

ACCOUNTING FOR DETECTION HETEROGENEITY AND HOST
MOVEMENTS IN A HOUSE FINCH-MYCOPLASMA GALLISEPTICUM
DISEASE SYSTEM

A Dissertation

Presented to the Faculty of the Graduate School
of Cornell University

In Partial Fulfillment of the Requirements for the Degree of
Doctor of Philosophy

by

Christopher Shane Jennelle

August 2007

© 2007 Christopher Shane Jennelle

ACCOUNTING FOR DETECTION HETEROGENEITY AND HOST
MOVEMENTS IN A HOUSE FINCH-MYCOPLASMA GALLISEPTICUM
DISEASE SYSTEM

Christopher Shane Jennelle, Ph. D.

Cornell University 2007

During the course of my dissertation research, I made use of capture-recapture methods to investigate local house finch (*Carpodacus mexicanus*) demography and movements in the context of understanding seasonal *Mycoplasma gallisepticum* (MG) infection dynamics. Capture-recapture design, estimation, and modeling explicitly accounts for variable detectability of individuals and provides a framework for making multi-model inference, thereby incorporating inherent model selection uncertainty (via Akaike information criterion) into the inferential process. The biological focus throughout my research has generally been centered on the relationship between local spatial scale host population structure, movements, and host-pathogen dynamics. Broadly, my work illustrates the importance of accounting for animal detection probabilities when estimating epidemiological statistics and parameters. I also highlight the importance of considering different forms of animal movements (either biologically induced or as a consequence of sampling design) with respect to understanding dynamics in the finch-MG system (specifically), but also applicable to other host-pathogen systems (generally). I estimate host transient movements, completely observable within-study area movements, proportional recruitment, and temporary movements from the study area (representing partially observable movements); all of which are very important elements to consider for understanding the dynamics of highly mobile animal populations (especially in the presence of a

virulent pathogen). My research, conducted at a local spatial scale in Ithaca, NY complements analyses using House Finch Disease Survey data (Dhondt et al. 1998) at a broader spatial scale, and provides a point of entry for understanding the critical linkage of scale dependent processes influencing finch-MG dynamics. Throughout this dissertation, I have sought to characterize the structure of this local finch population, and establish how both host population structure and movements lead to a better overall understanding of MG infection dynamics. As such, the complete body of work produced here represents the most comprehensive investigation of wildlife disease dynamics to date, which has incorporated and accounted for sampling and biologically driven heterogeneity in host encounter probabilities. Beyond the proximate benefits that this research contributes to understanding of the finch-MG system, my hope is that this work will in part serve as a precedent for future empirical investigations of wildlife-pathogen dynamics.

BIOGRAPHICAL SKETCH

Christopher Jennelle earned a Bachelor of Science degree with a focus in Wildlife Science from Rutgers University (Cook College), along with a GIS certification in 1997. Continuing on with graduate education, he attended the University of Arkansas (Fayetteville) and graduated with a Master of Science degree in Avian Ecology in 2000. After working as a temporary Biologist at Patuxent Wildlife Research Center (under the direction of Dr. William Kendall), he started a Ph.D. program in the Department of Natural Resources at Cornell University under Dr. Evan Cooch in Fall 2001. Following completion of his Ph.D., Chris now resides in Madison, WI studying Chronic Wasting Disease transmission potential as a post-doctoral researcher under Dr. Michael Samuel.

I dedicate this work to my Mom (Mary Wills Langdana) and Dad (Farrokh Langdana), for all of my achievements leading up to and including this dissertation would not have been possible without their love, support, and encouragement. I share this accomplishment with them and any small measure of limelight (although perhaps a handful of people will ever pick up this book). I also dedicate this dissertation to the rest of my family and friends who have supported me through these years.

ACKNOWLEDGMENTS

Many thanks to my parents, family members, friends, fellow graduate students, and department staff for their help and support in so many ways. I thank my advisor, Evan Cooch, for his guidance and support. Not only did he provide me with an arsenal of quantitative and technical skills, but also he was instrumental in guiding my thought processes as a scientist. The capacity to conceive of, address, and package broadly relevant ecological questions are invaluable skills (and arts) that are earned with careful thought and experience. I gratefully thank Evan for setting me upon that path. I also thank my committee members André Dhondt, William Kendall (for invaluable technical support and guidance with capture-recapture analysis), Cliff Kraft (for discussion of broad ecological considerations), and Patrick Sullivan (for valuable statistical guidance and direction) for their support and many fruitful discussions. I especially acknowledge André for being a first-rate mentor; in effect he functioned as a second major advisor. I thank the Cornell Department of Natural Resources, Laboratory of Ornithology, and NSF (under grant DEB no. 0094456) for financial support. Of course I must acknowledge the millions of House Finches out there, some of which unwillingly took part in my research. I have gained a great respect for their social complexity, amazing endurance, and resilience in the face of numerous environmental, physiological, and anthropogenic challenges.

TABLE OF CONTENTS

BIOGRAPHICAL SKETCH.....	<i>iii</i>
DEDICATION	<i>iv</i>
ACKNOWLEDGMENTS.....	<i>v</i>
LIST OF FIGURES.....	<i>viii</i>
LIST OF TABLES	<i>ix</i>
CHAPTER 1.....	<i>10</i>
Modeling disease dynamics	10
Structure of the thesis	12
Implications of my research	18
Limitations and future considerations	19
Collaborations	26
REFERENCES	27
CHAPTER 2.....	32
Introduction	33
Methods	40
Results	48
Discussion	53
APPENDIX A. General Formula for Corrected Prevalence	67
APPENDIX B. Approximate Conditional Variance for Prevalence Estimator	68
APPENDIX C. Bias Evaluation of an Approach to Prevalence Estimation in a Special Case.	70
APPENDIX D. Weekly Counts, Estimated Encounter Probabilities, Apparent Prevalence, Corrected Prevalence, and Percent Relative Bias	76
APPENDIX E. Bias in Apparent Prevalence as a Function of a Range of Differences in Health State-specific Detection Probabilities	77
REFERENCES	80
CHAPTER 3.....	88
Introduction	89
Methods	94
Results	107
Discussion	125
APPENDIX A. Map of Study Area	138
APPENDIX B. Capture Data	139

APPENDIX C. Estimated Apparent Resident Survival of House Finches	141
APPENDIX D. Estimated Detection Probabilities of House Finches	143
APPENDIX E. Seasonal Age and Gender Profiles of House Finches	145
REFERENCES.....	146
CHAPTER 4.....	153
Introduction.....	154
Methods.....	160
Results	172
Discussion	185
REFERENCES.....	192

LIST OF FIGURES

<i>Figure 2.1: Effect of detection probabilities on apparent prevalence</i>	<i>39</i>
<i>Figure 2.2: Apparent versus corrected prevalence of finches in Ithaca, NY</i>	<i>50</i>
<i>Figure 2.3: Apparent versus corrected prevalence in hypothetical scenarios.....</i>	<i>52</i>
<i>Figure C2.1: Evaluation of special case of multistate modeling.....</i>	<i>75</i>
<i>Figure E2.1: State specific detection probabilities and bias in apparent prevalence ..</i>	<i>79</i>
<i>Figure 3.1: Estimated proportion of finch transients</i>	<i>112</i>
<i>Figure 3.2: Estimated site-specific finch movement probabilities.....</i>	<i>116</i>
<i>Figure 3.3: Corrected disease prevalence of finches in Ithaca, NY</i>	<i>118</i>
<i>Figure 3.4: Telemetry distribution of finch multivariate median locations.....</i>	<i>124</i>
<i>APPENDIX 3C: Estimated finch resident survival probabilities</i>	<i>141</i>
<i>APPENDIX 3D: Estimated finch detection probabilities.....</i>	<i>143</i>
<i>APPENDIX 3E: Estimated finch age and gender profiles for Ithaca, NY.....</i>	<i>145</i>
<i>Figure 4.1: Estimated finch emigration probabilities.....</i>	<i>176</i>
<i>Figure 4.2: Proportional contributions to diseased finch population growth.....</i>	<i>183</i>
<i>Figure 4.3: Proportional contributions to healthy finch population growth</i>	<i>184</i>

LIST OF TABLES

<i>APPENDIX 2D: House finch counts, encounter probabilities, and disease prevalence</i>	76
<i>Table 3.1: AIC results of multistate site analysis.....</i>	110
<i>Table 3.2: Logistic regression results of factors influencing disease prevalence.....</i>	122
<i>APPENDIX 3B: Numbers of finch captures, recaptures, and resights in Ithaca, NY.</i>	139
<i>Table 4.1: Estimable parameters from MSORD models.....</i>	167
<i>Table 4.2: AIC results from MSORD analysis.....</i>	174
<i>Table 4.3: Results of reverse time multistate analysis of symptomatic finches.....</i>	180
<i>Table 4.4: Results of reverse time multistate analysis of asymptomatic finches.....</i>	182

CHAPTER 1

SUMMARY OF FINDINGS

Modeling disease dynamics

Over the past two decades, there has been increasing interest in the ecological impacts and evolutionary consequences of wildlife diseases (for excellent reviews of wildlife disease dynamics see Rollinson and Anderson 1985, Grenfell and Dobson 1995, Hudson et al. 2002). Epidemiological models, developed as an extension from work conducted by Ross and Hudson (1917) and Kermack and McKendrick (1927), are commonly used tools to make predictions about the driving mechanisms and resulting patterns of disease transmission in populations. This approach to modeling disease dynamics generally divides a population into several compartments including susceptible individuals (*S*; available for infection), exposed individuals (*E*; a latent class that are infected, but not infectious), infectious individuals (*I*; infected and infectious), and removed individuals (*R*; a broad category including recovered individuals with permanent or temporary immunity, or dead individuals).

Such disease models are often broadly dichotomized between ‘*SIR*’ and ‘*SIS*’ models. In *SIR* models, susceptible individuals may become infected and subsequently removed from further re-infection, either by death or recovery with permanent immunity. This class of model is typically useful in providing a framework for characterizing epidemic (sudden outbreak of infectious disease in a host) patterns of disease incidence in a population (Brauer and Castillo-Chavez 2001).

In contrast, in *SIS* models, which may be more generally applicable, susceptible individuals become infected, and then (conditional on surviving) may have only temporary immunity (i.e., they are not permanently ‘removed’) before re-entering the

susceptible class. Such *SIS* models are generally useful for predicting and explaining endemic patterns of disease incidence, when some constant proportion of infected individuals is maintained in a population (Brauer and Castillo-Chavez 2001).

In both model frameworks, the dynamics of disease incidence in the population are governed by the rate at which susceptible individuals become infected (β); however, they differ in that removal rate (γ) in *SIS* models indicates the rate at which infected individuals resolve back to the susceptible class, whereas in *SIR* models it indicates the rate of either recovering and attaining permanent immunity or mortality (which are not demographically equivalent). In some cases, both primary parameters (β and γ) may be estimated with controlled experiments; for example, if the mean time an individual is infected is known (or can be estimated), then under some assumptions, you can estimate β since the mean time of infection is given approximately as $1/-\ln(\beta)$. More often, however, it is very difficult to attain estimates of these parameters, either in laboratory or field sampling situations, because of the inherent complexity and covariance of factors that interact in unpredictable ways to drive disease dynamics.

The recent application of capture-mark-recapture (CMR) models provides a theoretically and empirically grounded analytical framework with which to provide both parameter estimates for epidemiological models (e.g., survival, infection, recovery, movement), as well as the ability to test biological hypotheses regarding impacts and drivers of disease dynamics (Faustino et al. 2004). Some of the primary strengths of CMR methods include the ability to account for variable encounter probabilities of individuals when certainty of detection cannot be guaranteed (often encountered in field studies), a readily applicable information theoretic framework for evaluating competing biological hypotheses, and tools to account for model selection

uncertainty when estimation of biological effect size is of interest (for excellent reviews see Lebreton et al. 1992, Burnham and Anderson 2002, Williams et al. 2002).

While CMR methods are a useful tool for estimating demographic parameters that drive population dynamics, animal populations are spread out across a landscape, and depending upon mode of transportation, preferred habitat, and social interactions (among other things), interact with the environment at different spatial scales (for review see Peterson and Parker 1998). As finches are capable of extensive movements (as much as 1000 km; Able and Belthoff 1998) and exhibit seasonal changes in social structure (Hill 1993), there are conceivably many spatial scales (likely some distribution) over which finches are organized. Broad spatial scale analysis of finch movements (Able and Belthoff 1998), MG-induced disease prevalence (Altizer et al. 2004), and the interplay between group size and prevalence (Hochachka and Dhondt 2006) have set the stage for a more detailed examination of finch demography and *Mycoplasma gallisepticum* (MG) dynamics at a local scale. CMR approaches are ideally suited for this task, and at a local scale provide the quantitative means to begin to bridge the gap in understanding between local and broad scale dynamics of finch social structure (hence interactions) and disease dynamics. In general, my research has entailed making use of CMR methods to investigate local finch demography and movements in the context of understanding seasonal finch-MG dynamics.

Structure of the thesis

Each of the three chapters in this thesis (and to a larger degree, the additional research collaborations I've made during my dissertation) has both a methodological and biological focus.

The primary methodological issue throughout my research, which strongly conditions the biological questions addressed, concerns the differential detectability of

organisms. In most studies of wildlife populations, sampling of individuals is imperfect, resulting in encounter probabilities that are less than unity. While this is a common problem in population studies, the most robust statistical means to handle such problems are by using capture-recapture sampling, modeling, and estimation (Pollock et al. 1990, Lebreton et al. 1992, Williams et al. 2002).

Surprisingly, perhaps, most studies in wildlife disease ecology conducted before 2004 did not explicitly account for encounter probabilities in the estimation of pertinent demographic parameters (e.g., survivorship and population size) or indices of disease burden (e.g., incidence and prevalence). While it has been known for some time that neglecting the issue of detectability can induce significant bias in demographic parameter estimates (see Williams et al. 2002 for review), only recently has it been explicitly demonstrated that many of the classical diagnostics of wildlife health (e.g., prevalence) can be similarly affected (Jennelle et al. 2007). As such, the complete body of work produced here represents the most comprehensive investigation of wildlife disease dynamics to date, which has incorporated and accounted for sampling and biologically driven heterogeneity in host encounter probabilities.

Yet, despite the general advantages of CMR methods, difficulties regarding parameter estimation and the invocation of assumptions (in various circumstances) do arise and require a discussion of the caveats that must be considered. I briefly discuss several issues (relating either directly or indirectly to parameter estimation) in more detail below that required consideration during the course of my research. These included the notion of using conjunctivitis as a proxy for disease state, the potential for non-identification or misclassification of disease state, restrictions imposed in the use of multistate models, and challenges associated with unobservable states. Preceding

this treatment of caveats with respect to parameter estimation, I will highlight the major findings and broader implications of my research.

The biological focus throughout my research has generally been centered on the relationship between local spatial scale population structure, movements of House Finches, and host-pathogen dynamics. Complementing work conducted using House Finch Disease Survey data (Dhondt et al. 1998) at a broader spatial scale, my work provides a point of entry for understanding the critical linkage of scale dependent processes influencing finch-MG dynamics. The work conducted in this study is centered on a set of local scale study sites in Ithaca, NY, which have been explored as a natural microcosm of broader spatial scale dynamics of the finch-MG system. In this dissertation, I have sought to characterize the structure of this local population, and establish how both structure and host movements lead to a better overall understanding of MG infection dynamics.

The purpose of Chapter 2 (published – *Ecological Applications*; Jennelle et al. 2007) was to establish the fundamental necessity for accounting for encounter probabilities in studies of wildlife disease dynamics. As detection heterogeneity can arise from diverse sources such as sampling design, disease state, demographic stratification, and season among other things, it is of paramount importance that whenever study objectives involve estimation of demographic processes or counts, that researchers use a sampling design and appropriate statistical methods for accounting for the detection process. Given that mathematical models of disease dynamics (see Hudson et al. 2002 for examples) have been built upon count data collected without considering the detection process (in systems where variable detection is likely; red grouse (*Lagopus lagopus*), Hudson et al. 1992), it calls into question the validity of associated inferences about respective patterns and processes. To make this point clear, I provide several simple scenarios that demonstrate how

heterogeneity in encounter probabilities can lead to stark differences in patterns of disease prevalence.

The purpose of chapter 3 (In review – *Ecology*) was to apply capture-mark-recapture (CMR) methods accounting for variable detectability to more fully characterize the structure of the population within the Ithaca study area. I extend upon work conducted in Faustino et al. (2004), which was conducted to examine disease-induced survivorship effects and provide field based estimates of infection and recovery rates. Specifically, I characterize the local population (in terms of age and sex proportions), estimate site-to-site movements and the proportion of transients (a form of permanent emigration), and determine primary risk factors associated with conjunctivitis risk. In addition, I used radio telemetry data to examine the spatial distribution of daytime locations (i.e., foraging activity) as a function of release site and disease state of finches. While the telemetry analysis confirmed that birds clustered around their respective release location, asymmetric movement probabilities towards the Golf course site (associated with greater densities of coniferous trees – confirmed roost sites; Dhondt et al. 2007) suggested that not only bird feeders, but the distribution of suitable roosting sites influences the distribution of house finch aggregations.

With respect to the impact of MG infection on movements of finches, analysis of transient proportions (transient individuals are those that are observed once and never again, and represent a form of permanent emigration; Pradel et al. 1997) revealed that there were lower proportions of symptomatic finches (as opposed to asymptomatic) in seasons when a transient effect was supported by the data (typically in the autumn months, during which mass migrations occur). Within study area analysis of site-to-site movements showed that there was some evidence of reduced movement probabilities of symptomatic finches. However, I was only able to assign disease

status as a seasonal covariate (a binary response indicating if an animal was observed with conjunctivitis over the course of a given season). As disease state is a dynamics variable (and many times birds are observed to recover within a season), this dampens the disease effect on movement probabilities, and underestimates the true difference between symptomatic and asymptomatic birds.

The risk factor analysis revealed that juvenile finches are consistently more likely to express conjunctivitis compared with adults (in autumn), in some cases in spring as well. Furthermore, there was evidence that female birds are at higher risk compared with males. The former result corroborates theoretical predictions that cite pulses of juveniles as the driving force of autumn peaks in disease prevalence (Hosseini et al. 2004).

This third chapter provides partial confirmation of results from theoretical studies (Hosseini et al. 2004) suggesting a significant driver of disease dynamics in this system on a broad spatial scale, namely empirical support for the role of asymptomatic carriers as likely spreaders of MG between finch populations, and highlights the discrete structural nature of daytime finch assemblages.

Chapter 4 considered movement between two discrete locations, and permanent emigration of transient individuals. However, as finches are highly mobile animals (Able and Belthoff 1998), I needed to consider the possibility of that birds temporarily move into and out of the study area, since this form of population mixing has direct implications on disease dynamics. The purpose of chapter 4 (Jennelle et al., In prep) was to estimate both a ubiquitous type of movement (temporary emigration) and contributions to population growth of the local Ithaca, NY population (which also accounts for new recruits to the population). The Ithaca study area did not limit the spatial range over which wild House Finches in the local system could forage. As such, finches could easily undergo temporary movements out of and back into the

study area. Upon initial reaction, this sampling induced form of temporary emigration may appear to be completely irrelevant from a biological perspective in understanding the dynamics of the finch-MG system (although from a technical perspective it eliminates bias in survivorship and detection probabilities). Considering that backyard bird feeders in large part sustain wintering populations of finches in the northeastern US, and can serve as a focal activity zone for finch aggregations (Chapter 3), in fact estimates of temporary emigration in this system can be used as a proxy for the degree of mixing between host subpopulations or aggregations. This has direct implications for understanding seasonal changes in disease spread and maintenance in finch populations. It also provides a useful application of this class of capture-mark-recapture (CMR) model in other wildlife disease studies with mobile hosts.

Estimates of contributions to population growth from *in situ* disease transmission and immigration permitted me to examine the seasonal sources of infection from different population components; to my knowledge, this is the first application of ‘temporal symmetry’ models (*sensu* Nichols et al. 2000) to partition contributions to different disease states in a wild population. This set of analyses provided a context with which to evaluate the relative importance of different components of the population to growth of both the symptomatic and asymptomatic segments of the local population. Results from this set of analyses supported the notion that MG dynamics are largely driven from individuals within the local population. Taken together, the results of this chapter suggest that the spatial arrangement of bird feeders may serve to structure local aggregations of finches. Not only does this have important implications for disease dynamics in the finch-MG system (as it may be possible to manipulate spacing of aggregations across the landscape, and resulting disease transmission dynamics), but it also may be an important consideration in the spatial structuring of other bird species that rely heavily on bird feeders in the non-breeding season.

Implications of my research

Going beyond the proximate benefits that this research contributes to understanding of the finch-MG system, my hope is that my work will in part serve as a precedent for future empirical investigations of wildlife-pathogen dynamics. Capture-recapture design, estimation, and modeling explicitly accounts for variable detectability of individuals and provides a framework for making multi-model inference, thereby incorporating inherent model selection uncertainty (via Akaike information criterion) into the inferential process. The necessary linkage and interplay between theoretical and empirical investigations places a particular onus on empirical studies to produce robust and unbiased parameter estimates of interest. If empirical research is to sufficiently inform theoretical models and concepts of the biological realities of field systems, then the rigorous statistical framework embodied in capture-recapture design, estimation, and modeling should be encouraged as a standard practice in the field of wildlife epidemiology.

Furthermore, my work highlights the importance of considering different forms of animal movements (either biologically induced or as a consequence of sampling design) with respect to understanding dynamics in the finch-MG system (specifically), but also applicable to other disease systems (generally). Transient movements, completely observable within study area movements, proportional recruitment, and temporary movements outside of a study area (representing partially observable movements) are all very important elements to consider for understanding the dynamics of highly mobile animal populations (especially in the presence of a virulent pathogen).

Another important outcome of my dissertation research is in highlighting the technical challenges of robust parameter estimation in wild studies. While specific

details are addressed in each chapter, in the following I provide a brief summary of the key issues.

Limitations and future considerations

In my research on the finch-MG system, disease state was assigned by evaluating visual symptoms (conjunctivitis), and while this has been shown to be a reliable predictor of the presence of MG (Hartup et al 2001), there are several problems with using this diagnostic as a proxy for disease status. First and foremost is the fact that presence of conjunctivitis does not necessarily imply infectiousness of the afflicted individual. It is likely that there is a delay between first infection with the MG organism and onset of clinical conjunctivitis. If this delay varies by season (e.g., influenced by temperature stress), social status, age (older birds may have had previous MG challenge, recovered, and produced a temporary immunity; Sydenstricker et al. 2005), gender, or some other non-random factor, then it is possible that important periods associated with infection (and by extension the associated mechanisms) may be missed. Alternatively, birds may have residual cases of conjunctivitis, despite being free of the MG pathogen. In any case, there could be subclinical effects of MG infection, either during the incubation period or upon clearing the organism.

Two diagnostic tests for the presence of MG in finches are PCR (Polymerase Chain Reaction; which can identify whether an MG organism (living or dead) was present in blood samples) and RPA (Rapid Plate Agglutination; which can evaluate the degree of pathogen-specific antibody response). While these tests are useful and relatively efficient, they cannot establish the density of living MG organisms in tissue samples. Culture of live MG organisms would be optimal, as it would permit assessment of density (hence pathogen challenge) both between and within individuals. Despite its use in restricted situations, culture of MG (in particular) cannot

be readily and reliably carried out. Future microbiological work in this area would be very useful for both field and laboratory studies.

Under the assumption that conjunctivitis status is an appropriate proxy for assessing disease state in this system given the aforementioned considerations, then there is the issue of the intensity of infection (i.e., the severity of conjunctivitis). While conjunctivitis was recorded in field captures and resightings of birds using an ordinal index of severity, I could not account for this dynamic variable in my analyses, as there was insufficient data for symptomatic finches to do this. Realistically, this continuum of disease severity likely has some linear or nonlinear relationship to encounter probabilities, survivorship, and other demographic estimates produced. As this could not be accounted for, this effective loss of information reduced the strength of inference that could be achieved and subdivides an essentially continuous process.

All of this relates to the notion of how we actually define a disease. It poses a larger question about what stage(s) during the exposure, infection, and recovery phases an animal should be considered diseased or not? With respect to the finch-MG system, all field components have based on assessment of disease state on conjunctivitis status. While the statistical inferences used in this thesis (and those of many researchers in this system) are dependent upon this form of disease classification, the general notion of a visual clinical sign of disease raises several other important issues.

While clinical signs of infection have permitted (relatively) rapid assessment of the footprint of infection in finch populations (and may function similarly in other disease systems), it has also functioned to catalyze public concern over the population status of house finches (which evidence suggests are not threatened by regional extirpation). While readily observable pathogen infection in a gregarious and popular backyard bird certainly garners support for public awareness of avian conservation issues, it veils a

weakness pertinent to public outreach efforts and generates a question relevant to the study of wildlife diseases that the scientific community will likely have to address in the future. Many micro- and macro-parasites do not induce readily observable clinical symptoms in affected wildlife hosts (for examples see Hudson et al. 2002), which can mask their importance as potential conservation or human health threats. This weakness (effectively an observation bias from a monitoring perspective) should not only be addressed within a public policy/education perspective, but naturally leads to the question of how scientists are to effectively monitor (potentially) highly virulent and mobile wildlife diseases that do not present readily observable visual symptoms? In light of the present threat of avian influenza throughout the world, efforts should be made to design and test monitoring programs that can address this concern. While there is no omnibus solution to this looming problem, use of volunteer networks (akin to the House Finch Disease Survey; Dhondt et al. 1998) potentially provide one economically feasible framework with which to proceed.

During resighting events in the study, it was possible to identify an individual by its color-band combination, while not being able to assess disease state in one or both of its eyes (either because of a bird's spatial orientation on a feeder, or sudden flight from the feeder). In some cases this situation was rectified, as unknown state birds (a form of unobservable state) would return to feeders later during a given resighting event, permitting assessment of disease state. In situations when this was not possible, an individual could not reliably be placed into a discrete disease state category. Simulations conducted in Faustino et al (2004), which included unknown state individuals found that significant bias can occur in transition probabilities, while survival estimates remained unbiased. Exclusion of the unknowns only resulted in a loss of precision on parameter estimates, but effectively translated to a loss of potentially valuable information. By redirecting more effort to fewer field sites, and

changing the protocol of resighting events to place priority on identification of disease states of finches helped reduce the likelihood of non-identification of disease state during resighting events (generally non-identification only occurred in <5% of resighting observations).

A related problem associated with assessment of disease state during resighting events involves the notion of misclassification. While ordinal rankings of eye scores were reduced to a binary option (symptomatic or asymptomatic), relaxing the burden of disease rank misclassification, it surely does not eliminate the problem altogether. While field workers made every effort to carefully assign disease states of finches, there was certainly a non-zero probability of misclassification. Preliminary field tests showed that with increasing severity of conjunctivitis, there was increasing correspondence between observer state assignments, indicating a consistent ability to correctly classify higher conjunctivitis scores (2-moderate and 3-severe). As such, the probability of falsely assigning a symptomatic bird as asymptomatic is likely to be greater than the probability of assigning an asymptomatic bird as symptomatic (i.e., $\text{Pr}(\text{false negative}) > \text{Pr}(\text{false positive})$), and these types of errors most likely occurred with birds whose true eye score was in the range of <1 (for a given eye). Since my field test indicated greater variation in assigning scores of 0 (asymptomatic) or 1 (mildly symptomatic), observers were instructed to take a conservative approach in classifying disease state if uncertain. Thus, finches were classified as asymptomatic if there was doubt regarding state assignment. This effectively increased type II error rates, and with respect to parameter estimation, would be expected to reduce the estimated effects of infection on survivorship and precision in estimates of transition probabilities in multistate models.

Regarding the nature of parameter estimation in multistate models, there are two important assumptions that must be made that bear heavily on the resulting inferences that can be made regarding disease dynamics. First is the assumption that all state transitions are first-order Markovian. In other words, the probability of a finch making a transition between disease (or physical sites) states from time i to $i+1$ is dependent only on its state at time i . This model excludes the possibility that there are higher orders of dependence (i.e. ‘memory’, where transition between disease states is dependent on an animal’s state at not only time i , but also at time $i-1$; Hestbeck et al. 1991). As there is some evidence of partial immunity or reduced intensity following reinfection in finches challenged with MG in the laboratory (Sydenstricker et al. 2005), it is likely that higher orders of disease state dependence do occur. This may explain in part why estimates of infection probability (asymptomatic to symptomatic transitions) are much greater than recovery probability (symptomatic to asymptomatic) (Faustino et al. 2004). In any case, the relative sparseness of the dataset (specifically, the relatively low frequency of symptomatic birds) precluded a ‘memory’ analysis. These models are parameter rich, and thus extremely ‘data hungry’.

Another assumption built into the formulation of the relationship between state-specific survival and transition probabilities in the general Arnason-Schwarz multistate model (Arnason 1973, Schwarz et al. 1993) is that survival is conditional on an individual’s departure state (at time i), and transition to the arrival state (at time $i+1$) is dependent on survival in state i with the state transition occurring just prior to time $i+1$. In essence, timing of state transitions of individuals is assumed to be known (at the end of an interval in my case), and is homogeneous within stratified groups. Thus, the multistate analyses that I have conducted do not account for the possibility of different transition patterns within the interval of time i to $i+1$. While it is possible to allow random transitions within the interval using a uniform distribution (Joe and

Pollock 2002), estimator bias has been found to be generally low under the Arnason-Schwarz model. Presumably, as long as the probability of encounter with individuals in either state is random with respect to timing during the interval (although it may differ among states), state transition estimates will be qualitatively robust.

Despite the strong constraints required for robust estimation of state-specific survival and transition probabilities may be obtained, the demography of animals in host-pathogen systems can be reasonably approximated using multistate systems (minimally composed of a binary health state) and this tool is likely to see future use within this context. The notion of unobservable states, either driven by biological or sampling conditions, where a given individual is not available for observation or capture in the study area, poses particular difficulties in many types of field studies (including host-pathogen systems).

Temporary emigration can bias estimates of detection probabilities and survival. Extensions to Pollock's robust design (Pollock 1982, Kendall et al. 1997) account for this source of heterogeneity in field studies. Use of the robust design within a multistate framework increases the dimensionality of potentially estimable parameters, and the complexity of modeling and potential for biased parameters. This stems from the fact that certain constraints on parameters must be emplaced. Since the multistate framework separates survival from transition probabilities, survival of unobservable individuals must be set equal to the respective observable group (e.g., see Crespin et al. 2006). In Chapter 4, I was forced to impose this constraint in my analyses. However, I think this assumption was reasonably justified in this instance since my study area effectively represents one realization of a distribution of (for the most part) similarly available finch habitat (suburban areas with backyard bird feeders). Problems arise when considering completely and partially observable state transitions. The conditional nature of transition probabilities in multistate models imposes some degree

of covariance between state transitions. For example, in a two state system with states \mathbf{A} (observable) and \mathbf{a} (unobservable), since transitions are conditional on state at time (i), transitions from \mathbf{A} to \mathbf{A} and \mathbf{A} to \mathbf{a} must sum to 1. In my analyses, I was forced to impose constraints on transition probabilities involving transitions between partially observable and completely unobservable states in order to estimate parameters in the model set. Despite the fact that setting constraints allowed estimation of parameters of interest (temporary movements of birds within a given disease state), the nature of the constraints imposed on ‘nuisance’ transitions could bias my parameters of interest. As the use of MSORD models is very new, simulations are required on a case-by-case basis to explore the effects of constraints on the estimability and unbiasedness of parameters of interest. With respect to my research, I have made efforts to simulate conditions encountered in my data to verify the validity of inferences I make regarding MSORD models. Future research with respect to the degree of bias in parameters, given sets of constraints is necessary and will likely reveal new insights with respect to estimation under this model design.

With respect to future use of MSORD models in studies of disease dynamics, if host-pathogen systems can be approximated with standard *SIR* dynamics, then the implicit degree of determinism (permanent transition from infectious to a removed class) can reduce the dimensionality of state-specific transitions (i.e., any transitions to the susceptible from the removed class would be justifiably set to zero), leading to a better chance of unbiased estimation for transitions of interest. Yet if dynamics of a disease system are better approximated by *SIS* or *SIRI* dynamics, as in the finch-MG system, then the increased dimensionality of unrestricted state transitions will make parameter estimation increasingly difficult (especially with increasing numbers of unobservable states). However, if there is information about the distribution of the time course of disease, and records of when individuals first acquired infection, then

some degree of partial determinism could be incorporated into modeling transition parameters (thereby improving estimability of parameters).

Collaborations

Over the course of my dissertation research, I have collaborated with a number of researchers here at Cornell. The interdisciplinary nature of the finch-MG project provided me an opportunity to participate in projects with several members of the finch group. These projects were focused on experimental work conducted in aviaries. In particular, we characterized the horizontal transmission of MG infection within fixed social groups, explored the interactions of social dominance, behavior, and sex-specific immunocompetence, and provided the first confirmation of MG transmission via fomites. The net results of these collaborations have lead to several publications, with further manuscripts still in preparation. The published works include:

Dhondt, A.A., S. Altizer, E.G. Cooch, A.K. Davis, A. Dobson, M.J.L. Driscoll, B.K.

Hartup, D.M. Hawley, W.M. Hochachka, P.R. Hosseini, C.S. Jennelle, G.V.

Kollias, D.H. Ley, E.C.H. Swarthout, and K.V. Sydenstricker. 2005. Dynamics of a novel pathogen in an avian host: Mycoplasmal conjunctivitis in house finches.

Acta Tropica 94:77-93.

Dhondt, A.A., K.V. Dhondt, D.M. Hawley, and C.S. Jennelle. in press. Experimental evidence for fomite transmission of *Mycoplasma gallisepticum* in house finches.

Avian Pathology.

Hawley, D.M., C.S. Jennelle, K.V. Sydenstricker, and A.A. Dhondt. in press.

Pathogen resistance and immunocompetence covary with social status in house finches (*Carpodacus mexicanus*). *Functional Ecology*.

Sydenstricker, K.V., A.A. Dhondt, D.M. Hawley, C.S. Jennelle, H.W. Kollias, and G.V. Kollias. 2006. Characterization of experimental *Mycoplasma gallisepticum* infection in captive house finch flocks 50:39-44.

REFERENCES

- Arnason, A.N. 1973. The estimation of population size, migration rates, and survival in a stratified population. *Researches in Population Ecology* 15:1-8.
- Able, K.P., and J.R. Belthoff. 1998. Rapid 'evolution' of migratory behaviour in the introduced house finch of eastern North America. *Proceedings of the Royal Society of London B* 265:2063-2071.
- Altizer, S.M., W.M. Hochachka, and A.A. Dhondt. 2004. Seasonal dynamics of mycoplasmal conjunctivitis in eastern North American House Finches. *Journal of Animal Ecology* 73:309-322.
- Anderson, R.M., and R.M. May. 1978. Regulation and stability of host-parasite population interactions. I. Regulatory processes. *Journal of Animal Ecology* 47: 219-247.
- Brauer, F., and C. Castillo-Chavez. 2001. *Mathematical Models in Population Biology and Epidemiology*, Springer-Verlag, New York.
- Burnham, K.P., and D.R. Anderson. 2002. *Model selection and multimodel inference: A practical information-theoretic approach*. Springer-Verlag, New York.
- Crespin, L, M.P. Harris, J.D. Lebreton, M. Frederiksen, and S. Wanless. 2006. Recruitment to a seabird population depends on environmental factors and on population size. *Journal of Animal Ecology* 75:228-238.
- Dhondt, A.A., D.L. Tessaglia, and R.L. Slothower. 1998. Epidemic mycoplasmal conjunctivitis in house finches from eastern North America. *Journal of Wildlife Diseases* 34:265-280.

- Dhondt, A.A., M.J.L. Driscoll, and E.C.H. Swarthout. 2007. House finch *Carpodacus mexicanus* roosting behaviour during the non-breeding season and possible effects of mycoplasmal conjunctivitis. *Ibis* 149:1-9.
- Faustino, C.R., C.S. Jennelle, V. Connolly, A.K. Davis, E.C. Swarthout, A.A. Dhondt, and E.G. Cooch. 2004. *Mycoplasma gallisepticum* infection dynamics in a house finch population: seasonal variation in survival, encounter and transmission rate. *Journal of Animal Ecology* 73:651-669.
- Grenfell, B.T., and A.P. Dobson. 1995. Ecology of infectious diseases in natural populations. Cambridge University Press, Cambridge, UK.
- Hartup, B.K., J.M. Bickal, A.A. Dhondt, D.H. Ley, and G.V. Kollias. 2001. Dynamics of conjunctivitis and *Mycoplasma gallisepticum* infections in House Finches. *Auk* 118:327-333.
- Hestbeck, J.B., J.D. Nichols, and R.A. Malecki .1991. Estimates of movement and site fidelity using mark-resight data of wintering Canada geese. *Ecology* 72:523-533.
- Hill, G.E. 1993. House finch (*Carpodacus mexicanus*). *The Birds of North America*, no. 46 (eds A. Poole and F. Gill), pp. 1-24. Academy of Natural Sciences, Philadelphia and American Ornithologists' Union, Washington, D.C.
- Hochachka, W.M., and A.A. Dhondt. 2006. House finch (*Carpodacus mexicanus*) population- and group-level responses to a bacterial disease. *Ornithological Monographs* 60:30-43.
- Hosseini, P.R., A.A. Dhondt, and A. Dobson. 2004. Seasonality and wildlife disease: how seasonal birth, aggregation and variation in immunity affect the dynamics of *Mycoplasma gallisepticum* in house finches. *Proceedings of the Royal Society B* 271:2569-2577.

- Hudson, P. J., D. Newborn, and A. P. Dobson. 1992. Regulation and stability of a free-living host–parasite system, *Trichostrongylus tenuis* in red grouse. I. Monitoring and parasite reduction experiments. *Journal of Animal Ecology* 61: 477–486.
- Hudson, P. J., A. P. Rizzoli, B. T. Grenfell, J. A. P. Heesterbeek, and A. P. Dobson. 2002. *The ecology of wildlife diseases*. Oxford University Press, New York, New York, USA.
- Jennelle, C.S., E.G. Cooch, M.J. Conroy, and J.C. Senar. 2007. State-specific detection probabilities and disease prevalence. *Ecological Applications* 17: 154-167.
- Joe, M., and K.H. Pollock. 2002. Separation of survival and movement rates in multi-state tag-return and capture-recapture models. *Journal of Applied Statistics* 29:373-384.
- Kendall, W.L., J.D. Nichols, and J.E. Hines. 1997. Estimating temporary emigration and breeding proportions using capture-recapture data with Pollock’s robust design. *Ecology* 78:563-578.
- Kermack, W.O., and A.G. McKendrick. 1927. Contributions to the mathematical theory of epidemics. 1. *Proceedings of the Royal Society of Edinburgh*. A115:700-721. (reprinted in *Bulletin of Mathematical Biology* 53:33-55).
- Lebreton, J.-D., K.P. Burnham, J. Clobert, and D.R. Anderson. 1992. Modeling survival and testing biological hypothesis using marked animals: A unified approach with case studies. *Ecological Monographs* 66:67-118.
- May, R.M., and R.M. Anderson. 1978. Regulation and stability of host-parasite population interactions: Destabilising processes. II. *Journal of Animal Ecology* 47: 249-267.

- May, R.M. 1983. Parasitic infections as regulators of animal populations. *American Scientist* 71: 36-45.
- Nichols, J.D., J.E. Hines, J-D. Lebreton, and R. Pradel. 2000. Estimation of contributions to population growth: A reverse time capture-recapture approach. *Ecology* 81:3362-3376.
- Peterson, D.L., and V.T. Parker. 1998. *Ecological Scale: Theory and applications*. Columbia University Press, New York, USA.
- Pollock K.H. 1982. A capture-recapture design robust to unequal probability of capture. *Journal of Wildlife Management* 46:752-757.
- Pollock, K.H., J.D. Nichols, C. Brownie, and J.E. Hines. 1990. Statistical inference for capture-recapture experiments. *Wildlife Monographs* 107:1-97.
- Pradel, R., J.E. Hines, J.-D. Lebreton, and J.D. Nichols. 1997. Capture-recapture survival models taking account of transients. *Biometrics* 53:60-72.
- Rollinson, D., and R.M. Anderson (editors). 1985. *Ecology and Genetics of Host-parasite Interactions*. Academic Press, London.
- Ross, R., and H. P. Hudson. 1917. An application of the theory of probabilities to the study of a priori pathometry. Parts II and III. *Proceedings of the Royal Society of London A* 93:212-240.
- Schwarz C.J., J.F. Schweigert, and A.N. Arnason 1993. Estimating migration rates using tag-recovery data. *Biometrics* 49:177-193.
- Sydenstricker, K.V., A.A. Dhondt, D.H. Ley, and G.V. Kollias. 2005. Re-exposure of captive house finches that recovered from *Mycoplasma gallisepticum* infection. *Journal of Wildlife Diseases* 41:326-333.

Williams, B.K., J.D. Nichols, and M.J. Conroy. 2002. *Analysis and Management of Animal Populations*. Academic Press, San Diego.

CHAPTER 2
STATE-SPECIFIC DETECTION PROBABILITIES AND DISEASE
PREVALENCE*

Abstract. Investigations of disease dynamics in wild populations often use estimated prevalence or incidence as a measure of true disease frequency. Such indices, almost always based solely on raw counts of diseased and healthy individuals, are often the basis for analysis of temporal and spatial dynamics of diseases in wild populations. Generally, such studies do not account for potential differences in observer detection probabilities of host individuals stratified by biotic and/or abiotic factors. I demonstrate the potential effects of heterogeneity in state-specific detection probabilities on estimated disease prevalence using mark-recapture data from previous work in a house finch (*Carpodacus mexicanus*) - *Mycoplasma gallisepticum* system. In this system, detection probabilities of uninfected finches were generally higher than infected individuals. I show that the magnitude and seasonal pattern of variation in estimated prevalence corrected for differences in detection probabilities differed markedly from uncorrected (apparent) prevalence. When the detection probability of uninfected individuals is higher than infected individuals (as in this study), apparent prevalence is negatively biased and vice versa. In situations where state-specific detection probabilities strongly interact over time, I show that the magnitude and pattern of apparent prevalence can change dramatically; in such cases, observed variations in prevalence may be completely spurious artifacts of variation in detection probability, rather than changes in underlying disease dynamics. Accounting for differential detection probabilities in estimates of disease frequency removes a potentially confounding factor in studies seeking to identify biotic and/or abiotic

* Chapter 2 published in *Ecological Applications* 17:154-167.

drivers of disease dynamics. Given that detection probabilities of different groups of individuals are likely to change temporally and spatially in most field studies, these results underscore the importance of estimating and incorporating detection probabilities in estimated disease prevalence (specifically), and more generally, any ecological index used to estimate some parameter of interest. While a mark-recapture approach makes it possible to estimate detection probabilities, it is not always practical, especially at large scales. I discuss several alternative approaches, and categorize the assumptions under which analysis of uncorrected prevalence may be acceptable.

Key-words: *Carpodacus mexicanus*; disease; ecological index; detection probability; mark-recapture; *Mycoplasma gallisepticum*; prevalence.

Introduction

Of significant importance to disease ecologists and epidemiologists in wildlife disease studies are the factors or mechanisms that drive disease dynamics in a host-pathogen system. In most cases, field data are collected to produce a diagnostic measure of disease burden in the sampled population. It is often prohibitive both financially and logistically to enumerate and maintain records of every diseased and susceptible case in a population, thus complete census data are rarely obtainable in human or wildlife disease studies. As such, disease ecologists and epidemiologists often must rely on incomplete counts of individuals or indices for estimation of prevalence and/or incidence. *Prevalence* is usually defined as the proportion of all individuals in a target population that are infected at some time period, whereas *incidence* is the proportion of susceptible individuals in a target population that are infected for the first time (Mausner and Bahn 1974). In many cases, incidence is usually estimated as a rate over

some user-defined period of time. Collection of basic disease frequency data is not only useful for monitoring the health of animal populations and evaluating the effects of disease control efforts (Wobeser 2002), but also for making inferences about the possible drivers of disease dynamics in wild populations (Altizer et al. 2004a, Joly and Messier 2004, Atkinson et al. 2005, Loot et al. 2005, Salkeld and Schwartzkopf 2005). Given the limited budget of most wildlife disease studies, collection and analysis of disease frequency data is at the least a useful starting point for studying disease dynamics in wild animal populations.

The use of indices is widespread in the field of ecology, employed to represent a diverse array of biological information such as abundance (avian point counts), community structure (species diversity), and ecological integrity (various bioindicators). In general an index is considered to be a value, which relates linearly or non-linearly, to a parameter of interest. A number of papers have discussed the risks associated with using indices (Anderson 2001, MacKenzie and Kendall 2002, Anderson 2003), so I will only highlight the major points presented in these works. The critical assumption of an index is that variation in its value (e.g., raw count) represents true variation in the value of the parameter of interest (e.g., population abundance). In order for this assumption to be met, detection probabilities across time, space, observer, and species (if multiple species are being counted) must be equal and invariant across all factors. Here I define detection (encounter) probability as the probability of the observer detecting (by some sampling method) an individual of a species or group at time t , conditional on the individual being in the sampling area during time t .

The Importance of Estimating Detection Probabilities in Wildlife Disease Studies

Capture-mark-recapture (CMR) methods (Otis et al. 1978, Pollock et al. 1990, Lebreton et al. 1992, Williams et al. 2002) provide a general framework for estimating detection probabilities in field studies. The theory and application of these methods is very well developed and robust under many field situations, allowing not only estimation of pertinent demographic parameters of interest, but also the incorporation of model selection uncertainty in resulting estimates. When complete detectability is not possible in a study, CMR methods are ideal. Maximum likelihood estimates of parameters, such as detection probability, are generated based on comparison of observed individual encounter histories and the underlying expected probabilistic structure imposed by *a priori* models conceived by the investigator.

In the field of human epidemiology, it has been recognized that estimation of prevalence and/or incidence using incomplete counts alone results in biased estimates (McCarty et al. 1993, also see International Working Group for Disease Monitoring and Forecasting 1995*a,b* for review). Borrowing from the fields of ecology and biostatistics, human epidemiologists ‘discovered’ the usefulness of CMR methods, which provide a tool for estimating the unobserved disease cases in studies using incomplete counts of individuals (International Working Group for Disease Monitoring and Forecasting 1995*a,b*). Although the field of wildlife biology has an extensive history of CMR usage with strong theoretical and empirical underpinnings, the empirical study of wildlife diseases is a relatively newer avenue of investigation in this field. In this burgeoning area of interest, a standard method for estimating disease prevalence has been to establish a sampling framework around a target population and report proportions of infected individuals from that sample under the assumption that detection probabilities are invariant temporally, spatially, and between relevant health-

states of individuals (e.g., Dobson and Meagher 1996, Delahay et al. 2000, Van Riper III et al. 2002, Fallon et al. 2003, Joly et al. 2003).

It has long been recognized that the detection probability of organisms can vary as a function of numerous biotic and abiotic factors. For instance, vulnerability of waterfowl to harvest can be influenced by disease state (e.g., effect of lead poisoning in waterfowl; Bellrose 1959) or body condition (Hepp et al. 1986) and evidence suggests there can be a condition bias in trapped samples of birds as well (Weatherhead and Greenwood 1981), but it is likely difficult to ensure a purely representative sample of a given population (Burnham and Nichols 1985; but see Weatherhead and Ankney 1985). Likewise, it has been well established and documented in studies using marked individuals that animal detection probabilities can vary as a function of other factors besides disease state and condition such as time, space, age, gender, group size, environmental covariates, capture method, observers (e.g., in point counts), effort, and other types of stratification (Samuel and Pollock 1981, Lebreton et al. 1992, Domenech and Senar 1997, Domenech and Senar 1998, Tuyttens et al. 1999, Senar et al. 1999, Nichols et al. 2000, Tracey et al. 2005). Despite recognition by population ecologists of potential sources of heterogeneity in animal detection probabilities and subsequent efforts to correct population estimates for various sources of bias, in studies of wildlife disease dynamics to date only two that I am aware of have acknowledged the potential for bias in estimates of disease prevalence due to detectability issues (Tuyttens et al. 1999, Senar and Conroy 2004) and only one (Senar and Conroy 2004) has incorporated detection heterogeneity into estimates of disease frequency. Another study evaluated competing models using AIC to evaluate potential bias in harvest-based prevalence estimates of chronic wasting disease in mule deer (*Odocoileus hemionus*) (Conner et al. 2000); however, their approach did not explicitly account for potential differences in detection probabilities

between groups of animals stratified by health state, age, gender, or other possible sources of heterogeneity.

Failure to account for state-specific differences in observer detection probabilities can potentially result in reported patterns of disease frequency (e.g., prevalence), which are entirely an artifact of host encounter dynamics (which may be a function of variability in the sampling process, state-induced host behavioral changes, environmental conditions, or demographic stochasticity). It is often assumed that a stationary (unchanging) pattern of variation in prevalence (either temporally or spatially) is consistent with an underlying dynamical driver(s) (e.g., seasonal or latitudinal temperature changes); however, such variation might also reflect stationary patterns in observer detection probabilities for individuals in either disease state, and have little to do with underlying disease dynamics. For example, consider a situation where the true prevalence of a disease is constant over time, but where the probability of detecting (say) a diseased individual varies seasonally. Such seasonal variation in detection is entirely plausible in many situations, since disease state may induce a behavioral response changing the sampling probability of animals in a given state. In such a case, there would be seasonal variation in apparent prevalence driven by seasonal variation in state-specific detection probabilities, which would suggest a seasonal cyclic pattern in prevalence when in fact the true prevalence was constant over time (Fig. 2.1). This of course leads to the critical question of whether or not systematic variation in apparent prevalence reflects true variation in the proportions of individuals in each disease state, or variation in the probability of detecting individuals in each disease state. Assuming that a stationary pattern of variation in apparent prevalence reflects the underlying mechanism(s) driving observed disease dynamics without considering the host encounter process is expedient, but is a clear example of inferring process from pattern. In this case, such an inference would be biased by the

fact that the pattern (stationary or not) could reflect heterogeneity in sampling, and have little to do with variation in the disease dynamics at all.

Because disease frequency data are collected and often used for the basis of inferences in wildlife disease studies, in this paper I assess the importance of incorporating heterogeneous detection probabilities in estimates of disease prevalence (and easily extended to incidence) and provide a simple calculation to facilitate estimation. I demonstrate the potential consequences of heterogeneity in detection probabilities using data from an intensive study of a local house finch (*Carpodacus mexicanus*) population exposed to the pathogen (*Mycoplasma gallisepticum*). I use this model disease system along with simulations to show that potentially misleading inferences about host-pathogen dynamics can be made when estimating prevalence without accounting for potential differences in detection probabilities among disease states (although other sources of detection heterogeneity can also induce bias in estimates of disease frequency and can easily be accommodated with this approach). In some cases the observed pattern of variation in prevalence (based on simple count data of relative numbers of diseased and healthy individuals) can potentially be a strongly biased estimate of variation in true prevalence. I also evaluate a special case of a recently described approach for estimating prevalence (Senar and Conroy 2004), which is suitable for studies conducted under a multistate CMR modeling framework. Although the examples I consider concern avian diseases, the underlying ideas are relevant to any taxa under study. I conclude by presenting recommendations for estimation of detection probabilities for small- and large-scale studies when standard CMR methods are not used.

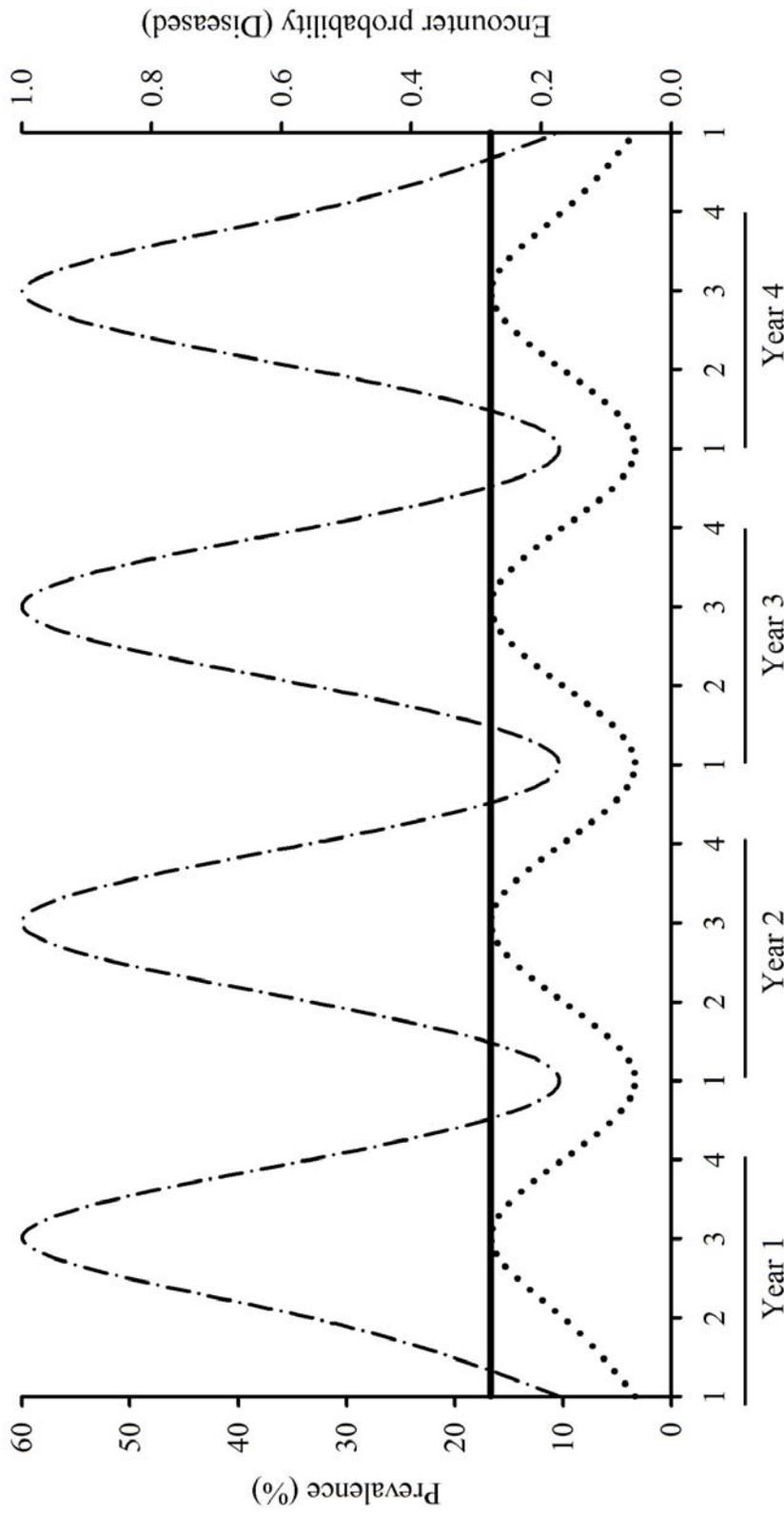


Figure 2.1. Illustration showing how cyclic patterns of apparent (observed) prevalence (···) may be an artifact of cyclic patterns in detection probabilities (—·—) of one or more groups of animals, which can result in misleading inferences about the pattern of corrected (true) prevalence (—). In this case only the detection probability of diseased animals varies temporally, while detection probability of healthy (with respect to the condition under study) animals is time invariant (=1.0).

Methods

Subset of Data Used

Data were collected as part of a larger effort to study the disease dynamics of *Mycoplasma gallisepticum* infection in eastern house finches (hereafter finches), which were introduced to the eastern US around 1940 (Hill 1993). General field methods are described in Faustino et al. (2004). Trapping and resighting data were collected from encounters with individual finches from August to April of 2001 to 2005 in Ithaca, NY (located at approximately 42.5°N 76.5°E). Each newly captured bird was fitted, under permit, with a 9-digit numbered aluminum leg band (Bird Banding Laboratory, Laurel, Maryland, USA) and a combination of three colored plastic leg bands. Individual birds were scored for infection status at each encounter, using a binary ranking: ‘I’ (infected) indicating some level of the disease, or ‘U’ (uninfected) indicating that conjunctivitis was not observed. I stress that for the purposes of this paper, I explicitly define an MG-infected individual as expressing observable symptoms of infection, namely conjunctivitis. I only used data from 2002-03 to demonstrate the methodology.

Estimation of Detection Probabilities and Corrected Prevalence

I contrast estimates of MG prevalence obtained using the standard approach (termed *apparent prevalence* following Senar and Conroy 2004) with estimates accounting for differences in detection probabilities as a function of disease state and calculate the associated percent relative bias (%RB) of apparent prevalence as

$$\%RB = \frac{(\hat{\delta}_i^A - \hat{\delta}_i^C) \times 100}{\hat{\delta}_i^C}$$

where,

$\hat{\delta}_i^A$ = estimated apparent prevalence at time i ,

$\hat{\delta}_i^C$ = estimated corrected prevalence at time i .

Apparent prevalence was estimated as the sum of unique infected finches divided by the total sum of unique finches resighted in a given week. Although I obtained capture data from both live capture (via mist nets and cage traps) and resighting events, I only used information from resightings to eliminate the possible confounding effects of capture heterogeneity due to trap type (*sensu* Domenech and Senar 1997,1998, Davis 2005). To correct estimates of weekly apparent prevalence, I used estimates of weekly detection probabilities of infected and uninfected finches generated from an intensive mark-recapture study in Ithaca, NY (Faustino et al. 2004). Detection probabilities were estimated using multistate mark-recapture models (Williams et al. 2002, and references therein) in program MARK (White and Burnham 1999).

Multistate models are an extension of the classical Cormack-Jolly-Seber (CJS) live mark-encounter, open-population models that allow individuals in the population to be distributed across multiple sites or among multiple ‘states’. Such models allow for robust estimation of transition probabilities (i.e., survival, movement among states) under conditions where the probability of observing an individual on a particular sampling occasion is <1 . Under the assumption that survival from time i to $i+1$ depends only on the state (stratum) at time i , then separate estimation of survival from transition probabilities 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 $i+1$,

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 time $i+1$,

and

$$\phi_i^{rs} = S_i^r \psi_i^{rs}$$

where,

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

In the context of wildlife diseases, state refers to alternative disease states (i.e., I = infected and U = uninfected), and transition among disease states corresponds to probabilities of infection (transition from U to I ; ψ_i^{UI}) and recovery (transition from I to U ; ψ_i^{IU}).

There are several important assumptions regarding multistate models to consider within the context of a study on wildlife diseases aside from other standard CJS model assumptions (Williams et al. 2002, and references therein). First, standard methods for multistate analysis assume that all transitions are first-order Markovian. In other words, they assume that the probability of an animal 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 the models). The statistical interpretation of a transition probability under this assumption is that an animal must survive from time i to $i+1$ in state x before it can make a transition to state y immediately before time $i+1$. In reality an animal can make a state transition at any time between time i and $i+1$, and care must be taken to ensure that the time span between sampling periods is at most the average duration of time it is expected for an animal to make a transition from a diseased to healthy state. It is not clear how variation in time spans between sampling periods might induce bias in estimated transition probabilities. In the context of a disease study, it is possible that the probability of an animal surviving and making a transition between disease states is dependent on its state at not only time i , but also at times $i-1$,

i-2, and so on. In most cases, however, I expect that there will not be sufficient data to model state transitions as a higher-order Markov process ('memory models', *sensu* Hestbeck et al. 1991); such models are parameter rich, and thus extremely 'data hungry'. Since it is possible that some diseases might impose acute mortality in hosts, this could be tested with covariate models, random effect models, or by parameterizing the model with a transience structure (Pradel et al. 1997). A standard assumption of CJS models is that emigration (which can be considered an unobservable state) is permanent, causing it to be confounded with true survival probability. In many cases, it is possible that animals may move in and out of the study area over the course of sampling and to accommodate this, Kendall et al. (1997) have developed temporary emigration models that make use of Pollock's robust design (1982) with extensions that incorporate a multistate framework (Bailey et al. 2004, Schaub et al. 2004). Furthermore, multistate modeling assumes that state can be assigned with complete certainty upon encounter with an individual. The potential for misspecification of disease state, expressed as uncertainty in assigning the correct disease state to an individual is certainly a reality that should be considered in any study of diseases. The degree of misclassification will likely induce proportional bias in state transition probabilities, which can result in researchers making incorrect inferences about estimates of the force of infection and recovery probabilities in a disease system. In some cases it is possible to correct for misclassification bias in transition probabilities using a modification of multistate models that incorporates the robust design both in cases when state can change stochastically (Kendall et al. 2003) and when state is deterministic (Nichols et al. 2004). A key assumption to consider when using CJS models is that individuals are independent of each other with respect to survival and detection probability (Pollock et al. 1990). In many biological systems, there is likely to be dependence between individuals (e.g., family groups), which at the

least will induce overdispersion in variance of some parameters. All in all, researchers must carefully consider the underlying assumptions of multistate models with respect to how violation may induce bias in resulting parameter estimates and influence the inferences that can be made.

For my analyses I sought relative estimates of N for infected and uninfected finches as in relation to the expression $E(C) = p N$, where $E(C)$ is the expected value of a count statistic C , p is the probability of encountering an individual in a given time period, and N is the population size (Conroy 1996, MacKenzie and Kendall 2002, Williams et al. 2002). For the purpose of estimating prevalence in the study area, the specification of the population I sampled is not important as long as the sampled finches adequately represent the numbers of infected and uninfected birds in the biological population.

Evidence from Faustino et al. (2004) indicated in some cases marked differences in detection probabilities between infected and uninfected finches (from 0 to 80%) that varied over time. These estimates of state- and time-specific detection probabilities obtained from Faustino et al. (2004) were incorporated into corrected prevalence estimates. Given that observations of finches at my study sites represent an incomplete count of finches, if I account for differential detection probabilities between infected and uninfected finches, the true finch count for a given disease state at a given time can be expressed as

$$E(C_i^s) = \hat{p}_i^s \hat{N}_i^s \tag{1}$$

where,

C_i^s = observed count of finches in health state s (infected or uninfected) at time i ,

\hat{p}_i^s = the estimated detection probability of a finch in health state s at time i ,

\hat{N}_i^s = the estimated population size of finches in health state s at time i .

This expression can be rearranged to estimate \hat{N}_i^s as

$$\hat{N}_i^s = \frac{C_i^s}{\hat{p}_i^s}. \quad (2)$$

Since prevalence is defined as the proportion of infected individuals in a population, I can derive an expression that corrects estimates of apparent prevalence to account for differences in detection probability between disease states. If the only source of heterogeneity in detection probability is disease state, then

$$\hat{\delta}_i^R = \frac{\hat{N}_i^I}{\hat{N}_i^I + \hat{N}_i^U} = \frac{\frac{C_i^I}{\hat{p}_i^I}}{\frac{C_i^I}{\hat{p}_i^I} + \frac{C_i^U}{\hat{p}_i^U}} = \frac{C_i^I \hat{p}_i^U}{C_i^I \hat{p}_i^U + C_i^U \hat{p}_i^I} \quad (3)$$

where,

$\hat{\delta}_i^R$ = corrected disease prevalence at time i for reduced state space (disease-state only),

C_i^s = observed count of focal species in health state s (I : infected or U : uninfected) at time i ,

\hat{p}_i^s = the estimated detection probability of the focal species in health state s at time i .

The development of the expression for corrected prevalence is analogous to that presented for estimation of breeding proportions as presented in Nichols et al. (1994). This expression can be generalized to account for other sources of heterogeneity in detection probabilities that may be orthogonal to disease state (e.g., age, gender;

Appendix A). In this paper, I use equation (3), and assume the primary source of heterogeneity in detection probability is disease state.

An approximation for the conditional variance of corrected prevalence $\hat{\delta}_{ijk}^R$ is given as

$$\text{var}(\hat{\delta}_{ijk}^R | C_{ijk}^U, C_{ijk}^I) = \frac{C_{ijk}^I{}^2 C_{ijk}^U{}^2 \left(\hat{p}_{ijk}^U{}^2 \text{var}(\hat{p}_{ijk}^I) + \hat{p}_{ijk}^I{}^2 \text{var}(\hat{p}_{ijk}^U) - 2\hat{p}_{ijk}^U \hat{p}_{ijk}^I \text{cov}(\hat{p}_{ijk}^U, \hat{p}_{ijk}^I) \right)}{\left(C_{ijk}^U \hat{p}_{ijk}^I + C_{ijk}^I \hat{p}_{ijk}^U \right)^4}$$

(see Appendix B) where,

C_{ijk}^s = observed count of finches in health state s (I : infected or

U : uninfected) in year i , month j , and week k ,

\hat{p}_{ijk}^s = the estimated detection probability for a finch in health state s (I :

infected or U : uninfected) in year i , month j , and week k .

Note that the estimated variance and covariance for state-specific detection probabilities can be obtained directly from programs MARK (White and Burnham 1999) or MSSURVIV (Hines 1994).

Bias Evaluation of Simulated Detection Process

In addition to correcting estimates of prevalence for a subset of the empirical data (2002-03 field season), I present three hypothetical scenarios using the same finch count data. In place of estimated detection probabilities from Faustino et al. (2004), I assign hypothetical detection probabilities for infected and uninfected individuals that interact over time and are additive with time. In each scenario, I evaluate the bias of apparent prevalence, which assumes that state-specific detection probabilities are equal. The first scenario represents a situation where detection probabilities of infected and uninfected individuals interact over time by assigning uninfected individuals a

higher detection probability than infected individuals during the first half of the study period and inverting this trend for the latter half of the study period. To show how the magnitude of bias changes with increasing differences in detection probabilities between health states, I provide a gradient of corrected prevalence functions based on the following pairs of detection probabilities ($p_i^U = 0.55$, $p_i^I = 0.45$; $p_i^U = 0.65$, $p_i^I = 0.35$; $p_i^U = 0.75$, $p_i^I = 0.25$). If the study period spanned an autumn and winter season, respectively, then I could hypothesize that infected individuals have a lower detection probability in the autumn because conjunctivitis (in the case of finches) impairs their vision, and subsequently reduces mobility. Uninfected finches are not constrained by the limitations of conjunctivitis (reduced vision and lethargy), permitting them to exploit more feeding sites (backyard feeders) during this season. In winter, the encounter relationship could switch where the detection probability of infected finches is higher in winter due to the increased energetic demands of this period causing these handicapped birds to rely more heavily on stable (and stationary) food resources, whereas uninfected individuals would still be able to exploit widespread natural resources and bird feeders. Given that mobility of infected finches remains low over both seasons, it is plausible for infected finches to have a lower detection probability during autumn as these birds may have access to a readily available natural food source that is more easily accessible than food that is provided at feeder stations. As natural sources of food are depleted or rendered inaccessible by precipitation, detection probabilities (at feeders) of infected finches can increase in winter if these individuals find bird feeders despite their overall reduced mobility. The importance of these values is reflected by the relative difference in detection probabilities between health states, rather than the actual values of the detection probabilities used.

In a different scenario, I present a situation in which detection probabilities of infected and uninfected individuals are additive with time and show that the resultant direction of bias in prevalence is a function of whether p_i^I is greater or less than p_i^U . I hypothetically assign infected individuals a higher detection probability than uninfected individuals and again provide a gradient of corrected prevalence functions based on the following pairs of detection probabilities ($p_i^U = 0.45$, $p_i^I = 0.55$; $p_i^U = 0.35$, $p_i^I = 0.65$; $p_i^U = 0.25$, $p_i^I = 0.75$). I could hypothesize that infected individuals have a consistently higher detection probability because they rely heavily on easily obtained food at baited feeding stations throughout the study period (a scenario supported by Senar and Conroy (2004)). For completeness, I also provide the reverse scenario with consistently higher detection probabilities for uninfected compared to infected individuals.

In addition to presenting a corrected estimator for disease prevalence, I evaluated the bias of an approach to prevalence estimation given in Senar and Conroy (2004) under conditions when only time invariant infection probabilities are available (Appendix C). The general framework for the approach in Senar and Conroy (2004) makes use of state-specific survival and transition parameters estimated using CMR multistate models, and offers considerable flexibility in practical usage.

Results

From November 2002 through mid-March 2003, weekly counts of marked finches ranged from 13 to 193 birds, while apparent prevalence ranged from 4.4% to 26.7% (Appendix D). Weekly estimates of detection probability for infected and uninfected finches were obtained from Faustino et al. (2004) (Appendix D). -

Fig. 2.2 compares the pattern of apparent prevalence observed from actual counts of infected and uninfected finches versus prevalence corrected for health state specific detection probabilities. Since detection probabilities for uninfected finches were generally higher than those for infected birds (Appendix D), the estimator for apparent prevalence was negatively biased (Fig. 2.2). Conjunctivitis in finches due to MG infection affects visual acuity, which likely makes it difficult for infected finches to find bird feeders. Since feeder stations were used to attract birds during capture and resighting events, difficulty in finding feeders might be responsible for lower estimated detection probabilities of infected finches. The degree of bias varied over the course of the seasons with average %RB equal to -25%. The magnitude of %RB was greatest during the week of Dec 14 (-47%) and in early February and March (maximum -61%) (Appendix D). In general, the trend in bias increased as the difference between estimates of state-specific detection probabilities increased. The overall pattern in prevalence did not change appreciably, except for a spike in corrected prevalence in mid-February and early March 2003 (Fig. 2.2).

In Fig. 2.3, I considered a hypothetical example using the same finch count data that were used to generate Fig. 2.2. In Fig. 2.3A, I show how bias in apparent prevalence changes with three series of corrected prevalence values. In each series, the difference in detection probabilities that interact over time between uninfected and infected finches increased. Under this scenario, both the pattern and magnitude of corrected prevalence changed directly with the difference in state-specific detection probabilities (Fig. 2.3A). Thus, increasing differences in state-specific detection probabilities lead to increased bias in apparent prevalence values.

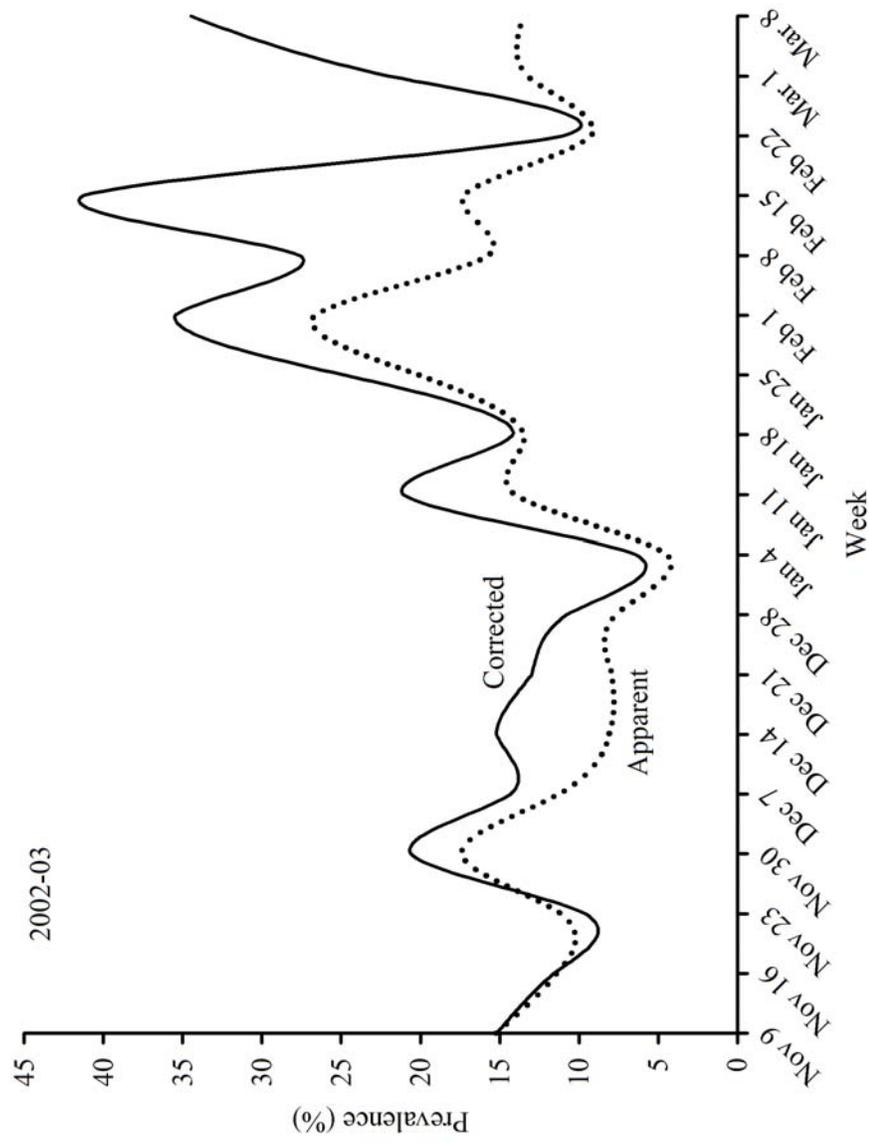
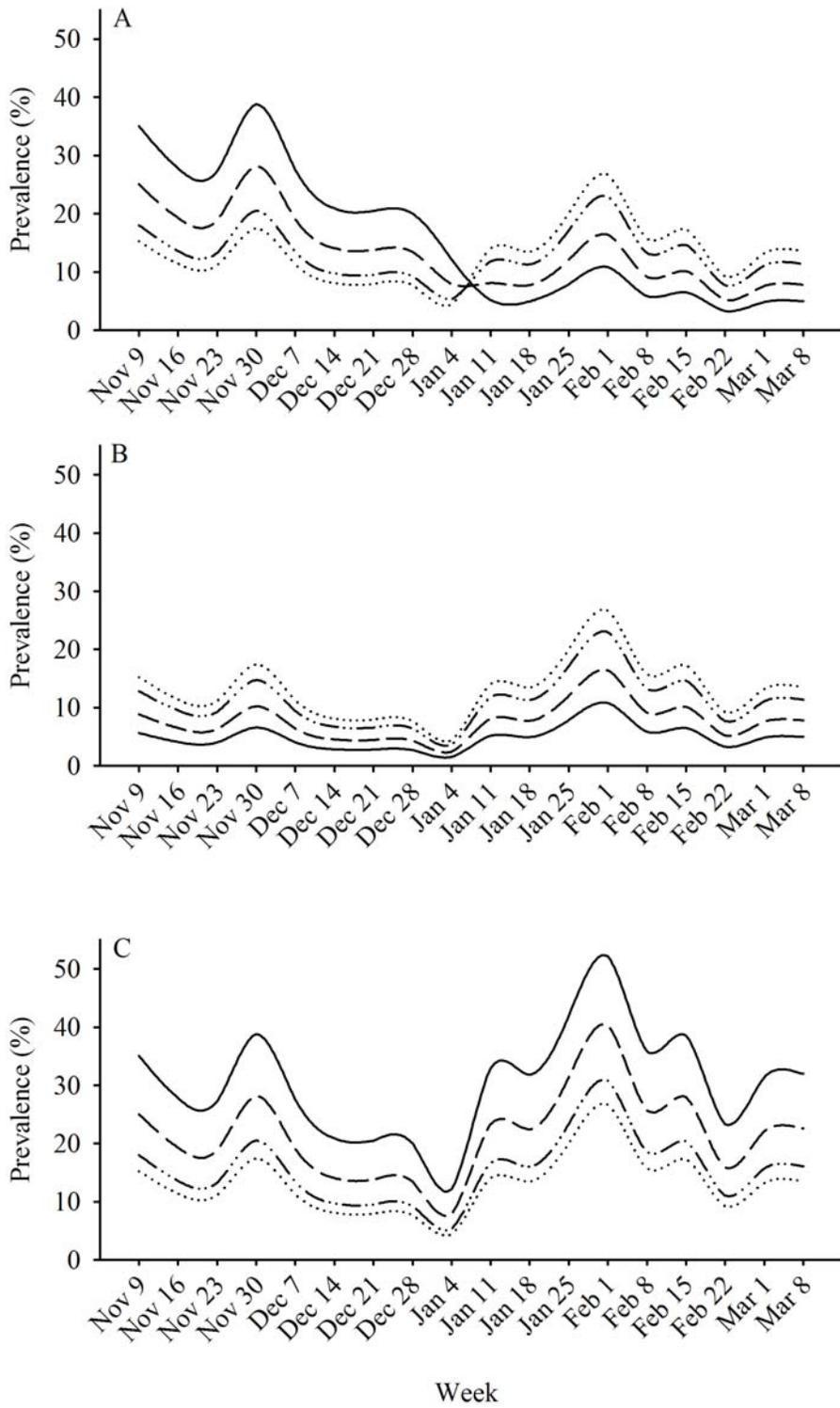


Figure 2.2. Comparison of apparent prevalence (dotted line), based on observed counts of finches, to corrected prevalence (solid line) incorporating differential detection probabilities (from Faustino et al. 2004) between infected and uninfected house finches for data collected between the weeks of 9 November 2002 and 8 March 2003; data were collected in Ithaca, NY, USA.

Figure 2.3. Comparison of apparent prevalence (\cdots) based on observed counts of finches to corrected prevalence incorporating differential detection probabilities between infected and uninfected house finches under hypothetical scenarios of differential detectability. Corrected prevalence is shown for (A) an interaction over time, where $p_i^I = 0.45$ and $p_i^U = 0.55$ ($-\cdots-$); $p_i^I = 0.35$ and $p_i^U = 0.65$ ($---$); $p_i^I = 0.25$ and $p_i^U = 0.75$ ($---$) for the first half of the study period, with this relationship reversed for the second half, (B) an additive effect over time, where $p_i^I = 0.55$ and $p_i^U = 0.45$ ($-\cdots-$); $p_i^I = 0.65$ and $p_i^U = 0.35$ ($---$); $p_i^I = 0.75$ and $p_i^U = 0.25$ ($---$), and (C) an additive effect over time, where $p_i^I = 0.45$ and $p_i^U = 0.55$ ($-\cdots-$); $p_i^I = 0.35$ and $p_i^U = 0.65$ ($---$); $p_i^I = 0.25$ and $p_i^U = 0.75$ ($---$) (p_i^s is detection probability of animals at time i in state s , where I = infected, U = uninfected).



In Figs. 2.3B and 2.3C, I considered alternative hypothetical scenarios, again using the same finch count data that was used to generate Fig. 2.3A. In these scenarios I again plotted three series of corrected prevalence values, whose state-specific detection probabilities were additive with time, such that $p_i^I > p_i^U$ in Fig. 2.3B and $p_i^I < p_i^U$ in Fig. 2.3C. When the detection probability of infected individuals was greater than uninfected individuals, I found that the observed pattern of prevalence was positively biased (Fig. 2.3B). The bias increased as the difference in state-specific detection probabilities increased. Alternatively, when detection probabilities of infected individuals were lower than uninfected individuals, the observed pattern of prevalence was negatively biased (Fig. 2.3C), again depending on the magnitude of difference between state-specific detection probabilities.

Discussion

Valid Use of Indices

Indices are viewed as simple and cost efficient in studies where large-scale surveillance or monitoring is an objective. The results show that the trend and magnitude of disease prevalence estimates based on raw counts of individuals that are uncorrected for state-specific detection probabilities can be biased (Fig. 2.3), leading investigators to make potentially incorrect inferences about disease dynamics in wildlife populations. It is clear that using uncorrected prevalence data can lead investigators to report (i) exaggerated seasonal peaks in prevalence (Fig. 2.3A), (ii) inflated levels of disease prevalence (Fig. 2.3B), or (iii) gross underestimates of disease prevalence (Fig. 2.3C).

If an index (such as disease prevalence) is used for making ecological inferences, I espouse the view put forth by MacKenzie and Kendall (2002) that at the least

detection probabilities should be assumed different, and the burden of proof should be placed on determining equality. Environmental fluctuations, observer or sampling differences, and the impact of a disease on the behavior of animals are intuitively going to exacerbate differences in detection probabilities. Given that wildlife diseases can devastate fragile populations and in some cases be transmitted to humans, it is reasonable to take this conservative approach. The application of bioequivalence testing as outlined in MacKenzie and Kendall (2002) should be strongly considered by investigators considering the use of indices. Inferences made using indices are predicated on the validity of the underlying assumptions implicit in their constituent elements. Every effort should be made to test these assumptions with the expectation that detection probabilities will have to be estimated and incorporated explicitly into calculation of an index.

I acknowledge the difficulty inherent in some studies to produce sufficient sample sizes of animals in different disease states, as well as finer levels of stratification. Disease systems which require blood or tissue samples of individuals to assess health status will clearly require more funding and logistical support to undertake. Some organisms are very difficult to capture in practice, and/or may be distributed throughout a landscape at such low densities that resulting captured sample sizes are small despite a great amount of effort. Under these circumstances, I cannot expect researchers to be able to correct estimates of disease frequency for differential detection probabilities. If an unadjusted index is used as the basis for inference, I urge investigators to take caution when interpreting resulting temporal and/or spatial patterns. There is likely to be some form of heterogeneity in the temporal and/or spatial component of a study, and in some cases the scale of measurement itself might be associated with the driver(s) of disease dynamics (e.g., climatic or elevational

gradients; Michael Samuel *personal communication*). The use of indices (e.g., prevalence) requires extra precautions besides those associated with standard sampling designs. Care must be taken to ensure that a meaningful relationship exists between an index and a metric of interest and entails calibration of the index with the metric (Conroy 1996). When inferences are based upon a single study site and health-state related detection probabilities (and/or other categories of detection stratification) remain stationary (i.e. invariant) over time, then temporal covariation of an index can be compared. On the other hand, to make inferences about differences in the value of an index temporally and/or spatially, state-specific detection probabilities (in the case of prevalence) must be equal or known with certainty. Otherwise, the magnitude of observed differences could be due solely to unequal detection probabilities of individuals rather than a true difference in the effect of interest. If an index is used to investigate the long-term trend of some parameter, then inferences can be made if it were known or determined that detection probabilities of the individuals in question varied randomly about some invariant mean value. The time series in this case must be of considerable length, as random variation in detection probabilities over few points along a time series can artificially induce significant bias between the value of an index and a parameter of interest (William Kendall *personal communication*).

Although the use of indices may be valid for making inferences with certain data types and under certain conditions (if assumptions are adequately tested), direct methods are generally preferred, as the misuse of an index can produce erroneous results and incorrect inferences (Conroy 1996, Jennelle et al. 2002). In fact, there are powerful arguments posed that indices should be abandoned altogether, and that direct and more robust methods of parameter estimation be used when needed (Anderson 2003).

Sampling of Populations

Often funding is a limiting factor precluding the establishment and maintenance of multiple field sites for sampling animal populations. Thus, random sampling and replication of field sites under this constraint may not be achievable. Furthermore, in such situations a complete biological population may not be encompassed in the sampling frame of the disease study, which precludes inference to the population. If one is constrained to such a limited sampling frame, then inferences based on the sampled locations are all that can be made. Despite the limitation of a small number of field sites in a disease study, if temporal replication of sampling can be maintained over multiple seasons or cycles of disease, then under the assumption that these field sites are a random sample of the population of possible sampling sites, variation in disease dynamics at these sites can be a proxy for the dynamics of the population if detection probabilities of relevant biological and environmental groups that influence local dynamics are estimated and incorporated into disease frequency data. If replication of field sites, possibly stratified by animal populations, environmental gradients, and/or some other geographic feature is possible, then use of a modified Horvitz-Thompson population estimator (Steinhorst and Samuel 1989, Samuel et al. 1992) may be used to estimate corrected disease prevalence (Michael Samuel *personal communication*). This estimator couples inclusion or detection probabilities of different groups of animals with probabilities of sampling different sites within a population to produce an unbiased estimator of population size. Using this approach to estimate the population of animals in each state-specific group, it is possible to estimate detection corrected prevalence in a similar manner as I have shown here. Estimation of the sampling variance of prevalence, however, is still a non-trivial problem. Although it is possible to approximate a sampling variance estimator using the delta method (Seber 1982), the theoretical validity for its use holds up only under

large-sample theory. Work in this area is needed to produce a more robust sampling variance estimator.

The scientific literature is rife with studies providing evidence for a myriad of factors, which can influence detectability of an organism (Lebreton et al. 1992, Williams et al. 2002, and references therein). I urge researchers to consider *a priori* which biotic and/or abiotic factors they know or expect to influence detection probabilities of the host species under study and design their sampling program (with consideration to appropriate capture methods and the temporal and spatial frame of capture events) to capture sufficient samples of animals within each relevant category. This pre-stratification strategy will better ensure successful estimation of detection probabilities of individuals within each grouping, and subsequent disease prevalence as a function of those groups.

Detection Probabilities, Bias, and Estimation of Disease Prevalence

It was demonstrated in Faustino et al. (2004) that detection probabilities between health-related states in the house finch–*M. gallisepticum* system are different (Appendix D). In a serin (*Serinus serinus*)–avian pox disease system, it was also shown that detection probabilities between pox-infected and uninfected individuals differed (Senar and Conroy 2004). In contrast to the house finch-MG system, detection probabilities of pox-infected serins were consistently higher than uninfected individuals. The magnitude of the difference between detection probabilities that I used for the hypothetical scenarios (e.g., the extreme values of $p_i^s = 0.75$ and 0.25) are not implausible; these values are quite realistic as Senar and Conroy (2004) found that the average difference of estimated detection probabilities between serin health states was even more extreme ($p^I = 0.81$ compared with $p^U = 0.21$). In both of these avian disease systems, the authors suspect that variation in detection probabilities as a

function of health state reflects a true underlying difference in behavior due to infection (possibly confounded with other sources of heterogeneity). Studies of diseases in other systems have shown that infection elicits behavioral modification of the host (Berdoy et al. 2000, Stentiford et al. 2001, Meadows and Meadows 2003), which can potentially influence the detectability of individuals.

Variation in detection probabilities due to health state alone is not the only source of heterogeneity that can induce bias in estimates of prevalence. Heterogeneity in detection due to age, gender, and/or other biological or environmental stratification may similarly induce bias in estimates of apparent prevalence. For example, consider a disease system in which it is established that detection probabilities of juvenile (J) and adult (A) animals are 1.0 and 0.25, respectively, and I obtain the following sample from the population: Infected (J = 20, A = 5) and uninfected (J = 20, A = 10). If I calculate disease prevalence unadjusted for age-specific detection probabilities then I obtain 0.45, but when I correct for detection probabilities then prevalence is 0.40, resulting in bias of apparent prevalence of 0.05. Of course, detection probabilities may vary as a more complicated function of interactions between biological and environmental covariates making it less clear how such variation in detection of individuals will influence bias. In any case, when it is demonstrated that parasitic infection and/or other possible sources of detection heterogeneity do not induce changes in detection of a host, then it may be reasonable to justify estimation of prevalence in its standard form. Yet, it is preferable to test this supposition explicitly to provide definitive evidence for or against a capture effect due to biological and/or environmental covariates.

Depending upon how disease state is defined in a study, it is possible that infected hosts that are misdiagnosed as uninfected and vice versa can further contribute to bias

in estimates of prevalence. A first-approximation to correcting estimates of disease frequency for such conditional state assignment errors requires a segment of the study population to be tested with both a gold standard (reference) along with the standard field diagnostic. Based on this sample of the population, a standard misclassification matrix (used to estimate sensitivity and specificity) can be created to estimate the complement of the positive and negative predictive values. Using these probabilities, I can derive the following statistics

$$C_i^{U'} = C_i^U - (C_i^U (1 - NPV)) + (C_i^I (1 - PPV))$$

$$C_i^{I'} = C_i^I - (C_i^I (1 - PPV)) + (C_i^U (1 - NPV))$$

where,

$C_i^{s'}$ = corrected observed count of the focal species in health state s (I : infected or U : uninfected) at time i accounting for misclassification bias,

C_i^s = observed count of the focal species in health state s (I : infected or U : uninfected) at time i ,

NPV = negative predictive value or the probability of an uninfected individual given a negative test result,

PPV = positive predictive value or the probability of an infected individual given a positive test result.

To account for misclassification bias in estimates of prevalence, these corrected counts can be substituted for the observed health-state specific counts of individuals in equation 3. I stress that state-specific detection probabilities in this case must be estimated using only the segment of the population assessed for disease status with the gold standard, since the unknown identities of the misclassified marked sample will bias detection probabilities.

In addition, for some diseases the detection probability of infected individuals may be intensity-dependent, meaning that the detectability of an individual is a function of the severity of illness it is suffering from. Given data limitations, epidemiologists and disease ecologists typically create discrete health classes of individuals, and with respect to detectability, in all likelihood the true detection function of an individual covaries continuously with a gradient in health status. Based upon preliminary simulations where I assigned disease-intensity dependent detection probabilities to individuals, I found that such detection heterogeneity (if sufficiently large) can result in additional bias of unadjusted estimates of prevalence. As such, I urge researchers to consider estimating detection probabilities and applying subsequent corrections to prevalence estimators at the finest possible scale that their data will allow in order to further reduce bias.

There are many other potential biases that can affect estimates of prevalence and scientists have been quick to point out many sources, which span from the study sampling frame (Delahay et al. 2001) to the model assumptions underlying parameter estimation of detection probabilities (Michael Samuel *personal communication*). In this paper I have focused on one potentially serious source of bias in estimates of prevalence, differential detection probabilities of animals stratified by health state (easily extended to other sources of capture heterogeneity), which involves a relatively straightforward correction.

To help researchers obtain a general idea of the potential bias that might accompany estimates of prevalence uncorrected for state-specific detection probabilities, I provide the following calculation for expected bias. The inputs are simply the counts of individuals and respective detection probabilities stratified by disease state. Using the values of state-specific counts obtained in field studies and detection probabilities that researchers might expect for their particular study species

as inputs in the following expression will give an idea of the potential bias of apparent prevalence if health state related detection probabilities are not accounted for. The formulation of expected bias is easily extendable to account for multiple groups of individuals with differential detection probability (for simplicity I use health state differences). The expected bias of an estimated parameter is defined as

$$bias = E(\hat{\theta}) - \theta$$

where,

$E(\hat{\theta})$ = the expected value of the estimated parameter of interest; in this case apparent prevalence,
 θ = the value of the parameter of interest; in this case the value of estimated prevalence corrected for differential detection probability (equation 3).

This expression can be applied to estimating bias in apparent disease prevalence as

$$bias = \left(\frac{C_i^I C_i^U (\alpha_i - 1)}{(C_i^I + C_i^U)(C_i^I + \alpha_i C_i^U)} \right)$$

where,

C_i^s = observed count of the focal species in health state s (I : infected or U : uninfected) at time i ,

α_i = the ratio of estimated detection probability of the focal species in the infected state to the detection probability in the uninfected state at time i ($\hat{p}_i^I / \hat{p}_i^U$).

When the value of α_i is greater than one, then apparent prevalence is positively biased and vice versa. Using this formulation, I provide an evaluation of bias and percent relative bias for a range of differences in health state-specific detection probabilities in Appendix E.

When CMR studies are implemented to permit estimation of demographic parameters under multistate type models (Brownie et al. 1993, Williams et al. 2002), it is possible to use health-state specific survival and transition probabilities to estimate disease prevalence (Senar and Conroy 2004). The general approach outlined in Senar and Conroy (2004) implicitly incorporates variation in detection probabilities between health states into estimates of prevalence and only requires a starting point (some baseline estimate of prevalence in the time series) to initiate their recursive prevalence function. In disease systems with clear epidemic cycles, this approach is simple to use. If there is a non-zero value of apparent prevalence at the beginning of a study period, then the methodology outlined in this paper can be used to generate a starting point.

Alternative Approaches for Estimating Detection Probabilities

Planning and conducting a CMR study can be financially and logistically prohibitive on a large scale, so it is unrealistic to expect researchers to be able to conduct multiple CMR studies across the spatial and temporal scale typically encountered in large-scale disease surveillance or monitoring studies. With frequently limited funding and field assistance, how can wildlife disease investigators expect to estimate detection probabilities? With careful study design, moderate effort, and current theoretical and empirical advances in statistical methodology, even large-scale studies can be adequately designed to produce robust estimates of population size that account for detection probabilities (Nichols et al. 2000, Royle and Nichols 2003), which can subsequently be used to estimate disease prevalence or incidence.

In recent years, there have been several robust approaches developed for estimating abundance using traditional avian point-count and presence-absence data. If the primary focus is on estimating disease prevalence for some host species, then it is possible to partition diseased and healthy individuals as different groups. The use of these particular sampling techniques for estimating abundance, when used expressly for estimating disease prevalence, are conditional on an observer's ability to assess the disease state visually in the field. If data can be collected in a manner similar to avian point counts (Ralph et al. 1995), then several possibilities exist. The double-observer approach developed in Nichols et al. (2000) provides robust methods for estimating abundance and/or density using point-count data. Given there are two observers at a given sampling point, each person can collate independent counts of individuals, which can be used to estimate detection probabilities of the groups of interest. Using information theoretic methods, specifically the Akaike information criterion (Akaike 1973), then heterogeneity in detection probability can be modeled spatially and temporally by comparing alternative models (Burnham and Anderson 2002). An alternative approach to abundance estimation amenable to point-count or presence-absence data is described in Royle and Nichols (2003). This method takes advantage of the inherent heterogeneity in abundance associated with heterogeneity in detection probabilities. Given repeated sampling of multiple locations over multiple time periods, estimates of abundance (of infected and healthy individuals) corrected for detection probability can be produced.

It may be possible to estimate disease prevalence with a large-scale citizen science sampling framework similar to that described in Altizer et al. (2004b) using patch-occupancy models (MacKenzie et al. 2002). The recent advent of this class of CMR models allows for robust estimation of community dynamic parameters such as species occupancy, colonization, and extinction rates when detection probabilities are

less than unity. Instead of treating physical locations as patches, one could substitute time periods (days), thereby providing a means of incorporating differential detection probabilities into estimates of prevalence without having to mark individuals.

In a study conducted by Samuel et al. (1992), the goal was to determine if various factors influenced the visibility of elk from aerial surveys, which in turn would affect estimates of population size. By attaching radio telemetry devices to a sample of animals and recording a number of covariates associated with elk captured with and without the use of tracking equipment, they were able to determine that both vegetation cover and group size influenced the visibility of elk from aerial surveys using competing models within a logistic regression framework. Using this approach, they were able to construct sightability models to predict in essence the detection probability of observing elk groupings. A similar approach with a double-observer modification (akin to Nichols et al. 2000) could be used in wildlife disease systems to estimate the detectability of individuals stratified by expected sources of detection heterogeneity. At the time of marking and attachment of radio telemetry devices, animals would be assessed (via blood or tissue sample, etc.) for their disease state (among other things). In the case of an aerial survey sampling approach, a telemetry operator and regular observer would simultaneously record sighted animals. Conditioning on the sample of radio-marked animals (whose disease state and other biological attributes are known), it would be possible to determine the state-specific detection probability of animals sampled by the regular observer for a given sampling occasion.

Concluding Remarks

At the very least, even a minimal effort to estimate health-state specific detection probabilities (by way of a shortened mark-recapture study) would be useful in

providing investigators with some idea of whether differential detection probabilities occur between healthy and diseased individuals (an obvious first choice as a major source of detection heterogeneity). This approach is not limited to host-pathogen systems for which clinical signs can be visually assessed. The necessary requirements include that the host species be capturable by some means (preferably the most efficient and successful method maximizing the likelihood of capture) and that some pathogen diagnostic test be available for captured animals (via blood, fecal, or tissue sample). If a field diagnostic test is used, the test procedure should be administered to all captured animals regardless of in-hand assessment of disease state, to ensure that subsequent trap effects (if imposed) due to capture/handling are more likely homogeneous across animals in each health state. Even if small sample sizes of marked individuals are obtained with moderate to high detection probabilities, state-specific detection probabilities can be constrained to be time-invariant with a simple structure imposed for apparent survival and health state transition rates (in the case of multistate CMR models). Standard Cormack-Jolly-Seber CMR models (Williams et al. 2002) could be used also, with health state treated as a time-varying binary covariate for each individual. Despite the simple model structure, estimates of detection probabilities will be more likely to converge and be of value to researchers in correcting estimates of disease frequency.

Both the House Finch-*M. gallisepticum* system that I use in this paper and the Serin-avian pox system (Senar and Conroy 2004) that I reference are unique in that (i) health status can be inferred from visual observation of birds and (ii) an intensive CMR effort has afforded the opportunity to directly estimate detection probabilities for animals in alternative health states. I recognize that this type of situation may be uncommonly encountered in wildlife disease studies and embodies the confluence of both beneficial circumstances and exceptional data collecting opportunities. Yet, the

data from both systems clearly demonstrate the importance of accounting for detection probabilities in wildlife disease studies, and I am confident this knowledge will help investigators produce more reliable estimates of disease frequency.

Acknowledgments

I would like to thank Cristina Faustino for providing estimates from her CMR analyses; Cristina Faustino, Veronique Connolly, Andy Davis, Melanie Driscoll, Dana Hawley, and Elliott Swarthout for their efforts collecting field data. Special thanks to Michael Samuel and Mary Conner for helpful reviews of this manuscript. Thanks to Sonia Altizer, Andre Dhondt, Andy Dobson, Wes Hochachka, and William Kendall for providing useful comments on earlier versions of this manuscript. I thank James Hines (USGS - Patuxent) for use of his program MARKWAIT in simulations. I gratefully acknowledge all of the ‘citizen scientists’ that contributed survey data to the HFDS. Also, I thank the National Science Foundation (under Grant No. DEB-0094456). Any opinions, findings and conclusions or recommendations expressed in this material are those of the author and do not necessarily reflect the views of the National Science Foundation. I also thank the Georgia Cooperative Fish and Wildlife Research Unit.

APPENDIX A. General Formula for Corrected Prevalence

In many cases, it will be possible to partition sources of heterogeneity in detection probabilities into one or more discrete classes of individuals. For example, consider a situation where detection probabilities vary as a function of gender, age, and health status. Incorporating class specific detection probabilities, a corrected estimate of prevalence can be calculated as

$$\begin{aligned}\hat{\delta}_i &= \frac{\sum_{k=1}^2 \sum_{l=1}^a \hat{N}_i^{I,kl}}{\sum_{k=1}^2 \sum_{l=1}^a \hat{N}_i^{I,kl} + \sum_{k=1}^2 \sum_{l=1}^a \hat{N}_i^{U,kl}} = \frac{\sum_{k=1}^2 \sum_{l=1}^a \frac{C_i^{I,kl}}{\hat{p}_i^{I,kl}}}{\sum_{k=1}^2 \sum_{l=1}^a \frac{C_i^{I,kl}}{\hat{p}_i^{I,kl}} + \sum_{k=1}^2 \sum_{l=1}^a \frac{C_i^{U,kl}}{\hat{p}_i^{U,kl}}} \\ &= \frac{\left(\prod_{k=1}^2 \prod_{l=1}^a \hat{p}_i^{U,kl} \right) \left[\sum_{k=1}^2 \sum_{l=1}^a C_i^{I,kl} \left(\frac{\prod_{y=1}^2 \prod_{z=1}^a \hat{p}_i^{I,yz}}{\hat{p}_i^{I,kl}} \right) \right]}{\sum_{j=1}^2 \sum_{k=1}^2 \sum_{l=1}^a C_i^{jkl} \left(\frac{\prod_{x=1}^2 \prod_{y=1}^2 \prod_{z=1}^a \hat{p}_i^{xyz}}{\hat{p}_i^{jlk}} \right)}\end{aligned}$$

where,

$\hat{\delta}_i$ = corrected disease prevalence at time i ,

C_i^{jkl} = observed count of focal species in health state j (I : infected or U : uninfected), gender k , and age l (up to a age classes) at time i ,

\hat{p}_i^{jkl} = the estimated detection probability of focal species in health state j , gender k , and age l at time i ,

\hat{p}_i^{xyz} = the estimated detection probability of focal species in health state x , gender y , and age z (up to a age classes) at time i ,

Extending the expression for additional states is straightforward.

APPENDIX B. Approximate Conditional Variance for Prevalence Estimator

Estimated prevalence is a derived parameter, expressed as the ratio of constants (raw counts) multiplied by state-specific detection probabilities with some level of uncertainty. I used the Delta Method (Seber 1982) to derive the approximate conditional sampling variance for the prevalence estimator. Estimates of weekly detection probability were obtained from Faustino et al. (2004).

The expression for the approximate conditional sampling variance of the reduced form of estimated prevalence (equation 3), is

$$\text{var}(\hat{\delta}_{ijk}^R | C_{ijk}^U, C_{ijk}^I) = \left(\frac{\partial \hat{\delta}_{ijk}^R}{\partial \hat{p}_{ijk}^s} \right) \times \hat{\Sigma} \times \left(\frac{\partial \hat{\delta}_{ijk}^R}{\partial \hat{p}_{ijk}^s} \right)^T, \quad (\text{B1})$$

where,

$\hat{\delta}_{ijk}^R$ = Reduced form expression for corrected estimate of prevalence in year i

(i = 2002-03), month j (j = Nov-Mar), and week k (1, ..., 4),

C_{ijk}^s = observed count of animals in health state s (I: infected or

U: uninfected) in year i, month j, and week k,

\hat{p}_{ijk}^s = the detection probability for an animal in health state s (I: infected or

U: uninfected) in year i, month j, and week k,

$\hat{\Sigma}$ = the variance-covariance matrix of the detection probabilities, \hat{p}_{ijk}^s .

Using matrix algebra, the Delta Method yields

$$\text{var}(\hat{\delta}_{ijk}^R | C_{ijk}^U, C_{ijk}^I) = \begin{bmatrix} -\frac{C_{ijk}^I C_{ijk}^U \hat{p}_{ijk}^U}{\left((C_{ijk}^U \hat{p}_{ijk}^I) + (C_{ijk}^I \hat{p}_{ijk}^U) \right)^2} & \frac{C_{ijk}^I C_{ijk}^U \hat{p}_{ijk}^I}{\left((C_{ijk}^U \hat{p}_{ijk}^I) + (C_{ijk}^I \hat{p}_{ijk}^U) \right)^2} \\ -\frac{C_{ijk}^I C_{ijk}^U \hat{p}_{ijk}^U}{\left((C_{ijk}^U \hat{p}_{ijk}^I) + (C_{ijk}^I \hat{p}_{ijk}^U) \right)^2} & \frac{C_{ijk}^I C_{ijk}^U \hat{p}_{ijk}^I}{\left((C_{ijk}^U \hat{p}_{ijk}^I) + (C_{ijk}^I \hat{p}_{ijk}^U) \right)^2} \end{bmatrix} \times \hat{\Sigma} \times \begin{bmatrix} -\frac{C_{ijk}^I C_{ijk}^U \hat{p}_{ijk}^U}{\left((C_{ijk}^U \hat{p}_{ijk}^I) + (C_{ijk}^I \hat{p}_{ijk}^U) \right)^2} & \frac{C_{ijk}^I C_{ijk}^U \hat{p}_{ijk}^I}{\left((C_{ijk}^U \hat{p}_{ijk}^I) + (C_{ijk}^I \hat{p}_{ijk}^U) \right)^2} \\ -\frac{C_{ijk}^I C_{ijk}^U \hat{p}_{ijk}^U}{\left((C_{ijk}^U \hat{p}_{ijk}^I) + (C_{ijk}^I \hat{p}_{ijk}^U) \right)^2} & \frac{C_{ijk}^I C_{ijk}^U \hat{p}_{ijk}^I}{\left((C_{ijk}^U \hat{p}_{ijk}^I) + (C_{ijk}^I \hat{p}_{ijk}^U) \right)^2} \end{bmatrix}^T,$$

where,

C_{ijk}^s = observed count of finches in health state s (I: infected or U: uninfected) in year i, month j, and week k,

\hat{p}_{ijk}^s = the estimated detection probability for a finch in health state s (I: infected or U: uninfected) in year i, month j, and week k,

$\hat{\Sigma}$ = the variance-covariance matrix of the detection probabilities, \hat{p}_{ijk}^s .

The approximate conditional sampling variance of the prevalence estimator in equation (B1) is thus

$$\text{var}(\hat{\delta}_{ijk}^R | C_{ijk}^U, C_{ijk}^I) = \frac{C_{ijk}^I{}^2 C_{ijk}^U{}^2 \left(\hat{p}_{ijk}^U{}^2 \text{var}(\hat{p}_{ijk}^I) + \hat{p}_{ijk}^I{}^2 \text{var}(\hat{p}_{ijk}^U) - 2\hat{p}_{ijk}^U \hat{p}_{ijk}^I \text{cov}(\hat{p}_{ijk}^U, \hat{p}_{ijk}^I) \right)}{\left(C_{ijk}^U \hat{p}_{ijk}^I + C_{ijk}^I \hat{p}_{ijk}^U \right)^4}.$$

APPENDIX C. Bias Evaluation of an Approach to Prevalence Estimation in a Special Case.

As the approach presented in Senar and Conroy (2004) and in this paper are dependent upon the temporal resolution allowable from the data (i.e., resolution in the sense that there is sufficient data to estimate demographic parameters and detection probabilities with time variation if present, which in turn become the constituent inputs that produce the prevalence estimator), I was particularly interested in situations where a true underlying temporal trend in infection probability existed, while only time invariant infection probability was estimable given the data. To this end, I evaluated bias in estimated prevalence with respect to this special condition under two scenarios (which represent truth) with state-specific and time invariant apparent survival, detection, and recovery probabilities over 15 time steps ($\phi_i^U = 0.9$, $\phi_i^I = 0.6$; $p_i^U = 0.5$, $p_i^I = 0.7$; $\psi_i^{IU} = 0.3$). In scenario (i) true prevalence is a function of a decreasing trend in ψ_i^{UI} (infection probability) of the functional form $\psi_i^{UI} = 0.3333e^{-0.1054 \cdot i}$, $i = 1$ to 15 , which corresponds to a 10% decrease in time-specific infection probability per time step (Fig. C2.1*i*). This scenario is representative of a newly introduced pathogen into a naïve population of susceptible individuals, where less resistant genotypes are removed from the susceptible pool earlier in the disease cycle. In scenario (ii) true prevalence is a function of an increasing trend in ψ_i^{UI} (infection probability) of the functional form $\psi_i^{UI} = 0.2857e^{0.0488 \cdot i}$, $i = 1$ to 15 , which corresponds to a 5% increase in time-specific infection probability per time step (Fig. C2.1*ii*). This scenario could arise when there is an increase in the virulence or transmission of some pathogen in a population. The decreasing trend in ‘true’ prevalence under scenario (i) would also be observed in distinct scenarios with a trend in increasing recovery probability or a decreasing trend in survival probability of diseased animals. Likewise, the increasing trend in ‘true’ prevalence under scenario

(ii) would also be observed in distinct scenarios with a decreasing trend in recovery probability or decreasing trend in survival probability of healthy animals. As such, the two scenarios I consider here can be considered qualitatively representative of the trends in prevalence expected under a variety of demographic circumstances (assuming all other parameters are set constant).

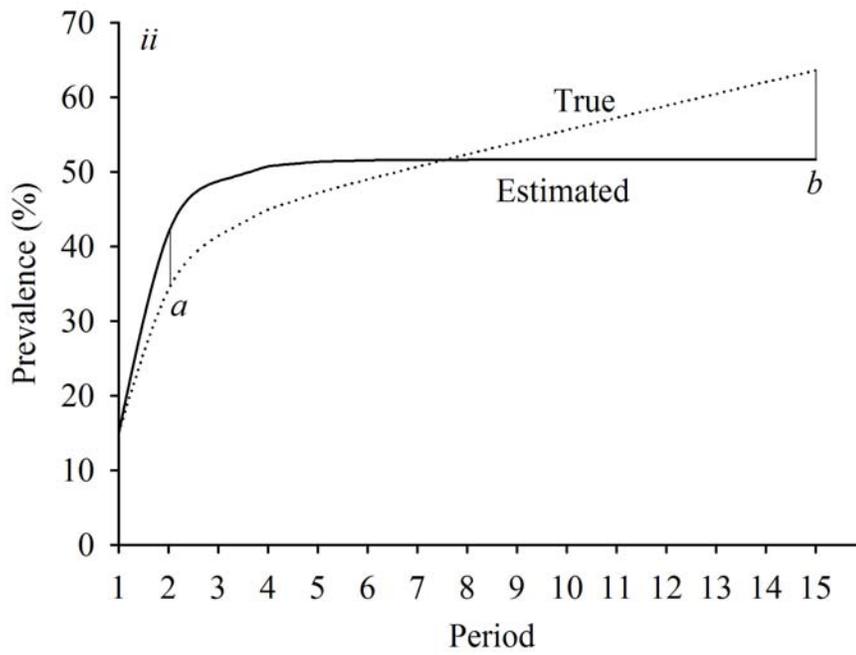
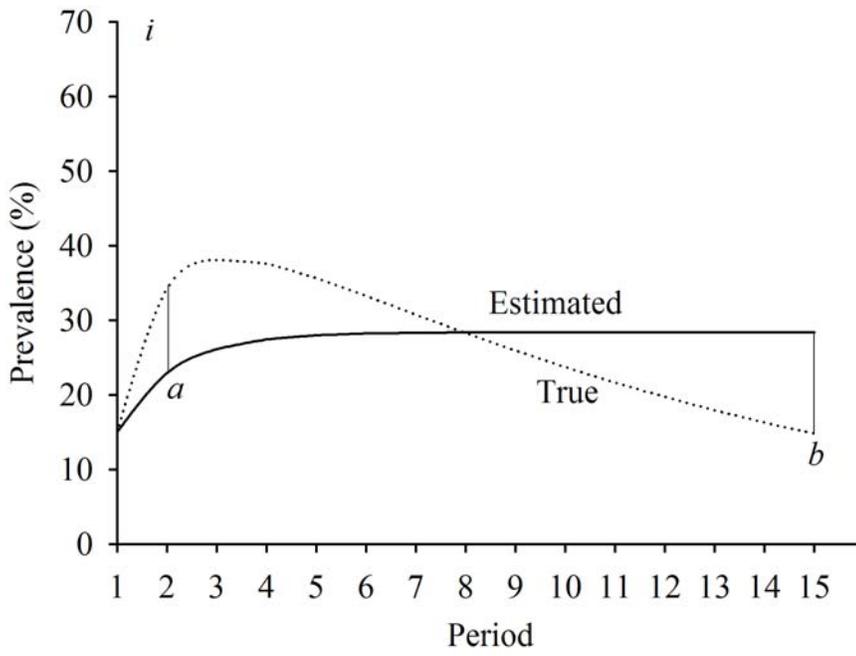
Using SAS software (v.9 SAS Institute), I simulated encounter histories under the aforementioned demographic scenarios starting each simulated population with 15% diseased individuals and for convenience assigned 1000 newly released (newly marked) individuals at each time step. I was not interested in determining a lower bound for the number of marked individuals necessary for adequate support of models with time-dependent variation. Rather, I used 1000 newly released ‘simulated’ individuals in each cohort in the bootstrap samples only to facilitate the convergence of time-invariant parameter estimates. I simulated 200 encounter histories under each scenario and estimated state-specific survival and transition probabilities using program MARK v.4.2 (White and Burnham 1999) under the model $\{ \phi(\text{disease state}) p(\text{disease state}) \psi^{UI}(\cdot) \psi^{IU}(\cdot) \}$ (note the period surrounded by parenthesis indicates time-invariance). I used program MARKWAIT designed by James E. Hines of Patuxent Wildlife Research Center in Laurel, MD to automate the creation of sample encounter histories in SAS, and subsequently estimate parameters under the aforementioned model in MARK. I suspect that in a fair number of wildlife disease studies, a sufficient number of recaptures and observed state transitions will not be available for convergence of mark-recapture models with time-dependent survival and/or state transition parameters. As previously mentioned, multistate models are considerably ‘data hungry’, so I have conducted these simulations under the special case that estimates of survival and state transitions are time invariant. With time invariant parameter estimates obtained from the bootstrap samples, I estimated the

value of disease prevalence at each time step using the Senar and Conroy (2004) approach and compared this quantity with ‘true’ prevalence calculated using the two exponential models of infection probability (with all other parameters equal) at each respective time step under both scenarios. For each scenario I plotted and calculated the two maximal values of percent relative bias in estimated prevalence.

Projecting disease prevalence over 15 time steps under scenario (i) produced a declining trend in ‘true’ prevalence (Fig. C2.1*i*), while projection under scenario (ii) produced an increasing trend in ‘true’ prevalence (Fig. C2.1*ii*). The expected value of infection probability under scenario (i) was 0.1740, and that under scenario (ii) was 0.3854. Using the given values of survival and recovery probabilities, along with the ‘true’ value of infection probability obtained from the exponential equations under each scenario I calculated ‘true’ prevalence, and estimated prevalence using the Senar and Conroy (2004) multistate model approach using the same given set of parameter values along with the expected value of infection probability generated using a bootstrap. I compared ‘true’ prevalence with estimated prevalence under the Senar and Conroy (2004) approach, and found significant bias in the pattern and magnitude of estimated prevalence when time dependence in infection probability was not taken into account (Fig. C2.1). The simulations show that when time invariant infection probability is used in the calculation of disease prevalence with the Senar and Conroy (2004) approach in situations when there is a decreasing or increasing trend in ‘true’ infection probability, respectively, that important temporal trends in disease prevalence will not be detected. If a trend in true disease prevalence is mediated by a concomitant trend in infection probability, recovery probability, or state-specific survival, and if CMR data are not sufficient (plentiful) to support models with time variation in the trending parameter, then this type of inferential error will occur. This phenomenon could also occur when using the prevalence estimator presented in the

body of the paper if a true trend in state-specific detection probability is not discernable given the data. As such, I urge researchers to be very cautious when making inferences about patterns in estimated disease prevalence when data is sparse.

Figure C2.1. Evaluation of a special case of the Senar and Conroy (2004) multistate model approach to prevalence estimation reveals that under certain conditions, researchers may falsely interpret patterns of estimated prevalence to be time invariant, when in fact a temporal trend exists. The following figures show the potential bias in estimated prevalence when a temporal trend in ‘true’ prevalence is mediated by a trend in decreasing (*i*) or increasing infection probability (*ii*). Points *a* and *b* in each panel indicate where maximal levels of percent relative bias (%RB) in estimated prevalence occur; in panel (*i*) %RB at points *a* and *b* are -33% and 92%, respectively, while in panel (*ii*) %RB at points *a* and *b* are 22% and -19%, respectively. The failure to detect a true temporal trend in estimated prevalence will occur if there is insufficient mark-recapture data to support models with time-dependence in the demographic parameters that are truly time-dependent when using the Senar and Conroy (2004) approach to estimating prevalence.



APPENDIX D

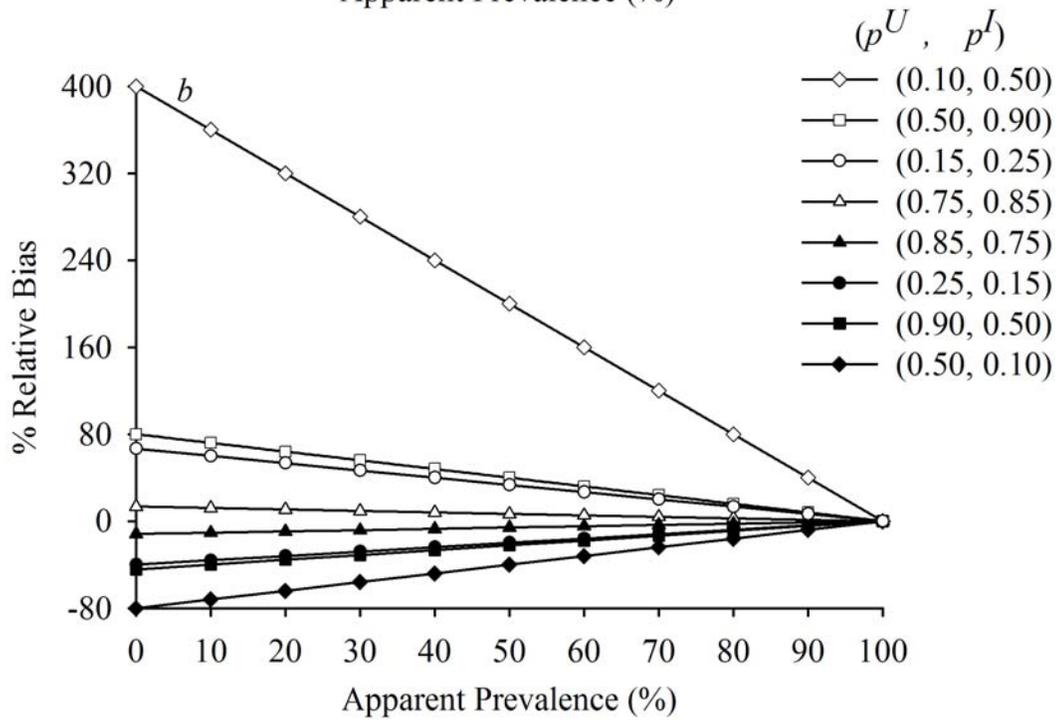
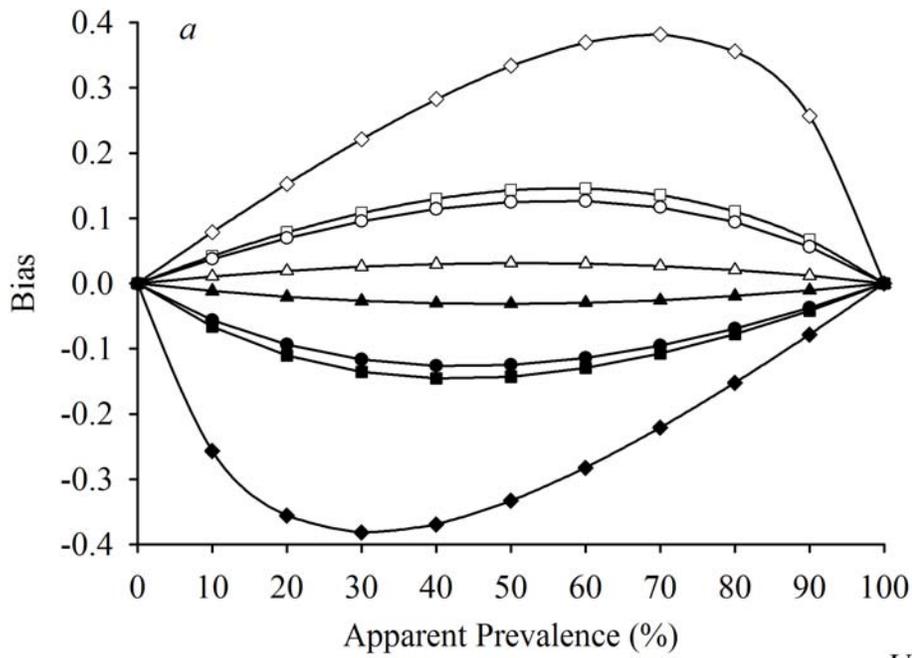
Weekly counts (C), estimated encounter probabilities (\hat{p} : U = uninfected and I = infected) taken from Faustino et al. (2004), apparent prevalence ($\hat{\delta}^A$), corrected prevalence ($\hat{\delta}^C$), and percent relative bias (%RB) from November through March 2002-03 in Ithaca, NY, USA.

Week	C^U	C^I	\hat{p}^U	\hat{p}^I	$\hat{\delta}^A$	$\hat{\delta}^C$	%RB
Nov 9	139	25	0.887	0.892	15.2	15.2	0.5
Nov 16	171	22	0.105	0.103	11.4	11.6	-1.7
Nov 23	40	5	0.466	0.549	11.1	9.6	15.8
Nov 30	90	19	0.777	0.631	17.4	20.6	-15.5
Dec 7	126	16	0.722	0.549	11.3	14.3	-21.3
Dec 14	102	9	0.866	0.426	8.1	15.2	-46.7
Dec 21	-	-	0.865	0.426	-	-	-
Dec 28	12	1	0.616	0.425	7.7	10.8	-28.6
Jan 4	65	3	0.843	0.573	4.4	6.4	-30.6
Jan 11	104	17	0.450	0.275	14.0	21.1	-33.4
Jan 18	45	7	0.647	0.610	13.5	14.2	-4.9
Feb 1	22	8	0.700	0.463	26.7	35.5	-24.8
Feb 8	102	19	0.542	0.265	15.7	27.6	-43.1
Feb 15	43	9	0.779	0.232	17.3	41.3	-58.1
Feb 22	69	7	0.715	0.588	9.2	11.0	-16.1
Mar 1	59	9	0.997	0.539	13.2	22.0	-39.9
Mar 8	83	13	0.595	0.177	13.5	34.5	-60.7

APPENDIX E. **Bias in Apparent Prevalence as a Function of a Range of Differences in Health State-specific Detection Probabilities**

To give readers an idea of the general form of bias to be expected in estimated apparent prevalence when health state-specific detection probabilities differ, I calculated both expected bias (Fig. E2.1a) and percent relative bias (%RB) (Fig. E2.1b) for eight pairs of differential detection probability representing a range of potential differences across probability space. There are three major influences on the magnitude of bias in apparent prevalence when considering health state as the primary source of detection heterogeneity. These include i) the magnitude of estimated apparent prevalence, ii) the magnitude of the difference between health state-specific detection probabilities, and iii) the location of detection probabilities in probability space (0-1.0). Relating to point (i), as estimated apparent prevalence approaches 0.5 for a given pair of detection probabilities, the magnitude of bias increases. With respect to point (ii), bias is positively related to the magnitude of difference in detection probability between infected and uninfected individuals. As for point (iii), for a given absolute difference in detection probability (e.g., 0.10), bias will generally be higher when detection probabilities are low (e.g., examine bias in apparent prevalence as a function of $p^U = 0.15$, $p^I = 0.25$ vs. $p^U = 0.75$, $p^I = 0.85$). I highlight these results to show researchers that the relationship between detection probabilities and bias in apparent prevalence may not be straightforward.

Figure E2.1. Bias in apparent prevalence as a function of a range of differences in health state-specific detection probabilities (a) and percent relative bias (%RB) as a function of a range of differences in health state-specific detection probabilities (b). In the figure key, (p^U , p^I) are the values for detection probability of uninfected and infected individuals, respectively.



REFERENCES

- Akaike, H. 1973. Information theory and an extension of the maximum likelihood principle. Pages 267-281 in B. N. Petran and F. Csaki, editors. International symposium on information theory, 2nd edn. Akadémiai Kiadó, Budapest, Hungary.
- Altizer, S., A. K. Davis, K. C. Cook, and J. J. Cherry. 2004a. Age, sex, and season affect the risk of mycoplasmal conjunctivitis in a southeastern house finch population. *Canadian Journal of Zoology* **82**:1-9.
- Altizer, S., W. M. Hochachka, and A. A. Dhondt. 2004b. Seasonal dynamics of mycoplasmal conjunctivitis in eastern North American House Finches. *Journal of Animal Ecology* **73**:309-322.
- Anderson, D. R. 2001. The need to get the basics right in wildlife field studies. *Wildlife Society Bulletin* **29**:1294-1297.
- Anderson, D. R. 2003. Response to Engeman: Index values rarely constitute reliable information. *Wildlife Society Bulletin* **31**:288-291.
- Atkinson, C. T., J. K. Lease, R. J. Dusek, and M. D. Samuel. 2005. Prevalence of pox-like lesions and malaria in forest bird communities on leeward Mauna Loa volcano, Hawaii. *Condor* **107**:537-546.
- Bailey, L. L., W. L. Kendall, D. R. Church, and H. M. Wilbur. 2004. Estimating survival and breeding probability for pond-breeding amphibians: A modified robust design. *Ecology* **85**:2456-2466.
- Bellrose, F. C. 1959. Lead poisoning as a mortality factor in waterfowl populations. *Illinois Natural History Survey Bulletin* **27**:235-288.
- Berdoy, M., J. P. Webster, and D. W. MacDonald. 2000. Fatal attraction in rats infected with *Toxoplasma gondii*. *Proceedings of the Royal Society of London* **267**:1591-1594.

- Brownie, C., J. E. Hines, J. D. Nichols, K. H. Pollock, and J. B. Hestbeck. 1993. Capture-recapture studies for multiple strata including non-Markovian transitions. *Biometrics* **49**:1173-1187.
- Burnham, K. P., and J. D. Nichols. 1985. On condition bias and band-recovery data from large-scale waterfowl banding programs. *Wildlife Society Bulletin* **13**:345-349.
- Burnham, K. P., and D. R. Anderson. 2002. Model selection and multimodel inference: A practical information-theoretic approach (2nd edition). Springer, New York, New York, USA.
- Conner, M. M., C. W. McCarty, and M. W. Miller. 2000. Detection of bias in harvest-based estimates of chronic wasting disease prevalence in mule deer. *Journal of Wildlife Diseases* **36**:691-699.
- Conroy, M. J. 1996. Abundance indices. Pages 179-192 in D. Wilson, F. R. Cole, J. D. Nichols, R. Rudran, and M. S. Foster, editors. *Measuring and monitoring of biological diversity: standard methods for mammals*. Smithsonian Institution Press, Washington, D.C., USA.
- Davis, A. K. 2005. A comparison of age, size and health of house finches captured with two trapping methods. *Journal of Field Ornithology* **76**:339-344.
- Delahay, R. J., S. Langton, G. C. Smith, R. S. Clifton-Hadley, and C. L. Cheeseman. 2000. The spatio-temporal distribution of *Mycobacterium bovis* (bovine tuberculosis) infection in a high-density badger population. *Journal of Animal Ecology* **69**:428-441.
- Delahay, R. J., C. L. Cheeseman, and R. S. Clifton-Hadley. 2001. Wildlife disease reservoirs: The epidemiology of *Mycobacterium bovis* infection in the European badger (*Meles meles*) and other British mammals. *Tuberculosis* **81**:43-49.

- Dobson, A., and M. Meagher. 1996. The population dynamics of brucellosis in the Yellowstone National Park. *Ecology* **77**: 1026-1036.
- Domenech, J., and J. C. Senar. 1997. Trapping methods can bias age ratio in samples of passerine populations. *Bird Study* **44**:348-354.
- Domenech, J., and J. C. Senar. 1998. Trap type can bias estimates of sex ratio. *Journal of Field ornithology* **69**:380-385.
- Fallon, S. M., E. Bermingham, and R. E. Ricklefs. 2003. Island and taxon effects in parasitism revisited: Avian malaria in the Lesser Antilles. *Evolution* **57**:606-615.
- Faustino, C. R., C. S. Jennelle, V. Connolly, A. K. Davis, E. C. Swarthout, A. A. Dhondt, and E. G. Cooch. 2004. *Mycoplasma gallisepticum* infection dynamics in a House Finch population: Empirical analysis of seasonal variation, encounter and transmission rate. *Journal of Animal Ecology* **73**:651-669.
- Hepp, G. R., R. J. Blohm, R. E. Reynolds, J. E. Hines, and J. D. Nichols. 1986. Physiological condition of autumn-banded mallards and its relationship to hunting vulnerability. *Journal of Wildlife Management* **50**:177-183.
- Hestbeck, J. B., J. D. Nichols, and R. A. Malecki. 1991. Estimates of movement and site fidelity using mark-resight data of wintering Canada geese. *Ecology* **72**:523-533.
- Hill, G. E. 1993. House finch (*Carpodacus mexicanus*). Pages 1-24 in A. Poole, and F. Gill, editors. *The Birds of North America*, no. 46. Academy of Natural Sciences, Philadelphia and American Ornithologists' Union, Washington, D.C.
- Hines, J. E. 1994. MSSURVIV Users Manual. National Biological Service, Patuxent Wildlife Research Center, laurel, MD 20708-4017.
- International Working Group for Disease Monitoring and Forecasting. 1995a. Capture-recapture and multiple-record systems estimation I: History and theoretical development. *American Journal of Epidemiology* **142**:1047-1058.

- International Working Group for Disease Monitoring and Forecasting. 1995*b*.
Capture-recapture and multiple-record systems estimation II: Applications in human diseases. *American Journal of Epidemiology* **142**:1059-1068.
- Jennelle, C. S., M. C. Runge, and D. I. MacKenzie. 2002. The use of photographic rates to estimate densities of tigers and other cryptic mammals: A comment on misleading conclusions. *Animal Conservation* **5**:119-120.
- Joly, D. O., C. A. Ribic, J. A. Langenberg, K. Beheler, C. A. Batha, B. J. Dhuey, R. E. Rolley, G. Bartelt, T. R. Van Deelen, and M. D. Samuel. 2003. Chronic wasting disease in free-ranging Wisconsin white-tailed deer. *Emerging Infectious Diseases* **9**: 599-601.
- Joly, D. O., and F. Messier. 2004. Factors affecting apparent prevalence of tuberculosis and brucellosis in wood bison. *Journal of Animal Ecology* **73**:623-631.
- Kendall, W. L., J. D. Nichols, and J. E. Hines. 1997. Estimating temporary emigration using capture-recapture data with Pollock's robust design. *Ecology* **78**:563-578.
- Kendall, W. L., J. E. Hines, and J. D. Nichols. 2003. Adjusting multistate capture-recapture models for misclassification bias: Manatee breeding proportions. *Ecology* **84**:1058-1066.
- Lebreton, J.-D., K. P. Burnham, J. Clobert, and D. R. Anderson. 1992. Modeling survival and testing biological hypothesis using marked animals: A unified approach with case studies. *Ecological Monographs* **66**:67-118.
- Loot, G., M. Aldana, and S. A. Navarrete. 2005. Effects of human exclusion on parasitism in intertidal food webs of central Chile. *Conservation Biology* **19**:203-212.
- MacKenzie, D. I., and W. L. Kendall. 2002. How should detection probability be incorporated into estimates of relative abundance? *Ecology* **83**:2387-2393.

- MacKenzie, D. I., J. D. Nichols, G. B. Lachman, S. Droege, J. A. Royle, and C. A. Langtimm. 2002. Estimating site occupancy rates when detection probabilities are less than one. *Ecology* **83**:2248-2255.
- Mausner, J. S., and A. K. Bahn. 1974. *Epidemiology: An introductory text*. W. B. Saunders Company, London, U.K.
- McCarty, D. J., E. S. Tull, C. S. Moy, C. K. Kwoh, and R. E. LaPorte. 1993. Ascertainment corrected rates: applications of capture-recapture methods. *International Journal of Epidemiology* **22**:559-565.
- Meadows, D. W., and C. M. Meadows. 2003. Behavioral and ecological correlates of foureye butterflyfish, *Chaetodon capistratus*, (Perciformes : Chaetodontidae) infected with *Anilocra chaetodontis* (Isopoda : Cymothoidae). *Revista de Biologia Tropical* **51**:77-81.
- Nichols, J. D., J. E. Hines, K. H. Pollock, R. L. Hinz, and W. A. Link. 1994. Estimating breeding proportions and testing hypotheses about costs of reproduction with capture-recapture data. *Ecology* **75**:2052-2065.
- Nichols, J. D., J. E. Hines, J. R. Sauer, F. W. Fallon, J. E. Fallon, and P. J. Heglund. 2000. A double-observer approach for estimating detection probability and abundance from point counts. *Auk* **117**:393-408.
- Nichols, J. D., W. L. Kendall, J. E. Hines, and J. A. Spindel. 2004. Estimation of sex-specific survival from capture-recapture data when sex is not always known. *Ecology* **85**:3192-3201.
- Otis, D. L., K. P. Burnham, G. C. White, and D. R. Anderson. 1978. Statistical inference from capture data on closed animal populations. *Wildlife Monographs* **62**:1-135.
- Pollock, K. H. 1982. A capture-recapture design robust to unequal probabilities of capture. *Journal of Wildlife Management* **46**:752-757.

- Pollock, K. H., J. D. Nichols, C. Brownie, and J. E. Hines. 1990. Statistical inference for capture-recapture experiments. *Wildlife Monographs* **107**:1-97.
- Pradel, R., J. E. Hines, J.-D. Lebreton, and J. D. Nichols. 1997. Capture-recapture survival models taking account of transients. *Biometrics* **53**:60-72.
- Ralph, C. J., S. Droege, and J. R. Sauer. 1995. Managing and monitoring birds using point counts: standards and applications. Pages 161-168 *in* C. J. Ralph, J. R. Sauer, and S. Droege, editors. *Monitoring bird populations by point counts*. U.S. Forest Service General Technical Report PSW-CTR-149.
- Royle, J. A., and J. D. Nichols. 2003. Estimating abundance from repeated presence-absence data or point counts. *Ecology* **84**:777-790.
- Salkeld, D. J., and L. Schwarzkopf. 2005. Epizootiology of blood parasites in an Australian lizard: a mark-recapture study of a natural population. *International Journal for Parasitology* **35**:11-18.
- Samuel, M. D., and K. H. Pollock. 1981. Correction of visibility bias in aerial surveys where animals occur in groups. *Journal of Wildlife Management* **45**:993-997.
- Samuel, M. D., E. O. Garton, M. W. Schlegel, and R. G. Carson. 1992. Visibility bias during aerial surveys of elk in northcentral Idaho. *Journal of Wildlife Management* **51**:622-630.
- Schaub, M., O. Gimenez, B. R. Schmidt, and R. Pradel. 2004. Estimating survival and temporary emigration in the multistate capture-recapture framework. *Ecology* **85**:2107-2113.
- Seber, G. 1982. *The estimation of animal abundance and related parameters*. Charles Griffin and Company, London, U.K.
- Senar, J. C., M. J. Conroy, L. M. Carrascal, J. Domenech, and I. Mozetich. 1999. Identifying sources of heterogeneity in capture probabilities: An example using the great tit, *Parus major*. *Bird Study Supplement* **46**:248-252.

- Senar, J. C., and M. J. Conroy. 2004. Multi-state analysis of the impacts of avian pox on a population of Serins (*Serinus serinus*): The importance of estimating recapture rates. *Animal Biodiversity and Conservation* **27**:133-146.
- Steinhorst, R. K., and M. D. Samuel. 1989. Sightability adjustment methods for aerial surveys of wildlife populations. *Biometrics* **45**:415-425.
- Stentiford, G. D., D. M. Neil, and R. J. A. Atkinson. 2001. Alteration of burrow-related behavior of the Norway lobster, *Nephrops norvegicus* during infection by the parasitic dinoflagellate *Hematodinium*. *Marine and Freshwater Behavior and Physiology* **34**:139-156.
- Tracey, J. P., P. J. S. Fleming, and G. J. Melville. 2005. Does variable probability of detection compromise the use of indices in aerial surveys of medium-sized mammals? *Wildlife Research* **32**:245-252.
- Tuytens, F. A. M., D. W. MacDonald, R. Delahay, L. M. Rogers, P. J. Mallinson, C. A. Donnelly, and C. Newman. 1999. Differences in trappability of European badgers *Meles meles* in three populations in England. *Journal of Applied Ecology* **36**:1051-1062.
- Van Riper III, C. I., S. G. Van Riper, and W. R. Hansen. 2002. Epizootiology and effect of avian pox on Hawaiian forest birds. *Auk* **119**:929-942.
- Weatherhead, P. J., and H. Greenwood. 1981. A critical assumption of band-recovery models may often be violated. *Wildlife Society Bulletin* **12**:198-199.
- Weatherhead, P. J., and C. D. Ankney. 1985. Condition bias and band recovery data: A reply to Burnham and Nichols. *Wildlife Society Bulletin* **13**:349-351.
- White, G. C., and K. P. Burnham. 1999. Program MARK: Survival estimation from populations of marked animals. *Bird Study* **46** Supplement:120-138.
- Williams, B. K., J. D. Nichols, and M. J. Conroy. 2002. *Analysis and Management of Animal Populations*. Academic Press, San Diego, USA.

Wobeser, G. 2002. Disease management strategies for wildlife. *Revue scientifique et technique (International Office of Epizootics)* **21**:159-178.

CHAPTER 3
MOVEMENT DYNAMICS, DEMOGRAPHIC RISK FACTORS, AND
HABITAT PARTITIONING IN AN AVIAN DISEASE SYSTEM

Abstract. I conducted a local scale study to investigate the potential risk factors associated with Mycoplasmal conjunctivitis infection in house finches (*Carpodacus mexicanus*), the effects of this disease on host movement dynamics, and how local spatial scale dynamics relate to regional scale patterns. I used multistate capture-mark-recapture (CMR) models to analyze a subset of banding data collected over three years in Ithaca, NY. In this study, I found that juvenile and female house finches in particular are at greater risk of having Mycoplasmal conjunctivitis (symptomatic). Symptomatic birds have both lower rates of local site-to-site movement and exhibit a lower likelihood of undergoing transient movements through the study area. My analysis suggests that adult finches have the greatest propensity for transient movements, which highlights the importance of this particular group as a potentially major contributor to the rapid spread of Mycoplasmal conjunctivitis infections in finches throughout the eastern US. This work demonstrates the ability of an infectious agent to alter the movement behavior of an avian host, and demonstrates that local scale subpopulations or groups exhibit considerable habitat partitioning likely as some function of the availability of roosting and feeding sites. I synthesize my findings with other studies of this host-pathogen system conducted at broader spatial scales, and comment on the correspondence between disease patterns and hypothesized driving processes.

Key-words: capture-recapture; *Carpodacus mexicanus*; disease; movement; *Mycoplasma gallisepticum*; risk factors; scale.

Introduction

The impacts of seasonal variation in demographic, biological, behavioral, and environmental components of a population can play a role in driving disease dynamics in wild animals (Dobson and Hudson 1995, Hosseini et al. 2006). Similarly, in many human diseases there are often marked patterns in disease incidence, which have been ascribed to changes in environmental conditions, host behavior, and physiological attributes (Dowell 2001). Many factors that may operate at different temporal and spatial scales contribute to the heterogeneous patterns of disease occurrence in animal populations. These factors can include biotic components of a host-pathogen system (e.g. age, sex, social structure, reproductive cycle) or abiotic components associated with the environment in which the host and pathogen live (e.g. environmental fluctuations, resource limitations). However, while it is a common practice to generate demographic profiles (e.g. age, sex) of disease prevalence (Pac and Frey 1991, Hartup et al. 2000, Hartup et al. 2001, Van Riper III et al. 2002), there is a paucity of information pertaining to whether such factors contribute to disease risk in wild animals (but see Altizer et al. 2004a).

Compounding these difficulties is the potential for heterogeneity or seasonal patterns in host movement. While the spatial distribution of hosts and pathogens is clearly an important consideration in the study of wildlife diseases (Bolker and Grenfell 1995, Mollison and Levin 1995, Hudson et al. 2002), it can be difficult to quantify and estimate the rate of movement of mobile hosts that have large home ranges. The rate of disease spread can be inferred in broad-scale disease monitoring projects, but these designs do not make it possible to attribute heterogeneity in movements to specific components of a host population. Movement of individuals within and between populations is clearly an important element to consider when

modeling a host-pathogen system, as host movements can influence the spread and maintenance of disease (Hudson et al. 2002). Heterogeneity in movement rates likely affects contact rates between infected and susceptible individuals, changing the rate of disease spread within and between disparate populations. In smaller scale studies, although more detailed information can be recorded about host population structure, oftentimes the scale, extent of sampling efforts, and traditional study design limits the inferences that can be made regarding host movements.

As such, methods for carrying out observational studies in wildlife disease ecology require careful thought. Aside from basic study design characteristics, which involve consideration of the target and sampled population, making inferences about a sampled population of animals requires robust methods that account for detectability of individuals. In many cases there is heterogeneity in the likelihood of capture for different subsets of a population and a multitude of biotic and abiotic factors, such as age, disease state, and site characteristics may contribute to this variation. A lack of consideration for animal detectability will likely lead to incorrect inferences about demographic parameter estimates, patterns in disease frequency, and ultimately the processes driving ecological patterns (Lebreton et al. 1992, Williams et al. 2002, Jennelle et al. 2007). Capture-recapture data collection and modeling are ideally suited for dealing with this complication in studies of wildlife disease ecology (Faustino et al. 2004, Senar and Conroy 2004), as a solid theoretical foundation supported by numerous empirical studies permits rigorous estimation of pertinent demographic parameters, while accounting for variability in the detection process (Pollock et al. 1990, Lebreton et al. 1992, Williams et al. 2002).

Since 1993-94, a novel strain of the bacterium *Mycoplasma gallisepticum* (MG) has become established as an endemic pathogen in eastern house finch (*Carpodacus mexicanus*; hereafter finch) populations. While other bird species can harbor the

pathogen, house finches are particularly susceptible to debilitating and lethal infections (for review, see Dhondt et al. 2005). Observable clinical symptoms of MG infection manifest as mild to severe conjunctivitis, which enables the disease to be readily tracked and studied in the wild.

In 1994 a broad-scale monitoring network (House Finch Disease Survey; HFDS) was established to track the spread of MG in finches across the eastern US (Dhondt et al. 1998), and has been used to make a number of discoveries about this host-pathogen system. Using HFDS and Christmas Bird Count data, Hochachka and Dhondt (2000) confirmed a causal relationship between declining finch abundance and Mycoplasmal conjunctivitis. Their work provided evidence for density-dependent finch mortality at a regional scale. Altizer et al. (2004b) examined regional trends in disease prevalence and found distinct bi-modal peaks in MG prevalence across the eastern US, which dampened with increasing latitude. Extending upon this work, Hosseini et al. (2004) found that the seasonal pattern of breeding and social aggregation in finches incorporating a latitudinal gradient and the effects of partial immunity can produce the observed regional patterns of MG prevalence as reported in Altizer et al. (2004b). Recently, Hosseini et al. (2006) used HFDS data to determine the direction (spatial) and rate (temporal) of spread of Mycoplasmal conjunctivitis in house finches. They found that dispersal movements of juvenile birds in July and asymptomatic carriers are likely to be important driving factors in the dynamics of this host-pathogen system at a large spatial scale.

An equally important factor in finch-MG dynamics is finch social structure. Hochachka and Dhondt (2006) examined differences in house finch group sizes across the landscape with HFDS data, and found there was no detectable correlation between group sizes and distance between groups. As such, they concluded that local site-

specific characteristics may play a significant role in structuring interacting groups or subpopulations of finches. While data from the House Finch Disease Survey (HFDS) has been used to make important contributions to understanding of Mycoplasmal conjunctivitis dynamics at a regional scale, there are few field studies published that make use of information about uniquely marked individuals at a local population scale.

Several local scale field studies of the finch-*Mycoplasma* system have been carried out, but they have not examined how the structure of populations in space interacts with host movements, and (with the exception of Faustino et al. 2004) have not fully utilized information on uniquely marked individuals to make inferences about host demography. The majority of field studies of this host-pathogen system have been carried out in the northeastern US and began with a general health survey in Tompkins County, NY to assess mycoplasmal conjunctivitis prevalence in house finches (Hartup et al. 2000). In a similar study conducted in New Jersey, Hartup et al. (2001) used raw count data to estimate seasonal prevalence, and found no differences in the proportion of diseased finches stratified by age or gender. Faustino et al. (2004) were the first to use multistate capture-recapture models to examine seasonal dynamics of house finch survival, recapture, infection, and recovery in Tompkins County, NY. While in a study conducted in Georgia, Altizer et al. (2004a) suggested that juvenile finches are at higher risk of MG infection compared with adults, supporting the notion that annual pulses of juveniles during the summer breeding season contribute a sufficient number of susceptible individuals to fuel autumn resurgence of conjunctivitis in finches. Since these studies (except Faustino et al. 2004) did not account for heterogeneous detection probabilities of individuals, inferences based upon raw data may reflect completely spurious artifacts of heterogeneity in detection of individuals rather than process drivers (Jennelle et al. 2007).

Despite a number of field studies, empirical work at a local scale (which accounts for detection heterogeneity) is still needed to confirm the mechanisms for spread and maintenance of MG among house finch populations. Although broad-scale monitoring programs like the HFDS permit the assessment of regional trends in MG dynamics of finches, information pertaining to individuals cannot be collected and there is considerable variability in the detection process along temporal and spatial gradients, as well as among observers. Furthermore, with the exception of Faustino et al. (2004), field studies of this disease system to date have not used a capture-recapture modeling approach to account for variation in detectability of individuals when testing hypotheses and estimating demographic parameters and disease frequency statistics. In order to further our understanding of the impacts of disease on wildlife populations and the risk factors that facilitate spread and maintenance within and between populations at a local scale, I extend upon the work carried out in Faustino et al. (2004).

The house finch is highly gregarious with pronounced seasonal variation in social structure (Hill 1993). Eastern house finches, unlike their native counterparts in the west, have become partial migrants and evidence suggests that young birds exhibit a stronger tendency for this recently evolved behavior (Able and Belthoff 1998). Typically in the autumn months, groups of juvenile individuals will form large nomadic flocks, and in winter and spring while loose aggregations of finches continue to mix, pair bonding occurs. Complex social and migratory behaviors, along with seasonal variation in population structure and movement propensity are likely to interact in driving the dynamics of Mycoplasmal conjunctivitis in house finches. As such, I sought to address whether there was significant seasonal variation in i) the role of demographic factors (age, gender, and disease state) and spatial structuring on local-scale movements and ii) risk factors (age and gender) associated with MG

infection, and how these components might interact to explain the observed pattern of MG dynamics in the local study area.

Methods

Data Collection and Study Area

A detailed description of the data collection techniques and study area is presented in Faustino et al. (2004), so here I will only highlight pertinent details relevant for this paper.

The data used in this paper are part of an intensive capture-mark-recapture (CMR) study in Ithaca, NY carried out from August 2000 to March 2005. I excluded data from the first year of study (2000-2001) and used only the data from January 2002 through December 2004, since data collection efforts over this time were standardized with weekly trapping and resighting periods at two fixed sites. Data were analyzed from 1 September through 15 April and within each year the dataset was divided into a Fall-Winter (FW: Sep-Dec) and Winter-Spring (WS: Jan-Apr) subset, each spanning 8 to 15 weeks. This was done to both reduce the number of parameters to be estimated in CMR models and to examine variability in seasonal demographic structure, which coincides with significant changes in house finch social structure and behavior (Hill 1993).

Encounter data were obtained from two different types of events, (i) physical recaptures (trapping), and (ii) live resightings of marked individuals. Physical recaptures (via mist nets and cage traps) were conducted under permits from the New York State Department of Environmental Conservation, the U.S. Fish and Wildlife Service, and the U.S. Geological Survey. All procedures involving live animals were implemented under the Animal Use Protocol #00-90 issued by the Cornell University

Institutional Animal Care and Use Committee. On each trapping occasion, birds were captured using a combination of two or three hand-built cylindrical wire-mesh cage traps and two or three 30mm mist-nets. Each newly captured bird was fitted under permit with a unique sequence of a 9-digit numbered aluminum leg band (Bird Banding Laboratory, Laurel, Maryland, USA) and a combination of three colored plastic leg bands. Standard demographic and morphological measurements were recorded (Pyle 1997) including age (finches were assigned as juveniles if it was their first winter, and adults if it was at least their second winter), gender, as well as disease state. Individual birds were assessed for disease status by recording the severity of conjunctivitis in both eyes at each encounter, using a binary ranking: ‘I’ (symptomatic) indicating some level of the disease, or ‘U’ (asymptomatic). The presence of conjunctivitis for assessing MG infection status correlates highly with PCR analysis of eye swab (conjunctival tissue) samples and culture for MG organisms (Hartup et al. 2001). For resighting events, a vehicle was used as a blind and a spotting scope and/or binoculars were used to resight birds. The color-band combination, gender, and disease state were recorded for each resighted bird. After 2003, resighting was conducted twice per week at each of the two banding sites.

Trapping and resighting sessions occurred two days per week, with trapping conducted every Tuesday and Wednesday on the two primary study sites (‘Golf course’ and Liddell Field Station known as ‘Beelab’), while resighting was conducted every Thursday and Friday (Appendix A). The current investigation is based solely on these two sites as they were consistently maintained over the study period. Although both sites are representative of suburban landscapes, the Golf course is composed of a matrix of neatly mowed golf greenways, interspersed with spruce (*Picea* spp.) trees, early successional scrub habitat, and housing developments. The Beelab on the other hand is surrounded by a matrix of agricultural fields and deciduous forest cover, with

fewer surrounding suburban developments. While these sites are approximately 1.5 km apart, they are oriented almost exactly along an east-west gradient. Overall, the Golf course is much closer in proximity to suburban housing developments as compared to the Beelab. This can be inferred by the density of roads in proximity to each field site (Appendix A).

I maintained and stocked tube-style feeders with black oil sunflower seeds at all trapping and resighting sites to attract finches. The use of baited stations is known to lead to potential bias in cases where baiting induces greater likelihood of encountering previously captured individuals than expected by random chance (Pradel 1993, Williams et al. 2002). 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 significant trap effects due to the use of baited feeders with respect to subsequent visual resighting encounters. However, I cannot rule out an influence of physical trapping on encounter histories in the data. Since there were many fewer live captures of finches relative to resightings, I believe this should not induce substantial bias in parameter estimates.

Analysis of Live Encounter Data

Live encounter data were analyzed using a multistate capture-recapture approach (*sensu* Faustino et al. 2004, Williams et al. 2002, and 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 (Arnason 1973, Schwarz et al. 1993). Such models allow for robust estimation of transition probabilities among physical sites in this case, under conditions where the probability of observing an individual on a particular sampling occasion is < 1 .

Under the assumption that survival from time i to $i+1$ depends only on the physical 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 $i+1$

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

$$\phi_i^{rs} = S_i^r \psi_i^{rs}$$

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 $i+1$.

Despite a reasonable number of observations of marked individuals (Appendix B), multistate models are typically “data hungry” (Williams et al. 2002), therefore I pooled the data into 7-day periods to increase estimator precision because most sampling events occurred on a weekly basis. Therefore, estimates reflect *weekly* apparent survival, detection, and site transition probabilities within a given year and season (FW or WS). A standard assumption for capture-recapture studies requires that capture/release sessions occur instantaneously, which translates to mean that there are no gains or losses to the sampled population during capture periods. Violation of this assumption typically results in the sample of marked individuals exhibiting some degree of heterogeneity in survival probabilities within capture occasions (Lindberg and Rexstad 2002). While pooling data into 7-day periods may violate the instantaneous release assumption, at worst this will introduce some level of overdispersion, which can be accounted for by a variance inflation factor (below).

Pooling data reduces the number of parameters in a given model and thus improves parameter precision, but it creates difficulty in assigning an ‘average state’. In my case I consider two binary states, location (Beelab and Golf course) and disease (symptomatic and asymptomatic), but I account for variation in estimated parameters as functions of these states quite differently.

Ideally, I would have assigned four states in the modeling process (a state representing each disease and location class); however, due to data constraints I organized the encounter histories to reflect only variation in location-specific states. Because the majority of the encounter histories for birds included only one sighting per 7-day period (fewer than 10% of all birds in the data were observed more than once in a given 7-day period) I used a first-encounter approach (*sensu* Faustino et al. 2004) 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 a daily encounter history of ‘00B00G0’ over a particular 7-day period, where **B** = Beelab, **G** = Golf course and **0** = not encountered, the state assigned for this 7-day period would be ‘Beelab’ or ‘**B**’) (Hargrove & Borland 1994). The one-week pooling interval chosen was acceptable in my study as very few observations of birds in different location states occurred within a given sampling period.

To account for variation in survival, detection, and movement due to disease state, I assigned each bird a binary covariate indicating whether it was observed with conjunctivitis in a given season. Since finches make observable transitions between disease states over the course of a given season, I was concerned that not accounting for a finer grain (for a review of ecological scale, see Peterson and Parker 1998) in temporal scale might result in indistinguishable state-specific differences in estimated parameters, thereby reducing the power in the dataset to detect real state-specific

effects. To evaluate the potential loss in inferential power associated with assigning a binary state-specific covariate, I performed CMR analyses (see details below) using two types of individual covariates: (1) a binary covariate indicating whether a finch was observed with conjunctivitis in a given season as mentioned above and (2) a covariate accounting for observable disease state heterogeneity within a given season, estimated as the proportion of observations that a finch was observed in the symptomatic state. Thus, if an individual was observed three out of four different occasions during a season in the symptomatic state, then I assigned this covariate as 0.75. Using both formulations of individual covariate, I found that there were negligible differences (quantitatively and qualitatively) in the parameters of interest and AIC ordering of models (see section on Model Selection below). Therefore, I report results based upon use of the binary covariate. While there is likely to be some loss of inferential power to assess disease state-specific differences in parameter estimates by not accounting for weekly disease state transitions, at worst the differences in reported parameters will be underestimating true heterogeneity due to variation in disease state.

Under the general Arnason-Schwarz model (Arnason 1973, Schwarz et al. 1993), partitioning φ into movement (ψ) and survival (S) generally assumes that timing of movement of individuals among states is known (typically at the beginning or end of an interval), and is equivalent among all individuals. Due to sparseness of data in this study, I was forced to pool encounter data as described earlier. In so doing, the time between a live encounter of an individual during interval (i) and the next potential encounter of that individual in interval ($i+1$) is a random variable. While it is possible to model movement that is completely random, with a uniform distribution (equivalent to an uninformative prior expectation of the distribution of individual movements; Joe and Pollock 2002), this assumes that the interval is fixed for all individuals, which is

unlikely to be the case in this study. In cases where movements do not occur at the same time among individuals, estimates of movement rates from the general Arnason-Schwarz model are likely to exhibit negative covariance among states, especially when movement rates are high (Joe and Pollock 2002). However, estimator bias has been found to be generally low, and I believe that as long as the probability of encounter with individuals in either state is random with respect to timing during the interval (although it may differ among states), estimates will be qualitatively robust. To account for uncertainty in my estimates due to pooling, and to minimize the chances of making a Type II error, I adopted a conservative strategy for evaluating and adjusting for lack of fit of the general models (cf. *Model selection* – GOF testing).

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 physical states from time i to $i+1$ is dependent only on its state at time i (i.e., - there is no ‘memory’ in these multi-state models). However, in the context of this study, it is possible that the probability of a bird making a transition between states is not only dependent on its state at time i , but also at its state at time $i-1$, or $i-2$ and so on. However, the data were not sufficient to model state transitions as a higher-order Markov process (‘memory models’, *sensu* Hestbeck et al. 1991).

Multistate modeling also assumes that individual state can be assigned with complete certainty upon each individual encounter. In this study, there was negligible uncertainty as to whether or not a bird was encountered at a given spatial location unless an error in transcription of a record occurred. Yet, there was uncertainty in assigning disease state, age class, and gender in some instances. In most cases I was able to assign disease scores for both eyes of an individual, but during resighting events this was not always possible as a bird’s orientation on a feeder might preclude

observation of one or both eyes. As such, these individuals were classified in the unknown health state category and were omitted from subsequent analyses. Likewise, it was not always possible to accurately assess the age of individuals upon capture, especially from November through April after skull ossification of first-year finches neared completion. Birds whose age could not be assessed with certainty were omitted from analyses; however, if these birds were recaptured or resighted in subsequent banding seasons, they were included in the adult group. In very few cases was I unable to determine the gender of finches in the selected dataset. House finches are a sexually dimorphic species with males displaying strong washes of red to yellowish coloration (depending upon diet) on the face, breast, and rump, while females typically lack strong coloration (Hill 1993). A few juveniles that had not completed their pre-basic molt were captured once and never again in September preventing me from assigning gender with certainty, so these individuals were also excluded from analyses.

To assess the effect of transient individuals on apparent survival probabilities, I compared the relative fit of ‘time-since-marking’ (TSM) ultra-structural models with standard Cormack-Jolly-Seber (CJS) models (note that TSM models are structurally equivalent to what are commonly referred to as ‘age-models’; *sensu* Pollock 1981. I adopt the TSM convention to prevent confusion with true chronological age). These models permit separate estimation of apparent survival probability of newly captured individuals (potentially containing transients, or birds seen only once and never seen again) and the apparent survival probability of individuals assumed to be residents (i.e., birds recaptured or resighted at least once after the initial marking event; Brownie and Robson 1983; Pradel et al. 1997; Cilimburg et al. 2002). Since multistate models have not been previously applied to study of transience, I performed a preliminary simulation to determine the influence of transient individuals on estimated survival, encounter, and transition probabilities. Consistent with expectations from

single-state models, I found that the presence of transient individuals only negatively biased survival estimates (equal to the proportion of transients simulated in the population), while encounter and transition probabilities were not affected. Thus, I applied the TSM structure to the survival parameter (S) only. Following Pradel et al. (1997), I derived an *ad hoc* estimate of the proportion of residents in the newly marked sample for each 7-day period by dividing the apparent survival probability for each age class and disease state estimated during the *first period following marking for a given release cohort* (where the sample is presumed to contain both residents and transients) by the apparent survival for that age and state estimated *for the same interval from previous release cohorts*; estimates from previous release cohorts will be at least 1 period after marking, and are assumed to consist entirely of residents. I then obtained the proportion of transients as (1 - the calculated proportion of residents). The proportion of transients was used in conjunction with estimates of transition probabilities between sites to make inferences about movement patterns of finches.

Model Structure

The physical sites chosen in this study represent locations within the study area where house finches could be reliably and consistently captured. I used CMR data to test for an effect of transience and estimate apparent survival, detection probabilities (used in estimation of disease prevalence and age ratios; details below), and site transition probabilities. In particular, I considered sources of variation that I expected might strongly influence the dynamics of this system including the factors of age (juvenile or adult), gender, site (Beelab or Golf course), and disease state (symptomatic or asymptomatic). While weekly variation in the values of estimated parameters is likely, I only consider seasonal differences in the temporal dimension.

My CMR model set consisted of 30 *a priori* competing models composed of different functional relationships between the factors of age, gender, site, and disease

state, including an effect of transience in the survival component of the models. I expected there to be significant variation in apparent survival probability between finches stratified by disease state given recent work by Faustino et al. (2004), so for each model considered, I included an additive disease effect. The model set for apparent survival included a disease effect only, and all single effect and two-way interactions between age, gender, and site. These models were tested with and without a transience effect. For the WS 2002 season, neither a disease effect nor transient structure could be accommodated by the data, so I removed models including these effects from the set. For detection and site transition probabilities, respectively, I tested eight model structures, which included all two-way and three-way interactions between site, age, and gender. I expected site variation in these parameters, so a site factor was included in each competing model. Each of these model structures was in turn tested with and without an additive disease effect. I was careful to ensure equal representation of each factor in the model set to protect against the risk of model redundancy, which results in artificially higher weighting (importance) to factors that are more represented in the model set.

While I made every effort to limit the *a priori* model set using prior knowledge of this system, I had to conduct CMR analyses in a piece-wise or hierarchal fashion. I first modeled variation in apparent survival, while maintaining the most general (most parameterized) structure for detection and site transition probabilities. In modeling detection probability, I used the most parsimonious model structure from the survival analyses, and maintained a general structure for site transitions. Likewise, when modeling site transition probabilities, I maintained a structure for apparent survival and detection probability based upon the most parsimonious model obtained for these parameters. Ideally, it would be optimal to analyze all possible combinations of the *a priori* model structures considered in each parameter, however, this would require

preparing and analyzing 896 model structures. While I recognize that this approach does not account for all model selection uncertainty in the parameters that are held constant, I believe this approach strikes a balance between computational tractability and rigorous model selection. This scenario is becoming more common in fact, and other workers have used a similar approach (Sandercock and Jaramillo 2002, MacKenzie 2006).

Model Selection

I used a median \hat{c} procedure (Cooch and White 2006) to estimate an overdispersion correction factor for my most general models (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).

I fit models to the data using program MARK (v. 4.3; White and 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 and 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 and Anderson 2002). The best model was that with the lowest QAIC_c values, and other models were ranked relative to deviations from the best model (Δ 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 and Anderson 2002). The ratio of w_i between any two models indicates the relative (proportional) difference between those two models.

When model selection is based on information-theoretic approaches (e.g., AIC), it is inappropriate to express differences among models in terms of nominal alpha levels

(i.e., *P*-values). For example, using AIC to rank models in the candidate model set, and then test (using a likelihood ratio test or equivalent) whether the best model is significantly better than the second best model, would be incorrect (since these classical tests are invalid if models are ranked by AIC; Royall 1997; Burnham and Anderson 2002; Anderson and Burnham 2002). Thus, I report comparisons among models in terms of relative degrees of support in the data.

For robust parameter estimates, I accounted for conditional model selection uncertainty (conditional on fixed model structures for parameters not under consideration; see above) by calculating an average value for a parameter (apparent survival, detection, or state transition probability) over all relevant (structurally consistent) models in the candidate set, weighted by normalized QAIC_c model weights.

With model-averaged parameter estimates of detection probability, I estimated both disease prevalence and the proportion of age/gender classes in each season of a given year. In almost all previous studies of wildlife diseases using marked individuals, group-specific detection probabilities are not accounted for when estimating prevalence and age/gender structure. I present estimates of time specific disease prevalence and seasonal age/gender proportions, adjusted for differences in detection rate (*sensu* Jennelle et al. 2007).

Risk Factor Analysis

To assess whether finch age and gender are significant risk factors associated with the presence of conjunctivitis, I evaluated four *a priori* defined models using logistic regression. I modeled presence or absence of conjunctivitis as a function of i) age interacting with gender, ii) age additive with gender, iii) age only, and iv) gender only. To evaluate the strength of each model, I ranked models using AIC, with models having lower AIC values given more inferential weight. I evaluated the goodness-of-

fit of each model using deviance and Pearson GOF statistics and conducted all analyses using SAS, v.9 software (SAS institute). Since detection probabilities varied between age, gender, disease state, site, and season, using raw counts of individuals could bias inferences about risk factors. To account for variable detection, I corrected raw counts for group-specific detection probabilities and used these frequency values as input data for the logistic regression by season and year. Since there can be considerable uncertainty in the underlying detection process, I weighted each age/gender/disease state group by the inverse of the variance for detection probabilities of each respective group. This gives greater weight to detection-corrected frequencies of individuals associated with less uncertainty in the detection process.

Analysis of Telemetry Data

To complement the banding component of the study, I attached 62 Holohil radio telemetry transmitters to both symptomatic and asymptomatic finches between November 2001 and March of 2002; however data from only 38 birds was usable due to unstandardized tracking protocols for several release cohorts (24 birds). Transmitters had an expected lifespan of 3-weeks, so from the day of release I attempted to obtain daily relocations of each bird within the Ithaca study area. Upon obtaining a reliable transmitter signal, I used triangulation and visual confirmation (if possible) to determine the location of each finch. Geographic positions (UTM coordinates) of finch relocations were recorded with a Global Positioning System unit (GPS Path-finder Pro XR, Trimble Navigation Ltd., Sunnyvale, California). Tracking was conducted between 07:20 and 15:45 on a given day, and finches were relocated randomly with no specific ordering assigned to tracked individuals. The disease state of each bird was determined both at initial capture and when relocations afforded a visual assessment of an individual. I assumed that disease state did not change

appreciably and that associated long-distance movements did not occur over the course of the three-week transmitter lifespan.

With this subset of data, I sought to address whether, i) birds marked and released at each release location were clustered with respect to daytime usage of the available habitat, ii) there was a difference in the dispersion between birds as a function of release location, and iii) there was a difference in the dispersion between asymptomatic and symptomatic finches. To address these questions, I used program BLOSSOM (Cade and Richards 2001) to implement a multi-response permutation procedure (MRPP). For each finch, I calculated the UTM coordinates for their respective multivariate medians. These coordinates were subsequently used in the MRPP procedures.

Results

Over the course of this study, I used information from 666 initial captures, 709 recaptures, and 5357 resightings to produce 1674 house finch encounter histories (Appendix B). The main limitation of this study system was obtaining sufficient encounters of symptomatic finches to partition variation in parameters as functions of disease state. As such, I was only able to impose a relatively simple structure on the model set with respect to partitioning variation in apparent survival, encounter, and state transition probabilities.

For each fall-winter season, the combined AIC weight for models including a transience structure (TSM) approached 100%, strongly supporting a transience effect in the data (Table 3.1). The data suggested only moderate support in the WS 2003 and WS 2004 seasons, as combined AIC weight for a transience effect was 25% and 67%, respectively (Table 3.1). There was not substantial support in the data for a gender

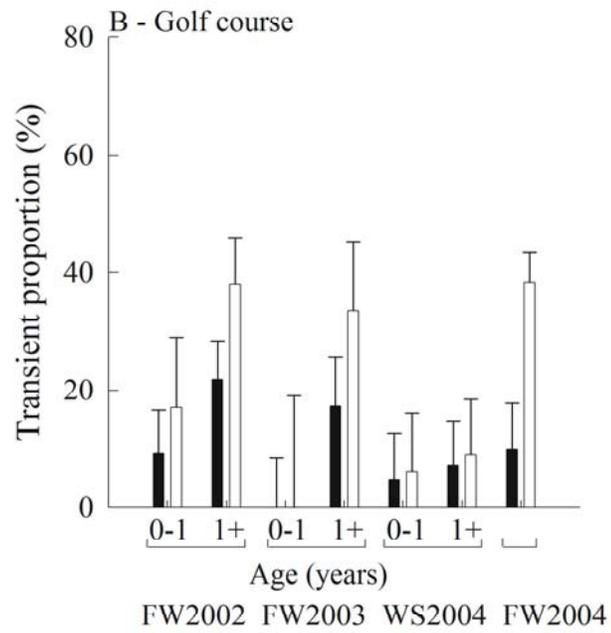
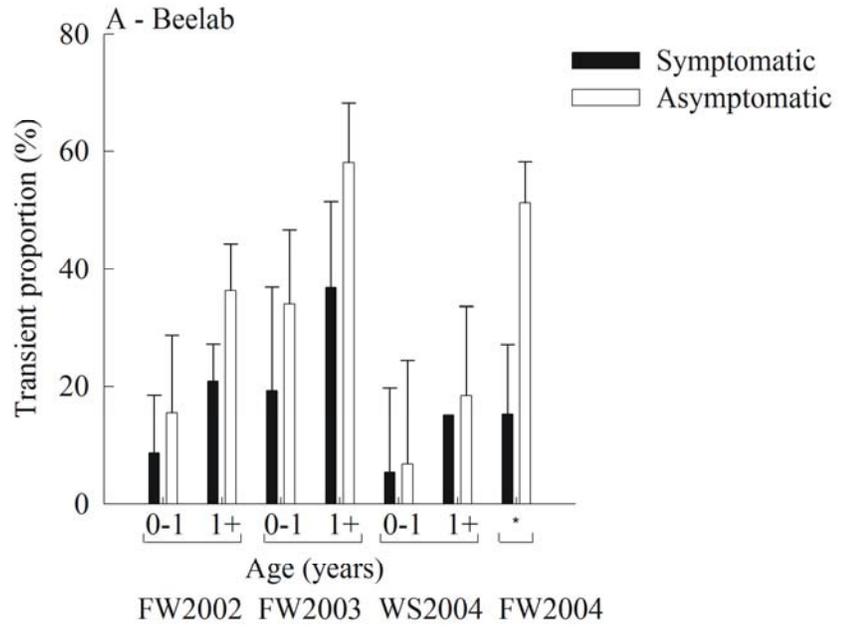
effect in transience structure as the greatest combined AIC weight with this effect was less than 10% for a given season (Table 3.1). Model averaged estimates of group-specific apparent survival in the period following marking (first TSM class) were lower than estimates in subsequent periods (i.e., second TSM class) suggesting that the first TSM class included a mixture of transient and resident individuals. Although the estimated proportion of transients was greater in FW seasons, general patterns emerged across seasons. There were consistently lower proportions of transients in the marked sample of symptomatic compared to asymptomatic finches and consistently greater proportions of transients in the marked sample of adult versus juvenile finches (Fig. 3.1).

Regarding resident apparent survivorship, there was a consistent pattern in the influence of age; with the exception of FW 2004, there was greater support in the data for an age effect during FW versus WS seasons (combined AIC weights: WS 2002 =26%, FW 2002=100%, WS 2003=20%, FW 2003=99%, WS 2004=47%, and FW 2004=4%) (Table 3.1). During FW seasons survival of adults was greater than juvenile finches, with no apparent differences during WS seasons (Appendix C). The influence of site on survivorship varied by season and year with support ranging from 8% for WS 2002, 31% for FW 2002, 17% for WS 2003, 79% for FW 2003, 55% for WS 2004, and 78% for FW 2004; however, no pattern emerged between sites. There was little influence of gender on survivorship. The most support for a gender effect occurred in the WS 2002 season, with combined AIC weight of 22% (Table 3.1), however, this had negligible influence on model averaged survival estimates. Counter to my expectations, resident apparent survival of symptomatic finches was consistently higher than that of asymptomatic finches across seasons, age groups, and sites (with the exception of WS 2003, and adult finches in FW 2003) (Appendix C). This difference in survival was greatest during the FW seasons.

Table 3.1. Summary of multistate analysis of live encounter data for 2002-04 in Ithaca, NY, USA. Each year is divided into subsets: *FW* = ‘Fall-Winter’ (Sep-Dec) and *WS* = ‘Winter-Spring’ (Jan-Mar for 2002; Jan-Apr for 2003-04), and each period spans seven days over which capture and resighting data were pooled. *Model notation:* Φ , survival; p , recapture; ψ , state transition; *Age* (a), juvenile or adult; *Disease* (d), asymptomatic or symptomatic; *Site* (s), Beelab or Golf Course; and *Gender* (g). Interaction effects indicated with ‘*’ sign. Lower ΔQAIC_c values show better fit. Only models comprising $\geq 95\%$ of QAIC_c weight are listed in decreasing order of parsimony. The Akaike weights indicate the relative support for each model, given the data. The Deviance is the difference in $-2 \cdot (\log \text{Likelihood})$ between a selected model and the saturated model (a model with the number of parameters equal to the sample size). Estimated corrections for overdispersion (\hat{c}) for each year and season starting from WS 2002 include 1.20, 1.09, 1.26, 1.10, 1.26, and 1.18, respectively.

Model components		Model Statistics				
Season	Φ, p, ψ	QAICc weight (%)	Δ QAICc	Deviance	Parameters	
<i>WS</i> ^a 2002	(.), (s), (s)	42.13, 74.85, 75.14	0.00, 0.00, 0.00	312, 318, 318	17, 10, 11	
	(a), (s*a), (s*g)	25.51, 17.16, 16.15	1.00, 2.95, 3.08	310, 316, 316	18, 12, 13	
	(g), (s*g), (s*a)	21.95, 07.68, 08.12	1.30, 4.55, 4.45	310, 318, 317	18, 12, 13	
	(s), (s*a*g), (s*a*g)	07.59, 00.32, 00.59	3.43, 10.94, 9.70	313, 314, 312	18, 16, 17	
<i>FW</i> 2002	M2(a+d), (s*g)+d, (s*a)	66.54, 52.62, 56.16	0.00, 0.00, 0.00	2763, 2770, 2763	23, 19, 18	
	M2(s*a)+d, (s*a*g)+d, (s*a)+d	30.54, 39.74, 25.71	1.56, 0.56, 1.56	2756, 2763, 2763	27, 23, 19	
	M2(a*g)+d, (s+d), (s)	02.74, 05.71, 10.51	6.38, 4.44, 3.35	2760, 2779, 2771	27, 17, 16	
	(s*a)+d, (s*a)+d, (s+d)	00.09, 01.93, 03.75	13.25, 6.61, 5.41	2776, 2777, 2771	23, 19, 17	
<i>WS</i> 2003	(d), (s*a), (s*a)+d	31.83, 31.00, 31.95	0.00, 0.00, 0.00	1020, 1024, 1024	15, 11, 09	
	M2(d), (s), (s+d)	13.94, 22.88, 18.29	1.65, 0.61, 1.12	1019, 1029, 1027	16, 09, 08	
	(g+d), (s*a*g), (s*g)+d	12.89, 11.17, 15.46	1.81, 2.04, 1.45	1019, 1020, 1025	16, 14, 09	
	(s+d), (s*a)+d, (s*a)	11.75, 10.76, 11.97	1.99, 2.12, 1.96	1020, 1024, 1026	16, 12, 09	
<i>M2</i> (a+d), (s+d), (s)	(a+d), (s*g), (s*a*g)+d	10.92, 08.89, 07.78	2.14, 2.50, 2.83	1020, 1027, 1022	16, 11, 11	
	(s*g)+d, (s*a*g)+d, (s*g)	09.23, 08.06, 06.10	2.48, 2.69, 3.31	1018, 1029, 1029	17, 10, 08	
	M2(g+d), (s*g)+d, (s*a*g)	04.65, 04.01, 05.45	3.85, 4.09, 3.54	1017, 1020, 1027	18, 15, 09	
	M2(g+d), (s*g)+d, (s*a*g)	01.89, 03.23, 03.01	5.65, 4.52, 4.72	1019, 1027, 1024	18, 12, 11	

Figure 3.1. Estimated weekly proportion of potential transients (± 1 SE) in the banded sample of house finches from Ithaca, NY, USA for the Fall-Winter (FW) seasons of 2002 and 2003 and Winter-Spring (WS) season of 2004, stratified by site (A – Beelab, B – Golf Course), age (juvenile (0-1), adult (1+)), and disease state (symptomatic ■, asymptomatic □). Transience estimates are based on the ratio of estimated apparent survival probabilities of the first TSM class to the second TSM class. *Notation:* Φ ; TSM=1 refers to the estimate for weekly apparent survival following the first release within a season (these are assumed to be a mixture of residents and transient individuals), and TSM=2 refers to the estimate of weekly apparent survival for finches captured ≥ 1 week after initial capture within a season (I assume these individuals to be residents within a season). In FW 2004, there was no variation in the transient proportion as a function of age (indexed with a “*”).



The factors important for explaining variation in detection probability between sites varied across seasons and years, but were generally some combination of the influence of disease state, age, and gender. Aside from site-specific differences, which I expected, combined AIC weights for models containing an age effect were 17%, 42%, 57%, 47%, 14%, and 100% for each season and year, respectively (Table 3.1). Overall, there was no general pattern with respect to the influence of age on group-specific detection probabilities. Combined AIC weights for models containing a gender effect were 8%, 93%, 27%, 5%, 54%, and 31% for each season and year, respectively (Table 3.1). Again, there was no consistency in the pattern of gender-specific detection probabilities. Total AIC weights for models containing a disease effect (from FW 2002 to FW 2004) were 100%, 27%, 42%, 42%, and 70% for each season and year, respectively (Table 3.1). In FW 2002, symptomatic finches had greater detection probabilities than asymptomatic finches, but this trend appeared to be reversed in FW 2004 (Appendix D). Estimates of detection probability were model averaged for each season and used to subsequently correct for the relative frequencies of each age/gender/disease state group in calculations of disease prevalence and age/gender proportions.

There were several consistent patterns in movement probabilities between sites. In general, movement probabilities were greater for finches making a transition from the Beelab to the Golf course (Fig. 3.2). The influence of age with respect to movement probabilities varied with combined AIC weights of 9%, 82%, 55%, 17%, 30%, and 100% for each season and year, respectively (Table 3.1). When age contributed to substantial variation in movement (FW 2002, WS 2003), the probabilities of site-to-site movement of juvenile finches were for the most part greater than those of adults (Fig. 3.2). Combined AIC weights for models containing a gender effect were 17%, 0%, 32%, 7%, 27%, and 92% for each season and year, respectively (Table 3.1);

however, no consistent pattern emerged in FW 2004 when there was substantial support in the data for this effect (Fig. 3.2). Total AIC weights for models containing a disease effect were (from FW 2002 to FW 2004) 29%, 74%, 70%, 33%, and 69% for each season and year, respectively (Table 1). When disease state contributed to substantial variation in movement (WS 2003, FW 2003, and FW 2004), symptomatic finches exhibited lower movement probabilities than asymptomatic finches (Fig. 3.2).

Using counts of finches stratified by site, age, gender, and disease state and corrected for group-specific detection probabilities, I estimated disease prevalence (Fig. 3.3) and the age/gender proportions (Appendix E) of finches stratified by season and site. Since site-specific estimates of prevalence were qualitatively similar, I combined data across both sites. Corrected disease prevalence did not exceed 42% over the course of the study and with the exception of the 2001-02 seasons (which exhibited a unimodal distribution) tended to exhibit a bimodal distribution (Fig. 3.3). Given significant variation in weekly estimates of age and gender proportions, I chose to estimate these statistics over the course of an entire season. There were two consistent trends that emerged from the data. Namely, there were greater proportions of juvenile compared with adult finches in the FW seasons (at the Beelab location), with an inverse trend during WS seasons. In addition, the proportion of females declined between FW and WS seasons of each year.

Figure 3.2. Estimated weekly site transition probabilities (± 1 SE) of house finches stratified by disease state, age (juvenile = J and adult = A), gender (F = female and M = male), and site from 2002-2004 in Ithaca, NY; asymptomatic finches/Beelab to Golf course (○), symptomatic finches/Beelab to Golf course (●), asymptomatic finches/Golf course to Beelab (▽), symptomatic finches/Golf course to Beelab (▼). Estimates were averaged over all models in the candidate set, when combined QAICc support for a given factor was at least approximately 33%.

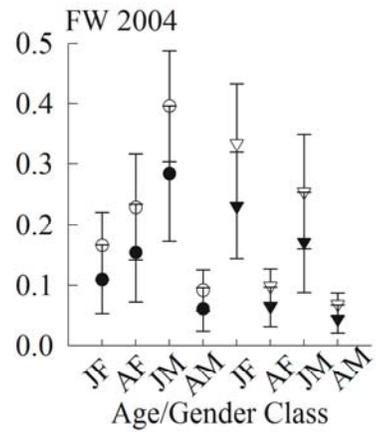
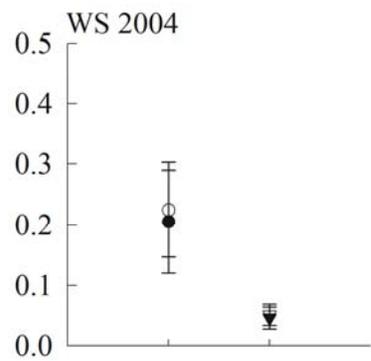
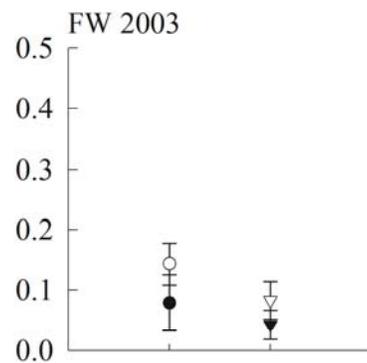
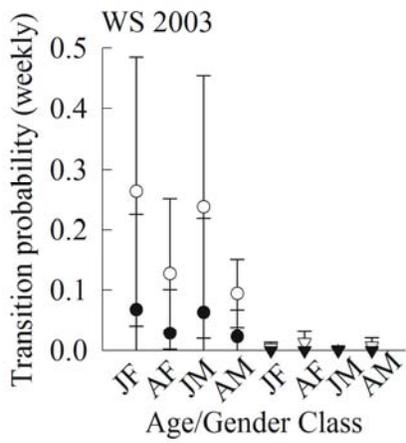
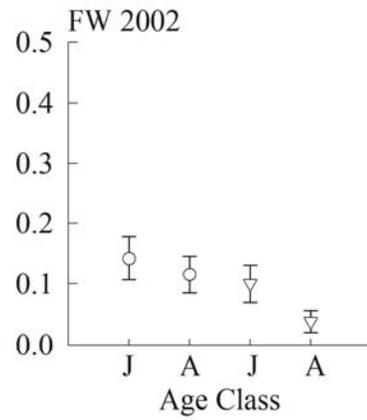
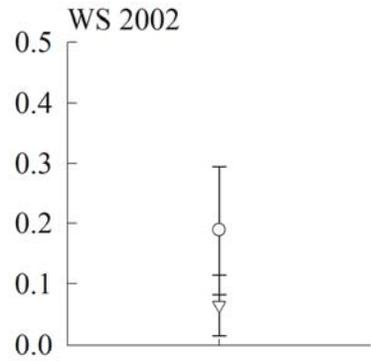
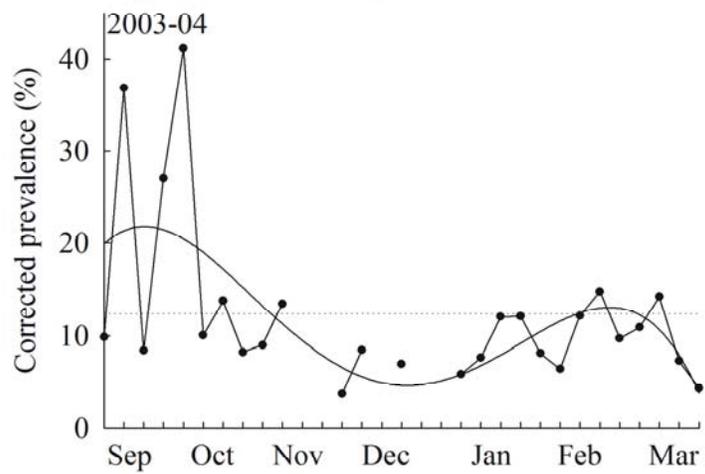
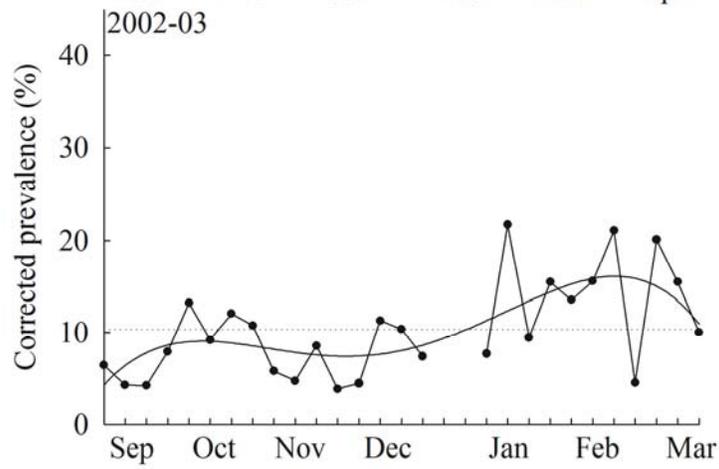
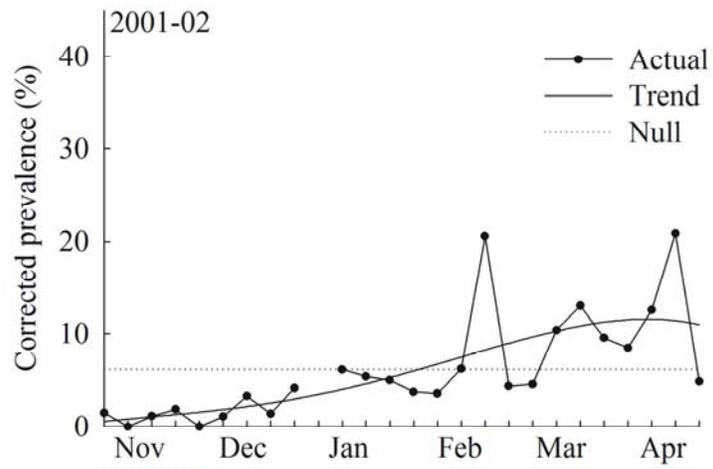


Figure 3.3. Estimated disease prevalence corrected for estimated detection probabilities stratified by disease state, age, and gender from 2001-2004 in Ithaca, NY. Data from the two primary sampling sites were combined, as the qualitative patterns of prevalence were similar. For each year, the null values of estimated prevalence represent the time invariant average disease prevalence from approximately September through early April. The trend values of estimated prevalence represent the fit of a fourth degree polynomial with respect to time. To evaluate the relative fit of the two models, I calculated Akaike information criterion (Akaike 1973) corrected for small sample sizes. Models with smaller values of AICc are better supported by the data. In 2001-02: AICc (null) = 87.89, AICc (trend) = 83.80; 2002-03: AICc (null) = 90.64, AICc (trend) = 86.91; 2003-04: AICc (null) = 115.77, AICc (trend) = 113.09.



For seasons other than FW 2001 and WS 2002 (insufficient data), I used the frequencies of individuals stratified by disease state, age, and gender corrected for group-specific detection probabilities to run four plausible logistic regression models to evaluate the influence of age and gender as potential risk factors of MG infection. Age and gender appeared to play more of a role in affecting conjunctivitis risk during the autumn seasons (Table 3.2). When these effects were supported by the data, estimated odds ratios indicated that juvenile and female finches exhibited a higher risk of infection with MG (Table 3.2).

Between November 27 2001 and March 28 2002, I attached 37 Holohil radio receivers to 31 asymptomatic and 6 symptomatic house finches stratified in four release cohorts. For the 21 days following initial attachment, I made every effort to locate each bird during daytime hours within the Ithaca, NY study area. I was not able to relocate each bird every day; finches were relocated on unique days between five and nine times within the 21-day lifespan of a transmitter battery. Given the limited battery life of radios and logistical limitations on the total number of finches I was able to track during a given time period, I did not stratify the limited sample by age, gender, or time. I pooled birds across cohorts stratified by disease state and release location, respectively, only after testing that the multivariate median distributions between group specific cohorts were the same.

I obtained 394 finch locations for all birds fitted with radio receivers in the sample, and determined the multivariate median UTM location for each bird. Using these derived coordinates, I found that the average distance between all pairs of individuals when stratified by release site was ($\delta=1400\text{m}$), and the probability of a smaller or equal value was 0.0002 suggesting that the distribution between multivariate medians of finches based on release site was different. I found that the average distance to the multivariate (overall UTM) median for birds released at the Beelab and Golf course

were 1111.49m and 678.34m, respectively (Fig. 3.4A). There was reasonable evidence that the dispersion of finch locations based on release at the Golf course was significantly smaller than that around the Beelab ($n=37$; standardized test statistic = -1.93; $p = 0.0536$) (Fig. 3.4A). Since there was evidence of a difference in dispersion between finches based upon release site, I had to condition my analysis of dispersion as a function of disease state on releases at the Golf course. I did this to maximize the power of the permutation test, as the sample size of finch releases was greatest at this site. The average distance to the bivariate median differed between symptomatic (552.6m) and asymptomatic finches (708.8m), however the permutation procedure revealed that the dispersion around the multivariate medians of finches based on disease state were not different ($n=19$; standardized test statistic = 0.648; $p = 0.727$) (Fig. 3.4B).

Table 3.2. Results of logistic regression analysis of conjunctivitis risk factors in house finches for the fall-winter (FW) 2002-2004 and winter-spring (WS) 2003-2004 seasons in Ithaca, NY, USA. Observations for age, gender, and disease state specific groups were corrected for estimated detection probabilities prior to analysis. To account for uncertainty in the detection process, the counts for each group were weighted by the inverse of the variance of group specific detection probabilities. Akaike information criterion (AIC) was used as a basis for ranking the set of four *a priori* models per season. Results are shown for models that are < 3.0 AIC units from the most parsimonious model (i.e., having the lowest AIC value). *Model notation:* A, age (juvenile or adult); G, gender; additive and interaction effects indicated with ‘+’ and ‘*’ signs, respectively. Values within parentheses i) under “Model Parameters” are *p*-values for the test of the null hypothesis that the respective parameter coefficient is equal to zero, and ii) under “Odds Ratios” are the 95% confidence intervals for conditional odds ratios, where OR_{age} is the relative odds that juvenile vs. adult finches have conjunctivitis, and OR_{gender} is the relative odds that female vs. male finches have conjunctivitis.

Season	Model	AIC	MODEL PARAMETERS				ODDS RATIOS	
			β_0	β_{age}	β_{gender}	$\beta_{age*gender}$	OR _{age}	OR _{gender}
FW2002	<i>Age+Gender</i>	838.8	-3.765 (<0.01)	1.062 (<0.01)	0.695 (<0.01)	-	2.89 (1.9, 4.5)	2.00 (1.4, 3.0)
FW2002	<i>Age*Gender</i>	839.4	-3.613 (<0.01)	0.799 (0.01)	0.325 (0.41)	0.527 (0.26)	*	*
WS2003	<i>Age</i>	427.4	-1.758 (<0.01)	0.373 (0.30)	-	-	1.45 (0.8, 2.8)	-
WS2003	<i>Gender</i>	428.1	-1.744 (<0.01)	-	0.207 (0.50)	-	-	1.23 (0.7, 2.2)
FW2003	<i>Age*Gender</i>	422.1	-3.087 (<0.01)	1.369 (0.01)	1.030 (0.03)	-0.877 (0.12)	*	*
FW2003	<i>Age+Gender</i>	422.5	-2.781 (<0.01)	0.905 (<0.01)	0.434 (0.10)	-	2.47 (1.4, 4.3)	1.54 (0.9, 2.6)
FW2003	<i>Age</i>	423.3	-2.606 (<0.01)	0.969 (<0.01)	-	-	2.6 (1.5, 4.5)	-
WS2004	<i>Gender</i>	130.6	-3.250 (<0.01)	-	1.877 (0.01)	-	-	6.54 (2.5, 17.0)
WS2004	<i>Age+Gender</i>	131.0	-3.402 (<0.01)	0.652 (0.21)	1.801 (<0.01)	-	1.92 (0.7, 5.3)	6.06 (2.3, 15.9)
FW2004	<i>Age+Gender</i>	226.3	-4.304 (<0.01)	1.697 (0.01)	0.810 (0.05)	-	5.46 (2.5, 11.9)	2.25 (1.0, 5.0)
FW2004	<i>Age*Gender</i>	227.4	-4.560 (<0.01)	2.202 (<0.01)	1.229 (0.05)	-0.780 (0.344)	*	*
FW2004	<i>Age</i>	228.4	-3.925 (<0.01)	1.843 (<0.01)	-	-	6.31 (2.9, 13.5)	-

Figure 3.4. Distribution of multivariate median locations for House Finches released as a function of site (panel A: ○ - Beelab, ○ - Beelab origin, ● - Beelab multivariate median, △ - Golf course, △ - Golf course origin, ▲ - Golf course multivariate median) and as a function of disease state at the Golf course (panel B: △ - Golf course, □ - asymptomatic finch, ■ - bivariate median for asymptomatic finches, × - symptomatic finch, ✕ - bivariate median for symptomatic finches).

Discussion

Disease Prevalence, Conjunctivitis Risk Factors, and Population Structure

Pathogens can impose limiting and regulatory constraints upon host populations, thus it is important to determine how disease influences host demography and behavior. Likewise, the structure of host populations can influence pathogen transmission and persistence. In this study, I evaluated the structure of a local-scale house finch population, used capture-recapture data to assess the impact of *Mycoplasma gallisepticum* (MG) infection and host properties on local-scale and transitory movements of birds, and evaluated the influence of host age and gender on conjunctivitis risk. The study of this host-pathogen system has received considerable attention in the last decade due to the sudden emergence of a novel strain of *Mycoplasma* previously known to infect poultry (for a review see Dhondt et al. 2005). The dynamics of this system are particularly interesting because of the speed with which MG has spread and the cyclic nature of apparent seasonal outbreaks throughout the eastern range of the house finch (Dhondt et al. 1998, Altizer et al. 2004b, Hosseini et al. 2006).

Descriptions of patterns in MG prevalence at regional spatial scales suggest that there are bimodal peaks in prevalence between the months of August and March in the eastern United States, which dampen in magnitude with increasing latitude (Altizer et al. 2004b). From a local scale perspective this phenomenon was not apparent in the southeastern US (Altizer et al. 2004a), but data from the study area does corroborate this pattern during 2002-03 and 2003-04 (Fig. 3.3). In 2001-02 the data suggested a mild single peak in disease prevalence in late March and April, and I believe this delayed peak was in part driven by warmer than usual local average temperatures and lower than average snowfall accumulation (Ithaca weather station data). When the data is stratified by season, there is greater disease prevalence during the Fall-Winter

compared with Winter-Spring seasons. In concordance with this trend, juvenile finches had a greater risk of expressing conjunctivitis (Table 3.2), which supports earlier suggestions that annual pulses of juvenile finches provide a pool of susceptible individuals that in part drive the seasonal dynamics of MG infections (Hosseini et al. 2004). A study by Altizer et al. (2004a) conducted in Georgia cited similar results, and when considered together with this work suggests the ubiquitous influence of juvenile pulses to finch-MG dynamics. I also found reasonable evidence that a gender effect was additive with age, where female finches were consistently at higher risk of expressing conjunctivitis during the FW season (Table 3.2); a result not strongly supported in Altizer et al (2004a). While adult females might be particularly vulnerable due to the energetic stress of the previous breeding season, juvenile females may be more susceptible because of their smaller body size (thus increased energetic demands during colder periods in the northeastern US) and likely lower levels of immunocompetence (compared with adults). During winter-spring seasons there was no consistency with respect to the factors accounting for significant variation in conjunctivitis risk, but if age or gender were supported in models then juvenile and female finches were always at higher risk (Table 3.2).

By and large, there was a consistent annual pattern in the age structure of the population. As would be expected, there were comparable or greater proportions of juveniles in the fall-winter, compared with winter-spring seasons (Appendix E). This result is corroborated by the lower estimated survivorship of juveniles (Appendix C). This is not surprising, as pulses of juveniles following the breeding season will increase their proportional representation in the population. There was also a consistent pattern in the gender profile of the population, where there were a greater proportion of adult males during the WS seasons at both sites (Appendix E). This supports the body size hypothesis espoused by Belthoff and Gauthreaux (1991), which

predicts that sexual segregation in house finches occurs over a latitudinal gradient because males, due to their larger body size, are better able to cope with the energetic demands of colder environments. I did not detect a gender effect in my analysis of transience though, which would lend more credibility to this hypothesis.

Transience Analysis

Recent work by Hosseini et al. (2006) has suggested that asymptomatic carriers can facilitate the spread of MG at a regional spatial scale. My analysis of the transient movements of marked individuals in this study appears consistent with this hypothesis on a local spatial scale as I estimated greater proportions of asymptomatic as opposed to symptomatic transients (Fig. 3.1). I go further to provide evidence that there are also significant differences in transitory movements of finches as a function of season and age. A transient effect is functionally interpreted as a difference between apparent survival of birds between the first time interval since marking (i.e., TSM) and subsequent intervals for each cohort (Pradel et al. 1997). With these estimates, one can produce an *ad hoc* estimator of the proportion of transient individuals in the marked population, where a transient is defined as an individual that is seen only once and never again within a given season. My results demonstrated that there were lower proportions of transients in the symptomatic disease state across both age classes and all seasons, when a transient effect was supported in the data (Fig. 3.1). This apparent disease-induced reduction in local scale movements could result from an interaction between reduced visual acuity due to conjunctivitis, lethargy associated with MG infection (Kollias et al. 2004), and increased energetic demands due to disease and colder temperatures (in northern latitudes) (Dawson et al. 1983). Given these stressors, symptomatic finches may remain more faithful to sites that have stable food supplies (continuously stocked bird feeders as in my study). Recent work by Dana Hawley and

colleagues (unpublished data) supports this notion in part, as they found that finches with conjunctivitis spent more time foraging at bird feeders.

In four of the six seasons analyzed in this study, the data strongly suggested a transient effect. As I expected, the proportion of transients in the marked population was higher during the FW compared with the WS season (Fig. 3.1). Following the breeding season, eastern finches are known to undergo short-distance migrations (Able and Belthoff 1998) and in general tend to aggregate and form large roaming flocks (Hill 1993).

Counter to my expectations, adult finches exhibited a greater degree of transitory behavior as compared with juveniles, stratified by both disease states (Fig. 3.1). This might appear counter-intuitive at first because one might expect that adult birds are more sedentary as they are more likely to have an established home range.

Furthermore, analyses of banding recovery data suggest that juvenile house finches have a greater tendency to migrate distances greater than 80 km (Able and Belthoff 1998). How can my apparently contradictory results be explained? My estimates of transience are conditional on the spatial frame of reference for my study area (two sites approximately 1.5km apart). Adult finches likely have better knowledge of the surrounding areas (with respect to feeding and roosting sites), and may tend to exploit alternative food resources (backyard bird feeders) that might offer greater protection from harsh environmental conditions and reduced intraspecific competition. Juvenile birds on the hand most likely have limited knowledge of the surrounding areas and given that my banding sites were consistently stocked with sunflower seeds, I would expect that non-migratory juvenile birds have a greater tendency to be faithful to a local site with reliable food resources. Consequently, I hypothesize that adult finches have a larger 'ecological neighborhood' (Addicott et al. 1987) than juveniles during the non-breeding season, and that the spatial scale of my study area is capturing a

smaller fraction of that extent for adults. While juvenile finches may indeed have a greater tendency to migrate long distances, the fine spatial scale of my study would explain the results. This does not preclude the possibility that adult finches are engaging in shorter distance migratory events at a higher frequency than juveniles. My results, however, underscore the importance of considering heterogeneity in house finch movements over multiple spatial and temporal scales when modeling this disease system, and highlight the need in general to design ecological studies that are able to identify scale-specific linkages.

Apparent Survival, Detection, and Movements

In contrast with results from Faustino et al (2004), I found for the most part that estimates of apparent resident survival for symptomatic finches were greater than those of asymptomatic finches (Appendix C). I believe that this result occurred for several reasons. I used a one-week period to define a transient individual and realistically there is some unknown distribution of time over which a given transient animal will reside in an area before moving permanently. While I reasoned that a one-week interval was appropriate for capturing the majority of transient individuals in the marked sample, this time interval may not be sufficient for capturing all of the transient dynamics of finches. As MG infection is likely to inhibit movement propensity (supported by lower proportions of symptomatic transients in the marked sample), this is an especially likely scenario for birds in the asymptomatic disease state. Since transience is a form of permanent emigration (confounded with true mortality) and a greater proportion of asymptomatic transients were estimated in the marked sample, this could explain the discrepancy with results in Faustino et al. (2004). A limitation of this study is that I was constrained to assign disease state as an individual seasonal covariate (i.e., if an individual was observed with conjunctivitis at least once, it was assigned as symptomatic), which reduces the statistical power that I

had to make inferences about true differences in state-specific survival, detection, and movement probabilities. Disease state is a dynamic variable and in some cases individual birds were observed in both states during a season, providing information about finer-scale levels of heterogeneity in state-specific detection probability. I could not accommodate this additional state structure because an exponential increase in the amount of data is required with each additional state included in multistate CMR models (Williams et al. 2002). The byproduct of the way I accounted for disease state most likely reduces the state-specific differences in the parameters that I report, and coupled with unaccounted heterogeneity in detection probabilities may in part be responsible for spurious estimates of resident survival. I also wish to point out that there were a greater number of observations in the dataset used in Faustino et al. (2004), which included information from temporary sites that were not considered (as the analyses required fixed sites and consistent sampling). In effect, their study area was larger, which likely encompassed a greater proportion of the home range of asymptomatic finches. Any changes in the distribution of study sites (even within a given study) will change the spatial scale over which a transient is defined.

There was a distinct seasonal pattern in apparent resident survivorship of finches as a function of age, where survival of juvenile birds was lower than that of adults during the FW seasons (Appendix C). Other studies have found that juvenile survival can be lower than that of adult birds (Ralph and Fancy 1995, Sandercock and Jaramillo 2002) or higher (Hagen et al. 2005). I expected juvenile finches to have lower survivorship as this age group is less experienced at foraging, finding adequate roosting sites, and evading predators. As reported in Faustino et al. (2004), I found virtually no evidence of a gender effect in survivorship (even accounting for a transience and disease effect) for this northeastern finch population. This contrasts with results in Nolan et al. (1998), where they show that female finches have a

survival advantage over males in the presence of an MG outbreak. Assuming that their estimates of survivorship were not biased (as they did not account for heterogeneity in detection probabilities of finches), the virulence of MG may have declined or the ability of finches to mount an immune response may have increased over time. This is certainly a possible scenario since earlier studies of captive finches exposed to MG reported high levels of mortality (Luttrell et al. 1998, Nolan et al. 1998), whereas recent experimental infections have shown the opposite results (Kollias et al. 2004, but see Sydenstricker et al. 2006). Since finches undergo significant behavioral changes (Hill 1993), coupled with gender-specific physiological constraints during the breeding season, further study is warranted during this time of year to determine whether similar patterns of survivorship emerge.

Although typically regarded as a nuisance parameter, group-specific encounter probabilities can have profound implications on the inferences that can be made from purely observational types of data (Jennelle et al. 2007). I accounted for variation in detection probability as a function of disease state, age, gender, and site in the construction of prevalence, age, and gender profiles by following the general methodology outlined in Jennelle et al. (2007). In Faustino et al. (2004), it was demonstrated that detection probabilities of house finches varied both temporally and as a function of disease state. Similarly in this study, I found that detection probabilities varied by disease state, age, gender, and study site (Appendix D). Significant variation in detection probability as a function of disease state was not discernable in every season as shown in Faustino et al (2004); likely because of the way in which I defined a symptomatic individual (a binary covariate indicating whether an individual was observed with conjunctivitis during a given season). I note, however, that the subset of data was different than that used in Faustino et al. (2004), thus I cannot expect model selection results to align in perfect agreement.

The dominant pattern that consistently emerged after evaluating movement probabilities was a distinct asymmetry between sites, where Beelab to Golf course movement (east to west) was always greater (Fig. 3.2) (with the exception of juvenile females in FW 2004). This difference was accentuated in the WS seasons (coupled with greater proportions of transients at the Beelab), and I know from analyses of banding recovery data that short-distance migrations occurring over a northeast to southwest spatial gradient occur in autumn (Able and Belthoff 1998). If I consider the two study sites as a simplified microcosm of migratory space, then it is highly unlikely that the movement patterns that I detect are capturing short distance migrations (the majority of these events are subsumed within estimates of transience). In addition, the telemetry analysis of finches stratified by release site produced clear evidence that there is site-specific daytime partitioning of the study area (Fig. 3.4A). There is also considerably more dispersion of daytime habitat usage around the Beelab compared with the Golf course site. The synthesis of these results suggests that there are considerable differences in habitat quality between the two sites. The Golf course is much closer in proximity to housing developments (with bird feeders) and equally as important; higher densities of coniferous trees. Dhondt et al. (2007) showed that house finches make a shift from using primarily deciduous to exclusively coniferous trees as roosting sites in the northeastern USA during winter. Not surprisingly, it has been shown that conifers provide substantial energy savings to passerine birds by reducing convective and radiative heat losses to the environment (Buttemer 1985). While Dhondt et al. (2007) suggest (and I agree) that MG transmission events occur more readily and frequently at feeding sites as there are higher observed rates of finch contact and activity, I believe that roosting sites during winter (at least in the northeastern USA) function as a limiting factor that may in part drive heterogeneity in the density of foraging finches at nearby feeders. The availability of roosting and

feeding locations appear to play a major role in structuring local subpopulations, which lays a foundation for the local scale interactions among individuals that Hochachka and Dhondt (2006) suggest drive disease dynamics in this system.

There was no consistent difference in the age or gender profiles between sites (Appendix E), so it is unclear what mechanism(s) determine the membership within the two patches. In any case, the concept of coniferous trees functioning as a limiting factor in the distribution of wintering house finches warrants further investigation. If confirmed through experimental procedures, this information could contribute significantly to refinements in disease transmission models, and efforts to predict and control MG outbreaks in house finches.

During three seasons (WS 2003, FW 2003, and FW 2004) there was a clear distinction in movement probabilities between symptomatic and asymptomatic finches with the former group exhibiting reduced probabilities of movement (Fig. 3.2). A similar pattern occurred in the WS 2004 season, but the difference was negligible. Although movement probabilities of asymptomatic finches were never greater than symptomatic birds, I believe that the lack of consistency in the pattern was again due to the way in which I defined a symptomatic finch. Lacking sufficient data to include disease state as a dynamic variable within a given season certainly reduced the magnitude of the differences I could detect in movement probabilities between finches as a function of disease state. Yet my results coupled with findings from Dhondt et al. (2007), which show that infected finches consistently travel shorter distances between roost and feeding sites suggests MG infection limits local and dispersal movement capacity of house finches.

To a lesser degree there was some evidence (FW 2002, WS 2003, and FW 2004) that juvenile finches had higher probabilities of movement compared with adults. Yet, there was negligible variation in movement probabilities as a function of gender (with

the exception of FW 2004). Taken together, I believe that at a local spatial scale, movements as a function of gender and age do not contribute significantly to MG dynamics. Rather, larger scale movements and dispersal events as a function of age (as evidenced by estimates of transience) likely contribute more to spread and maintenance of MG within and between finch populations.

While conjunctivitis may inhibit visual acuity and thereby rates of movement and dispersal (as suggested by transience estimates in Fig. 3.1 and state-specific movement probabilities in Fig. 3.2), it is not clear whether local daytime movements are limited (Fig. 3.4B). I was not able to find statistical evidence of a disease effect on the dispersion of daytime movements, but my sample of infected finches was very small (4 birds) and lacks adequate power for making strong inferences from the data. Yet in a recently published study of the roosting behavior of house finches, Dhondt et al. (2007) reported that movement distances between roost sites and subsequent daytime locations or vice versa were significantly lower for symptomatic finches. Thus, at least during winter it is likely that conjunctivitis exacerbates visual impairment of finches at dawn and dusk when lighting conditions are marginal and imposes substantial energetic constraints, which would explain the shorter distances they travel to and from roosting sites.

My work demonstrates the ability of an infectious agent to limit the rate and spatial scale of movement of an avian host. It further suggests that adult (particularly asymptomatic) finches may have a more significant role in the spread and possible maintenance of MG infections in disparate populations. Evidence from my study suggests that symptomatic finches are less mobile and are likely to remain in a familiar area (at least while expressing clinical symptoms of MG infection) (Fig. 3.1). As there are regional differences in the behavior (Hill 1993) and movements (Able and Belthoff 1998, Hosseni et al. 2006) of finches, whether the patterns found in this data

hold true for other parts of their range warrants investigation. In large part, this paper not only presents novel results regarding the ecology and dynamics of the house finch-MG system, but it also synthesizes results across local and regional spatial scales.

Studies at multiple scales are needed to identify the ecological patterns and the processes driving them (Addicott et al. 1987) as quite often patterns and processes may be scale-dependent (Wiens 1989, Levin 1992). This study highlights the scale dependent correspondence between patterns in disease prevalence (double peaks in prevalence, Fig. 3.3; see Altizer et al. 2004b), major sources of disease spread (i.e., asymptomatic/juvenile finches; see Hosseini et al. 2004, 2006), and hypothesized risk factors (i.e., juvenile/female finches). Coupling these results with the finding that there is distinct structuring of local populations across the landscape (Fig. 3.4a) elucidates important linkages in the factors that drive MG dynamics across multiple spatial scales. Various combinations of processes can produce similar ecological patterns, making the task of assigning causality to one or more drivers a non-trivial endeavor (Cale et al. 1989); however, the similarity between these findings and work in this disease system conducted at a broader scale (using independent datasets) lends credibility to the inferences that I can make about drivers of Mycoplasmal conjunctivitis in house finches. It is possible that the similarity in disease patterns and purported driving processes across both regional and local spatial scales may stem from the highly mobile nature of house finches. Several researchers have noted that the population dynamics of metapopulations formed by highly mobile organisms are less likely to be affected by local habitat characteristics (Fahrig and Paloheimo 1988, Wiens 1989). Perhaps the potential for spatially extensive ‘ecological neighborhoods’ and resultant overlap/mixing of individuals between local subpopulations increases the likelihood that scale-dependent patterns and driving processes will correspond.

Carefully designed studies to test this hypothesis would be very useful in furthering our overall understanding of potential scaling laws in ecology.

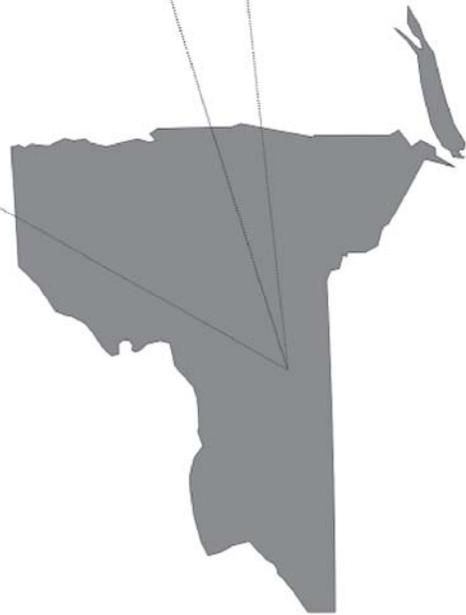
In this paper, I have sought to explain how movements and demographic factors contribute to disease dynamics at a local scale. Regional studies (Altizer et al. 2004*b*, Hochachka and Dhondt 2006, Hosseini et al. 2004, 2006) have relied upon data from the House Finch Disease Survey (Dhondt et al. 1998) to make inferences about patterns and processes. Further studies of this system should consider a design intermediate in spatial scale between this local study and HFDS monitoring, which might incorporate both citizen science and traditional capture-recapture design in order to confirm inferences about the linkages between related patterns and processes across a spatial gradient in this disease system.

Acknowledgements

This research was funded by the US National Science Foundation under DEB no. 0094456, and support was given from the Department of Natural Resources, Cornell University. I thank Veronique Connolly, Melanie Driscoll, Cristina Faustino, Sarah Goodwin, Dana Hawley, Mari Kimura, Tom Muscato, and Elliott Swarthout for field assistance. I also thank Evan Cooch, Andre Dhondt, William Kendall, Cliff Kraft, and Patrick Sullivan for valuable discussion and comments on this manuscript.

APPENDIX A

Map of study area located in Ithaca, NY, USA. The two primary banding/resighting sites include Liddell Field Station (Beelab) and Robert Trent Jones Golf course (Golf course).



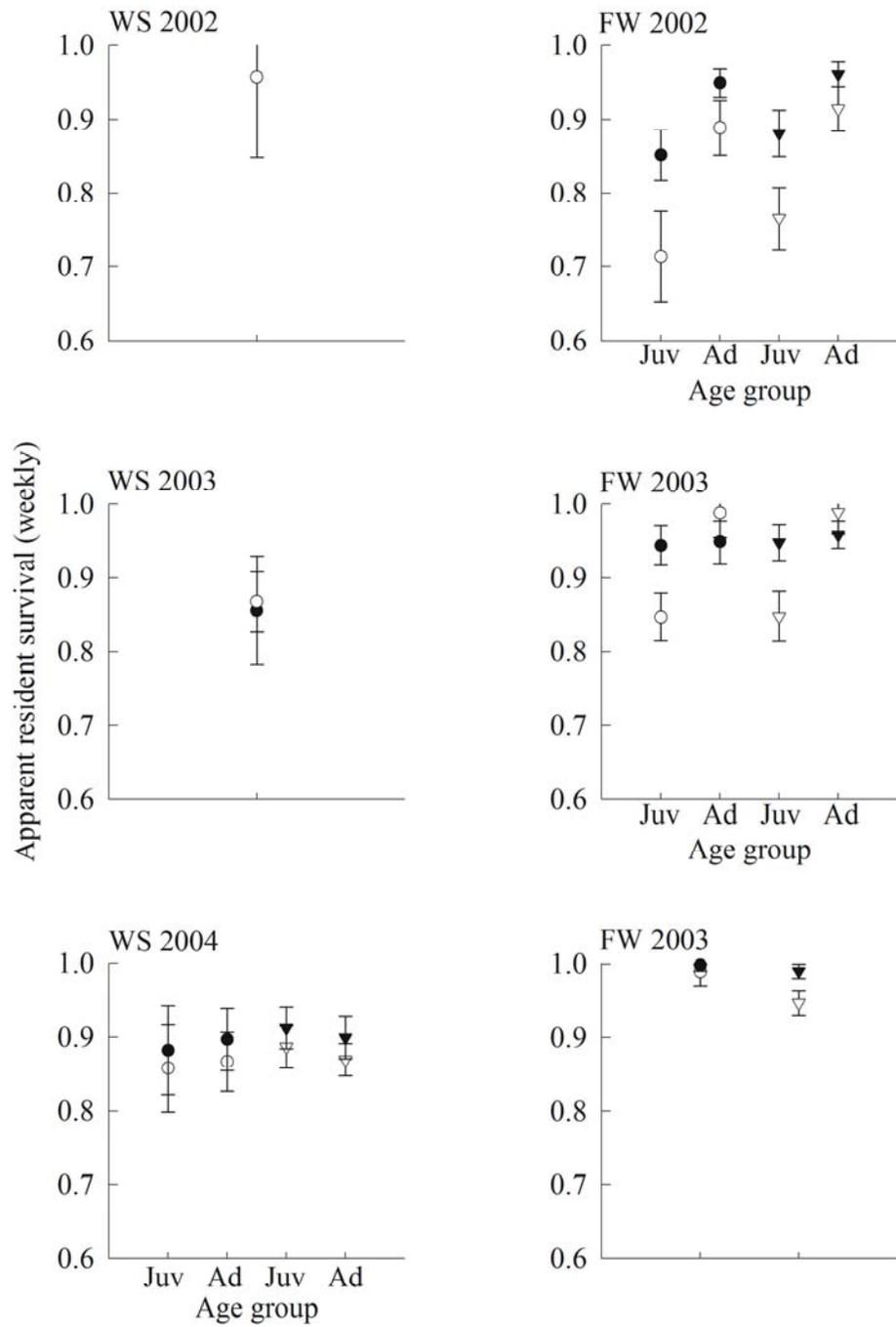
APPENDIX B

Numbers of asymptomatic (symptomatic) initial captures, recaptures, and resights stratified by age (HY = hatch year; AHY = after hatch year) and gender (F = female; M = male) for fall-winter (FW) and winter-spring (WS) subsets for the Beelab and Golf course banding sites from 2001-04, in Ithaca, NY, USA. Note that recaptured and especially resighted birds may be individuals that were banded at other locations in the study area or at other time periods.

	FW2001	WS2002	FW2002	WS2003	FW2003	WS2004	FW2004
<i>Initial captures</i>							
Juv, F	51(2)	0(0)	131(6)	0(0)	37(2)	0(0)	44(2)
Juv, M	37(0)	0(0)	103(5)	0(0)	28(1)	0(0)	44(2)
Ad, F	1(0)	0(0)	32(2)	0(0)	12(0)	0(0)	32(1)
Ad, M	0(0)	0(0)	54(0)	0(0)	11(1)	0(0)	25(0)
<i>Recaptures</i>							
Juv, F	55(3)	10(1)	96(49)	5(2)	26(4)	16(1)	32(3)
Juv, M	44(0)	5(2)	62(22)	11(4)	28(1)	20(1)	33(0)
Ad, F	22(0)	5(0)	34(6)	6(2)	12(0)	6(1)	21(1)
Ad, M	19(0)	10(0)	38(2)	22(2)	11(1)	24(0)	15(0)
<i>Resights</i>							
Juv, F	20(2)	18(9)	265(95)	46(10)	154(35)	173(59)	282(29)
Juv, M	20(0)	28(2)	133(51)	69(9)	114(36)	221(9)	375(38)
Ad, F	27(0)	33(0)	165(9)	112(11)	92(10)	259(39)	370(11)
Ad, M	43(0)	48(0)	205(21)	228(30)	177(4)	674(21)	460(6)

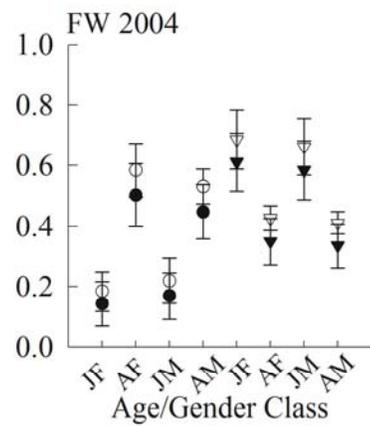
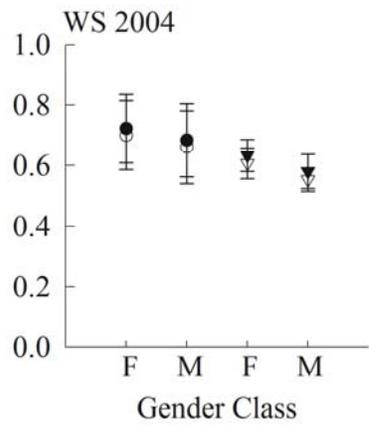
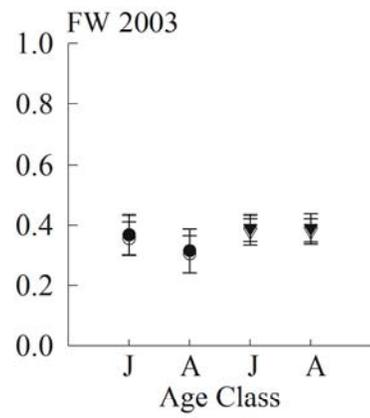
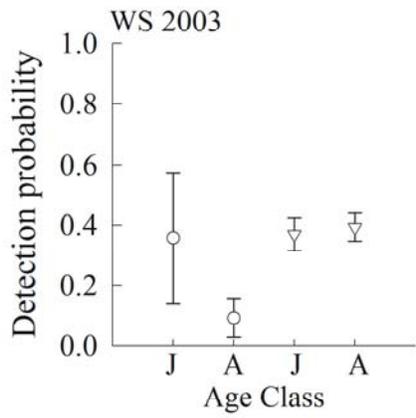
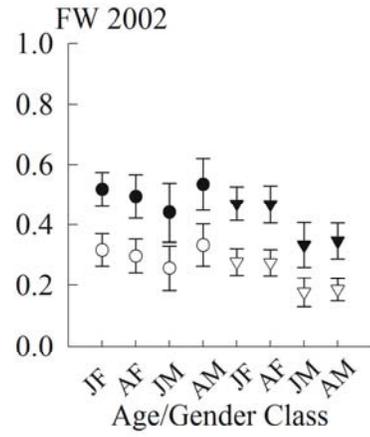
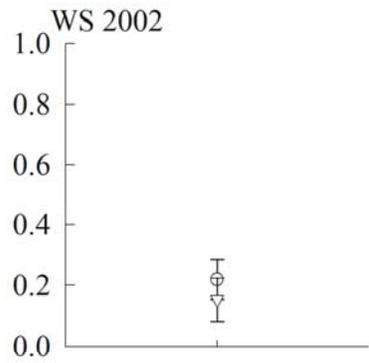
APPENDIX C

Estimated weekly apparent resident survival (± 1 SE) of house finches stratified by disease state, age (Juv = juveniles and Ad = adults), or site from 2002-2004 in Ithaca, NY; asymptomatic finches/Beelab (\circ), symptomatic finches/Beelab (\bullet), asymptomatic finches/Golf course (∇), symptomatic finches/Golf course (\blacktriangledown). Estimates were averaged over all models in the candidate set, when combined QAICc support for a given factor was at least approximately 33%.



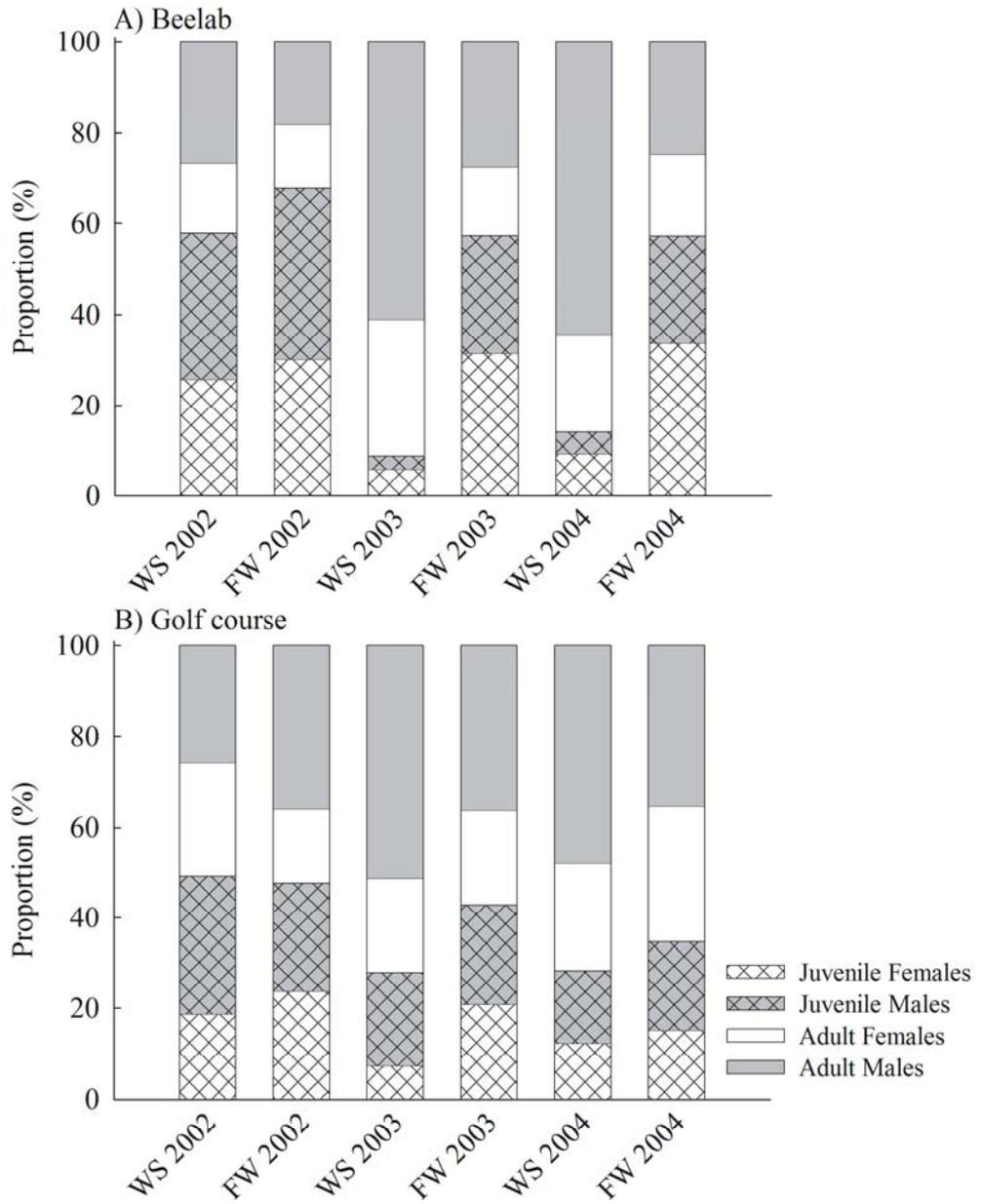
APPENDIX D

Estimated weekly detection probabilities (± 1 SE) of house finches stratified by disease state, age (J = juveniles and A = adults), gender (F = females and M = males), and site from 2002-2004 in Ithaca, NY; asymptomatic finches/Beelab (○), symptomatic finches/Beelab (●), asymptomatic finches/Golf course (▽), symptomatic finches/Golf course (▼). Estimates were averaged over all models in the candidate set, when combined QAICc support for a given factor was at least approximately 33%.



APPENDIX E

Seasonal (FW = fall-winter, WS=winter-spring) age and gender profiles of house finches corrected for disease state, age, gender, and site specific detection probabilities in Ithaca, NY 2002-2004. Panels A and B represent the Beelab and Golf Course sites, respectively.



REFERENCES

- Able, K.P., and J.R. Belthoff. 1998. Rapid 'evolution' of migratory behaviour in the introduced house finch of eastern North America. *Proceedings of the Royal Society of London B* **265**:2063-2071.
- Addicott, J.F., J.M. Aho, M.F. Antolin, D.K. Padilla, J.S. Richardson, and D.A. Soluk. 1987. Ecological neighborhoods: Scaling environmental patterns. *Oikos* **49**:340-346.
- Altizer, S., A.K. Davis, K.C. Cook, and J.J. Cherry. 2004a. Age, sex, and season affect the risk of Mycoplasma conjunctivitis in a southeastern house finch population. *Canadian Journal of Zoology* **82**:1-9.
- Altizer, S.M., W.M. Hochachka, and A.A. Dhondt. 2004b. Seasonal dynamics of mycoplasma conjunctivitis in eastern North American House Finches. *Journal of Animal Ecology* **73**:309-322.
- Anderson, D.R., and K.P. Burnham. 2002. Avoiding pitfalls when using information theoretic methods. *Journal of Wildlife Management* **66**:912-918.
- Arnason, A.N. 1973. The estimation of population size, migration rates, and survival in a stratified population. *Research in Population Ecology* **15**: 1-8.
- Belthoff, J.R., and S.A. Gauthreaux, Jr. 1991. Partial migration and differential winter distribution of House Finches in the eastern United States. *Condor* **93**:374-382.
- Bolker, B., and B. Grenfell. 1995. Space, persistence and dynamics of measles epidemics. *Philosophical Transactions of the Royal Society of London B* **348**:309-320.
- Brownie, C., and D.S. Robson. 1983. Estimation of time-specific survival rates from tag-resighting samples: A generalization of the Jolly-Seber model. *Biometrics* **39**:437-453.

- Burnham, K.P., and D.R. Anderson. 2002. Model selection and multimodel inference: A practical information-theoretic approach. Springer-Verlag, New York.
- Buttner, W.A. 1985. Energy relations of winter roost-site utilization by American goldfinches (*Carduelis tristis*). *Oecologia* **68**:126-132.
- Cade, B.S., and J.D. Richards. 2001. User manual for BLOSSOM Statistical Software. Fort Collins, CO: U.S. Geological Survey, Midcontinent Ecological Science Center. 107 pp.
- Cale, W.G., G.M. Henebry, and J.A. Yeakley. 1989. Inferring process from pattern in natural communities. *BioScience* **39**:600-605.
- Cilimburg, A.B., M.S. Lindberg, J.T. Tewksbury, and S.J. Hejl. 2002. Effects of dispersal on survival probability of adult yellow warblers (*Dendroica petechia*). *Auk* **119**:778-789.
- Cooch, E.G., and G.C. White. 2006. A Gentle Introduction to Program MARK (5th Edition). [online] <http://www.phidot.org/software/mark/docs/book>.
- Dawson, W.R., R.L. Marsh, and M.E. Yacoe. 1983. Metabolic adjustments of small passerine birds for migration and cold. *American Journal of Physiology – Regulatory, Integrative, and Comparative Physiology* **245**:755-767.
- Dhondt, A.A., D.L. Tessaglia, and R.L. Slothower. 1998. Epidemic mycoplasmal conjunctivitis in house finches from eastern North America. *Journal of Wildlife Diseases* **34**:265-280.
- Dhondt, A.A., S. Altizer, E.G. Cooch, A.K. Davis, A. Dobson, M.J.L. Driscoll, B.K. Hartup, D.M. Hawley, W.M. Hochachka, P.R. Hosseini, C.S. Jennelle, G.V. Kollias, D.H. Ley, E.C.H. Swarthout, and K.V. Sydenstricker. 2005. Dynamics of a novel pathogen in an avian host: Mycoplasmal conjunctivitis in house finches. *Acta Tropica* **94**:77-93.

- Dhondt, A.A., M.J.L. Driscoll, and E.C.H. Swarthout. 2007. House finch *Carpodacus mexicanus* roosting behaviour during the non-breeding season and possible effects of mycoplasmal conjunctivitis. *Ibis* **149**:1-9.
- Dobson, A.P., and P.J. Hudson. 1995. Microparasites: Observed patterns in wild animal populations. Pages 52-89 in B.T. Grenfell and A.P. Dobson, editors. *Ecology of infectious diseases in natural populations*. Cambridge University Press, Cambridge, UK.
- Dowell, S.F. 2001. Seasonal variation in host susceptibility and cycles of certain infectious diseases. *Emerging Infectious Diseases* **7**:369-374.
- Fahrig, L., and J. Paloheimo. 1988. Effect of spatial arrangement of habitat patches on local population size. *Ecology* **69**:468-475.
- Faustino, C.R., C.S. Jennelle, V. Connolly, A.K. Davis, E.C. Swarthout, A.A. Dhondt, and E.G. Cooch. 2004. *Mycoplasma gallisepticum* infection dynamics in a house finch population: seasonal variation in survival, encounter and transmission rate. *Journal of Animal Ecology* **73**:651-669.
- Hagen, C.A., J.C. Pitman, B.K. Sandercock, R.J. Robel, and R.D. Applegate. 2005. Age-specific variation in apparent survival rates of male Lesser Prairie-chickens. *Condor* **107**:78-86.
- Hargrove, J.W., and Borland, C.H. 1994. Pooled population parameter estimates from mark-recapture data. *Biometrics* **50**:1129-1141.
- Hartup, B.K., G.V. Kollias, and D.H. Ley. 2000. Mycoplasmal conjunctivitis in songbirds from New York. *Journal of Wildlife Diseases* **36**:257-264.
- Hartup, B.K., J.M. Bickal, A.A. Dhondt, D.H. Ley, and G.V. Kollias. 2001. Dynamics of conjunctivitis and *Mycoplasma gallisepticum* infections in House Finches. *Auk* **118**:327-333.

- Hestbeck, J.B., J.D. Nichols, and R.A. Malecki. 1991. Estimates of movement and site fidelity using mark-resight data of wintering Canada geese. *Ecology* **72**: 523-533.
- Hill, G.E. 1993. House finch (*Carpodacus mexicanus*). *The Birds of North America*, no. 46 (eds A. Poole and F. Gill), pp. 1-24. Academy of Natural Sciences, Philadelphia and American Ornithologists' Union, Washington, D.C.
- Hochachka, W.M., and A.A. Dhondt. 2000. Density-dependent decline of host abundance resulting from a new infectious disease. *Proceedings of the National Academy of Sciences* **97**:5303-5306.
- Hochachka, W.M., and A.A. Dhondt. 2006. House finch (*Carpodacus mexicanus*) population- and group-level responses to a bacterial disease. *Ornithological Monographs* **60**:30-43.
- Hosseini, P.R., A.A. Dhondt, and A. Dobson. 2004. Seasonality and wildlife disease: how seasonal birth, aggregation and variation in immunity affect the dynamics of *Mycoplasma gallisepticum* in house finches. *Proceedings of the Royal Society B* **271**:2569-2577.
- Hosseini, P.R., A.A. Dhondt, and A.P. Dobson. 2006. Spatial spread of an emerging infectious disease: Conjunctivitis in house finches. *Ecology* **87**:3037-3046.
- Hudson, P. J., A. P. Rizzoli, B. T. Grenfell, J. A. P. Heesterbeek, and A. P. Dobson. 2002. *The ecology of wildlife diseases*. Oxford University Press, New York, New York, USA.
- Jennelle, C.S., E.G. Cooch, M.J. Conroy, and J.C. Senar. 2007. State-specific detection probabilities and disease prevalence. *Ecological Applications* **17**: 154-167.

- Joe, M., and K.H. Pollock. 2002. Separation of survival and movement rates in multi-state tag-return and capture-recapture models. *Journal of Applied Statistics* **29**:373-384.
- Kollias, G.V., K.V. Sydenstricker, H.W. Kollias, D.H. Ley, P.R. Hosseini, V. Connolly, and A.A. Dhondt. 2004. Experimental infection of house finches with *Mycoplasma gallisepticum*. *Journal of Wildlife Diseases* **40**:79-86.
- Lebreton, J.-D., K.P. Burnham, J. Clobert, and D.R. Anderson. 1992. Modeling survival and testing biological hypothesis using marked animals: A unified approach with case studies. *Ecological Monographs* **66**:67-118.
- Levin, S.A. 1992. The problem of pattern and scale in ecology: The Robert H. MacArthur award lecture. *Ecology* **73**:1943-1967.
- Lindberg, M., and E. Rexstad. 2002. Capture-recapture sampling designs. Pages 251-262 in A.H. El-Shaarawi and W.W. Piegorsch, editors. *Encyclopedia of Environmetrics*, John Wiley & Sons, Ltd, Chichester, UK.
- Luttrell, M.P., D.E. Stallknecht, J.R. Fischer, C.T. Sewell, and S.H. Kleven. 1998. Natural *Mycoplasma gallisepticum* infection in a captive flock of house finches. *Journal of Wildlife Diseases* **34**:289-296.
- MacKenzie, D.I. 2006. Modeling the probability of resource use: The effect of, and dealing with detecting a species imperfectly. *Journal of Wildlife Management* **70**:367-374.
- Mollison, D., and S.A. Levin. 1995. Spatial dynamics of parasitism. Pages 384-398 in B.T. Grenfell and A.P. Dobson, editors. *Ecology of infectious disease in natural populations*. Cambridge University Press, Cambridge, UK.

- Nolan, P.M., G.E. Hill, and A.M. Stoehr. 1998. Sex, size and plumage redness predict house finch survival in an epidemic. *Proceedings of the Royal Society of London B* **265**:961-965.
- Pac, H.I., and K. Frey. 1991. Some population characteristics of the Northern Yellowstone bison herd during the winter of 1988-89. Montana Department of Fish, Wildlife, and Parks, Bozeman, Montana, USA.
- Peterson, D.L., and V.T. Parker. 1998. *Ecological Scale: Theory and applications*. Columbia University Press, New York, USA.
- Pollock, K.H. 1981. Capture-recapture models allowing for age-dependent survival and capture rates. *Biometrics* **37**:521-529.
- Pollock, K.H., J.D. Nichols, C. Brownie, and J.E. Hines. 1990. Statistical inference for capture-recapture experiments. *Wildlife Monographs* **107**:1-97.
- Pradel, R. 1993. Flexibility in survival analysis from recapture data: handling trap-dependence. Pages 29–37 in J.-D. Lebreton and P. M. North, editors. *Marked individuals in the study of bird populations*. Birkhäuser Verlag, Basel, Switzerland.
- Pradel, R., J.E. Hines, J.-D. Lebreton, and J.D. Nichols. 1997. Capture-recapture survival models taking account of transients. *Biometrics* **53**:60-72.
- Pyle, Peter. 1997. *Identification Guide to North American Birds. Part I: Columbidae to Ploceidae*. Slate Creek Press, Bolinas, California.
- Ralph, C.J., and S.G. Fancy. 1995. Demography and movements of Apapane and Iiwi in Hawaii. *Auk* **97**:729-742.
- Royall, R. M. 1997. *Statistical Evidence: A Likelihood Paradigm*. Chapman and Hall, London, UK.

- Sandercock, B.K., and A. Jaramillo. 2002. Annual survival rates of wintering sparrows: Assessing demographic consequences of migration. *Auk* **119**:149-165.
- Schwarz, C.J., J.F. Schweigert, and A.N. Arnason. 1993. Estimating migration rates using tag-recovery data. *Biometrics* **49**:177-193.
- Senar, J.C., and M.J. Conroy. 2004. Multi-state analysis of the impacts of avian pox on a population of Serins (*Serinus serinus*): The importance of estimating recapture rates. *Animal Biodiversity and Conservation* **27**:133-146.
- Sydenstricker, K.V., A.A. Dhondt, D.M. Hawley, C.S. Jennelle, H.W. Kollias, and G.V. Kollias. 2006. Characterization of experimental *Mycoplasma gallisepticum* infection in captive house finch flocks. *Avian Diseases* **50**:39-44.
- White, G.C., and K.P. Burnham. 1999. Program MARK: Survival estimation from populations of marked animals. *Bird Study* **46**:120-139.
- Wiens, J.A. 1989. Spatial scaling in ecology. *Functional Ecology* **3**:385-397.
- Williams, B.K., J.D. Nichols, and M.J. Conroy. 2002. *Analysis and Management of Animal Populations*. Academic Press, San Diego.
- Van Riper III, C. I., S. G. Van Riper, and W. R. Hansen. 2002. Epizootiology and effect of avian pox on Hawaiian forest birds. *Auk* **119**:929-942.

CHAPTER 4

TEMPORARY MOVEMENTS AND CONTRIBUTIONS TO POPULATION GROWTH OF HOUSE FINCHES IN A HOST-PATHOGEN SYSTEM

Abstract. In host-pathogen systems, disease transmission via direct and indirect contacts between individuals occurs (in part) as a function of the degree of mixing across a gradient of spatial scale-dependent aggregations of the host. In a local scale house finch (*Carpodacus mexicanus*)-*Mycoplasma gallisepticum* (MG) system, I examined several forms of local movements critical for a thorough understanding of the seasonal capacity of finch populations to mix with conspecifics. Previous work in this system found evidence for restricted movement capacity of house finches infected with MG within a local study area. Using capture-recapture software programs MARK and MSSURVIV, respectively, I used multistate open robust design (MSORD) models to estimate temporary emigration and multistate reverse-time analysis to estimate contributions to population growth as a function of age and disease state. Contrary to my expectations, I found that temporary movements outside of the study area were not influenced by disease state or age. Rather season (autumn versus winter) was the most important source of variation in temporary movements, Greater temporary movements from the study area occurred in autumn, in combination with a broad scale tendency for finches to migrate. I also found that while juvenile finches contribute proportional more to population growth of the symptomatic (infected) class, disease dynamics are sustained primarily from within subpopulations of house finches. This work complements broad spatial scale analysis of this disease system, which used House Finch Disease Survey data, and provides a linkage between detailed local scale demographic analysis and broad patterns of disease prevalence.

Introduction

A novel strain of the pathogen *Mycoplasma gallisepticum* (MG), previously known to infect domestic poultry was documented in wild House Finch (hereafter referred to as finch) (*Carpodacus mexicanus*) populations in the winter of 1993 around Washington, D.C. (Ley et al. 1996, Luttrell et al. 1996, Fischer et al. 1997). The infection caused by the bacterium is recognized by a number of symptoms in afflicted birds including mild to severe swelling around one or both eyes, conjunctivitis, nasal and ocular discharge, lethargy, weakness, possible blindness, and in some cases death (Luttrell et al. 1998). Specific modes of MG transmission have been partially confirmed, and are associated with direct and indirect contact (via fomites) facilitated through social interactions at bird feeders (Dhondt et al. in press). Researchers have also hypothesized that disease transmission may occur through contact with infected seed in bird feeders (Luttrell et al. 1998) or conspecific contacts at roost sites (Fischer et al. 1997), although very low densities of roosting finches (Dhondt et al. 2007) suggest the later scenario may not be very important in the dynamics of this system.

Shortly after emergence of the novel strain of MG in wild birds, the spread of the bacterium reached epidemic proportions and within a few years of the first documented finch infections, the disease had spread throughout the eastern United States (Dhondt et al. 1998). Citizen science initiatives, such as the House Finch Disease Survey (HFDS) and Project FeederWatch coordinated by the Cornell Laboratory of Ornithology have been used to track the spread of MG infections in finches across the USA. Using data from the HFDS, Hartup et al. (1998) were able to broadly classify the risk periods of infections. They found that high-risk periods (defined as periods of time during which >30% of reported finch sightings included signs of conjunctivitis) extended from September through March, with low risk

periods extending from April to August of any given year. Analysis of regional scale data (across the eastern US) indicates two distinct peaks in disease prevalence occurring roughly between September-October and January-March (Altizer et al. 2004a), with a dampening in the magnitude of peaks with increasing latitude. During the early stages of the epidemic, evidence suggested that MG infections were causing widespread deaths of house finches across the eastern USA. Hochachka and Dhondt (2000) confirmed a causal relationship between declining finch abundance and mycoplasmal conjunctivitis, and their work provided evidence for density-dependent finch mortality at a regional spatial scale. Although the House Finch is not the only wild bird vulnerable to infection with MG (see Hartup et al. 2000, Mikaelian et al. 2001), it is the only species (as yet) that has been severely impacted.

Recent findings suggest that the rapid spread of MG infection observed throughout the eastern United States is likely linked to asymptomatic MG carriers and subsequent movements of these individuals (Hosseini et al 2006, Jennelle et al. submitted). The endemic cycles of disease occurrence, on the other hand, are likely maintained by an interaction of the release of newly susceptible birds (juveniles following the breeding season; Hosseini et al. 2004, Jennelle et al. submitted), seasonal aggregation at bird feeders (Hosseini et al. 2004), and a lack of permanent immunity following recovery of infections (Sydenstricker et al. 2005).

While finch migration in the eastern US (Able and Belthoff 1998) may account in part for disease spread among populations, local scale movements along with population structuring warrants further investigation with respect to understanding disease dynamics in the finch-MG system (Jennelle et al. submitted). In fact, analysis of data from the HFDS revealed that interactions among finches at a local spatial scale are important for understanding the impacts of MG in this disease system (Hochachka

and Dhondt 2006). This notion is supported to some degree by the tendency of finches to discretely partition the available habitat (Jennelle et al. submitted).

With the combination of a very mobile host, and a relatively restricted study area size (typical in many field research projects), there are several general scales of host movement that need to be addressed. This is particularly important in the study of host-pathogen systems, as different types of movements can influence the dynamics of the system (with respect to intra- and inter-specific host contacts and mixing). Depending upon the extent of the study area and partitioning of the available habitat by the study species, there can be inter-patch movements that within a disease transmission context can be characterized leading to inferences on the rates of mixing between patches. There can also be transient movements of individuals through a study area (i.e., migrants of some form), which enter temporarily and after some period of time, permanently leave. Individuals undergoing this form of movement have the potential to be carriers of disease or potentially contribute to the pool of susceptible individuals in another location. Another form of movement (either driven by biology or sampling design) is temporary emigration, which is manifest in the capacity of individuals to move outside of the observable boundaries of a study area (unavailable for capture), but returning later (Kendall et al. 1997). The degree and extent of such temporary movements from a patch(es) provides a useful measure for evaluating the degree of potential mixing between animals inside and outside of a given study area.

In a local-scale population, Faustino et al. (2004) found consistent evidence that survival of house finches varied as a function of disease state, with a negative effect of MG infection (expressing conjunctivitis) on survivorship. In addition, they showed that infection probabilities were consistently lower than recovery probabilities, which

in light of the recent finding that finches can become re-infected with MG in a laboratory setting (Sydenstricker et al. 2006), supports the modeling of this host-pathogen system under *SIS* or *SIRI* (*S*-susceptible, *I*-infectious, *R*-removed; for review of epidemiological models, see Anderson and May 1992) dynamics (Hosseini et al. 2004). Analyses in Faustino et al. (2004) represent the first use of multistate capture-mark-recapture (CMR) models to make inferences about the survival, infection, and recovery processes of a host-pathogen system. One of the strengths of multistate CMR models lies in the partitioning of combined survival and transition probability (\square) into separate probabilities of survival (*S*) and transition (\square) (Williams et al. 2002).

Work conducted by Jennelle et al. (submitted) conducted in the same study area as Faustino et al. (2004), also made use of multistate models, but focused on examining the influence of demographic and disease state effects on inter-patch movements of finches within a local scale study area. In addition, Jennelle et al. (submitted) show that finches exhibit significant variation in transient movements (functionally when an individual is observed once and never again inside the study area; a form of permanent emigration) as a function of disease state and age. Their work supports the notion that asymptomatic carriers play a critical role in the spread of MG across disparate populations (Hosseini et al 2004).

While Faustino et al. (2004) were able to establish basic differences in survivorship as a function of disease state and Jennelle et al. (submitted) estimated local within-patch movements and transient dynamics, two unresolved issues remained from this body of work. State-specific apparent survival estimates reported in Faustino et al. (2004) were negatively biased when comparing expected with observed counts of birds in subsequent seasons, which to some extent were accounted for in a transience analysis conducted in Jennelle et al. (submitted). Yet a key feature of finch behavior that must be accounted for is the phenomenon of temporary

emigration from the study area. In many field studies of vertebrates (particularly birds), individuals most likely move in and out of a given sampling area (especially when study areas are not defined by distinct geographic barriers), which effectively changes their availability for capture. From a parameter estimation perspective, increasing probabilities of random temporary emigration (the probability of emigrating from a study area in time i is not dependent on whether an animal was inside or outside the study area in time $i-1$) from a study area biases detection probabilities (p ; if it is defined as probability of detection, given presence in the study area), while the degree of bias due to Markovian emigration (which conditions the probability of emigrating from a study area in time i as a function of where an animal was before) depends on the relative tendency of animals to leave versus return to the study area (Kendall et al. 1997). Within the context of finch-MG dynamics, disease transmission is likely a function of direct contact with infectious individuals (Sydenstricker et al. 2006) and materials (Dhondt et al. in press). As such, it is a logical extension to infer that intraspecific mixing among finch aggregations increases (increasing the likelihood of disease transmission) as the rate of movement increases (with respect to movements within a study area and temporary movements out of a study area).

As finches exhibit seasonal variation in movements, and in particular I expect the Markovian form of temporary emigration (see Methods), it is necessary to account for this form of movement from both a technical and biological perspective. Thus, to account for the special form of capture heterogeneity induced by this type of movement, while simultaneously modeling state-specific temporary emigration and immigration of house finches at a local scale, I used multistate open robust design (MSORD) models (see Methods). This class of capture-mark-recapture (CMR) model is a very recent addition to the population biologist's toolkit, and this study represents one of the first applications of this methodology that I am aware of.

As previously noted, at a local spatial scale it has been shown that there is substantial structuring of house finch populations, likely as a function of the consistency of food supplies during the autumn and winter months and the distribution of adequate roosting sites (coniferous trees) (Dhondt et al. 2007, Jennelle et al. submitted). In addition, similar trends in disease prevalence emerge both at a local (Jennelle et al. submitted) and a regional spatial scale (Altizer et. 2004a), suggesting that the mechanism(s) driving these patterns can be resolved at a local spatial scale. As the existing body of work in Ithaca, NY suggests this study area is a representative microcosm of broader spatial scale finch-MG dynamics, I extended upon previous empirical works in this system and complemented previous analysis of temporary emigration by applying another recent methodological advance in CMR approaches (multistate reverse-time models; Nichols et al. 2000) to address a generally interesting problem in population studies: estimation of contributions to population growth. The recurring theme of uncertain correspondence between study area extent and activity area covered by a study species of interest, especially in the context of host-pathogen dynamics, warrants the estimation of proportional contributions to population growth of the infected and uninfected (in this case symptomatic and asymptomatic) components of a population. Estimation of this quantity complements the analysis of temporary emigration, as it provides a proportional measure of the degree of recruitment to the study area (a form of movement mentioned earlier). Regarding finch-MG dynamics, the estimation of recruitment to the local study area provides a quantitative means of assessing the degree to which the symptomatic and asymptomatic components of the local population are sustained from within and outside the study area. Overall, this approach permits inference on the most important direct determinants (drivers) of population dynamics in a system without having to directly estimate population sizes (Nichols et al. 2000).

Thus, I sought to complement the analysis of inter-patch movements within the local study area (Jennelle et al. in review) in order to synthesize the different forms of house finch movement occurring at a local spatial scale, ultimately to further our collective understanding of finch-MG dynamics. My overall objectives in this study were to (1) assess whether season, disease state, and age affect probabilities of temporary emigration and immigration of house finches from the study area, (2) estimate the proportional contribution of individuals to growth of the symptomatic and asymptomatic components of the population from within and outside the study area as a function of finch age and season, and (3) synthesize how this information contributes to understanding of disease dynamics in this system, and more generally how multistate robust design and reverse-time capture-recapture models can be used to account for different types of animal movements in ecological studies.

Methods

Study Area and Sampling

A detailed description of the data collection techniques and study area is presented in Faustino et al. (2004), so I will only highlight relevant details for this paper.

I used a subset of data from an intensive capture-mark-recapture (CMR) study in Ithaca, NY spanning from January 2002 through December 2004. Although data collection began in 2000, I excluded data from the first two years of study, since field efforts over this time were not standardized with weekly trapping and resighting periods at two fixed sites. Data were analyzed from 1 September through 15 April and within each year the dataset was divided into a Fall-Winter (FW: Sep-Dec) and Winter-Spring (WS: Jan-Apr) subset, each spanning 8 to 15 weeks, which coincides with significant changes in house finch social structure and behavior (Hill 1993). This framework was established to reduce the number of estimated parameters in CMR

models and to assess seasonal differences in movements and contributions to population growth.

Encounter data were derived from two types of events, (i) physical captures and recaptures (trapping), and (ii) resightings of marked individuals. Physical captures (via mist nets and cage traps) were conducted under permits from the New York State Department of Environmental Conservation, the U.S. Fish and Wildlife Service, and the U.S. Geological Survey. All procedures involving live animals were implemented under the Animal Use Protocol #00-90 issued by the Cornell University Institutional Animal Care and Use Committee. On a trapping occasion, I used a combination of two or three hand-built cylindrical wire-mesh cage traps and two or three 30mm mist-nets to capture birds. Each newly captured finch was fitted under permit with a unique sequence of a 9-digit numbered aluminum leg band (USGS Bird Banding Laboratory, Laurel, Maryland, USA) and a combination of three colored plastic leg bands. Standard demographic and morphological measurements were recorded (Pyle 1997) including age (finches were assigned as juveniles if it was their first winter, and adults if it was at least their second winter), gender, as well as disease state. Each bird was evaluated for disease status by recording the severity of conjunctivitis in both eyes at each encounter, using a binary ranking: 'I' (symptomatic) indicating some level of conjunctivitis, or 'U' (asymptomatic). Conjunctivitis is a very useful proxy for assessing MG infection status, and correlates highly with PCR analysis of eye swab (conjunctival tissue) samples and culture for MG organisms (Hartup et al. 2001). During resighting events, observers recorded color band combinations and disease state of marked finches from inside a vehicle using a high power spotting scope and/or binoculars. After 2003, resighting was conducted twice per week at each of the two primary banding sites. In some cases only one eye of a given marked individual could be assessed for disease state, and if asymptomatic, resulted in uncertainty in assigning

a final classification (as the other eye could be symptomatic). Relative to the dataset, these instances were very infrequent (<5% of resighting observations), but were treated as non-observations when present (thus representing a loss of information).

Each week, trapping was conducted every Tuesday and Wednesday on the two primary study sites ('Golf course' and Liddell Field Station known as 'Beelab'), while resighting was conducted every Thursday and Friday. Both sites are representative of suburban landscapes, with the Golf course composed of mowed greenways, interspersed with spruce (*Picea* spp.) trees, early successional scrub habitat, and housing developments. The Beelab, however, is surrounded by a matrix of agricultural fields and deciduous forest cover, with fewer surrounding housing developments. While these sites are approximately 1.5 km apart, they are oriented almost exactly along an east-west gradient.

I maintained and stocked between 8 to 10 tube-style feeders with black oil sunflower seeds at the field sites to attract finches. While baiting stations can lead to bias in detection probabilities as baiting can increase the likelihood of encountering previously captured individuals (Pradel 1993, Williams et al. 2002), finches are backyard 'feeder birds'. In the eastern finch populations, birds rely almost solely on feeders for subsistence during the non-breeding season (Hill 1993), and there is little reason to expect significant trap effects due to the use of baited feeders with respect to subsequent visual resighting encounters. I cannot rule out, however, the influence of physical trapping on the extent of trap shyness or affinity. Since there were many fewer live captures of finches relative to resightings, I believe this should not induce substantial bias in parameter estimates.

Analysis of Encounter Data: Estimation of Temporary Emigration/Immigration

To account for state-specific probabilities of temporary emigration and immigration in the local study area, I used multistate open robust design (MSORD) models, which combine multistate models (Arnason 1973, Brownie et al. 1993) with an extension to Pollock's robust design models (Pollock 1982, Schwarz and Stobo 1997, Kendall et al. 1997, Kendall and Bjorkland 2001). Standard robust design models incorporate two sources of information, utilizing data collected over k primary sampling periods to estimate survival rates and l secondary periods to estimate capture probabilities and population size. Sufficiently long periods of time are maintained between i primary sampling periods, making use of the Jolly-Seber (JS) live recapture model (Jolly 1965, Seber 1965), coupled with sampling over closely spaced time intervals (typically 1 day) over l secondary periods, which is amenable for estimation of capture probabilities using closed-population models (Otis et al. 1978). In Kendall and Bjorkland (2001), the assumption of a closed population (with respect to emigration/immigration, not mortality) during the secondary sampling periods is relaxed, allowing for flexibility in the CMR sampling design. Multistate models on the other hand allow for estimation of state-specific apparent survival, detection, and transition probabilities between physical, physiological, or behavioral states (Arnason 1973, Brownie et al. 1993).

As house finches are very mobile organisms (Hill 1993, Able and Belthoff 1998) and are likely to have a home range that exceeds the boundaries of the study area, I used MSORD models to avoid the restrictive closure assumptions of standard robust design models (Kendall and Nichols 1995, Kendall et al. 1997) during sampling within primary periods. These models are predicated on the assumption that the sampled population is closed to both additions (births and immigration) and deletions (deaths and emigration) for all samples within each primary period. However, Kendall

(1999) demonstrated that if the closure assumption is violated by either immigration or emigration into or out of the study area (which can be a frequent occurrence in many studies), then use of robust design models (Kendall et al. 1997) are still appropriate for estimating temporary emigration. By using open robust design models, individuals are allowed to make one entry and one exit from the study area, while mortality and recruitment are not permitted within each primary period (Kendall and Bjorkland 2001). Aside from standard CMR and multistate assumptions (see Williams et al. 2002, and Jennelle et al. submitted for treatment of multistate assumptions with respect to this study system), the MSORD model assumes that all animals (of a given stratification), even those entering the study area, have the same survival probability within a given primary period (Kendall and Bjorkland 2001).

The open robust design is nested within a general multistate framework. In this study, four states are required to specify the system, including asymptomatic individuals in the sampling area (**U**), asymptomatic individuals outside the sampling area (**u**), symptomatic individuals inside the study area (**I**), and symptomatic individuals outside the study area (**i**). Birds located in the study area are by definition potentially observable (with probability p) and temporary emigrants are considered unobservable (if an animal is outside the study area, it is not available for observation), which is treated implicitly in the modeling of the four aforementioned states.

The field sampling procedures could only accommodate the structure for a robust design CMR analysis for the final year of the study. Resighting data were organized and collated into individual encounter histories by season. Both the winter-spring season (WS: Jan-Apr) and the fall-winter season (FW: Sep-Dec) of 2004 contained 13 primary periods (1-week duration per primary period) with two embedded secondary periods (24hr duration between secondary periods) per primary.

I fixed survival of observable and unobservable disease state-specific probabilities to be equal (i.e., $S_i^U = S_i^u$, $S_i^I = S_i^i$), with variation modeled as a function of disease state and age (only for asymptomatic birds). I believe the survival constraint was justifiable since I expected the study area represented a subsample from a matrix of available finch habitat (i.e., that survival within a disease state should be independent of whether the individual was observable – by virtue of falling within the segment of the habitat matrix being sampled – or unobservable). I also accounted for a one-period duration transient effect (*sensu* Jennelle et al. submitted), with the exception of symptomatic finches in FW2004 (due to insufficient data). Similarly, primary observable encounter probabilities were modeled as an interaction between disease state and age (unobservable encounter probabilities were set to zero), while observable (within the study area) infection (transition from the asymptomatic state to the symptomatic state between time i and $i+1$) and recovery probabilities (transition from the symptomatic state to the asymptomatic state between time i and $i+1$) were held constant over time (Faustino et al. 2004, Jennelle et al. submitted). Partially observable infection and recovery probabilities were set equal, respectively ($\psi_i^{Ui} = \psi_i^{ui}$), ($\psi_i^{Ii} = \psi_i^{ii}$) to facilitate parameter convergence, but were not readily interpretable as movement was confounded with disease state transition. Partial observability in this context is simply defined as a case where either the source or destination state, but not both, is observable. Completely unobservable (source and destination state are unobservable) infection and recovery probabilities were set equal to zero ($\psi_i^{ui} = \psi_i^{ii} = 0$), as these transition parameters were inestimable. Given only two secondary occasions per primary period and insufficient data over the secondary occasions in most cases, I set finch probabilities of entry into the study area ($pent_{i1}^s$, Table 4.1 - program MARK convention equivalent to β_{ij} in Kendall and Bjorkland 2001) within primary periods equal to zero (since the first entry probability is

estimated by subtraction in program MARK, all $pent_{i1}^s$ parameters were effectively 1.0, meaning there were no new entries of individuals over the secondary occasions). I also constrained ϕ_{ija}^s to be constant across disease state, age, and time within primary periods (this parameter was later set to 1, as estimates were very close to unity).

The parameters of interest in the model set were ψ_i^{Uu} (probability of a marked asymptomatic finch emigrating from the study area between time i and $i+1$), ψ_i^{uU} (probability of a marked asymptomatic finch immigrating into the study area between time i and $i+1$), ψ_i^{Ii} (probability of a marked symptomatic finch emigrating from the study area between time i and $i+1$), and ψ_i^{iI} (probability of a marked symptomatic finch immigrating into the study area between time i and $i+1$).

Specifically, I was interested in addressing the hypothesis that temporary movements of symptomatic finches are less than those of asymptomatic finches, i.e.,

$H_a: (\psi_i^{Uu} > \psi_i^{Ii}, \psi_i^{uU} > \psi_i^{iI})$. Given that finches are largely dependent on bird feeders for sustenance during the non-breeding season (at least in the northeastern US), and the decision to enter or leave a feeder site poses a tradeoff between resource attraction and competition (intra- and inter-specific), I expected temporary emigration and immigration probabilities to be first-order Markovian as opposed to random. First-order Markovian movements (in this case) imply that emigration or immigration probabilities in time i are dependent on the location of an individual in time $i-1$. Thus, a total of 38 models were built in the set to evaluate combinations of the equality of these parameters and whether they varied as a function of age.

Table 4.1. Estimable parameters from multistate open robust design (MSORD) model for the primary and secondary sampling levels. Adapted from robust design chapter 16 in Cooch and White (2006). For consideration in our application of MSORD models, the states r and s can be U (asymptomatic inside the study area), u (asymptomatic outside the study area), I (symptomatic inside the study area), and i (symptomatic outside the study area).

Primary	
level	Definition
S_i^r	Survival probability in state r from time i to $i+1$
ψ_i^{rs}	Probability that an animal in state r at primary period i is in state s at primary period $i+1$, given it is alive at $i+1$
p_i^r	The probability that an observable individual in the study area in state r is encountered in primary period i
Secondary	
level	Definition
ϕ_{ija}^s	Probability that an individual in the study area in state s during primary period i and capture occasion j , who first arrived in the study area a capture occasions previously, is still in the study area at capture occasion $j+1$.
$pent_{ij}^s$	Probability that an individual in state s in primary period i is a new arrival (within that primary period) to the study area at capture occasion j . Note that the first entry probability is derived by subtraction in program MARK.

There are no formal goodness-of-fit tests available for MSORD models, nor can a bootstrap or median \hat{c} approach be used to estimate an overdispersion correction factor (\hat{c}) to account for the potential of extra binomial variation in the data. To account for this possibility, I examined the relative rank order and degree of support (QAIC_c weight) of models by varying \hat{c} from 1.0 to 2.0. Increasing values of \hat{c} imply greater lack of fit, and incorporation of larger values of \hat{c} in QAIC_c calculations is more conservative, increasing the support for reduced parameter models. While the ordering and relative support for models did not change appreciably within the range of \hat{c} values incorporated, I assigned a conservative value of 1.5 for both seasonal model sets to account for factors (e.g., non-independence of individuals, sparse encounters with symptomatic birds) that might exacerbate a lack of fit of the most general models to the data (see Faustino et al. 2004 and Jennelle et al. submitted for details).

Analyses were conducted for each season separately to make modeling more tractable and all models were fit to the data using program MARK (v. 4.3; White and Burnham 1999). Selection among models in the candidate 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 and Anderson 2002). This particular information criterion was used to select the best approximating model for the data based on the principles of parsimony, which balances the trade-off between under- and over-fitting models (Burnham and Anderson 2002). The model in the candidate model set closest to “truth” was that with the lowest QAIC_c values, and other models were ranked relative to deviations from this model (Δ QAIC_c). I made comparisons among models in the candidate set by evaluating an index of relative plausibility, based upon normalized Akaike weights (w_i ; Burnham and Anderson 2002). When Δ QAIC_c values (of respective models) are organized in ascending order,

the ratio of w_i between any two models indicates the relative (proportional) support for the model with greater Akaike weight.

To account for model selection uncertainty, I used model averaging (weighting model-specific parameter estimates by normalized QAICc weights) over the set of candidate models to arrive at robust estimates of state-specific emigration and immigration probabilities.

Analysis of Encounter Data: Contributions to Population Growth

To estimate contributions to the growth of the local population I used a reverse-time capture-recapture approach (Nichols et al. 2000) with program MSSURVIV (Hines 1994). The basic methodology in the case of one state involves reversing the time sequence of encounter histories and analyzing the data with a Cormack-Jolly-Seber (CJS) open population model (Pollock et al. 1974). While estimation of apparent survival and detection probability under the CJS model is conditioned on the first release of individuals, the reverse-time approach conditions on the last time an animal was captured or seen (although a full likelihood approach is presented in Pradel 1996). An extension to account for multiple physical or physiological states is easily accommodated and proceeds in the same fashion as a standard multistate analysis (Arnason 1973, Hestbeck et al. 1991, Brownie et al. 1993, Schwarz et al. 1993).

In a reverse time multistate framework, the emphasis is not on estimation of state-specific survival, but rather on the parameter γ_i^{rs} , which is the probability that a member of the population in state r at time i was a member of the population in state s at time $i-1$. This is equivalent to estimating the product of state-specific survival and transition on the reversed encounter histories. The parameter γ_i^{rs} in this case can be interpreted as the contribution of animals in state s at time $i-1$ to population growth (realized λ) of animals in state r at time i (Nichols et al. 2000).

I was primarily interested in estimating contributions to population growth as a function of the disease state of individuals, but I also stratified the analyses by different age classes (juvenile or adult), as this factor contributed to both variation in the proportion of transients and accounted for significant variation in the probability of expressing conjunctivitis in house finches (Jennelle et al. submitted). In order to estimate the contributions to population growth of both asymptomatic and symptomatic disease classes of finches, while accounting for age differences, I conducted two separate analyses.

The first analysis was conducted in order to estimate contributions to population growth of symptomatic finches from both asymptomatic birds stratified by age within the local population and symptomatic immigrants. Encounter data were stratified into three states (asymptomatic adults (A), asymptomatic juveniles (J), and a general symptomatic class (I)), organized in reverse time order, and analyzed in program MSSURVIV. Over the course of a given season, the age of a given individual did not change, so treatment of age as a static state was justified.

I evaluated three models in each year and season to estimate contributions to population growth of symptomatic finches (γ_i^{Is}) in the local population from asymptomatic adults (γ_i^{IA}) and juveniles (γ_i^{IJ}), resident symptomatic cases (γ_i^{II}), and symptomatic recruits from outside the study area ($1 - (\gamma_i^{IA} + \gamma_i^{IJ} + \gamma_i^{II})$). The three models included $\{\gamma_i^{IA}, \gamma_i^{IJ}, \gamma_i^{II}\}$, $\{\gamma_i^{IA} = \gamma_i^{IJ}, \gamma_i^{II}\}$, and $\{\gamma_i^{IA} = \gamma_i^{IJ} = \gamma_i^{II}\}$, and were designed to test for equality in the contributions of the different state-specific components of the population as well as symptomatic recruits. For each of these models, I maintained state-specific variation in γ_i^{As} and γ_i^{Js} . Previous work in this system has shown that encounter probabilities can vary by disease state, age, and time (Faustino et al. 2004, Jennelle et al. submitted), so variation in encounter probabilities

was modeled as a function of an interaction between asymptomatic finches, age, and time with symptomatic finches invariant over time (due to small sample sizes).

The second reverse-time analysis was conducted in order to estimate contributions to population growth of asymptomatic finches from both symptomatic birds stratified by age within the local population and asymptomatic immigrants. Again, encounter data were stratified into three states (symptomatic adults (a), symptomatic juveniles (j), and a general asymptomatic class (U)), organized in reverse time order, and analyzed in program MSSURVIV (Hines 1994).

As before I evaluated three models in each year and season, to estimate contributions to population growth of asymptomatic finches (γ_i^{Us}) in the local population from symptomatic adults (γ_i^{Ua}) and juveniles (γ_i^{Uj}), resident asymptomatic cases (γ_i^{UU}), and asymptomatic recruits $1 - (\gamma_i^{Ua} + \gamma_i^{Uj} + \gamma_i^{UU})$ from outside the study area. The three models included $\{\gamma_i^{Ua}, \gamma_i^{Uj}, \gamma_i^{UU}\}$, $\{\gamma_i^{Ua} = \gamma_i^{Uj}, \gamma_i^{UU}\}$, and $\{\gamma_i^{Ua} = \gamma_i^{Uj} = \gamma_i^{UU}\}$, and were designed to test for equality in the contributions of the different state-specific components of the population as well as asymptomatic recruits. For each of these models, I maintained state-specific variation in γ_i^{as} and γ_i^{js} , while variation in encounter probabilities was modeled as a function of an interaction between asymptomatic finches and time, with encounter probabilities of symptomatic adult and juvenile finches invariant over time (due to small sample sizes).

As with the analysis of temporary emigration/immigration, I used QAICc (Burnham and Anderson 2002) to evaluate the relative fit of models to the data, and all structurally consistent parameters were model averaged to account for model selection uncertainty.

Results

In this study, I used information from 666 initial captures, 709 recaptures, and 5357 resightings of house finches in Ithaca, NY to produce 562 and 1655 encounter histories for the MSORD and reverse-time analyses, respectively. For the MSORD analyses, only the information from resighting occasions from the WS2004 and FW2004 seasons were used. Similarly as in previous CMR analyses of this system (Faustino et al. 2004, Jennelle et al. submitted), I was only able to impose a relatively simple structure on the model sets with respect to partitioning variation in parameters of interest. This limitation was primarily due to the disproportionately lower number of captures of symptomatic as opposed to asymptomatic finches.

Temporary Emigration/Immigration

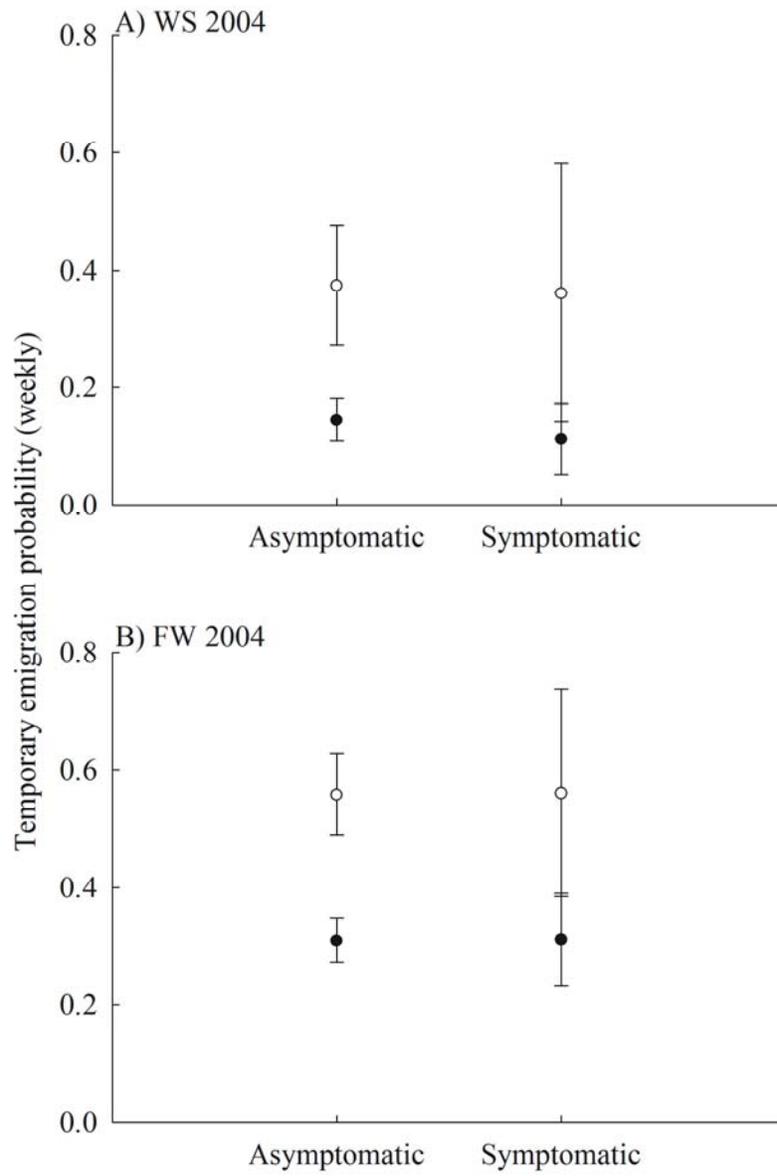
Since entry probabilities (within primary periods) were set to zero, and ϕ was estimated as 0.95 in WS2004 and set to 1.0 in FW2004, the models essentially mimicked a closed-robust design.

In the WS2004 season, the best fitting model ($\{\psi^{Lu} = \psi^{Li}(\cdot) \psi^{Uu} = \psi^{Uj}(\cdot)\}$; QAIC_c weight = 27.6%) partitioned separate emigration and immigration probabilities, but there was no difference in these movements as a function of either disease state or age (Table 4.2). While there was no single model structure that was overwhelmingly supported by the data, there was overall very little support for age or disease effects in the data (Table 4.2). Model averaged estimates over all structurally relevant disease state-specific movement probabilities revealed that while there were no differences between symptomatic and asymptomatic finches, differences between emigration and immigration probabilities were quite distinct (Fig. 4.1A). In fact, emigration movements were half as likely as immigration.

Table 4.2. Summary of multistate open robust design (MSORD) analysis of live encounter data for 2004, in Ithaca, NY, USA. Each season was analyzed separately in subsets: *WS* = ‘Winter-Spring’ (Jan-Mar) and *FW* = ‘Fall-Winter’ (Sep-Dec). Each of the 13 primary periods spanned seven days, while the time between two embedded secondary occasions was 24 hours. Only variation in state-dependent temporary emigration and immigration probabilities were modeled, and a model set was generated that explored variation in these parameters as functions of disease state and age. *Model notation:* ψ^{Uu} (emigration of asymptomatic finches between times i to $i+1$), ψ^{uU} (immigration of asymptomatic finches between times i to $i+1$), ψ^{Ii} (emigration of symptomatic finches between times i to $i+1$), and ψ^{iI} (immigration of symptomatic finches between times i to $i+1$). The symbol “.” within parenthesis indicates time invariance of the modeled parameter. Lower ΔQAIC_c values show better fit. Only models comprising $\geq 75\%$ of QAIC_c weight are listed in decreasing order of parsimony. The Akaike weights indicate the relative support for each model, given the data. The Deviance is the difference in $-2 \cdot (\log \text{Likelihood})$ between a selected model and the saturated model (a model with the number of parameters equal to the sample size). A correction for overdispersion (\hat{c}) was conservatively assigned as 1.5 for each season.

Model components				Model Statistics				
Season	ψ^{Uu}	ψ^{Ji}	$\psi^{\mu U}$	ψ^{Il}	Δ QAIC _c	QAIC _c weight (%)	Deviance	Parameters
<i>WS2004</i>	$\psi^{Uu} = \psi^{Ji}(\cdot)$		$\psi^{\mu U} = \psi^{Il}(\cdot)$		0.00	27.6	3495	18
	$\psi^{Uu}(\cdot)$	$\psi^{Ji}(\cdot)$	$\psi^{\mu U} = \psi^{Il}(\cdot)$		1.75	11.5	3495	19
	$\psi^{Uu} = \psi^{Ji}(\cdot)$		$\psi^{\mu U}(\cdot)$	$\psi^{Il}(\cdot)$	1.99	10.2	3495	19
	$\psi^{Uu} = \psi^{Ji} = \psi^{\mu U} = \psi^{Il}(\cdot)$				2.68	7.2	3500	17
	$\psi^{Uu}(\cdot)$	$\psi^{Ji} = \psi^{Il}(\cdot)$	$\psi^{\mu U}(\cdot)$		2.70	7.2	3496	19
	$\psi^{Uu} = \psi^{Ji}(\text{age})$		$\psi^{\mu U} = \psi^{Il}(\cdot)$		3.70	4.3	3495	20
	$\psi^{Uu}(\cdot)$	$\psi^{Ji}(\cdot)$	$\psi^{\mu U}(\cdot)$	$\psi^{Il}(\cdot)$	3.73	4.3	3495	20
	$\psi^{Uu}(\cdot)$	$\psi^{Ji} = \psi^{Il}(\cdot)$	$\psi^{\mu U}(\text{age})$		4.19	3.4	3493	21
<i>FW2004</i>	$\psi^{Uu} = \psi^{Ji}(\cdot)$		$\psi^{\mu U} = \psi^{Il}(\cdot)$		0.00	36.1	2580	18
	$\psi^{Uu} = \psi^{Ji}(\cdot)$		$\psi^{\mu U}(\cdot)$	$\psi^{Il}(\cdot)$	1.95	13.6	2579	19
	$\psi^{Uu}(\cdot)$	$\psi^{Ji}(\cdot)$	$\psi^{\mu U} = \psi^{Il}(\cdot)$		1.99	13.4	2580	19
	$\psi^{Uu}(\cdot)$	$\psi^{Ji} = \psi^{Il}(\cdot)$	$\psi^{\mu U}(\cdot)$		2.49	10.4	2580	19
	$\psi^{Uu} = \psi^{Ji}(\cdot)$		$\psi^{\mu U} = \psi^{Il}(\text{age})$		3.82	5.4	2579	20
	$\psi^{Uu}(\cdot)$	$\psi^{Ji}(\cdot)$	$\psi^{\mu U}(\cdot)$	$\psi^{Il}(\cdot)$	3.93	5.1	2579	20

Figure 4.1. Estimated weekly probabilities (± 1 SE) of emigration (●; probability that an individual inside the study area in time i is outside the study area in time $i+1$) and immigration (○; probability that an individual outside the study area in time i is inside the study area in time $i+1$) of house finches in Ithaca, NY. Panel (A) represents estimates from the winter-spring (WS) season of 2004, and panel (B) represents estimates from the fall-winter (FW) season of 2004.



In the FW2004 season, again the structure of the best fitting model ($\{\psi^{Uu} = \psi^i(.)\}$ $\psi^{uU} = \psi^i(.)\}$; QAIC_c weight = 36.1%) partitioned variation in the data as a function of separate emigration and immigration probabilities, but did not contain a disease or age effect (Table 4.2). Though there was some support for models with disease effects for the movement parameters, age effects were very weakly supported. Model-averaged estimates over structurally relevant movement parameters showed a similar overall pattern to the WS2004 season, where emigration was approximately half as likely as immigration in any given week (Fig. 4.1B). There were marked seasonal differences in estimates of emigration and immigration probability; however, with winter-spring movements almost half as likely as fall-winter movements (Fig. 4.1).

Contributions to Population Growth

For the WS2002 and FW2002 seasons, the data completely supported (QAIC_c weight=100%) the model for contributions to population growth of symptomatic house finches as a function of state (Table 4.3). In the remaining four seasons (WS2003 through FW2004), the data did not unequivocally support the state-specific model, and there was varying degrees of support for equal contributions to population growth of symptomatic finches from juvenile and adult asymptomatic birds (Table 4.3).

In all of the FW seasons, juveniles were more important to population growth of the symptomatic class as compared with adult finches (Fig. 4.2). With the exception of WS2002, adults contributed equally or more to population growth of symptomatic birds in the population during WS seasons, as compared to juvenile finches. During all years and seasons, resident symptomatic finches contributed the most weight proportionally to overall maintenance and growth of the symptomatic class of finches in the local population (Fig. 4.2). In none of the seasons analyzed did symptomatic

recruits contribute more than approximately 10% to overall population growth of the symptomatic class.

For every year and season (except FW2004), the data supported a model for equal contribution to population growth of the asymptomatic class of house finches from symptomatic adults and juveniles (QAIC_c=100%; Table 4.4). In FW2004, the state-specific model for contributions to the asymptomatic class was overwhelmingly supported by the data (QAIC_c=100%; Table 4.4). During all years and seasons, contributions to population growth of the asymptomatic class of house finches from symptomatic adults and juveniles were less than 7% (Fig. 4.3). Contributions to population growth from asymptomatic recruits were always greater during the FW seasons (not exceeding 20%), and were twice as important compared with subsequent WS seasons. The greatest contributions to population growth of asymptomatic finches in all years and seasons were from resident asymptomatic individuals (>75%; Fig. 4.3).

Table 4.3. Summary of reverse-time multistate analysis of live encounter data from 2002 to 2004, in Ithaca, NY, USA. This analysis was designed to evaluate contributions to population growth of symptomatic house finches. Each season was analyzed separately in subsets: *WS* = ‘Winter-Spring’ (Jan-Mar) and *FW* = ‘Fall-Winter’ (Sep-Dec). Each season was composed of 16 periods (except *WS* 2004 - 13 periods), which in turn spanned seven days. All data were pooled over period (an individual seen multiple times was counted only once). Variation in detection probabilities was fixed according to major sources of variation in this system (Faustino et al. 2004, Jennelle et al. submitted). It was modeled as a function of an interaction between state A (asymptomatic adult finches), state J (asymptomatic juvenile finches), and period, while state I (all symptomatic finches) was time invariant. Only variation in γ^{IS} was modeled (the probability that an individual alive and in the symptomatic state I at time i was in state S at time $i-1$). Three models consisting of all possible combinations of state-specific variation in γ^{IS} were included in the set. Lower ΔQAIC_c values show better fit. Only models comprising $\geq 99\%$ of QAIC_c weight (cumulative over the set) are listed in decreasing order of parsimony. The Akaike weights indicate the relative support for each model, given the data. A correction for overdispersion (\hat{c}) was estimated and is accounted for in QAIC_c calculations in program MSSURVIV.

Model components		Model Statistics			
Season	γ^{RS}	QAICc		Likelihood	Par
		weight (%)	Δ QAICc		
<i>WS</i> 2002	$\gamma^A(\text{state})\gamma^J(\text{state})\gamma^I(\text{state})$	100	0.00	-150.072	40
<i>FW</i> 2002	$\gamma^A(\text{state})\gamma^J(\text{state})\gamma^I(\text{state})$	100	0.00	-479.387	40
<i>WS</i> 2003	$\gamma^A(\text{state})\gamma^J(\text{state})\gamma^I(\text{state})$	82.5	0.00	-233.138	40
	$\gamma^A(\text{state})\gamma^J(\text{state})\gamma^I(\text{IA} = \text{IJ}, \text{II})$	17.5	3.10	-235.926	39
<i>FW</i> 2003	$\gamma^A(\text{state})\gamma^J(\text{state})\gamma^I(\text{IA} = \text{IJ}, \text{II})$	57.6	0.00	-288.990	39
	$\gamma^A(\text{state})\gamma^J(\text{state})\gamma^I(\text{state})$	42.4	0.61	-288.110	40
<i>WS</i> 2004	$\gamma^A(\text{state})\gamma^J(\text{state})\gamma^I(\text{IA} = \text{IJ}, \text{II})$	75.1	0.00	-259.346	33
	$\gamma^A(\text{state})\gamma^J(\text{state})\gamma^I(\text{state})$	24.9	2.21	-259.345	34
<i>FW</i> 2004	$\gamma^A(\text{state})\gamma^J(\text{state})\gamma^I(\text{state})$	59.7	0.00	-379.149	40
	$\gamma^A(\text{state})\gamma^J(\text{state})\gamma^I(\text{IA} = \text{IJ}, \text{II})$	28.5	1.48	-380.979	39
	$\gamma^A(\text{state})\gamma^J(\text{state})\gamma^I(\text{IA} = \text{IJ} = \text{II})$	11.8	3.25	-382.954	38

Table 4.4. Summary of reverse-time multistate analysis of live encounter data from 2002 to 2004, in Ithaca, NY, USA. This analysis was designed to evaluate contributions to population growth of asymptomatic house finches. Each season was analyzed separately in subsets: *WS* = ‘Winter-Spring’ (Jan-Mar) and *FW* = ‘Fall-Winter’ (Sep-Dec). Each season was composed of 16 periods (except *WS* 2004 - 13 periods), which in turn spanned seven days. All data were pooled over period (an individual seen multiple times was counted only once). Variation in detection probabilities was fixed according to major sources of variation in this system (Faustino et al. 2004, Jennelle et al. submitted). It was modeled as a function of an interaction between the U-state (all asymptomatic finches) and period, and time invariance for symptomatic adult finches (state a) and symptomatic juvenile finches (state j). Only variation in γ^{US} was modeled (the probability that an individual alive and in the asymptomatic state U at time i was in state S at time $i-1$). Three models consisting of all possible combinations of state-specific variation in γ^{US} were included in the set. Lower $\Delta QAIC_c$ values show better fit. Only models comprising $\geq 99\%$ of $QAIC_c$ weight (cumulative over the set) were listed in decreasing order of parsimony. The Akaike weights indicate the relative support for each model, given the data. A correction for overdispersion (\hat{c}) was estimated and is accounted for in $QAIC_c$ calculations in program MSSURVIV.

Model components		Model Statistics			
Season	γ^{RS}	QAICc weight (%)	Δ QAICc	Likelihood	Par
<i>WS</i> 2002	$\gamma^a(\text{state})\gamma^j(\text{state})\gamma^U(\text{Ua} = \text{Uj}, \text{UU})$	100	0.00	-127.276	23
<i>FW</i> 2002	$\gamma^a(\text{state})\gamma^j(\text{state})\gamma^U(\text{Ua} = \text{Uj}, \text{UU})$	100	0.00	-424.068	23
<i>WS</i> 2003	$\gamma^a(\text{state})\gamma^j(\text{state})\gamma^U(\text{Ua} = \text{Uj}, \text{UU})$	100	0.00	-196.039	23
<i>FW</i> 2003	$\gamma^a(\text{state})\gamma^j(\text{state})\gamma^U(\text{Ua} = \text{Uj}, \text{UU})$	100	0.00	-264.577	23
<i>WS</i> 2004	$\gamma^a(\text{state})\gamma^j(\text{state})\gamma^U(\text{Ua} = \text{Uj}, \text{UU})$	100	0.00	-240.648	20
<i>FW</i> 2004	$\gamma^a(\text{state})\gamma^j(\text{state})\gamma^U(\text{state})$	100	0.00	-293.913	24

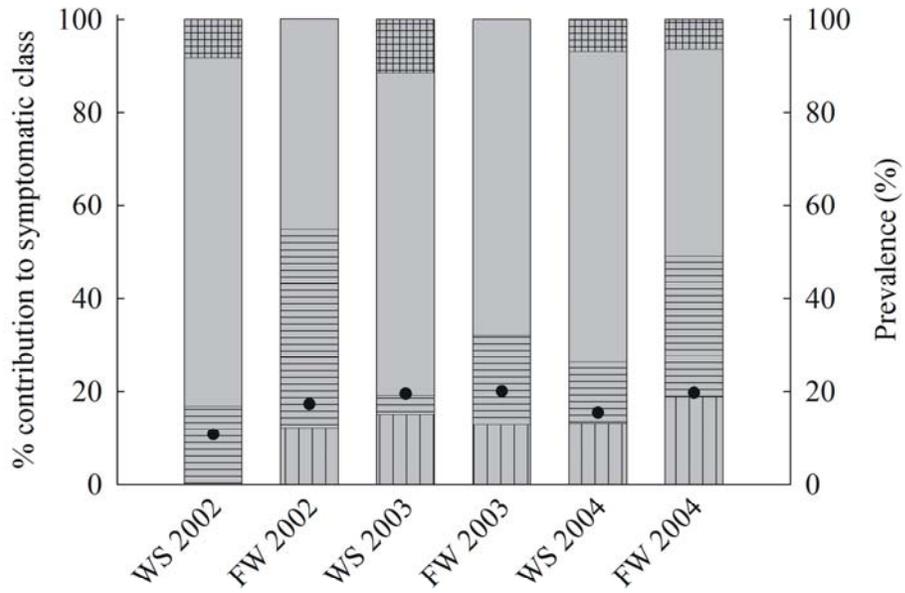


Figure 4.2. Estimated proportional contributions (weekly) to the symptomatic component of the Ithaca, NY house finch population by season (WS: winter-spring, FW: fall-winter) and year (2002-2004). Sources of population growth are from asymptomatic adults (γ^{IA} ; ▨), asymptomatic juveniles (γ^{IJ} ; ▩), symptomatic finches (no age specificity; γ^{II} ; ▧), and symptomatic recruits from outside the study area ($1-\gamma^{IA}-\gamma^{IJ}-\gamma^{II}$; ▤). Superimposed is average seasonal disease prevalence (•).

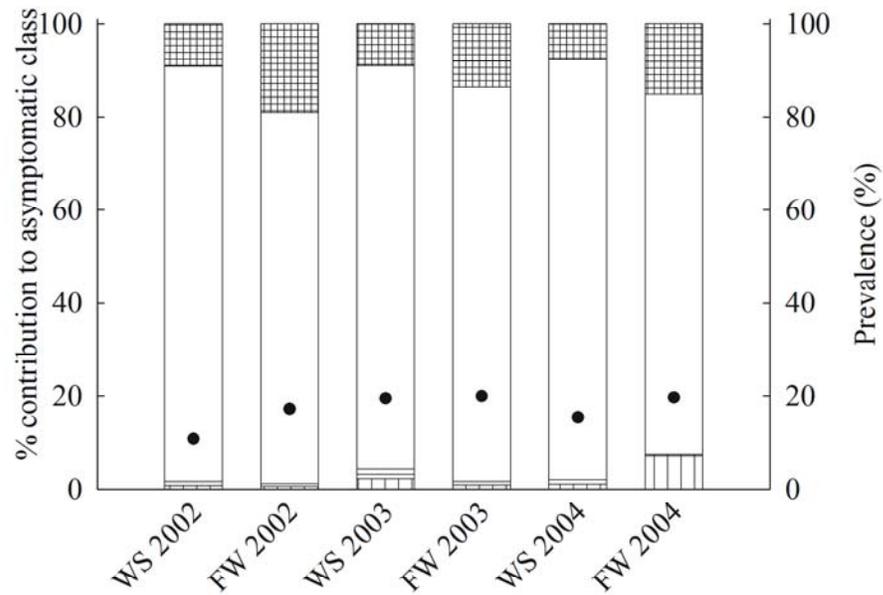


Figure 4.3. Estimated proportional contributions (weekly) to the asymptomatic component of the Ithaca, NY house finch population by season (WS: winter-spring, FW: fall-winter) and year (2002-2004). Sources of population growth are from symptomatic adults (γ^{Ua} ; ▧), symptomatic juveniles (γ^{Uj} ; ▨), asymptomatic finches (no age specificity; γ^{UU} ; ▩), and asymptomatic recruits from outside the study area ($1-\gamma^{Ua}-\gamma^{Uj}-\gamma^{UU}$; ▦). Superimposed is average seasonal disease prevalence.

Discussion

Implications of Temporary Emigration and Immigration

Although this study did not focus explicitly on addressing survival differences between symptomatic and asymptomatic house finches, the model structure for survivorship that I imposed (based upon work from Faustino et al. 2004 and Jennelle et al. submitted) expressed differences in apparent survival of finches as a function of disease state and a transient effect. While the effect of transient individuals can negatively bias survival estimates (Pradel et al. 1997) and was a significant factor in the finch-MG system (Jennelle et al. submitted), temporary emigration from a study area can produce similar bias (Kendall et al. 1997). Although I did not find evidence for a disease state or age effect in the current analyses of these types of temporary movement, there were significant seasonal differences in the probabilities of temporary emigration and immigration in the system (Fig. 4.1). Accounting for this source of detection heterogeneity and a transient effect (a form of permanent emigration from a study area; Pradel et al. 1997) improved estimates of state-specific survivorship (mitigating bias from temporary emigrants and transient individuals), which was a point of concern in Faustino et al. (2004). Estimates of survivorship for both disease states (after accounting for transience) from MSORD models were always greater (by as much as 35%) than analogous estimates reported in Faustino et al (2004). As in Faustino et al. (2004), symptomatic finches exhibited lower survivorship than asymptomatic birds.

With respect to finch-MG dynamics, the seasonal analysis of temporary movements has several important implications. Within the spatial context of the study area, temporary movements of birds did not appear to be a function of disease state or age. While MG infection does not limit such local scale movements of finches,

substantially higher probabilities of temporary emigration and immigration during the fall-winter season (Fig. 4.1) supports the notion that there are distinct seasonal patterns in the degree of mixing between finch aggregations. The greater rate of mixing and/or spatial activity range in the autumn (as suggested by the analysis) highlights an important feature of finch behavior that contributes to seasonality (Hosseini et al. 2004) in the dynamics of this host-pathogen system. All in all, the results imply that permanently stocked bird feeders, coupled with adequate roost sites (Dhondt et al. 2007, Jennelle et al. submitted) likely serve to attract and retain finches to a given area (given consistently greater probabilities of immigration; see also discussion of contributions to population growth below), thus serving as an artificial spacing mechanism.

Hawley et al. (2006) found that experimentally increased intraspecific competition (resulting in increased aggression between house finches) lowered the immune response of individuals. By extension, I would expect that increasing the density of bird feeders at a site would reduce intraspecific competition (thereby dampening competition-mediated immune suppression) and perhaps diffuse the density of infectious *M. gallisepticum* on a confirmed fomite (bird feeders; Dhondt et al. in press) (decreasing the likelihood of successful infection of finches). Considering this together with the finding that the study area (with permanently stocked feeders) attracts and retains aggregations of finches, I believe that manipulation of the spatial arrangement and density of bird feeders (in large part) across the suburban landscape could serve to slow or arrest the spread and maintenance of MG induced conjunctivitis in house finches. This multiscale effect of bird feeders (at the subpopulation or aggregate level and individual level) is worth considering if MG evolves to a more virulent form (affecting finches and/or other host birds), which warrants management or containment strategies.

With respect to the application of MSORD models in the study, I encountered several technical limitations to the analysis. First, I could only accommodate two secondary sampling occasions within each primary sampling period, and this precluded me from exploring hypotheses about arrival and departure probabilities (Table 4.1; useful parameters with respect to studies of migration and colonial breeding among others). This study also suffered from disproportionately fewer encounters of symptomatic than asymptomatic finches, which was simply due to relatively low disease prevalence in the population (Figs 4.2 & 4.3). Compounded with the high dimensionality of potentially estimable parameters in the models, I was forced to constrain models with relatively simple structures, restricting the use of models with parameters that included interactions between factors and temporal variation. In addition, the complicated relationships between state transitions with increasing number of unobservable states calls into question the estimability of parameters of interest and potential bias of these quantities associated with the use of various constraints. This issue of parameter estimability and bias associated with the use of constraints in various partially or fully unobservable transitions is applicable to any study in which there are unobservable states. Kendall and Bjorkland (2001) and Kendall and Nichols (2002) evaluate some of the situations in which unbiased estimation can be achieved, but currently researchers should take a precautionary approach and simulate their study system with known parameter values in order to evaluate parameter bias and estimability.

Contributions to Population Growth

With respect to contributions to population growth of the symptomatic component of the population, several distinct patterns emerged, which corroborate previous findings as well as provide new insights into the dynamics of this disease system. As suggested by studies conducted in the northeastern (Jennelle et al. submitted) and

southeastern US (Altizer et al. 2004b), asymptomatic juvenile finches contribute more than adults to the growth of the symptomatic component of the Ithaca, NY population during FW seasons (Fig. 4.2). As juvenile birds are largely immunologically naïve to MG, this demographic component serves as a deterministic (annual) pool of susceptible individuals with which MG can be maintained and spread between finch populations. Presumably, once the surge of infection has spread throughout a large proportion of juvenile finches in FW seasons, adult birds contribute at least as much to population growth of symptomatic individuals during WS seasons (WS2003 and WS2004; Fig. 4.2). While there is experimental evidence to suggest temporary immunity following recovery from MG infection in house finches, which in part may explain the greater importance of juvenile birds to population growth of the symptomatic class, reinfection or recrudescence of symptoms is possible (Sydenstricker et al. 2005).

Comparing symptomatic to asymptomatic finches, Jennelle et al. (submitted) showed that the former group were less likely to undergo transient movements (a form of permanent emigration) and exhibited lower rates of local site-to-site movement, complementing my finding that symptomatic recruits contribute proportionally very little (< 11% at most) to population growth of the symptomatic component of the population (Fig. 4.2). While recruitment of symptomatic finches (presumably infectious) or asymptomatic carriers may serve to “seed” MG-naïve populations of house finches in the spread of this pathogen across the range of the host, the greatest contribution to maintenance and spread of MG within host populations are from resident symptomatic individuals (Fig. 4.2).

House finches infected with MG have been found to recover both in the field (Faustino et al. 2004) and in laboratory experiments (Kollias et al. 2004, Sydenstricker et al. 2006). Despite a greater probability of recovery than infection (Faustino et al

2004), finches can remain symptomatic (i.e., with conjunctivitis) for a long period of time (median of 42 days and range of 1-172 days; Sydenstricker et al. 2006). My results suggest that survival of infected finches coupled with associated prolonged expression of clinical signs is the primary contributor to maintenance of apparent endemic levels of MG in wild house finch populations (at least in the northeastern US).

With respect to contributions to the population growth of asymptomatic finches, there were surges in the contribution from asymptomatic recruits during the FW seasons (Fig. 4.3). There was almost negligible contribution to the asymptomatic class from symptomatic juveniles and adults (i.e., individuals that recovered). At first this result seems surprising, but after careful consideration, it conforms clearly to what would be expected in the system. While recovery probability of house finches infected with MG has been shown to be on average greater than infection probability (Faustino et al. 2004), recovered individuals by definition are ‘produced’ from a small pool of infected individuals (relative to uninfected birds) (see prevalence; Figs 4.2 & 4.3). Thus, one can conceptualize that relative to both existing asymptomatic finches and new recruits, recovered birds contribute proportionally very little to the asymptomatic pool of birds. Of primary importance to the population growth and maintenance of the asymptomatic class are asymptomatic residents. By extension, I can infer that resident birds (in large part) serve as the source pool of susceptible individuals with which the finch-MG dynamics plays out. Despite modest contributions from outside the local area, my findings corroborate those of Jennelle et al. (submitted), which suggest that there is a core structure to house finch assemblages (as represented by the Ithaca, NY study area). If this is a ubiquitous trend across the range of introduced house finches, such structure suggests an organizational complexity on a metapopulation scale that has not been previously appreciated.

Concluding Remarks

This study suggests that while significant mixing of finch aggregations occurs particularly in the autumn, which coincides with post-breeding season movements (of juveniles and adults; Hill 1993, Jennelle et al. submitted) and migration (Able and Belthoff 1998), maintenance of the symptomatic component of the Ithaca, NY population is sustained predominantly by resident prevalent cases and new infections from within the local study area. Adding to previous work in this system (Fausinto et al. 2004, Jennelle et al. submitted), this work highlights the notion that there is relatively greater closure of local scale finch aggregations during the winter-spring seasons. The potential for reinfection following recovery (Sydenstricker et al. 2005), coupled with evidence of the relative closure of finch aggregations suggests the possibility that the oft cited second seasonal peak in disease prevalence occurring during Spring seasons (Altizer et al. 2004a, Hosseini et al. 2004, Jennelle et al. submitted) may be in part driven by aggregate reinfection/recrudescence of MG in finches.

In many investigations of wildlife populations, study areas are often arbitrarily defined and lack delineation by physical boundaries. Such lack of geographic closure and by extension demographic closure, complicate the study of population dynamics as temporary emigration and immigration (either biologically or sampling driven) movements can occur (Kendall et al. 1997). In wildlife population studies, these types of movements (influencing detectability) along with sampling and biologically driven variation in detectability require the use of robust capture-recapture models (see Williams et al 2002 for review) to make strong inferences about the biological patterns and processes of interest. Capture-recapture designs formulated for addressing questions using MSORD and reverse-time multistate models are especially powerful tools for simultaneously accounting for various sources of heterogeneity in

detectability, temporary movements of organisms, and exploring hypotheses regarding state-specific (in this case disease state) variation in contributions to population growth.

Acknowledgements

This research was funded by the US National Science Foundation under DEB no. 0094456, and support was given from the Department of Natural Resources, Cornell University. I thank Veronique Connolly, Melanie Driscoll, Sarah Goodwin, Dana Hawley, Mari Kimura, Tom Muscato, and Elliott Swarthout for field assistance. I also thank Evan Cooch, Andre Dhondt, William Kendall, Cliff Kraft, and Patrick Sullivan for comments and discussion on earlier drafts of this manuscript.

REFERENCES

- Able, K.P., and J.R. Belthoff. 1998. Rapid 'evolution' of migratory behaviour in the introduced house finch of eastern North America. *Proceedings of the Royal Society of London B* 265:2063-2071.
- Altizer, S.M., W.M. Hochachka, and A.A. Dhondt. 2004a. Seasonal dynamics of mycoplasmal conjunctivitis in eastern North American House Finches. *Journal of Animal Ecology* 73:309-322.
- Altizer, S., A. K. Davis, K.C. Cook, and J.J. Cherry. 2004b. Age, sex, and season affect the risk of mycoplasmal conjunctivitis in a southeastern house finch population. *Canadian Journal of Zoology* 82:1-9.
- Anderson, R.M., and R.M. May. 1992. *Infectious Diseases of Humans: dynamics and Control*. Oxford Science Publications.
- Arnason, A.N. 1973. The estimation of population size, migration rates, and survival in a stratified population. *Researches in Population Ecology* 15:1-8.
- Brownie, C., J.E. Hines, J.D. Nichols, K.H. Pollock, and J.B. Hestbeck. 1993. Capture-recapture studies for multiple strata including non-Markovian transitions. *Biometrics* 49:1173-1187.
- Burnham, K.P., and D.R. Anderson. 2002. *Model selection and multimodel inference: A practical information-theoretic approach*. Springer-Verlag, New York.
- Cooch, E.G., and G.C. White. 2006. *A Gentle Introduction to Program MARK (5th Edition)*. [online] <http://www.phidot.org/software/mark/docs/book>.
- Dhondt, A.A., D.L. Tessaglia, and R.L. Slothower. 1998. Epidemic mycoplasmal conjunctivitis in House Finches from eastern North America. *Journal of Wildlife Diseases* 34:265-280.

- Dhondt, A.A., M.J.L. Driscoll, and E.C.H. Swarthout. 2007. House finch *Carpodacus mexicanus* roosting behaviour during the non-breeding season and possible effects of mycoplasmal conjunctivitis. *Ibis* 149:1-9.
- Dhondt, A.A., K.V. Dhondt, D.M. Hawley, and C.S. Jennelle. In press. Experimental evidence for transmission of *Mycoplasma gallisepticum* in house finches by fomites. *Avian Pathology*.
- Faustino, C.R., C.S. Jennelle, V. Connolly, A.K. Davis, E.C. Swarthout, A.A. Dhondt, and E.G. Cooch. 2004. *Mycoplasma gallisepticum* infection dynamics in a house finch population: seasonal variation in survival, encounter and transmission rate. *Journal of Animal Ecology* 73:651-669.
- Fischer, J.R., D.E. Stallknecht, M.P. Luttrell, A.A. Dhondt, and K.A. Converse. 1997. Mycoplasmal conjunctivitis in wild songbirds: The spread of a new contagious disease in a mobile host population. *Emerging Infectious Diseases* 3:69-72.
- Hartup, B.K., H.O. Mohammed, G.V. Kollias, and A.A. Dhondt. 1998. Risk factors associated with mycoplasmal conjunctivitis in House Finches. *Journal of Wildlife Diseases* 34:281-288.
- Hartup, B.K., G.V. Kollias, and D.H. Ley. 2000. Mycoplasmal conjunctivitis in songbirds from New York. *Journal of Wildlife Diseases* 36:257-264.
- Hartup, B.K., J.M. Bickal, A.A. Dhondt, D.H. Ley, and G.V. Kollias. 2001. Dynamics of conjunctivitis and *Mycoplasma gallisepticum* infections in house finches. *Auk* 118:327-333.
- Hawley, D.M., K. Lindstrom, and M. Wikelski. 2006. Experimentally increased social competition compromises humoral immune responses in house finches. *Hormones and Behavior* 49:417-424.

- Hestbeck, J.B., J.D. Nichols, and R.A. Malecki. 1991. Estimates of movement and site fidelity using mark-resight data of wintering Canada geese. *Ecology* 72:523-533.
- Hill, G.E., 1993. House finch (*Carpodacus mexicanus*). In *The Birds of North America*, No.46, A. Poole and F. Gill (eds.). The Academy of Natural Sciences, Philadelphia, Pennsylvania; The American Ornithologists' Union, Washington, D.C. pp.1-24.
- Hines, J.E. 1994. MSSURVIV Software to compute estimates of survival-movement and capture probability for open capture-recapture models. USGS-PWRS. <http://www.mbr-pwrc.usgs.gov/software/mssurviv.html>
- Hochachka, W.M., and A.A. Dhondt. 2000. Density-dependent decline of host abundance resulting from a new infectious disease. *Proceedings of the National Academy of Sciences* 97:5303-5306.
- Hochachka, W.M., and A.A. Dhondt. 2006. House finch (*Carpodacus mexicanus*) population- and group-level responses to a bacterial disease. *Ornithological Monographs* 60:30-43.
- Hosseini, P.R., A.A. Dhondt, and A. Dobson. 2004. Seasonality and wildlife disease: how seasonal birth, aggregation and variation in immunity affect the dynamics of *Mycoplasma gallisepticum* in house finches. *Proceedings of the Royal Society B* 271:2569-2577.
- Hosseini, P.R., A.A. Dhondt, and A.P. Dobson. 2006. Spatial spread of an emerging infectious disease: Conjunctivitis in house finches. *Ecology* 87:3037-3046.
- Jennelle, C.S., C.R. Faustino, E.G. Cooch, and A.A. Dhondt. submitted. Movement dynamics, demographic risk factors, and habitat partitioning in an avian disease system.

- Jolly, G. M. 1965. Explicit estimates from capture-recapture data with both death and immigration - stochastic model. *Biometrika* 52: 225-247.
- Kendall, W.L., and J.D. Nichols. 1995. On the use of secondary capture-recapture samples to estimate temporary emigration and breeding proportions. *Journal of Applied Statistics* 22:751-762.
- Kendall, W.L., J.D. Nichols, and J.E. Hines. 1997. Estimating temporary emigration and breeding proportions using capture-recapture data with Pollock's robust design. *Ecology* 78:563-578.
- Kendall, W.L. 1999. Robustness of closed capture-recapture methods to violations of the closure assumption. *Ecology* 80:2517-2525.
- Kendall, W.L., and R. Bjorkland. 2001. Using open robust design models to estimate temporary emigration from capture-recapture data. *Biometrics* 57:1113-1122.
- Kendall, W.L., and J.D. Nichols. 2002. Estimating state transition probabilities for unobservable states using capture-recapture/resighting data. *Ecology* 83:3276-3284.
- Kollias, G.V., K.V. Sydenstricker, H.W. Kollias, D.H. Ley, P.R. Hosseini, V. Connolly, and A.A. Dhondt. 2004. Experimental infection of House Finches with *Mycoplasma gallisepticum*. *Journal of Wildlife Diseases* 40:79-86.
- Ley, D.H., J.E. Berkhoff, and J.M. McLaren. 1996. *Mycoplasma gallisepticum* isolated from house finches (*Carpodacus mexicanus*) with conjunctivitis. *Avian Diseases* 40:480-483.
- Luttrell, M.P., J.R. Fischer, D.E. Stallknecht, and S.H. Kleven. 1996. Field investigation of *Mycoplasma gallisepticum* infections in house finches (*Carpodacus mexicanus*) from Maryland and Georgia. *Avian Diseases* 40:335-341.

- Luttrell, M.P., D.E. Stallknecht, J.R. Fischer, C.T. Sewell, and S.H. Kleven. 1998. Natural *Mycoplasma* infection in a captive flock of House Finches. *Journal of Wildlife Diseases* 34:289-296.
- Mikaelian, I., D.H. Ley, R. Claeau, M. Lemieux, and J.P. Berube. 2001. Mycoplasmosis in evening and pine grosbeaks with conjunctivitis in Quebec. *Journal of Wildlife Diseases* 37:826-830.
- Nichols, J.D., J.E. Hines, J.-D. Lebreton, and R. Pradel. 2000. Estimation of contributions to population growth: A reverse time capture-recapture approach. *Ecology* 81:3362-3376.
- Pollock K.H., D.L. Solomon, and D.S. Robson. 1974. Tests for mortality and recruitment in a K-sample tag-recapture experiment. *Biometrics* 30:77-87.
- Pollock K.H. 1982. A capture-recapture design robust to unequal probability of capture. *Journal of Wildlife Management* 46:752-757.
- Pradel, R. 1993. Flexibility in survival analysis from recapture data: handling trap-dependence. Pages 29–37 in J.-D. Lebreton and P. M. North, editors. *Marked individuals in the study of bird populations*. Birkhäuser Verlag, Basel, Switzerland.
- Pradel, R. 1996. Utilization of capture-mark-recapture for the study of recruitment and population growth rate. *Biometrics* 52:703-709.
- Pradel, R., J.E. Hines, J.-D. Lebreton, and J.D. Nichols. 1997. Capture-recapture survival models taking account of transients. *Biometrics* 53:60-72.
- Schwarz C.J., J.F. Schweigert, and A.N. Arnason 1993. Estimating migration rates using tag-recovery data. *Biometrics* 49:177-193.

- Schwarz, C.J., and W.T. Stobo. 1997. Estimating temporary emigration using the robust design. *Biometrics* 53:178-194.
- Seber, G. A. F. 1965. A note on the multiple-recapture census. *Biometrika* 52: 249-259.
- Sydenstricker, K.V., A.A. Dhondt, D.H. Ley, and G.V. Kollias. 2005. Re-exposure of captive house finches that recovered from *Mycoplasma gallisepticum* infection. *Journal of Wildlife Diseases* 41:326-333.
- Sydenstricker, K.V., A.A. Dhondt, D.M. Hawley, C.S. Jennelle, H.W. Kollias, and G.V. Kollias. 2006. Characterization of experimental *Mycoplasma gallisepticum* infection in captive House Finch flocks. *Avian Diseases* 50:39-44.
- White, G.C., and K.P. Burnham. 1999. Program MARK: Survival estimation from populations of marked animals. *Bird Study* 46:120-139.
- Williams, B.K., J.D. Nichols, and M.J. Conroy. 2002. *Analysis and Management of Animal Populations*. Academic Press, San Diego.