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Seasonal dynamics of mycoplasmal conjunctivitis in eastern North American house finches

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Summary

1. *Mycoplasma gallisepticum* is an emerging eye disease that spread rapidly among wild house finches (*Carpodacus mexicanus*) in the eastern United States following initial reports of infected birds in 1994. The hallmark signs of infection have allowed systematic monitoring of disease at both local and continent-wide scales for more than 7 years since the onset of the epidemic.
2. Using data collected by a network of citizen science volunteers, we examined both long-term trends and seasonal dynamics of mycoplasmal conjunctivitis in three different climatic regions in eastern North America over a 77-month period (November 1994–March 2001).
3. Time-series prevalence data from all three regions suggest that following establishment, marked seasonal fluctuations in prevalence each year were characterized by autumn–winter epidemics (in October and February) and consistent summer declines (with prevalence falling close to zero from May to July).
4. The maximum peak, annual rates of increase and timing of epidemics varied among three geographical locations that were delineated by minimum winter temperatures. Annual autumn prevalence in the South increased more rapidly, and maximum prevalence was nearly three times greater in the South than in the colder North and Central regions.
5. Longer-term trends showed evidence for multiyear fluctuations in prevalence that were characterized by greater amplitude in the southern region.
6. Finally, monthly estimates of house finch flock sizes derived from a similar citizen science data set showed that winter flock sizes were associated positively with average monthly prevalence in the northern and central regions, although regional differences in flock sizes did not correspond to regional differences in maximum prevalence.
7. This study represents the first evidence of multiyear fluctuations, regional differences and highly predictable annual outbreaks of this recently emerged wildlife pathogen. Several factors associated with house finch life history and behaviour are likely to contribute to temporal and spatial variation in prevalence, including annual changes in host reproduction, social behaviour and environmental effects on host stress or immunocompetence.

Key-words: avian disease, *Carpodacus mexicanus*, citizen science, geographical variation, host–parasite dynamics, *Mycoplasma gallisepticum*.

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Introduction

In recent years, ecologists and conservation biologists have become increasingly concerned with emerging infectious diseases and the threats they pose to free-living wildlife (Daszak, Cunningham & Hyatt 2000; Friend, McLean & Dein 2001; Cleaveland *et al*. 2002). New and re-emerging pathogens such as canine distemper virus, seal morbillivirus and fungal chytridiomycosis have caused devastating declines of African lions, seals, and amphibians, respectively (Daszak *et al*. 1999, 2000; Packer *et al*. 1999; Jensen *et al*. 2002). Among birds, avian pox and malaria have been linked to marked losses in several Hawaiian forest birds (van Riper *et al*. 1986). Most recently, American crows and related species in North America have died in unusually large

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numbers following the emergence of West Nile virus in 1999 (O'Leary *et al*. 2002; Hochachka *et al*. 2003). Understanding the impacts, population dynamics and factors affecting the transmission of infectious diseases in natural populations will become increasingly important for wildlife conservation. However, despite detailed studies of the dynamics of human diseases increasingly sophisticated host–pathogen models, we know relatively little about factors driving changes in disease prevalence in wildlife populations (but see Hudson, Dobson & Newborn 1998; Begon *et al*. 1999; Hochachka & Dhondt 2000).

According to simple epidemiological models, periodic outbreaks or cyclical epidemics can result from intrinsic properties of the host and pathogen, including short infectious periods, rapid transmission, long latent phases and high pathogen-induced mortality (Anderson & May 1979, 1991). External drivers or seasonal factors can also lead to annual or multiyear outbreaks (Pascual *et al*. 2000; Dowell 2001). For example, seasonal changes in incidence have been reported for human diseases such as measles, rubella, whooping cough and influenza, with epidemics occurring at about the same time each year (reviewed in Dowell 2001). Among wildlife diseases, pathogens such as skunk rabies (Gremillion-Smith & Woolf 1988), helminth parasites infecting wood mice (Montgomery & Montgomery 1988), cowpox virus in voles and wood mice (Begon *et al*. 1999) and avian pox in California house finches (McClure 1989) also cause annual outbreaks, with rapid changes in prevalence coinciding with seasonal events. Despite the pervasiveness of annual epidemics and environmental factors that affect host–pathogen ecology, seasonal or climate-mediated drivers are not often incorporated into host–pathogen models (but see Bjørnstad, Finkenstädt & Grenfell 2002; Keeling & Grenfell 2002; Rodo *et al*. 2002).

Several factors might trigger seasonal changes in prevalence, including annual changes in climate or photoperiod that affect host or pathogen biology. For example, seasonal variation in host breeding biology (including hormonal changes or mating displays) could affect host immunocompetence or disease susceptibility (Hillgarth & Wingfield 1997; Dowell 2001; Duckworth, Mendonca & Hill 2001). Seasonal host reproduction could generate a 'pulse' of immunologically naive juveniles recruited into the population at approximately the same time each year. Annual changes in host social behaviour might alter parasite transmission through increased contacts or crowding of susceptible hosts (Gremillion-Smith & Woolf 1988; Bolker & Grenfell 1995). Finally, annual environmental changes in temperature, rainfall and winds have been linked directly to pathogen transmission and survival in other systems (Dowell 2001; Harvell *et al*. 2002).

Mycoplasma gallisepticum (Edward & Kanarek) is a new wildlife pathogen with major impacts on its avian host and complex local dynamics following establishment (Hochachka & Dhondt 2000; Hartup *et al*. 2001a). Following initial reports during the winter of 1993–94, this pathogen spread rapidly throughout house finch populations in eastern North America. The mycoplasmal conjunctivitis epidemic in house finches has been monitored almost continuously since its inception via a citizen science programme coordinated at the Cornell Laboratory of Ornithology (Dhondt, Tessaglia & Slothower 1998; Dhondt *et al*. 2001), providing the opportunity to examine host–pathogen dynamics on an unprecedented geographical and temporal scale. Recent work indicates that *M. gallisepticum* has persisted post-invasion and exhibits sharp fluctuations in prevalence at both local and regional scales (Hartup, Kollias & Ley 2000; Hartup *et al*. 2001a). The timing of these fluctuations is of great potential significance in revealing components of host ecology that trigger rapid changes in disease prevalence.

Our main objective was to characterize changes in the prevalence of conjunctivitis in wild house finches, focusing on both seasonal and long-term patterns. We also examined geographical variation in parasite dynamics among three regions in eastern North America to determine whether the timing and magnitude of epidemics varied among regions defined by climatic differences. Most importantly, we examined the timing of prevalence changes in each region relative to existing knowledge of house finch biology to identify a list of possible factors that might drive temporal and spatial variation in the disease prevalence.

Methods

NATURAL HISTORY OF THE HOST–PATHOGEN SYSTEM

Native to western North America, house finches (*Carpodacus mexicanus* Muller) spread rapidly throughout the eastern United States after a small number of birds were released in New York in 1940 (Bock & Lepthien 1976; Hill 1993). Although house finches in their native range occupy open habitats ranging from undisturbed deserts to urban areas, their affinity for bird feeders and habitual nesting around buildings has made them one of the most commonly sighted backyard birds in the eastern United States (Hill 1993; Hill, Nolan & Stoehr 1999). Whereas western house finches show no seasonal migration, the eastern population has developed a partial migration following introduction (Belthoff & Gauthreaux 1991; Able & Belthoff 1998) that involves south-western movement to wintering sites from November to February. House finch breeding biology is similar throughout their eastern range: pair formation begins in winter flocks between January and March, and breeding populations reach their maximum size by early April (Hill 1993). Egg laying occurs from March to July, with females laying up to six clutches throughout the summer and fledging as many as three broods of young (Hill 1993).

The increasing abundance of house finches in eastern North America has slowed or stopped in recent

years following the emergence and spread of mycoplasmal conjunctivitis (Luttrell *et al*. 1996; Hochachka & Dhondt 2000). This disease is caused by a novel strain of the bacterium *Mycoplasma gallisepticum* (MG), a common pathogen of domestic poultry (Ley & Yoder 1997) that was first recovered during the winter of 1993–94 from wild house finches near Washington DC (Fischer *et al*. 1997). Infected house finches usually develop eye infections (characterized by conjunctival swelling and discharge) within days following exposure, and clinical signs can persist for many weeks (Roberts, Nolan & Hill 2001a). This newly emerged pathogen has resulted in a decline in house finch abundance, so that in parts of their range, house finch numbers were only 40% of numbers expected if the disease had not appeared (Hochachka & Dhondt 2000). Some birds persist in an infectious carrier state, and recent evidence indicates that infected birds can recover in the wild (Roberts *et al*. 2001b).

Transmission of MG is thought to require contact between susceptible and infected hosts, although contact with contaminated feeders or fomites might also spread the disease (Ley & Yoder 1997). The tendency of house finches to flock and gather at feeding stations will probably increase host contact rates and transmission during winter months. Transmission at bird feeders could also contribute to spillover infections from house finches to other passerine birds (Hartup *et al*. 2001b), and other carduline finches appear to be susceptible, including American goldfinches, purple finches, and grosbeaks (Mikaelian *et al*. 2001).

LARGE-SCALE MONITORING AND THE HOUSE FINCH DISEASE SURVEY

Mycoplasmal conjunctivitis is well suited for citizen science monitoring because hosts commonly visit bird feeders throughout North America, and the bacterium is detected easily by the presence of clinical signs (Dhondt *et al*. 1998; Hartup *et al*. 2001a, 2001b). We tracked fluctuations in disease prevalence using data from the House Finch Disease Survey (HFDS), a citizen science project coordinated by the Cornell Laboratory of Ornithology and initiated in November 1994 (Fischer *et al*. 1997; Dhondt *et al*. 2001). Volunteer participants report monthly sightings of infected and healthy house finches at their bird feeders, and data are collated into a large database (currently over 300 000 observations) that includes one record per observer per month for each of several bird species (Dhondt *et al*. 1998). Additional data fields include each observer's geographical location, identification code, month, number of observation days and the number of days that healthy and infected birds were seen. (An online description of the HFDS is available at www.birds.cornell.edu/hofi.)

To analyse long-term trends in house finch–*Mycoplasma* dynamics, we examined records from over 45 000 individual observations collected between November 1994 and March 2001. We focused on observers located within eastern North America, east of –95° longitude and south of 50° latitude (Fig. 1). We included only data from participants that submitted data for 6 or more months to minimize biases caused by infrequent reports (i.e. participants who submit

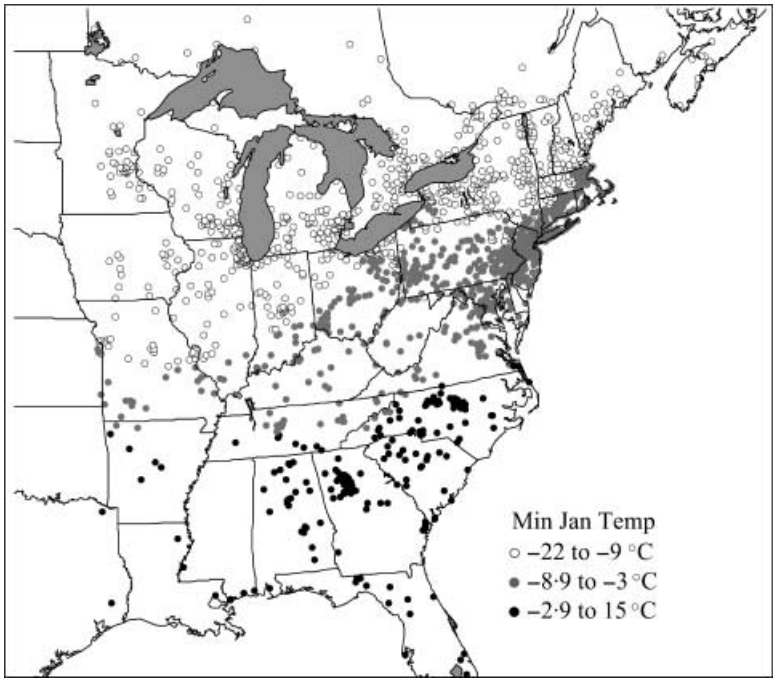

Fig. 1. Geographic locations of volunteer HFDS participants divided into three climatic regions (North, Central and South) using mean minimum January temperatures (e.g. Karl *et al*. 1990). For the North (open circles), temperatures were equal to or below –9 °C; for the Central region (grey circles), temperatures were greater than –9 °C but less than or equal to –3 °C; for the South (black circles), temperatures exceeded –3 °C.

information only when they observe infected birds). We also screened the database to include only records where observers watched their feeders for 4 or more days per month and actually saw house finches, thus reducing our data set to approximately 25 000 observations over 72 months.

ESTIMATING PREVALENCE

Because most observers provided presence/absence information only (and did not count numbers of healthy and diseased individuals), we determined first the best estimate of prevalence. Recent analyses of the geographical and temporal changes in MG abundance have used a site-based prevalence measure (Dhondt *et al*. 1998; Hartup *et al*. 2001a) based on the proportion of observers in a defined area reporting infected birds. Although effective at tracking geographical disease spread, this site-based measure might be insensitive to fine-scale changes in the proportion of infected birds. An alternative estimate is the proportion of days that infected birds were seen relative to the total number of days that house finches were observed each month.

For a subset of months (December 1997–October 2000, or 2750 individual reports) observers recorded both the number of days that healthy and infected birds were seen and the actual number of healthy and infected birds. Comparison of monthly averages for true prevalence and the site- and day-based prevalence estimates showed a strong linear relationship between true prevalence and the proportion of days infected birds were seen ($y = 0{\cdot}008 + 2{\cdot}91x$; SE slope = 0·19; $t = 15{\cdot}01$, d.f. = 34, $P < 0{\cdot}0001$), with the proportion of days potentially overestimating true prevalence by a factor of 2–3. The site-based prevalence estimate was also biased upwards (overestimating true prevalence by a factor of 5–20), and was best fit as a quadratic function, suggesting that its utility is greatest during the early stages of an epidemic when prevalence is low (polynomial regression: $y = 0{\cdot}113 + 5{\cdot}80x - 18{\cdot}86x^2$; SE slope$_1$ = 0·98; $t_1 = 5{\cdot}89$, $P_1 < 0{\cdot}0001$; SE slope$_2$ = 6·84; $t_2 = -2{\cdot}76$, $P_2 = 0{\cdot}009$). Because the proportion of days that observers reported diseased birds was available for the entire data set and provided a linear index of actual prevalence, we used this variable in all analyses described below.

STATISTICAL ANALYSES OF REGIONAL AND TEMPORAL TRENDS

Regional differences in mycoplasmal conjunctivitis were examined because previous studies indicated that prevalence was lowest in the extreme North, where winter climates are most severe (Dhondt *et al*. 1998). To compare geographical variation in prevalence, we divided the entire sampling area into three climatic regions based on average January minimum temperatures (Fig. 1; e.g. Karl *et al*. 1990). Within each region we calculated the average proportion of days that infected birds were seen at each site on a monthly basis from November 1994 to March 2001.

Regionally based monthly changes in prevalence were analysed in three ways. First, we used logistic regression to examine the main effects and interactions of region, year and month on estimated prevalence. Secondly, we used temporal autocorrelation to examine similarity in prevalence among successive months and to identify significant lag intervals. Finally, we characterized seasonal patterns relative to long-term trends using the X-11 Census Method for classical seasonal decomposition (Makridakis & Wheelwright 1989; Yaffee & McGee 2000; SAS Institute 2001). This method has been refined by the US Census Bureau to decompose time-series data into (1) a seasonal component, (2) a longer-term trend-cycle component and (3) noise or random error. The seasonal decomposition algorithm first applies a large-window moving-average function to the original time-series to estimate long-term trends. The original time-series is then divided by these smoothed values to obtain a combined seasonal-irregular component, from which the seasonal component is isolated as the medial average for each month. After calculating seasonally adjusted values (by dividing the original series by the seasonal component), a final long-term trend-cycle is approximated by applying a second smoothing algorithm to the seasonally adjusted series. Finally, the random component is calculated by dividing the seasonally adjusted series by the final trend-cycle component.

POTENTIAL CONFOUNDING FACTORS

Several confounding factors might contribute to seasonal or geographical variation in prevalence, especially temporal variation in observer effort. The potential indices of observer bias that we examined were: (1) the number of observation days each month, (2) the number of days house finches were seen and (3) the number of participants reporting data. To consider the contribution of sampling biases to differences in prevalence, we examined seasonal changes in each of these three variables in addition to correlations between estimated prevalence and potential observer bias (e.g. Figure 2).

We found seasonal variation in all three indices of potential bias, but not in a way that would change conclusions qualitatively regarding disease dynamics. Each year, the number of participants declined during the summer and early autumn (April–October). This is explained best by a subset of observers that regularly participate only during the time that a parallel monitoring survey (Project FeederWatch, described below) is in place. Despite their regular occurrence, seasonal changes in observer effort and house finch visitation are unlikely to cause the variation in prevalence we report here for two reasons. First, prevalence did not vary systematically with the number of regular participants submitting data each month (e.g. linear regression of arcsine square root-transformed monthly prevalence

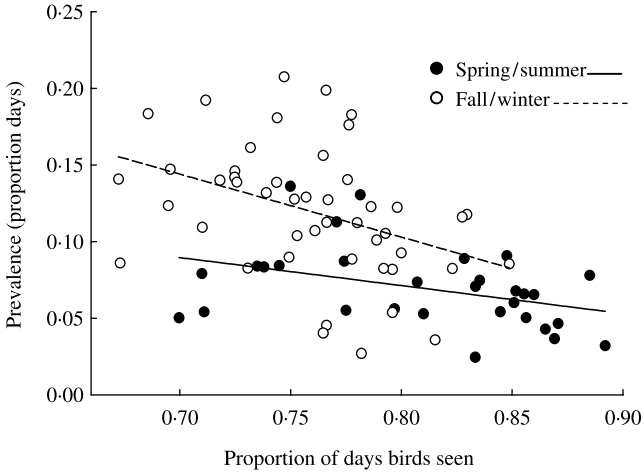

Fig. 2. Prevalence vs. the proportion of days house finches were seen. Solid circles represent summer months (April–August) and open circles represent winter months (September–March). Solid and dashed lines show least-squares regression for summer ($y = 0{\cdot}218 - 0{\cdot}183x$, $t_{slope} = -2{\cdot}26$, $P = 0{\cdot}030$) and winter months ($y = 0{\cdot}432 - 0{\cdot}411x$, $t_{slope} = -2{\cdot}87$, $P = 0{\cdot}006$), respectively. For both plots, points represent averages throughout all of eastern North America. For regression analyses, prevalence values were arcsine square root-transformed, but are shown in plots as untransformed values.

vs. number of observers showed no significant relationship; $y = 0{\cdot}121 + 0{\cdot}00x$, $t_{slope} = -0{\cdot}985$, $P = 0{\cdot}328$). Secondly, observers who watched feeders for more days per month or who saw house finches more regularly were not more likely to see infected birds. Rather, prevalence appeared to decline with increasing observer effort and the probability of seeing house finches (Fig. 2). Because annual patterns of lower summer prevalence are clearly not caused by failure of observers to see house finches, we did not include potential measures of observer bias as covariates in the analyses described below.

REGIONAL VARIATION IN FLOCK SIZES AND FEEDER USE

House finch feeder use and flocking behaviour are two additional factors that could influence seasonal and regional differences in prevalence. To examine whether these factors were associated with disease prevalence, we used reports of the numbers of house finches observed at feeders in eastern North America from Project FeederWatch (PFW), a volunteer-based monitoring programme coordinated by the Cornell Laboratory of Ornithology. From November to April, PFW observers report the maximum number of individuals of each species seen at one time at backyard feeding stations. The total number of monthly participants for PFW varied between 3800 and 5400 throughout all of North America, and previous studies have used PFW data to examine temporal and spatial variation in the abundance of winter feeding birds (Wells *et al.* 1998; LePage & Francis 2002). (An online description of PFW is available at http://birds.cornell.edu/pfw.)

We used PFW data from five consecutive winter observation seasons that overlapped with years of HFDS reports (1995–96, 1996–97, 1997–98, 1998–99, 1999–2000). We divided observations into the same three regions in eastern North America using temperature data as described earlier (e.g. Figure 1). We estimated feeder use as the proportion of observers reporting any house finches, and flock size as the maximum number of finches seen together at one time averaged over observers. For estimates of flock size, we excluded extreme observations (where > 100 house finches were reported), and 'zero' observations (where no house finches were seen). Analysis of variance (SAS, PROC GLM) and logistic regression (SAS, PROC GENMOD and PROC LOGISTIC) were used to examine how flock size and the proportion of feeders visited varied as a function of sampling year, month, region and all two-way interactions.

Finally, we used a generalized linear model with binomial errors (SAS, PROC MIXED, glimmix macro) to examine how temporal changes in prevalence covaried with mean flock sizes and feeder use within each region. In this case, we assumed that prevalence estimates followed a time-decaying covariance process, so that correlations between prevalence estimates fell linearly with increasing time (months) between samples (i.e. by using the REPEATED statement with a first-order autoregressive process). We explored model fit for different assumptions of how the covariance structure changed among regions and observation seasons (years), as determined by comparing Akaike information criteria corrected for small sample sizes (AICc). Our statistical model included the main effects of region, observation season, flock size and feeder use, as well as two-way interactions between flock size, feeder use and region.

Results

PRELIMINARY ANALYSES

Long-term monitoring records demonstrated that following initial establishment, *M. gallisepticum* has persisted in house finch populations and exhibited marked fluctuations in prevalence at a continent-wide

Table 1. Logistic regression of the effects of region, month, year and all two-way interactions on mean prevalence (measured as the proportion of days infected birds were seen). All explanatory variables were treated as categorical factors and analysis of the full model relative to the null model (intercept only) yielded a likelihood ratio score of 17410 (d.f. = 114, $P < 0{\cdot}001$)

Effect	d.f.	ΔD (Wald χ^2)	$P > \chi^2$
Region	2	1629·96	< 0·0001
Year	7	2307·92	< 0·0001
Month	11	679·23	< 0·0001
Region × month	22	1660·87	< 0·0001
Region × year	14	1698·71	< 0·0001
Year × month	58	2156·91	< 0·0001

scale, with mean monthly prevalence throughout eastern North America varying from 3·5% to 22·5%. Logistic regression analysis of the effects of region, month and year on prevalence showed that all main effects and two-way interactions were significant (Table 1). Thus, changes in prevalence occurred over both long (years) and short (months) time periods. Prevalence also varied significantly among geographical regions, so that over the 77-month monitoring period mean prevalence was higher in the central (0·135 ± 0·006 SE) and southern (0·119 ± 0·011 SE) regions relative to the North (0·083 ± 0·005 SE; Fig. 3).

Significant interaction effects between region, year and month demonstrated that the timing and magnitude of seasonal and long-term prevalence changes differed among regions (Table 1). Annual prevalence cycles occurred in all three regions, with autumn (September–November) and late winter (January–March) epidemics followed by a sharp decline in prevalence during the summer months (Fig. 3). Differences in annual minima and maxima were, on average, more extreme in the South. Comparison of 3-month averages for summer (May–July) vs. autumn (September–November) across all 6 years showed that summer to autumn prevalence increases were most extreme in the South, lower in the northern region and smallest in the central region (Table 2).

Temporal autocorrelation of monthly prevalence from November 1994 to March 2001 showed further evidence for annual cycles in two of the three geographical regions. Time-series data were characterized by large positive correlation coefficients at 12-month lags, with large negative coefficients at intermediate lag intervals in the North and South (Table 3). Most autocorrelation coefficients for the central region were smaller and non-significant (Table 3).

SEASONAL DECOMPOSITION ANALYSIS

Seasonal decomposition of monthly prevalence (using the multiplicative procedure of the X-11 census method)

Table 2. Annual variation in summer (May–July) vs. autumn (September–November) prevalence of conjunctivitis in all three regions, measured as the proportion of days that infected birds were seen. Mean prevalence represents 3-month averages over 6 years (1995–2000). The mean number of regular participants per month is shown in parentheses

Region	Mean summer prevalence	Mean autumn prevalence	Difference	% Increase from summer to autumn
1 (North)	0·042 (152)	0·100 (158)	0·058	238%
2 (Central)	0·091 (146)	0·156 (173)	0·065	171%
3 (South)	0·065 (31)	0·206 (32)	0·141	317%

Table 3. Temporal autocorrelation coefficients and standard errors based on time-series prevalence data for each of the three regions shown in Fig. 1. *Coefficients exceeding the 95% confidence limits

	North		Central		South	
Lag (months)	Autocorr.	SE	Autocorr.	SE	Autocorr.	SE
1	0·774*	0·112	0·653*	0·112	0·552*	0·112
2	0·490*	0·111	0·209	0·111	0·146	0·111
3	0·208	0·110	−0·067	0·110	−0·088	0·110
4	−0·020	0·110	−0·147	0·110	−0·004	0·110
5	−0·173	0·109	−0·103	0·109	0·162	0·109
6	−0·252*	0·108	−0·012	0·108	0·168	0·108
7	−0·235*	0·107	−0·006	0·107	0·044	0·107
8	−0·075	0·107	−0·034	0·107	−0·208	0·107
9	0·140	0·105	−0·073	0·106	−0·383*	0·106
10	0·345*	0·104	−0·077	0·105	−0·260*	0·105
11	0·466*	0·103	0·042	0·104	0·038	0·104
12	0·451*	0·103	0·108	0·103	0·273*	0·103
13	0·277*	0·102	−0·059	0·102	0·022	0·103
14	0·055	0·102	−0·200	0·102	−0·243*	0·102
15	−0·148	0·101	−0·262*	0·101	−0·392*	0·101

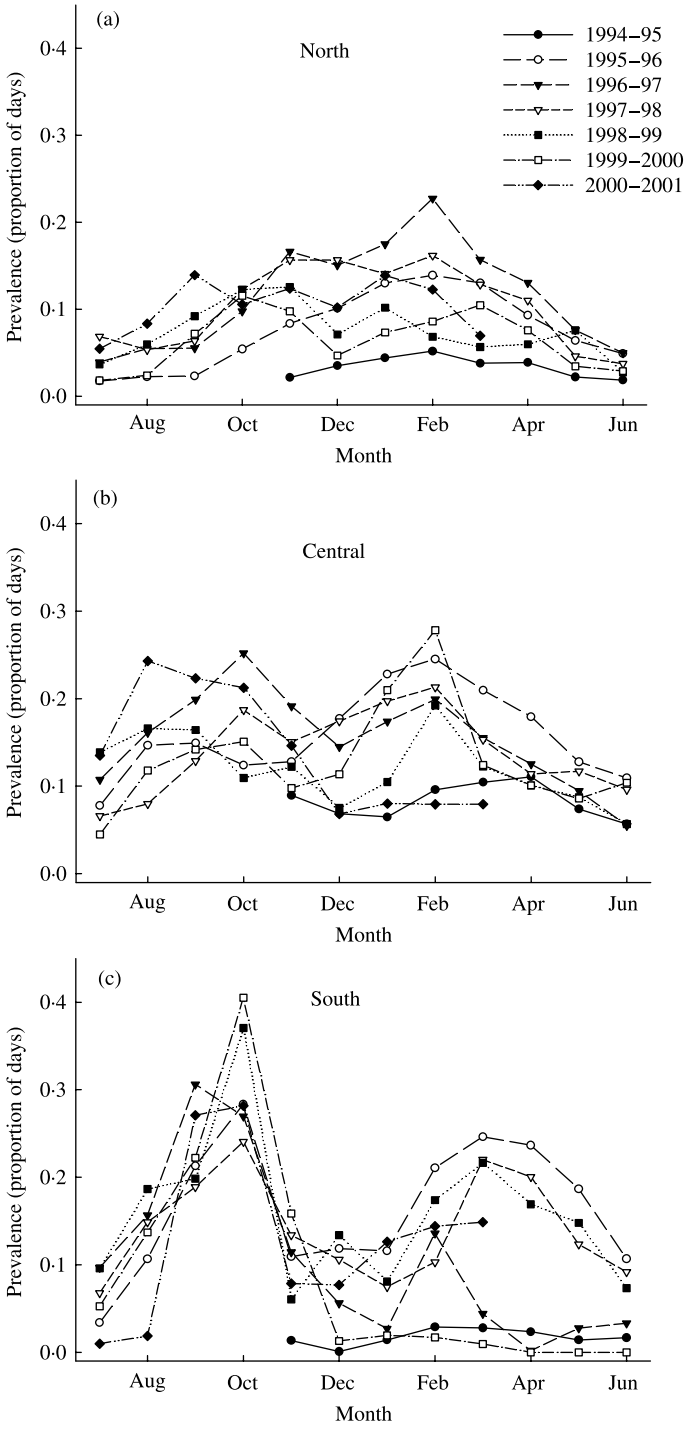

Fig. 3. Monthly changes in prevalence (from June to July) in each of the three climatic regions represented in Fig. 1: (a) North (b) Central and (c) South. Each line represents data from a single year of the HFDS, beginning in November 1994 (data from partial years were included). Note that the solid line represents the first year of the study when the pathogen was still increasing in geographical range.

showed that both seasonal factors and long-term trend-cycles varied among the three climatic regions. Removing seasonal factors and noise to reveal the longer term trend-cycle (Fig. 4) demonstrated that mycoplasmal conjunctivitis has not increased or declined monotonically following its initial invasion, but showed evidence of multiyear fluctuations in all three regions. Longer-term peaks in prevalence occurred at 2–3-year intervals, with more extreme long-term oscillations in the South (solid line in Fig. 4) and asynchrony among regions. For example, following a period of unusually low prevalence from March 1999 to August 2000, prevalence in the South increased rapidly during the winter of 2000–01.

Seasonal factors from decomposition analysis (shown in Fig. 5 as multiplicative percentages relative to baseline prevalence) showed two components that were common to all three regions: a clear summer decline in prevalence and an autumn–winter increase in prevalence

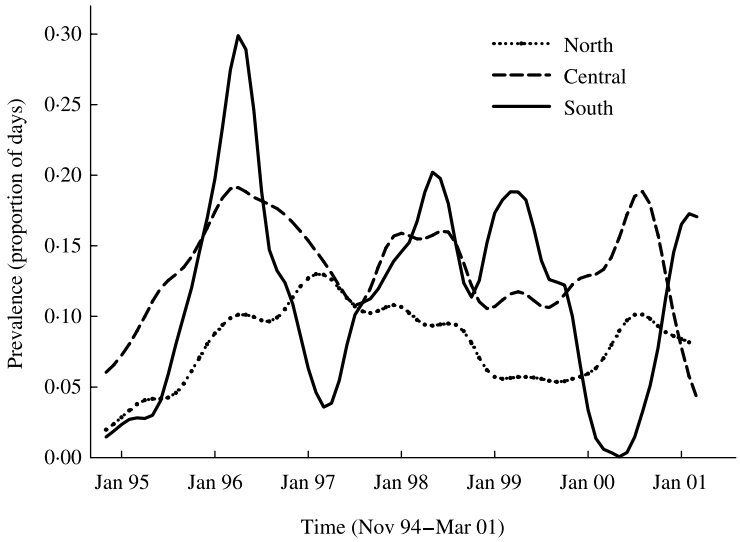

Fig. 4. Long-term trend-cycle from Census X-11 decomposition of monthly prevalence time-series data in each of the three climatic regions from November 1994 to March 2001. The dotted line represents the North, long dash represents the Central region and solid line represents the South. Note that the pathogen was still establishing and expanding in geographical range prior to 1996, and shows evidence of multiyear fluctuations after that time. The first maximum in each region was as follows: March 1996 (North), April 1996 (Central) and April 1996 (South). The most recent multiyear maximum occurred in August to September 2000 (North and Central regions) and in February to March 2001 (South).

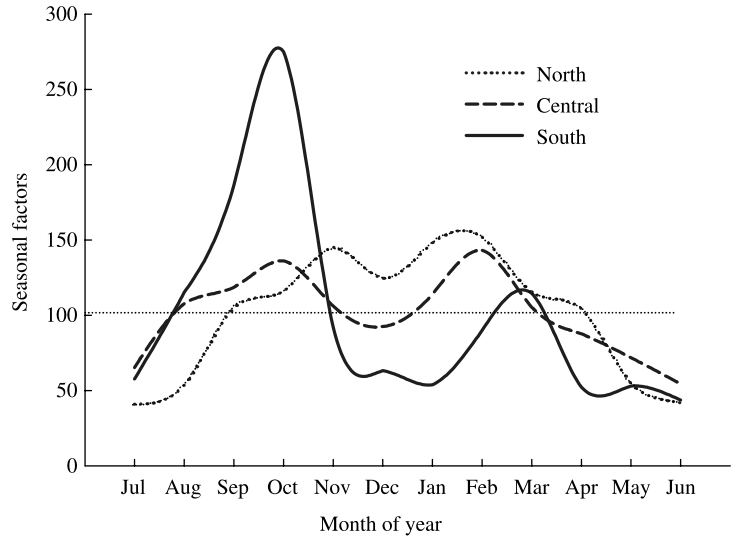

Fig. 5. Final seasonal factors from Census X-11 decomposition of monthly prevalence for each of the three climatic regions, shown as a percentage (multiplicative) deviation from the long-term trend cycle, where the dashed line represents baseline prevalence at 100%. The dotted line represents the North, long dash represents the Central region, and solid line represents the South.

(between October and March). On average, two 'winter peaks' in prevalence occurred in each region: during the autumn, prevalence peaked in October–November, and a second winter peak occurred between February and March. Differences between winter maxima and summer minima were smallest in the central region (Fig. 5). Annual autumn outbreaks occurred earlier (by approximately 1 month) and were most extreme in the South. Separations between autumn and late winter peaks were notable in both southern and central regions, but were relatively minor in the North.

REGIONAL VARIATION IN FLOCK SIZES AT FEEDERS

Mean flock sizes (measured as the maximum number of house finches observed at one time) varied significantly among years, regions and months (Table 4; Fig. 6a). In most years, flock sizes peaked during the coldest winter months (December–January) and declined sharply in early spring (Fig. 6a). Except for one year (1995–96), flock sizes in the South were notably lower than in the North and Central regions. Beginning in 1997–98, flock

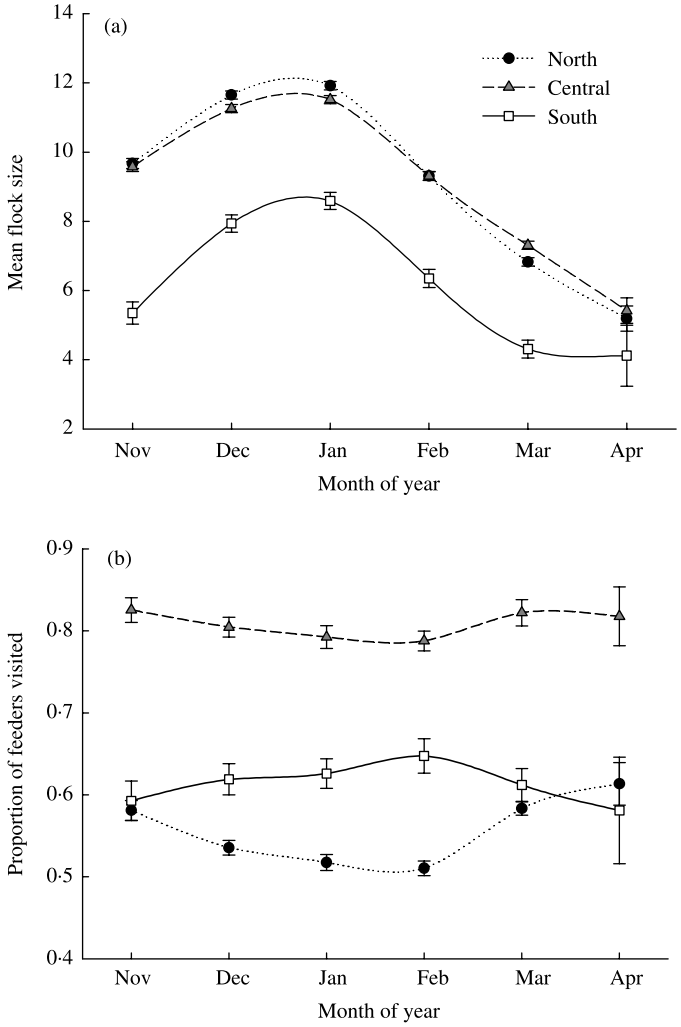

Fig. 6. Temporal and regional variation in (a) house finch flock sizes and (b) feeder use based on Project FeederWatch observations from November to April. Feeder use was measured as the proportion of participants who reported seeing house finches at their feeders, and flock sizes represent the mean counts of house finches seen together at feeders at any one time. Least-squares mean estimates (LSMEANS in PROC GENMOD and PROC GLM, SAS Institute 2001) were obtained from five seasons of data (1995–96, 1996–97, 1997–98, 1998–99 and 1999–2000). In each figure, the dotted line represents the North, long dashed line represents the Central region and solid line represents the South. Error bars represent 95% confidence intervals from least squares means estimates. LSMEANS for feeder use were back-transformed from a logit scale based on the following expression: Proportion = EXP(LSMEAN)/(1 + EXP(LSMEAN)).

Table 4. Analysis of variance of house finch flock size as a function of year, region and month. PFW data were selected from five consecutive years beginning in 1995–96, where year is defined as a 6-month observation interval from November to April. Counts were log-transformed prior to analysis to yield normally distributed observations. Results are based on Type III sums of squares for individual effects of each categorical variable (ANOVA model: flock size = year + region + month + region × month + region × year + year × month)

Effect	d.f.	Type III SS	*F*	*P* > *F*
Year	4	260·96	68·17	< 0·0001
Region	2	329·07	171·92	< 0·0001
Month	5	1190·15	248·70	< 0·0001
Region × month	10	62·87	6·57	< 0·0001
Region × year	8	384·80	50·26	< 0·0001
Year × month	20	173·96	9·09	< 0·0001
Error	103845	103380·28		

sizes in the South also showed less within-season variation than flock sizes in the north and central regions. Analysis of variance showed that all three main effects and two-way interactions explained significant variation in the number of house finches seen at one time (Table 4).

In all years, feeder use was highest in the central region, with over 75% of observers reporting house finches each month (Fig. 6b). Fewer observers in the North and South reported seeing house finches at their feeders (proportion of feeders visited varied between 50 and 60%; Fig. 6b). Logistic regression analysis showed that all main effects and two-way interactions were associated significantly with variation in feeder use (Table 5), so that the proportion of feeders visited depended strongly on observation season (year), month within year and region.

Table 5. Logistic regression table for effects of time and region on house finch feeder use. PFW data were selected from five consecutive year seasons beginning in 1995–96, where year is defined as a 6-month interval from November to April. Results are shown as Wald χ^2 for each effect (logistic regression model: proportion visited = year + region + month + region × month + region × year + year × month), where effects were treated as categorical factors. For the full model, likelihood ratio tests were highly significant ($\chi^2 = 10393{\cdot}68$; d.f. = 49; $P < 0{\cdot}0001$)

Effect	d.f.	ΔD (Wald χ^2)	$P > \chi^2$
Year	4	153·27	< 0·0001
Region	2	3505·37	< 0·0001
Month	5	43·33	< 0·0001
Region × month	10	108·38	< 0·0001
Region × year	8	476·00	< 0·0001
Year × month	20	222·60	< 0·0001

Table 6. Generalized linear model results for effects of region, flock size, and feeder use on monthly prevalence (SAS, GLIMMIX macro; logistic model: proportion of days = region + year + flock size + feeder use + flock size × region + feeder use × region). The REPEATED statement was used to specify that correlations between prevalence estimates should decay linearly with increasing time between sampling months. Comparison of model fit parameters showed that a constant covariance structure across regions and year provided the best fit to the data. Because model fit parameters were also better by assuming that prevalence was associated with flock sizes and feeder use in the previous month (rather than the current month), only type 3 tests of fixed effects for this time-lagged scenario are shown below. Num d.f. and Den d.f. refer to degrees of freedom for numerator and denominator of *F*-statistic

Effect	Num d.f.	Den d.f.	*F*	*P*
Region	2	8	0·70	0·524
Year	4	8	1·02	0·451
Flock (lagged)	1	68	17·12	< 0·001
Feeder (lagged)	1	68	0·001	0·964
Flock × region	2	68	3·28	0·044
Feeder × region	2	68	1·40	0·253

PREVALENCE, FLOCK SIZE AND FEEDER USE

The annual decline in prevalence from February to April corresponded closely to the timing of flock size declines in the North and Central regions, despite the fact that months when flock sizes peaked did not correspond to months when prevalence was highest (Fig. 6a vs. Fig. 5). Fitting a logistic mixed model to prevalence showed that mean flock sizes, but not feeder use, explained differences in monthly prevalence and that this association varied among regions (Table 6; Fig. 7). Our generalized linear model assumed that correlations between prevalence estimates within each region and year decayed linearly with increasing time between sample months (treating month as a repeated measure), and that the same time-decaying covariance structure occurred within all regions and years. This assumption was supported by comparison of AICc among models with different covariance structures, which showed that the simplest assumption (constant covariance structure) provided the best fit to the data (e.g. AICc for model assuming constant covariance = 74·4 vs. AICc for model assuming that covariance structures differed among regions = 84·4).

Because the expression of severe clinical signs may occur 1–4 weeks post-transmission, we examined whether prevalence was associated more strongly with

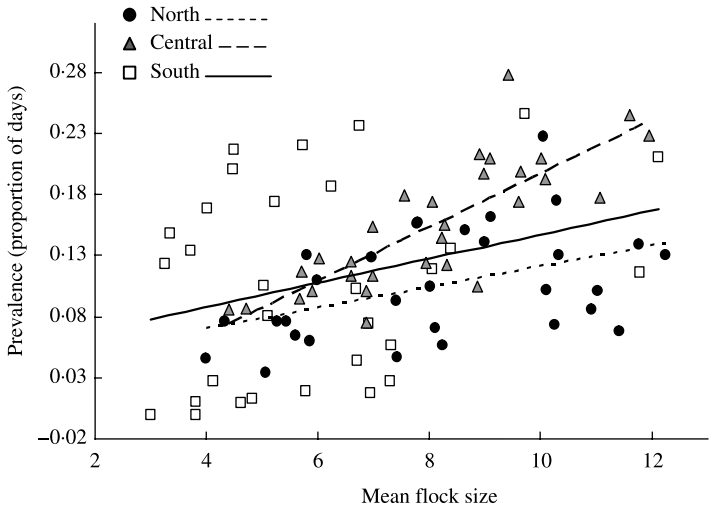

Fig. 7. Monthly prevalence estimates (from the HFDS, based on the proportion of days diseased birds were seen) relative to mean flock size in the previous month (from PFW data). Data shown are region-wide monthly prevalence averages for Dec–May, and region-wide monthly flock size averages for Nov–April over five observation seasons. Regions (North, Central and South) correspond to observation sites in Fig. 1. Lines highlight the slope from a least-squares regression of prevalence on mean flock size, where the dotted line represents the North (slope = 0·008, $t = 2{\cdot}26$, $P = 0{\cdot}032$, d.f. = 29), dashed line represents the central region (slope = 0·022, $t = 7{\cdot}19$, $P < 0{\cdot}001$, d.f. = 29), and solid line represents the South (slope = 0·01, $t = 1{\cdot}60$, $P = 0{\cdot}122$, d.f. = 29). As in the logistic analysis described in the results text, flock sizes were associated positively with estimated prevalence, with the strongest association in the central region and weakest association in the South.

flock sizes and feeder use from the same month vs. the previous month. We compared AICcs for models using one of the two sets of explanatory variables. For the lagged scenario, we excluded prevalence data for November but included prevalence data for May, as flock size and feeder use data were available for April but not for October. Using the same number of observations, model fit parameters were better for the time-lagged scenario (AICc = 74·4) than for the same-month scenario (AICc = 81·7), and this was true when tested across all covariance structure assumptions. Thus, in the final model with lagged flock sizes and feeder use, both the main effects of flock size and the interaction between flock size and region were statistically significant (Table 6). Visual comparison of monthly prevalence estimates with flock sizes showed a significant positive relationship in the central region, with little or no relationship in the North and South (Fig. 7).

Discussion

Consistent monitoring of mycoplasmal conjunctivitis in house finches at a continent-wide scale has provided an unprecedented opportunity to examine seasonal, geographical and long-term temporal variation in the dynamics of this wildlife pathogen. Our analysis showed that following emergence and initial spread, the complicated dynamics exhibited by this disease were characterized by both seasonal and longer-term cycles. Regular seasonal cycles occurred in all three geographical regions in eastern North America and were characterized by autumn–winter epidemics and very low prevalence during the summer months. Longer-term variation in prevalence was characterized by multiyear fluctuations.

FACTORS ASSOCIATED WITH SEASONAL TRENDS

Several factors might cause seasonal changes in the prevalence of mycoplasmal conjunctivitis within each region, including annual variation in host density or abundance at feeders, seasonality of breeding (causing changes in host stress or immunocompetence and generating seasonal pulses of juvenile recruitment), and environmental factors that influence pathogen persistence or virulence. The most probable explanation for low prevalence during the summer months is that seasonal reproduction limits contact rates among hosts. In spring and summer, house finches disperse to breeding sites where they interact primarily within family groups and do not form large flocks (Hill 1993). Indeed, multiyear comparisons of changes in flock size (Fig. 6a) showed that the number of birds seen together at feeders is lowest in March and April, when house finches form mating pairs and begin nesting, and largest during the coldest midwinter months. The decline in flock sizes from February to April corresponded closely with a rapid decrease in prevalence each spring (Figs 5 and 6), and within the northern and central regions prevalence was significantly positively associated with mean flock size (Fig. 7).

Autumn epidemics of mycoplasmal conjunctivitis have been noted in a number of previous studies (Hartup *et al*. 2001a; Roberts *et al*. 2001a, 2001b) and could be due to a combination of factors, including the recruitment of immunologically naive juveniles into the population towards late summer, and a shift in host social behaviour (with breeding pairs giving way to larger social aggregations). Alternative explanations for an autumn increase in prevalence relate to increased host susceptibility brought on by late-summer moulting, end-of-breeding season stress or long-distance migration. House finches moult once annually, during early July to mid-September (Hill 1993). Moulting has been shown to be energetically costly (Lindstrom, Visser & Serge 1993) and might increase susceptibility to disease due to increased stress or reduced temperature insulation (Ginn & Melville 1983; Svensson & Merila 1996). Moreover, increased reproductive effort in other avian species has been shown to correlate with greater parasite burdens and lower antibody production and cell-mediated immunity (Moreno *et al*. 2001; Møller & Petrie 2002). Interestingly, seasonal epidemics punctuated by winter outbreaks and summer declines have also been reported for another pathogen (avian pox) infecting house finches in coastal California (McClure 1989).

Limited data suggest that two potentially important factors – seasonal migration and temporal changes in feeder use – are not likely to trigger annual autumn–winter epidemics. First, the timing of the autumn epidemic in the South and Central regions precedes southward migration by over a month (Belthoff & Gauthreaux 1991), so that neither an annual influx of migrants nor migration stress is likely to trigger the sharp autumn epidemic in the South. Secondly, estimates of house finch feeder use over all three regions in eastern North America did not change in a consistent direction from November to April (Fig. 6b), and in some cases the proportion of feeders visited increased slightly in the early spring, during the same time that prevalence declined in most regions. Statistical analysis further suggested that feeder use was not associated strongly with temporal variation in prevalence during the winter months. Thus, although exposure at bird feeders has been suggested as a potentially important transmission route for this disease, we found no consistent evidence that seasonal variation in feeder visitation contributes to annual outbreaks of this disease.

We were surprised to observe a late winter epidemic (from February to April) that occurred in the South and Central regions (Fig. 7), although this second epidemic was not observed every year (e.g. Fig. 5). The timing of this second winter peak coincides with the formation of breeding pairs and courtship in February and March (Hill 1993). One mechanism underlying a late winter outbreak could be that mating stress and

hormonal changes lower host immunocompetence – especially among males entering breeding condition. The link between testosterone and immune response is well established in other species (Marsh 1992), and past work in house finches has shown that experimentally elevating testosterone in captive males increased susceptibility to coccidial infections (Duckworth *et al*. 2001). Thus, a hormonally induced decrease in host immunocompetence might cause prevalence in early spring to increase more rapidly among males than females. Alternatively, late winter epidemics could be initiated by an increase in aggressive (male : male) or courtship-related contacts (bill rubbing, feeding) that accompany the formation of breeding pairs in February to March. Finally, it is important to emphasize that the two annual epidemics (autumn and late winter) are caused probably by independent factors, as we have not identified a single mechanism that coincides with the timing of both seasonal outbreaks.

FACTORS ASSOCIATED WITH REGIONAL DIFFERENCES

Our analysis showed that annual autumn epidemics of mycoplasmal conjunctivitis increased more rapidly and were of greater amplitude in the South than the North and Central regions (e.g. Figs 5 and 7). Moreover, autumn–winter epidemics showed stronger evidence of bimodality (with distinct autumn and late winter outbreaks) in the South and, to a lesser degree, Central regions, with little evidence for multiple distinct winter epidemics in the North. One cause for extreme autumn epidemics in the South could relate to more rapid recruitment of previously unexposed juveniles through a breeding season of longer duration than in the North and Central regions. For example, studies of nematode outbreaks in Soay sheep have shown that magnitude of late-summer peaks in worm larvae depended on the number of lambs produced earlier that year (Gulland & Fox 1992). If this mechanism were important to disease spread in the house finch–MG system, the majority of new infections each autumn should occur in juvenile birds. An alternative possibility is that pathogen transmission is higher in the South due to a longer duration of infectiousness (if, for example, infected hosts live longer due to a milder climate and greater food availability) or longer persistence of pathogen infectious stages outside of the host (for example, greater persistence of fomites on bird feeders). Future studies addressing regional differences in house finch behaviour, survival and pathogen persistence in the environment might identify which of these factors influence observed epidemiological patterns most strongly. Interestingly, our results did not support geographical variation in flock sizes or feeder use as an underlying cause of regional prevalence differences. In fact, mean flock sizes from November to April were lowest in the South and did not correspond to temporal changes in prevalence in this region, although it is important to note that flock size data from August to October (when prevalence increased most rapidly in the South) were not available. It could be that more sensitive measures of flocking behaviour and feeder visitation or continuous year-round flock size data are required to conduct regional comparisons of both spatial and temporal variation in prevalence.

LONG-TERM FLUCTUATIONS

Over the 77-month monitoring period, multiyear epidemics occurred within each region, and continued monitoring should reveal whether long-term oscillations show regular periodicity. These oscillations were moderately asynchronous among regions, with the 2000–01 increase in prevalence in the South occurring 6 months after peak prevalence in the Central region. We also observed more extreme multiyear fluctuations in disease prevalence in the South. Similar multiyear cycles in prevalence occur in other human and wildlife systems (Childs *et al*. 2000; Pascual *et al*. 2000) and could be driven by high disease-induced mortality, long-lasting host immunity, a short duration of infectiousness, long incubation periods or longer-term environmental trends (Anderson & May 1991).

In summary, our study indicates that factors underlying rapid changes in diseases prevalence can vary over both space and time in natural host–pathogen interactions. Evidence suggests that in the house finch–MG system, conditions leading to increased disease spread, including higher numbers of susceptible birds, increased social contacts or lower host immunocompetence manifest themselves during the autumn and winter in eastern North America. The more extreme and earlier autumn epidemics in the south-eastern United States point to more rapid disease transmission further south, or greater host susceptibility in this region. Observed variation in prevalence further suggests that epidemiological models used to capture disease dynamics should consider both annual and longer-term prevalence changes and address potential causes of regional variation.

An important possibility is that seasonal changes in house finch ecology and behaviour could sustain longer-term cycles of *M. gallisepticum* and could influence its population-wide persistence. Regular fluctuations in population dynamics can be synchronized or reinforced by the extrinsic environmental effects on biological systems (Hudson & Cattadori 1999; Koenig 1999; Lloyd & May 1999; Bjørnstad 2000). For example, population cycles of snowshoe hares might track solar activity through secondary effects on climate (Sinclair *et al*. 1993), and school year seasonality has been implicated in regular and synchronous outbreaks of measles in Britain (Ranta *et al*. 1995; Bolker & Grenfell 1996). Thus, extrinsic factors affecting disease dynamics, including those that generate seasonal pulses of susceptible juveniles and annual variation in host social behaviour, could have overriding importance for large-scale spatial and

temporal patterns of disease prevalence in this and other host–pathogen systems.

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