

Spatial and Temporal Development



Dead harbour seal
(Photo H.H. Dietz)

The Phocine Distemper Virus Outbreak of 2002 amongst Harbour Seals in the North Sea and Baltic Sea: Spatial and Temporal Development, and Predicted Population Consequences

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Introduction

An unusually high mortality amongst harbour seals started on Anholt in April/May 2002. This appeared to be the start of a virus epizootic caused by a phocine distemper virus (PDV), like in 1988 (Jensen et al. 2002). The epizootic spread in summer northwards and leaped to the western part of the Wadden Sea in mid-June, from where it spread eastwards throughout the Wadden Sea (Reineking 2002a). The first victims in the UK were found in mid-August in the Wash (SMRU 2002).

In the North Sea and Baltic Sea together at least 22,500 seals were found dead (Reineking 2002b). It is the intention of this paper to discuss the spreading of the disease in space and time, for the different areas in the North Sea, with additional reference to the duration of the epizootic. Furthermore, differences in mortality pattern, duration of the epizootic, and relative mortality will be specifically elaborated for the Kattegat/Skagerrak area and the entire Wadden Sea (Denmark, Schleswig-Holstein, Lower Saxony and the Neth-

erlands). Finally, the possible population consequences of this renewed and perhaps to be expected future recurrent PDV-outbreaks will be addressed by modelling how harbour seal population growth in the Wadden Sea will be affected under different scenarios for mortality, probability of infection and frequency of occurrence. Emanating intriguing management questions and particular implications for future persistence of the population and related anticipatory conservation management will be addressed.

Spatial and Temporal Development of the Epizootic

The first unusual mortality amongst harbour seals was reported from Anholt (Danish Kattegat) on May 4th 2003. An overview of the spatial and temporal spreading is given in Figure 1. Details on the chronology of the first occurrence of unusual mortality have been given by Reineking (2002a).



Figure 1: Spatial and temporal spreading of the PDV epizootic amongst harbour seals in the Baltic and North Sea in 2002.

*In the context of this paper it suffices to present the general pattern (in monthly intervals) and focus on the geographical spreading.

The disease spread rather quickly from the Danish Kattegat to the north. Within about a month, seal deaths were reported from nearly all sites in the Kattegat/Skagerrak area and the Oslofjord. Rather intriguing is the isolated observation of the first case (with confirmed PDV) in the Wadden Sea, notably in the western part of the Dutch Wadden Sea (Reineking 2002a). Such an isolated case is remarkable given our knowledge about dispersal patterns of harbour seals in the Wadden Sea (e.g. Nørgaard 1996) and assuming infected harbour seals are the vectors for the disease. An explanation may be provided by considering that another carrier has brought the disease to the Dutch Wadden Sea, either of anthropogenic or marine animal (e.g. grey seal) origin. Irrespective the origin, this pattern is in contrast with the pattern found in 1988, where the epizootic spread along the northern coast of Denmark into the Danish Wadden Sea. From the Wadden Sea onwards it arrived in different areas in the Wadden Sea, and the general pattern was rather a north-west and east-west spreading throughout this area. Striking is the observation that the population in the Limfjord was affected only on September 16th 2002. This indicates that this seal stock has little exchange with the Kattegat/Skagerrak colonies, at least not in the summer. The subsequent spreading of the disease after the Wadden Sea to the Wash and later on to Scotland, Wales, N-Ireland

and the Republic of Ireland, as well as from the Wadden Sea to the Delta area (SW-Netherlands) and further on to the Belgium and French coastal waters, is rather similar to what was observed during the 1988 epizootic (Dietz et al. 1989).

Temporal and Spatial Pattern in Registered Seals Found Dead

It is obvious that the number of reported dead seals is strongly influenced by the timely accuracy and consistency of the reporting system over time, and moreover by environmental conditions. In the latter category is particular the wind, direction as well as force, of direct influence on the drift of the moribund and dead seals. The raw reporting data are therefore rather variable and to account for the largely unknown influence of the afore mentioned factors, the data have been transformed into 3-day moving averages. The moving averages of the daily number of seals found dead, expressed as a percentage of the estimated total population, are given for the Dutch, Lower Saxon, Schleswig-Holstein and Danish part of the Wadden Sea in Figure 2.

The influence of wind direction and force is e.g. demonstrated by the unlikely drop in animals found dead in the Netherlands, around September 1st, followed by an increase in the 3rd week of September. In the neighbouring area Lower Saxony, a similar pattern is seen though less dramatic. In addition the daily numbers of seals found

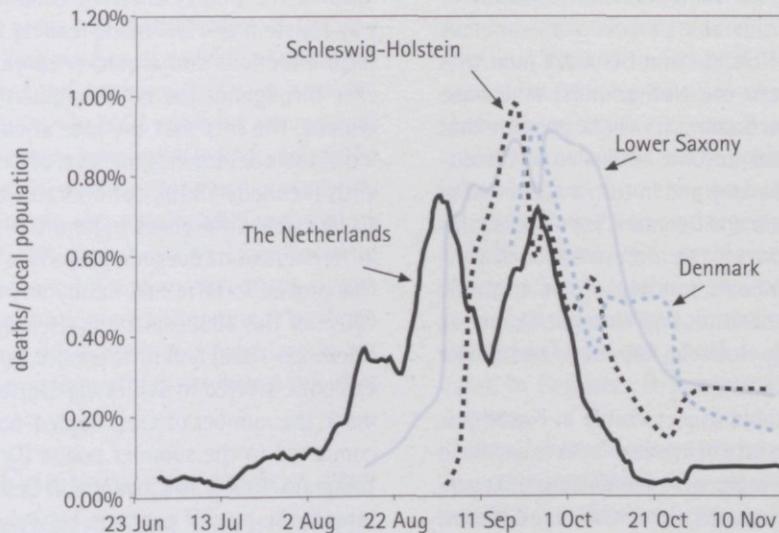


Figure 2: Moving averages of the number of seals found dead in the Wadden Sea, expressed per local population size, for the Netherlands, Lower Saxony, Schleswig-Holstein and Denmark, from June until November 2002.

dead stayed high for a longer period compared to the other regions in the Wadden Sea. This could be explained by the prevailing, strong south-easterly winds during the first two weeks of September. These might have blown a large part of the drifting dead and moribund seals offshore. Changing winds (north-westerly) in the second half of September would blow the corpses back. In practice this means that dead seals were more likely to drift away from the Dutch/Lower Saxon Wadden Sea into the North Sea early in September, and that later in September the situation returned to former conditions. It is therefore postulated that a larger proportion of the animals found dead late September early October, actually died in the first half of September and drifted into the North Sea. Lower Saxony is the region that probably received dead seals from both the Netherlands and from Schleswig-Holstein and Denmark. This is supported by the finding that after such a period of offshore winds, the animals found dead were usually in a worse condition (longer time to death) than those found in earlier periods (M. Stede, pers. comm.), indicating they had drifted some time at sea before arriving at the coast and being collected.

The other conclusion drawn from the data in Figure 2, is that the maximum percentage of daily deaths per total population, never exceeded 1%, and was rather equal amongst the four regions. This may indicate that no real outburst or strong pulses in mortality did occur.

Duration of the Epizootic and Severity of the Death Toll

Besides the question about the duration of the epizootic, it is in view of the future of the population, essential to assess how many animals in the population became victim of the epizootic. Both aspects, duration of the epizootic and extent of mortality, have been investigated by expressing the numbers of animals found dead over time, as a percentage of the local population size. The patterns have been synchronised by taking the start of the epizootic in each region as day zero. To address these questions of time span and severity on a more European scale, a comparison with the findings in the Kattegat/Skagerrak area have been incorporated. By the time these analyses were carried out, the epizootic still continued in England, Scotland and Ireland and virtually only stopped around mid-December (SMRU 2003). Those data could therefore not be included here. The transformed data on seals found dead (percentage of the population) are given in Figure 3 for the Wadden Sea and the Kattegat/Skagerrak area. It can be deduced that the duration of the epizootic differs between the Kattegat/Skagerrak plus Netherlands and Lower Saxony, Schleswig-Holstein and Denmark. For the entire Wadden Sea, the Netherlands and Kattegat/Skagerrak it lasted about 130 days, for Schleswig-Holstein and Denmark around 70 days, whereas Lower Saxony lies

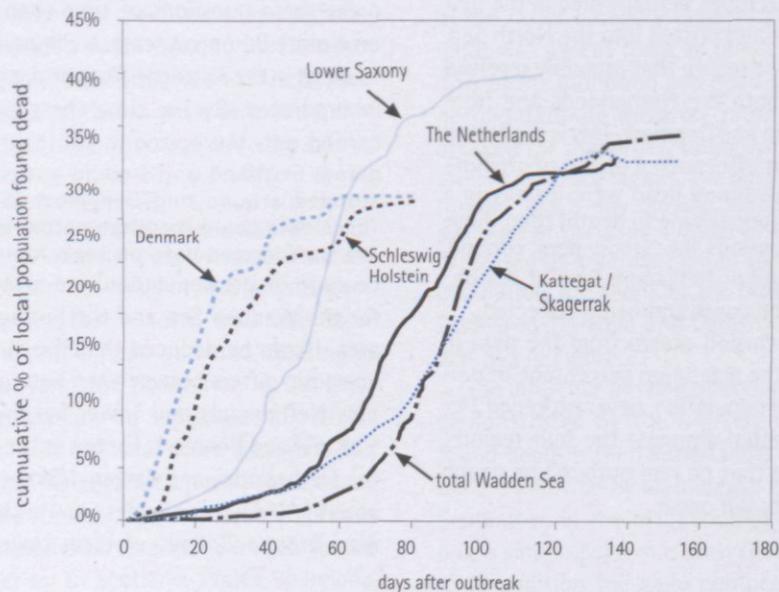
in between with around 105 days. Under the assumption that in the third and fourth quartile of September a considerable portion of the animals from Schleswig-Holstein and Denmark (and to a lesser degree from the Netherlands) may have ended up in Lower Saxony, it may be possible that this event has led to some extension of the duration in Lower Saxony and in turn a reduction in Schleswig-Holstein and Denmark. Even in case this supposition is correct, the afore mentioned considerable difference in duration of the epizootic between the Netherlands and Kattegat/Skagerrak versus Schleswig-Holstein, Denmark and Lower Saxony would still exist.

The other notable aspect visible in Figure 3 is the differences in rate of increase in % found dead in the respective regions. Again Kattegat/Skagerrak and the Netherlands are similar, but different from both Schleswig-Holstein and Denmark, being similar as well, and Lower Saxony is again somewhat in between. These differences could be explained by the time the disease arrived and the numbers of vectors carrying on the disease by infecting conspecifics. As elaborated in an earlier section of this paper, the first observation of dead seals and victim of the PDV, in the Wadden Sea was in the Netherlands and from there it spread to Lower Saxony, and arrived rather late in Schleswig-Holstein and Denmark. The infection in the Netherlands can actually be considered as a point source. By the time the disease arrived in Lower Saxony, there were more vectors (infected seals) that entered different colonies in Lower Saxony, creating different focal infections thereby enhancing the spread (speed) of infection in that region.

Subsequently, an even larger source of vectors reached the (more) different colonies in Schleswig-Holstein and Denmark, leading to even more focal infections and a quicker spread of the disease throughout the entire populations in those regions. The fact that the rate of contact among individuals determines the level of exposure to the virus (Kennedy 1990), corroborates this hypothesis. Possible differences in herd size between the different regions does not play a role here because this proved to have had hardly any effect on the cause of the 1988-epizootic (Heide-Jørgensen & Härkönen 1992) and furthermore, by the time the epizootic started in Schleswig-Holstein and Denmark, the number of seals hauled-out were lower compared to the summer period (Drescher 1979, Tougaard 1990). This change in behaviour counteracts the rate of contacts between individuals. The number of animals found dead and expressed per total population varies from just below 30% in Schleswig-Holstein and Denmark, to just 40% in Lower Saxony. In the other areas (Kattegat/Skagerrak, the Netherlands as well as the entire Wadden Sea) this amounted to approximately 35%. As mentioned before, the percentage for Lower Saxony may be artificially increased respectively reduced for Schleswig-Holstein and Denmark. It is therefore justified to conclude that in most areas the numbers of seals found dead and reported, amounted to around one-third of the respective populations increase.

It is complicated to assess the actual number of seals that died and hence what portion of the population fell victim to the PDV-epizootic. Modelling (Harding et al. 2002) and surveys late au-

Figure 3: Cumulative number of seals found dead, expressed as a percentage of the local population, since the start of the epizootic in the Netherlands, Lower Saxony, Schleswig-Holstein, Denmark, the entire Wadden Sea, and the Kattegat/Skagerrak area in 2002.



tumn in Kattegat/Skagerrak area (T. Härkönen pers. comm.) revealed that around 53% of the population in those areas had died because of the disease. For the Wadden Sea, our preliminary modelling shows that this percentage ranges from 48-52%. Aerial surveys in 2003 will bring the required data to enable more conclusive remarks about the actual impact. Nevertheless it can be prudently concluded that the epizootic in 2002 was slightly less severe (5-7% probably lower) compared to the 1988 epizootic. Assuming that the survivors of the 1988 epizootic were all immune, it has been calculated that this difference maybe largely attributed to the number of those survivors still alive in 2002.

Consequences for the Wadden Sea Population on Longer Term

To assess the longer term consequences of the 2002 epizootic on the harbour seal populations in the Wadden Sea, we modelled the population development for the next 35 years. Two scenarios are distinguished: a scenario where no recurrent PDV-epizootic would occur and a scenario where recurrent epizootics with different cycle length would occur. These calculations on population development are not meant to predict exactly the actual development, they are rather used to demonstrate the magnitude of effect on the populations when different scenarios would be operative.

The modelling is based on the population parameters obtained over the past years since the last epizootic (Reijnders et al. 1997, Reijnders & Brasseur 2003, Reijnders et al. 2003) and it is assumed that the combination of parameters found in the period 1990-2002 are also valid for the period of our calculations. The epidemiological modelling is based on the method used by Grenfell et al. (1992) and Heide-Jørgensen & Härkönen (1992), whereby the specific parameters for the Wadden Sea population (population size, mortality due to the epizootic, intrinsic growth rate, *per capita* birth rate, *per capita* death rate) as elaborated in Reijnders & Brasseur (2003) have been applied.

Population development has been calculated for scenarios where the epizootic cycle length would be respectively two, seven and 14 years, and a scenario where no epizootic would occur. The results are shown in Figure 4a-d. The cycle of two years has been chosen because it was calculated that only after this point of time a new epizootic could theoretically happen. The period of 14 years being a representation of the period between the last two epizootics, and seven years is the mid-value thereof.

Figure 4a shows a rapid recovery of the population to its pre-epizootic level of around 27,000 seals and a level of approximately 70,000 would be reached in 35 years. Under the two-year cycle (Fig. 4b), the epizootic would finally damp out and the population will slightly decrease and amount to approximately 15,000 animals in 2038. The 7-

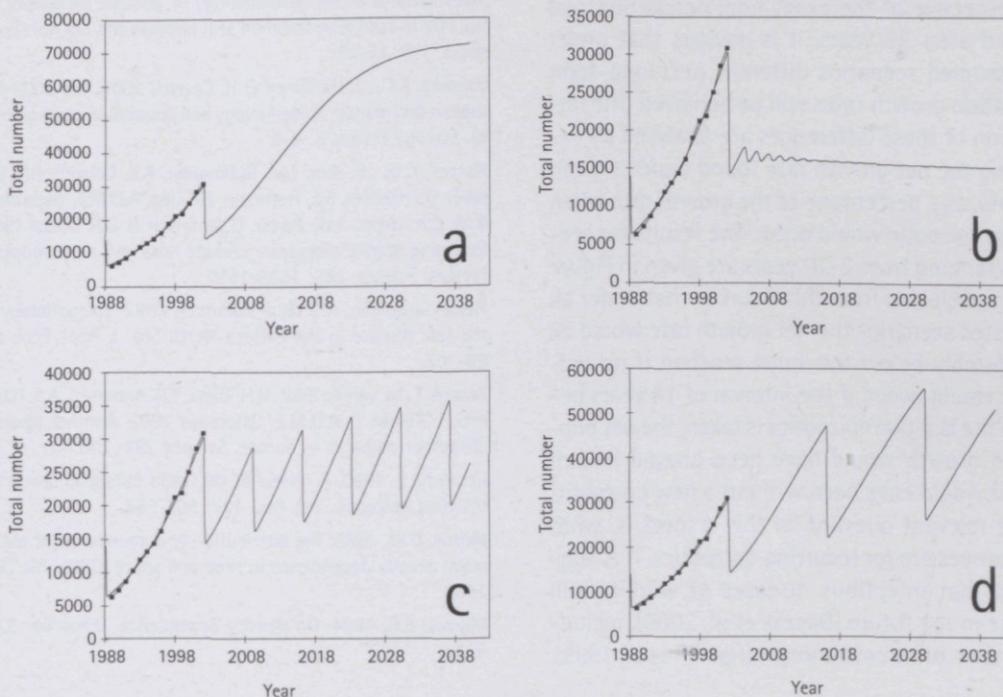


Figure 4: Modelled developments of the population of harbour seals in the entire Wadden Sea: from 1989-2001 based on actual counts (black squares), the epizootic in 2002, and from 2002 onwards under different scenarios for recurrency of PDV-epizootics. Fig.4a: with no epizootic after 2002, fig. 4b-d: with an epizootic cycle length of respectively 2, 7 and 14 years.

year cycle (Fig. 4c) would result in an overall slight increase and the 14-year cycle (Fig. 4d) would result in a stronger overall increase.

It is emphasised that all the shown population developments, with the exception of the two-year cycle, can only be realised if our estimates about density dependent regulation are correct. These are based on the estimated disease free equilibrium of the population (see Grenfell et al. 1992), on the growth rates observed between 1990 and 2001 (Reijnders & Brasseur 2003), and subject to the assumptions that the *Allee Effect* (Emlen 1984) will hold here as well. Taking into account the scientific debate (Murray 1994, Sinclair & Pech 1994, Morris 1996) on density dependence in e.g. time and space, related to environmental stochasticity, compensatory processes, we still continue further modelling to obtain the range of confidence intervals around the estimated carrying capacity given the variance in the data used under the aforementioned assumptions.

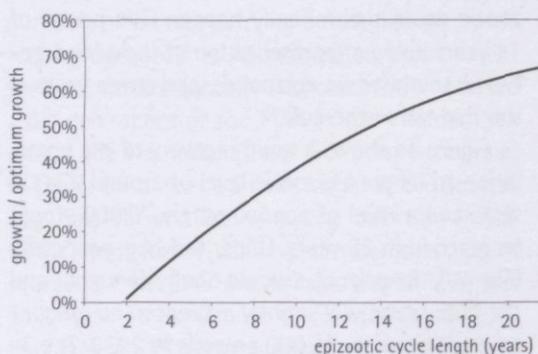


Figure 5: Modelled net growth expressed as percentage of growth if no epizootic would occur, in relation to epizootic cycle length.

Irrespective of the exact final population size reached after 35 years, it is obvious that under the assumed scenarios different net, long-term population growth rates will be achieved. The implication of these differences are analysed by expressing the net growth rate found under a given scenario, as a percentage of the growth rate when no new epizootic would occur. The results for scenarios ranging from 2–20 years are given in Figure 5. The conclusion from this figure is that under all the tested scenarios the net growth rate would be considerably below the value reached if no epizootic would occur. If the interval of 14 years between the last two epizootics is taken, the net population growth would have been around half of what it would have been without a new epizootic.

The relevant question in this respect is, what the chances are for recurring epizootics. It is suggested that infectious diseases of wildlife will emerge in the future (Daszak et al. 2000), including in the marine environment (Harvell et al. 1999).

These are brought about by both climatic changes as well as anthropogenic factors, including (un)intentional global transport of species and inherent pathogenic commensals. This observation, the elegant study by Harding et al. 2002 who expressed the consequences of PDV-epizootics amongst the harbour seal population in the Kattegat/Skagerrak in terms of an increased quasi-extinction risk, and our results on the reduced growth rate in the Wadden Sea harbour seal population caused by recurrent epizootics, have large consequences for future management of those populations. In designing management plans for harbour seals in both the Wadden Sea and the Baltic Sea, it has to be taken into account that these populations are subject to unexpected and timely unpredictable events such as epizootics. Any other factor that reduces population growth *viz.* size – e.g. hunting, culling or resource limitation – will put the population now at a higher risk (such as extinction, inbreeding) than hitherto assumed on the basis of predicting the impact of a single intervention.

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