Morbillivirus infections in European seals before 1988

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THE authors have shown that the recent epizootic among harbour seals (*Phoca vitulina*) in north-west Europe, was caused by a morbillivirus closely related to canine distemper virus and rinderpest virus (Mahy and others 1988, Osterhaus and Vedder 1988, Osterhaus and others 1988, 1989a). This virus has been called the phocine distemper virus. There is evidence that a similar morbillivirus infection occurred in Lake Baikal seals one year earlier (Grachev and others 1989, Osterhaus and others 1989b). Recently, Dietz and others (1989) reported that morbillivirus-specific antibodies were detected in serum samples from harp seals (*Pagophilus groenlandicus*) and ringed seals (*Phoca hispida*) in Greenland, before the presence of a morbillivirus was demonstrated in Europe and lake Baikal.

Serum samples taken from harbour seals admitted to the seal sanctuary in Pieterburen from 1984 to 1988 have now been screened for the presence of antibodies to canine distemper virus and rinderpest virus (Fig 1). As reported previously (Osterhaus and Vedder 1988) neutralising antibodies to canine distemper virus were found in virtually all seals which had developed clinical signs of disease after the start of the outbreak. In addition, one sample collected in 1986 showed a low titre of antibody in this test (titre 30), but it was negative in the neutralisation test for rinderpest virus (Anderson and others 1982) and could not be confirmed as positive in either distemper virus or rinderpest virus antibody ELISAs (Anderson and others 1983, De Vries and others 1988). Of 30 serum samples collected before the outbreak which were negative in the distemper virus-neutralisation test, five contained low titres of rinderpest virus-neutralising antibodies (titres 30 to 60). Three of these were also positive in the rinderpest virus-antibody ELISA.

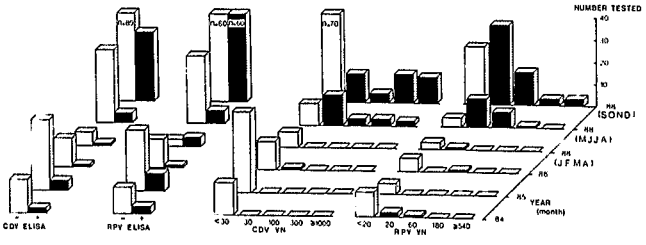

FIG 1: Morbillivirus-specific antibodies in harbour seals admitted to the seal sanctuary in Pieterburen from 1984 to 1988. Canine distemper virus (CDV) and rinderpest virus (RPV) ELISA + or −, are positive and negative serum samples in these tests. CDV− or RPV− virus neutralisation (VN) titres are given as reciprocals of serum dilutions. □ negative, ■ positive. Initials of months are given in brackets

Two serum samples had been collected from one of the above rinderpest virus-seropositive seals in 1986, one when it arrived in the sanctuary and the other two weeks later, after the animals had developed what was to be a fatal seal herpesvirus infection (Osterhaus and others 1985). The first of these samples was negative in neutralisation tests for canine distemper virus and rinderpest virus, but the second sample was positive in the neutralisation test for rinderpest virus (titre 60) but negative in the neutralisation test for canine distemper virus (titre <30). This second sample was also positive in both the rinderpest and canine distemper virus-antibody ELISAs, whereas the first sample was negative in both ELISAs. This indicates that an active morbillivirus infection occurred in this animal during its stay in the sanctuary. Although this seal had been kept under the same conditions which in 1988 allowed the rapid spread of phocine distemper virus in the sanctuary (Osterhaus and Vedder 1988) no other seal developed clinical signs that suggested infection with this virus.

Negative results in the ELISA for canine distemper virus were established in 14 sera from harbour seals, which had remained free from infection with phocine distemper virus (Osterhaus and others 1989a) while in captivity, and in 17 sera from Weddell seals (*Leptonychotes weddelli*) from the Antarctic (Osterhaus and Vedder 1988) where no signs of morbillivirus infection had been found. Similarly, the threshold negative value for the rinderpest virus ELISA was established by using 24 sera, which were collected during 1976 and which were assumed to be free from antimorbillivirus antibodies. Sera collected from seals that developed disease during the recent outbreak all contained titres of antibody in the canine distemper virus and rinderpest virus ELISAs which were significantly above the negative values established above. These results confirm the specificity of these ELISAs for the presence of antibodies to a morbillivirus in the sera of infected seals. Seal sera collected before the outbreak in 1988 were either negative in all four tests or showed significant titres in the canine distemper and rinderpest virus-antibody ELISAs while remaining negative in the corresponding neutralisation tests. In contrast, sera collected from the majority of infected seals during the 1988 epidemic were positive in all four tests. The absence of neutralisation activity in sera collected before 1988 suggests that there is a qualitative difference between the types of antibodies produced before and after 1988, which may reflect antigenic differences between the viruses circulating in the seals of north-west Europe at these times. The virus circulating in the current outbreak is distinguished by having increased pathogenicity in harbour seals, which has enabled the identification of the morbillivirus disease problem; the problem might not have been noticed had the morbillivirus only infected grey seals, which show milder signs of disease. An analogous situation has been demonstrated for an isolate of rinderpest virus, which is non-pathogenic in sheep and goats but which results in high mortality in cattle (Al Hassan and others 1989, Diallo and others 1989).

The sudden appearance of a seal virus with altered biological properties could have been due to a mutation which altered the pathogenicity of a pre-existing non-pathogenic virus within the host species, or to a mutation that changed the host-species range of the virus. The observed differences between the antibodies present in sera collected from seals before and during the outbreak may reflect these processes, but detailed studies on virus isolates will be required to resolve their origins and variation.

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