Ecological Applications, 17(1), 2007, pp. 154–167

STATE-SPECIFIC DETECTION PROBABILITIES AND DISEASE PREVALENCE

Christopher S. Jennelle,[1,4] Evan G. Cooch,[1] Michael J. Conroy,[2] and Juan Carlos Senar[3]

[1]*Department of Natural Resources, Fernow Hall, Cornell University, Ithaca, New York 14853 USA*
[2]*Georgia Cooperative Fish and Wildlife Research Unit, University of Georgia, Athens, Georgia 30602 USA*
[3]*Evolutionary and Behavioural Ecology Associate Unit, CSIC, Natural History Museum, P. Picasso s/n, 08003 Barcelona, Spain*

Abstract. Investigations of disease dynamics in wild animal populations often use estimated prevalence or incidence as a measure of true disease frequency. Such indices, almost always based solely on raw counts of infected and uninfected individuals, are often used as the basis for analysis of temporal and spatial dynamics of diseases. Generally, such studies do not account for potential differences in observer detection probabilities of host individuals stratified by biotic and/or abiotic factors. We 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*) and *Mycoplasma gallisepticum* system. In this system, detection probabilities of uninfected finches were generally higher than infected individuals. We 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 our study), apparent prevalence is negatively biased, and vice versa. In situations where state-specific detection probabilities strongly interact over time, we 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 drivers of disease dynamics. Given that detection probabilities of different groups of individuals are likely to change temporally and spatially in most field studies, our 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. We discuss several alternative approaches and categorize the assumptions under which analysis of uncorrected prevalence may be acceptable.

Key words: *capture–recapture;* Carpodacus mexicanus; *conjunctivitis; detection probability; disease; ecological index; House Finch; mark–recapture;* Mycoplasma gallisepticum; *prevalence.*

Introduction

Of significant importance to disease ecologists and epidemiologists studying wildlife diseases 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 must often rely on incomplete counts of individuals or indices for estimating 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 animal populations (Altizer et al. 2004*a*, 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 are, at the least, a useful starting point for studying disease dynamics.

Manuscript received 26 September 2005; revised 10 April 2006; accepted 12 April 2006; final version received 9 May 2006. Corresponding Editor: R. S. Ostfeld.
[4] E-mail: csj9@cornell.edu

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 nonlinearly, 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 we 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 we 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 the 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 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., effects of lead poisoning in waterfowl; Bellrose 1959) or body condition (Hepp et al. 1986). Evidence suggests that there can be a condition bias in samples of trapped 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, 1998, Senar et al. 1999, Tuyttens et al. 1999, Nichols et al. 2000, Tracey et al. 2005). Despite population ecologists' recognition of potential sources of heterogeneity in animal detection probabilities and the subsequent efforts to correct population estimates for various sources of bias in studies of wildlife disease dynamics, to date we are only aware of two efforts that 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 the Akaike information criterion (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 temporal or spatial) is consistent with an underlying

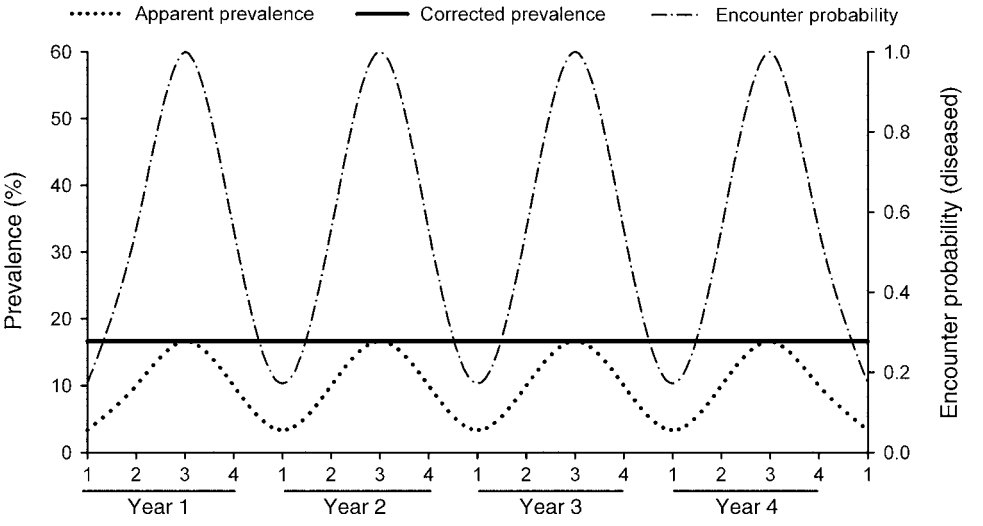

FIG. 1. Illustration showing how cyclic patterns of apparent (observed) prevalence may be an artifact of cyclic patterns in detection probabilities of one or more groups of animals, which can result in misleading inferences about the pattern of corrected (true) prevalence. In this case, only the detection probability of diseased animals varies temporally, while the detection probability of healthy animals (with respect to the condition under study) is time invariant (=1.0).

dynamical driver (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 may have little to do with underlying disease dynamics. Consider a situation where the true prevalence of a disease is constant over time but where the probability of detecting, for example, 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; this would suggest a seasonal, cyclic pattern in prevalence, when in fact, the true prevalence was constant over time (Fig. 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 an expedient assumption, but it 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 we assess the importance of incorporating heterogeneous detection probabilities in estimates of disease prevalence (and easily extended to incidence); we also provide a simple calculation to facilitate estimation. We 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*. We 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 our 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. We 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 we consider concern avian diseases, the underlying ideas are relevant to any taxa under study. We conclude by presenting recommendations for estimation of detection probabilities for small- and large-scale studies when standard CMR methods are not used.

METHODS

Subset of data used

Data were collected as part of a larger effort to study the disease dynamics of *Mycoplasma gallisepticum* (MG) infection in eastern House Finches (hereafter finches), which were introduced to the eastern United States

around 1940 (Hill 1993). General field methods used are described in Faustino et al. (2004). Trapping and resighting data were collected from encounters with individual finches from August to April of 2001–2005 in Ithaca, New York, USA (located at approximately 42.5° N 76.5° E). Each newly captured bird was fitted, under permit, with a nine-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. We stress that for the purposes of this paper, we explicitly define an MG-infected individual as expressing observable symptoms of infection, namely conjunctivitis. We only used data from 2002 to 2003 to demonstrate the methodology.

Estimation of detection probabilities and corrected prevalence

We 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; next, we calculate the associated percentage of relative bias (%RB) of apparent prevalence as

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

where $\hat{\delta}_i^{\mathrm{A}}$ = estimated *apparent* prevalence at time i and $\hat{\delta}_i^{\mathrm{C}}$ = 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 we obtained capture data from both live capture (via mist nets and cage traps) and resighting events, we 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, we used estimates of weekly detection probabilities of infected and uninfected finches generated from an intensive mark–recapture study in Ithaca, New York (Faustino et al. 2004). Detection probabilities were estimated using multistate mark–recapture models (Williams et al. 2002, and references therein) in program MARK, version 4.1 (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. If we assume that survival from time i to time $i+1$ depends only on the state (stratum) at time i, then separate estimation of survival from transition probabilities is possible where S_i^r is 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} is 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} is 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 (in our case I stands for infected and U stand for 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; $\psi_{\mathrm{i}}^{\mathrm{IU}}$).

There are several important assumptions to consider when conducting multistate CMR analysis of wildlife disease data, 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 $i+1$. Care must be taken to ensure that the time span between sampling periods is, at most, the average time expected for an animal to be infected. 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 not only at time i, but also at times $i-1$, $i-2$, and so on. In most cases, however, we 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; to accommodate this, Kendall et al. (1997) have developed temporary emigration models that make use of Pollock's robust design (Pollock 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, as violation of these assumptions may induce bias in resulting parameter estimates and influence the inferences that can be made.

For our analyses, we sought relative estimates of N for infected and uninfected finches in relation to the expression $E(C) = pN$, 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 our study area, the specification of the population we sampled is not important as long as the sampled finches adequately represent the number 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 our corrected prevalence estimates. Given that observations of finches at our study sites represent an incomplete count of finches, if we 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 is the observed count of finches in health state s (infected or uninfected) at time i, $\hat{p}_i^s$ is the estimated detection probability of a finch in health state s at time i, and $\hat{N}_i^s$ is 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

$$\hat{N}_i^s = \frac{C_i^s}{\hat{p}_i^s}. \tag{2}$$

Since prevalence is defined as the proportion of infected individuals in a population, we can derive an expression that corrects our estimate 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

$$\hat{\delta}_i^{\mathrm{R}} = \frac{\hat{N}_i^{\mathrm{I}}}{\hat{N}_i^{\mathrm{I}} + \hat{N}_i^{\mathrm{U}}} = \frac{\dfrac{C_i^{\mathrm{I}}}{\hat{p}_i^{\mathrm{I}}}}{\dfrac{C_i^{\mathrm{I}}}{\hat{p}_i^{\mathrm{I}}} + \dfrac{C_i^{\mathrm{U}}}{\hat{p}_i^{\mathrm{U}}}} = \frac{C_i^{\mathrm{I}} \hat{p}_i^{\mathrm{U}}}{C_i^{\mathrm{I}} \hat{p}_i^{\mathrm{U}} + C_i^{\mathrm{U}} \hat{p}_i^{\mathrm{I}}} \tag{3}$$

where $\hat{\delta}_i^{\mathrm{R}}$ is the corrected disease prevalence at time i for reduced state space (disease state only), C_i^s is the observed count of focal species in health state s at time i, and $\hat{p}_i^s$ is the estimated detection probability of the focal species in health state s at time i.

The development of our expression for corrected prevalence is analogous to that presented for estimation of breeding proportions 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, we use Eq. 3 and assume that the primary source of heterogeneity in detection probability is disease state.

An approximation for the conditional variance of corrected prevalence $\hat{\delta}_{ijk}^{\mathrm{R}}$ is given by Eq. 4 (at the bottom of the page; see Appendix B for equation derivation) where C_{ijk}^s is the observed count of finches in health state s in year i, month j, and week k, and $\hat{p}_{ijk}^s$ is the estimated detection probability for a finch in health state s 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 the 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 (2002–2003 field season), we

$$\operatorname{var}\left(\hat{\delta}_{ijk}^{\mathrm{R}} \mid C_{ijk}^{\mathrm{U}}, C_{ijk}^{\mathrm{I}}\right) = \frac{(C_{ijk}^{\mathrm{I}})^2 (C_{ijk}^{\mathrm{U}})^2 [(\hat{p}_{ijk}^{\mathrm{U}})^2 \operatorname{var}(\hat{p}_{ijk}^{\mathrm{I}}) + (\hat{p}_{ijk}^{\mathrm{I}})^2 \operatorname{var}(\hat{p}_{ijk}^{\mathrm{U}}) - 2\hat{p}_{ijk}^{\mathrm{U}} \hat{p}_{ijk}^{\mathrm{I}} \operatorname{cov}(\hat{p}_{ijk}^{\mathrm{U}}, \hat{p}_{ijk}^{\mathrm{I}})]}{(C_{ijk}^{\mathrm{U}} \hat{p}_{ijk}^{\mathrm{I}} + C_{ijk}^{\mathrm{I}} \hat{p}_{ijk}^{\mathrm{U}})^4} \tag{4}$$

present three hypothetical scenarios using the same finch count data. In place of estimated detection probabilities from Faustino et al. (2004), we assign hypothetical detection probabilities for infected and uninfected individuals that interact over time and are additive with time. In each scenario, we 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, we provide a gradient of corrected prevalence functions over the course of two partial seasons (autumn and winter) using the following pairs of detection probabilities ($p_i^U = 0.55$, $p_i^I = 0.45$; $p_i^U = 0.65$, $p_i^I = 0.35$; $p_i^U = 0.75$, $p_i^I = 0.25$). We 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 to where the detection probability of infected finches is higher in winter due to the increased energetic demands of this period causing 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, we 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. In this case, we 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 ($p_i^U = 0.45$, $p_i^I = 0.55$; $p_i^U = 0.35$, $p_i^I = 0.65$; $p_i^U = 0.25$, $p_i^I = 0.75$, respective to the two halves of the study period). We 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, we also provide a third scenario, the reverse of the previous scenario, with consistently higher detection probabilities for uninfected, compared to infected, individuals.

In addition to presenting a corrected estimator for disease prevalence, we 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 multistate CMR 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 of disease 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 compares the pattern of apparent prevalence observed from actual counts of infected and uninfected finches vs. 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). 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 December 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).

In Fig. 3, we considered a hypothetical example using the same finch count data that were used to generate Fig. 2. In Fig. 3A, we 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 increases. Under this scenario, both the pattern and magnitude of corrected prevalence changed directly with the difference in state-

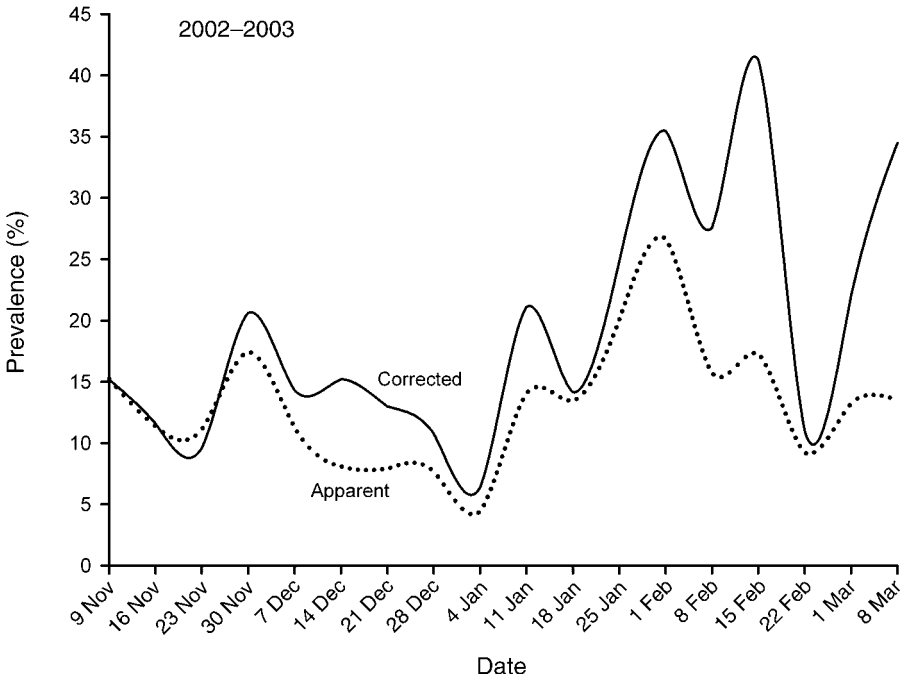

FIG. 2. Comparison of apparent prevalence (dotted line; based on observed counts of finches) to corrected prevalence (solid line) incorporating differential detection probabilities (from Faustino et al. 2004) between infected and uninfected House Finches for data collected between the weeks of 9 November 2002 and 8 March 2003; data were collected in Ithaca, New York, USA.

specific detection probabilities (Fig. 3A). Thus, increasing differences in state-specific detection probabilities led to increased bias in apparent-prevalence estimates.

In Fig. 3B and 3C, we considered alternative hypothetical scenarios, again using the same finch-count data that were used to generate Fig. 3A. In these scenarios, we 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. 3B and $p_i^I < p_i^U$ in Fig. 3C. When the detection probability of infected individuals was greater than uninfected individuals, we found that the observed pattern of prevalence was positively biased (Fig. 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. 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. Our results show that the magnitude and trend of disease prevalence estimates based on raw counts of individuals that are uncorrected for state-specific detection probabilities can be biased (Fig. 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 (1) exaggerated seasonal peaks in prevalence (Fig. 3A), (2) inflated levels of disease prevalence (Fig. 3B), or (3) gross underestimates of disease prevalence (Fig. 3C).

If an index (such as disease prevalence) is used for making ecological inferences, we 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 the calculation of an index.

We 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

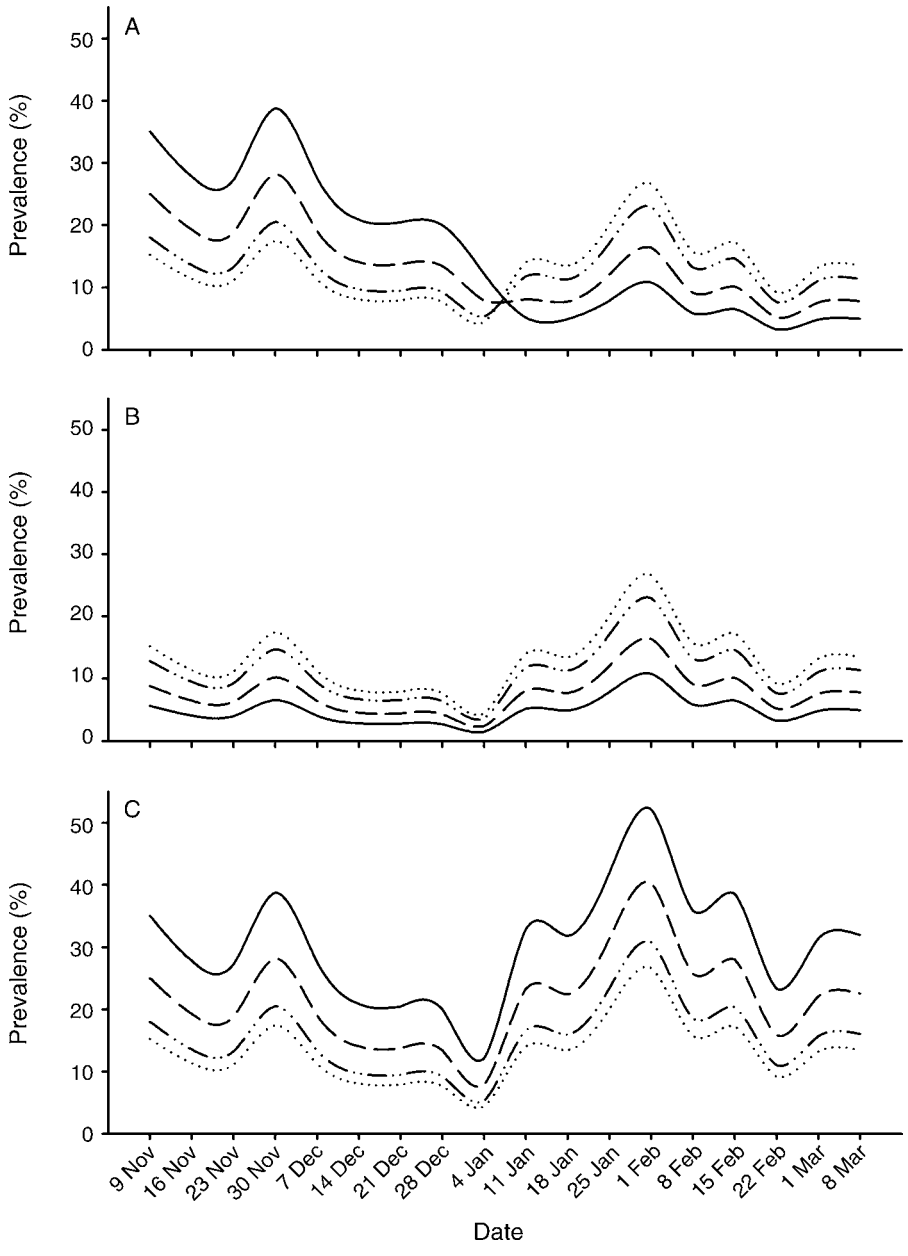

FIG. 3. Comparison of apparent prevalence (···; based on observed counts of finches) to corrected prevalence incorporating differential detection probabilities between infected and uninfected House Finches under hypothetical scenarios of differential detectability. Corrected prevalence is shown for (A) an interaction over time, where $p_i^{\mathrm{I}} = 0.45$ and $p_i^{\mathrm{U}} = 0.55$ (– ·· –), $p_i^{\mathrm{I}} = 0.35$ and $p_i^{\mathrm{U}} = 0.65$ (– –), and $p_i^{\mathrm{I}} = 0.25$ and $p_i^{\mathrm{U}} = 0.75$ (—) for the first half of the study period, with this relationship reversed for the second half; (B) an additive effect over time, where $p_i^{\mathrm{I}} = 0.55$ and $p_i^{\mathrm{U}} = 0.45$ (– ·· –), $p_i^{\mathrm{I}} = 0.65$ and $p_i^{\mathrm{U}} = 0.35$ (– –), and $p_i^{\mathrm{I}} = 0.75$ and $p_i^{\mathrm{U}} = 0.25$ (—); and (C) an additive effect over time, where $p_i^{\mathrm{I}} = 0.45$ and $p_i^{\mathrm{U}} = 0.55$ (– ·· –), $p_i^{\mathrm{I}} = 0.35$ and $p_i^{\mathrm{U}} = 0.65$ (– –), and $p_i^{\mathrm{I}} = 0.25$ and $p_i^{\mathrm{U}} = 0.75$ (—) (p_i^s is detection probability of animals at time i in state s, where I = infected, U = uninfected).

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 the resulting captured sample sizes are small despite a great amount of effort. Under these circumstances, we 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, we 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; M. 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 (W. 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 instead (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 (M. 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 corrected prevalence in a similar manner as we 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). We 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 *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 (*Serinus serinus*) were consistently higher than uninfected individuals. The magnitude of the difference between detection probabilities that we used for our 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 ($p^{\mathrm{I}} = 0.81$ compared with $p^{\mathrm{U}} = 0.21$). In both of these avian

disease systems, the authors suspected 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 we obtain the following sample from the population: infected ($J = 20$, $A = 5$) and uninfected ($J = 20$, $A = 10$). If we calculate disease prevalence unadjusted for age-specific-detection probabilities, then we obtain 0.45, but when we account for detection probabilities, corrected prevalence is 0.40, resulting in a positive bias of apparent prevalence. 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 hosts that are misdiagnosed as uninfected or 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, we can derive the following statistics:

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

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

where $C_i^{s'}$ is the 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 is the observed count of the focal species in health state s at time i, NPV is the negative predictive value or the probability of an uninfected individual given a negative test result, and PPV is the 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 Eq. 3. We 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 we assigned disease-intensity-dependent detection probabilities to individuals, we found that such detection heterogeneity (if sufficiently large) can result in additional bias of unadjusted estimates of prevalence. As such, we 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 (M. Samuel, *personal communication*). In our paper we 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, we 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 we use health-state differences). The expected bias of an estimated parameter is defined as

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

where $E(\hat{\theta})$ is the expected value of the estimated parameter of interest, in our case apparent prevalence. θ is the value of the parameter of interest, in our case the value of estimated prevalence corrected for differential detection probability (Eq. 3).

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

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

where C_i^s is the observed count of the focal species in health state s (I, infected or U, uninfected) at time i; α_i is the ratio of estimated detection probabilities of the focal species $(\hat{p}_i^{\text{I}}/\hat{p}_i^{\text{U}})$, where I represents infected and U represents uninfected at time i.

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

When CMR (capture–mark–release) 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 our primary focus is on estimating disease prevalence for some host species, then we can partition and treat 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 that 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. By using information theoretic methods, specifically the AIC (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. (2004*b*) 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, for example) 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.

Conclusion

At the very least, even a minimal effort to estimate health-state-specific detection probabilities (by way of a shortened capture–recapture study) would be useful in providing investigators with some idea of whether differential detection probabilities occur between infected and uninfected 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 also be used, 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–MG system that we consider in this paper and the Serin–avian pox system (Senar and Conroy 2004) that we reference are unique in that (1) health status can be inferred from visual observation of birds and (2) an intensive CMR effort has afforded the opportunity to directly estimate detection probabilities for animals in alternative health states. We 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 we are confident this knowledge will help investigators produce more reliable estimates of disease frequency.

Acknowledgments

We thank Cristina Faustino for providing us with estimates from her CMR analyses; Veronique Connolly, Melanie Driscoll, Cristina Faustino, Sarah Goodwin, Dana Hawley, Mari Kimura, and Elliott Swarthout for their efforts collecting field data. Special thanks to Mary Conner, Michael Samuel, and an anonymous reviewer for helpful reviews of this manuscript. Thanks to Sonia Altizer, André Dhondt, Andy Dobson, Wes Hochachka, and William Kendall for providing useful comments on earlier versions. We thank James Hines (USGS: Patuxent) for use of his program MARKWAIT in simulations. We gratefully acknowledge all of the "citizen scientists" who contributed survey data to the HFDS. Also, we thank the National Science Foundation (under grant number DEB-0094456). Any opinions, findings and conclusions, or recommendations expressed in this material are those of the authors and do not necessarily reflect the views of the National Science Foundation. We also thank the Georgia Cooperative Fish and Wildlife Research Unit and the Ministerio de Ciencia y Tecnología of the Spanish Research Council (BOS 2003-09589).

Literature Cited

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. Second edition. Akadémiai Kiadi, Budapest, Hungary.

Altizer, S., A. K. Davis, K. C. Cook, and J. J. Cherry. 2004*a*. 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. 2004*b*. 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*. Proceed-

ings of the Royal Society of London B: Biological Sciences 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 D. R. Anderson. 2002. Model selection and multimodel inference: a practical information-theoretic approach. Second edition. Springer, New York, New York, USA.

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.

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 Institute 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., 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.

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.

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. Number 46. Academy of Natural Sciences, Philadelphia, Pennsylvania, USA and American Ornithologists' Union, Washington, D.C., USA.

Hines, J. E. 1994. MSSURVIV Users Manual. National Biological Service, Patuxent Wildlife Research Center, Laurel, Maryland, USA.

International Working Group for Disease Monitoring and Forecasting. 1995*a*. 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., and F. Messier. 2004. Factors affecting apparent prevalence of tuberculosis and brucellosis in wood bison. Journal of Animal Ecology 73:623–631.

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.

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.

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.

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, UK.

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. Spendelow. 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., 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.

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.

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, UK.

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.

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 46(Supplement):248–252.

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.

Tuyttens, 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, C. I., III, 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 C. D. Ankney. 1985. Condition bias and band recovery data: a reply to Burnham and Nichols. Wildlife Society Bulletin 13:349–351.

Weatherhead, P. J., and H. Greenwood. 1981. A critical assumption of band-recovery models may often be violated. Wildlife Society Bulletin 12:198–199.

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, California, USA.

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

APPENDIX A

A general formula for corrected prevalence (*Ecological Archives* A017-008-A1).

APPENDIX B

The derivation of the approximate conditional variance for the reduced form prevalence estimator presented in the text (*Ecological Archives* A017-008-A2).

APPENDIX C

An evaluation of bias of an approach to prevalence estimation in a special case (*Ecological Archives* A017-008-A3).

APPENDIX D

A table of the weekly counts of House Finches, estimated encounter probabilities, estimated prevalence, and relative bias in apparent prevalence (*Ecological Archives* A017-008-A4).

APPENDIX E

Figures showing the bias in apparent prevalence as a function of a range of differences in health-state-specific detection probabilities (*Ecological Archives* A017-008-A5).

***Ecological Archives* A017-008-A4**

Christopher S. Jennelle, Evan G. Cooch, Michael J. Conroy, and Juan Carlos Senar. 2007. State-specific detection probabilities and disease prevalence. *Ecological Applications* 17:154–167.

Appendix D. Weekly counts (C) of house finches, estimated encounter probabilities ($\hat{p}$: U = uninfected; I =infected) taken from Faustino et al. (2004), estimated prevalence (δ: A = apparent; C = corrected), and % relative bias (%RB) from November through March 2002–2003 in Ithaca, New York, USA.

Week	C^U	C^I	$\hat{p}^U$	$\hat{p}^I$	δ^A	δ^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

LITERATURE CITED

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.

[Back to A017-008]

Ecological Archives **A017-008-A3**

Christopher S. Jennelle, Evan G. Cooch, Michael J. Conroy, and Juan Carlos Senar. 2007. State-specific detection probabilities and disease prevalence. *Ecological Applications* 17:154–167.

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), we were 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, we 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 (A) 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. C1A). 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 (B) 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. C1B). 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 (A) 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 (B) 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 we 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), we 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. We were not interested in determining a lower bound for the number of marked individuals necessary for adequate support of models with time-dependent variation. Rather, we used 1000 newly released 'simulated' individuals in each cohort in our bootstrap samples only to facilitate the convergence of time-invariant parameter estimates. We 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 { ϕ (disease state) *p*(disease state) ψ^{UI}(.) ψ^{IU}(.)} (note the period surrounded by parenthesis indicates time-invariance). We used program MARKWAIT designed by James E. Hines of Patuxent Wildlife Research Center in Laurel, Maryland, to automate the creation of sample encounter histories in SAS, and subsequently estimate parameters under the aforementioned model in MARK. We 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 we 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, we 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 we plotted and calculated the two maximal values of percent relative bias in estimated prevalence.

Projecting disease prevalence over 15 time steps under scenario (A) produced a declining trend in 'true' prevalence (Fig. C1A), while projection under scenario (B) produced an increasing trend in 'true' prevalence (Fig. C1B). The expected value of infection probability under scenario (A) was 0.1740, and that under scenario (B) 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 we 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. We 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. C1). Our 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, we urge researchers to be very cautious when making inferences about patterns in estimated disease prevalence when data is sparse.

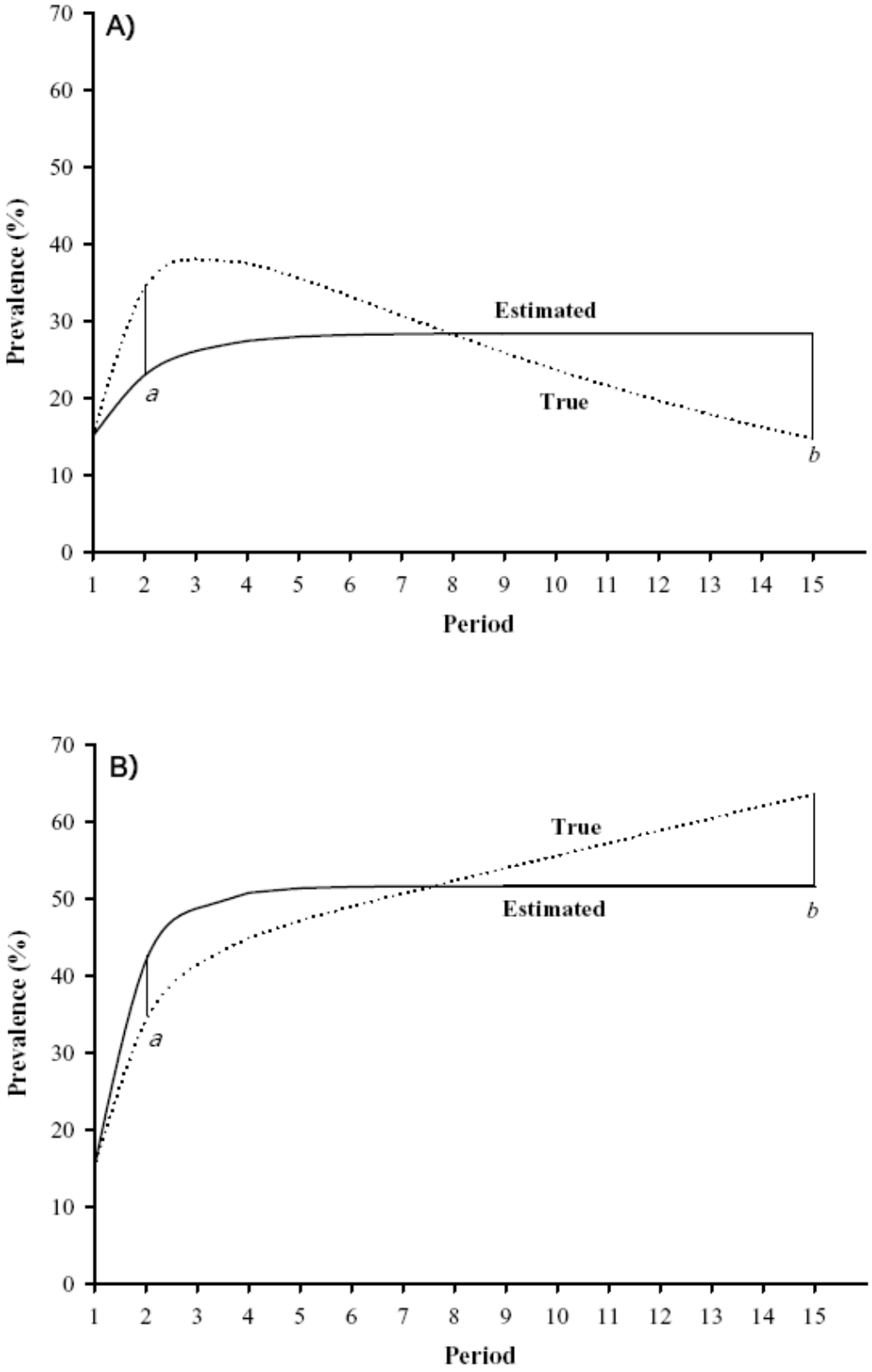

FIG. C1. Evaluation of a special case of the Senar and Conroy (2004) multistate model approach to prevalence estimation reveals that under certain conditions researchers may falsely interpret patterns of estimated prevalence to be time invariant, when in fact a temporal trend exists. The figures show the potential bias in estimated prevalence when a temporal trend in 'true' prevalence is mediated by a trend in decreasing (C1A) or increasing infection probability (C1B). Points *a* and *b* in each panel indicate where maximal levels of percent relative bias (%RB) in estimated prevalence occur; in panel (C1A) %RB at points *a* and *b* are -33% and 92%, respectively, while in panel (C1B) %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.

LITERATURE CITED

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.

White, G. C., and K. P. Burnham. 1999. Program MARK: Survival estimation from populations of marked animals. Bird Study **46** (Supplement):120–138.

[Back to A017-008]

Ecological Archives **A017-008-A2**

Christopher S. Jennelle, Evan G. Cooch, Michael J. Conroy, and Juan Carlos Senar. 2007. State-specific detection probabilities and disease prevalence. *Ecological Applications* 17:154–167.

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. We used the Delta Method (Seber 1982) to derive the approximate conditional sampling variance for our 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 (Eq. 3), is

$$\mathrm{var}(\hat{\delta}^{R}_{ijk} \left| C^{U}_{ijk}, C^{I}_{ijk} \right.) = \left(\frac{\partial \hat{\delta}^{R}_{ijk}}{\partial \hat{p}^{s}_{ijk}} \right) \times \hat{\Sigma} \times \left(\frac{\partial \hat{\delta}^{R}_{ijk}}{\partial \hat{p}^{s}_{ijk}} \right)^{T},$$

where,

$\hat{\delta}^{R}_{ijk}$ = 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^{s}_{ijk} = observed count of animals in health state *s* (*I*: infected or *U*: uninfected) in year *i*,

month *j* , and week *k*,

$\hat{p}^{s}_{ijk}$ = 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, $\hat{p}^{s}_{ijk}$.

Using matrix algebra, the Delta Method yields

$$\mathrm{var}(\hat{\delta}^{R}_{ijk} \left| C^{U}_{ijk}, C^{I}_{ijk} \right.) = \left[-\frac{C^{I}_{ijk} C^{U}_{ijk} \hat{p}^{U}_{ijk}}{\left(\left(C^{U}_{ijk} \hat{p}^{I}_{ijk} \right) + \left(C^{I}_{ijk} \hat{p}^{U}_{ijk} \right) \right)^{2}} \quad \frac{C^{I}_{ijk} C^{U}_{ijk} \hat{p}^{I}_{ijk}}{\left(\left(C^{U}_{ijk} \hat{p}^{I}_{ijk} \right) + \left(C^{I}_{ijk} \hat{p}^{U}_{ijk} \right) \right)^{2}} \right] \times \hat{\Sigma} \times$$
$$\left[-\frac{C^{I}_{ijk} C^{U}_{ijk} \hat{p}^{U}_{ijk}}{\left(\left(C^{U}_{ijk} \hat{p}^{I}_{ijk} \right) + \left(C^{I}_{ijk} \hat{p}^{U}_{ijk} \right) \right)^{2}} \quad \frac{C^{I}_{ijk} C^{U}_{ijk} \hat{p}^{I}_{ijk}}{\left(\left(C^{U}_{ijk} \hat{p}^{I}_{ijk} \right) + \left(C^{I}_{ijk} \hat{p}^{U}_{ijk} \right) \right)^{2}} \right]^{T},$$

where,

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

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

$\hat{p}^{s}_{ijk}$ = 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, $\hat{p}^{s}_{ijk}$.

The approximate conditional sampling variance of the prevalence estimator is thus

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

LITERATURE CITED

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.

Seber , G. 1982. The estimation of animal abundance and related parameters. Charles Griffin and Company, London, UK.

[Back to A017-008]

Ecological Archives **A017-008-A1**

Christopher S. Jennelle, Evan G. Cooch, Michael J. Conroy, and Juan Carlos Senar. 2007. State-specific detection probabilities and disease prevalence. *Ecological Applications* 17:154–167.

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, we can calculate a corrected estimate of prevalence as

$$\hat{\delta}_i = \frac{\sum_{k=1}^{2}\sum_{l=1}^{a}\hat{N}_i^{I,kl}}{\sum_{k=1}^{2}\sum_{l=1}^{a}\hat{N}_i^{I,kl}+\sum_{k=1}^{2}\sum_{l=1}^{a}\hat{N}_i^{U,kl}} = \frac{\sum_{k=1}^{2}\sum_{l=1}^{a}\frac{C_i^{I,kl}}{\hat{p}_i^{I,kl}}}{\sum_{k=1}^{2}\sum_{l=1}^{a}\frac{C_i^{I,kl}}{\hat{p}_i^{I,kl}}+\sum_{k=1}^{2}\sum_{l=1}^{a}\frac{C_i^{U,kl}}{\hat{p}_i^{U,kl}}}$$

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

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.

[Back to A017-008]

Ecological Archives **A017-008-A5**

Christopher S. Jennelle, Evan G. Cooch, Michael J. Conroy, and Juan Carlos Senar. 2007. State-specific detection probabilities and disease prevalence. *Ecological Applications* 17:154–167.

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, we calculated both expected bias (Fig. E1) and percent relative bias (%RB) (Fig. E2) 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 we consider 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. $p^U = 0.75$, $p^I = 0.85$). We highlight these results to show researchers that the relationship between detection probabilities and bias in apparent prevalence may not be straightforward.

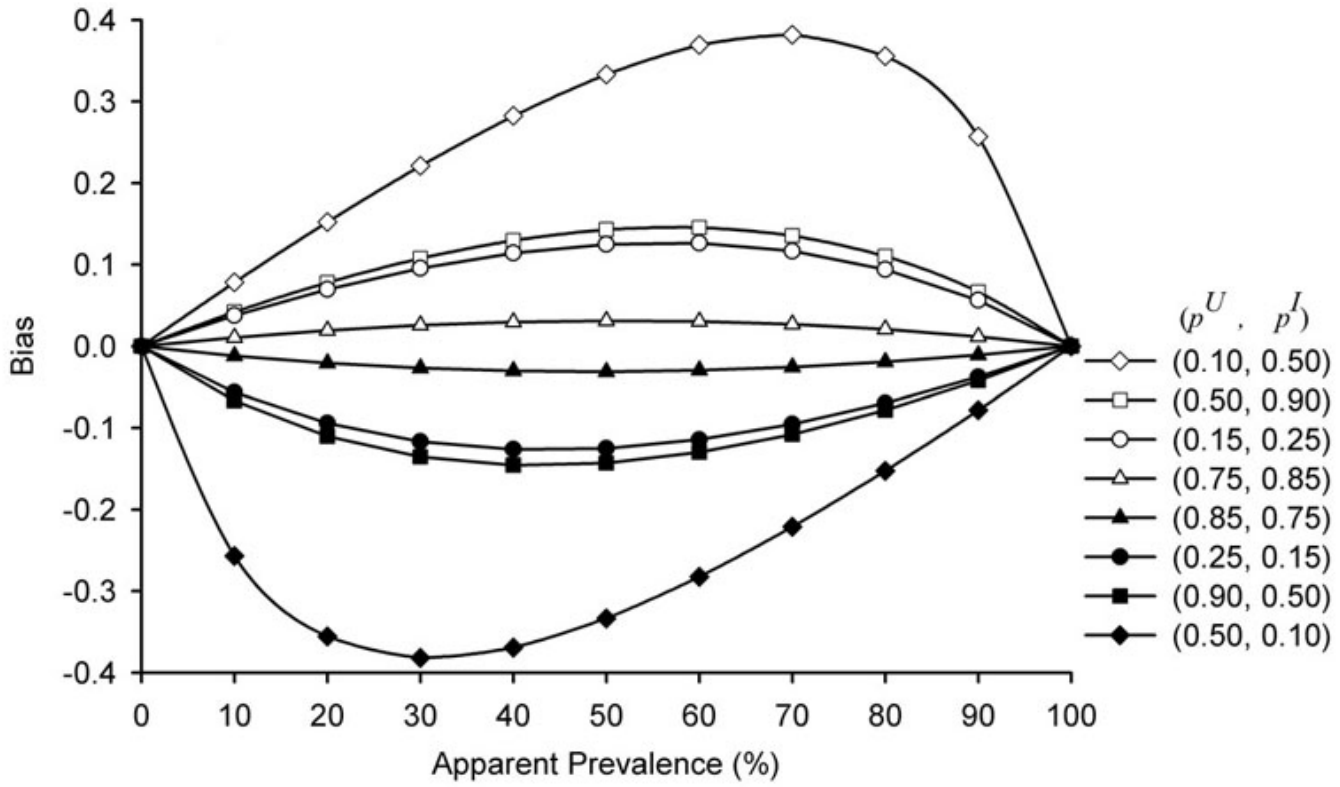

FIG. E1. Bias in apparent prevalence as a function of a range of differences in health state-specific detection probabilities. In the figure key, (p^U, p^I) are the values for detection probability of uninfected and infected individuals, respectively.

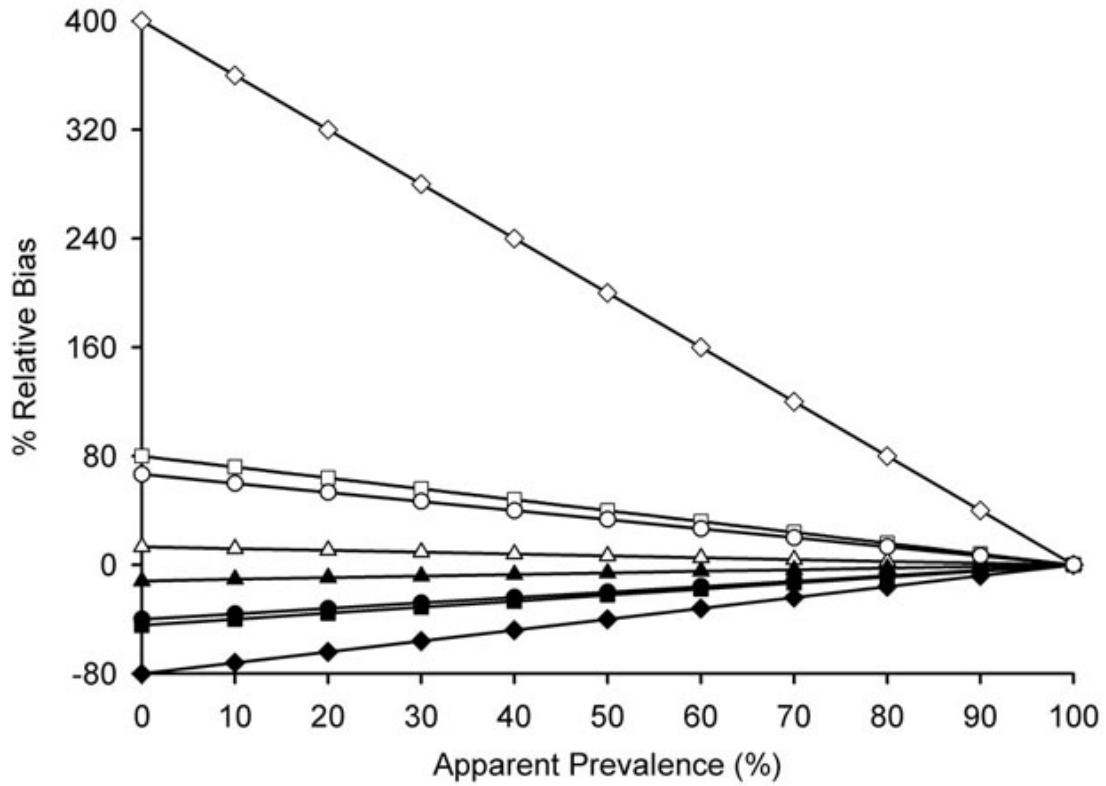

FIG. E2. Percent relative bias (%RB) as a function of a range of differences in health state-specific detection probabilities. In the figure key, (p^U, p^I) are the values for detection probability of uninfected and infected individuals, respectively.

[Back to A017-008]