HOST RANGE AND DYNAMICS OF MYCOPLASMAL CONJUNCTIVITIS AMONG BIRDS IN NORTH AMERICA

Authors: Hartup, Barry K., Dhondt, André A., Sydenstricker, Keila V., Hochachka, Wesley M., and Kollias, George V.

Source: Journal of Wildlife Diseases, 37(1) : 72-81

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-37.1.72

Journal of Wildlife Diseases, 37(1), 2001, pp. 72–81

HOST RANGE AND DYNAMICS OF MYCOPLASMAL CONJUNCTIVITIS AMONG BIRDS IN NORTH AMERICA

Barry K. Hartup,[1,3] André A. Dhondt,[2] Keila V. Sydenstricker,[2] Wesley M. Hochachka,[2] and George V. Kollias[1]

[1] Division of Wildlife Health, College of Veterinary Medicine, Cornell University, Ithaca, New York 14853, USA
[2] Bird Population Studies, Laboratory of Ornithology, Cornell University, Ithaca, New York 14850, USA
[3] Corresponding author. Present address: International Crane Foundation, E-11376 Shady Lane Road, Baraboo, Wisconsin 53913, USA; E-mail: hartup@savingcranes.org

ABSTRACT: An epidemic of conjunctivitis among house finches (*Carpodacus mexicanus*) caused by *Mycoplasma gallisepticum* (MG) bacterial infections was first described in 1994. The disease exhibits high primary host specificity, but has been isolated from a limited number of secondary avian hosts at various times and locations. We used records from the House Finch Disease Survey, a continent-wide, volunteer monitoring project, to document the host range of conjunctivitis in birds at feeding stations and to investigate how disease in house finches might influence the spread of conjunctivitis to other hosts. Between 1994 and 1998, participants recorded 675 cases of conjunctivitis in 31 species other than house finches in eastern North America. Seventy five % of these cases were observed among three species: American goldfinches (*Carduelis tristis*), purple finches (*Carpodacus purpureus*) and house sparrows (*Passer domesticus*). The proportion of sites with diseased wintering populations of the three species increased over the 4 yr study and coincided with range expansion of conjunctivitis in house finches. Sites with diseased house finches present were significantly more likely to report conjunctivitis in each of the three species during the same month. These observations are most consistent with transmission of an infectious agent (presumably MG) from house finches to these secondary hosts via spillover of localized epidemics, rather than sustained interspecific transmission.

Key words: *Carduelis tristis*, *Carpodacus mexicanus*, *Carpodacus purpureus*, conjunctivitis, host range, epidemiology, *Mycoplasma gallisepticum*.

INTRODUCTION

Host specificity, or the degree to which a symbiotic relationship between host and parasite has evolved, is the result of several factors, including microhabitat requirements and virulence of the parasite, prevailing environmental conditions, host ecology, and host-parasite co-evolution (Adamson and Caira, 1994; Levin, 1996). These factors combine to shape adaptations among infectious organisms for optimal transmission of disease. Yet, parasites with high host specificity (single primary or narrow host range) occasionally infect other, secondary hosts, sometimes with devastating results. Such instances may reflect changes in ecological relationships that increase transmission probabilities between the two hosts, changes in the susceptibility of the secondary host to disease, or novel microbial adaptations to the secondary host (Morse, 1995; Cohen, 1998). Under these circumstances, secondary hosts may become maintenance hosts where the infectious agent persists and acquires an expanded host range. These infections may instead represent spillover from a primary reservoir into hosts in which the disease is not maintained (Bhattacharya et al., 1986; O'Reilly and Daborn, 1995; Nel et al., 1997). "Spillover" infections are normally distinguished by a more sporadic pattern of disease occurrence among secondary hosts, and are generally in close association with sudden or sustained increases in incidence among the primary host range. Spillover events are important because they provide opportunities for the emergence of new host ranges of parasites (Morse, 1995).

A recent example of an emergent bacterial disease in wild birds is conjunctivitis in house finches (*Carpodacus mexicanus*) caused by *Mycoplasma gallisepticum* (MG) infection (Ley et al., 1996; Luttrell et al., 1996; Fischer et al., 1997). Of 23 mycoplasma species described from domestic and free-ranging avian sources, MG

is one of four pathogenic species common in domestic poultry (Jordan, 1996). *Mycoplasma gallisepticum* is not considered a naturally occurring pathogen in wild birds and sustained reservoirs have never been identified. The MG strain isolated from house finches since early 1994 appears to be unique and not related to historical or current poultry isolates or vaccines (Ley et al., 1997). Confirmed diagnoses of MG-associated conjunctivitis from other hosts have been infrequent during this period. Single isolations of MG have been obtained from a purple finch (*Carpodacus purpureus*), downy woodpecker (*Picoides pubescens*), blue jay (*Cyanocitta cristata*), evening grosbeak (*Coccothraustes vespertinus*) and pine grosbeak (*Pinicola enucleator*) in various seasons and locations throughout eastern North America (Hartup et al., 2000). *Mycoplasma gallisepticum* has also been isolated from fewer than ten American goldfinches (*Carduelis tristis*) with conjunctivitis, but each isolate was genetically similar to the strain affecting house finches (Ley et al., 1997). In addition, survey-derived estimates showed the frequency of conjunctivitis in four sympatric species at feeding stations appeared to be low compared to house finches early in the epidemic (Hartup et al., 1998). This limited data suggests that MG infections in North American birds appear to be highly specific to house finches. Host specificity is considered a hallmark of the mycoplasma-host relationship (Tully, 1996).

Is mycoplasmal conjunctivitis emerging as a disease of other North American birds, or are infections only occasionally transmitted from house finches to secondary hosts? Based on the above findings, we hypothesized that sporadic MG infections in non-house finch hosts were the result of spillover infections arising from infected house finch populations, rather than alterations in ecological barriers to disease transmission between house finches and other hosts, lowered susceptibility to disease in other hosts, or the appearance of newly adapted mycoplasma strains. Under these alternative hypotheses, disease patterns in secondary hosts should suggest persistence at local levels and show a more contiguous pattern of occurrence, as well as spread independently of conjunctivitis in house finches. The longitudinal and distribution data required to evaluate predictions such as these, however, have rarely been sufficient in any single wild animal host, let alone between two of them (Gulland, 1995).

In this study, we used observations from the House Finch Disease Survey (HFDS) of the Cornell Laboratory of Ornithology (Ithaca, New York, USA), a volunteer-based disease monitoring tool, to evaluate the competing hypotheses outlined above for several species. The method capitalizes on the apparent strong relationship between MG infection and the easily detected host response (in this case conjunctivitis), and provides a mechanism to sample the study populations on a continental scale. We first used HFDS records to document the occurrence of conjunctivitis in bird species other than house finches, and to describe trends in the prevalence of conjunctivitis observations at feeding stations visited by these species. Secondly, we evaluated the relationship of conjunctivitis reports in American goldfinches, purple finches and house sparrows (*Passer domesticus*) with the presence of diseased house finches. If spillover occurred, we expected that the presence of diseased house finches must enter the models developed to explain conjunctivitis in these species.

MATERIALS AND METHODS

The methodology of the HFDS has been thoroughly described elsewhere (Dhondt et al., 1998; Hartup et al., 1998). Briefly, the survey was initiated in November of 1994 to follow the spread of conjunctivitis in eastern house finches and other birds common to bird feeders. The HFDS provides a valid index to the prevalence of disease in house finches because the presence of conjunctivitis in birds at feeders is closely correlated with active MG infections (Hartup et al., 2000; Hartup et al., unpubl. data). We assumed a similar relationship be-

tween disease status and MG infection in the other species surveyed for this study.

The survey makes use of experienced volunteers to provide year-round observations of diseased house finches, purple finches, chickadees (*Parus atricapilus* and *P. carolinensis*), house sparrows and dark-eyed juncos (*Junco hyemalis*) (hereafter referred to as target species) across eastern North America via a questionnaire. The survey also requested description of disease in other species (non-target species). Daily observations of healthy and conjunctivitis-affected birds were summarized to provide the following monthly totals at each bird feeding site monitored: days participants made observations, days the target species were observed, and days where one or more individuals with conjunctivitis of each target species were observed. Reports of conjunctivitis in non-target species were noted and evaluated as described below. Each monthly report was considered an independent observation of a dynamic study population of birds, similar to Hartup et al. (1998).

We used 29,266 monthly HFDS observations made by 3,489 participants from 37 states and six Canadian provinces to detect conjunctivitis in birds between November 1994 and October 1998 (4 yr). Observations from states and provinces (n = 546) west of W103° longitude were excluded from our analysis due to the historical presence of the disease in the central and eastern portions of the continent only. The presence of conjunctivitis among a species at a site (referred to as a case) was defined by one of two criteria: where the observer described signs and behavior consistent with mycoplasmal conjunctivitis (Luttrell et al., 1996) for one or more days as determined by independent analysis of two wildlife veterinarians familiar with this syndrome, or where the observer provided no description of abnormal birds, but made two or more daily observations of conjunctivitis-affected birds. The first criterion was used to identify the presence of conjunctivitis in both target and non-target species. The second criterion was used only for the target species, and was designed to guard against single, false observations with no supportive description by the observer. Other bacterial and viral etiologies may underlie the observations of conjunctivitis in these species by HFDS participants (Williams, 1997). We limited potential observer bias by excluding reports of conjunctivitis that did not pass the screening protocol.

For the target species, we were able to obtain data on the presence of healthy individuals in order to provide information on disease distribution and to serve as a referent population in epidemiological analyses. No such information, however, was available for the non-target species. In order to provide additional referent data, we extracted records from Project Feeder Watch (PFW), a volunteer-based survey that generates counts of birds visiting feeders between November and March in the study area (Wells et al., 1998). Data from PFW consists of average maximum counts of birds visiting feeders each month as an index to bird abundance at a site. We were able to match 15,375 HFDS observations with PFW counts by linking data provided by 2,484 observers at the same sites across the four years of the study.

The data for the target species and American goldfinches were stratified by winters (November 1994 to March 1995 = W1, and so on to W4) and analyzed for a linear trend in proportions to determine if conjunctivitis was becoming more common among sites with these species present (Schlesselman, 1982; EpiInfo, version 6.04, Centers for Disease Control and Prevention, Atlanta, Georgia, USA). Logistic regression models were then developed to evaluate the association of conjunctivitis in house finches (healthy house finches only versus a case being present at each site) with the likelihood of conjunctivitis observation in goldfinches, purple finches, and house sparrows (Hosmer and Lemeshow, 1989). These three species were chosen because each had more than 100 cases of conjunctivitis reported during the survey. Several potential confounding factors were considered for entry into each model including: month (November to March) and region in which observations were made (Northeast, MidAtlantic, Southeast, Midwest and Great Plains; see Dhondt et al., 1998), number of days participants monitored their feeders each month, and corresponding PFW monthly counts of house finches and the three test species ($\log_{10}$ transformed to approximate normality). We used a forward selection process ($P < 0.10$) followed by backward elimination of nonsignificant variables ($P > 0.15$; BMDP, version 7.0, Los Angeles, California, USA). Final variable inclusion was confirmed with a backward elimination procedure. Biologically interpretable interaction terms were allowed to enter the models under the criteria above if both corresponding first order terms remained in the model.

RESULTS

Non-target species

Survey participants observed 292 cases of conjunctivitis in 27 non-target species from 15 avian families at feeding stations during the study period (Table 1). The

TABLE 1. Frequency and HFDS regional distribution of conjunctivitis observations in non-target species, 1994–98.

Species	Number of reports	HFDS regions[a]
Cedar waxwing (*Bombycilla cedrorum*)	1	SE
Lazuli bunting (*Passerina amoena*)	1	GP
Rose-breasted grosbeak (*Pheucticus ludovicianus*)	2	NE, MW
Mourning dove (*Zenaida macroura*)	9	MA, SE, MW
Rock dove (*Columba livia*)	1	MA
Blue jay (*Cyanocitta cristata*)	2	MW
American tree sparrow (*Spizella arborea*)	6	MA, MW
Chipping sparrow (*Spizella passerina*)	4	MA, SE
Northern cardinal (*Cardinalis cardinalis*)	23	MA, SE, MW, GP
Song sparrow (*Melospiza melodia*)	1	MW
White-throated sparrow (*Zonotrichia albicollis*)	5	MA, SE
American goldfinch (*Carduelis tristis*)	187	MA, SE, NE, MW, GP
Common redpoll (*Cardeulis flammea*)	1	MW
Evening grosbeak (*Coccothraustes vespertinus*)	1	NE
Pine siskin (*Cardeulis pinus*)	2	MA, MW
Brown-headed cowbird (*Molothrus ater*)	7	MA, SE, MW
Common grackle (*Quiscalus quiscula*)	6	SE, MW
Red-winged blackbird (*Agelaius phoeniceus*)	4	MA, SE, GP
Northern shrike (*Lanius excubitor*)	1	MA
Tufted titmouse (*Parus bicolor*)	9	MA
Downy woodpecker (*Picoides pubescens*)	5	MA, SE, MW
White-breasted nuthatch (*Sitta carolinensis*)	1	MW
European starling (*Sturnus vulgaris*)	6	MA, MW
Ruby-throated hummingbird (*Archilochus colubris*)	1	MA
Carolina wren (*Thyothorus ludovicianus*)	1	MW
American robin (*Turdus migratorius*)	1	MW
Eastern bluebird (*Sialia sialis*)	1	SE

[a] MA = Mid-Atlantic, SE = Southeast, NE = Northeast, MW = Midwest, GP = Great Plains.

number of cases among most of the species was quite low ($n < 10$), with single cases noted from 12 species. The non-target species cases were identified from 687 disease reports; we excluded 395 reports due to incomplete descriptions of signs and behavior or descriptions inconsistent with mycoplasmal conjunctivitis. Several reports described clinical signs and behaviors consistent with other diseases commonly observed at bird feeding stations such as avian poxvirus infection or salmonellosis.

One hundred eighty-seven cases of conjunctivitis were reported in goldfinches. Seventy percent (139/187) of the cases were reported between November and March and occurred in all regions (Fig. 1). In W1, multiple cases were observed in a five-state region including Massachusetts, New York, Pennsylvania, Ohio and Indiana. Multiple cases were later recorded from New Jersey, Maryland, Virginia and North Carolina (W2), Illinois, Oklahoma and Texas (W3), and Michigan, Iowa and Missouri (W4). The original focus of cases in W1 persisted through the end of the study with small fluctuations in the number of cases recorded per state each winter. The maximum number of cases reported in a given winter was eight from both Maryland and North Carolina in W2. The proportion of sites in winter with goldfinch cases increased over the study period ($\chi^2_1 = 44.0$, $n = 10{,}212$, $P < 0.001$).

The presence of a case of conjunctivitis among house finches at a site increased

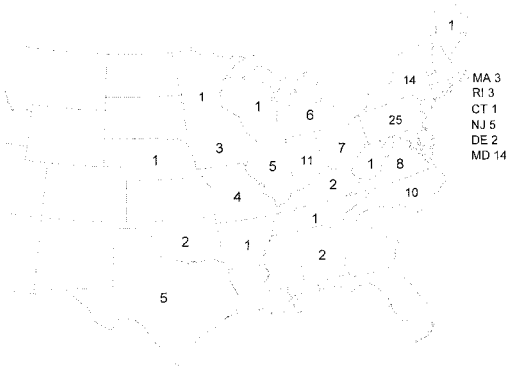

FIGURE 1. Distribution and total number of conjunctivitis cases by state in wintering American goldfinches, 1994–98. Totals for various northeastern and mid-Atlantic states appear at the right of the figure. One case from Ontario, Canada is not shown.

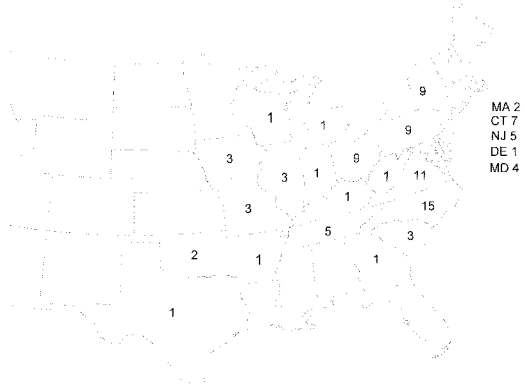

FIGURE 2. Distribution and total number of conjunctivitis cases by state in wintering purple finches, 1994–98. Totals for various northeastern and mid-Atlantic states appear at the right of the figure.

the odds of observing conjunctivitis in goldfinches by over seven times (Table 2). In addition, the probability of observing a case among goldfinches increased at sites with greater numbers of house finches and goldfinches, and during the last two winters of the study. Increasing counts of goldfinches at a site in W2, however, lowered the probability of observing a case of conjunctivitis in goldfinches.

Target species

Thirty-three cases of conjunctivitis were reported in both chickadees and dark-eyed juncos during the study period. Chickadee cases were detected in all regions except the Great Plains, but few cases were well described. There was no trend in the proportion of sites in winter with chickadee cases during the study ($\chi^2_1 = 0.06$, $n = 14{,}009$, $P = 0.80$). By contrast, junco cases were reported from all five regions monitored by the HFDS and were comparatively well described. The proportion of sites with junco cases approached an increasing trend during the survey ($\chi^2_1 = 3.2$, $n = 11{,}984$, $P = 0.07$).

Participants observed 125 cases of conjunctivitis in purple finches; 80% (100/125) occurred in winter months (Fig. 2). Conjunctivitis in purple finches was detected in all HFDS regions. In W1, multiple cases were observed in New Jersey, New York and Pennsylvania only. Multiple cases were noted from several additional states the following winter, including Connecticut, Maryland, Virginia, North Carolina, Tennessee, Ohio, Indiana and Illinois. A single participant in Oklahoma reported cases one month apart in W3, and multiple cases were first reported from Iowa and Missouri in W4. The original focus of conjunctivitis cases in purple finches did not persist over the four years of the study; no cases were reported in New Jersey, New York or Pennsylvania in W4. The maxi-

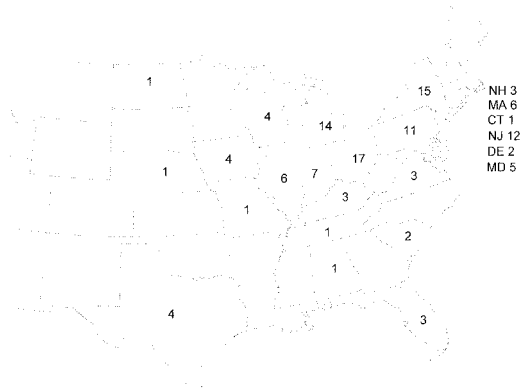

FIGURE 3. Distribution and total number of conjunctivitis cases by state in wintering house sparrows, 1994–98. Totals for various northeastern and mid-Atlantic states appear at the right of the figure. Fourteen cases from Ontario, Canada are not shown.

mum number of cases reported in any winter was seven from North Carolina in W2. The proportion of sites with purple finch cases in winter increased over the study period ($\chi^2_1 = 9.5$, $n = 3{,}010$, $P < 0.01$). Results from the multivariate analysis showed that participants were 2.5 times as likely to observe a case among purple finches when a house finch case was present (Table 2). Participants were also more likely to observe cases among purple finches in W2 and W3 compared to W1.

Survey participants reported 192 cases of conjunctivitis in house sparrows across all HFDS regions, and 72% (138/192) of cases were reported in winter months (Fig. 3). In W1, multiple cases among house sparrows were reported from 12 states: nine Great Lakes states between Illinois and New York, and New Jersey, Maryland, and Florida. First reports of multiple cases were later reported from Massachusetts and Ontario (W2), Delaware, Texas, and Virginia (W3), and Kentucky, Iowa, and Wisconsin (W4). The maximum number of cases was reported from Ontario in W2 ($n = 11$) from six different sites. There was a significant linear increase in the proportion of sites with house sparrow cases over the study period ($\chi^2_1 = 13.0$, $n = 8{,}743$, $P < 0.001$). The multivariate analysis showed that HFDS participants were more than twice as likely to observe conjunctivitis in house sparrows when a case among house finches was present (Table 2). Additionally, cases involving house sparrows were more likely to be observed during W3, the month of December each winter, and at sites with greater numbers of house sparrows.

DISCUSSION

The HFDS detected numerous cases of conjunctivitis in several species other than house finches, despite being widely distributed across time and space. The majority of cases occurred in American goldfinches, purple finches and house sparrows, but they comprised less than 2% of the total observations submitted for each species. By comparison, Dhondt et al. (1998) showed 20% of all surveyed sites in W2 reported cases of conjunctivitis in house finches. The increasing frequency of cases in secondary hosts, however, suggests the disease spread and may have increased in prevalence among their populations. These findings corroborate the view that MG has parasitized a limited range of free-ranging avian hosts, but is most prevalent in house finches (Hartup et al., 2000).

Results of the multivariate analyses showed that conjunctivitis in each of the three species was significantly related to exposure to diseased house finches. These findings suggest diseased house finches transmitted the causative agent of conjunctivitis (presumably MG) to the secondary hosts. Though an infected, disease-free carrier state may exist in some individuals, the rate of MG infection among house finches with conjunctivitis is considerably greater than infection rates in normal appearing house finches (Luttrell et al., 1998), and there is excellent agreement between the appearance of clinical conjunctivitis and MG infection in wild house finches (Hartup et al., unpubl. data). Transmission of pathogenic MG to secondary hosts most likely occurs through indirect means via contaminated bird feeder surfaces, or directly through competition at feeding stations and increased contacts with house finches (Hartup et al., 1998). Without a more detailed survey, however, we cannot clarify to what extent these species truly interacted with one another and the extent of MG transmission among all hosts.

The HFDS was designed to detect and document a common host response to MG infection in house finches and presumably other birds common to feeding stations, not the underlying cause of observed disease. There was no other evidence or diagnostic information made available by survey participants to suggest other infectious or non-infectious etiologies for the cases reported. Questionable or incom-

TABLE 2. Variables associated with conjunctivitis observations in three songbird species using a logistic regression model, winters 1994–98.

Species	Variable	Coefficient (±SE)	OR (95% CI)[a]
American goldfinch	Constant	−8.849 (0.883)	—
	House finch disease status		
	Healthy only	—	1
	Case present	1.962 (0.662)**	7.09 (1.94–26.04)
	Year W1	—	1
	W2	1.033 (1.33)	2.80 (0.21–37.7)
	W3	2.557 (0.995)*	12.88 (1.83–90.9)
	W4	2.900 (1.05)**	18.18 (2.34–141.2)
	Month		
	November	—	1
	December	−0.728 (0.396)	0.48 (0.22–1.05)
	January	0.049 (0.335)	1.05 (0.55–2.02)
	February	0.387 (0.332)	1.47 (0.77–2.82)
	March	−0.243 (0.357)	0.79 (0.39–1.58)
	PFW house finch count[b]	0.605 (0.222)**	—
	PFW goldfinch count	2.002 (0.520)***	—
	House finch disease status × year		
	Case × W1	—	1
	Case × W2	1.181 (1.210)	3.26 (0.30–35.0)
	Case × W3	−0.859 (0.789)	0.42 (0.09–1.99)
	Case × W4	−1.244 (0.820)	0.29 (0.06–1.44)
	PFW goldfinch count × year		
	Count × W1	—	1
	Count × W2	−1.708 (0.627)**	0.18 (0.53–0.62)
	Count × W3	−0.542 (0.638)	0.58 (0.17–2.03)
	Count × W4	−0.464 (0.695)	0.63 (0.16–2.45)
Purple finch	Constant	−6.686 (0.887)	—
	House finch disease status		
	Healthy only	—	1
	Case present	1.060 (0.352)**	2.89 (1.45–5.75)
	Year W1	—	1
	W2	1.778 (0.794)*	5.90 (1.25–28.1)
	W3	1.943 (0.805)*	6.90 (1.43–33.6)
	W4	−0.649 (1.35)	0.52 (0.04–7.35)
	Month		
	November	—	1
	December	−0.693 (0.782)	0.50 (0.11–2.32)
	January	0.170 (0.633)	1.18 (0.34–4.10)
	February	1.084 (0.583)	2.96 (0.94–9.26)
	March	0.494 (0.589)	1.64 (0.52–5.21)
	PFW purple finch count	1.025 (0.959)	—
	PFW purple finch count × year		
	Count × W1	—	1
	Count × W2	−0.285 (1.060)	0.75 (0.09–6.02)
	Count × W3	−1.782 (1.220)	0.17 (0.01–1.84)
	Count × W4	2.827 (1.460)	16.9 (0.96–298.5)

TABLE 2. Continued.

Species	Variable	Coefficient (±SE)	OR (95% CI)[a]
House sparrow	Constant	−5.663 (0.364)	—
	House finch disease status		
	Healthy only	—	1
	Case present	0.887 (0.221)***	2.40 (1.56-3.70)
	Year W1	—	1
	W2	0.519 (0.267)	1.68 (1.00–2.83)
	W3	1.005 (0.278)***	2.73 (1.58–4.72)
	W4	0.545 (0.370)	1.72 (0.83–3.56)
	Month		
	November	—	1
	December	−0.922 (0.360)*	0.40 (0.20–0.81)
	January	−0.181 (0.304)	0.83 (0.46–1.51)
	February	0.095 (0.302)	1.10 (0.61–1.99)
	March	0.444 (0.328)	0.64 (0.34–1.22)
	PFW house sparrow count	0.637 (0.192)**	—

[a] OR = odds ratio, CI = confidence interval.
[b] $\log_{10}$ transformed.
* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

plete data, or reports describing other syndromes common to birds at feeders were excluded to limit potential bias in the data.

Another avian mycoplasma, *M. sturni* (MS), has been associated with a single case of conjunctivitis in a free-ranging European starling (*Sturnus vulgaris*), and several captive blue jays (*Cyanocitta cristata*) and northern mockingbirds (*Mimus polyglottos*) (Forsyth et al., 1996; Ley et al., 1998). Both starlings and blue jays with conjunctivitis were described by HFDS participants, and may be attributable to MS infection; yet, no epidemic of conjunctivitis in free-ranging individuals of any species has been attributed to MS. The low prevalence of MG-associated conjunctivitis in the secondary host species surveyed by the HFDS has been confirmed by field and laboratory studies (Hartup et al., 2000; M.P. Luttrell unpubl. data; C.B. Thomas unpubl. data), suggesting these hosts experience less exposure to MG (lower transmission rates), are less likely to develop conjunctivitis than house finches following MG infection (lower susceptibility), or both.

The small number and patchy distribution of conjunctivitis in each secondary host appear to be most consistent with spillover of a transmissible agent (presumably MG) from house finches. Alternative explanations, such as sustained transmission of disease within the secondary species at low levels or inherently lowered susceptibility to infection, or the emergence of new host-parasite relationships, should have resulted in different patterns of disease occurrence, as well as independence of disease in house finches in the epidemiological models.

If the secondary species experienced less frequent transmission or were inherently less susceptible to MG but sustained the parasite, conjunctivitis should have been more consistently reported in local (or state level) populations once established, and the rate of disease spread among each species should have lagged behind that observed in house finches. Instead, we observed a patchy and inconsistent distribution of cases at local and regional levels in each of the three species examined, though long distance movement of MG infected birds could occur with secondary host dispersal or migration. Addi-

tionally, the initial ranges of conjunctivitis suggested by the distribution of W1 cases (especially in goldfinches and purple finches), together with subsequent range expansion, coincides closely with the rate and direction of spread of mycoplasmal conjunctivitis among eastern house finches (Dhondt et al., 1998).

To our knowledge, no significant changes have occurred in the ecology of house finches and the other species surveyed that would result in increased transmission of an infectious agent between them. We indirectly controlled for changes in the population dynamics and range of each species by incorporating data from PFW. For example, house finch populations have declined throughout much of eastern North America after the emergence of mycoplasmal conjunctivitis (Hochachka and Dhondt, 2000), and the purple finch is noted for quasicyclical irruptions across its winter range that likely impact its contact with house finches and exposure to pathogenic organisms. Bird feeding activity by the general public, however, was assumed to be unchanged throughout the survey period though this may represent a significant risk factor for the transmission of conjunctivitis in songbirds (Hartup et al., 1998). The impact of larger ecological alterations, such as climate change, was beyond the scope of this study.

Finally, there remains no compelling documentation to support the emergence of novel MG strains in these secondary hosts. Ongoing molecular epidemiological surveillance of isolates made from goldfinches and purple finches show no dissimilarities with DNA fingerprints of house finch isolates (Ley et al., 1997; Hartup et al., 2000).

The concurrent observations of conjunctivitis in American goldfinches, purple finches and house sparrows over time and space with epidemics among house finches, demonstrated epidemiological associations with exposure to diseased house finches, and available diagnostic findings lead us to believe there is a common etiology involved in these cases and that spillover of infections from house finches underlie their appearance. Additional study is needed, however, to confirm mycoplasmal conjunctivitis in house sparrows and other species recorded with multiple cases of conjunctivitis (cardinals for example). At the levels detected in the present study, conjunctivitis is unlikely to have a deleterious impact on any of the secondary host populations affected, though increases in local or regional mortality rates may occur in the short-term due to spillover events. We believe continued monitoring of this disease is warranted to detect further changes in host range and specificity.

ACKNOWLEDGMENTS

We thank the thousands of House Finch Disease Survey participants from throughout North America for their enthusiasm and meticulous contributions. We also thank M. Barker, L. Field, H. Freiberger, L. Kammermeier, S. Kelling, D. Tessaglia-Hymes, and other Laboratory of Ornithology staff for their efforts towards this project. H. Mohammed provided generous assistance in the development of the statistical models for this project. This research was, in part, funded by a Hatch grant NYC-171403.

LITERATURE CITED

ADAMSON, M. L., AND J. N. CAIRA. 1994. Evolutionary factors influencing the nature of parasite specificity. Parasitology 109: S85–S95.

BHATTACHARY, A. S., S. K. CHAKRABORTY, S. CHAKRABORTY, K. K. GHOSH, A. PALIT, K. K. MUKHERJEE, M. S. CHAKRABORTY, N. TANDON, AND A. K. HATI. 1986. Density of *Culex vishnui* and appearance of Japanese encephalitis antibody in sentinel chicks and wild birds in relation to Japanese encephalitis cases. Tropical and Geographical Medicine 38: 46–50.

COHEN, M. L. 1998. Resurgent and emergent disease in a changing world. British Medical Bulletin 54: 523–532.

DHONDT, A. A., D. L. TESSAGLIA, AND R. L. SLOTHOWER. 1998. Epidemic mycoplasmal conjunctivitis in house finches from eastern North America. Journal of Wildlife Diseases 34: 265–280.

FISCHER, J. R., D. E. STALLKNECHT, M. P. LUTTRELL, A. A. DHONDT, AND K. A. CONVERSE. 1997. Mycoplasmal conjunctivitis in wild songbirds: The spread of a new contagious disease in a mobile host population. Emerging Infectious Diseases 3: 69–72.

FORSYTH, M. H., J. G. TULLY, T. S. GORTON, L. HINCKLEY, S. FRASCA, H. J. VAN KRUININGEN, AND S. J. GEARY. 1996. *Mycoplasma sturni* sp. nov., from the conjunctiva of a European starling (*Sturnus vulgaris*). International Journal of Systematic Bacteriology 46: 716–719.

GULLAND, F. M. D. 1995. The impact of infectious diseases on wild animal populations—a review. *In* Ecology of infectious diseases in natural populations, B. T. Grenfell and A. P. Dobson (eds.). Cambridge University Press, Cambridge, UK, pp. 20–51.

HARTUP, B. K., G. V. KOLLIAS, AND D. H. LEY. 2000. Mycoplasmal conjunctivitis in songbirds from New York. Journal of Wildlife Diseases 36: 257–264.

———, H. O. MOHAMMED, G. V. KOLLIAS, AND A. A. DHONDT. 1998. Risk factors associated with mycoplasmal conjunctivitis in house finches. Journal of Wildlife Diseases 34: 281–288.

HOCHACHKA, W. M., AND A. A. DHONDT. 2000. Density-dependent decline of host abundance resulting from a new infectious disease. Proceedings of the National Academy of Sciences, USA 97: 5303–5306.

HOSMER, D. W., AND S. LEMESHOW. 1989. Applied logistic regression. John Wiley and Sons, New York, New York, 307 pp.

JORDAN, F. T. W. 1996. Avian mycoplasmosis. *In* Poultry diseases, 4th Edition, F. T. W. Jordan and M. Pattison (eds.). W. B. Saunders, London, UK, pp. 81–93.

LEVIN, B. R. 1996. The evolution and maintenance of virulence in microparasites. Emerging Infectious Diseases 2: 93–102.

LEY, D. H., J. E. BERKHOFF, AND J. M. MCLAREN. 1996. *Mycoplasma gallisepticum* isolated from house finches (*Carpodacus mexicanus*) with conjunctivitis. Avian Diseases 40: 480–483.

———, ———, AND S. LEVISOHN. 1997. Molecular epidemiologic investigations of *Mycoplasma gallisepticum* conjunctivitis in songbirds by random amplified polymorphic DNA analyses. Emerging Infectious Diseases 3: 375–380.

———, S. J. GEARY, J. E. BERKHOFF, J. M. MCLAREN, AND S. LEVISOHN. 1998. *Mycoplasma sturni* from blue jays and northern mockingbirds with conjunctivitis in Florida. Journal of Wildlife Diseases 34: 403–406.

LUTTRELL, M. P., J. R. FISCHER, D. E. STALLKNECHT, AND S. H. KLEVEN. 1996. Field investigation of *Mycoplasma gallisepticum* infections in house finches (*Carpodacus mexicanus*) from Maryland and Georgia. Avian Diseases 40: 335–341.

———, D. E. STALLKNECHT, J. R. FISCHER, C. T. SEWELL, AND S. H. KLEVEN. 1998. Natural *Mycoplasma gallisepticum* infection in a captive flock of house finches. Journal of Wildlife Diseases 34: 289–296.

MORSE, S. S. 1995. Factors in the emergence of infectious diseases. Emerging Infectious Diseases 1: 7–15.

NEL, L., J. JACOBS, J. JAFTHA, AND C. MEREDITH. 1997. Natural spillover of a distinctly canidae-associated biotype of rabies virus into an expanded wildlife host range in Southern Africa. Virus Genes 15: 79–82.

O'REILLY, L. M., AND C. J. DABORN. 1995. The epidemiology of *Mycobacterium bovis* infection in animals and man: A review. Tubercle and Lung Disease 76 (Suppl. 1): 1–46.

SCHLESSELMAN, J. J. 1982. Case control studies: Design, conduct, analysis. Oxford University Press, Oxford, UK, 354 pp.

TULLY, J. G. 1996. Mollicute-host interrelationships: Current concepts and diagnostic implications. *In* Molecular and diagnostic procedures in mycoplasmology, Vol. pp. 2, J. G. Tully and S. Razin (eds.). Academic Press, San Diego, California, 1–21.

WELLS, J. V., K. V. ROSENBERG, E. H. DUNN, D. L. TESSAGLIA-HYMES, AND A. A. DHONDT. 1998. Feeder counts as indicators of spatial and temporal variation in winter abundance of resident birds. Journal of Field Ornithology 69: 577–586.

WILLIAMS, D. 1997. Ophthalmology. *In* Avian medicine: Principles and application. Abridged edition, B. W. Ritchie, G. J. Harrison and L. Harrison (eds.). Wingers Publishing, Lake Worth, Florida, pp. 352–360.

Received for publication 14 February 2000.