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Comparison of the 1988 and 2002 phocine distemper epizootics in British harbour seal *Phoca vitulina* populations

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ABSTRACT: In 1988 and 2002 dramatic and well-documented phocine distemper epizootics occurred in Europe. While their progression and impact were remarkably similar and consistent over much of Europe, mortality in the UK varied greatly between and within the 2 epizootics. We use antibody levels in blood samples to show that 51% (Bayesian 95% CI: 41 to 61%) of the individuals alive in 5 UK harbour seal populations at the end of the 1988 epizootic had been exposed to the virus, and that the equivalent figure after the 2002 outbreak was 22% (95% CI: 16 to 30%). Antibody prevalence was significantly higher in females than males after the 2002 epizootic. Combining these estimates with information on reductions in the numbers of animals observed hauled out during surveys of the Wash, Moray Firth, and Orkney populations and a simple epidemiological model, suggests that the differences between the 2 epizootics were primarily due to a 27% (95% CI: 8 to 43%) fall in R_0, the basic reproductive rate of the virus. The large geographic variation in population effects observed within the UK during each epizootic appears to have been mainly due to differences in case mortality, with R_0 being remarkably similar in all the populations investigated.

KEY WORDS: Epidemic · Epidemiology · Mathematical model · Pinniped

INTRODUCTION

The European phocine distemper epizootics of 1988 and 2002 were sudden, dramatic, and apparently very similar (Hall et al. 2006, Härkönen et al. 2006). Widespread public and scientific interest led to detailed study and provided an opportunity to explore viral transmission in a free-living population. On both occasions unusual numbers of abortions and mortalities were first noticed on the island of Anholt in Denmark in the spring, and the virus spread rapidly around northern Europe. It has been estimated that in 1988 > 50% of the harbour seals *Phoca vitulina* in most areas died, along with smaller numbers of grey seals *Halichoerus grypus* (Dietz et al. 1989, Heide-Jørgensen & Härkönen 1992). In 2002 the outbreak started one month later than in 1988 and progressed less smoothly around the coast (Harding et al. 2002, Jensen et al. 2002). The most obvious exception to the general pattern occurred in the UK, where mortality was much lower and varied more within and between years than elsewhere in Europe.

In both 1988 and 2002 the impact of the disease seems to have decreased as it moved north around the UK. In 1988 the population in the Wash, a large estuary on the east coast of England, suffered around 50% mortality (Thompson et al. 2005), close to the levels observed in the rest of Europe. However, mortality in the Moray Firth, on the Scottish east coast, was much lower (Thompson & Miller 1992), and in Orkney no change in population size was detected (Harwood 1990, Thompson et al. 2001). In 2002 mortality in the

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Wash was around 22% (Thompson et al. 2005), and very few dead seals were reported from Scotland (Härkönen et al. 2006). While different proportions of carcasses may have washed ashore in different places, and their recording largely depended on reports from the public rather than systematic surveying, it seems unlikely that large numbers of animals could have died unnoticed everywhere in Scotland. There are 2 potential explanations for these variations in mortality. Either different proportions of the various populations were exposed to the virus, or the case mortality rate (the proportion of infected individuals killed by the disease) varied between populations and outbreaks (Grenfell et al. 1992).

Using the course of the epizootic to choose between these explanations would require the recovery of large numbers of carcasses, the availability of detailed information on the patterns of mixing within each area, and strong assumptions about the duration and intensity of infectivity of individuals. Instead, we used the prevalence of antibodies in blood samples, combined with estimates of mortality in the populations, to identify differences in the impact of the disease.

MATERIALS AND METHODS

Blood samples were taken from animals, handled under UK Home Office licence, after each epizootic had concluded. After the first outbreak, samples were collected from surviving seals between November 1988 and February 1989, and after the second outbreak between March and June 2003. The straightline, nose-to-tail tip length of each animal was recorded. Pups (<0.9 m standard length) were excluded from the analysis as they could have maternally derived antibodies. In 2003 each animal was also weighed with a spring balance. Serum was separated and frozen before being diluted and used in a Virus Neutralisation Test (Biobest Laboratories) with canine distemper virus following the method of Appel & Robson (1973), as described in Thompson et al. (1992). Following these authors, a titre of 1:90 was taken as the threshold indicating seropositivity.

A compartmental model (Kermack & McKendrick 1927, Grenfell et al. 1992) was used to represent viral transmission within each local area. These were treated as containing large, perfectly mixed closed populations. Individuals were classified as susceptible (s), infected (i), or recovered and permanently immune to the virus (r). The introduction of the virus was assumed to initiate a deterministic simple epizootic, whose course satisfies:

$$ds/dt = -\beta si \quad (1)$$

$$di/dt = \beta si - \alpha i \quad (2)$$

$$dr/dt = (1-\delta)\alpha i \quad (3)$$

Here β controls the transmission of the virus between individuals, α the rate at which infected individuals recover or die, and δ is the case mortality. These equations assume that the area occupied by the population remains constant, and therefore that the density of susceptible individuals falls over the course of the epizootic. The form of the equations implies that the duration and pattern of spread of the virus is independent of the absolute size of these populations. The proportion, p, of an initially naïve population that will be infected during a disease outbreak, then satisfies the equation:

$$R_0 p = -\ln(1-p) \quad (4)$$

Where R_0, the basic reproductive rate of the virus within the population, equals $\beta s_1/\alpha$ for an initial population size s_1, and is the number of animals an infected individual would directly infect within such a naïve population. R_0 must be >1 for a significant disease outbreak to occur. Life-long immunity seems usual after recovery from infection by morbilliviruses, though antibody concentrations decline over time (Thompson et al. 2002), so individuals were assumed not to reacquire or transmit the virus on re-exposure, but just mount an immune response. The presence of immune individuals reduces the rate of viral transmission by effectively diluting R_0 in proportion to their numbers. This means that after one epizootic has concluded, another cannot occur until birth, natural death, and immigration/emigration have increased the proportion of susceptible individuals in the population beyond the inverse of the original viral reproductive rate (Grenfell & Dobson 1995, Longergan & Harwood 2003).

The antibody prevalence, q, measured at the end of an epidemic, differs from the proportion of the initial population infected, p, because some animals die as a result of the disease. If the population was s_1 before the outbreak and s_2 afterwards, these values are related by:

$$(1-p)s_1 = (1-q)s_2 \quad (5)$$

and the proportion of infected individuals that die, δ, is given by:

$$\delta = (s_1 - s_2)/(ps_1) \quad (6)$$

A Bayesian approach was taken to estimate antibody prevalence within the local populations. An uninformative prior was used, with all proportions of seropositive animals initially considered equally likely. The probability of getting the observed results was then calculated and, using Bayes theorem, the most likely proportion identified, along with the range of proportions containing the central 95% of the likelihood. The statistical significance of differences between estimated parameter values was assessed by simulation.

In each case, 1000 draws were made from each of the relevant distributions. The reported probability values are the proportion of pairs for which the sign of the difference was the reverse of that between the point estimates. These, therefore, are estimates of the likelihood that the true difference between the values had the opposite sign to the difference in the estimates, given the observed data.

RESULTS

Table 1 shows the number of seropositive individuals and the estimated percentages of the populations with antibodies to phocine distemper virus (PDV) in each local population. It appears that approximately half the animals alive after the 1988 outbreak had been exposed to the virus. After the second outbreak this proportion was around one-quarter. One exception was the west coast of Scotland, where no antibodies were detected in 2003. However, these particular samples were all collected within a small area around the islands of Islay and Jura, and so may not be representative of the wider region. None of the differences (except those involving the west coast of Scotland), neither between regions within epizootics nor between epizootics within regions, was statistically significant. However, when all regions were combined, the difference between epizootics was significant ($p < 0.001$). Excluding the west coast results from the aggregated analysis increased the 2002 prevalence estimates to 27%, but did not change these conclusions.

Imperfect mixing, or differing immune responses, might be expected to result in different antibody prevalences in different sections of the populations. Both Harwood et al. (1989) and Thompson et al. (1992) reported significantly smaller proportions of seropositive juveniles than adults after the 1988 epizootic. We did not have accurate ages for the individuals in our sample, so we were not able to test this directly. Instead, as younger animals are generally smaller and lighter, we used weight as a proxy for age and split the 2002 sample at a threshold chosen to lie below the weights of the majority of individuals but leave sufficient smaller animals for comparison. The threshold was set at 55 kg for females and 65 kg for males. Antibody prevalences in the samples from the 2002 epizootic were not inconsistent with uniformity across the size classes. Antibody prevalence was, however, significantly lower in males than females ($p = 0.04$) (Table 2).

Estimates of mortality in the Wash population found by Thompson et al. (2005) were combined with our estimates of post-epizootic antibody prevalence to investigate the percentage of the pre-epizootic populations that was exposed to the virus, the case mortality rate, and R_0 for PDV in each outbreak within this area (Table 3). The results for 2002 were lower than the equivalent ones for 1988, although the change in the percentage infected ($p < 0.005$) was much clearer than that in case mortality ($p < 0.05$).

Equivalent estimates were also made for the Moray Firth (Table 3). The means (±SE) of repeated counts carried out there during the pupping season (see Thompson & Miller 1992 and Thompson et al. 1997 for details of survey methodology) in 1988 and 1989 were 982 (±28) and 740 (±39), respectively; the equivalent values in 2002 and 2003 were 683 (±37) and 634 (±36), respectively (P. Thompson unpubl. data). These suggest declines of 25% (95% CI: 16 to 33%) and 7% (95% CI: −8 to +20%) over the 2 outbreaks, with the second decline significantly smaller than the first ($p < 0.02$). The estimated population change between 2002 and 2003, which implies a 17% chance that the population actually increased over this period, may

Table 1. Prevalence of distemper antibodies in harbour seals *Phoca vitulina* after the 1988 and 2002 phocine distemper virus (PDV) epizootics. Positive: number with titres of at least 90 and the total numbers sampled each time. Antibody prevalence: most likely value for the % of seropositive individuals in each population and its Bayesian 95% CI; nd: no data

	Post-1988		Post-2002	
	Positive	Antibody prevalence (%)	Positive	Antibody prevalence (%)
Wash	8/17	47 (26–69)	6/20	30 (15–52)
Scotland				
Tay	5/7	71 (35–91)	8/33	24 (13–41)
Moray Firth	19/41[a]	46 (32–61)	8/26	31 (16–50)
Orkney	11/15	73 (48–89)	5/20	25 (11–47)
West coast	2/8	25 (7–60)	0/22	0 (0–15)
All Scottish	37/71	52 (41–63)	21/101	21 (14–30)
All Great Britain	45/88	51 (41–61)	27/121	22 (16–30)
Northern Ireland	8/12	67 (39–86)	nd	nd

[a]Source: Thompson et al. (1992)

Table 2. Antibody prevalence in different size and sex classes of harbour seal *Phoca vitulina*. Data collected after the 2002 outbreak from all regions have been combined. Females <55 kg and males <65 kg were classified as small. For explanation of terms see Table 1

	Males		Females		Total	
	Positive	Antibody prevalence (%)	Positive	Antibody prevalence (%)	Positive	Antibody prevalence (%)
Small	1/12	8 (2–36)	5/13	39 (18–65)	6/25	24 (12–44)
Large	7/46	15 (8–28)	14/50	28 (17–42)	21/96	22 (15–31)
Total	8/58	14 (7–25)	19/63	30 (20–42)	27/121	22 (16–30)

Table 3. Mean and Bayesian 95% CI for harbour seal *Phoca vitulina* population changes, and the case mortality, % of pre-epizootic populations infected, and R_0 (the basic reproductive rates) of the virus within the populations, implied by combining these values with the post-epizootic antibody prevalences in Table 1

	1988				2002			
	Population decrease (%)	Case mortality (%)	Infected (%)	R_0	Population decrease (%)	Case mortality (%)	Infected (%)	R_0
Wash	52[a] (44–59)	70 (58–82)	75 (63–86)	1.8 (1.6–2.3)	22[a] (9–33)	47 (24–70)	47 (30–64)	1.3 (1.2–1.6)
Moray Firth	25 (16–33)	41 (27–55)	60 (47–71)	1.5 (1.4–1.8)	7 (0–20)	19 (0–49)	37 (20–56)	1.3 (1.2–1.5)
Orkney	0[b]	0	71 (48–89)	1.7 (1.4–2.5)	0[b]	0	27 (11–47)	1.2 (1.1–1.4)
	5[b]	7 (6–10)	73 (51–90)	1.8 (1.4–2.6)	5[b]	17 (10–32)	30 (16–50)	1.2 (1.1–1.4)
	15[b]	20 (16–27)	76 (56–91)	1.9 (1.5–2.6)	15[b]	40 (27–61)	38 (25–55)	1.3 (1.1–1.5)

[a]Source: Thompson et al. (2005); [b]3 possible scenarios for the population impact of the epizootics

overstate the effects of the disease, as the population has been declining since 1993 (Lonergan et al. 2007, Thompson et al. 2007).

In 1988 both the case mortality and R_0 were lower in the Moray Firth than the Wash ($p < 0.01$; $p < 0.05$), but they were indistinguishable in 2002 ($p > 0.2$; $p > 0.05$). The decrease in R_0 between years in the Moray Firth was statistically significant, but the reduction in case mortality is not ($p < 0.05$; $p > 0.10$).

For Orkney, where carcasses were reported during both epizootics but no change in seal numbers was detected, we investigated 3 scenarios in which the disease killed 0, 5, or 15% of the population. These levels of mortality result in estimates of case mortality and exposure to the virus that are consistent with those observed in the other regions (Table 3). Unless the actual proportion of the population that died in Orkney in 2002 was substantially higher than in 1988, it would seem clear that a much lower proportion of the population was exposed to the virus in the second outbreak.

Overall, these results indicate that, within each epizootic, R_0 was similar across all 3 regions, but also that it was substantially lower in 2002 than in 1988.

DISCUSSION

The relatively small sample sizes in this study and the assumptions required for the analysis limit both the precision of the results and the weight that can be put on small differences within them. However, the overall pattern is clear: after the 1988 phocine distemper outbreak around half the individuals within all the British harbour seal populations carried antibodies to PDV; the equivalent figure after 2002 was one-quarter. Similar proportions of all the populations appear to have been exposed to the virus, suggesting that the conditions for its transmission were comparable everywhere.

Antibodies to PDV, indicating recent exposure to the virus, were detected in grey seals on the west coast of Scotland in 2002–2003 (Pomeroy et al. 2005), suggesting that the virus had recently spread within the region. Their absence in all the west coast harbour seals examined here may indicate that the virus did not reach this population, or may just be due to chance and the limited sample size.

The age distribution of carcasses recovered during the 1988 epizootic suggests that pre-epizootic annual

adult harbour seal survival in Europe was around 90% (Härkönen & Heide-Jørgensen 1990). With the 6% annual population growth rate observed in the Wash between 1989 and 2002 (Thompson et al. 2005), this suggests that approximately 10% of the individuals alive at the start of the 2002 outbreak had been born before 1988. Three-quarters of these would have been exposed to the virus then, so only 8% of the 2002 population might be expected to have residual antibodies from the 1988 epizootic. Thus, the 27% difference (95% CI: 8 to 43%) between the estimated R_0 values in 1988 and 2002 cannot be explained by the dilution effect of immune survivors. Some of the change could be due to seasonal variation in the proportion of time individuals spend hauled out (Thompson et al. 2005), or other behavioural changes between the years. In Scotland, where it seems unlikely that harbour seal numbers increased between 1988 and 2002 (Lonergan et al. 2007), up to 20% of the populations in 2002 may have been already immune to the virus, halving the discrepancy needing explanation.

Antibody prevalence was significantly higher in females than males after the 2002 outbreak. This could be due to either differences in the exposure of the sexes to the virus or physiological differences in their susceptibility to it. Mortality among males was higher than in females in 1988 (Hall et al. 1992), and both epizootics reached a peak in the UK around late August (Härkönen et al. 2006), when British harbour seals moult. Harbour seals spend a larger proportion of the time out of the water during the moult than at other times of year. Mature females moult first, followed by immature animals, and, lastly, mature males (Thompson & Rothery 1987). The intensity as well as probability of individuals' exposure to the virus may therefore have varied between groups within the population.

Our analysis also suggests that the greatest difference between the outbreaks in the Wash and those in continental Europe, where case mortality was estimated to be 65% among the 95% of individuals exposed to the virus (Heide-Jørgensen & Härkönen 1992), was in the transmission of the virus. It seems likely, given the similarity in viral transmission characteristics throughout the UK and the apparent discrepancies in population effects, that case mortality from PDV is highly variable across the British populations. It is difficult to ascribe this to differences in the timing of the local outbreaks, given the uniformity of R_0 within each epizootic. However, we cannot rule out the possibility that seasonal differences in behaviour resulted in more intense, rather than more frequent, contact between infectives and susceptibles. If so, this may result in differences in viral dose that could account for the observed differences in the case fatality rate. We do not have sufficient data on how interactions between individuals, particularly at haulouts, vary by season. However, our observation (corroborated by De Koeijer et al. [1998] for the Wadden Sea population) is that the nearest-neighbour distance in harbour seal haulouts is relatively constant and independent of haulout size, provided space is not a limiting factor. The higher mortality rates observed in the faster-growing populations, where animals might have been expected to be in better condition, is consistent with the suggestion that differences in, for example, genetic makeup or pollutant burdens may have had an important effect (Heide-Jørgensen et al. 1992). Hall et al. (2006) provide a detailed discussion of the possible role of these individual factors and the potential mechanisms involved. The observed variability will also complicate attempts to predict the potential effects of future epizootics (Harding et al. 2002, Lonergan & Harwood 2003).

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