

Dynamics of Mycoplasmal Conjunctivitis in the Native and Introduced Range of the Host

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Abstract: In 1994, *Mycoplasma gallisepticum*, a common bacterial poultry pathogen, caused an epidemic in house finches in the eastern part of their North American range where the species had been introduced in the 1940s. Birds with mycoplasmal conjunctivitis were reported across the entire eastern United States within 3–4 years. Here we track the course of the *Mycoplasma gallisepticum* epidemic as it reached native, western North American populations of the house finch. In 2002, *Mycoplasma gallisepticum* was first observed in a native house finch population in Missoula, MT, where it gradually increased in prevalence during the next 2 years. Concurrently, house finches with conjunctivitis were reported with increasing number in the Pacific Northwest. In native populations of the host, the epidemic expanded more slowly, and reached lower levels of prevalence than in the eastern, introduced range of the species. Maximal prevalence was about half in the Missoula population than in local populations in the East. Although many factors can contribute to these differences, we argue that it is most likely the higher genetic heterogeneity in western than in eastern populations caused the lower impact of the pathogen.

Key words: *Mycoplasma gallisepticum*, *Carpodacus mexicanus*, house finch, genetic bottleneck, epidemic, House Finch Disease Survey

INTRODUCTION

Identifying variation in host-pathogen dynamics may allow researchers to generate hypotheses regarding key features affecting the influence of pathogens on host populations. One host-disease system in which spatial variation can be examined is the interaction between the bacterium *Myco-*

plasma gallisepticum (MG) and a wild songbird, the house finch (*Carpodacus mexicanus*). In 1994, a novel strain of a common poultry pathogen MG, which causes severe conjunctivitis in house finches, emerged in the Washington, DC area and spread rapidly across the eastern North American range of the host species (Fischer et al., 1997; Dhondt et al., 1998). Within a year after its emergence, mycoplasmal conjunctivitis was found across a region of roughly 800,000 km², and within 3 years after its emergence, the disease had spread over most of the eastern range

of the host species, roughly 3,000,000 km² (Fischer et al., 1997; Dhondt et al., 1998). The epidemic caused a severe decrease in host abundance both in local populations (Nolan et al., 1998) and across the entire eastern range of the species (Hochachka and Dhondt, 2000). Using a volunteer-based monitoring scheme, the House Finch Disease Survey (HFDS), Dhondt et al. (1998) were able to document this rapid expansion of the epidemic on a monthly basis. Once the disease had become established, regular seasonal variation in prevalence occurred in local populations (Hartup et al., 2001, Dhondt et al., 2005) and at a regional scale (Altizer et al., 2004).

An apparent difference in host-pathogen dynamics has emerged following the first reports in April 2002 of mycoplasmal conjunctivitis caused by the house-finch strain of MG in western, native house finches. These reports came from a finch population in Missoula, MT that had been studied continuously since 1993 (Duckworth et al., 2003). By April 2004, MG was also confirmed in house finches from Portland, OR (Ley et al., in press). In 2000, we expanded the HFDS in anticipation of the arrival of MG in western North America to document the expansion of the pathogen once it reached the native range of the host species. In this article, we describe the geographic expansion of mycoplasmal conjunctivitis into native, western house finches using HFDS data, and then contrast the disease dynamics between eastern, introduced house finches and western, native populations using prevalence data collected at both local and broad population scales.

MATERIALS AND METHODS

Western Local Population Study

Prevalence data from Missoula, MT were collected in the context of a detailed, long-term population study using individually banded and re-observed house finches in a local population studied since 1993. Detailed study methods are described in Badyaev and Martin (2000). For that population, there are observations for all months except November and December. As a measure of disease prevalence, we calculated the percentage of individuals that were observed with conjunctivitis in the population in any given half-month.

Broad Scale Disease Survey

To describe the expansion of mycoplasmal conjunctivitis in house finches and to measure its prevalence, we used

data collected by thousands of volunteers who participated in the House Finch Disease Survey (HFDS), and Project Feeder Watch (PFW) (Wells et al., 1998; Lepage and Francis, 2002). The methodology of the HFDS is described in detail in Dhondt et al. (1998). In brief, participants can report on a daily basis if they observed house finches and if any observed finches had signs of conjunctivitis. PFW data are reports of observations made at weekly (minimum) intervals, in which observers entering their data over the Internet can specify both the total number of house finches observed as well as the number of house finches with conjunctivitis. PFW disease survey data are only used when observers explicitly report a number of diseased birds (zero or more). In western North America, we defined three geographic regions that had roughly equal numbers of participants: the Northwest (British Columbia, Washington, Oregon, Alberta, Montana, Idaho, and Wyoming), California, and the Southwest (Nevada, Utah, Colorado, Arizona, and New Mexico). Following Altizer et al. (2004), we calculated the proportion of “sick-days” as a monthly index of disease prevalence in each region as the number of days on which a participant observed one or more diseased house finches divided by the number of days on which the participant saw any house finches. We combined data from all participants in a region during a single month and we only included months for which at least 30 observers reported in a region, thereby limiting data on prevalence to the period November–April. The percentage of birds with conjunctivitis in a local population is correlated with the proportion of participants reporting diseased birds (Dhondt et al., 1998) and with the proportion of sick-days in a region (Altizer et al., 2004), so that variation in all three measures reflects variation in disease prevalence, although the absolute levels around which prevalence values vary cannot be compared directly.

Statistical Analyses

We used SigmaStat 3.1 (Systat Software, Point Richmond, CA, 2004) to carry out χ^2 tests and a two-way analysis of variance with the Holm-Sidak test for pairwise posteriori comparisons of means. We used SAS 9.0 to carry out a logistic regression to compare the change in disease prevalence between regions, modeling the probability of disease as a function of year, region, and a year \times region interaction. A significant interaction term would indicate that the change in prevalence from the year before to the year after

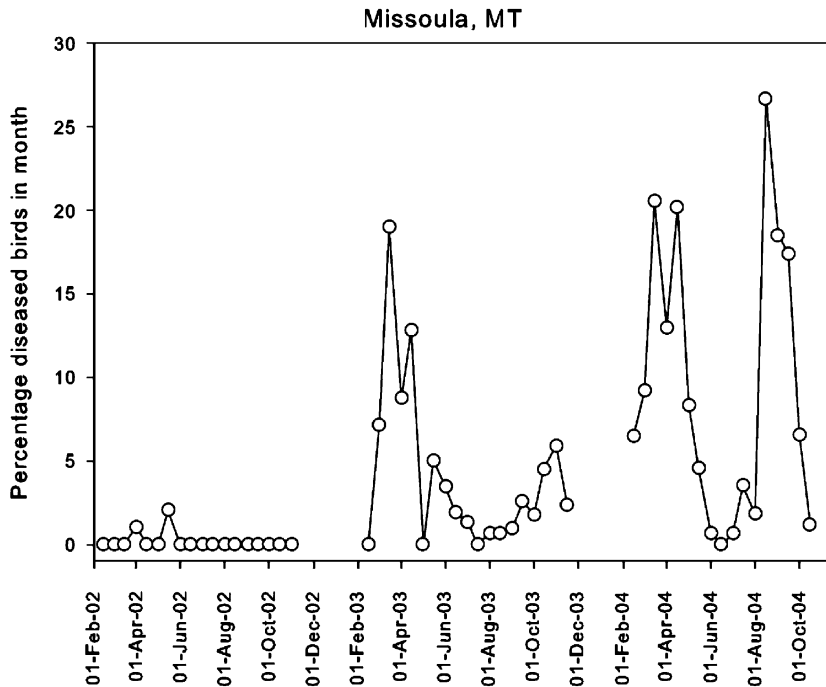


Figure 1. Prevalence of conjunctivitis calculated as the percentage of house finches with disease in a local population in Missoula, Montana (MT). All birds were individually banded, and re-observed frequently. The percentages were calculated per half month. Note how the disease prevalence increases slowly, reaching peak values in spring in all 3 years.

the epidemic started in a region would differ between regions. The critical value for acceptance of statistically significant results was set at $\alpha = 0.05$.

RESULTS

Prevalence in a Local Western Population

In Missoula, MT diseased birds were observed for the first time during April–June 2002 when eight birds were seen with conjunctivitis consistent with MG infection (confirmed by MG PCR) (Duckworth et al., 2003). No diseased birds were observed between July 2002 and February 2003, after which prevalence rose rapidly to a peak of 19% diseased individuals during the second half of March (Fig. 1; difference spring 2002–2003: $\chi^2 = 23.6$, 1 df, $P < 0.0001$). Prevalence gradually decreased during the breeding season and again returned to 0% in July. This was followed by a low fall peak reaching 5.9% in the first half of November (July–Nov 2003: $\chi^2 = 30.4$, 1 df, $P < 0.0001$). In spring 2004, prevalence peaked at 20.5% during the second half of March, a level similar to that in the previous year (spring 2003–2004: $\chi^2 = 0.02$, 1 df, NS) and declined again to 0% in the second half of June. The fall of 2004 peak, however, was much higher than in the previous year reaching 26.7% in the second half of August (fall 2003–fall 2004: $\chi^2 = 28.7$, 1 df, $P < 0.001$). In 2004, seasonal variation in disease prevalence was similar to that in eastern populations (Al-

tizer et al., 2004; Dhondt et al., 2005) with both fall and spring peaks, and breeding season and mid-winter minima.

Even 3 years after the arrival of MG in the Missoula population, the maximum prevalence values remained much lower than those in a banded eastern population where a prevalence of 60% was observed within less than a year after conjunctivitis was first observed (Nolan et al., 1998).

Prevalence at a Regional Scale

Although MG was not confirmed in western house finches until 2002, frequent observations of house finches with conjunctivitis were already reported in 2000 in all western regions (Table 1). Between 2000 and 2002, the proportion of sick-days per winter averaged 5.84% in the Northwest, 3.16% in California, and 2.73% in the Southwest. We believe that this background level of conjunctivitis reflects avian pox lesions around the eyes, whose physical signs may easily be mistaken for MG infections, even by trained observers. We use these background levels as a baseline and assume that any dramatic increases in baseline levels following 2002 (when MG was first documented) represent changes in MG prevalence. In November 2003, about 15 months after the first reported case in Missoula, a rapid increase in conjunctivitis prevalence was observed in the Northwest with prevalence values of up to 14.0% in late winter and early spring. A similar pattern unfolded in the

Table 1. Conjunctivitis Prevalence per Winter in Three Western Regions

Region mean winter	CA		SW		NW	
	SE	Mean	SE	Mean	SE	Mean
2000–01	3.41	0.69	4.37	0.58	7.25	0.83
2001–02	2.28	0.52	1.87	0.63	6.07	0.80
2002–03	3.78	0.67	1.95	0.27	4.21	0.75
2003–04	2.77	0.64	1.97	0.70	11.35	0.97
2004–05	5.10	1.20	1.98	0.22	10.11	0.66

CA, California; SW, southwestern region as defined in Materials and Methods; NW, northwestern region.

Table 2. Results of a Two-way Analysis of Variance Comparing Prevalence per Winter in Three Western Regions Using Monthly Prevalence Values

Source of variation	DF	SS	MS	F	P
Region	2	486.658	243.329	79.725	<0.001
Winter	4	91.846	22.962	7.523	<0.001
Region × winter	8	169.548	21.193	6.944	<0.001
Residual	75	228.907	3.052		
Total	89	976.958	10.977		

DF, degrees of freedom; SS, sum of squares; MS, mean square.

following winter 2004–2005. Through the course of both winters, prevalence increased about twofold above background levels in the Northwest, while it remained at background level in California and in the Southwest (Table 1). The more formal analysis of the data, with a two-way analysis of variance with winter and region as factors, showed that both main effects (year and region) and their interaction were significant (Table 2). A pairwise comparison of the means at the 5% level showed no significant differences between winters in California or in the Southwest. In the Northwest, however, the mean prevalence values of winters 2003–2004 and 2004–2005 did not differ from each other, but were significantly different from the winters 2000–2001, 2001–2002, and 2002–2003, confirming that prevalence increased only in that region in the winter 2003–2004.

At the regional scale (Fig. 2), prevalence increased much more rapidly in eastern, introduced populations than in western, native populations (Fig. 2). Thus, for example, the proportion of sick-days in Ohio, Illinois, and Indiana combined increased from 0.87% in February 1995 to 27.90% in February 1996, compared to a rise in the Northwest between February 2002 and February 2003 from

6.8% to 14.2% (logistic regression year × region interaction term Wald- $\chi^2 = 9.311$, $df = 1$, $P = 0.0023$). The rate of geographic expansion also differed between introduced and native regions: the epidemic covered 800,000 km² 10 months after first being observed in the eastern United States and almost 3,000,000 km² 3 years after its emergence (Dhondt et al., 1998). In the western United States, the disease still did not cover more than about 1,000,000 km² 3 years after first being observed in the region.

DISCUSSION AND CONCLUSIONS

Our data suggest that the expansion of the MG epidemic was slow in western North America. After the first cases of mycoplasmal conjunctivitis appeared in house finches in 2002 in Missoula, MT, the epidemic gradually expanded further west during subsequent years. Conjunctival samples in April 2004 and in March 2005 from house finches with conjunctivitis captured in Portland, OR, 800 km west of Missoula, were diagnosed PCR-positive for MG (Ley et al., in press). MG in house finches thus reached western coastal states, and it is therefore likely that the increase in preva-

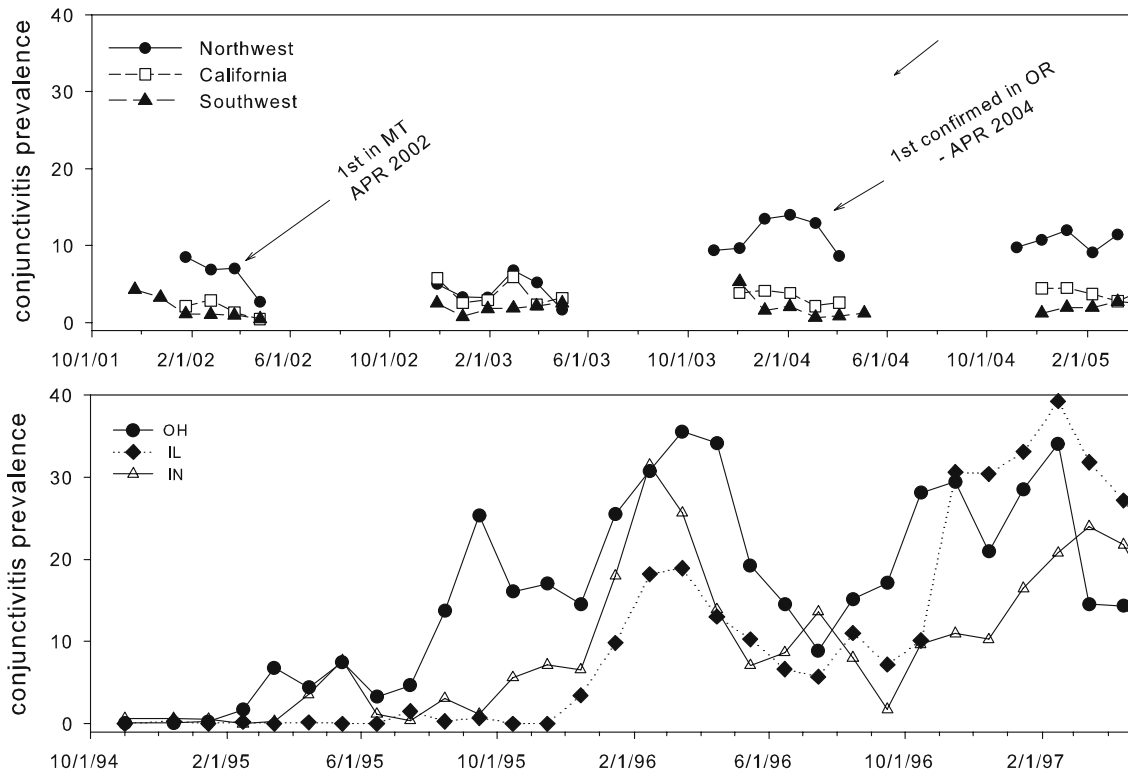


Figure 2. Prevalence of conjunctivitis expressed as the percentage of sick days (see text) in three regions of western North America and in three eastern states (OH = Ohio, IL = Illinois, IN = Indiana). Avian pox can cause lesions around the eyes that are very similar to those caused by *Mycoplasma gallisepticum* (MG) infections, and we argue

that the relatively high pre-MG levels of conjunctivitis reported by participants of the House Finch Disease Survey reflect this. Beginning in the winter of 2003–2004, prevalence of conjunctivitis increased markedly in the Northwest (where MG had been detected in MT and in Oregon [OR]).

lence of conjunctivitis in house finches in the Northwest was the result of the westward progression of the epidemic.

Similarly, even at a local level, the increase in disease prevalence and maximum prevalence in Missoula, MT (Fig. 1) was slower than that seen in intensively-studied eastern populations. Less than a year after the first birds with conjunctivitis were observed in Auburn, AL, 60% of the house finches captured for banding and observed at feeders showed signs of mycoplasmal conjunctivitis (Nolan et al., 1998). In another eastern population in Atlanta, GA, in 2001, 6–7 years after MG reached the area, the peak prevalence still surpassed 50% of the birds trapped (Dhondt et al., 2005). In contrast, in Missoula, 1 year after the epidemic started, only about 20% of the birds showed disease, and 2.5 years after mycoplasmal conjunctivitis was first observed, only 26% of individuals had conjunctivitis. In the Missoula population, therefore, prevalence increased at a much lower rate and reached a much lower maximum level than in eastern populations (Fig. 1). Not only did the epidemic expand much more slowly in the West and

debilitated a smaller proportion of individuals in local populations, we also have no evidence, so far, that house finch abundance declined in the Northwest following the MG epidemic. It may, however, be too soon to detect such an effect because, in the East, a 60% decline in house finch numbers was apparent 2.5–3 years after the epidemic reached high prevalence levels (Hochachka and Dhondt, 2000).

In the West, the presence of avian pox complicated description of changing prevalence of MG-induced conjunctivitis. Avian pox may cause physical signs that are difficult to distinguish from those caused by MG. McClure (1989) described the prevalence of avian pox based on 11,000 house finches trapped in Ventura County, CA. He noted a high prevalence of pox, especially in winter. Although most lesions were on the legs or feet, about 10% of infected birds showed lesions around the bill and 8% around the eyes. These latter lesions could easily be confounded with the physical signs caused by MG, even by a trained observer. In fact, we sampled the eyes of three

house finches showing distinct signs of conjunctivitis in Lafayette Co., CA during March of 2004, but all tested negative for MG by our PCR-based assay. This led us to examine changes in prevalence of conjunctivitis relative to baseline (presumably pox-induced) levels of conjunctivitis in western North America (Table 1) before MG was found in this region. In the eastern United States, pox lesions are rare (Hartup et al., 2004) [and own observations]. We believe HFDS data described the expansion of the MG epidemic in the western United States because HFDS data did not yet show an increase in conjunctivitis prevalence in California nor in the Southwest (Fig. 2), whereas we found a substantial increase in prevalence in the Northwest, where the presence of MG had also been confirmed by PCR testing both in Montana and in Oregon.

Understanding the factors that mediate the likelihood of pathogen epidemics in host populations remains a question of general and timely importance in disease ecology (Woolhouse et al., 2005). The factors that influence the likelihood that pathogens cause major epidemics in their hosts include variation in host ecology, cross immunity, behavior, and genetics, as well as changes in pathogen genetics (Woolhouse et al., 2005). A small number of field studies have shown that in the same population, genetically less variable individuals are more likely to be affected by pathogens than those that are more genetically variable (Lively et al., 1990; Coltman et al., 1999; Acevedo-Whitehouse et al., 2003; MacDougall-Shackleton et al., 2005). Similarly, only a small number of studies have related genetic diversity at the population-level in geographically distinct populations with parasite burdens (Meagher, 1999) or pathogen susceptibility (Pearman and Garner, 2005). Assuming that pathogen success is mitigated by host genetic variation, it is particularly interesting to compare epidemics in different parts of a host's range between which genetic diversity differs, although no studies have done this so far.

Although our evidence is indirect, and other factors certainly contributed to differences in MG expansion in eastern and western populations, we believe that our data strongly suggest that genetic factors have played a role. Five sampled eastern populations of house finches harbor, on average, 17.5% lower allelic richness and 4.9 % lower heterozygosity levels than four western populations (Hawley et al., 2006). These genetic differences almost certainly resulted from the strong demographic bottleneck that occurred during the introduction of house finches to the eastern United States (Elliott and Arbib, 1953). Further-

more, variation in disease susceptibility among individuals in the eastern population has been directly linked to differences in heterozygosity (Hawley et al., 2005), indicating that genetic variation at the scale of the individual mediates disease susceptibility in this system. Our disease prevalence data suggest that relationships between genetic variation and disease susceptibility may be equally important at the population level. Our results are consistent with the idea that the epidemic of the emerging mycoplasmal conjunctivitis expanded more slowly, and that a smaller proportion of birds developed disease among genetically more variable native birds than among the genetically less variable introduced host populations that passed through a demographic bottleneck after introduction.

However, as Woolhouse et al. (2005) pointed out, non-genetic factors can also contribute to differences in R_0 and hence in the rate at which emerging diseases spread. These include host behavior, host ecology, pathogen genetics, pathogen ecology, and host cross immunity. As regards host ecology, two factors could slow down the rate at which the epidemic spread among western house finches compared to eastern birds: western populations, if they migrate at all, exhibit lower prevalence of migration than eastern birds (Able and Belthoff, 1998). The geography of the West, with high mountain ranges running North–South could reduce dispersal from East to West. These factors could explain differences in the rate of spread of the pathogen, but would not explain why the increase in prevalence in a local population was slower. Additionally, house finches in the West are less clumped in urban and suburban environments than in the East, and overall density and social group sizes appear to be lower (unpublished data from PFW). Both factors would affect transmission rates and pathogen persistence, and could explain differences both in the rate of spread and in the prevalence level reached.

Pathogen-related differences may also account for some of the observed differences in disease dynamics. We know very little about pathogen genetics and ecology including whether the MG strain that arrived in the West is genetically different. Given that genetic variation is appearing among MG samples taken in the East, examining the genetics of the pathogen in native house finch populations as well as its resulting disease course in the host could yield important insights (Pillai et al., 2003; Cherry et al., in press). Finally, climatic conditions in the West could be sufficiently different from those in the East to affect pathogen fomite survival outside the host and thereby influence the probability of disease transmission. MG sur-

vival on dry surfaces, as are characteristic in the western United States, is longer at lower temperatures than at higher temperatures in experimental conditions (Nagatomo et al., 2001). Finally, the higher prevalence of other pathogens in western populations (such as avian pox) might be partially protective against the novel pathogen, thereby slowing down its spread and reducing the R_0 value.

Although some or all of these factors might contribute to differences in the epidemic in eastern and western house finches, the slow rate at which disease prevalence increased in the local population in Missoula, where the density is high and where avian pox has not been observed, strongly suggests that host genetic factors are the main cause of the differences in pathogen-host dynamics in the different parts of the host range. Further research, however, is needed to determine the relative importance of all factors that may contribute to the observed differences in disease expression in native and introduced parts of the host range.

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