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EPIDEMIOLOGIC AND PATHOLOGIC ASPECTS OF *SALMONELLA* TYPHIMURIUM INFECTION IN PASSERINE BIRDS IN NORWAY

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ABSTRACT: Septicemic salmonellosis caused by *Salmonella* Typhimurium 4, 12: i : 1, 2 was diagnosed in 94 (64.8%) of 145 small passerines comprising nine species, examined in Norway during 1999–2000. The birds were found dead at private feeding places throughout the country. The bullfinch (*Pyrrhula pyrrhula*), Eurasian siskin (*Carduelis spinus*), common redpoll (*Carduelis flammea*), and Eurasian greenfinch (*Carduelis chloris*) were the most frequently affected species. Pathologic findings in 94 carcasses included poor body condition (84%), enlarged spleen (73%), and necrosis of crop/esophagus (78%), liver (53%), spleen (46%), proventriculus (13%), and intestine (5.3%). Histologically, necrosis consisted of debris, fibrin, inflammatory cells, and aggregates of Gram-negative bacteria and occasionally giant cells. Based on information from questionnaires sick and dead birds were observed at feeding places from December to June, with a distinct peak during February and March. The duration of recorded outbreaks varied from less than 1 wk to 4 mo. In a separate study, 1,990 apparently healthy passerines caught at feeding places established for bird-ringing purposes were surveyed for cloacal carriage of *Salmonella* spp. Forty (2.0%) of the birds examined, representing sampling sites both in southern and northern parts of the country, harbored *S.* Typhimurium 4, 12: i : 1, 2 in their intestines. The carrier species largely reflected the species most often suffering from fatal infection.

Key words: Epidemiology, passerines, pathology, salmonella epizootic, *Salmonella* Typhimurium, salmonellosis, wild birds.

INTRODUCTION

Epizootics in wild-living passerine birds (*Passeriformes*) caused by *Salmonella* Typhimurium were first reported from Switzerland in the 1950s (Bouvier et al., 1955). Epizootics also have been reported in Great Britain (Wilson and MacDonald, 1967), Germany (Schaal and Ernst, 1967), Sweden (Hurvell et al., 1974), Denmark (Nielsen and Clausen, 1975), USA (Hudson and Tudor, 1957) and Canada (Wobeser and Finlayson, 1969). This disease was first recognized in Norway in 1969 and has since been diagnosed regularly. The disease is invariably associated with *S.* Typhimurium 4, 12: i : 1, 2 and occurs at private feeding places during winter (Refsum et al., 2002). *S.* Typhimurium has also been the most common serovar found in bird species other than small passerines in Norway and Sweden (Borg, 1985; Refsum et al., 2002). Nevertheless, small passerines constituted the vast majority of the postmortem avian cases (93.8%) at the National veterinary institute in Norway during 1969–2000.

In passerines, *S.* Typhimurium usually causes subacute septicemic infection (Daoust et al., 2000). Tits (*Paridae*) seem to be less susceptible to the infection than finches (*Fringillidae*) and sparrows (*Ploceidae, Emberizidae*) (Englert et al., 1967; Schaal and Ernst, 1967; Cornelius, 1969; Hurvell et al., 1974). Species differences are also reflected in the lesions; necrosis in esophagus and crop occurs in finches and sparrows (Kösters and Scheer, 1967; Wobeser and Finlayson, 1969; Daoust et al., 2000) but not in tits (Englert et al., 1967; Hurvell et al. 1974). These differences may be due to greater resistance in tits, or they may be less exposed to the bacteria due to their feeding behavior (Englert et al., 1967; Cornelius, 1969; Hurvell and Jevring, 1974). Systematic and detailed de-

scriptions of lesions associated with salmonellosis in passerine species are rather scarce; only a few reports describe histopathologic findings (Hurvel, 1973; Routh and Sleeman, 1995; Pennycott et al., 1998; Daoust et al., 2000; Hudson et al., 2000).

Healthy carriers of *S.* Typhimurium in passerine populations are considered to be a major source of fatal infections (Greuel and Arnold, 1971; Hurvell et al., 1974; Pennycott et al., 1998; Daoust et al., 2000). Salmonella carriage in passerines varying from 0–8.3% (in special cases 14–43%); sparrows and starlings (*Sturnus vulgaris*) are the most common carriers (Wilson and MacDonald, 1967; Goodchild and Tucker, 1968; Marx, 1969; Tizard et al., 1979; Brittingham et al., 1988; Čížek et al., 1994; Morishita et al., 1999).

In this study, we report pathologic findings and epidemiologic data linked to fatal salmonellosis in passerine birds at private feeding places in Norway. Additionally, we present results of a survey of salmonella-carriage among passerines.

MATERIALS AND METHODS

Pathologic examination

During 1998–2000, the public was encouraged, through requests published in periodicals of the Norwegian Zoological Society and the Norwegian Ornithological Society, to collect birds found dead at private feeding places, and to forward the carcasses to the National Veterinary Institute for pathologic and bacteriologic examination. The carcasses were collected from January 1998 to April 2000 and kept frozen up to 2 mo by the consignors until submission to the laboratory. All carcasses received during 1999 and 2000 (145) were subjected to extensive postmortem examination. The birds received in 1998 (34) were subjected to less extensive postmortem examination, and therefore included only in the bacteriologic part of the study. At necropsy, species, sex, weight, body condition, and pathologic findings where recorded. From the birds submitted in 1999, specimens of the lungs, heart, liver, spleen, kidneys, and other organs with gross findings, were fixed in 10% buffered formalin. Fixed specimens were processed routinely, embedded in paraffin, sectioned at 5 μm, and stained with hematoxylin and eosin for histologic examination (Culling et al., 1985). Sections that included gross lesions were also Gram stained (Culling et al., 1985). Histologic examination was not conducted on birds received in 2000.

Questionnaires

Epidemiologic information was obtained by requesting the consignors of dead birds to fill in a questionnaire. However, only questionnaires from consignors who submitted birds which laboratory examination subsequently confirmed to be cases of salmonellosis were included in the study. The questionnaires from consignors, whose dead birds were assigned other diagnosis than salmonellosis, were too few to make a comparable statistical analysis of the two groups, and thus excluded from the study. Data concerning time and duration of the incidents, species and numbers of sick and dead birds, the different species at the feeding place, type of feed, feeding routines, as well as earlier episodes with sick or dead birds, were collected and analyzed using the computer program Epi Info (version 6.04 b/c, Centers for Disease Control and Prevention, Atlanta, Georgia, USA). Salmonellosis incidents were classified in two categories; single cases representing one dead bird, and outbreaks involving at least two sick or dead birds. Species observed at the feeding place at least once a week during the winter were classified as common species.

Carriers of salmonella

Cloacal swabs were collected from apparently healthy passerines caught at 21 feeding places established for bird-ringing purposes during the winters of 1998–2000. The feeding places were located in 15 of 19 Norwegian counties, including southern and northern Norway. The swabs were kept in Stuart's transport medium (Statens Serum Institut, Copenhagen, Denmark), cooled in refrigerator before submission, and examined at the laboratory within 3–4 days after collection.

Bacteriologic examinations

Bacteriologic examination was performed on all bird carcasses (179) received during 1998–2000. Samples from liver, lungs, and heart blood were separately inoculated on two blood agar (BA) plates (Bacto Blood Agar Base No 2, Difco Laboratories, Detroit, Michigan, USA) containing 5% bovine blood, and on one bromothymol-blue lactose sucrose agar (BBLSA) plate (Bacto heart infusion agar 40.0 g, lactose 120 g, saccharose 120 g, $Na_2S_2O_3$*$5H_20$ 12 g, bromothymol-blue 0.96 g, and crystal-violet 0.06 g in 1,000 ml distilled water). The two BA plates were incubated in

5% CO_2 atmosphere and in anaerobic conditions, respectively, while the BBLSA plate was incubated aerobically. All plates were incubated overnight at 37 C.

Cloacal swabs from live birds, and intestines from carcasses, were cultured for *Salmonella* according to the Nordic Committee on Food Analysis (Anonymous, 1991). Briefly, cloacal swabs were individually incubated in 9.9 ml buffered peptone water (BPW) (Oxoid CM509, Oxoid Limited, Hampshire, England) at 37 C for 24 hr. One hundred μl from each of five samples were further pooled into 49.5 ml Rappaport-Vassiliadis Soya broth (RVS) (Oxoid CM866) and incubated at 42 C for 24 hr. Finally, the pooled RVS-samples were inoculated on modified brilliant green agar (Difco 218801) and BBLSA and incubated at 37 C for 24 hr. Meanwhile, the BPW-incubated swabs were kept in a refrigerator for possible later investigation. Intestines with contents were incubated in BPW in a ratio of 1:10 (weight/volume), after which 100 μl from each sample were transferred to 10 ml RVS. Further the procedures were otherwise identical to those describe above. Presumptive salmonella colonies were further characterized by inoculation on triple sugar iron (Difco 226540) and urease medium (Difco 228310). The cultivation procedures were repeated to identify the individual sample from positive pooled samples. Serologic typing was done by use of poly- and univalent sera (Statens Serum Institut, Copenhagen, Denmark) according to Popoff and Le Minor (1992).

RESULTS

Bacteriologic findings in bird carcasses

Salmonella Typhimurium 4, 12: i : 1, 2 was isolated from 123 of 179 birds (68.7%) examined bacteriologically during 1998–2000 (Table 1). The salmonella-positive birds originated from 87 private feeding places located across the country. In 115 of the salmonella-positive birds, the bacterium was present in lung, heart blood, liver, and intestine, indicating septicemic infection. In four birds, one bullfinch (*Pyrrhula pyrrhula*) and three Eurasian siskins (*Carduelis spinus*), the bacterium was present in lung, heart blood, and liver, but not in the intestine, indicating septicemic infection without intestinal colonization. The remaining salmonella-infected birds; two Eurasian siskins, one bullfinch, and one Eurasian greenfinch (*Carduelis chloris*), had intestinal infection only (carrier birds) and had died from trauma.

Pathologic findings in bird carcasses

From a total of 145 birds subjected to thorough postmortem examination in 1999 and 2000, 94 had died from septicemic salmonellosis (Table 2). Affected birds were equally distributed with regard to sex, and represented nine species. Most were emaciated or in poor body condition (84.0%) with wasted pectoral muscles and no visible fat. Affected birds (91.5%), except for three Eurasian siskins, three common redpolls (*Carduelis flammea*), one tree sparrow (*Passer montanus*) and one great tit (*Parus major*), also had marked organ lesions (Table 2). The most constant finding was yellow-white, multifocal to confluent necrosis in the wall of the esophagus and crop (77.7%) (Fig. 1). Scattered foci of necrosis were also common on the surface of the liver (53.2%) and spleen (45.7%), and less frequently in the wall of the proventriculus (12.8%) and intestines (5.3%). The spleen was enlarged in most cases (73.4%). In all 78 birds (83.0%) had necrotic lesions in one or several parts of the upper digestive tract (esophagus/crop/proventriculus), and 55 (58.5%) of these also had similar lesions in the liver and/or spleen. With the exception of one Eurasian siskin with necrosis in the proventriculus only, all birds with gross lesions in the proventriculus or intestine also had necrosis in other organs. Eight birds (8.5%) had necrosis only in the liver and/or spleen. Fifty-one birds that did not die of salmonellosis, died either from trauma (23%), emaciation (5%), or other causes (7%).

Histologically, necrosis in the crop/esophagus affected the entire mucosa, and commonly the submucosa and lamina muscularis. Necrotic areas consisted of fibrin, debris, heterophilic and mononuclear inflammatory cells, and large amounts of Gram-negative bacteria. The necrosis seen in proventriculus and intestine were similar to those in the crop/esophagus, but normally less extensive. Hepatic and splen-

TABLE 1. *Salmonella* Typhimurium 4, 12 : i : 1, 2-infection in birds found dead at 87 private feeding places by species, Norway 1998–2000, common species observed at 70[a] feeding places, and isolation of *S.* Typhimurium 4, 12 : i : 1, 2 from carrier birds.

Species	Carcasses		Common species number of feeding places (%)	Carriers	
	Number of birds examined	Number of birds infected (%)		Number of birds examined	Number of birds infected (%)
Great tit (*Parus major*)	13	2 (15)	69 (99)	303	0
Bullfinch (*Pyrrhula pyrrhula*)	63	49 (78)	60 (86)	281	8 (2.8)
Blue tit (*Cyanistes caeruleus*)	6	2 (33)	56 (80)	138	0
Eurasian greenfinch (*Carduelis chloris*)	10	8 (80)	53 (76)	380	2 (0.5)
Black-billed magpie (*Pica pica*)	—	—	40 (57)	1	0
Willow tit (*Poecile montanus*)	—	—	39 (56)	26	0
Common redpoll (*Carduelis flammea*)	31	26 (84)	29 (41)	348	12 (3.4)
Coal tit (*Parus ater*)	—	—	28 (40)	16	0
Yellow hammer (*Emberiza citrinella*)	—	—	27 (39)	51	1
Eurasian siskin (*Carduelis spinus*)	40	33 (83)	25 (36)	159	14 (8.8)
Wood nuthatch (*Sitta europaea*)	—	—	25 (36)	20	0
Common blackbird (*Turdus merula*)	1	0	24 (34)	26	0
House sparrow (*Passer domesticus*)	1	0	24 (34)	25	2 (8)
Eurasian jay (*Garrulus glandarius*)	—	—	20 (29)	0	—
Great spotted woodpecker (*Dendrocopos major*)	—	—	18 (26)	3	0
Carrion crow (*Corvus corone*)	—	—	14 (20)	0	—
Marsh tit (*Parus palustris*)	—	—	13 (19)	2	0
European robin (*Erithacus rubecula*)	—	—	11 (16)	11	0
Eurasian tree sparrow (*Passer montanus*)	4	1 (25)	11 (16)	40	0
Brambling (*Fringilla montefringilla*)	1	1 (100)	9 (13)	77	0
Bohemian waxwing (*Bombycilla garrulus*)	2	0	4 (6)	8	1 (13)
Chaffinch (*Fringilla coelebs*)	1	0	4 (6)	0	—
Blackcap (*Sylvia atricapilla*)	—	—	3 (4)	3	0
Hawfinch (*Coccothraustes coccothraustes*)	2	1 (50)	0 (—)	5	0
Common crossbill (*Loxia curvirostra*)	2	0	0 (—)	0	—
Other species	2	0	—	67	0
	179	123		1,990	40

[a] One missing value.

ic lesions were characterized by extensive necrosis with accumulation of inflammatory cells, and occasional multinucleated giant cells, in combination with small acute foci of necrosis with no inflammatory response. Aggregates of Gram-negative bacteria were seen in association with necrosis, but also occurred elsewhere in hepatic and splenic parenchyma. Pulmonary, renal, and myocardial findings included scattered aggregates of Gram negative bacteria, usually located within vessels, although in some sites, the aggregations were found in combination with small foci of necrosis. Four of eight birds with septicemic salmonellosis and no grossly visible lesions, had acute focal necrosis with little or no reactive change nor bacteria.

Questionnaires

Questionnaires were received from 71 (82%) of 87 feeding places with confirmed cases of salmonellosis during 1998–2000, representing 17 single cases and 54 outbreaks. Sick and dead birds were observed from December to June, with a distinct peak during February and March (Fig. 2). The duration of the outbreaks varied from less than 1 wk up to 4 mo (median=1.35 mo, mean=1 mo).

Overall, 205 birds were reported found dead; 121 of these birds were submitted

TABLE 2. Sites of gross lesions (necrosis) in 94 passerines with fatal *S.* Typhimurium 4, 12 : i : 1, 2-infection, Norway 1999–2000.

Species	Number of birds infected	Number of birds (%) with necrosis in organs					
		Pharynx	Crop/esophagus	Proventriculus	Liver	Spleen	Intestines
Bullfinch (*Pyrrhula pyrrhula*)	33	1 (3)	32 (97)	3 (9)	29 (88)	17 (52)	4 (12)
Eurasian siskin (*Carduelis spinus*)	27	—	18 (67)	4 (15)	12 (44)	12 (44)	1 (4)
Common redpoll (*Carduelis flammea*)	24	—	19 (79)	3 (13)	3 (13)	10 (48)	—
Eurasian greenfinch (*Carduelis chloris*)	5	—	2 (40)	1 (20)	5 (100)	4 (80)	—
Hawfinch (*Coccothraustes coccothraustes*)	1	—	1	1	—	—	—
Brambling (*Fringilla montefringilla*)	1	—	1	—	—	—	—
Eurasian tree sparrow (*Passer montanus*)	1[a]	—	—	—	—	—	—
Great tit (*Parus major*)	1[a]	—	—	—	—	—	—
Blue tit (*Cyanistes caeruleus*)	1	—	—	—	1	—	—
Total	94	1 (1)	73 (78)	12 (13)	50 (53)	43 (46)	5 (5)

[a] Extensive postmortem autolysis.

and in 111 the cause of death was septicemic salmonellosis. The number of dead birds found at each feeding place during outbreaks varied from one to 23 (median=2). In 13 outbreaks (25%), only one dead bird was found, but additional sick birds were observed. Twenty-eight consignors (39%) had observed one or several episodes of disease in recent years. The most common species of birds observed at the feeding places are given in Table 1.

All except one consignor fed the birds sunflower seed, most often in combination with other kinds of feed, like tallow (48%), bread (35%), nuts (23%), balls of mixed fat and seed (21%), oat sheaf (a Christmas tradition in Norway) (20%), seed mixture (17%), and grain (16%). More than 80% of the consignors used one to three feeding systems, the remaining using more than three systems. The feed was most often suspended (78%), or offered from hanging feeders (69%), on roofed bird tables (59%), or on the ground (35%). Most feeding systems were cleaned (brushed or scraped) at least once during the winter.

Carriers of salmonella

Salmonella Typhimurium 4, 12: i : 1, 2 was isolated from cloacal swabs from 2.0% of 1,990 passerines examined, comprising seven species (Table 1). The 40 carriers came from nine (43%) of 21 feeding places where birds were sampled. If only samples from feeding places where carriers were detected are included (1,014), prevalence rises to 4%. Additionally, during the 3 yr study, salmonellosis was confirmed in carcasses from four of these nine feeding places.

DISCUSSION

Septicemic salmonellosis, caused by *S.* Typhimurium 4, 12: i : 1, 2, was the main cause of death in small passerines found at Norwegian feeding places in the present study; the occurrence of disease peaked during February and March. Similar findings have been reported from Sweden (Hurvell et al., 1974; Borg, 1985).

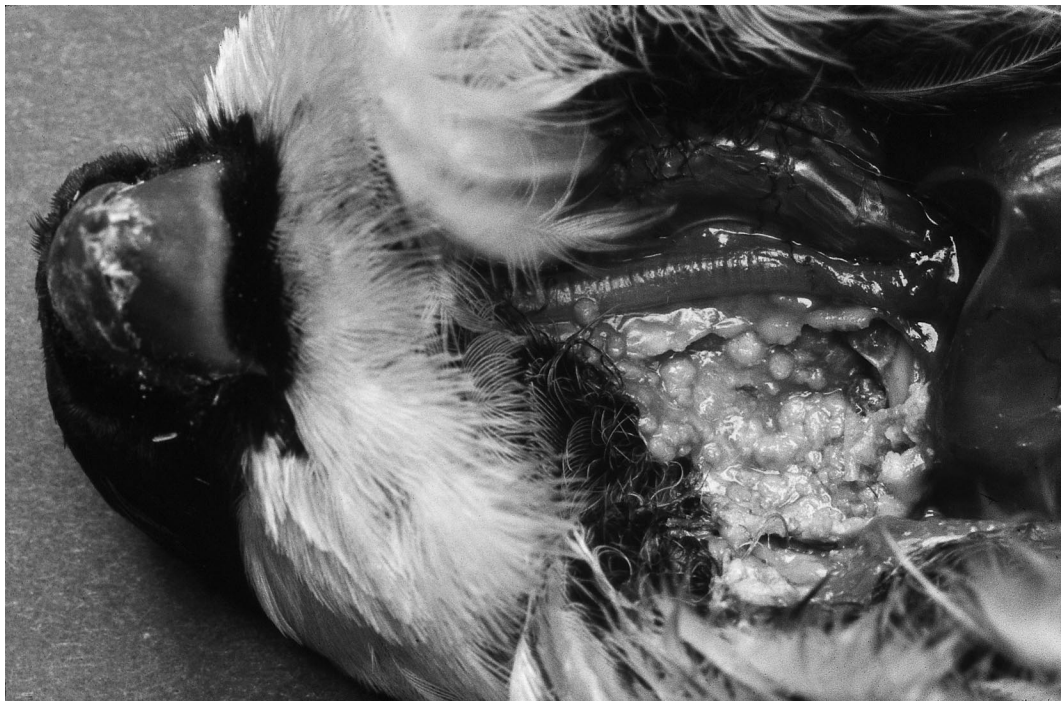

FIGURE 1. Bullfinch (*Pyrrhula pyrrhula*) with extensive and confluent necrosis in the crop wall protruding into the lumen.

Our study supports earlier findings that finches, like bullfinch, Eurasian siskin, common redpoll, and greenfinch, are particularly susceptible to *S.* Typhimurium infection (Englert et al., 1967; Schaal and Ernst, 1967; Borg, 1985; Routh and Sleeman, 1995; Daoust et al., 2000). Reports of disease in common redpolls are restricted to the Scandinavian peninsula (Borg, 1985) and North America (Daoust et al., 2000), probably reflecting the circumpolar distribution of this species (Cramp and Simmons, 1994). Although the great tit and blue tit (*Cyanistes caeruleus*) were common species at most of the feeding places in the present study, salmonellosis was confirmed only in four individuals. In Norway and Sweden, only 5% of dead great tits examined during more than 30 yr died from salmonellosis (Borg, 1985; Refsum et al., 2002). The explanation for the lower number of fatal infections in tits, compared to finches, may lie in a greater resistance to infection in tits, or behaviour at the feeding place resulting in less exposure to the bacterium. Gregarious species like finches, siskins, and sparrows often seek feed on the ground, potentially contaminated by droppings from infected birds. These birds also probably run a greater risk of being infected, since they often stay for a prolonged time at the feeding place (Englert et al., 1967; Cornelius, 1969; Hurvell and Jevring, 1974; Daoust et al., 2000). Tits usually pick food, often

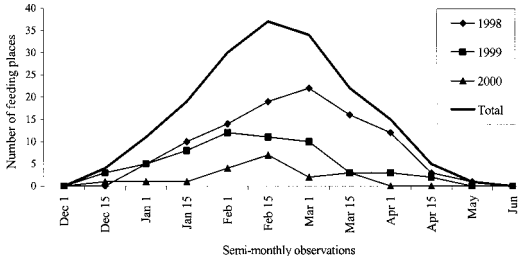

FIGURE 2. Observations of sick and dead birds at feeding places by month, reported by consignors of dead birds confirmed to be cases of salmonellosis, Norway 1998–2000.

from suspended feed or hanging feeders, and fly off the feeding place to consume it elsewhere. In addition, the way the birds eat seed might be important. Finches and siskins can hold several seeds in their bills and husk them one at a time (Newton, 1972), while tits process feed by pecking it while held between the feet. Thus, in contrast to finches, tits may be less exposed to potentially contaminated hulls. Whether there actually are species differences in resistance or not, needs further clarification.

Common lesions in cases of fatal salmonellosis were necrosis in the crop/esophagus, liver, and spleen, as well as splenomegaly. These lesions were reported in other studies (Kösters and Scheer, 1967; Englert et al., 1967; Hurvell et al., 1974; Pennycott et al., 1998; Daoust et al., 2000). We also found necrosis in the proventriculus. The most constant and extensive lesion was necrosis in the crop/eosophagus. In four birds, the bacterium was isolated from lung, heart blood and liver, but not in intestine, suggesting that the bacterium can be invasive in the upper part of the digestive tract. This was proposed by Daoust et al. (2000). The crop stores food when the gizzard is full (King and McLelland, 1984), providing the opportunity for bacterial invasion through the crop mucosa. Species, such as finches, that stay at the feeding place and fill up the crop in a short time may be prone to invasion of the crop by the bacterium, compared to species, such as tits, that seize food and fly off to consume it elsewhere.

The great majority of the salmonella-infected birds in this study had gross organ lesions that histologically represented sites of subacute inflammatory processes. These observations support the work of Daoust et al. (2000) that septicemic infections in passerines caused by *S.* Typhimurium normally take a subacute course. However, in the present study, there were also a few birds with no grossly visible lesions, the infection being characterized by acute necrosis in the organs. Thus the course of disease may be acute, or even peracute in some birds.

The overall prevalence of salmonella carriage found in the present study (2.0%) was probably an underestimation, due to sub-optimal sampling method, using cloacal swabs. The primary carrier species were identical to those species most often suffering from salmonellosis. No carriers were found among 485 tits (*Parus* sp.) examined. This result supports our recent hypothesis, that tits are less exposed to the bacterium. Other surveys of passerines have shown varying prevalences from 0–8.3 % (Wilson and MacDonald, 1967; Goodchild and Tucker, 1968; Tizard et al., 1979; Čížek et al., 1994; Morishita et al., 1999). However, with one exception (Tizard et al., 1979), none of these studies provide explicit information as to whether or not the samples were collected from birds caught at private feeding places.

The high prevalence of salmonella infection in passerines during winter in the present study is in accordance with earlier studies from Europe (Englert et al., 1967; Cornelius, 1969; Hurvell et al., 1974; Borg, 1985;) and North America (Faddoul et al., 1966; Wobeser and Finlayson, 1969; Daoust et al., 2000). The apparent seasonal occurrence may be biased to some extent by the fact that the public mainly feed, and thus closely observe, the birds during winter. On the other hand, most birds live dispersed during summer, reducing the possibility of salmonella transmission between birds. Additionally, they are in good condition in summer due to the availability of varied nutrient-rich feed, which presumably makes them less susceptible to fatal infection. However, the occurrence of sporadic or epizootic cases of fatal salmonellosis has been reported during the summer (Hudson and Tudor, 1957; Faddoul et al., 1966; Schaal and Ernst, 1967; Wobeser and Finlayson, 1969; Pennycott et al., 1998; Daoust et al., 2000). All cases were found at feeding places. Contaminated feeding places may facilitate transmission of pathogens be-

tween birds at any time of the year, but summer feeding of birds is not common in Norway.

Contaminated feeding places seem to play an important role in the epizootiology of salmonellosis in passerines (Greuel and Arnold, 1971; Locke et al., 1973; Borg, 1985; Pennycott et al., 1998). Crowds of birds visit feeding places during the winter season, running a risk of being infected indirectly from healthy carriers or sick birds. In our study, carrier birds and disease outbreaks occurred all over the country. Approximately half the consignors reported previous observations of sick or dead birds consistent with fatal salmonellosis, thus indicating a possible continuous local source of infection. In a follow-up investigation of two feeding places, at which more than one outbreak was reported in recent years, we isolated *S.* Typhimurium 4, 12: i : 1, 2 from feed on the ground and on the bird table, from remnants of old food, and from the soil (data not shown). Similar findings have been reported from Germany (Kösters and Scheer, 1967; Schaal and Ernst, 1967). The bacteria may survive for months in moist soil, feces, and water (Hess et al., 1974; Murray, 1991; Böhm, 1993). Thus, the bacterium may survive from one year to another, potentially causing new infections in birds.

In most studies, salmonella have not been isolated from fresh bird feed in Norway (Anonymous, 1965–2000) nor elsewhere (Englert et al., 1967; Schaal and Ernst, 1967; Wobeser and Finlayson, 1969; Borg, 1985). Moreover, if feed constituted a primary source of infection, one would expect several serovars to be involved. Only *S.* Typhimurium 4, 12: i : 1, 2 has been isolated from birds suffering from salmonellosis in Scandinavia (Anonymous, 1965–2000; Borg, 1985). In our study, all but one consignor fed sunflower seed. This may enhance the risk of salmonella exposure, since the birds often scatter this seed on the ground where it can be easily contaminated by droppings.

The present study has shown that carrier birds are probably important in maintaining a source of infection, causing sporadic cases or local epizootics of salmonellosis in passerines by contamination of feeding places. However, the influence of environmental factors in the epidemiology of extensive epizootics still remains poorly understood.

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LITERATURE CITED

ANONYMOUS. 1965–2000. Records of bacteriology and wildlife pathology. National Veterinary Institute, Oslo, Norway.

ANONYMOUS. 1991. Nordic Committee on Food Analysis. Salmonella; Detection in food. Method no. 71, 4th Edition, National Veterinary Institute, Oslo, Norway.

BÖHM, R. 1993. Behavior of selected salmonellae in the environment. Deutsche Tierärztliche Wochenschrifte 100: 275–278 [In German].

BORG, K. 1985. Spread of infection through wild animals-account of a 35-year study. Svensk Veterinärtidning 37: 111–128 [In Swedish].

BOUVIER, G., H. BURGISSER, AND P. A. SCHNEIDER. 1955. Observations on diseases in game, birds and fish in 1953 and 1954. Schweizer Archiv für Tierheilkunde 97: 318–325 [In French].

BRITTINGHAM, M. C., S. A. TEMPLE, AND R. M. DUNCAN. 1988. A survey of the prevalence of selected bacteria in wild birds. Journal of Wildlife Diseases 24: 299–307.

ČÍŽEK, A., I. LITERÁK, K. HEJLÍ ČEK, F. TREML, AND J. SMOLA. 1994. Salmonella contamination of the environment and its incidence in wild birds. Journal of Veterinary Medicine 41: 320–327.

CORNELIUS, L. W. 1969. Field notes on salmonella infection in greenfinches and house sparrows. Journal of Wildlife Diseases 5: 142–143.

CRAMP, S., AND K. E. L. SIMMONS. 1994. Handbook of the birds of Europe, the Middle East and North Africa, Vol. VIII, Oxford University Press, Oxford, UK, pp. 639–661.

CULLING, C. F. A., R. T. ALLISON, AND W. T. BARR. 1985. Cellular pathology technique, 4th Edition, Butterworth & Co. Ltd., London, UK, pp. 155–180.

DAOUST, P. Y., D. G. BUSBY, L. FERNS, J. GOLTZ, S. MCBURNEY, C. POPPE, AND H. WHITNEY. 2000. Salmonellosis in songbirds in the Canadian Atlantic provinces during winter-summer 1997–98. Canadian Veterinary Journal 41: 54–59.

ENGLERT, H. K., K. HAASS, J. SCHNEIDER, AND M. SCHNETTER. 1967. Enzootic salmonellosis in birds in Baden. Berliner und Münchener Tierärztliche Wochenschrift 80: 277–279 [In German].

FADDOUL, G. P., G. W. FELLOWS, AND J. BAIRD. 1966. A survey on the incidence of Salmonellae in wild birds. Avian Diseases 10: 90–94.

GOODCHILD, W. M., AND J. F. TUCKER. 1968. Salmonellae in British wild birds and their transfer to domestic fowl. British Veterinary Journal 124: 95–101.

GREUEL, E., AND J. ARNOLD. 1971. Epidemiologic studies on the occurrence of salmonellosis in song-birds. Berliner und Münchener Tierärztliche Wochenschrift 84: 292–294 [In German].

HESS, E., G. LOTT, AND C. BREER. 1974. Sewage-sludge and transmission cycles of Salmonellae. Zentralblatt für Bakteriologie, Parasitenkunde, Infektionskrankheiten und Hygiene. Erste Abteilung Originale. Reihe B: Hygiene, Präventive Medizin 158: 446–455 [In German].

HUDSON, C. B., AND D. C. TUDOR. 1957. *Salmonella typhi-murium* infection in feral birds. Cornell Veterinarian 47: 394–395.

HUDSON, C. R., C. QUIST, M. D. LEE, K. KEYES, S. V. DODSON, C. MORALES, S. SANCHEZ, D. G. WHITE, AND J. J. MAURER. 2000. Genetic relatedness of *Salmonella* isolates from nondomestic birds in southeastern United States. Journal of Clinical Microbiology 38: 1860–1865.

HURVELL, B. 1973. *Salmonella typhi-murium* in small passerines in Sweden. Svensk Veterinärtidning 25: 683–687 [In Swedish].

———, AND J. JEVRING. 1974. *Salmonella typhi-murium* in small passerines in Sweden. Nordisk Veterinærmedicin 26: 392–399 [In Swedish].

———, K. BORG, A. GUNNARSON, AND J. JEVRING. 1974. Studies on *Salmonella typhi-murium* infections in passerine birds in Sweden. International Congress of Game Biologists 11: 493–497.

KING, A. S., AND J. MCLELLAND. 1984. Oesophagus. *In* Birds their structure and function, 2nd Edition, Baillière Tindall, London, UK, pp. 90–94.

KÖSTERS, J., AND M. SCHEER. 1967. Salmonellosis in wild finches. Eine Tierärztliche Umschau 22: 66–71.

LOCKE, L. N., R. B. SHILLINGER, AND T. JAREED. 1973. Salmonellosis in passerine birds in Maryland and West Virginia. Journal of Wildlife Diseases 9: 144–145.

MARX, M. B. 1969. Two surveys of salmonella infection among certain species of wildlife in northern Virginia (1963 and 1965–1966). American Journal of Veterinary Research 30: 2003–2006.

MORISHITA, T. Y., P. P. AYE, E. C. LEY, AND B. S. HARR. 1999. Survey of pathogens and blood parasites in free-living passerines. Avian Diseases 43: 549–552.

MURRAY, C. J. 1991. Salmonellae in the environment. Revue Scientifique et Technique 10: 765–785.

NEWTON, I. 1972. Feeding ecology. *In* Finches. Williams Collins Sons & Co Ltd, London, UK, p. 100.

NIELSEN, B. B., AND B. CLAUSEN. 1975. The incidence of *Salmonella* bacteria in Danish wildlife and in imported animals. Nordisk Veterinærmedicin. 27: 633–640.

PENNYCOTT, T. W., H. M. ROSS, I. M. MCLAREN, A. PARK, G. F. HOPKINS, AND G. FOSTER. 1998. Causes of death of wild birds of the family Fringillidae in Britain. The Veterinary Record 143: 155–158.

POPOFF, M., AND L. LE MINOR. 1992. Antigenic formulas of the *Salmonella* serovars. WHO Collaborating Centre for References and Research on Salmonella. Institut Pasteur, Paris, France.

REFSUM, T., K. HANDELAND, D. L BAGGESEN, G. HOLSTAD, AND G. KAPPERUD. 2002. Salmonellae in avian wildlife in Norway 1969–2000. Applied and Environmental Microbiology. In press.

ROUTH, A., AND J. M. SLEEMAN. 1995. Greenfinch mortalities. The Veterinary Record 136: 500.

SCHAAL, E., AND H. ERNST. 1967. Enzootic occurrence of salmonellosis in local wild birds. Berliner und Münchener Tierärztliche Wochenschrift 80: 13–16 [In German].

TIZARD, I. R., N. A. FISH, AND J. HARMESON. 1979. Free flying sparrows as carriers of salmonellosis. Canadian Veterinary Journal 20: 143–144.

WILSON, J. E., AND J. W. MACDONALD. 1967. Salmonella infection in wild birds. British Veterinary Journal 123: 212–218.

WOBESER, G. A., AND M. C. FINLAYSON. 1969. *Salmonella typhimurium* infection in house sparrows. Archives of Environmental Health 19: 882–884.

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