RE-EMERGENCE OF PHOCINE DISTEMPER IN THE HARBOUR SEAL POPULATION OF NORTHERN EUROPE

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In the spring of 2002, increased mortality of harbour seals (Phoca vitulina) was observed in Northern Europe, with a total of 1160 dead seals along Scandinavian coasts, and over 50 in the Netherlands by the end of June. Clinical signs included those of respiratory and nervous disease. Samples from necropsied animals were examined for evidence of phocine distemper virus (PDV) infection by RT-PCR, virus isolation, serology, histology, and immunohistochemistry. The results showed that PDV was present in both areas. Based on these findings, together with the known severity of this virus infection in harbour seals, we concluded that PDV infection was the primary cause of mortality of seals in Northern Europe, with over 20.000 seals reported dead. Monitoring of the seal population in the Netherlands for the past ten years by serology and RT-PCR indicated that PDV has not been circulating in this population after the 1988 outbreak. The mortality of harbour seals during the 1988 outbreak in the above areas was estimated at 40 to 60 % of the population. Different factors, including specific immunity (less than 5 % seropositive animals in The Netherlands), pollutant load, and general health status, may have influenced the mortality rate during the 2002 PDV epidemic.

IMMUNOPHENOTYPIC CHARACTERIZATION OF THE HEPATIC INFLAMMATORY CELL INFILTRATES IN COMMON DOLPHINS (*DELPHINUS DELPHIS*)

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This work describes the cellular distribution of T lymphocytes (CD3), immunoglobulin-bearing plasma cells, macrophages, MHC class II antigen and S-100 protein in hepatic lesions observed in 14 common dolphins (*Delphinus delphis*). Non specific reactive hepatitis was identified in 12 dolphins, whereas chronic parasitic cholangitis with lymphoid proliferation was observed in three other dolphins. Non specific reactive hepatitis showed small clusters of CD3+ T cells either in portal areas and hepatic sinusoids. The anti-S100 polyclonal antibody reacted with a variable number of lymphocytes from portal areas and hepatic sinusoids, as well as with Kupffer cells and epithelial cells of the bile ducts. The majority of plasma cells observed in portal areas and hepatic sinusoids were IgG+. In lymphonodular lesions of chronic parasitic cholangitis, the distribution of immunoreactive cells was similar to that found in the cortex of lymph nodes. The presence of stellate cells similar to follicular dendritic and interdigitating cells expressing S-100 protein and MHC class II antigen in lymphonodular lesions suggested that these are highly organized structures developed to enhance antigen presentation to B and T cells.