Density-dependent decline of host abundance resulting from a new infectious disease

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Although many new diseases have emerged within the past 2 decades [Cohen, M. L. (1998) *Brit. Med. Bull.* 54, 523–532], attributing low numbers of animal hosts to the existence of even a new pathogen is problematic. This is because very rarely does one have data on host abundance before and after the epizootic as well as detailed descriptions of pathogen prevalence [Dobson, A. P. & Hudson, P. J. (1985) in *Ecology of Infectious Diseases in Natural Populations*, eds. Grenfell, B. T. & Dobson, A. P. (Cambridge Univ. Press, Cambridge, U.K.), pp. 52–89]. Month by month we tracked the spread of the epizootic of an apparently novel strain of a widespread poultry pathogen, *Mycoplasma gallisepticum*, through a previously unknown host, the house finch, whose abundance has been monitored over past decades. Here we are able to demonstrate a causal relationship between high disease prevalence and declining house finch abundance throughout the eastern half of North America because the epizootic reached different parts of the house finch range at different times. Three years after the epizootic arrived, house finch abundance stabilized at similar levels, although house finch abundance had been high and stable in some areas but low and rapidly increasing in others. This result, not previously documented in wild populations, is as expected from theory if transmission of the disease was density dependent.

T heory predicts that the effects of disease on abundance of hosts can be highly variable (1), with effects ranging from highly unstable oscillations in abundance of the host to stable, density-dependent regulation of hosts. However, this entire range of host–disease dynamics has not been observed in the field, and the importance of disease in determining abundance of wild animals is not well understood (2). In extreme cases (3–5), the effects of disease on host abundance are obvious: host populations collapse, and potentially, species may go extinct (6). However, subtler effects of disease may be more common than these extreme cases, with changes in size of host populations leading to changes in the rate of disease transmission and thus to oscillating or smaller but stable host populations (1). However, these less obvious effects of disease can be difficult to detect. Diseases have only rarely been linked with cyclic changes in host abundance (7), and to date, we are unaware of any studies that have demonstrated nonoscillatory density-dependent control of host abundance by a disease. Potential reasons for this lack of empirical evidence of diseases regulating host populations (8) are that diseases are endemic in host populations well before research begins, and the presence of these diseases may be difficult to detect.

Among the many new diseases that have emerged within the past 2 decades (9) is one system involving the bacterium *Mycoplasma gallisepticum* and an avian host, the house finch (*Carpodacus mexicanus*). This system is unusual (10) because data are available on host abundance before and after the start of the epizootic together with detailed descriptions of pathogen prevalence. The house finch is a small (20 g) passerine bird species native to the western half of North America with an approximate generation time of 1.5–2 years. In 1940, a small number of house finches originating from the west coast of the United States were released in Long Island, NY (40.8° N, 73.3° W). After the cold winter of 1947–1948, only about 80 individuals survived (11).

Beginning in the 1960s, the population rapidly expanded its range and by 1995, the eastern population met the eastward-expanding natural population of the species at about 95° west longitude (12). In many parts of their range, but especially in the Midwestern states, house finch numbers were still increasing rapidly in the mid-1990s [ref. 13; http://www.mbr-pwrc.usgs.gov/bbs/bbs.html (accessed June 29, 1999)]. In the 1993–1994 winter, cases of conjunctivitis began being reported in house finches in the region around Washington, DC. The disease was caused by a previously undescribed strain (14) of *M. gallisepticum*, a pathogen of poultry with a worldwide distribution that was first described in North America in 1936 and that had not previously been considered a pathogen of wild passerines (15). This pathogen apparently also infects American goldfinch (*Carduelis tristis*), purple finch (*Carpodacus purpureus*), and house sparrow (*Passer domesticus*) (16), although at far lower rates than house finches. House finches naturally infected with *M. gallisepticum* and held in captivity with ad libitum food developed conjunctivitis within 2–4 weeks, lost weight rapidly, and many (17) or all (18) died. This led to the suggestion (18) that the epizootic should have major and very rapid effects on the numbers of free-living birds in areas of high *M. gallisepticum* prevalence.

In our research, we have monitored the spread of mycoplasmal conjunctivitis and its effects on house finches across the extent of the epizootic, an area encompassing much of the eastern United States and areas of southeastern Canada. We are able to show that, wherever house finches were previously abundant, they have declined in numbers after the arrival of the disease. However, population declines have not been as catastrophic as predicted by studies of captive birds (17, 18). Instead, populations only declined when at higher densities, and numbers decreased gradually and seemed to have stabilized at a new lower level. This pattern is consistent with mycoplasmal conjunctivitis causing density-dependent limitation of house finches.

Methods

We were able to associate mycoplasmal conjunctivitis with population declines in house finches because of an established bird-monitoring program and the rapid initiation of a new program to monitor mycoplasmal conjunctivitis. The North American Christmas Bird Count (CBC) (19) was used to monitor abundance of house finches, with abundance indexed as the number of birds per group of participants per hour (19). We tracked the epizootic month by month from November 1994 onward by using data from the House Finch Disease Survey (HFDS) (20). The HFDS asked volunteer participants to report

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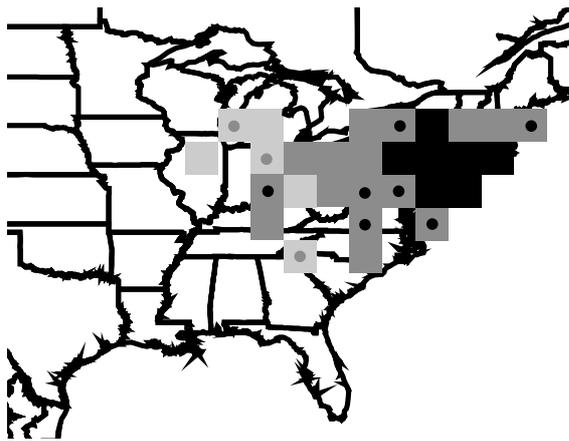
Fig. 1. Time when ≥ 20% disease prevalence was reached in 29 2° × 2° blocks in eastern North America from which data were analyzed. The darkest shading indicates ≥ 20% prevalence in 1994, midtone shading indicates ≥ 20% in 1995, and lightest shading indicates ≥ 20% prevalence in 1996. Darker circles within the center of a square indicate that the epizootic achieved ≥ 20% prevalence in the first half of a year. The data from each block were treated as one observation unit.

the presence or absence of house finches exhibiting clinical signs of conjunctivitis at a specific location, generally a bird-feeding station in the yard of the reporter. Each reporting period for each survey was a single calendar month within a year, with observers sending in surveys for multiple periods, up to 48 months.

Initial host population sizes, host population dynamics, and disease dynamics all varied across eastern North America. To deal with this spatial variability, we divided the area under study into 2° × 2° blocks (equal to 222.2 km × 157.6 km at 40° latitude), centered on odd-numbered latitudes and longitudes. Data within each of these blocks (Fig. 1) were treated as separate time series for all analyses.

We had to choose which of two data sets we would use to provide our indices of abundance of house finches. The two available data sets describing continentwide bird abundance in North America are: the North American Breeding Bird Survey (BBS) (21) describing breeding season abundance and the CBC (19) describing winter abundance. Changes in abundance of house finches from the two surveys are highly correlated; the median $r^2 = 0.68$ across the 29 blocks in eastern North America for which both BBS and CBC data were available (Fig. 1). BBS data in the analysis were averages of raw BBS counts within regions, and CBC data were regional averages of birds per party per hour. Because CBC and BBS data are highly correlated and CBC data are available including the 1998–1999 winter but the BBS data are only available through 1997, we used the former to describe changes in house finch numbers among years.

House finches are still colonizing many parts of eastern North America, and the abundance of the finches has been increasing even in areas in which the finches have been established for over 2 decades. Thus, we could not measure the impact of the epidemic on host abundance by simply comparing abundance of house finches pre- and postconjunctivitis. Instead, we compared observed abundance to the abundance expected if the disease had not had any effect on house finch numbers. The earliest data used to calculate expected population sizes came from 1966, the year in which the BBS was initiated (21). In blocks in which house finches were established before 1966, data were used from a CBC count circle only if that count was established in or before 1966. Where house finches colonized a block after 1966, CBC count circles were only used if they were initiated during or previous to the year that house finches arrived. The annual index

of house finch abundance within each 2° × 2° block was the average number of birds per party per hour over all suitable count circles in that block. We fit Gompertz growth equations (by using PROC NLIN of SAS) (22) to the preconjunctivitis time series from each 2° × 2° block. We determined that all obvious and systematic changes in population size followed forms that were part or all of an increasing sigmoid curve trajectory, by plotting our data. We used Gompertz growth equations, which fit a wide range of sigmoid shapes and are commonly used to describe asymptotic growth (23), to describe population growth trajectories. Our regression equations allowed us to extrapolate expected population sizes of house finches in postdisease years assuming the epizootic had not had any effect on abundance, and express the observed house finch abundance as a percentage of the calculated expected abundance. We only used data from the 29 2° × 2° blocks in which the Gompertz regression explained better than 90% of the variance in the data and for which house finch abundance reached sustained levels of greater than one bird per party per hour. Blocks that did not meet these criteria appeared to contain data with substantial variation resulting from sampling error.

To relate disease prevalence to changes in house finch abundance within 2° × 2° blocks of latitude and longitude, we calculated a "regional prevalence" (the chance to see a diseased bird during a given month at a given site in a 2° × 2° block) of the disease. The presence-absence data from individual feeders were converted to a smoothed map of regional prevalence for each month of the survey by using the procedure described (20). For each 2° × 2° block, we were able to determine in which month and year the regional prevalence reached an arbitrary but biologically informative (20) threshold of 20%. Although only presence-absence data are available, work (20) has shown that a larger proportion of house finches are infected in regions in which a greater proportion of feeders is visited by infected birds. Thus, all references to high regional prevalence of mycoplasmal conjunctivitis also indicate a high prevalence of the disease among birds at individual feeders.

The epizootic began at different times in different locations in eastern North America (20, 24, 25), and within two and one-half years after the disease appeared, the epizootic had spread through most of the house finch range in eastern North America, reaching and maintaining high levels of prevalence everywhere. Regional prevalence of the disease could have initially reached threshold levels in any month of the year. However, we treated time of arrival as a semicontinuous variable, treating time since arrival of the disease as either full or half years. If the disease threshold value was reached during January–June of the year of the December CBC count (12 blocks), we considered the disease to have been present for one year. If the disease threshold value was reached during July–December of the year of the December CBC count (17 blocks), we considered the disease to have been in high prevalence 0.5 year at the time of the CBC. The threshold prevalence was reached in seven blocks in 1994, seven during the first half of 1995, nine during the second half of 1995, and in another three each in the first and last half of 1996 (Fig. 1). There were no "control" blocks in which mycoplasmal conjunctivitis failed to reach threshold prevalence.

Data were analyzed by using PROC MIXED of SAS (26). In our analysis of population trends in Fig. 2, we treated the number of years since disease arrival as a continuous log-transformed variable (ln[N_Years + 0.5]), and year of disease arrival as a categorical variable. The interaction term between these variables was also included in the model. Changes in indices of abundance with year of disease arrival (Fig. 3) were tested as follows. The year of disease arrival was treated as a continuous variable, the time period (immediately before disease, 2.5–3 years after disease) as categorical. An analysis of covariance with interaction term was used. In both ANCOVAs, repeated measures

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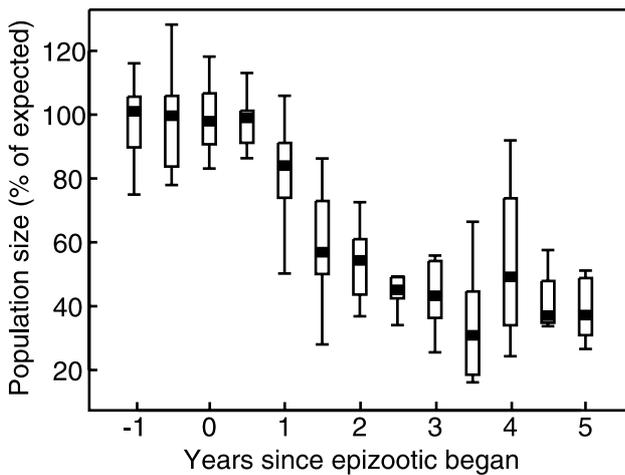
Fig. 2. Changes in abundance of house finches after the arrival of conjunctivitis in a region. Note that changes are expressed as a percentage of the expected abundance if mycoplasmal conjunctivitis did not affect house finch numbers (see *Methods*). Thick horizontal lines indicate medians and boxes span the interquartile range. The year 0 represents house finch abundance in the last year before the epizootic reaching the 20% threshold value in that 2° × 2° block. Data from five independent time series (three on integer years, two on half years; see text for details) are represented.

within 2° × 2° blocks were treated as a nested effect, and the analyses were run assuming unstructured covariance matrices.

Results

Geographic variation in arrival time of the disease allowed us to examine the impact of mycoplasmal conjunctivitis on host abundance in replicate areas, and uncoupled the effects of disease from those of calendar year *per se*. The abundance of house finches relative to their expected abundance declined at a decelerating rate in the 3 years after the disease reached 20% regional prevalence (Fig. 2). This pattern of slowing decline was statistically significant ($F = 10.4$; $df = 1,26$; $P < 0.003$; see *Methods* for description of analysis). This analysis also suggested that the pattern of population change may have differed geo-
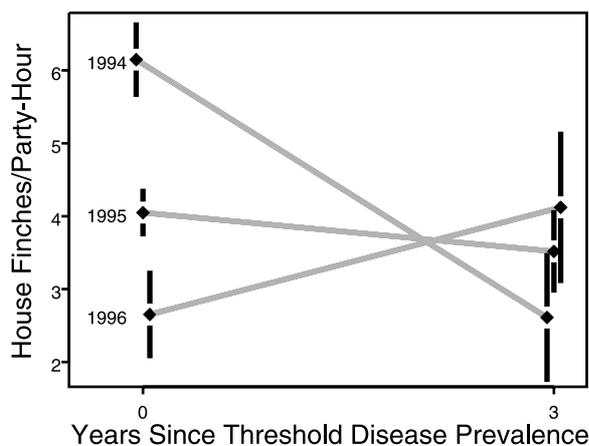
Fig. 3. Changes in house finch abundance in the three groups of 2° × 2° blocks where threshold disease prevalence was reached in 1994, and the first halves of 1995 and 1996. Abundance (± 1 SEM from an ANCOVA) of house finches is plotted as birds per party per hour (from CBC) immediately before and three years after regional prevalence of conjunctivitis reached 20%. The significant interaction term (see text) reflects the existence of significant differences in abundance before the epidemic, but the absence of significant differences 3 years later.

graphically because there was a nonsignificant but suggestive interaction between time of disease arrival and pattern of decline ($F = 2.5$; $df = 2,26$; $P = 0.11$). Areas which experienced the epizootic later also had lower abundances of house finches ($r^2 = 0.35$; $df = 1,25$; $P = 0.001$ in linear regression); thus, any geographic variation in population decline could also have been caused by population density of house finches and not time *per se*.

The variation in the pattern of decline was consistent with the predicted existence of density-dependent variation in the rate of disease transmission (1, 2). Population sizes were found to decrease in areas in which the disease arrived earliest while remaining stable or even tending to increase in the region reached latest by the disease ($F = 10.8$; $df = 1,25$; $P = 0.003$ for interaction term, in an ANCOVA comparing population sizes immediately before and 3 years after initial high prevalence of the disease; Fig. 3). Note that we used year of disease arrival as a surrogate for house finch abundance in our model because the abundance of house finches is the dependent variable in our model.

Discussion

Our results suggest both that declines in abundance of house finches were caused by mycoplasmal conjunctivitis, and that these declines were density dependent. We can causally associate the disease with population declines of house finches (Fig. 2), because the declines started in different years and occurred over a very large area (Fig. 1). The higher rate of decline in blocks in which the disease arrived earliest (Fig. 3) suggests density-dependent transmission of the disease, because house finch abundance was also generally higher in blocks in which the disease was present earliest. We could not directly relate the rate of decline to initial population density of house finches because such an analysis would not be interpretable. The resulting analysis would have been biased because the initial density of house finches would be both an independent variable and part of the dependent variable.

An alternative explanation for the apparent density dependence is a change in virulence of *M. gallisepticum* through time, as has been found for other diseases (3). However, we feel that this alternative is unlikely for three reasons. First, there is no evidence that the disease has undergone genetic change over the course of the epizootic (27). Second, over 2 years after arrival of the disease in one site, a high proportion of birds that were infected with *M. gallisepticum* also displayed clinical signs of the disease [ref. 27; http://www.vet.uga.edu/ivcvm/1999/hartup/hartup.htm (Feb. 2000)]; our interpretation is that when the bacterium is present, it continues to be virulent. Third, house finch populations in high-density areas continued to decline 2 years after the first appearance of the disease (Fig. 2), which is the same time period in which lower-density populations of house finches first encountered the disease but failed to show systematic declines (Fig. 3). Continued high virulence near the point of origin with lower virulence farther from this point seems unlikely.

Our results, in addition to suggesting density dependence in host mortality, also provide the first large-scale evidence for the impact of a nonintroduced disease on the population dynamics of a host. Whereas other diseases of wild animals have been studied at large spatial scales (3), all previous studies have been on disease organisms deliberately chosen for their high transmission rates and virulence. The efficiency with which introduced diseases kill their hosts (4, 5) results in local extinction of the disease organism in the short term, and potentially evolution of lower virulence through time (3). In contrast, mycoplasmal conjunctivitis was, from the beginning, either less virulent or with lower transmission rates than many diseases (3–5). Given the evidence (17, 18) of high virulence from laboratory studies,

and the density-dependent variation in the rate of population decline (Fig. 3), our data may indicate that low rates of disease transmission caused much of the observed slow decline and apparent subsequent stability (Fig. 2) in host numbers. Our large-scale monitoring can identify patterns and infer causes; studies of narrower scope are now needed to confirm the mechanisms inferred by our work.

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