Multi–state analysis of the impacts of avian pox on a population of Serins (*Serinus serinus*): the importance of estimating recapture rates

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Abstract

Multi–state analysis of the impacts of avian pox on a population of Serins (Serinus serinus)*: the importance of estimating recapture rates.*— Disease is one of the evolutionary forces shaping populations. Recent studies have shown that epidemics like avian pox, malaria, or mycoplasmosis have affected passerine population dynamics, being responsible for the decline of some populations or disproportionately killing males and larger individuals and thus selecting for specific morphotypes. However, few studies have estimated the effects of an epidemic by following individual birds using the capture–recapture approach. Because avian pox can be diagnosed by direct examination of the birds, we are here able to analyze, using multistate models, the development and consequences of an avian pox epidemic affecting in 1996, a population of Serins (*Serinus serinus*) in northeastern Spain. The epidemics lasted from June to the end of November of 1996, with a maximum apparent prevalence rate > 30% in October. However, recapture rate of sick birds was very high (0.81, range 0.37–0.93) compared to that of healthy birds (0.21, range 0.02–0.32), which highly inflated apparent prevalence rate. This was additionally supported by the low predicted transition from the state of being uninfected to the state of being infected (0.03, SE 0.03). Once infected, Serin avian pox was very virulent with (15–day) survival rate of infected birds being of only 0.46 (SE 0.17) compared to that of healthy ones (0.87, SE 0.03). Probability of recovery from disease, provided that the bird survived the first two weeks, however, was very high (0.65, SE 0.25). The use of these estimates together with a simple model, allowed us to predict an asymptotic increase to prevalence of about 4% by the end of the outbreak period, followed by a sharp decline, with the only remaining infestations being infected birds that had not yet recovered. This is in contrast to the apparent prevalence of pox and stresses the need to estimate recapture rates when estimating population dynamics parameters.

Key words: Avian pox, Epidemics, Serin, *Serinus serinus*, Survival, Capture–recapture.

Resumen

*Análisis mediante modelos de multiestados del impacto de la viruela aviar sobre una población de Verdecillos (*Serinus serinus*): la importancia de estimar las tasas de recaptura.*— Las enfermedades infecciosas son una de las fuerzas evolutivas que modulan a las poblaciones animales. Estudios recientes han puesto de manifiesto como epidemias como la viruela aviar, la malaria o la mycoplasmosis afectan a la dinámica de las poblaciones de passeriformes, siendo responsables de dramáticas reducciones en el tamaño de algunas poblaciones, o de la muerte desproporcionada de machos o de los individuos de mayor tamaño, seleccionando de ese modo en favor de determinados morfotipos. Sin embargo, pocos estudios han estimado los efectos de una epidemia mediante el seguimiento de los distintos individuos utilizando las técnicas de captura–recaptura. Debido al hecho de que la viruela aviar puede ser diagnosticada mediante el examen directo de los individuos, hemos podido analizar, utilizando modelos de multiestado, el desarrollo y consecuencias de una epidemia de viruela aviar que afectó en 1996, a una población de Verdecillos en el nordeste de España. La epidemia afectó a los Verdecillos desde junio hasta finales de noviembre, con una prevalencia aparente máxima de > 30% en octubre. Sin embargo, la tasa de recaptura de los individuos enfermos fue muy alta (0,81, rango 0,37–0,93), comparada con la de los individuos sanos (0,21, rango

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0,02–0,32), lo cual exageraba en gran medida la tasa de prevalencia aparente. Este resultado estaba adicionalmente apoyado por la baja tasa estimada de transición del estado de no infectado al estado de infectado (0,03, SE 0,03). Una vez un Verdecillo quedaba infectado, la viruela aviar resultó muy virulenta, siendo la tasa de supervivencia (a 15 días) de los individuos enfermos de tan solo 0,46 (SE 0,17), comparada con la de los individuos no infectados (0,87, SE 0,03). La probabilidad de recuperación de la enfermedad, siempre y cuando el individuo hubiera sobrevivido las dos primeras semanas, fue sin embargo, muy alta (0,65, SE 0,25). Estos valores fueron utilizados para construir un modelo que permitió predecir el valor real de prevalencia de la enfermedad. Según el modelo, el porcentaje de individuos infectados después del brote debió incrementarse de forma asintótica hasta el 4%, manteniéndose en ese valor, hasta que se produjo una abrupta reducción en el número de individuos infectados al final de la epidemia, siendo estos los individuos que todavía no se habían recuperado de la enfermedad. Estos valores contrastan con los valores aparentes de prevalencia de la viruela y enfatiza la necesidad de estimar la tasa de recaptura cuando se realizan estimaciones de los distintos parámetros de dinámica de poblaciones.

Palabras clave: Viruela aviar, Epidemia, Verdecillo, *Serinus serinus*, Supervivencia, Captura–recaptura.

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Introduction

It is increasingly recognised that infectious disease may shape animal populations (May, 1983; Scott, 1988; Clayton & Moore, 1997; Newton, 1998; Hudson et al., 2001). In North America during 1994 mycoplasmosis was recorded to have spread throughout the east coast in just two years (Fischer et al., 1997; Hochachka & Dhondt, 2000) and to have killed some 225 million birds (Nolan et al., 1998). Avian pox and malaria were responsible for the decline of several Hawaiian bird populations (Ralph & Fancy, 1995; Van Ripper III et al., 2002; Benning et al., 2002) and of some continental bobwhite quail and wild turkey populations in the southeastern United States (Hansen, 2004), and recent models of metapopulation dynamics consider disease as an important factor to have into account for the conservation of endangered populations (Woodroffe, 1999; Gog et al., 2002; Hess, 2003). Recent studies have shown that epidemics like avian pox and mycoplasmosis have affected passerine population dynamics, disproportionately killing males and larger individuals and thus selecting for specific morphotypes (Thompson et al., 1997; Nolan et al., 1998; Brawner et al., 2000). Avian pox and mycoplasmosis are also responsible for shifts in mean plumage colour of whole populations, which may have important consequences on the strength of sexual selection in these populations (Thompson et al., 1997; Zahn & Rothstein, 1999). Given the importance of disease in animal populations, several national programs have been developed to follow up infection dynamics (e.g., House finch Conjunctivitis survey) (Dhondt et al., 1998; Hartup et al., 2001). However, most studies have to rely on the establishment of prevalence (% infected birds) of the disease and few studies have estimated the effects of an epidemic by following individual birds by capture–recapture techniques (e.g., Faustino et al., in press). This is of importance because sick birds may have higher or lower probabilities of capture/recapture (e.g., McClure, 1989; Faustino et al., in press), thus biasing estimates of prevalence of the infection (Williams et al., 2002).

Avian pox is viral infection of birds caused by *Poxvirus avium*. The disease is worldwide in distribution (Hansen, 1987; Van Ripper III et al., 2002), and occurs in two forms; (1) most commonly, a skin form with warty lesions, mostly on the unfeathered body; and (2) a diptheritic form, which involves the mouth and respiratory tract. Transmission may occur either directly, by contact among infected birds, or with mechanical transfer via biting insects, especially mosquitoes (Hansen, 1987; Van Ripper III et al., 2002). Although usually not directly lethal, the disease may increase the vulnerability of birds to other risks, such as predation or secondary infections (Hansen, 1987; Van Ripper III et al., 2002; Gortazar et al., 2002). Infection is thought to confer immunity to the disease that lasts 12–18 months (Boch & Schneidawind, 1994), although this figure may vary among species, sometimes immunity being permanent (Arnall & Keymer, 1975; Del Pino, 1977). Recovery time is variable, but generally within 25 days of exposure (Del Pino, 1977).

In our study we used capture–mark–recapture (CMR) and multistate models, to model the development and consequences of an avian pox epidemic affecting a population of Serins (*Serinus serinus*) in northeastern Spain. Our approach allows the estimation of probabilities of infection and of disease recovering, and of survival rate of infected and uninfected birds, parameters that in natural populations are otherwise very difficult to estimate.

Methods

Field methods

The study was carried out at the Desert de Sarria, a ringing station (3 Ha) within the suburban area of Barcelona (NE Spain). The area is formed by orchards, small pine woods (*Pinus halepensis*) and gardens, which conforms a typical Serin habitat (Senar, 1986). Serins have been trapped there since 1985 on a weekly basis using platform and funnel traps, clap–nets and mist nets, all of them except mist–nets, associated with baited feeders; the use of several trapping devices allowed to reduce biases in trapping probabilities of different sex and age classes, allowing to obtain a representative sample of birds in the population (Yunick, 1971; Domènech & Senar, 1997, 1998; Conroy et al., 1999; Domènech et al., 2001). In June 1996 we had an outbreak of avian pox. From this time forward we implemented special procedures for the handling of care infected birds. We employed dedicated containers and measuring devices for the infected birds, and wore clinical gloves, disinfecting hands with Halamid™ after handling birds. We also disinfected traps after each capture and several times during the following week. Avian pox typically causes discrete, warty and proliferative lesions on the skin of legs, feet, eyelids and the base of the beak, and so can easily be diagnosed by visual inspection of the birds (Hansen, 2004). We confirmed we were dealing with avian pox by histopathologic examination of a bird which showed the typical eosinophilic intracytoplasmic inclusions diagnostic of avian pox (Laboratorio de Diagnóstico Veterinario) (Gortazar et al., 2002; Hansen, 2004). Hence, here "infected" denotes exhibiting signs of the disease, i.e., symptomatic, and "uninfected" denotes the absence of symptoms. Other studies have found good correspondence between exhibition of lesions and actual prevalence of avian pox; for instance Van Riper III et al. (2002) confirmed presence of avian pox in 20 of 22 histopathological examination of tissue from birds exhibiting pox–like lesions. Nevertheless, we recognise that some infected birds may have been asymptomatic and thus our analysis potentially incorrectly classi-

fies some birds as uninfected. In fact, some pox virus have developed strategies to minimise external appearance (Seet et al., 2003). Later, we discuss implications of misclassification for interpreting our results.

Statistical analyses

Incidence of pox was confined to Jun–Nov of 1996 (table 1); therefore, we confined our analyses to birds captured and or recaptured during May–December. To avoid issues of unidentified juvenile sex classes and transience (Conroy et al., 1999) analyses were confined to within–year recaptures, with the years constituting a grouping variable along with age and sex class. Within each year we identified 5 age–sex classes based on plumage characteristics: adult male (AM), adult female (AF), subadult male (SM), subadult female (SF), and juveniles (J), for which sex could not be identified. These were later grouped into 3 categories, in which adult and subadult classes were combined and identified by sex (M, F) and juveniles considered separately (J).

For 1996, we constructed multistate recapture models using program MARK (White & Burnham, 1999). Captures and recaptures were grouped by 15–day periods from 1 May–31 December. At each sampling occasion individuals were classified as exhibiting active pox lesions (P) or not (N), and modelled state–specific survival, capture, and transition probabilities. As discussed below, the state N may actually be a mixture of birds who are susceptible to the disease, and those who have been infected and are now immune. Specifically, we used the multistate data structure, by 15–day period and stratified by age–sex categories, to estimate $S_t^{s,as}$ the probability of 15–day survival for birds in state *s* (1 = uninfected or 2 = infected), age–sex as = AM, AF, SM, SF, or J over [*t*, *t*+1], *t* = 1,...,15; $P_t^{s,as}$, the probability of capture at occasion of *t* = 2,...,16 for birds state *s* and age–sex *as*; and $\psi_t^{s,r,as}$ the probability of movement to state *r* at *t*+1 for birds in state *s* at *t*, age–sex *as*, at sampling period *t* = 1,...,15. We employed 15–day periods as the shortest interval over which data could be grouped while providing sufficient data for estimation. Because we had no data on recovery times for pox–infected Serins, we desired this interval to be as short as possible so as to allow estimation of rates of recovery. Data from other similar species suggests recovery period to be of about 25 days (Del Pino, 1977), which validates our analysis interval in detecting rates of recovery. Unlike the CJS analysis (described below), this analysis focussed on modelling survival, capture, and transition processes within a single year. However, our *a priori* expectation was that age, sex, and state (infected or not) and other individual attributes (considered below) would account for greater variability in survival and capture rates, than would variation among 15–day periods. In addition, data were sparse, particularly captures and recaptures of infected birds. We thus constructed a number of models in which additive age–sex and state effects were modelled, using the design matrix feature in MARK. We also attempted to fit a "global" model in which group effects (age–sex and year) interacted with time (recapture occasions), for comparison to constrained–parameter models. We used *c* as a measure of model fit/ over dispersion, estimated by

$$\hat{c} = \chi^2 / df,$$

where χ^2 is the deviance (–2 $\log_e$ [likelihood]) statistic and *df* is computed as the number of independent multinomial cells minus the number of parameters estimated. However, sparse data render deviance–based statistics unreliable as measures of fit, and we therefore conducted 250 bootstrap simulations under a highly–parameterized ("global") model and compared the mean of the bootstrap estimate of *c* under this model to that under the corresponding estimated model to obtain $\hat{c}$ as

$$\hat{c} = \frac{\hat{c}_{sample}}{\hat{c}_{bootstra}} \quad .$$

Because bootstrap goodness of fit tests are not currently available in MARK, we developed a bootstrap program using a modification of the SAS code (simulate.sas) provided by G. White as part of MARK, integrated via a SAS macro with a batch version of MARK; this code is available from the second author at http://coopunit.forestry.uga.edu/conroy/software/bootstrap.txt. We then used this adjustment in program MARK to compute $QAIC_c$ (quasilikelihood–adjusted AIC, corrected for small sample size; (Burnham & Anderson, 2002) and $\Delta QAIC_c$, where

$$\Delta QAIC_c(i) = \Delta QAIC_c(i) - \Delta QAIC_c(\min)$$

and $\Delta QAIC_c(\min)$ was the model under consideration having the lowest value for $\Delta QAIC_c$. This statistic was in turn used to compute model weights (w_i) for each competing model as

$$w_i = \frac{\exp(-\Delta QAIC_c(t)/2)}{\sum_{j=1}^{R} \exp(-\Delta QAIC_c(j)/2)},$$

where *R* is the number of models in the set of candidate models. We then used model averaging (Burnham & Anderson, 2002) to obtain estimates of parameters by

$$\bar{\theta} = \sum_{i=1}^{k} w_i \hat{\theta}_i$$

and of unconditional standard errors by

$$se(\bar{\theta}) = \sqrt{\sum_{i}^{k} w_i \left[(\theta_i - \bar{\theta})^2 + \mathrm{var}(\hat{\theta}_i \,|\, \theta_i) \right]}$$

where $\hat{\theta}_i$ and $\mathrm{var}(\hat{\theta}_i \,|\, \theta_i)$ are the estimates of θ and its conditional (sampling) variance under model *i*. These were used to create asymptotic normal confidence intervals by multiplication with the 0.05 and 0.95 standard normal deviates.

Table 1. Monthly frequency of pox incidence for Serins captured and recaptured during 1996: [a] Infection status determined by external examination (presence or absence of warty lesions characteristic of avian pox). "Not infected" birds may include some birds that have been previously infected and are likely immune (see text).

Tabla 1. Incidencia mensual de viruela aviar en los Verdecillos capturados y recapturados durante 1996: [a] Estatus de infección determinado mediante inspección externa (presencia o ausencia de lesiones ulcerosas características de la viruela aviar). La categoría de "no infectado" puede incluir a algunas aves previamente infectadas pero que han desarrollado inmunidad (ver texto).

	Not infected[a]			Infected		
Month	Male	Female	Juvenile	Male	Female	Juvenile
January	37	20	78	0	0	0
February	30	13	71	0	0	0
March	56	28	111	0	0	0
April	28	13	68	0	0	0
May	42	30	49	0	0	0
June	24	13	116	0	0	1
July	12	9	56	0	0	1
August	12	11	85	2	4	2
September	12	11	128	0	2	17
October	11	9	59	1	0	27
November	12	11	81	0	1	4
December	7	4	51	0	0	0
Total	283	172	953	3	7	52

To provide background estimates of survival rate and of annual variation in within–year survival, and to aid in the interpretation of state transitions for asymptomatic ("1") birds (see below), we conducted a Cormack–Jolly–Seber (CJS) analyses of captures and recaptures at 15–day intervals from 1 May to 31 December in all years except 1996. As with the multistate analysis, we wished to avoid issues of unidentified juvenile sex classes and transience. Thus, the CJS analyses were confined to within–year recaptures, with the years constituting a grouping variable along with age and sex class. Within each year we identified 5 age–sex classes as above, later grouped into males, females, and juveniles. The parameters of the CJS model were survival probabilities $\phi_t^{as,y}$ and capture probabilities $p_t^{as,y}$ where t = 1,...,16 sampling occasions correspond to the 15–day intervals, as = 1,...,5 are the age–sex categories, and y = 1,...,10 are the years (1990–2000 excluding 1996). Because we anticipated greater year–to–year variation in survival probability than variation among 15–day intervals within year, we constructed a number of constrained–parameter models using MARK. We were particularly interested in modelling year–to–year variation in age–sex specific survival for comparison to survival during the year of pox epidemic. We therefore constructed a number of models in which additive age–sex and year effects were modelled, using the design matrix feature in MARK. We also attempted to fit a "global" model in which group effects (age–sex and year) interacted with time (within–year recapture occasions), for comparison to constrained–parameter models. Because of sparse data, goodness of fit based either on RELEASE or on deviance statistics were unreliable. We therefore conducted 500 bootstrap simulations under a "global" model and compared the mean of the bootstrap deviance to compute statistics under this model to the deviance under the corresponding estimated model to obtain $\hat{c}$ as described above, with computations performed within MARK.

We were also interested in the possible relationship of individual covariates (measured upon first capture) to state–specific survival and to the probability of transition between states. In particular, we identified mass, and body size as measured by wing length, tail length, and length of P3, as potentially influencing one or both of these rates. We used the design matrix feature of MARK to incorporated predictive relationships of the form

$$\log\left[\frac{S_i^s}{1-S_i^s}\right] = \beta_0^s + \sum_{i=1}^{k}\beta_i X_i \ ,$$

and

$$\log\left[\frac{\psi_i^s}{1-\psi_i^s}\right] = \alpha_0^s + \sum_{j=1}^{k}\alpha_j X_j \ ,$$

where S_i^s is predicted survival over [t, t+1] for individual i in state s, ψ_i^s is predicted transition over [t, t+1] to the alternate state (s') for individual i in state s at time t, β_0^s, α_0^s are state–specific effects on survival and transition, j = 1,...,k are individual covariates, β_j, α_j are coefficients including dummy variables expressing levels for age–sex and (potentially) capture occasion, and slopes for the covariate effects. For covariates we used standardized, individual mass, wing length, tail length, and P3 length and interactions of these with grouping variables (age–sex, state) as appropriate. Standardization was within age–sex class, with resulting predictors represent deviation from the–within–class mean. Because individual covariates were not taken for all individuals, we formed 3 subsets of the data, in which (1) mass, (2) mass and wing length, and (3) all covariates were recorded. For each subset, we selected the top–ranked multistate model (covariates absent) as a baseline model, and modified this model to incorporate covariate effects. We performed model evaluation and selection as above, with based on the bootstrap results from the multistate, no covariate model.

Results

During May–December 1996 we captured 428 individual Serins for a total of 1,470 capture–recapture events. We captured birds in the "infected" state on 62 occasions, representing 42 individuals (total prevalence of 9.8%), and the majority (52 captures of 37 birds) were of juvenile birds (table 1). Avian pox appeared from June to November. When stratifying by 15–day periods, apparent prevalence raised by the second half of October to 33% of birds trapped being infected (fig. 1). Birds differed in the part of the body infected: 13% of birds had legs infected, 34% the eyelids and 53% the base of the beak, with 10 individuals having both infected eyelids and the base of the beak (n = 42).

Data on the 1,470 capture–recapture events was used in the multistate modelling. Due to sparse data we were unable to estimate parameters under a global model incorporating time and group effects for all parameters. We instead used model $S_{pox}p_{pox}\psi_{pox+as}$ incorporating state and age–sex effects for survival and transition, and additive state and time effects for capture. We compared for this model (11.28) to the mean from 250 bootstrap simulations (4.65) to obtain an estimate of $\hat{c}$ = 2.43 for use in model evaluation and comparison (table 2). We selected model $S_{pox}p_{pox+t}\psi_{pox}$ as the best candidate model, allowing for state–specific effects on survival, capture, and transition, and additive time effects on capture probabilities. Several other models had non–negligible $QAIC_c$ weights; thus we used model averaging to obtain estimates and unconditional confidence intervals of state–specific survival and transition and of state and time–specific capture probabilities (table 3). These results show, first, a notably higher survival rate for birds captured as "uninfected" ($\hat{S}$ = 0.868, SE = 0.025) than "infected" ($\hat{S}$ = 0.458, SE = 0.17) and second, a higher rate of transition from "infected to uninfected" than the reverse ($\hat{\psi}^{S,N}$ = 0.654, SE = 0.254 *vs.* $\hat{\psi}^{N,S}$ = 0.032, SE = 0.030, table 3).

The CJS analysis confirms that birds in the "infected" state had an unusually low probability of survival, taking into account yearly variation in these rates. We selected model $\phi_{as+y+t}p_{as+y+t}$, incorporating age–sex, year–to–year, and within–year time variation on survival and capture probabilities; all other models had negligible credibility ($\Delta QAIC_c$ > 36). Because we were interested in comparison of our point estimate of state–specific survival to yearly variation in group–specific survival, we computed annual estimates of survival as the average over the 15 within–year capture occasions, with confidence intervals based on the ordinary variance among these empirical estimates. The confidence intervals thus include components of both sampling and temporal variation in within–year survival. We plotted these estimates over the years of the CJS analysis, together with the state–specific estimates of survival for 1996 from the multistate analysis. Birds captured in the "infected" state had a clearly lower probability of survival, lower than even the most extreme (early) years of the CJS analysis (fig. 2). We note that both the CJS and multistate models likely underestimate "true" survival, in that permanent emigration is confounded in these estimates of "apparent survival"; nevertheless we take these analyses as strong evidence of a state–specific influence on survival. However, these results are affected both by 1) potential misclassification of states (N or P) and 2) the fact that some asymptomatic (state N) birds may have been immune, due to previous infection. We discuss both of these issues in more detail below.

For the covariate analysis, we used recaptures of 417 birds for which mass was recorded; 333 birds for which both mass and wing length were recorded, and 323 birds for which all covariates (mass, wing length, tail, and P3) were recorded. These data subsets were used to fit series of covariate models, summarized in table 4. Although several of the covariate models are close competitors to the "baseline" (no covariate) model, the baseline model was the top–ranked model in 2 of the 3 data subsets. Model averaging resulted in unconditional estimates of covariate effects with large standard errors and confidence intervals widely dispersed near the origin, indicative of weak effects (table 5). Furthermore, coefficient signs for comparable models sometime differed among data subsets (e.g., $\hat{\beta}_{wt}$ positive for the first and third but negative for the second subset). We conclude that the evidence for covariate effects on survival and transition is weak for this study, probably due to the relatively small sample of the "infected" state. On the other hand,

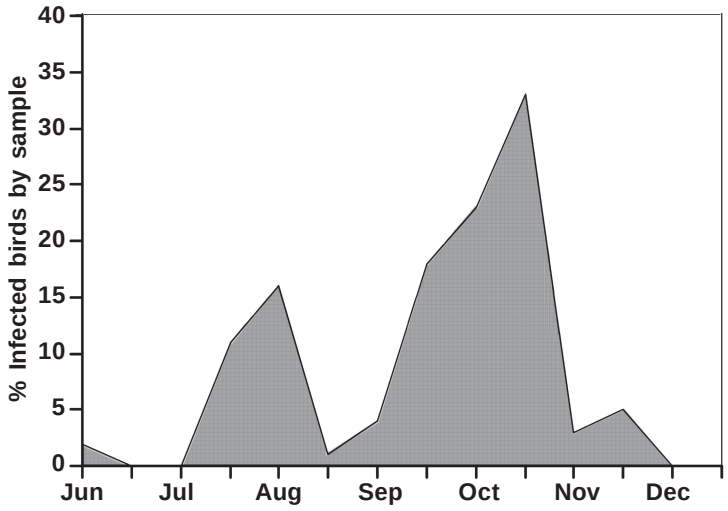

Fig. 1. Apparent prevalence rate of Serin avian pox, computed as the percentage of infected birds from total number trapped by 15–day periods (*n* = 799 birds).

Fig. 1. Tasa de prevalencia aparente de la viruela aviar en el Verdecillo, computado como el porcentaje de aves infectadas sobre el total de capturadas para periodos de 15 días (n = 799 aves).

other individual attributes, notably the state of pox infection, were clearly related to survival rates; furthermore, transition between states is clearly asymmetric, with birds surviving from pox more likely ($\hat{\psi}^{2,1}$ = 0.654) to move to the uninfected states than the reverse ($\hat{\psi}^{1,2}$ = 0.032).

Discussion

The Serin avian pox outbreak mainly affected birds from September to November, which seems to be the most common period of high prevalence for this kind of disease (Davidson et al., 1980; Van Ripper

Table 2. Multistate models of state–specific survival, capture, and transition for Serins captured May–December 1996. [a] Factors used to model variation in 15–day survival (*S*), capture (*p*) and transition probabilities (ψ): pox. Classified as uninfected (1) or infected (2) at time of release; *as. A*ge–sex groupings (male, female, and juvenile [unsexed]); *t.* 15–day capture; [b] Quasilikelihood $\hat{c}$ = 2.43.

*Tabla 2. Modelos de multiestado con tasas de supervivencia, captura y transición dependientes del estado, para los datos de Verdecillo capturados de mayo a diciembre de 1996. [a] Factores utilizados para modelar la variación en la tasa de supervivencia a 15 días (*S*), tasa de captura (*p*) y probabilidades de transición probabilities (ψ): pox (viruela). Clasificada como no infectados (1) o infectados (2) en el momento de la liberación;* as. *Agrupación según edad–sexo (macho, hembra, y juvenil [no sexado]);* t. *Período entre capturas de 15 días, [b] Quasi razón de verosimilitud $\hat{c}$ = 2,43.*

Model[a]	$QAIC_c$ [b]	$QAIC_c$ weights	Num. Par
$S_{pox}\ p_{pox+t}\ \psi_{pox+as}$	0	0.46837	20
$S_{pox}\ p_{pox+t}\ \psi_{pox+as}$	0.9563	0.29035	22
$S_{pox+as}\ p_{pox+t}\ \psi_{pox}$	2.7874	0.11623	22
$S_{pox+as}\ p_{pox+t}\ \psi_{pox+as}$	3.8465	0.06844	24
$S_{pox}\ p_{pox+t}\ \psi_{as}$	5.343	0.03239	20
$S_{as}\ p_{pox+t}\ \psi_{pox}$	6.0193	0.02309	21
$S_{pox}\ p_{pox+t}\ \psi_{as}$	12.0621	0.00113	22

Table 3. Model–averaged estimates and unconditional confidence intervals for state–specific 15–day survival, capture, and transition for Serins captured May–December 1996: [a] Infection status determined by external examination (presence or absence of warty lesions characteristic of avian pox). "Not infected" birds may include some birds that have been previously infected and are likely immune (see text).

Tabla 3. Estimas promediadas entre modelos e intervalos de confianza no condicionados para las tasas específicas de cada estado de supervivencia a los 15 días, de captura, y de transición para los Verdecillos capturados de mayo a diciembre de 1996. [a] Estatus de infección determinado mediante inspección externa (presencia o ausencia de lesiones ulcerosas características de la viruela aviar). La categoría de "No infectado" puede incluir a algunas aves previamente infectadas pero que han desarrollado inmunidad (ver texto).

					95% CI	
Parameter	State[a]	Period	θ	SE	Lower	Upper
Survival (*S*)	Uninfected		0.868	0.025	0.809	0.910
	Infected		0.458	0.171	0.180	0.765
Capture (*p*)	Uninfected	2	0.194	0.089	0.074	0.422
		3	0.242	0.079	0.121	0.426
		4	0.234	0.072	0.122	0.400
		5	0.154	0.051	0.078	0.282
		6	0.019	0.017	0.003	0.104
		7	0.048	0.031	0.013	0.158
		8	0.253	0.065	0.147	0.400
		9	0.342	0.071	0.218	0.492
		10	0.246	0.062	0.145	0.387
		11	0.315	0.074	0.190	0.474
		12	0.108	0.047	0.045	0.240
		13	0.210	0.067	0.108	0.369
		14	0.391	0.092	0.231	0.578
		15	0.041	0.031	0.009	0.168
		16	0.296	0.083	0.162	0.479
	Infected	2	0.861	0.215	0.153	0.995
		3	0.886	0.176	0.205	0.9957*
		4	0.882	0.181	0.198	0.996
		5	0.832	0.254	0.123	0.994
		6	0.373	0.444	0.014	0.961
		7	0.602	0.424	0.045	0.980
		8	0.890	0.167	0.221	0.996
		9	0.916	0.126	0.304	0.996
		10	0.887	0.169	0.224	0.995
		11	0.910	0.135	0.289	0.996
		12	0.774	0.306	0.100	0.991
		13	0.870	0.198	0.178	0.995
		14	0.926	0.111	0.342	0.997
		15	0.570	0.509	0.022	0.987
		16	0.905	0.146	0.257	0.996
Transition (ψ)	Uninfected		0.032	0.030	0.005	0.178
	Infected		0.654	0.254	0.173	0.945

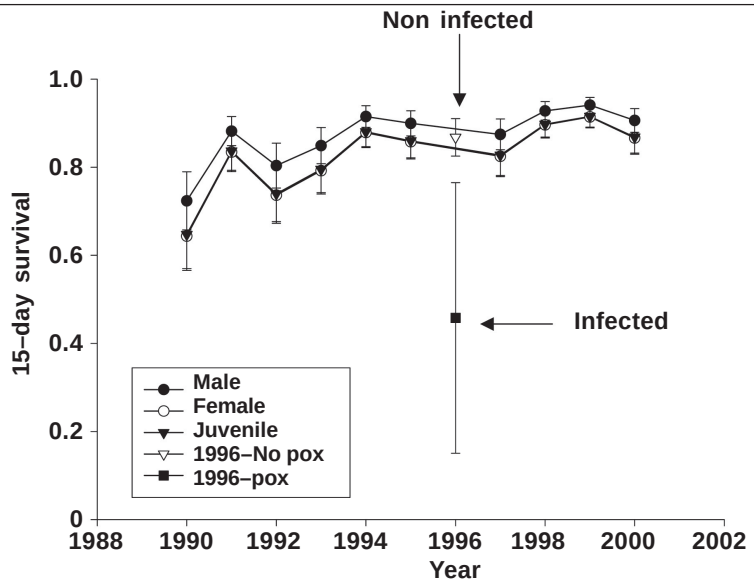

Fig. 2. Estimates of mean 15–day survival and empirical confidence intervals (within year) based on CJS modelling for Serins by age–sex group for 1990–2000 (excluding 1996). Superimposed estimated state–specific survival rates for 1996: Uninfected. Probability of survival for Serins uninfected by pox at time *t*; Infected. Probability of survival for infected birds.

Fig. 2. Estimas de la tasa de supervivencia quincenal media e intervalos de confianza empíricos (dentro del año) basado en modelos para los Verdecillos de CJS, por grupo de edad–sexo para el periodo 1990–2000 (excluyendo 1996). Superpuesto se proporciona la estima de la tasa de supervivencia específica del estado para 1996: No infectado. Probabilidad de supervivencia para los Verdecillos no infectados por la viruela en el tiempo t; Infectado. Probabilidad de supervivencia para las aves infectadas.

III et al., 2002; Buenestado et al., in press). Serin avian pox seemed to be very virulent, reducing 15–day survival from 0.87 in healthy birds to 0.46 in infected birds, which means that more of 50% of sick birds did not survive to the first two weeks after infection. Nevertheless, if infected birds survive, probability to recovery from pox seemed quite high (0.65). This is in accordance with data from other species, which have reported a high percentage (15–60%) of birds with healed lesions from previous infections, which reflects that the bird survived to the disease (McClure, 1989; Van Ripper III et al., 2002). A higher recovery than infection rate has also been reported for the Mycoplasma outbreak in eastern United States (Faustino et al., in press). Models involving the effects of age and sex, and interaction with disease state, generally were not supported. This does not necessarily indicated that such effects did not occur, and may be due to the sparseness of our data.

As noted earlier, two aspects of our data collection have implication for these analyses. First, because not all captured birds were subjected to histopathology, we may have incorrectly classified some birds as "not infected" that in fact had the disease; conversely, some birds exhibiting lesions may have been falsely identified as "infected" (although this is less probable). Second, birds that have been previously infected with pox may exhibit no visible lesions, and are thus indistinguishable form birds that have not been infected. This potentially creates an indistinguishable mixture of previously exposed (and thus, presumed to be immune) birds, and birds that have not been exposed (and are therefore at risk to the disease). We consider both of these issues, and their implications for our study, below.

Misclassification of states

Van Riper et al. (2002) found that more than 90% (N = 22) of birds with lesions were histopathologically positive for the disease. We only histologically examined 1 bird, and so cannot compute an estimate of a "false positive" rate; however, we are confident that most if not all birds with lesions were either currently infected with pox, or had recently been infected and were recovering. We think that it is much more likely that we misclassified as uninfected, perhaps because they were mildly symptomatic (e.g., had few or no lesions; Seet et al., 2003) and these were missed during our field examinations. Classifying some infected birds as healthy should have caused our state–specific survival rates

Table 4. Multistate models of covariate relationships to state–specific survival, capture, and transition for Serins captured May–December 1996: [a] Quasilikelihood $\hat{c}$ = 2.43.

Tabla 4. Modelos de multiestados de las relaciones de las covariantes con la tasa de supervivencia, de captura y de transición, dependientes del estado, para los Verdecillos capturados de mayo a diciembre de 1996: [a] Quasi razón de verosimilitud $\hat{c}$ = 2,43.

Covariates included	Model	$\Delta QAIC_c$	$QAIC_c$ weights	Num. Par
Mass	$S_{pox}\ p_{pox+t}\ \psi_{pox}$	0.000	0.338	19
	$S_{pox+wt}\ p_{pox+t}\ \psi_{pox}$	0.679	0.241	20
	$S_{pox}\ p_{pox+t}\ \psi_{pox+wt}$	1.451	0.164	20
	$S_{pox+wt}\ p_{pox+t}\ \psi_{pox+wt}$	2.390	0.102	21
	$S_{pox*wt}\ p_{pox+t}\ \psi_{pox}$	2.796	0.084	21
	$S_{pox}\ p_{pox+t}\ \psi_{pox*wt}$	3.520	0.058	21
	$S_{pox*wt}\ p_{pox+t}\ \psi_{pox*wt}$	6.553	0.013	23
Mass, wing length	$S_{pox}\ p_{pox+t}\ \psi_{pox*wt+wng}$	0.000	0.414	22
	$S_{pox}\ p_{pox+t}\ \psi_{pox}$	1.406	0.205	20
	$S_{pox+wt+wng}\ p_{pox+t}\ \psi_{pox+wt+wng}$	2.571	0.114	24
	$S_{pox+wt}\ p_{pox+t}\ \psi_{pox+wt}$	2.573	0.114	22
	$S_{pox+wng}\ p_{pox+t}\ \psi_{pox+wng}$	3.038	0.091	22
	$S_{pox+wt+wng}\ p_{pox+t}\ \psi_{pox}$	3.948	0.058	22
	$S_{pox*wt+pox*wng}\ p_{pox+t}\ \psi_{pox*wt+pox*wng}$	9.109	0.004	28
Mass, wing length, tail, P3	$S_{pox}\ p_{pox+t}\ \psi_{pox}$	0.000	0.662	20
	$S_{pox+wt+wng}\ p_{pox+t}\ \psi_{pox+wt+wng}$	1.552	0.305	24
	$S_{pox+tail+p3}\ p_{pox+t}\ \psi_{pox+tail+p3}$	6.480	0.026	24
	$S_{pox+wt+wng+tail+p3}\ p_{pox+t}\ \psi_{pox+tail+p3}$	9.083	0.007	28

to be biased low, so that the actual relative survival of uninfected to infected birds would have been even greater than what we observed. On the other hand, our low estimated rates of infection (i.e., transition to infected from evidently uninfected) could have been partially an artefact of some infected birds being misclassified as uninfected. However, we think that the misclassification problem is relatively unimportant compared to the second problem related to immunity, discussed below. Additionally, if the presence of asymptomatic infected birds (see Seet et al., 2003) within Serins, had been very important, it should had reduced survival rate of "apparently" uninfected birds, but the comparison of this survival rate to that of birds in years with no pox, suggests that this is not the case.

Unidentified asymptomatic (immune) birds

Our statistical models, which are based on models of disease transmission (Bailey, 1975; Anderson & May, 1992; Clayton & Moore, 1997), differ from these in certain aspects that are critical to interpreting our results. Like our models, infectious disease models often define disease states, and model rates of transition between these states. Additionally, our models take into account imperfect and potentially heterogeneous detectability (capture), which otherwise could confound inferences. Disease models typically assume that living individuals are in 1 of 3 possible states: susceptible (*X*), i.e., never having been infected and thus having no immunity; infected (*Y*), and post–infected (*Z*), often (but not always) assumed immune (incapable of reinfection). We can model transition among these states by ϕ_i^{XY}, the probability that over the interval [*i*, *i*+1] a susceptible individual becomes infected ϕ_i^{YZ}, the probability that an infected animal becomes immune, and ϕ_i^{ZX}, ϕ_i^{ZY} the probabilities that a post–infected animal becomes either susceptible or reinfected. These last 2 probabilities are assumed zero in the case where exposure confers complete immunity; likewise it would ordinarily be assumed that $\phi_i^{XZ} = 0$. i.e., an animal must first become infected before becoming "post–infected" or immune. If (as we have done in this paper) we model transition as the product survival conditioned on the state at the first occasion and movement between states, then

Table 5. Model–averaged estimates and unconditional confidence intervals for estimates of covariate relationships for survival transition for Serins captured May–December 1996: LCI. Lower 95% confidence interval; UCI. Upper 95% confidence interval.

Tabla 5. Estimas promediadas para los distintos modelos e intervalos de confianza no condicionados para las estimas de la relación de las covariantes con la tasa de supervivencia, para los Verdecillos capturados de mayo a diciembre de 1996: LCI. Intérvalo de confianza inferior del 95%; UCI. Intérvalo de confianza superior del 95%.

Covariates included	Parameter	θ	SE	LCI	UCI
Mass	β_0	–0.149	0.638	–1.399	1.102
	β_{pox}	2.060	0.698	0.694	3.430
	β_{wt}	0.080	0.260	–0.429	0.589
	β_{pox*wt}	–0.001	0.254	–0.498	0.497
	γ_0	0.731	0.860	–0.954	2.416
	γ_{pox}	–3.744	0.898	–5.503	–1.984
	γ_{wt}	–0.112	0.415	–0.925	0.701
	γ_{pox*wt}	0.019	0.329	–0.626	0.664
Mass, wing length	β_0	–0.282	0.686	–1.627	1.063
	β_{pox}	–2.069	0.733	0.632	3.507
	β_{wt}	–0.071	0.173	–0.267	0.410
	β_{wng}	0.008	0.085	–0.157	0.174
	β_{pox*wt}	–0.002	0.103	–0.204	0.200
	$\beta_{pox*wng}$	–0.001	0.058	–0.114	0.113
	γ_0	0.786	1.053	–1.278	2.851
	γ_{pox}	–4.223	1.273	–6.719	–1.728
	γ_{wt}	–0.499	0.610	–1.695	0.698
	γ_{wng}	0.381	0.405	–0.413	1.176
	γ_{pox*wt}	–0.001	0.148	–0.291	0.289
	$\gamma_{pox*wng}$	0.007	0.132	–0.252	0.265
Mass, wing length, tail, P3	β_0	–0.194	0.713	–1.592	1.204
	β_{pox}	1.963	0.748	0.497	3.430
	β_{wt}	0.060	0.145	–0.225	0.344
	β_{wng}	0.021	0.102	–0.178	0.220
	β_{tail}	–0.003	0.048	–0.098	0.092
	β_{p3}	0.000	0.052	–0.101	0.102
	γ_0	0.929	1.014	–1.058	2.917
	γ_{pox}	–4.363	1.167	–6.651	–2.075
	γ_{wt}	–0.248	0.460	1.150	0.654
	γ_{wng}	0.199	0.354	–0.494	–0.892
	γ_{pox*wt}	–0.003	0.105	–0.208	0.202
	$\gamma_{pox*wng}$	0.014	0.127	0.235	0.262

$$\phi_i^{XY} = S_i^X \psi_i^{XY}$$
$$\phi_i^{XX} = S_i^X (1- \psi_i^{XY}),$$
$$\phi_i^{YZ} = S_i^Y \psi_i^{YZ},$$
$$\phi_i^{YY} = S_i^Y (1- \psi_i^{YZ}),$$

$$\phi_i^{ZZ} = S_i^Z \quad \text{(assuming immunity).}$$

For the Serin problem, it seems to us that we cannot completely observe these 3 states. Rather,

subject to misclassification, we believe that our observed state "2" corresponds to the state *Y* (infected), and retrospectively–i.e., when an observed capture of "2" is later followed by "1" (asymptomatic), we believe that we can infer *Z,* the post–infection state, but only if birds are recaptured and observed to be uninfected after having previously been observed to be infected. When birds are either observed for the first time as asymptomatic ("1"), recaptured as still asymptomatic (e.g., a capture history of 101), or never recaptured (e.g., 100), we think that these birds' actual state is unknown (either *X* or *Z*). In principle it might also be possible to infer previous infection from the existence of healed lesions; however we observed very few of these; nearly all birds either had active lesions, or were observed to have no lesions. Antibody tests (which we did not conduct) also could reveal that the bird had previously been exposed, and was now immune (*Z*). Thus we conclude that our observable states consisted of *Y* (infected), *Z* (based on previous capture in the state *Y*), and *U* (unknown–free of lesions but not known based on prior capture to be either susceptible or immune). The state *U* is thus an (unknown) mixture of either susceptible (*X*) or immune (*Z*)

$$N_U = \pi N_X + (1 - \pi)N_Z$$

with π the mixing proportion. We can model observable transitions (conditional on recapture) transitions for these 3 states as:

$$\Pr(UU) = \pi_i\phi_i^{XX} + (1 + \pi_i)\phi_i^{ZZ} = \pi_i S_i^X \psi_i^X + (1 - \pi_i) S_i^Z \psi_i^{ZZ},$$
$$\Pr(UY) = \pi_i\phi_i^{XY} + (1 + \pi_i)\phi_i^{ZY} = \pi_i S_i^X \psi_i^{XY}$$

(assuming complete immunity, i.e., $\psi^{ZY} = 0$,

$$\Pr(YY) = \phi_i^{YY} = S_i^Y (1 - \psi_i^{YZ}),$$
$$\Pr(YZ) = \phi_i^{YY} = S_i^Y \psi_i^{YZ},$$
$$\Pr(ZZ) = \phi_i^{ZZ} = S_i^Z,$$

(assuming complete immunity, i.e., $\psi^{ZY} = 0$).

If immunity is incomplete, then $\psi^{ZY} > 0$, $\psi^{ZY} < 1$, complicating Pr(*UY*) and Pr(*ZZ*).

We make the following conclusions regarding our inferences with CMR data:

First, our estimated survival rates $\hat{S}^1$ for negative (*s* = 1) birds unidentifiably mixes survival for *X* (susceptible) and *Z* (immune birds). Because the latter may be higher than the former this overall might be thought of as an overestimate. Second, the 'infection rate' ψ_i^{12} confounds π the probability of being susceptible (i.e., being a member of the N_X, with ψ_i^{XY} the probability of infection for susceptible animals; because in general $\pi < 1$ this should result in $\hat{\Psi}^{1,2}$ underestimating ψ_i^{XY}). Third, the survival rate for infected birds, and probability of recovery, should still be unbiased, at least under the assumption that birds become immune once infected and that virulence of avian pox in symptomatic and asymptomatic infected birds is similar. With regard to the first point, our CJS estimates of apparent survival spans for several years in which there were no observed outbreaks of pox, and we were confident that these estimates well represent background survival for non–infected (*X*) birds.

Our estimate of low apparent infection ($\hat{\Psi}^{1,2} = 0.03$) contrasts with the high apparent prevalence rate (> 30%) found at the peak of the infection, by simple inspection of number of infected individuals from the total trapped birds. As noted above, this estimate undoubtedly underestimates true probability of infection (i.e., ψ_i^{XY}) to the degree that $\pi < 1$. However, we think that a more likely explanation for this low estimate is fact that recapture rates of symptomatic birds were consistently higher than those of asympotamic birds. Even accounting for the fact that some misclassification likely occurred, this suggests that infected birds are more prone to capture, possibility a consequence of greater dependency on an easy food source (McClure, 1989). We believe that these relatively higher recapture rates result in inflated estimates of disease prevalence rate, when such estimates rely on unadjusted capture frequencies.

To illustrate this point we constructed a simple model, to predict the actual (versus apparent) incidence of pox; for the purposes of this illustration we assume that no misclassification occurs, and that confine inferences to the observable states of asymptomatic (1) and symptomatic (2). In terms of the parameters we have estimated, the probability of being in state *s* for a bird in the population at time *i* depends on 3 elements: (1) the state the bird was in (infected or not) at time *i* – 1, (2) the probability of survival, dependent on state at time *i* – 1, and (3) the probability of moving from one state to the next, given the state at time *i* – 1.

For the state *s* = 1 (asymptomatic), this can be written as

$$\Pr(s_i = 1) = \phi_{i-1}^{11} \Pr(s_{i-1} = 1) + \phi_{i-1}^{12} \Pr(s_{i-1} = 2)$$

Likewise, the probability of *s* = *2* (symptomatic) is

$$\Pr(s_i = 2) = \phi_{i-1}^{12} \Pr(s_{i-1} = 1) + \phi_{i-1}^{22} \Pr(s_{i-1} = 2)$$

Both of these event probabilities involve state–specific survival from *i* – 1 to *i*, because $\phi_i^{rs} = S_i^r \psi_i^{rs}$. The proportion of birds at time *i* that are infected, is therefore

$$P(i) = \frac{\Pr(s_i = 2)}{\Pr(s_i = 1) + \Pr(s_i = 2)} =$$
$$= \frac{\phi_{i-1}^{12} \Pr(s_{i-1} = 1) + \phi_{i-1}^{22} \Pr(s_{i-1} = 2)}{\left(\phi_{i-1}^{11} + \phi_{i-1}^{12}\right) \Pr(s_{i-1} = 1) + \left(\phi_{i-1}^{22} + \phi_{i-1}^{21}\right) \Pr(s_{i-1} = 2)}$$

Because of the recursive nature of this expression, it has no solution without imposing initial conditions. If we assume an initial period before the outbreak of pox (*t* = 0), in our case May or earlier, then we can specify that $\Pr(s_0 = 2) = 0$, $\Pr(s_0 = 1) = 1$. These leads to an ability to recursively predict *P*(*i*) from the above expression. We have done so using our point estimates of survival and

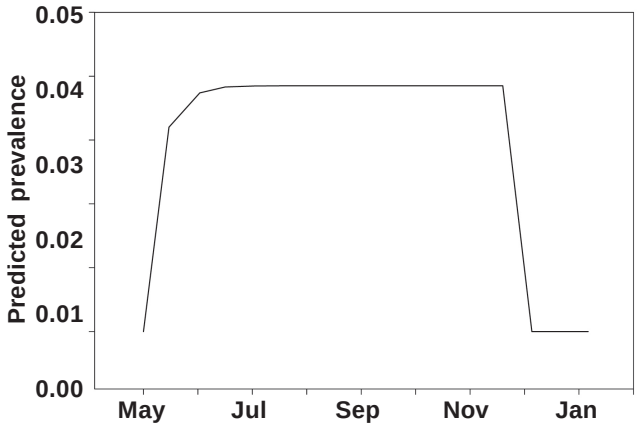

Fig. 3. Predicted incidence of pox based on estimates of state–specific survival and transition (table 3) and assuming 0 incidence before May and 0 probability of transition from uninfected to infected after November.

Fig. 3. Incidencia de la viruela predicha según las estimas de supervivencia y transición específicas del estado (tabla 3), asumiendo una incidencia 0 antes de mayo y una probabilidad 0 de transición del estado no infectado al infectado a partir de Noviembre.

transition parameters (table 3), using the additional assumption that of no further N to *S* transition following November (our last observed incidence of pox), that is $\psi_i^{12} = 0$ after this point. The predictions are displayed in figure 3, which shows a predicted asymptotic increase to a prevalence of about 4% by the end of the outbreak period, followed by a sharp decline (with the only remaining infestations being unrecovered, infected birds). This is in contrast to the apparent prevalence of pox (fig. 1), which does not properly take into account conditioning on survival, transition, and recapture rates, thus tending to overestimate prevalence. Our results indicate that caution is needed when estimating the prevalence of a disease in natural populations, either when relying on visual surveys at bird tables or on the capture of individuals (especially at baited traps). We stress the need to estimate recapture rates when estimating population dynamics parameters, a point that is repeatedly raised in all EURING conferences but that ecologists and evolutionary biologists are frequently reluctant to accept (Lebreton et al., 1993).

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