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LETTER

The 2002 European seal plague: epidemiology and population consequences

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Abstract

We present the first epidemiological data on the 2002 outbreak of phocine distemper virus (PDV) in European harbour seals (*Phoca vitulina*). The epizootic curve to date supports a mortality rate and probability of infection identical to that of the 1988 outbreak, which killed 58% of the population. Thus immunity is playing no significant role in the dynamics of the current outbreak. Because the timing of the outbreak is important in determining local mortality rates, we predict higher mortality rates on the European continent than in Great Britain or Ireland. A stochastic model is used to quantify how recurrent epizootics affect the long-term growth, fluctuation, and persistence of the population. Recurrent PDV epizootics with the observed frequency and severity would reduce the long-term stochastic growth rate of the harbour seal population by half, and significantly increase the risk of quasi-extinction.

Keywords

Epizootiology, extinction risk, Kattegat, mortality rates, phocine distemper virus, probability of infection, Skagerrak.

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INTRODUCTION

In 1988 an outbreak of phocine distemper ravaged populations of harbour seals (*Phoca vitulina*) in Europe. In April 1988 hundreds of dead harbour seals were observed on the Danish island of Anholt in central Kattegat. The 'seal plague' then spread north along the west coast of Sweden, south to the Netherlands, and west to England, Scotland, and Ireland, in what became the largest epizootic known in any marine mammal population (Dietz *et al.* 1989; Heide-Jørgensen & Härkönen 1992). At each colony, the outbreak lasted for 70–100 days and within 7 months all major European harbour seal colonies had been hit by the disease (Heide-Jørgensen *et al.* 1992). The death rate varied from 15% to 58% among regions. The mortality agent was a previously undescribed morbilli virus, the phocine distemper virus (PDV) (Cosby *et al.* 1988; Osterhaus & Vedder 1988; Bergman *et al.* 1990). Subsequent screenings revealed that, even before 1988, the PDV had been enzootic in arctic harp seals (*Phoca groenlandica*) which are little affected by the virus (Markussen & Have 1992). Migrating harp seals observed in Danish waters in 1987 and 1988 probably introduced PDV to the previously unexposed harbour seal population (Heide-Jørgensen *et al.* 1992).

In the 14 years since 1988 there has been no mortality due to PDV (Jensen *et al.* 2002). In May 2002, unusually high numbers of dead seals were reported from Danish colonies, and PDV infection was confirmed (Jensen *et al.* 2002). By mid September 2002, the death toll in the Kattegat and the Skagerrak exceeded 6000, and the disease had spread to the Baltic, the Wadden Sea and East Anglia (this study, Reineking 2002).

In this paper, our goal is to use information from the 1988 epizootic to predict the severity and dynamics of the ongoing 2002 outbreak. We are taking the unusual step of attempting to make these predictions while the epizootic is still in progress. This is risky, but provides a challenge to our analyses. Retrospective analyses will reveal how accurate our predictions are. We use the first detailed epizootic data to predict the eventual mortality rate and probability of infection, and to project the likely development of the 2002 epizootic. We use a stochastic model to analyse the long-term impact of recurrent PDV outbreaks on harbour seal population dynamics and persistence.

BIOLOGICAL DATA

Our population calculations are based on a time series of estimates of the size of the Kattegat–Skagerrak harbour seal

population between 1978 and 1998. These estimates are based on annual aerial surveys (Heide-Jørgensen & Härkönen 1988; Härkönen *et al.* 2002, present study). Analyses of infection and mortality are based on collections of dead seals from an ongoing intensive sampling programme along the west coast of Sweden. We compare the development of the present outbreak with information on the epidemiology from 1988 (Heide-Jørgensen & Härkönen 1992; Swinton *et al.* 1998).

INFECTION AND MORTALITY

Heide-Jørgensen & Härkönen (1992) analysed the 1988 epizootic using a discrete-time SIR model. They estimated the probability of infection (p) from the cumulative epizootic curve (i.e. the cumulative number of dead seals; see Fig. 1) and the observed mortality rate. We cannot estimate p for the 2002 outbreak until the outbreak is over. However, as a null hypothesis we suppose that all characteristics of the 2002 outbreak are the same as for the 1988 outbreak, but that the population differs in size. Heide-Jørgensen & Härkönen (1992) found that p scaled inversely with population size; this implies that the epizootic curve scales in direct proportion to population size. The ratio of the 2002 to the 1988 pre-outbreak population in southern Halland was 2294/1100 = 2.09. Thus the predicted epizootic curve in 2002 (see Fig. 1) is obtained by multiplying the 1988 curve by 2.09.

From Fig. 1 it is clear that our hypothesis predicts the 2002 epizootic closely. The correlation between observed and predicted numbers of deaths is 0.996. At the end of the epizootic, estimation of the epidemiological parameters will provide a more detailed comparison of the 1988 and 2002 dynamics, but so far the two are remarkably similar, suggesting that immunity plays no major role in the dynamics of the 2002 outbreak.

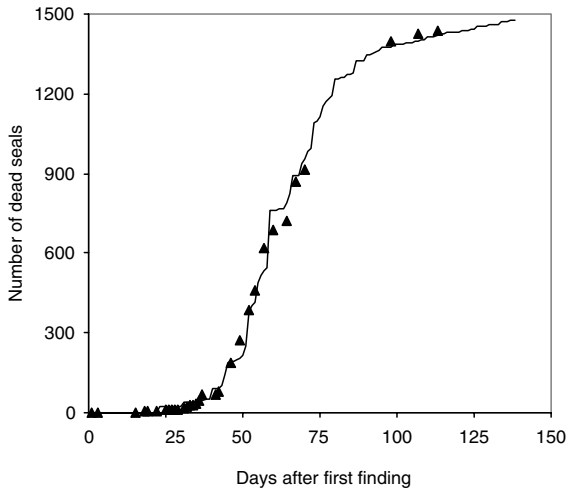

Figure 1 The epizootic curve (cumulative number of dead seals) in southern Halland (Sweden) in 2002 (triangles) follows the same pattern as the 1988 outbreak (solid line). Data from 1988 (Heide-Jørgensen & Härkönen 1992) scaled to present population size. The latest data point represents 11 September 2002.

EPIZOOTIC EFFECTS ON POPULATION DYNAMICS

The 1988 epizootic was a significant mortality event for European harbour seals, and we predict that the 2002 epizootic will be as well. Such stochastic events can have major impacts on long-term population growth and viability. Here we extend the stochastic Lewontin–Cohen model (Lewontin & Cohen 1969; see Caswell 2001; Section 14.3) to explore these impacts.

Stochastic population growth

The population grows as

$$N(t+1) = R(t)N(t) \tag{1}$$

where $N(t)$ is total population size. The growth rate $R(t)$ is a random variable determined by a stationary stochastic process reflecting both ordinary environmental variability in years without an epizootic, and the severity and frequency of epizootics.

The population will, with probability 1, eventually grow at the rate

$$\log \lambda_s = \lim_{t\to\infty} \frac{1}{t} \log \frac{N(t)}{N(0)}, \tag{2}$$

$$= E(\log\ R). \tag{3}$$

Asymptotically, $\log N(t)$ is normally distributed, with a mean growing as $t\log \lambda_s$ and a variance growing as $t\sigma^2$, where $\sigma^2 = V(\log R)$ is the variance in the growth rate.

Let $\log \lambda_s^{(n)}$ and $\sigma^{(n)2}$ be the growth rate and the variance in non-epizootic years. We obtained maximum likelihood estimates of these quantities by the method of Dennis *et al.* (1991), using the time series of annual population counts of the Kattegat–Skagerrak population, excluding the data from the 1988 outbreak. This assumes that the population is growing according to eqn 1 and that it has been doing so long enough to reach its stationary distribution. The resulting estimates

$$\log \lambda_s^{(n)} = 0.12 \tag{4}$$

$$\sigma^{(n)2} = 0.06 \tag{5}$$

indicate that the population was growing rapidly (an average of 12% per year) between 1978 and 1998, excluding the epizootic.

The population declined dramatically after the 1988 epizootic, but unfortunately no count data were available in 1987 to directly estimate the growth rate during the epizootic. Hence we estimated the growth rate R (1988) from

$$\log\frac{N(1988)}{N(1986)} = \log\hat{\lambda}_s(n) + \log\hat{R}(1988), \qquad (6)$$

We write $R(1988) = R^{(e)}$ where the superscript indicates an epizootic year. The probability s of surviving the epizootic is calculated from

$$\log R^{(e)} = \log\hat{\lambda}_s^{(n)} + \log s. \qquad (7)$$

Treating the four regions of the Kattegat–Skagerrak as independent subpopulations gives a mean $\log\hat{R}^{(e)} = -0.7477$ and $V(\log R^{(e)}) = 0.0259$ and mean $\log\hat{s} = -0.8675$; hence $\hat{s} = 0.42$ and the mortality due to the 1988 epizootic is estimated as 58%. We will use this value as a reference point for evaluating the long-term consequences of recurrent epizootics.

Epizootic effects on population growth

To model the effects of stochastically occurring epizootics, we define f as the long-run frequency of epizootics (because the process is scalar, autocorrelation has no effect). We neglect the effects of immunity (see further in discussion). The stochastic growth rate is now a function of f and s. By conditional probability

$$\log\lambda_s(f,s) = fE[\log R^{(e)}(t)] + (1-f)E[\log R^{(n)}(t)] \qquad (8)$$

where $R^{(e)}$ depends on s through eqn 7. The variance is also a function $\sigma^2(f,s)$ of f and s. We estimate it from the lognormality of $N(t)$. The expectation $E(N(t))$ grows at a rate $\log\mu$ given by $\log E(R)$. A consequence of the asymptotic lognormality of $N(t)$ is that $E(R) = \exp[E(\log R) + V(\log R)/2]$. Thus, by conditional probability

$$\log\mu(f,s) = \log\left[f\exp\left(\log\lambda_s^{(e)} + \frac{V(\log R^{(e)})}{2}\right) + (1-f)\exp\left(\log\lambda_s^{(n)} + \frac{V(\log R^{(n)})}{2}\right)\right]. \qquad (9)$$

Finally, we estimate the variance by

$$\sigma^2(f,s) = 2[\log\mu(f,s) - \log\lambda_s(f,s)]. \qquad (10)$$

Quasi-extinction probability

If $\log\lambda_s = 0$, eventual extinction is certain. If $\log\lambda_s > 0$ the population will eventually grow, but may decline temporarily. The probability that the population declines to a fraction θ of its initial size is called the quasi-extinction probability $P_q(\theta)$,

$$P_q(\theta) = \begin{cases} 1, & \log\lambda_s \le 0 \\ \exp\left(\frac{2\log\lambda_s\log\theta}{\sigma^2}\right) & \log\lambda_s > 0 \end{cases} \qquad (11)$$

(Lande & Orzack 1988; Dennis *et al.* 1991).

Population growth and epizootic frequency

We now have the necessary information to explore how the frequency and severity of epizootics affect population dynamics. Figures 2 and 3 show the stochastic growth rate $\log\lambda_s$, the variance σ^2, and the probability of quasi-extinction as functions of the frequency f and the severity $(1-s)$ of epizootics. As a point of reference, each figure notes the observed severity of the 1988 epizootic $(1-s = 0.58)$ and the frequency $f = 0.07$ corresponding to the interval of 14 years between the 1988 and 2002 epizootics.

At the 1988 mortality rate, there is a critical frequency $f = 0.14$ (an interval of 7.1 years) above which the population could not persist. At the observed interval of 14 years, there is a critical mortality of 0.81 above which the population could not persist. At the 1988 mortality rate and the 1988–2002 recurrence interval, the PDV epizootic reduces the stochastic growth rate $\log\lambda_s$ by half, from 0.12 to 0.06 (Fig. 2a) and it increases the variance σ^2 three-fold, from 0.06 to 0.16 (Fig. 2b). It increases the risk of a 50% population decline 10-fold, from 0.06 to 0.61 (Fig. 3). The risk of crashes to 10% of the current population size increases from negligible in the absence of epizootics ($P_q(0.1) = 1.0\times10^{-4}$) to a serious risk of 0.18 (Fig. 3). These are dramatic impacts on population performance.

DISCUSSION

Our analysis of the first epidemiological data from the 2002 PDV epizootic shows that the probability of infection is nearly identical to that in 1988, implying that immunity is of limited importance in 2002 (Fig. 1). Although it is generally believed that survivors of PDV develop life-long immunity (Kennedy 1990), we estimate that at most 7% of the current population are survivors of the 1988 epidemic, which would have a negligible impact on mortality. Thus, a full scale outbreak is at hand; of the 19 000 harbour seals in the Kattegat–Skagerrak, at least 17 500 are susceptible

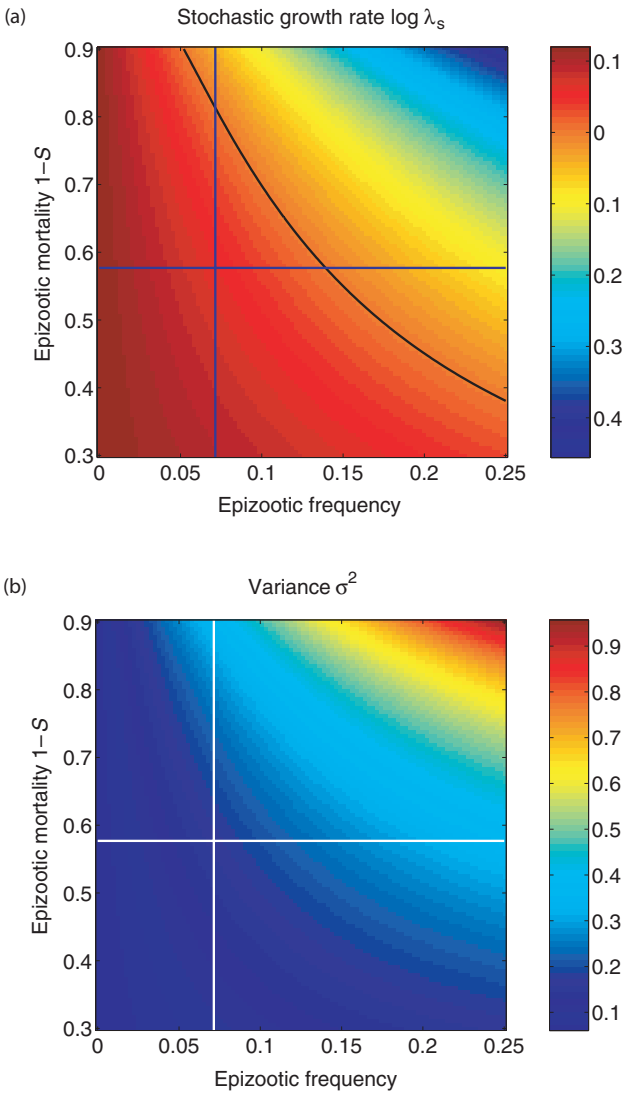

Figure 2 (a) The stochastic growth rate (logλ_s) of the harbour seal population as a function of epizootic frequency (*x*-axis) and the epizootic mortality rate (*y*-axis). The black curved line shows the critical limit (log $\lambda_s = 0$) below which extinction is certain. The present epizootic periodicity (14 years) and the 1988 mortality (58%) are shown for reference. Colours code values of log λ_s as shown in the colour bar at right. (b) The variance (σ^2) in the long term stochastic growth rate as a function of epizootic frequency (*x*-axis) and the epizootic mortality rate (*y*-axis). The present epizootic periodicity (14 years) and the 1988 mortality (58%) are shown for reference. Colours code values of σ^2 as shown in the colour bar at right.

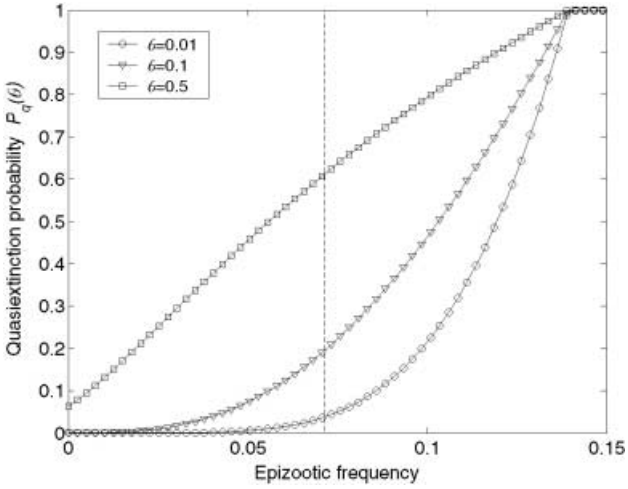

Figure 3 The probability of quasi-extinction ($Pq(\theta)$) of the harbour seal population as a function of epizootic frequency, assuming the 1988 mortality rate of 58%. Three different thresholds for quasi extinction are illustrated: squares $\theta = 0.5$; triangles $\theta = 0.1$; circles $\theta = 0.01$. The vertical line at $f = 0.07$ corresponds to the observed interval of 14 years.

(assuming all survivors from 1988 are immune), and we project an expected 10 000 deaths in 2002.

As expected, increasing epizootic frequency and/or increasing mortality rates have a profound effect on population growth rate and variance (Fig. 2a,b). A more detailed model would include reduced mortality at high epizootic frequencies due to immunity, which we neglect in this model. Thus Fig. 2 somewhat exaggerates the effect of the highest outbreak rates. However, at or above the observed epizootic interval, our simple model gives a good picture of the consequences of epizootics. (Even if all survivors of an epizootic are immune, few of them would be alive at the next outbreak.)

We evaluate the 'cost' of epizootics with the observed mortality rate (58%) in greater detail (Fig. 3). This is, to our knowledge, the first study where the consequences of epizootics have been evaluated in terms of quasi-extinction risk. At the observed mortality and epizootic frequency, the risk of a catastrophic decline to 1% of initial population size is very small. The 12% population growth rate between epizootics protects against such extreme declines. This is worth noting, as any factor that reduces population growth rate between epizootics, such as hunting, would increase quasi-extinction risk.

It is worrisome that the risk of a decline to 10% of initial population size is greatly increased (from 0.0001 to 0.18) at the observed frequency and severity of epizootics. Such a decrease is certainly undesirable, since the Kattegat–Skagerrak population is strongly spatially subdivided (Härkönen & Harding 2001) and the subpopulations could be at risk for demographic stochasticity and inbreeding.

One unanswered question after the 1988 PDV outbreak is why the mortality rates differed among regions. The Kattegat–Skagerrak and Wadden Sea populations

experienced mortalities of 50–60%, but mortality in England, Scotland, and Ireland was lower (10–20%; Dietz *et al*. 1989; Heide-Jørgensen *et al*. 1992, Thompson & Miller 1992). Some suggested that these differences were linked to pollution, because mortality rates were higher in regions with higher burdens of immuno-suppressive PCBs (De Swart 1995; De Koeijer *et al*. 1998), and because bone lesions, indicating PCB exposure (Bergman *et al*. 1992), were common in the Kattegat–Skagerrak (Mortensen *et al*. 1992), where mortality rates were high. Other authors have suggested a genetic contribution, following the discovery of pronounced genetic diversification among European harbour seal populations (Stanley *et al*. 1996, Goodman 1998). Theoretically, different frequencies of loci that determine susceptibility to pathogens in different local populations of seals could influence the mortality of local seal populations (Goodman 1998).

Mortality rates in 1988 were also influenced by seal behaviour (Härkönen *et al*. 1999; Harding 2000). The PDV is transferred to neighbours by coughing (De Koeijer *et al*. 1998) and the level of exposure to the virus depends on the rate of contact among individuals (Kennedy 1990). Colonies infected in late autumn, when seals spend less time on land, experienced low mortality rates. The Kattegat–Skagerrak colonies, which experienced mortalities of more than 50%, were infected in the summer (Dietz *et al*. 1989), when seals spend more time on land.

In 2002, several continental European populations were infected during August, when contact rates among individuals are peaking. Therefore, we predict high mortality rates (> 50%) in the Dutch Wadden Sea and the southern Baltic. However, since the 2002 epizootic began about 5 weeks later than that of 1988, we predict somewhat lower mortality rates in the German and Danish parts of the Wadden Sea, and substantially lower mortality rates in England, Scotland, and Ireland, since the disease will culminate in those populations in September and October, when contact rates among seals are low. Thus, we predict that the epizootic will subside by late November.

The models utilized here are purposely simple, making maximal use of the long-term count data in the Kattegat–Skagerrak region. Given that PDV is now a recurring risk for harbour seal populations, it will be important to develop more elaborate population models including age/sex structure, metapopulation structure and dispersal, seasonal behaviour, and epidemiological processes of contact, infection and immunity.

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