

Salmonella in Other Dense Bird Colonies

When birds gather in very large numbers within a restricted area so that massive fecal contamination occurs, then Salmonella numbers may build up to such an extent that they cause significant mortality. This process applies in wild birds just as much as in poultry houses. An excellent example of this was seen in 1995 to 1997 in the Izumi area of Japan where thousands of hooded cranes (*Grus monacha*) gather over the winter. Thus Maeda and coworkers found Salmonella in 29/420 fecal samples taken from these crane roost sites in late winter.[43] All were serotype Typhimurium and all appeared identical by plasmid profile analysis and antibiotic sensitivity. Since 80% of the world's population of hooded crane winter in this area, the potential for serious losses as a result of Salmonellosis is clear. Sporadic Salmonella cases occur in other crane species such as serotype Enteritidis in a whooping crane (*Grus americana*).[44] Other colonial waterbirds such as herons and egrets, especially when nesting in dense colonies may also experience salmonella outbreaks.[45] In 1999, Salmonellosis occurred in a colony of double-crested cormorants in Alberta, Canada.[46] Both mesogenic avian paramyxovirus 1 (Newcastle disease virus) and serovar Typhimurium were isolated from these birds. It is unclear what the initiating pathogen was in this case but it points out, yet again, that Typhimurium pathogenicity may be significantly increased by concurrent virus infections. In 2000 an outbreak of Salmonellosis occurred in a colony of black skimmers (*Rynchops niger*) in Texas. (W. Wigle, unpublished observations). The affected skimmers came from a very dense colony of these ground-nesting birds. Skimmer chicks began dying in large numbers and investigation showed Typhimurium to be the cause. It is likely that this is an example of dense bird populations leading to salmonella build-up in feces although the fresh water supply to the colony was a possible source. The water supply was limited and a popular bathing site for many other gulls and terns in the area.

Salmonella in Passerines

Although the avian Salmonella infections described above are significant, their importance is dwarfed by the occurrence of explosive outbreaks of this infection in small passerines attracted to bird feeders. These outbreaks not only kill very large numbers of wild birds, but may be significant sources of infection for humans and domestic animals.

Salmonella in Healthy Songbirds

It is essential to note the great differences in prevalence obtained by sampling apparently healthy songbirds as compared with examining moribund or dead birds. For example, Tizard, Fish, and Harmeson isolated serotype Typhimurium from 15% of healthy house sparrows in Ontario while Woebeser and Finlayson isolated the same organism from 90% of sick and dead sparrows submitted for necropsy from the same region.[47,48]

Many different surveys have been conducted to determine the level of carriage of Salmonellas in healthy songbirds. In North America, *S. enterica* serotype Typhimurium was isolated from the liver of 9/60 healthy house sparrows (*Passer domesticus*) trapped by mist netting in a garden in Ontario.[47] The phage types involved were DT 24, 40, and 160. On the other hand, Brittingham and coworkers were unable to isolate any Salmonellas from 364 healthy passerines and woodpeckers in Wisconsin.[49] Most of these samples (290) were obtained from black-capped chickadees (*Parus atricapillus*) captured using mist nets in wooded rural areas. In another survey, Morishita and coworkers sampled the cloacas of 1709 healthy passerines in Ohio.[50] They isolated Salmonella from 4/373 (1.07%) house sparrows and 62/868 (7.1%) of European starlings (*Sturnus vulgaris*). All other birds captured (mainly finch species and American robins) tested negative.

In Europe, Pinowska and coworkers examined 1124 male house sparrows from farms in northern Poland.[51] They found remarkable variation in the Salmonella prevalence rate. Thus on 4 farms, 20% to 50% of the birds tested positive; on 6 farms the prevalence was between 1.8% and 13%; and on 33 farms none of 660 birds tested positive. The predominant serotype isolated was Typhimurium followed by Dublin and Paratyphi. Passerine Salmonellosis has been well studied in Scandinavia. Thus Refsum and her colleagues sampled 1990 birds (mainly finches) captured at feeders in Norway during the winter of 1999 to 2000 and isolated serotype Typhimurium from 2% of them.[52] On the other hand, Hernandez and coworkers sampled 2377 actively migrating birds at different times of the year at a coastal banding station in southeast Sweden.[53] They isolated only one Salmonella, serotype Schleissheim, from a mistle thrush (*Turdus viscivorus*). This is a rare serotype previously only reported from Turkey. This is in agreement with an earlier study where Palmgren and her colleagues failed to isolate any Salmonella from 101 passerines captured immediately on arrival in Sweden during spring migration.[21]

It may be argued that sampling birds at farms where infected livestock are present results in a significant overestimation of Salmonella prevalence. For example, Cizek and colleagues isolated Salmonella from 7 house sparrows and 1 serin (*Serinus serin*) out of 31 birds examined on a farm where salmonellosis was occurring in calves.[19] All were serotype Typhimurium var Copenhagen. However, on farms where no livestock Salmonellosis was occurring, they only found Salmonella in 2 rock doves out of 2186 birds examined. No positive cases were found in 557 birds living in reed beds or 116 birds from military training areas. Although it might be expected that song birds living in and around sewage treatment plants may have an increased prevalence of Salmonellosis, such does not appear to be the case. Thus Plant found only 1 positive isolate among the feces of 599 birds captured at two sewage treatment plants in south-west England.[54] It is interesting to note that among the birds tested by Plant and found negative for Salmonella were 153 house sparrows, 20 greenfinch, and 14 redpoll. Goodchild and Tucker, also in England, took cloacal swabs from 511 apparently normal wild birds and recovered salmonellae from 2 house sparrows (0.39%).[55]

Sharma and coworkers, examined the intestinal contents of 799 wild birds in India and isolated Salmonella from 20 of these birds.[56] The serotypes involved included Saint Paul, Bareilly, Weltevrede, and Typhimurium. Bird species affected included 7/250 house sparrows, 1/30 "swallows," 1/1 gray partridge, 1/1 "parrot" and 9/100 "mynahs." Unfortunately it is unclear from this paper whether these were healthy birds or sick/dead birds submitted for necropsy.

With few exceptions therefore, it appears that the prevalence of Salmonella in healthy, wild migrating bird populations is low. This seems to be especially the case in birds that reside away from sites of human activity such as farms or sewage plants or which are not attracted to bird feeders.

Salmonella in Sick/Dead Songbirds

While the "background" infection rate of Salmonella in passerines is relatively low, it is not zero. As a result, major epidemics of this disease may occur under certain circumstances. As might be expected, wild bird mortality usually only comes to our attention when humans encounter significant numbers of carcasses and these carcasses are submitted to a diagnostic laboratory. Mortality occurring away from humans is much less likely to be noticed and investigated. Thus even the idea that bird mortality is associated with human contact, may well be, in part, an artifact of the way we encounter sick birds.

Given the attention that large numbers of sick, moribund, or dead songbirds attract, it is not surprising that many investigators have reported the prevalence of Salmonellosis in sick birds. It was only in 1957, however, in the northeastern United States, that Hudson and Tudor described several cases of salmonellosis in house sparrows, starlings, brown-headed cowbirds (*Molothus ater*), and rusty blackbirds (*Euphagus carolinus*) due to serotype Typhimurium.[57] These authors claimed that it was the first time this disease had been reported in wild birds, and implied that this was a new phenomenon. They suggested that the outbreaks were related to the birds concentrating at feeders during the winter.

Faddoul surveyed wild birds submitted to a diagnostic laboratory over a 1-year period in 1964 to 1965, in Massachusetts and Rhode Island.[58] He isolated Salmonella from 12 of 100 submissions. Eight of these isolations were from cowbirds, two from house sparrows and one each from a white-throated sparrow (*Zonotrichia albicollis*) and a herring gull. Locke, in 1973. described evening grosbeak (*Coccothraustes vespertinus*) and pine siskin (*Carduelis pinus*) deaths in Maryland and West

Virginia due to serotype Typhimurium.[59] Hudson and coworkers investigated the relationships between 22 Salmonella isolates from wild birds in the southeastern United States by the use of PFGE.[60] These fell into nine geographically restricted patterns. All the cowbird isolates and all the house sparrow isolates were identical. On the other hand, the isolates obtained from other bird species were heterogeneous.

In Britain, Wilson and MacDonald reported on Salmonella isolations in wild birds between 1939 and 1959.[61] Salmonella isolations were relatively rare so that in those 20 years they only isolated serotype Typhimurium from feral pigeons, Eurasian greenfinches (*Carduelis chloris*), a tawny owl (*Strix aluco*), a hooded crow (*Corvus cornix*), a rook (*C. frugilegus*), a red-throated loon (*Colymbus stellatus*), a mallard (*Anas platyrhynchos*) and a mute swan (*Cygnus olor*). They also isolated serotype London from a red grouse (*Lagopus scoticus*). In total they had 9 isolates from 1573 birds of 74 different species, a prevalence of 0.6%. Subsequently, things changed as the general public began to actively feed wild birds. Thus between 1960 and 1966 the same investigators isolated serotype Typhimurium 19 times, 10 in house sparrows, 1 each from a feral pigeon, a herring gull, a wood pigeon (*Columba palumbus*), 2 gannets (*Sula bassanus*) and 4 greenfinch.[62] It is interesting to note that they did not detect Salmonella in house sparrows until after 1960. In 1968, also in Britain, Goodchild and Tucker examined 382 birds that had been found dead and isolated salmonella from 2.88% of these birds.[55] These included redpolls (*C. flammea*), house and tree sparrows, a lesser whitethroat, and a dunnock (*Prunella modularis*). Many were serotype Typhimurium and eight of the Typhimurium isolates were phage type U17, one was DT14, and one was untypable. They did not report whether these isolates came from a single outbreak, nor did they note any seasonal differences. In 1995, Routh and Sleeman isolated serovar Typhimurium from dead goldfinch in the United Kingdom.[63] The birds showed no clinical signs, and the outbreak began and ended very suddenly. Pennicot surveyed of wild bird mortality in Scotland in 1994 to 1995.[64] He found that in some areas, *E. coli* O86 caused mortality. In other areas Typhimurium DT40 was the major cause of greenfinch mortality being isolated in 89% of 56 birds. Many of these birds had concurrent fungal infections or coccidiosis. He also described deaths due to serotype Typhimurium DT40 in greenfinches and a chaffinch (*Fringilla coelebs*) in southern Scotland in 1997.[65] Sixty-five isolations of Typhimurium DT40 were reported to the British central veterinary laboratory between 1990 and 1996.[66] Of these, 11 involved finches, 19 were from pheasants or partridges (15 in 1 year), 13 from horses, 7 from ducks or geese, 4 from chickens, and 4 were from cattle.

In Norway, Refsum and coworkers reported on avian deaths from Salmonellosis between 1969 and 2000.[52] Salmonella were isolated from 470 birds belonging to 26 species, 54% of these cases were in bullfinch (*Pyrrhula pyrrhula*). Other important species affected were greenfinch, siskin and redpoll. The only serotype isolated was Typhimurium except for a single isolate of Paratyphi-B from a hooded crow (*Corvus corone*). Random phage typing of these isolates identified 54% DT40, 35% U277, 6% DT99 and 4% DT110. Of these isolates, 96% were typical Typhimurium while the remainder were var. Copenhagen. Plasmid profile analysis detected six distinct profiles of which two accounted for 94% of isolates. As these outbreaks of Salmonellosis developed, a seasonal pattern emerged with greatest mortality between December and March.

Fecal Surveys

Since Salmonella-infected birds shed these organisms in their feces, many investigators have surveyed avian fecal samples for the presence of these organisms. Thus, Craven and coworkers isolated Salmonellae in from 0% to 33% of "wild bird" feces located near broiler chicken houses in Georgia.[67] Pennicott and coworkers, 2002 surveyed bird feces around feeders in south-west Scotland for 12 months. At one site where large numbers of birds were being fed throughout the year, serotype Typhimurium was isolated from 42% to 48% of feces at feeders, and from 33% of feces below a house sparrow roost site. The phage type isolated was DT56. Pennicott also surveyed a second site where birds were fed only in the winter months.[65] Here, in contrast, he found only two positive fecal samples, both were Typhimurium DT41. Six dead birds were recovered from the first site but none from the second site. Prescott and coworkers examined fecal samples from 129 winter bird feeders in the city of Guelph, Ontario on five occasions over the winter of 1997 to 1998.[68] No Salmonellae were isolated.

Explosive Salmonella Outbreaks

Outbreaks in Britain

As pointed out above, Wilson and MacDonald noted that, beginning in 1960, the frequency with which they isolated Salmonella from wild birds increased significantly.[61] Indeed, during the 1960s they identified three major outbreaks of salmonellosis in greenfinch and one in house sparrows. The greenfinch outbreaks involved large populations of birds attracted to feeders by abundant seed. The first epidemic lasted 6 weeks beginning in February 1956 and at its peak 20 to 30 birds died daily. It is interesting to note, however, that during this outbreak, both house sparrows and chaffinches fed beside the greenfinch but none were visibly affected. Other disease outbreaks occurred in April 1964 and April 1966 under circumstances that were similar to the first outbreak. They also reported on a disease outbreak that occurred in February 1965 where mortality was confined to house sparrows on a farm. Yet another major outbreak occurred in the south of England in 1967 and 1968 at a time when greenfinch numbers had recovered from a severe winter in 1962 to 1963.[61] Thus between January and April 1967, Typhimurium U218 caused multiple deaths in greenfinch and house sparrows. It broke out again in December 1967 with the same phage type also affecting sparrows and greenfinch. In January 1968, multiple outbreaks were in greenfinch and house sparrows occurring across southern England with four phage types, U165, U218, U239 and U 19. Toward the end of this time, the disease spread to dunnocks. Dunnocks are ground- feeding solitary birds that are attracted to seeds that accumulate under feeders and where fecal contamination may be high.

Salmonellosis has now become an annual epidemic in British songbirds affecting greenfinch, chaffinch, goldfinch, siskins, house sparrows and in 2001, tree sparrows (*P. montanus*). It has been speculated that Salmonellosis is an important contributory factor to the decline of these species in the United Kingdom. Thus woodland species have declined by 20% while farmland species have declined by 40% since the mid 1970s.[69] Three species known to be susceptible to Salmonellosis, redpoll, house sparrow and tree sparrow have declined by 92%, 58% and 87% respectively.[70] While it is premature to ascribe these declines to Salmonellosis, it is reasonable to assume that it has contributed to the problem.

Outbreaks in North America

Multiple Salmonella outbreaks have been described in North America, to such an extent that large numbers of wild birds die from this infection each winter somewhere in the United Sates. For example, the 1998 epidemic affected 16 states in the midwestern and northeastern United States and mainly affected common redpoll, pine siskins and goldfinches. The 1999 epidemic affected Michigan, North Carolina, Texas, Washington, Texas, Wisconsin and West Virginia, and mainly killed pine siskins and common redpolls. The 2000 epidemic affected Illinois, Georgia, Michigan, New York, North Carolina, Vermont, Virginia, and West Virginia and also killed pine siskins and common redpoll. In 2001, the states affected included California, Texas, Wisconsin, Georgia, Mississippi, and Oregon while the species involved included house sparrows, double-crested cormorants, white-throated sparrows (*Zonotrichia albicollis*), cowbirds and pine siskins.

In the winter of 1992 to 1993 salmonellosis was reported in pine siskins, evening grosbeaks (*Coccothraustes vespertinus*), house sparrows and purple finches (*C. purpureus*) in western North America ranging along the Pacific coast from British Columbia to California.[71] That winter had been characterized by unusually high rain and high snow levels and these may have driven the birds to feeders in unusually high numbers. In the winter and spring of 1994, an outbreak of Salmonellosis occurred on the eastern edge of the Rocky Mountains in Colorado. In the winter of 1997 to 1998, Salmonellosis affected a large number of songbirds in northeastern North America. The epidemic was well studied in the Atlantic Provinces of Canada.[71] Thus between December 1997 and August 1998, 73 cases of Salmonellosis were recorded in songbirds. (In the previous 10 years no more than 2-3 cases a year had been reported). Species affected in order of importance included common redpoll (*Carduelis flammea*), pine siskins, purple finches, evening grosbeaks, and American goldfinch (*Carduelis tristis*). Twenty of the 35 isolates characterized were identified as serotype Typhimurium DT40. Another eight lacked the O5 antigen and so were classified as Typhimurium, var Copenhagen also of phage type DT40. During the same period, Prescott and his colleagues identified Typhimurium DT40 as the predominant isolate from affected winter finches in Ontario.[72] They suggested that Typhimurium DT160 caused a sporadic winter bird infection limited to house sparrows. They also suggested that Typhimurium

DT 40 may have become adapted to wintering flocking songbirds.

Outbreaks in Scandinavia

In the winter of 1999 to 2000, septicemic Salmonellosis affected large numbers of small passerines in Norway.[51] Affected species included bullfinch (*Pyrrhula pyrrhula*), Eurasian siskin (*Carduelis spinus*), common redpoll and Eurasian greenfinch. Serotype Typhimurium was isolated in 64% of 145 birds examined. Deaths occurred at feeding sites from December to June with peak mortality during February and March.

Perhaps the clearest indication of the epidemiologic circumstances underlying outbreaks of Salmonellosis in passerines comes from the 1999 outbreak in Sweden.[73] This epidemic likely originated as a result of the failure to harvest large quantities of linseed in the summer of 1998. As a result of a cold, rainy summer in 1998, only 20% of 32,000 acres of linseed was harvested. Consequently, approximately 24,000 tons of linseed remained in the fields and served as a food source for finches in the autumn of 1998. The presence of these seeds together with an increased acreage of weedy fallow land permitted an unusually large number of birds to survive during the autumn and early winter. The disease developed within these enormous flocks, not when they fed in the fields, but when they congregated at garden feeders later that winter. Sick birds from these flocks, redpolls and siskin may have spread the infection to birds at garden feeders. The disease outbreak peaked in February 1999. The only serovar isolated was Typhimurium.

Outbreaks in New Zealand

In the winter and spring months of 2000 a major outbreak of Salmonellosis due to serotype Typhimurium DT160 occurred in New Zealand.[74] It caused extensive mortality in some passerines and the press reported "thousands" of dead house sparrows. Sparrows accounted for about 95% of cases with occasional cases among greenfinch, Eurasian goldfinch and blackbirds (*Turdus merula*). Central and northern areas of the South Island and the southern North Island were affected. Mortalities reached a peak in winter (July-August) and then decreased in spring and early summer. The high mortalities were most obvious in areas around grain silos. The importance of this outbreak is that it spread to humans and livestock. There were 80 associated human cases associated with hand feeding. There is also concern that the disease may spread to involve endangered indigenous species. The only significant mortality reported in native species was in silvereye (*Zosterops lateralis*) when 20 birds were found dead near Christchurch. Two sulfur-crested cockatoos (*Cocatua galerita*), a species that is not native to New Zealand, ate infected sparrows and became infected themselves. There was also at least one Salmonella death in a captive kaka, (*Nestor meridionalis*).

Pathology of Songbird Salmonellosis

The clinical signs of Salmonellosis in songbirds are brief, lasting for only 2 to 3 hours.[75] Affected sparrows are reluctant to fly and appear disorientated. They then collapse and die quickly. Birds that survive for 24 hours fluff their feathers and are reluctant to move. Diarrhea is not a feature of this disease but some birds have difficulty in swallowing as a result of their esophageal lesions. Weight loss is highly variable, some authors report that affected birds tend to be in poor body condition with a loss of body fat and pectoral muscle atrophy, while in other outbreaks such as the New Zealand epidemic, the dead birds may be in good condition. This likely depends on the speed at which affected birds die. In very acute disease death may occur in the absence of obvious gross lesions.

On necropsy, the most prominent lesions consist of an ingluvitis or esophagitis. Birds have characteristic multifocal areas of caseous necrosis and a fibrinopurulent exudation in the crop or esophagus. Thus in the New Zealand outbreak 40% of birds necropsied had crop lesions.[74] MacDonald and Cornelius also commented on the presence of multiple ulcers 1 to 2 mm in diameter in the lower part of the esophagus in about half of their greenfinch cases.[76] These ulcers can coalesce to form a large necrotic mass blocking the thoracic inlet. Hudson considered the characteristic lesions in passerines to be the necrotic plaques in esophagus and crop.[54] Kirkwood and coworkers, suggested that the lesions observed in greenfinch-(yellow nodules in crop walls), differed consistently from the lesions observed in house sparrows (diffuse crop lesions).[75] Wilson and Macdonald also described esophageal ulceration while Pennycott described multiple coalescing yellow nodules 1 to 4 mm in diameter in the mucosa of the esophagus that sometimes caused partial obstruction.[61,64] The ingluvitis and esophagitis associated with salmonellosis in songbirds is consistent and raises the question whether the crop and

esophagus represents a predilection site for this organism. It is possible that the organisms may be carried in these organs and cause opportunistic disease only when the animals are stressed for some reason. A few birds may have enteric lesions, but this is variable. Some have no obvious intestinal lesions while others may show necrosis of the large intestinal and cloacal mucosa. Many birds have hepatic lesions in the form of a swollen liver, with multiple areas of focal necrosis. Affected birds may also have pale, enlarged spleens. Small areas of necrosis are commonly seen in the liver, spleen and pectoral muscles. The brain may also be affected with lesions varying from areas of caseous necrosis to a mild mononuclear infiltration. It is also not uncommon to observe distention of the infraorbital sinuses as a result of inflammatory exudation.[29] Septicemia leading to disseminated intravascular coagulation and impaired blood coagulation may result in hemorrhage into the air spaces of the skull. Microscopic lesions consist of multifocal aggregates of heterophils mixed with fibrin and bacteria surrounded by necrotic cells. Salmonella organisms can be isolated in large numbers from most body organs including the crop, intestine, liver myocardium, and spleen implying that septicemia is the cause of death.

The lesions in cowbirds may be different from those in other passerines. Thus Faddoul described the lesions seen in cowbirds as swollen, congested livers and spleens, congested lungs, and abscesses in the brain or abdominal cavity.[43] This is consistent with our findings in diseased cowbirds in Texas.[77] These lesions in passerines are also distinctly different from Salmonellosis lesions in domestic poultry and rock doves where lesions in the lower GI tract are common and necrotic esophagitis has not been reported. Likewise, salmonellosis commonly affects adult wild birds while in chickens, young birds 1 to 7 days old are most commonly vulnerable.

The Role of Bird Feeders in Passerine Salmonellosis

The feeding of songbirds over the winter months, while always popular has become a very significant source of food for these birds. Since the 1960s, feeding of high-energy seeds, especially peanuts and sunflower seeds has increasingly attracted very large concentrations of songbirds to feeders. These birds in turn deposit relatively large quantities of feces in and around feeding sites. These feces may contain Salmonella and can thus spread to feeding birds leading to explosive outbreaks of Salmonellosis. (One advantage of being a bird is that they normally fly away from sites where they deposit their feces. By returning frequently to sites of massive fecal contamination, this advantage is lost.) Many investigators, such as for example, Kirkwood and Stroud and Friend have therefore suggested that these winter outbreaks of salmonellosis are a direct result of the accumulation of large populations and high densities of seed-eating birds at garden feeders.[78,79] This is, however, difficult to prove. Thus birds at feeders are more commonly observed by home owners and morbidity and mortality are correspondingly more likely to be observed. There is no data to confirm that deaths occur more commonly in birds in the vicinity of feeders rather than elsewhere. It is also abundantly clear that dead birds are very rapidly destroyed by scavengers so that the number of dead birds found certainly represents only a very small fraction of the population. Kirkwood provided data to suggest that deaths from infectious diseases occurred at a significantly greater rate near feeders where abundant food was provided as compared with feeders where there was less food.[78] Pennycott suggested that three conditions had to be met if a Salmonellosis outbreak were to occur.[64] First, the presence in a bird population of the appropriate species-adapted phage type of Typhimurium. Second, a large dense population of birds such that the organism can readily spread to new susceptible hosts. And third, a stress factor such as a sudden shortage of food, cold weather, or simply, a build up of the organisms to critical numbers.

The key to controlling Salmonellosis at bird feeders is therefore to avoid attracting overwhelming numbers of birds to feeders and so preventing the build-up of contaminated feces. The build-up of bird numbers tends to occur when feed is supplied in over-abundance. Reducing the amount of feed put out daily will restrict bird numbers without driving them away completely, Given that this build-up occurs over time, the simplest preventative may be to stop feeding birds in spring and summer when natural food sources are plentiful. It is also essential that feeders be kept as clean as possible. Bird lovers should use feeders with a design that minimizes fecal contamination and keep seed off the ground. Platform feeders that accommodate large numbers of birds at one time are most likely to become contami-

nated. It is also helpful to provide several feeders so that birds do not compete aggressively for the same food. It is also important to remove feces as it builds up. For example, bird feeders should be thoroughly cleaned and disinfected every 2 weeks. This should be done in a bucket rather than the kitchen sink. Feeders should be dismantled and scrubbed with a brush to ensure that bacteria do not persist in crevices and corners. Wooden feeders or feeders with wooden parts should be avoided since they are almost impossible to clean properly. They should be thoroughly cleaned in soap and water and then soaked in a 10% household bleach solution for 20 minutes followed by careful thorough rinsing with clean water. Reassemble and fill the feeders only after they are completely dry. Another solution is to use low-cost, recyclable feeders that can be discarded frequently. Bird baths are another potential source of infection. These should also be washed, bleached and rinsed at least once a week. Bird lovers should also be reminded that this infection may be transmissible to humans. Thus they should wear rubber gloves when cleaning feeders and bird baths. Rubber gloves should also be worn when handling dead birds and discarded with appropriate precautions. Hands should always be thoroughly washed in soapy water after handling or filling bird feeders.

Predisposing Causes of Outbreaks

During the Canadian epidemic of 1997 to 1998, it was noted that affected birds were in poor body condition.[71] It was not possible, however, to determine whether this poor condition was a predisposing factor for disease or a result of the disease. Food supplies through the winter of 1997 to 1998 appeared to be adequate, and tree seeds appeared to be available in normal quantities. As pointed out above, high concentration of songbirds at feeders appears to be a predisposing cause of many outbreaks. However, the Canadian outbreak continued well into the summer when much artificial feeding would have been discontinued. Indeed, during the 1997 to 1998 outbreak, mortality was seen not only in the Canadian Atlantic Provinces but also westward for hundreds of miles to Quebec and Ontario. This is a very wide geographical range and much wider than could be explained by local food shortages. Bacterial contamination of commercial seeds may explain some local outbreaks. Thus, if stored seeds have been contaminated by rodent feces, then this may, in theory, spread infection to birds. However, during the 1993 outbreak of Salmonellosis in birds in British Columbia, no Salmonella were isolated from several unopened bird seed packages.

Cowbirds as Reservoirs of Salmonellosis

Before European settlement, brown-headed cowbirds (*Molothrus ater*) were largely restricted to the great plains of North America where they associated with the buffalo herds. Clearance of forests has enabled cowbirds to spread eastward so that they are now found across the eastern United States and Canada. These birds are brood parasites and disperse in the summer. In the winter months, however, they gather in very large flocks. These flocks tend to congregate in areas where cattle are fed since they are attracted to creep feeders, large troughs placed in open fields and filled with grain. It is not uncommon to observe very large cowbird flocks swarming over these feeders as soon as they are filled. Thus the conditions necessary for Salmonella transmission are readily met. Studies in North America suggest that the cowbird is commonly affected by Salmonellosis. For example, in early 2001, a major outbreak of salmonellosis occurred in College Station and Houston, Texas.[77] It involved the presence on suburban streets of large numbers of sick and moribund cowbirds scattered at multiple sites. The severity of the outbreak and the presence of large numbers of dead and dying birds on the streets was such that it attracted media attention. Before the outbreak it was estimated that at least a million cowbirds roosted overnight at sites on the campus of Texas A&M University. Following the die-off in January-April 2001 cow bird numbers dropped to an estimated 600,000. The organisms isolated from birds on the campus were serotype Typhimurium, phage types u301 and DT 146. During this epidemic, serotype Typhimurium was also isolated from moribund and dead songbirds in four other locations, Fort Hood, Port Lavaca, Bandera, and Baton Rouge. The species affected included cowbirds, grackles (*Quiscalus mexicanus*), house sparrows, cardinals and black skimmers. The phage types involved were DT146, and U301. A similar disease outbreak among captive cowbirds at Fort Hood Texas in May of 2001 showed 3/4 of birds tested were infected by phage type DT146. The predisposing

causes of the 2001 Texas epidemic appeared to include unusually large and dense cowbird roosts containing, it was estimated, more than 1 million birds, and a hot spring season where water was limited.

Kirk and coworkers examined the prevalence of Salmonella in birds captured on dairy farms in California.[80] They isolated Salmonella from 2.5% of 892 birds sampled. It is interesting to note that the bird species with the highest prevalence were cowbirds 3.2% of 95 and house sparrows 3.1% of 451. In the other five species examined, only one bird in each was positive. It is also interesting to note that the serotypes isolated from the cowbirds were Meleagridis and Muenster. From the sparrows, the serotypes were Montevideo, Meleagridis, and Muenster. These are very different from the serotypes in sparrows from the northeastern United States and Europe and suggest multiple sources of infection. None of these serotypes, with the exception of Montevideo, are commonly found in dairy cattle and the authors concluded that these wild birds were not important sources of salmonellosis in cattle in this environment.

The importance of cowbirds as carriers of Salmonellosis was also seen in the survey by Radwan and Lampky, who surveyed wild birds in Michigan in 1970.[81] They examined the intestinal tract of 45 cowbirds and 11 other species. They found 11 Salmonella isolates in the cowbirds and four in various other species. The healthy cowbird isolates included the serotypes Typhimurium, paratyphi A, and paratyphi B. Snoeyenbos and coworkers examined several blackbird roosts in Massachusetts and cultured organs from healthy birds.[16] They found 11/299 cowbirds, 2/108 common grackles, and 13/148 European starlings to be positive. The vast majority of these isolates were Typhimurium.

Significance to Humans and Other Mammals

Salmonellosis in wild birds may be a source of infection for humans and domestic mammals. Indeed, many of the wild bird studies have been initiated to determine and quantitate this risk. For example, Tizard and Harmeson captured house sparrows at a large equine veterinary clinic where Salmonellosis was a recurring problem.[48] The sparrows were foraging in the horse feed and roosting in the rafters. They found 3/7 to be positive. Sparrows, of course, commonly frequent stables where grain is abundant. MacDonald and Bell pointed out that the Typhimurium phage types isolated from horses in the United Kingdom corresponded to those commonly isolated from greenfinch and house sparrows.[82] They also suggested that the pattern of salmonellosis seen in horses in the United Kingdom corresponded to the development of avian epidemics.

Cattle may also acquire infection from wild birds.[80] Thus, MacDonald and Bell pointed out that the 1968 outbreak in finches at bird feeders involved Typhimurium phage types, U218, U239 and U165.[82] Before 1967, these phage types had never been isolated from species other than wild birds. In 1967, U218 and U239 were isolated from pigs and sheep, respectively, and in 1970, U218 and U165 were isolated from turkeys and chicks. Human cases due to U218 and U165 occurred in 1972 and in one of these cases, the patient's cat is reported to have "caught a sparrow," while another had "handled" a small gull. The time sequence is suggestive.[19]

The New Zealand epidemic clearly spread to humans from songbirds.[74] Thus, Typhimurium DT160 had not been recorded in humans in New Zealand before 1998 or in domestic mammals before 2000. Yet there was a rapid rise in the frequency of these isolations as the epidemic progressed. These occurred in livestock (sheep, cattle horses and deer), domestic pets (cats and rabbits), and humans. Serotype Typhimurium has now been isolated from New Zealand poultry houses but has yet to cause confirmed disease. During 2000, 180 human cases of infection with DT160 were recorded and this was approximately 9% of the total cases of human Salmonellosis in New Zealand during that time. Interviews with patients suffering from Salmonellosis due to DT160 showed that direct handling of wild birds was a significant risk factor (13/119 patients handled wild birds while only 3/235 controls did so with an odds ratio of 12.28).[83]

In a much more direct example of bird to human transmission, Penfold and coworkers described an outbreak of Salmonella gastroenteritis in a hospital.[84] The food was prepared in a large, barn-like kitchen where house sparrows, flew freely in and out and roosted on the rafters. Some of these sparrows were trapped and tested for Salmonella. Serotype Typhimurium, phage type DT160 was isolated from both affected patients and sparrows. The problem disappeared once the kitchen was screened to exclude the sparrows. It

was assumed that sparrow droppings contaminated the patient's food.

Kapperud and coworkers have made a strong case that wild birds serve as a major reservoir for human serotype Typhimurium in Norway.[85] They analyzed sporadic human outbreaks in that country between 1966 and 1996. The human outbreaks were seasonal, with 78% of the cases occurring between January and April at a time corresponding to the annual avian outbreaks. The major epidemic strain isolated from these patients was identical to that encountered in wild birds and rarely encountered in other species. In a prospective case-control study, persons having direct contact with wild birds or their droppings clearly had an increased risk of infection. This was also true of an outbreak of Salmonellosis in 1987 that was originally believed to have originated in chocolate bars. This organism, however, also had the characteristic plasmid profile and phage type of wild bird strains. In addition, Refsum and coworkers examined the relationships between Norwegian isolates of Typhimurium from different sources by means of PFGE.[66] Eighty-five percent of the isolates fell into three main clusters that could be subdivided into 20 subclusters. Isolates from passerines, gulls, and pigeons clearly fell into five subclusters, while those from other, mammalian sources showed no predominance. There were two clusters restricted to passerines. However given that 32% of Norwegian human cases of Typhimurium also fall into these clusters, Refsum concluded that passerines constituted a major source of infection for humans in Norway while strains from gulls and pigeons were of much less significance.

"Songbird Fever"

Salmonellosis in pet cats is associated with eating wild songbirds and appears to be a regular springtime event in the northeastern United States.[86] It occurs at the time when cats predate on birds feeding at feeders. It is believed that hunting cats catch and eat moribund birds. The predominant serotype isolated from these cats is Typhimurium. Winter finches, especially the common redpoll appear to be the major avian source. The disease in cats is characterized by a 2- to 5-day incubation period. Affected cats show depression, anorexia, vomiting, and hemorrhagic enteritis plus a fever. The disease usually lasts for 2 to 7 days, Full recovery may take up to 3 weeks, and cats with impaired immunity may die. Treatment consists of isolation and supportive care. Antibiotic treatment is not recommended since they may affect the gut flora. There is obviously a potential for transmission to the cat's owner. Refsum's studies in Norway also provide evidence for the spread of Salmonella from birds to cats.[87] Thus they demonstrated the same PFGE patterns in Typhimurium isolates from cats as were found in small passerines.

Tauni and Osterlund investigated the Swedish outbreak in the winter of 1998 to 1999. They noted that in late February 1999 there was a marked increase in cats with fever and anorexia being presented at their veterinary clinic.[73] The cats were anorexic and lethargic, and many had vomiting and diarrhea. Overall, however, the clinical signs were nonspecific. Owners often informed the veterinarians that the cat had eaten a bird 1 to 2 days before the onset of disease. This timing corresponded, as described above, to the death of many small passerines in central Sweden as a result of Salmonellosis. Salmonella isolation was performed on 25 cats, and 20 of these yielded serotype Typhimurium. Eight of these 20 isolates were of phage type DT40, while the remainder were not typable. No similar cases in cats had been observed in this practice from 1994 to 1997 and there had been 5 cases in 1998. At the same time as this outbreak occurred in cats there was an outbreak of human Salmonellosis in the same area. There were two cases of Typhimurium phage type 29, 40 cases of Typhimurium phage type DT40, and 15 cases with untypable Typhimurium. Indeed, during the first 6 months of 1999, 57 of 242 domestically acquired human Salmonella cases in Sweden were caused by strains also found in wild birds and cats. In the previous 3 years, these strains accounted for 13/614 (2%), 11/585 (2%), and 44/452 (10%) of human cases, respectively. The evidence from phage typing suggests that the infection in humans likely originated in wild birds and was acquired by humans, either directly, or indirectly through cats.

Less direct evidence for bird human transmission has been reported by Heir and coworkers who used PFGE to analyze human Typhimurium strains in Norway.[88] They found that the strains characteristic of wild birds accounted for 32% of sporadic human cases. In addition, 76% of these cases occurred between January and April. On the other hand, pigeon and gull strains were very rarely detected in humans, although at least one small outbreak in humans was attributed to gulls

washing and roosting in a source of domestic water.

Conclusions

In conclusion, *Salmonella enterica* serovar Typhimurium is a relatively common infection in wild birds. Whether or not is causes significant disease likely depends on many different factors, but the most obvious are either the consumption of food containing this organism as occurs in raptors and in scavengers such as gulls, or alternatively, exposure to gross fecal contamination as occurs in house sparrows and winter finches at bird feeders. In the latter case, these circumstances together with other stress factors can lead to significant mortality. Under some circumstances, the infection can spread to domestic and wild animals. It is important to note however that the literature does not show evidence that wild birds are a significant source of infection for domestic poultry. Presumably, poultry houses, if properly constructed, should not admit significant numbers of wild birds.

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