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Mycoplasma gallisepticum infection dynamics in a house finch population: seasonal variation in survival, encounter and transmission rate

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Summary

1. We considered the impact of an emerging pathogen (*Mycoplasma gallisepticum* Edward and Kanarek) on apparent survival, encounter and transition rates in a population of a novel host (the house finch, *Carpodacus mexicanus* Müller). We used a multistate analysis of mark–encounter data from individually marked birds. Individual birds were categorized to a particular disease 'state'; transition rates among states, conditional on apparent survival, were analogous to rates of new infection and recovery from infection. We hypothesized that *M. gallisepticum* infection would reduce the apparent survival of infected individuals, and that the magnitude of this reduction would vary as a function of the physiological condition of the host (which was characterized in our analyses by including a demographic and an environmental surrogate as covariates).

2. We found consistent support for the hypothesis that *M. gallisepticum* infection resulted in lower apparent survival among infected individuals, and that recovery rates (from infected to non-infected) were greater than infection rates in this population. We also found strong evidence indicating that infected individuals were less likely to be encountered than were non-infected individuals. Although we predicted that both sex and temperature (proxies for physiological condition) would explain a significant proportion of the variation in our data, only marginal influences of both factors on apparent survival, encounter and state transition rates were detected.

3. Our analyses identified several factors that may be important to studies of disease in the wild. First, disease state assignment may be uncertain, which can complicate parameter estimation. Secondly, encounter rate for infected individuals in our study was low relative to that for non-infected individuals, reflecting possible behavioural changes in infected individuals. Low encounter rates reduces precision of estimated parameters, especially for multistate models. Finally, our results (and mark–recapture models in general) assume independence among individual birds. However, we are aware that there is a social structuring in house finches (and in general for many bird species). Accounting for such non-independence may be especially important for situations where the state transitions are directly related to the pattern of social contact.

Key-words: *Carpodacus mexicanus*, disease dynamics, multistate mark–recapture, sex effects, temperature effects.

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Introduction

There is increasing evidence that infectious diseases can strongly influence the dynamics of host populations (Saumier, Rau & Bird 1986; Hudson & Dobson 1991) and can limit population growth under some circumstances (Hochachka & Dhondt 2000; Tompkins *et al*. 2002). Generally, the impact of pathogens at a population level is to lower the survival rate and/or the reproductive rate of the infected hosts (Anderson & May 1978; Newton 1998), either of which may subsequently suppress population growth.

A recent example of such a relationship involves the emergence of a new pathogen, *Mycoplasma gallisepticum* (Edward and Kanarek, hereafter *Mycoplasma*), in a novel introduced host, the house finch (*Carpodacus mexicanus* Müller, hereafter finch). Finches are native to the western United States and Mexico and are an introduced species in the eastern half of the United States (Hill 1993). In February 1994, finches with swollen or crusty eyes were first observed at feeders in suburban Washington DC, USA. The bacterium causing this conjunctivitis was identified as *Mycoplasma* (Ley, Berkhoff & McLaren 1996), a costly pathogen of domestic poultry worldwide (Yoder 1991; Hartup *et al*. 1998; Levisohn & Kleven 2000). Since the initial observation of this transference from poultry to finches, the disease has spread rapidly throughout the eastern United States (Dhondt, Tessaglia & Slothower 1998).

The emergence of *Mycoplasma* is of potential significance in the population dynamics of finches, at several spatial and temporal scales. *Mycoplasma* is believed to have contributed to significant declines in the eastern finch populations, especially where they were previously abundant (Hochachka & Dhondt 2000); reductions in numbers of as much as 60% have been observed in parts of their range (Hochachka & Dhondt 2000; Altizer, Hochachka & Dhondt 2004). The disease seems to have stabilized at a lower prevalence level in the environment and finch abundance stabilized at approximately 40% of the level expected in the absence of the disease, suggesting density-dependent limitation of the host by the pathogen (Hochachka & Dhondt 2000). Temporally, the seasonal (within-year) dynamics of *Mycoplasma* in finches are typically bimodal: an autumn peak in disease (September–October), then a mid-winter low, another late winter peak (January–March), followed by a steep summer decline (Altizer *et al.* 2004). The magnitude of the differences in prevalence between the autumn–winter maxima and the summer minima was greatest at lower latitudes of the eastern United States and smallest at northern latitudes of the eastern United States.

The impacts of disease on the dynamics of the host population are, in part, governed by the rate at which susceptible individuals become infected, and the rate that infected individuals are removed from the population. Removal can occur either temporarily (due to transient immunity to the infection and/or behavioural changes which alter the rate of exposure to reinfection) or permanently (due to death or recovery with permanent immunity). In domestic poultry, transmission of *Mycoplasma* occurs via two major routes: (i) horizontally, by direct or indirect contact of susceptible individuals with infected carriers, contaminated surfaces or airborne particles or (ii) vertically (*in ovo*) from an infected breeder to its progeny (Yoder 1991; Hartup *et al*. 1998; Levisohn & Kleven 2000). However, the mode of transmission in wild finches is still unknown. It is suspected that the social and foraging behaviour of finches is the source of disease transmission (Hartup *et al*. 1998). For instance, *Mycoplasma* can spread much more rapidly when finches are aggregated in large feeding flocks during the autumn and winter. In addition, the eastern populations of finches currently depend on urban and suburban areas where they frequent backyard feeders (Hill 1993). It is possible that infected birds may contaminate feeders and pass the disease horizontally (through indirect contact) to other susceptible individuals.

While estimation of infection and removal rates has been a major focus of research (both clinically and empirically) in many human epidemiological studies, to date there have been relatively few attempts to derive similar estimates in wild animal populations. Here, we present results of an intensive field study of the seasonal dynamics of *Mycoplasma* infection in a local finch population, encountered during August to April, when *Mycoplasma* disease prevalence is greatest (Altizer *et al.* 2004). In this paper, we consider some of the factors that may influence, or be influenced by, the seasonal dynamics of *Mycoplasma* infection in the wild. In particular, we examine (i) the degree to which *Mycoplasma* infection has a measurable effect on survival and encounter rate, (ii) the pattern of variation in rates of infection and recovery and (iii) the degree to which this variation may be related to factors (sex, temperature) that we consider reasonable proxies for some of the variation in the physiological/immunological state of the birds.

Materials and methods

STUDY AREA AND DATA COLLECTION

We collected data from encounters with individual birds from August to April of 2000–03, at four different locations in Ithaca, NY, USA (Fig. 1). Our analyses were restricted to the months of November–April in years 1 and 2 (2000–01 and 2001–02, respectively) because we found that encounters of infected birds were highest in this time period (Fig. 2). In year 3 (2002–03) we shifted the time frame of our analyses to August–March because we encountered infected birds at high rates much earlier than in previous years (Fig. 2). The distances among our four main field sites ranged from 1·25 km to 2·9 km (Fig. 1). Ideally, finches should have been sampled randomly from all locations with suitable habitat

Fig. 1. Map of study area located in Ithaca, NY, USA. The four main trapping (T) and resighting (R) sites are indicated: (1) Liddell Field Station, (2) Robert Trent Jones Golf Course, (3) Cornell Laboratory of Ornithology and (4) private residence on 802 Dryden Road.

within the full extent of the sampling area. However, this was not practical logistically. The use of fixed sampling sites within a spatially heterogeneous habitat can contribute significantly to heterogeneity in encounters of marked individuals, especially if movement of individuals (or groups of individuals) is non-random with respect to the habitat. The implication of such heterogeneity on our results is discussed later.

Finches were sampled in two ways: live-trapping and live-resighting. Trapping was conducted under permits from the New York State Department of Environmental Conservation, the US Fish and Wildlife Service and the US Geological Survey. All procedures involving live animals were implemented under the Animal Use Protocol no. 00–90 issued by the Cornell University Institutional Animal Care and Use Committee. Each newly captured bird was fitted under permit with a nine-digit numbered aluminium leg ring (Bird Banding Laboratory, Laurel, MD, USA) and a unique combination of three coloured plastic leg rings.

In year 1, trapping and resightings were conducted on various days (with variable intervals between

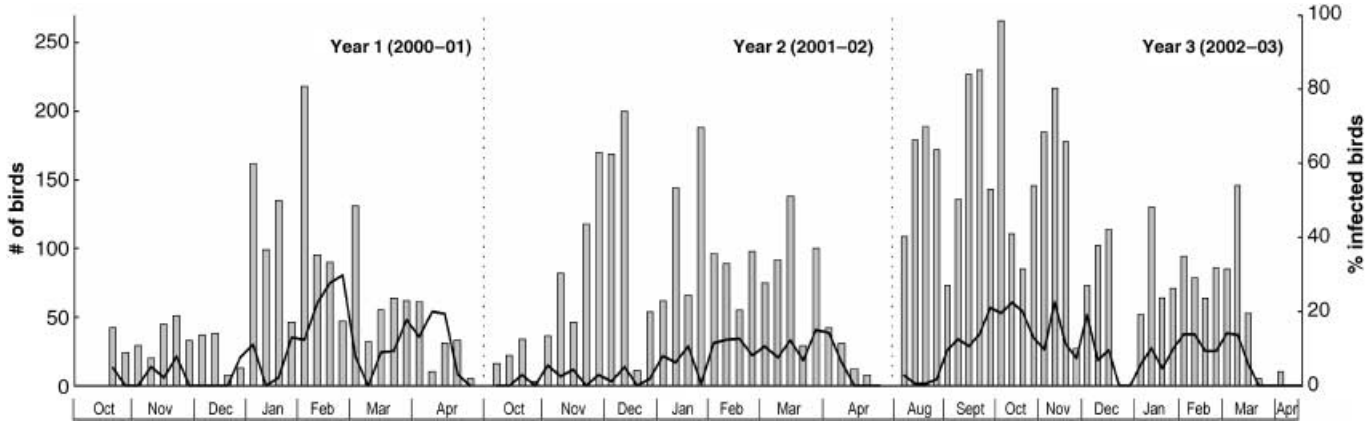

Fig. 2. Absolute number of marked individuals and prevalence (relative proportion) of MG infection in the Ithaca house finch population for year 1 (2000–01), year 2 (2001–02) and year 3 (2002–03). Vertical bars represent absolute number (incidence) of the infected and non-infected birds observed, pooled over each 7-day period from August to April. Solid line indicates prevalence (%) of infected birds observed in the marked sample. The gap in year 3 indicates a lull in HOFI activity where trapping was discontinued for 2 weeks; resightings efforts were continued, but few HOFI were resighted in these weeks.

trapping occasions) and locations throughout the study area to establish the most appropriate field methods. Based on our experiences in the first year, trapping and resighting sessions were standardized to 2 days per week during years 2 and 3, respectively. Trapping was conducted every Tuesday and Wednesday on two main sites ('Golf Course' and 'Liddell Field Station'), while resighting was conducted every Thursday and Friday at all four primary sites (Fig. 1). On each trapping occasion, birds were captured using a combination of two hand-built cylindrical wire-mesh cage traps and two 30-mm mist-nets. Precautions were taken to minimize the risk of transmitting *Mycoplasma* from handling infected birds. After each bird was handled, gloves were worn and washed and the 'holding bag' of each bird was discarded. It was not logistically practical to sterilize the mist-nets and cage traps each time a bird was trapped. Therefore, it is possible that indirect disease transmission could have occurred through a contaminated mist-net or cage trap. For resightings, a vehicle was used as a blind and a spotting scope and/or binoculars were used to resight the birds. Sex and disease state (discussed below) were recorded for each encountered bird.

It is important to note that we used feeders at our trapping and resighting sites to attract finches. The use of baited stations is known to lead to potential bias in cases where baiting induces a greater likelihood of encountering previously captured individuals than expected by random chance (Pradel 1993; Williams, Nichols & Conroy *et al*. 2002). In addition, the use of supplementary food could be affecting body condition and thus affecting survival and recovery rates. However, finches are 'feeder birds' and visiting bird feeders has become part of their natural history (Hill 1993), such that there is little reason to expect birds to become 'trap-happy' due to the use of baited feeders, at least on subsequent visual resighting encounters. Yet, 'trap-shyness' is a possibility if a bird leaves the sampling area due to the stress from being physically trapped. Determining the effect of trap-shyness will require implementing a different analysis approach (Faustino *et al.* in preparation).

The close inspection of the eyes for clinical signs of conjunctivitis, as part of our routine collection of morphological measurements of trapped birds, provided a robust indicator of *Mycoplasma* infection. Individual birds were ranked in terms of severity of infection using an ordinal scale from 0 to 3, but for analyses reported in this paper we collapsed the ordinal disease ranks into a binary one: 'Y' (yes: infected) indicating some level of the disease was observed (i.e. eye score of 1, 2 or 3 in one or both eyes), 'N' (no: not infected) indicating disease was not observed (i.e. eye score of 0 in both eyes), or 'U' indicating unknown disease state (discussed below). A bird in the 'N' state can either have had no previous infection, or have recovered from an existing infection. Because the disease is not always bilateral (40·4% of infected birds showed bilateral infections, $n = 643$), we also recorded whether a bird was infected in the left eye, right eye, or in both eyes. Hartup *et al*. (2001) found that in 95% of 586 house finches sampled there was agreement between an individual's clinical state and presence of *Mycoplasma* through laboratory isolation indicating excellent intermethod concordance for identification of *Mycoplasma*-associated conjunctivitis in finches.

ANALYSES OF LIVE ENCOUNTER DATA

Although our data set consisted of a relatively large total number of observations (8041; Table 1), only a small proportion of these observations included birds classified as infected ('Y') (9·34%; Table 1). This relative paucity of encounters with infected birds resulted in a very sparse encounter matrix when encounters were collated based on daily observations. Thus, to increase the precision of our estimates we pooled the data into 7-day periods because most of our sampling events occurred on a weekly basis. As such, our estimates reflect weekly apparent survival, encounter and transition rates. We then divided each year of data into 'summer–autumn' (August–October for year 3 only), 'autumn–winter' (November–January for all years) and 'winter–spring' (February–April for years 1 and 2; February–March for year 3) separately analysed subsets, with approximately

Table 1. Total numbers of infected, non-infected and unknown state house finches in for year 1 (2000–01), year 2 (2001–02) and year 3 (2002–03), in Ithaca, NY, USA. Each year is divided into subsets: S–A = 'summer–autumn' (August–October for year 3 only), A–W = 'autumn–winter' (November–January for all years) and W–S = 'winter–spring' (February–April for years 1 and 2; February–March for year 3) with approximately 12 7-day pooled periods within each subset. The extra subset, S–A, in year 3 was added due to the high encounters of infected birds earlier in this year than in previous years

	Year 1		Year 2		Year 3			
	A–W	W–S	A–W	W–S	S–A	A–W	W–S	Total
Non-infected (N)	653	761	1266	763	1718	899	484	6544
Infected (Y)	34	135	44	94	245	129	70	751
Unknown (U)	391	60	71	49	56	74	45	746
Total	1078	956	1381	906	2019	1102	599	8041
% infected (Y)	3·15	14·12	3·19	10·38	12·13	11·71	11·69	9·34
% unknown (U)	36·27	6·28	5·14	5·41	2·77	6·72	7·51	9·28

12 7-day pooled periods within each subset. Dividing each year into subsets reduced the number of parameters in the model, making modelling more tractable for some analyses. Marked individuals captured in subsequent subsets were treated as newly captured birds at their first re-encounter. Treatment of these individuals that were marked during a previous subset results in non-independence of subsets. To avoid this, one option would require dropping previously marked individuals that were *not* initially captured during that particular subset. However, this was not practical because our data were already sparse.

Pooled live encounter data were analysed using a multistate mark–encounter approach (Williams, Nichols & Conroy 2002; references therein). Multistate models are an extension of the classical Cormack–Jolly–Seber live mark–encounter, open-population models that allow individuals in the population to be distributed across multiple sites, or in this case among multiple disease states. Such models allow for robust estimation of transition probabilities among states, under conditions where the probability of observing an individual on a particular sampling occasion is < 1.

If we assume that survival from time i to $i + 1$, depends only on the disease state at time i, then separate estimation of survival from transition rates is possible, where:

S_i^r = the probability that an animal in state r at time i survives and remains in the study population until period $r + 1$;

Ψ_i^{rs} = the probability that an animal in state r at time i is in state s at time $i + 1$, given that the animal is alive at $i + 1$

and:

$$\phi_i^{rs} = s_i^{rs}\Psi_i^{rs}$$

where:

ϕ_i^{rs} = the combined probability that an animal alive in state r at time i is alive and in state s at time $i + 1$.

Standard methods for multistate analysis assume that all transitions are first-order Markovian. In other words, they assume that the probability of a bird making a transition between disease states from time i to $i + 1$ is dependent only on its state at time i (i.e. there is no 'memory' in these multistate models). However, in the context of our study, it is possible that the probability of a bird making a transition between disease states is not only dependent on its state at time i, but also on its state at time $i - 1$ or $i - 2$ and so on. For instance, if there is any degree of immunity towards this disease, a recovered bird that was currently infected at time $i - 2$, will have a lower probability of becoming infected than a bird that has never been infected with the disease. However, the sparseness of our data (in particular, the low frequency of infected birds; Table 1) prevented us from modelling state transitions as a higher-order Markov process ('memory models', *sensu* Hestbeck, Nichols & Malecki 1991). Multistate modelling also assumes that individual state can be assigned with complete certainty upon each individual encounter. However, in our study, there was uncertainty for birds that were resighted, because it was possible for the observer to identify an individual bird by its colour ring combination, but not able to assign the bird to either the 'Y' or 'N' disease state; such individuals with uncertain state were classified as 'U' state individuals.

Before analysing our data, we conducted a preliminary numerical simulation study to determine the influence of retaining observations of 'unknown' state ('U') on our estimates (Appendix I). Because 'U' state individuals are in reality either a 'N' or 'Y' state bird, retaining 'U' as a separate state may bias the demographic rate of interest. However, removing 'U' observations (which is in effect removing data concerning 'presence or absence' of the individual) may cause lower precision because 'U' state individuals do provide the information that the bird is alive. From our simulation, we found that the unknowns did not bias survival rates (S), although removal of the unknowns reduced estimate precision at high percentages of unknowns (reflecting a reduction in the amount of data upon which inference was based). We also found that encounter rates (p) were biased both with and without unknowns in the data, although estimates were less biased when unknowns were included, especially at low percentages of unknowns. However, there was little evidence that the magnitude of the bias varied systematically between disease states. In contrast, state transition rates (ψ) were biased when unknowns were included. Thus, estimates for the autumn–winter subset of year 1 will most probably be underestimated by the presence of unknown state birds because the percentage of unknowns is highest in this subset (Table 1).

Because survival rate estimates were unbiased and more precise and encounter rate estimates were less biased when the unknowns were included, we used a sequential two-step approach to model fitting of our field data. First, using the data including the 'U' state, we constructed a model set where both apparent survival (S) and encounter rate (p) were modelled as a function of several factors we believed a priori might be biologically relevant, such as disease state, sex, temperature and time (discussed below). During this first step, transition rates were constrained to vary only with disease state (i.e. constant over sex, temperature and time), which seemed to be the most biologically important factor. In the second step, we then used the apparent survival and encounter rate structure of the best model from the first stage model set and applied that to the data set where the unknowns were removed (i.e. unknowns transformed to unobserved, equivalent to transforming 'U' to '0' in the encounter histories). In this second-stage candidate model set, model structure for S and p, derived from the first stage of the analysis, was retained for all models, while transition rates among disease states (ψ) were constrained as functions of disease state, sex, temperature and time.

This general problem of state uncertainty (which is a consideration for all multistate analyses) was exacerbated in our study by the need to pool our daily encounter data. While pooling reduces the number of parameters in our models and thus improves parameter precision, it creates difficulty in assigning an 'average state'. If the true average time to transition among states is less than the length of the pooled period, it is possible for an individual to be observed more than once in different states within the same pooled period. We considered two different methods for deriving a representative state over a particular pooling period; (i) assigning each bird a state based on the first encounter during each period (e.g. given an encounter history of '00N00Y0' over a particular period, where N = not infected, Y = infected and 0 = not encountered, the 'average state' for this period would be 'not infected' or 'N') (Hargrove & Borland 1994) and (ii) a quasi-Bayesian approach that determined average state based on the likelihood of a given encounter history, calculated using all of the encounter data for a given bird over a given period (Jennelle *et al.* in preparation). Because the majority of the encounter histories for each individual bird included only one sighting per 7-day period (birds observed more than once in a given 7-day period is 1·48%, $n = 818$), the Bayesian pooling method produced very similar results as the first encounter method (Jennelle *et al.* in preparation). In addition, trapping was conducted earlier in the week and resighting later; thus, if a bird was trapped during a given period, then the correctly assigned state given during trapping would be the 'first encountered state'. Therefore, for simplicity, we used the 'first encounter' method to assign average state for each period in which an individual bird was encountered.

MODEL SELECTION

Our primary objective in this study was to quantify differences among groups of individual finches in terms of apparent survival, encounter and state transition rates. For convenience, we will refer occasionally to state transition rates as either recovery (ψ^{YN}) or infection (ψ^{NY}) rates, with the caveat that these designations are based on clinical signs, and may not reflect the underlying physiological (immunological) processes. For instance, what we called a 'recovered' bird may have still been infectious but not have shown physical signs of conjunctivitis. Similarly, we may have recorded a bird as 'infected', but it may have been infectious when it was not showing physical signs of conjunctivitis. However, as noted before, conjunctival signs are closely related to *Mycoplasma* infection (Hartup *et al*. 2001).

All models were fitted to the data using program MARK (v. 3·1; White & Burnham 1999). Selection among models in the candidate model set was based on comparison of the quasi-likelihood adjusted Akaike Information Criterion corrected for small sample sizes ($QAIC_c$) (Lebreton *et al*. 1992; Burnham & Anderson 2002). $QAIC_c$ values are used to select the best approximating (hereafter, best) model for the data, based on the principles of parsimony and trade-offs between under- and over-fitting models (Burnham & Anderson 2002). The best model was that with the lowest $QAIC_c$ value, and other models were ranked relative to deviations from the best model ($\Delta QAIC_c$). Comparisons among models in the candidate set were accomplished by deriving an index of relative plausibility, using normalized Akaike weights (w_i; Burnham & Anderson 2002). The ratio of w_i between any two models indicates the relative (proportional) support between those two models.

When model selection is based on information-theoretical approaches (e.g. AIC), it is inappropriate to express differences among models in terms of nominal alpha levels, or *P*-values (Royall 1997; Anderson & Burnham 2002a,b; Burnham & Anderson 2002). Thus, we report comparisons among models in terms of relative degrees of support in the data. In addition, we calculated effect sizes to determine the magnitude of differences between disease states and between sexes (where applicable) for apparent survival and encounter rate and between infection (ψ^{NY}) and recovery rates (ψ^{YN}) for transitions. Because the effect size is 'estimated', it will have an associated uncertainty that we can specify in terms of a confidence interval (CI). Because it is not possible to obtain a single effect size estimate for subsets that were influenced by models that had interactions with time and/or temperature, effect size estimates were not included for these subsets in the results. For robust parameter estimates, we accounted for model selection uncertainty by calculating an average value for a parameter over all relevant (structurally consistent) models in the candidate set, weighted by normalized $QAIC_c$ model weights (Burnham & Anderson 2002). Some models contained inestimable parameters that made it necessary to omit these parameters when calculating this average value.

At present, goodness-of-fit tests are not available for reduced parameter multistate models, although they have been developed recently for fully time-dependent models (Pradel, Wintrebert & Gimenez 2003). Thus, because our data were too sparse to fit a fully time-dependent multistate model (such that our most general models were reduced parameter models), we were unable to derive an estimate of lack of fit for the most general model in our candidate model sets. To compensate for this, we looked at the relative rank-ordering and degrees of support among models by varying the estimated lack of fit ($\hat{c}$) from 1·0 to 2·0 (increasing values of $\hat{c}$ imply increasing lack of fit; model selection with larger values of $\hat{c}$ is more conservative, with increasing support for reduced parameter models). Although the rank-ordering of models did not vary within the range of $\hat{c}$ values tested, we used a conservative value 1·5 for $\hat{c}$ to account for various factors that we believe would probably contribute to a lack of fit in our data (e.g. sparse encounters of infected birds, presence of transient birds; discussed later).

ECOLOGICAL COVARIATES

We are interested in potential differences in apparent survival and encounter rate between infected and non-infected birds and between infection and recovery rates. Differences in magnitude of recovery and infection rates can give us much information on the disease status of the population at the end of each year. In addition, we attempt to assess the within-season degree of recovery in the population. Previous work on captive finches has shown varying degrees of mortality and recovery. Earlier captive studies found that mortality associated with *Mycoplasma* is very high (Lutrell *et al*. 1998; Brawner, Hill & Sundermann 2000). This would imply that recovery in the wild would be unlikely because factors such as cold temperatures, lack of food and predation risk contribute to the mortality caused by the disease. Conversely, one recent study found that 73% of their captive flock recovered from the disease (Roberts, Nolan & Hill 2001).

In our study, we investigated the degree to which seasonal variation in apparent survival, encounter and transition rates might reflect differences in a demographic and environmental factor. In particular, we considered sources of variation that we expected might influence, or correlate with, differences in 'physiological state', where physiological stress might attenuate immune responses to *Mycoplasma* infection. For example, brood rearing could impose a greater physiological stress on the female and thus make them more susceptible to disease (Hõrak *et al*. 1998). Nolan, Hill & Stoehr (1998) found that *Mycoplasma* disproportionately killed male finches, due possibly to immunosuppression caused by elevated testosterone levels. Similarly, Dhondt *et al*. (1998) found that finch abundance decreased in areas with cold winters and high conjunctivitis prevalence, suggesting that survival decreases with colder temperature. To account for these possible sources of variation in our data, we included sex and an index to 'wind chill' (based on the standard formula from the US National Weather Service 2001) as a linear covariate in some of our models.

Although age is also a commonly used proxy for physiological and immunological condition (Apanius 1998; Woodworth, Faaborg & Arendt 1999), we did not include either variable in our model set, for several reasons. First, body mass can be assessed only on physical capture, and < 15% of our encounter data involve physical captures. Secondly, the relationship between age and immune state changes dynamically; as the year progresses, hatch-year birds are functionally (immunologically) equivalent to adult birds (Pyle 1997; Apanius 1998). In other words, a 10-month-old bird is still considered a hatch-year bird; however, it is just as susceptible to the disease as an adult bird. Our inability to control for effects of some sources of individual heterogeneity, such as age, may induce small negative bias in our estimates of survival (Prevot-Julliard, Lebreton & Pradel 1998; Williams *et al*. 2002). Effects of heterogeneous survival probabilities have not been well studied (Williams *et al*. 2002).

Another factor that may contribute to heterogeneity in our data is the possible presence of transient individuals. Individuals that are alive but have emigrated permanently from the sample can significantly negatively bias estimates of survival (Pradel *et al*. 1997; Sandercock & Jaramillo 2002). Eastern finches, unlike their western counterparts, have developed a partial migration following introduction (Able & Belthoff 1998), suggesting that transients might be present in our sample. In addition, several areas within the sampling region that contained suitable finch habitat were not sampled due to logistical limitations in the field. Such limited sampling, coupled with low probability of encounter of any individual in general, can lead to 'apparent transience'. While true transience can be accommodated by conditioning estimates of survival to be a function of time since marking (*sensu* Pollock 1981; Pradel *et al*. 1997; Sandercock & Jaramillo 2002; Perret *et al*. 2003) we did not adopt this approach, for two reasons. First, it is not clear how best to address the question of transience for pooled samples, especially for multistate models. Also, we need to differentiate between true transience and apparent transience, which is a form of temporary emigration where the probability of re-entering the sample approaches 0 (Faustino *et al.* in preparation).

The candidate model set consisted of (i) apparent survival rate and transition rate varying with disease state (*dis* = non-infected or infected), sex (*sex* = female or male), time and/or temperature (*temp*) and (ii) encounter rate varying only with disease state, time and/or temperature. We included additive models in the candidate model set to test the significance of the additive effect between disease state, sex and/or temperature. Because there was a very low proportion of infected birds for the autumn–winter subset of years 1 and 2, time variation models were excluded from these candidate model sets. Birds of unknown sex were omitted from the analysis. In the autumn–winter subset of year 3, there were a high proportion of hatch year birds trapped in this period, resulting in a relatively high numbers of birds of unknown sex (Table 1). To accommodate for this we modelled this subset in two stages. In the first stage, we omitted birds of unknown sex and determined that there was no effect of sex in this subset. In the second stage, we reincorporated the unknown sex birds, but did not allow any of the models to vary with sex.

Results

Over all 3 years of the study, we had 1387 initial captures, 941 recaptures and 5703 resights (Table 2). The winter–spring subset in years 1 and 2 and all subsets in year 3 exhibited a notably higher percentage of birds showing clinical signs of conjunctivitis (Fig. 2; Table 1), although the overall proportion of infected birds in the sample was still low (3–14%). The number of unknown state birds in the data was greatest in the autumn–winter subset of year 1. All other subsets had a relatively low percentage of unknowns (Table 1).

Table 2. Numbers of initial captures, recaptures and resights (broken down by sex) for year 1 (2000–01), year 2 (2001–02) and year 3 (2002–03) in Ithaca, NY, USA

	Year 1	Year 2	Year 3	Total
Initial captures	256	469	662	1387
Male	134	241	271	646
Female	113	228	271	612
Unknown sex	9	0	120	129
Recaptures	152	342	447	941
Male	77	172	172	421
Female	71	170	226	467
Unknown sex	4	0	49	53
Resights	1626	1473	2604	5703
Male	900	868	1316	3084
Female	659	602	1122	2383
Unknown sex	67	3	166	236

Because of marked differences between the 3 years of our study in seasonal patterns of *Mycoplasma* prevalence (Fig. 2), each year was analysed separately. The absolute numbers and relative frequencies of infected (Y) birds in our live encounter data were relatively low (Table 1), and thus our estimates of disease state transitions generally had poor precision, especially in the autumn–winter subset of the first 2 years of our study. This is reflected in generally wide confidence intervals for estimated differences in many of our analyses.

APPARENT SURVIVAL (S)

All the best models in every subset contained effects of disease state for weekly apparent survival, and had most of the support from the data (Table 3). The only exception occurs in the autumn–winter subset of year 2, where the two best models had a relatively low support from the data (40%; Table 3). However, the next three most parsimonious models (not shown) contained a disease state effect and these five best models had most of the support from the data (81·8%). After model averaging, we found that apparent survival of non-infected birds was greater than that of infected birds in six of seven subsets (Figs 3 and 4). In the winter–spring

Table 3. Summary of multistate analysis of live encounter data from HOFI population for year 1 (2000–01), year 2 (2001–02) and year 3 (2002–03), in Ithaca, NY, USA. Each year is divided into subsets: S–A = 'summer–autumn' (August–October for year 3 only), A–W = 'autumn–winter' (November–January for all years) and W–S = 'winter-spring' (February–April for years 1 and 2; February–March for year 3) with approximately 12 7-day pooled periods within each subset. The extra subset, S–A, in year 3 was added due to the high encounters of infected birds earlier in this year than in previous years. Model notation: S, survival; p, recapture; ψ, state transition; *dis*, diseases state; *sex*, sex differences; *time*, time differences and *temp*, temperature, modelled as real linear covariates. Additive effects among factors indicated with '+' sign. Interaction effects indicated with '*' sign. The left side of the table represents the first stage model set where S and p were modelled as a function of *dis*, *sex*, *time* and *temp* with 'unknowns' included in the data and ψ modelled only as a function of *dis*. The right side represents the second stage where ψ was modelled as a function of *dis*, *sex*, *time* and *temp* with 'unknowns' removed from the data and the structure of S and p is retained from the most parsimonious model of the first stage model set (see Methods: model selection). Lower Delta $QAIC_c$ values show better fit. Only models with $QAIC_c$ weights > 0·15 are listed with the most parsimonious model at the top. The Akaike weights indicate the relative support that a given model has from the data, compared to the other models in the set. The deviance is the difference in –2 log likelihood between the current model and the saturated model, the saturated model being the one with the number of parameters equal to the sample size. The number of estimated parameters is shown (no. par.). A $\hat{c}$ (estimated lack of fit) of 1·5 was used (see Methods)

			Model statistics					Model statistics			
Subset	S	p	$QAIC_c$ weight	Delta $QAIC_c$	Deviance	no. par.	ψ	$QAIC_c$ weight	Delta $QAIC_c$	Deviance	no. par.
Year 1 (2000–01)											
A–W	*dis* * *sex*	*dis* * *temp*	0·313	0·00	672·9	15	*temp*	0·622	0·00	239·7	9
	dis	*dis* * *temp*	0·287	0·17	675·2	14	*dis* * *time*	0·261	1·74	205·3	25
	dis + *temp*	*dis* * *temp*	0·170	1·22	676·2	14					
W–S	*dis* + *sex*	*dis* * *time*	0·986	0·00	593·9	34	*dis*	0·340	0·00	451·5	26
							temp	0·316	0·14	451·7	26
Year 2 (2001–02)											
A–W	*dis*	*dis* * *temp*	0·222	0·00	640·7	11	*dis*	0·990	0·00	463·0	2
	dis	*temp*	0·205	0·16	647·0	8					
W–S	*dis* + *time*	*dis* + *temp*	0·937	0·00	552·5	19	*dis* * *temp*	0·432	0·00	427·3	14
							dis + *temp*	0·362	0·35	427·6	14
Year 3 (2002–03)											
S–A	*dis* + *time*	*dis* + *temp*	0·992	0·00	798·1	20	*dis* * *temp*	0·545	0·00	696·6	12
							dis + *temp*	0·439	0·43	697·0	12
A–W	*dis* * *sex*	*dis* * *time*	0·483	0·00	674·4	35	*dis* + *sex*	0·325	0·00	475·9	24
	dis + *temp*	*dis* * *time*	0·438	0·19	679·0	33	*dis*	0·217	0·80	481·0	22
W–S	*dis* * *time*	*dis* * *time*	0·522	0·00	442·5	27	*dis* + *temp*	0·349	0·00	197·3	21
	dis * *temp*	*dis* * *time*	0·287	1·20	450·7	24	*dis*	0·311	0·23	197·5	21
							temp	0·203	1·09	200·3	20

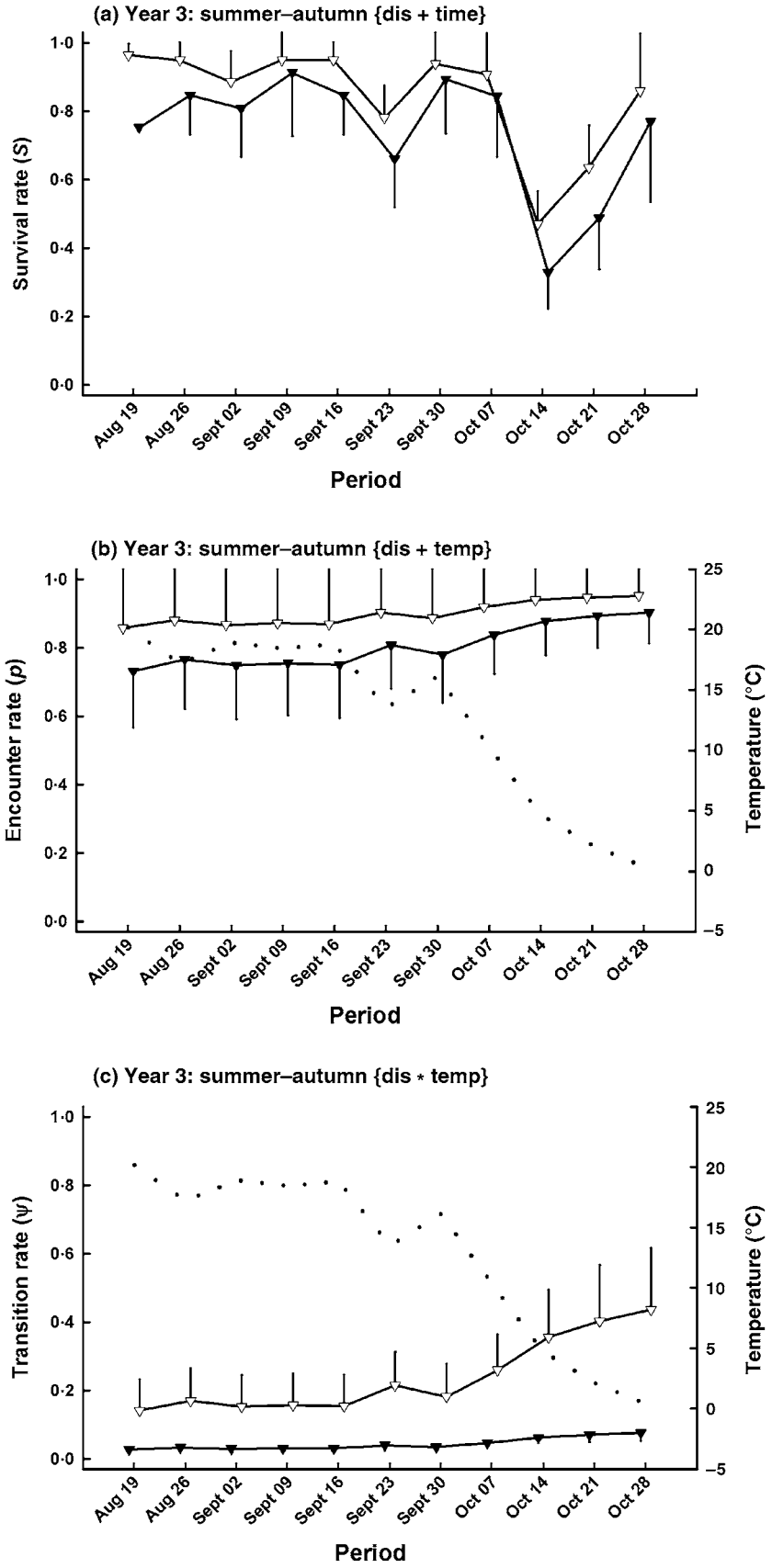

Fig. 3. Estimated house finch survival (S), encounter (p), and transition rates (ψ) for the summer–autumn (August–October) subset of year 3. (a,b) Estimates calculated per 7-day period, averaged over models in the candidate model set, for survival and encounter rate of non-infected (▽) and infected birds (▼). (c) shows estimates of infection rate, ψ^{NY} (▼)) and recovery rate, ψ^{YN} (▽). Estimates are shown with standard error (SE) bars (for clarity, only one orientation of the SE is indicated). Dotted line in (b) and (c) indicates per period value of the derived index of temperature expressed as real values; in these two subsets, estimates showed a pattern that could be associated with temperature. The best model for each subset is indicated in brackets {}.

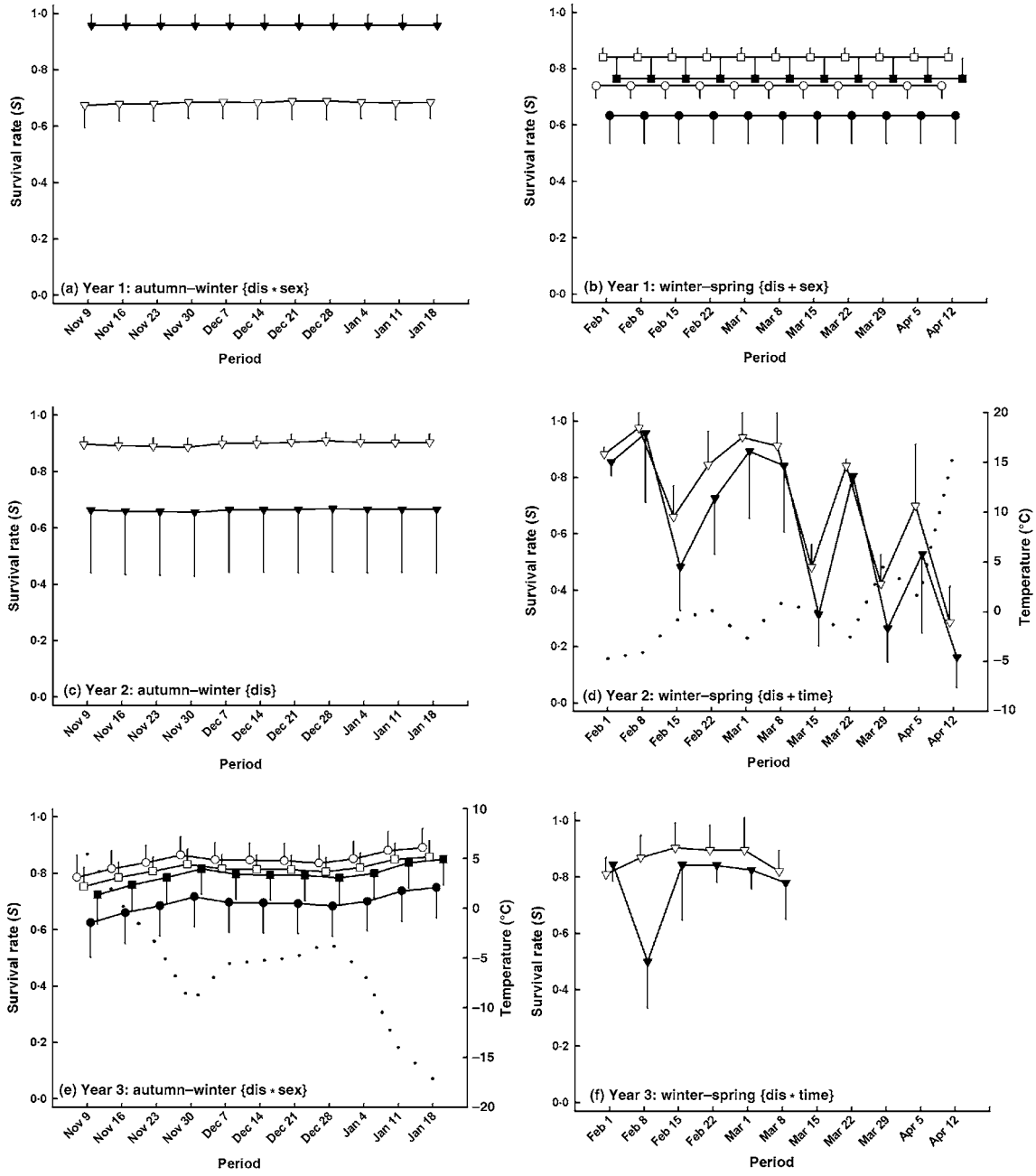

Fig. 4. Estimated house finch weekly survival rates (S) for autumn–winter (November–January) and winter–spring (February–March/April) subsets of the data, for year 1 (2000–01), year 2 (2001–02) and year 3 (2002–03). Estimates shown are calculated per 7-day period, averaged over models in the candidate model set, for non-infected, S_N (▽) and infected birds, S_Y (▼). (b, e) Estimates for non-infected (○) and infected (●) females, and non-infected (□) and infected (■) males; in these two subsets, there was support for models with differences in weekly survival rates between the sexes. Estimates are shown with standard error (SE) bars (for clarity, only one orientation of the SE is indicated). Dotted line in (d) and (e) indicates per period value of the derived index of temperature expressed as real values; in these two subsets, estimates showed a pattern that could be associated with temperature. The best model for each subset is indicated in brackets {}.

subset of year 1, apparent survival of non-infected birds was greater than that of infected birds (for females only, by 0·105 ± 0·379; 95% CI = −0·652–0·863; Fig. 4b). The autumn–winter subsets of years 2 and 3 show similar results (year 2: by 0·231 ± 0·504; 95% CI = −0·776–1·24; year 3: for females only, by 0·364 ± 0·339; 95% CI = −0·314–1·04; Fig. 4c,e). We found an exception in the autumn–winter subset of year 1, where infected birds had greater apparent survival than non-infected birds (by 0·283 ± 0·341; 95% CI = −0·399–0·966; Fig. 4a).

We found that three of seven subsets retained a sex effect as the best model for that subset (Table 3). However, after model averaging, only two subsets showed possible differences between sexes. In the winter–spring subset of year 1, apparent survival of males was greater than that of females (by 0·130 ± 0·424; 95% CI = −0·718–0·979; Fig. 4b). The autumn–winter subset of year 3 showed contrasting results; infected males had higher apparent survival than infected females (by 0·100 ± 0·501; 95% CI = −0·902–1·10; Fig. 4e), but non-infected

females had slightly higher apparent survival that non-infected males (by 0·0317 ± 0·403; 95% CI = −0·774–0·838; Fig. 4e). An effect of temperature was contained in models from three of seven subsets (Table 3). Of these subsets, only the autumn–winter subset of year 3 showed a pattern that could be associated with temperature; as temperature decreased, apparent survival increased (Fig. 4e). Although the best model in the winter–spring subset of year 2 did not contain a temperature effect, this subset showed a similar temperature pattern due to some weight given to the model, $\{S_{temp}\}$ (Fig. 4d).

ENCOUNTER RATE (P)

All of the best models contained a disease effect in each subset, strongly indicating a difference in encounter rate between disease states (Table 3). Model averaged values show that non-infected birds had a higher encounter rate than infected birds in six of seven subsets (Figs 3 and 5). One exception occurs in the winter–spring subset of year 2, where infected birds had a higher encounter rate than non-infected birds (by 0·0787 ± 0·403; 95% CI = −0·727–0·884; Fig. 5d). We were unable to derive effect sizes for any other subsets due to models of a moderate amount of weight having an interaction with time or temperature.

The best models in four of seven subsets contained a temperature effect (Table 3). Three of these subsets showed a similar pattern that could be associated with temperature; as temperature increases, encounter rate increases (Fig. 5a,c,d). However, for the autumn–winter subset of year 1, this pattern is true only for non-infected birds (Fig. 5a). The summer–autumn subset of year 3 shows an opposite pattern; as temperature increases, encounter rate decreases (Fig. 3b)

DISEASE STATE TRANSITIONS (ψ^{IJ})

Six of seven subsets strongly suggested a difference between transition rates and had most of the support from the data (Table 3). After model averaging, five of seven subsets showed that recovery rate was higher than infection rate (Figs 3 and 6). This difference in transition rate was smallest in the winter–spring subset of year 3 (by 0·0622 ± 0·249; 95% CI = −0·436–0·560; Fig. 6f) and was largest in the autumn–winter subset of year 3 (for females only; by 0·0364 ± 0·339; 95% CI = –0·314–0·104; Fig. 6e). Two exceptions occur in year 1, where the infection rate is greater than recovery rate in the autumn–winter subset, and where there is no real difference between transition rates in the winter–spring subset (Fig. 6a,b).

There was a sex effect in the best model of only the autumn–winter subset of year 3 (Table 3). Recovery rate for females was greater than that of males (by 0·112 ± 0·424; 95% CI = –0·735–0·961; Fig. 6e). There was no real difference between sexes of infection rate. Four of seven models showed an effect of temperature (Figs 3 and 6). However, after model averaging, three of these showed contradictory patterns that could be associated with temperature. The autumn–winter subset of year 1 showed that as temperature increases, both transition rates increases (Fig. 6a), while the winter–spring subset of year 2 and the summer–autumn subset of year 3 showed that as temperature increases, the recovery rate decreases (Figs 3c and 6d).

Discussion

There has been a long-standing interest in the role of disease on the dynamics of populations, particularly with reference to human populations, where infectious diseases have been documented as a significant component of morbidity and mortality for at least the past 10 000 years (Haldane 1949; May 1988). Diseases have also been known to afflict many animal and plant species, but their role in the dynamics of populations of these species has received comparatively little attention (May 1982; 1988; Newton 1998). Since the seminal publication of David Lack (1954), the general belief has been that pathogens evolved to have a rather benign effect on their host, thus ensuring a better chance of long-term survival of the infectious organisms (Hudson & Dobson 1991). Only relatively recently have the negative consequences of disease on host populations been considered; disease pathogens are dynamically engaged with the host population and can have a highly detrimental effect on the host (May 1983; Hudson *et al*. 2002).

In this study, we used live mark–encounter data to test the influence of three factors (disease state, sex and temperature) on apparent survival, encounter and transition rates between disease states. Although our analyses were complicated by both the low frequency of encounters with infected individuals (especially in the first half of the first 2 years of our study; Table 1), and uncertainty concerning disease state, we are able to draw several conclusions based on the cumulative evidence in our results. First, we found strong support for the hypothesis that disease state has an effect on apparent survival – in seven of seven subsets of the data the best models with $QAIC_c$ weight > 0·15 included a disease state effect (Table 3). Non-infected birds tended to have greater weekly apparent survival than did infected birds in six of seven subsets (Figs 3 and 4). The consistency of this trend strongly suggests that, in general, weekly apparent survival of infected birds is lower than that for non-infected birds. This is consistent with expectations from previous studies that show that *Mycoplasma* infection reduces finch survival.

Previous studies of captive birds (referenced earlier) clearly indicated a negative impact of *Mycoplasma* infection on finch survival. Thus, our results showing that *Mycoplasma* did have a negative effect on survival in the wild are perhaps not surprising. However, assessing the degree to which these differences in apparent survival, and the variation in infection and recovery rates is complicated by several factors. Our study population is open to both emigration and immigration.

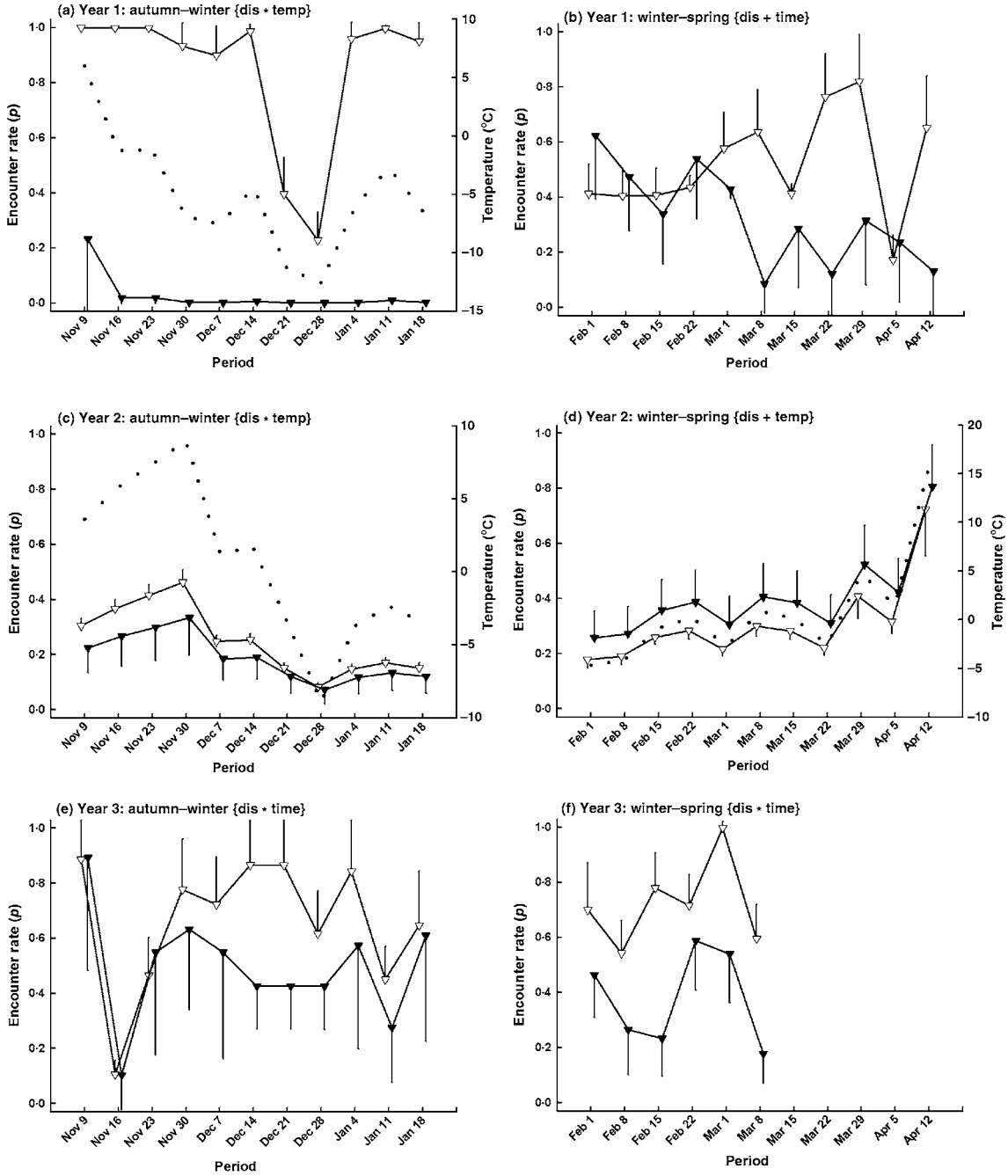

Fig. 5. Estimated house finch encounter rates (p) for autumn–winter (November–January) and winter–spring (February–March/April) subsets of the data for year 1 (2000–01), year 2 (2001–02) and year 3 (2002–03). Estimates shown are calculated per 7-day period, averaged over models in the candidate model set, for non-infected, p_N (▽) and infected birds, p_Y (▼)). Estimates are shown with standard error (SE) bars (for clarity, only one orientation of the SE is indicated). Dotted line in (a) (c) and (d) indicates per period value of the derived index of temperature expressed as real values; in these two subsets, estimates showed a pattern that could be associated with temperature. The best model for each subset is indicated in brackets {}.

More specifically, lower apparent survival of infected birds could be due to changes in behavioural interactions; it is plausible that infected birds are shunned from the flock, or are not able to compete with non-infected birds at the feeders. This could cause the infected bird to emigrate permanently from the study area. Lower apparent survival of infected birds on local abundance, or on local *Mycoplasma* prevalence, cannot be assessed without estimates of disease-state specific rates of immigration and emigration into the population. Deriving these estimates will require adopting a different sampling and analysis approach. Thus, we believe our estimates of weekly apparent survival are negatively biased. Based on weekly apparent survival estimates (from year 2) of 0·81 for non-infected individuals and 0·64 for infected individuals, and weekly recovery and infection rates of 0·03 and 0·15, respectively, few individuals (< 1%) would be expected to survive the 6-month

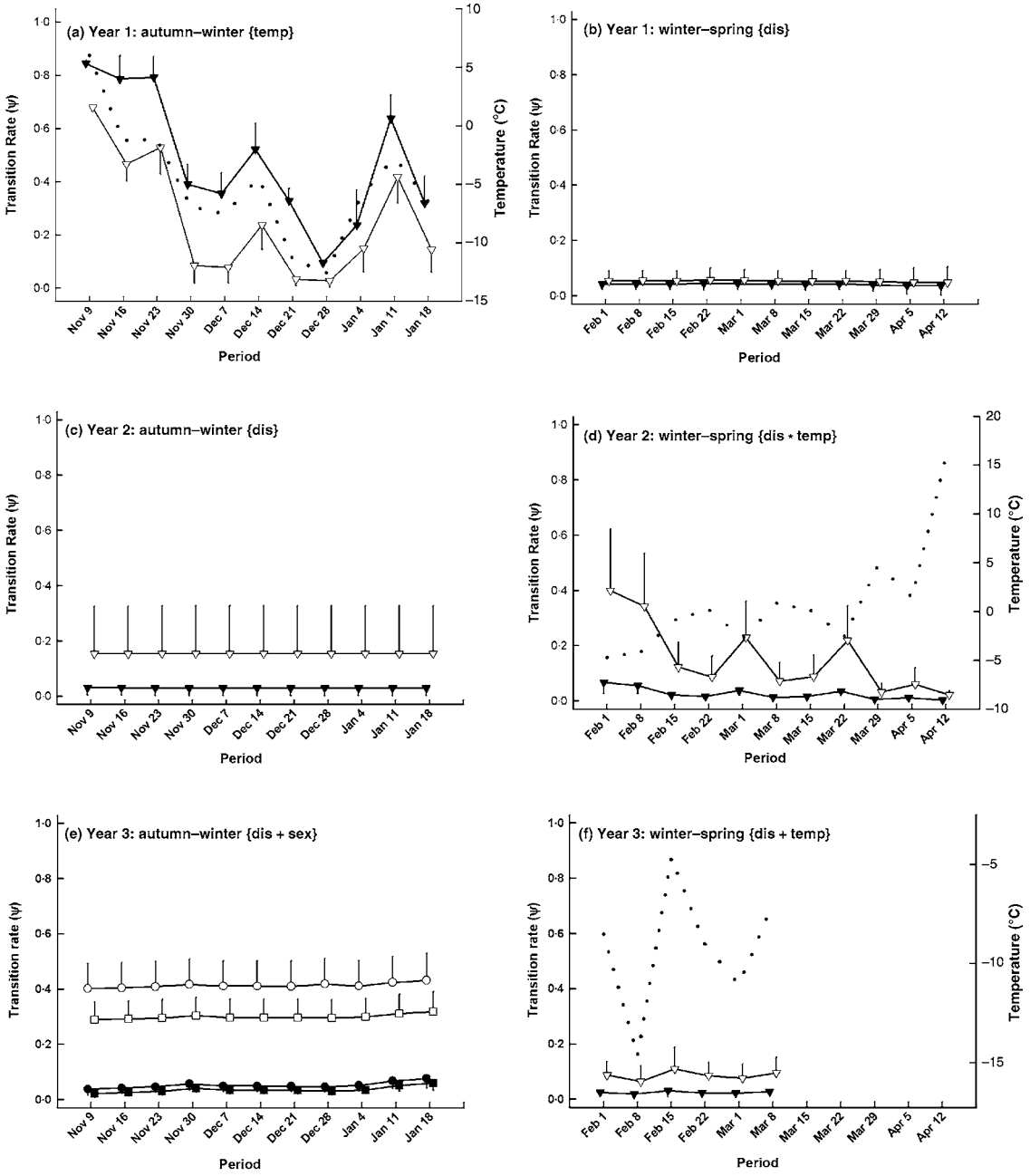

Fig. 6. Estimated house finch transition rates (ψ) for autumn–winter (November–January) and winter–spring (February–March/April) subsets of the data for year 1 (2000–01), year 2 (2001–02) and year 3 (2002–03). Estimates shown are calculated per 7-day period, averaged over models in the candidate model set, for infection rate, ψ^{NY} (▼) and recovery rate, ψ^{YN} (▽). (e) shows estimates of infection rate (●) and recovery rate (○) of females, and infection rate (■) and recovery rate (□) of males; in these two subsets, there was support for models with differences in weekly survival rates between the sexes. Estimates are shown with standard error (SE) bars (for clarity, only one orientation of the SE is indicated). Dotted line in (a) and (d) indicates per period value of the derived index of temperature expressed as real values; in these two subsets, estimates showed a pattern that could be associated with temperature. The best model for each subset is indicated in brackets {}.

period from November to April. However, in our data we found that 13·43% of individuals survive at least 1 year (based on the proportion of birds marked in the first year that were observed in the second year). This proportion is a minimum estimate of apparent survival because it does not account for individuals that were not encountered in the second year but are still alive.

The most likely reason for a negative bias in our estimates of apparent survival is heterogeneity among individuals in our sample. If a subset of our population contains temporary emigrants, this can result in significant heterogeneity of recapture probabilities. Temporary emigration coupled with low overall encounter rates can mimic true transience ('apparent transience'). True transience is an extreme form of heterogeneity, where the probability of encounter following the initial marking event is zero (i.e. never encountered after first capture). Like permanent emigration, true transience

can result in negatively biased survival rates. If the true encounter rate is low and temporary emigration rate is high, then the estimated encounter rate for some individuals can approach zero, which is equivalent to the condition for true transience. Transience in general leads to negatively biased estimates of survival, especially in the weeks immediately following marking (Sandercock & Jaramillo 2002).

The second conclusion based on cumulative evidence in our results is that our data also show clearly that recovery from *Mycoplasma* in the wild is possible; recovery rates over a 7-day period ranged from 2·29% to 67·9% (Figs 3 and 6). This is consistent with high recovery rates (73–80%) in captive finches infected with *Mycoplasma* (Roberts *et al*. 2001; Kollias *et al.* in review), yet contradictory to earlier studies that found high mortality in captive finches infected with *Mycoplasma* (Lutrell *et al*. 1998; Brawner *et al*. 2000). In general, recovery rates (i.e. the probability of making the transition from infected to non-infected) tended to be greater than infection rates (i.e. the probability of making the transition from non-infected to infected; Figs 3 and 6). Although similar results have been reported (Senar & Conroy in review) for a population of serins (*Serinus serinus*) infected by avian pox (*Poxvirus avium*), the greater recovery than infection rate suggested by our analyses was initially surprising. Because we do not see a greater infection rate at any point in either year and we have a very small pool of infected individuals, the greater recovery rate could be due to a bias from birds that become infected and die before they are encountered as infected, or are not seen due to a lower encounter rate (Figs 3 and 5; discussed below). If these infected birds were re-encountered, they would have been accounted for in estimates of infection rate. In addition, these transition rates could be biased due to individuals making rapid transitions between disease states. There are several examples of individual birds that have shown clinical signs of conjunctivitis, showed no clinical signs on the next observation, and then showed clinical signs again (or vice versa) in our data. Whether or not this is a true reinfection or an error in assigning disease states, these birds would be considered as having been infected twice, effectively increasing the infection rate.

Thirdly, we found that encounter rate differed as a function of disease state; overall, encounter rates for infected individuals were lower than those for non-infected individuals (Figs 3 and 5). The most likely explanation for the difference in encounter rate between diseased and non-diseased individuals in our study is the lower probability of feeder visits by infected birds. *Mycoplasma* infection is likely to reduce the visual acuity of infected individuals, and may reduce the chances of successfully visiting feeders. Additionally, Kollias *et al*. (in review) reported that acutely infected birds become less active, hence possibly reducing the encounter probability. The estimated encounter rate in our analyses is a product of the true encounter probability (the probability of encountering the individual conditional on it being in the sample area) and the non-zero probability of being in the entire study area (Kendall, Nichols & Hines 1997). If an individual bird temporarily leaves the sampling site near the feeders, then this will lower the estimated encounter rate. If the true encounter probability of infected and non-infected individuals is the same, then the lower estimated encounter rate for infected birds would suggest that infected birds are more likely to have temporarily emigrated from the sample than non-infected birds.

We detected only a very slight influence of sex on estimated apparent survival and transition rates. Similarly, in a recent study of the effects of avian pox (*Poxvirus avium*) on survival of serins (*S. serinus*), no interaction of sex, pox infection and survival was detected (Senar & Conroy in review). We had anticipated that the stress from breeding during the summer might make one sex more susceptible to *Mycoplasma* than the other. Our lack of a detectable sex effect might have several explanations. First, the data were rather sparse and a lack of power in our analyses could have prevented us from detecting any differences in apparent survival, encounter or transition rates between sexes. Secondly, it is possible that the physiological stress caused by breeding could have diminished by the time *Mycoplasma* prevalence begins to increase in the autumn. Finally, sex cannot be distinguished in hatch-year finches and the presence of significant numbers of hatch-year individuals in the sample may have minimized apparent differences in apparent survival and rates of infection or recovery between the sexes.

We also found marginal evidence of an influence of temperature on apparent survival, encounter and transition rates. The direction of the relationship between temperature and apparent survival that we observed was opposite to what we expected (Dhondt *et al*. 1998); in the year 2 winter–spring subset and the year 3 autumn–winter subset, as temperature increased, apparent survival decreased. Although the relationship between temperature and transition rates was inconsistent, a nearly consistent pattern was found for encounter rate; as temperature increased, encounter rate increased. However, these relationships were weak and may in fact reflect covariation of temperature with factors that are important, but not identified (e.g. population density, relative proportions of individuals breeding).

SEASONAL VARIATION IN *MYCOPLASMA* PREVALENCE: CONTEXT AND CONSEQUENCES

Previous analyses of *Mycoplasma* infection in finches indicated a bimodal seasonal pattern in disease prevalence (Altizer *et al.* 2004). We did not find bimodal seasonal variation, but observed higher disease prevalence in the winter–spring subsets for the first 2 years and in the summer–autumn subset of year 3 (Fig. 2). This might be explained by the observation that the magnitude of difference between the bimodal peaks

and the summer minima is lowest in northern latitudes of the United States (Altizer *et al.* 2004). Ithaca, NY would be considered as part of this region and would thus not exhibit the bimodal peaks as dramatically as in the more southern latitudes. In addition, the discrepancy could also be attributable to differing spatial scales over which data were collected. The bimodal seasonal patterns were observed over large geographical regions of the United States, while our data were collected in a 4·6-km^2 area.

We found that the higher disease prevalence in the winter–spring subsets of years 1 and 2 (Fig. 2) are not consistent with the results for apparent survival and transition rates. We found disease prevalence to peak at around February for both years and then decrease subsequently (Fig. 2). If the decrease in prevalence were caused by permanent emigration, our apparent survival results would reflect this; however, we found that apparent survival remains at relatively high levels (Fig. 4b,d). Similarly, if the decrease were caused by recovery of previously infected individuals, our estimates of recovery rate would reflect this; however, our estimates of recovery rates remain at relatively low levels (Fig. 6b,d). Therefore, it is likely that the decrease is caused by temporary emigration from the sample, providing more evidence for apparent transience in our study.

ASSESSING DISEASE DYNAMICS IN THE WILD

We believe our study has implications for analysis of the role of disease on finch dynamics, and in disease dynamics of wild populations in general. First, although *Mycoplasma* is harmless to humans and currently affects seriously only finches, an introduced species in the eastern United States, the disease had been identified recently in some native species such as American goldfinches (*Carduelis tristis* L.), purple finches (*C. purpureus* Gmelin), evening grosbeaks (*Coccothraustes vespertinus* Cooper) and pine grosbeaks (*Pinicola enucleator* L.), albeit at a lower rate than finches (Hartup *et al.* 2001; Mikaelian *et al.* 2001). In addition, *Mycoplasma* prevalence in finches continues to spread spatially; *Mycoplasma* has been confirmed in the native western range of finches (Duckworth *et al.* 2003). Further infection could be detrimental to that population. Secondly, the multistate mark–recapture methods used here are useful for estimating parameters such as survival, infection and recovery rates in studies of wildlife disease dynamics. In particular, use of multistate mark–recapture methods allowed us to control formally for any differences in encounter rates between disease states. These methods could be applied in studies of other wildlife infectious diseases, such as West Nile virus, chronic wasting disease and avian pox in native Hawaiian birds. To date, most studies on these diseases have been experimental, or studies that used counts to determine disease prevalence (Miller *et al.* 2000; McLean *et al.* 2001; Van Riper, Van Riper & Hansen 2002); however, these studies have not used the full power of mark–recapture methods for estimating demographic parameters related to disease dynamics.

More generally, our studies have indicated a variety of factors that could be relevant in the analysis of disease dynamics in the wild. First, we show clearly that encounter rate may differ among disease states. Assessment of prevalence in the wild (or in human populations) is conditional on assessing the relative proportions of individuals in each state. However, interpreting variation in these proportions is valid, conditional on knowing the relative detectability of each disease state; if, as is the case in our study, infected individuals are less likely to be encountered than non-infected individuals, then estimates of prevalence will be negatively biased. Secondly, our multistate modelling assumed that transitions were entirely first-order Markovian. For disease models in general, where the probability of recovery (or infection) may be a function of the preceding disease history, higher-order Markov models (i.e. memory models) may be more relevant. However, as noted, such models require extensive data to parameterize, and may be difficult to apply to studies such as ours with generally low encounter rates. Finally, our estimates are based upon the assumption of conditional independence among individuals (at least, within some level of stratification such as sex). Derivation of estimates for survival and transition given specific patterns of flock cohesion, flock structure and dominance behaviours are needed (Jennelle *et al.* in preparation; Hawley *et al.* in preparation). In particular, the degree to which patterns of movement into and out of our sampling regions may be influenced by particular patterns of social structuring which is currently unknown. This social structuring, may in turn be influenced by the pattern of disease prevalence and may be a generally important consideration for analysis of disease dynamics in any population with significant social structuring (Jennelle *et al.* in preparation).

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Appendix I

STATE UNCERTAINTY AND PARAMETER ESTIMATION

For some individuals, the 'average state' over a given 7-day period might be 'U' (unknown). To assess the degree to which the presence of an uncertain state might bias estimates of one or more parameters, we conducted a preliminary numerical simulation study. We created simulated encounter histories where survival, encounter and transition rates for infected and non-infected birds were known. We also simulated the process by which a bird is placed into the unknown state (discussed in Methods: Analysis of live encounter data). The simulated data were analysed in two ways. First, we analysed the data with the unknown state observations included in the data set. Thus, the encounter histories could consist of 'N' (not infected), 'Y' (infected), 'U' (unknown) or '0' (not seen). In this analysis, we modelled explicitly the unknown state observations as a discrete state. The global model, consisting of each parameter varying with disease and without time-dependence, $\{S_{dis}\, p_{dis} \psi_{dis}\}$, was analysed along with all nested models. The global model represented the correct structure of the simulated data. This analysis was followed by analysis of the data where unknown state observations were excluded (i.e. unknowns were replaced with zeroes, indicating that the bird was not seen at all); encounter histories could consist of 'N', 'Y' or '0'. The candidate model set for this analysis was identical. We considered both approaches because a bird that is classified as unknown state does provide partial information; while disease state is unknown, the bird is known to be alive. An observer that assigns the 'U' state usually is able to read the colour ring combination, but is not able to see the eyes of the bird at all before it flushes. Because no assessment of the eyes was made, we assumed that designation of the 'U' state was independent of disease state.

Based on a simulation analysis of 10 000 individuals, over seven occasions, we found that survival rates for both infected and non-infected individuals were essentially unbiased both with and without the unknowns in the data. When unknowns are removed, survival of infected birds (S_Y) began to stray slightly from the true value of S_Y when the percentage of unknowns was greater than 15% (Appendix, Fig. A1a). The encounter rate of infected (p_N) and non-infected birds (p_Y) was

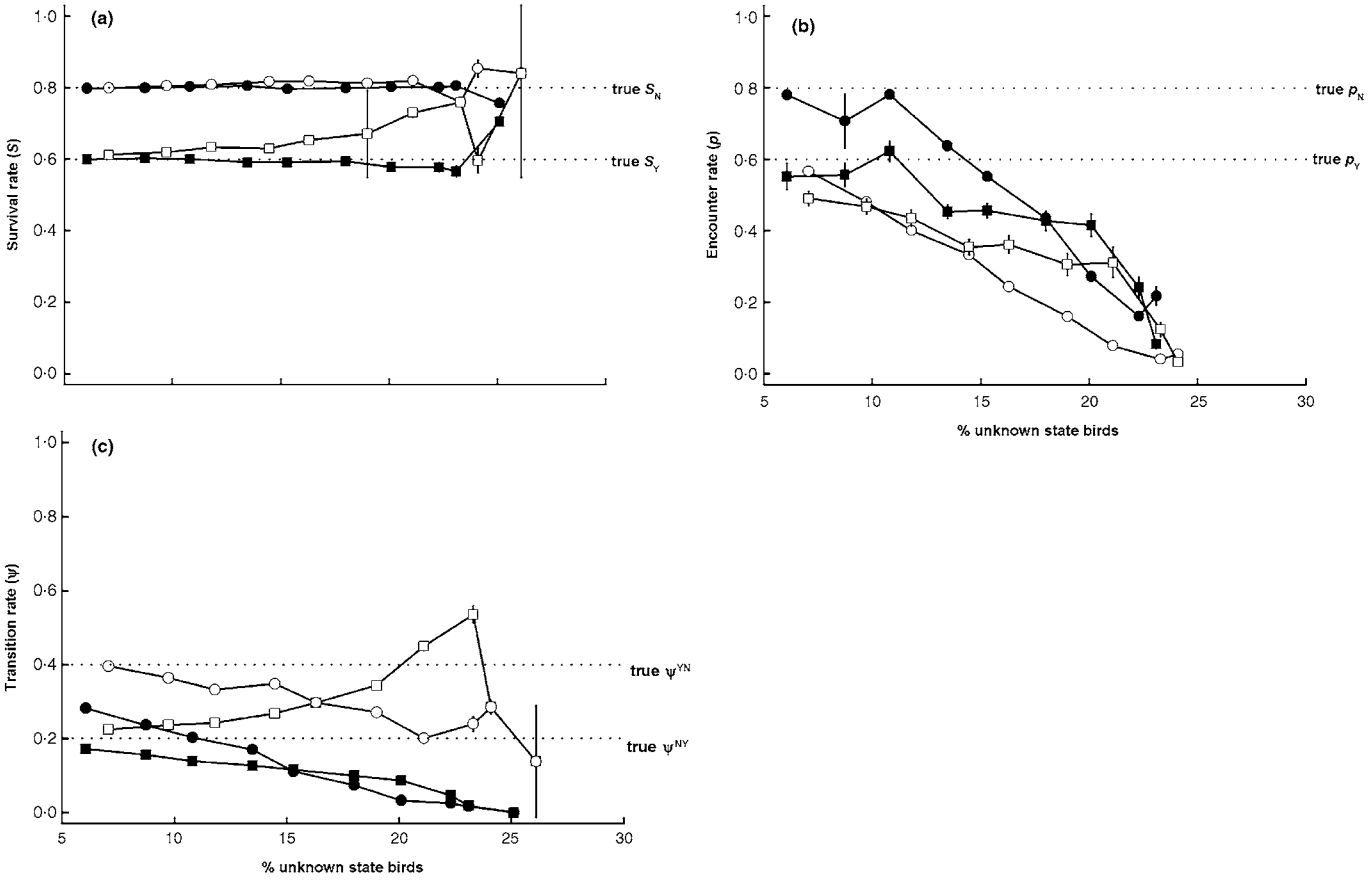

Fig. A1. Analysis of bias (deviance from true value) in house finch survival, encounter and transition rates estimated from simulated data, as a function of increasing percentage of unknown state birds in the data (see Fig. 3; Methods). Estimates are shown with standard error (SE) bars. (a) Estimated survival rate of infected, S_Y (■) and non-infected birds, S_N (●) with unknowns included in the data, and survival of infected (□) and non-infected birds (○) with unknowns removed from the data. (b) Estimated encounter rate of infected, p_Y (■) and non-infected birds, p_N (●) with unknowns included in the data and encounter rate of infected (□) and non-infected birds (○) with unknowns removed from the data. (c) Infection rate, ψ^{NY} (■) and recovery rate, ψ^{YN} (●) with unknowns included in the data and infection rate (□) and recovery rate (○) with unknowns removed from the data. For all three parameters, true values are indicated by dashed lines.

biased both with and without the unknowns in the data (Appendix, Fig. A1b). When unknowns were removed, the resulting data had fewer encounters, generating lower and more biased estimates of p. When unknowns were included, encounter rates were least biased at low percentages of unknowns (approx. < 12%). In contrast, the recovery rate (ψ^{YN}) was biased when unknowns were included in the data, but unbiased when (i) unknowns were treated as unobserved and (ii) at low percentages of unknowns (approx. < 15%). The infection rate (ψ^{NY}) for both unknowns included and unknowns removed showed a similar pattern with respect to the true value of ψ^{NY} (Appendix, Fig. A1c). Estimates for survival and transition rates were less precise (lower standard errors) when the unknowns were removed, especially at higher percentages of unknowns (approx > 12%). Encounter rates were slightly less precise with unknowns in the data.