Herpesvirus infections in seals: a summary of present knowledge

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Summary: This review summarises the occurrence of herpesvirus infections in pinnipeds and data from investigations carried out by the authors. These data demonstrate that herpesvirus isolates collected from harbour seals (Phoca vitulina Linnaeus, 1758) during the 1988 seal epidemic were almost indistinguishable from feline herpesvirus 1 and that Antarctic Weddell's seals (Leptonychotes weddelli Lesson, 1826) carry antibodies against seal herpesvirus from the north-western Europe seal epidemic. The significance of herpesvirus infections in seals is discussed.

KEYWORDS: Feline herpesvirus 1 – Harbour seal – Phocid herpesvirus 1 – Weddell's seal.

Since the devastating mass mortality of seals in 1988, interest has focused on virus infections of marine mammals. Hitherto, little was known about the major pathogens of these often endangered species. Herpesvirus infections, among others, are considered to be widespread in pinnipeds; however, their role has not yet been fully evaluated.

Terrestrial mammals, including man, are susceptible to indigenous herpesviruses or may be infected by viruses crossing the species barrier. These agents can cause severe disease, especially when immuno-compromising conditions occur. However, information is scarce regarding the significance of herpesvirus infections in marine mammals.

Herpesvirus infection in a pinniped was first recorded in 1985, when twenty-three harbour seal pups (Phoca vitulina Linnaeus, 1758) nursed in a Dutch seal orphanage all developed signs of acute disease (1, 6). In the course of this outbreak, eleven of the animals died with symptoms of pneumonia. A previously unknown alphaherpesvirus, serologically related to feline herpesvirus 1 (FHV1) and canine herpesvirus 1, was identified as the aetiological agent and designated seal herpesvirus (SeHV) or phocid herpesvirus 1. In 1986, a herpesvirus was isolated from the lung of a captive California sea lion (Zalophus californianus) (5). During the mass mortality of seals in 1988, SeHV was frequently isolated (2, 6, 8, 10) but was commonly regarded as being of minor importance. However, co-infection with SeHV and phocine distemper virus (PDV), as well as high titres of SeHV (>10^3 plaque-forming units per gram of organ, brain and lung) detected at post-mortem examination, indicated that SeHV played a considerable role in the mass mortality. This was supported by the fact that abortion in female animals was prominent, a phenomenon which is uncommon in morbillivirus infections, but which is indicative of infection with herpesviruses, such as infectious bovine rhinotracheitis (BHV1), pseudorabies (SHV1), equine abortion (EHV1) and feline rhinotracheitis (FHV1).
Transmission experiments performed on young harbour seals in 1988, using herpesvirus isolates from the ongoing epizootic, revealed a low pathogenicity for these animals (4). However, this does not necessarily prove the insignificance of SeHV infection for harbour seals, since the strain used may not have been identical to the virus which had been shown to be highly pathogenic earlier (7), or the harbour seal population may carry diverse herpesviruses of varying pathogenicity.

The results of investigations by the authors reveal a close relationship between SeHV and FHV1 on the antigenic and nucleic acid level (unpublished findings). To date, this study has included four SeHV isolates from different regions of the North Sea. Sera specific for SeHV and FHV1 showed strong cross reactivity in neutralisation tests and western blot antigen analysis. The similarity was striking with restriction enzyme analysis of the purified viral deoxyribonucleic acid (DNA) (Fig. 1). Only two of sixteen restriction

**FIG. 1**

**Analysis and comparison of purified seal herpesvirus (SeHV) and feline herpesvirus (FHV1) DNA using molecular fingerprinting**

The SeHV originated from the severely afflicted lung of a harbour seal found dead in German waters of the Wadden Sea. The FHV1 was kindly provided by Dr R. Gaskell, Bristol (United Kingdom). Both viruses were grown in feline embryo cells and plaque purified prior to DNA isolation.
enzymes generated slight differences in the genetic fingerprints: in one case, an altered fragment mobility was observed; in the other case, fragment duplication. The immunological and molecular data demonstrate that herpesvirus isolates from harbour seals represent FHV1 or a very closely related virus. If FHV1-like viruses had been recently introduced into seal populations and were widespread during the 1988 epizootic, herpesviruses may have made a considerable contribution, especially as FHV1 is known to have a pronounced uterus tropism (3). The origin of FHV1-like viruses in seals is not clear. A probable route for the infection of seals is contact with virus-contaminated sewage.

In addition, high neutralising antibody titres to SeHV and FHV1 were demonstrated in Antarctic Weddell’s seals (Leptonychotes weddelli Lesson, 1826) (9), thus indicating the presence of a serologically related virus in the Antarctic seal populations. Since no felids which could harbour herpesviruses are present in Antarctica, an evolutionary spread of SeHV can be presumed. However, other routes of infection may be possible, as Weddell’s seals are occasionally seen off the shores of South America, South Africa, Australia and New Zealand.

The data reviewed in this paper suggest that herpesviruses should also be considered as important, widespread pathogens in seals, which may cause severe illness. Herpesviruses almost certainly contributed to the 1988 seal epidemic in a more prominent way than was recognised at the time. Neither PDV nor SeHV can be regarded as the primary cause of the epizootic. It seems more likely that immunosuppression as a result of long-term exposure to pollutants such as organochlorines increased susceptibility to virus infections, leading to higher seal mortality.

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Résumé : Les auteurs font le point sur la fréquence des infections à herpèsvirus chez les pinnipèdes et présentent les résultats de leurs propres recherches. Les données présentées montrent que les souches de virus isolées chez les phoques veaux-marins (Phoca vitulina Linnaeus, 1758) durant l'épidémie de 1988 étaient presque impossibles à distinguer de l'herpèsvirus félin 1 et que des phoques de Weddell (Leptonychotes weddelli Lesson, 1826) sont porteurs d'anticorps spécifiques de l'herpèsvirus responsable de l'épidémie du nord-ouest de l'Europe. Les auteurs discutent de l'importance des infections à herpèsvirus chez les pinnipèdes.

MOTS-CLÉS : Herpèsvirus 1 des phocidés – Herpèsvirus 1 félin – Phoque de Weddell – Phoque veau-marin.

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Resumen: Los autores resumen los conocimientos acerca de la frecuencia de las infecciones por herpesvirus en los pinnípedos y presentan los resultados de sus propias investigaciones. Los datos que ofrecen muestran que las cepas de
herpesvirus aisladas en becerros marinos (Phoca vitulina Linnaeus, 1758) durante la epidemia de 1988 eran casi imposibles de distinguir del herpesvirus felino 1 y que focas de Weddell (Leptonychotes weddelli Lesson, 1826) son portadoras de anticuerpos específicos del herpesvirus responsable de la epidemia del noroeste europeo. Por último, discuten de la importancia de las infecciones por herpesvirus en los pinnípedos.

PALABRAS CLAVE: Becerro marino – Herpesvirus 1 de los pinnípedos – Herpesvirus 1 felino – Foca de Weddell.

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REFERENCES


