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EPIZOOTIOLOGY OF MORBILLIVIRUS INFECTION IN NORTH AMERICAN HARBOR SEALS (*PHOCA VITULINA*) AND GRAY SEALS (*HALICHOERUS GRYPUS*)

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ABSTRACT: A longitudinal study of morbillivirus infection among harbor (*Phoca vitulina*) and gray (*Halichoerus grypus*) seals on the Atlantic coast of North America was carried out between 1980 and 1994. Serology also was carried out on harbor seals from the Pacific northwest coast collected in 1992 and 1993. The prevalence of morbillivirus neutralizing antibodies was significantly ($P < 0.0001$) higher in gray (73%, $n = 296$) than in harbor seals (37%, $n = 387$) from the Atlantic. Titers were significantly ($P < 0.0001$) higher against phocine distemper (PDV) compared to any other morbillivirus. Antibodies were not detected in serum from Pacific harbor seals. During the winter of 1991 to 1992 an epizootic occurred among harbor seals on the northeast coast of the United States. The event was characterized by an increase in strandings and by a significant ($P = 0.001$) increase in PDV antibody prevalence to 83% ($n = 36$) in seals stranded that winter. Morbillivirus lesions and antigen were observed in six animals found stranded from southern Maine to Long Island, New York (USA), between November 1991 and April 1992. In addition, morbillivirus encephalitis was detected in tissues from a harbor seal that stranded in 1988. Enzootic infection appeared to be present in both seal species, although with a different prevalence of disease. We propose that enzootic infection among gray seals is facilitated by population size, high annual recruitment and innate resistance to clinical disease. Infection may be maintained in the smaller harbor seal population through casual contact with gray seals.

Key words: Harbor seal, *Phoca vitulina*, gray seal, *Halichoerus grypus*, morbillivirus, phocine distemper virus, serology, histopathology, enzootic, western Atlantic.

INTRODUCTION

Epizootics resulting in mass-mortality are not uncommon among pinnipeds and may play an important role in population dynamics (Harwood and Hall, 1990). Epizootics of undetermined etiology periodically have killed seals in British waters (Harwood and Hall, 1990) and, earlier this century, claimed substantial numbers of Icelandic harbor seals (*Phoca vitulina*) (Bardarson, 1931) and Antarctic crabeater seals (*Lobodon carcinophagus*) (Laws and Taylor, 1957). Scientific investigation of these events is a relatively recent phenomenon. The first confirmed viral-induced mass-mortality was an influenza epizootic that spread through aggregations of harbor seals in New England (USA) during the winter of 1979 to 1980 (Geraci et al., 1982). Since then, various strains of influenza have caused a low level of mortality among harbor seals in this region (Callan et al., 1995).

Despite the heightened awareness of viral disease, the scientific community was not prepared for the recent series of morbillivirus epizootics that spread rapidly through European marine mammal populations (Osterhaus, 1988). Eastern North Atlantic harbor seals were devastated by a canine distemper-like virus called phocine distemper virus (PDV) (Cosby et al.,

1988), that struck at the beginning of the 1988 breeding season (Heide-Jørgensen et al., 1992). Although the origin of PDV and the reason for its sudden appearance in European waters have been the subject of much debate, it now seems likely that the virus originated in harp seals (*Phoca groenlandica*) in which infection is enzootic (Markussen and Have, 1992). Unusual harp seal migrations into the North Sea may have precipitated the epizootic among harbor seals (Heide-Jørgensen et al., 1992). It is note-worthy that gray seals (*Halichoerus grypus*) in the eastern North Atlantic also were infected by PDV but were not as severely affected as the harbor seals (Kennedy et al., 1989). Similarly, gray seals emerged unscathed from the influenza epizootics in New England (Geraci et al., 1984).

Although pinniped morbilliviruses first came to scientific attention in Europe, they now are known to be more widespread. Phocine distemper-like disease recently was recognized in both harp (Daoust et al., 1993) and harbor seals (Duignan et al., 1993) from the Atlantic coast of North America. These findings corroborated serologic evidence of morbillivirus infection in phocids from eastern Canada (Henderson et al., 1992; Ross et al., 1992) and in Atlantic walruses (*Odobenus rosmarus rosmarus*) from the Canadian Arctic (Duignan et al., 1994). However, documentation of the prevalence and significance of infection in North American pinnipeds is sporadic. Our objectives were to determine the prevalence of morbillivirus infection in both harbor and gray seals and to document an epizootic among harbor seals on the U.S. northeast coast during the winter of 1991 to 1992.

MATERIALS AND METHODS

Harbor seals (n = 387) were sampled along the Atlantic coast between Sable Island, Nova Scotia, Canada, a major breeding site, and New York harbor, New York (USA) (Table 1). On Sable Island, free-ranging harbor seals that had hauled out on the beach were approached on all-terrain vehicles and captured individually in nets. While the seals were manually restrained, a blood sample was collected from the epidural vein into untreated glass tubes and allowed to clot for several hours (Geraci and Lounsbury, 1993). The serum was separated by centrifugation and stored at −20 C. Standard length measurements also were taken while the seals were restrained. Age was estimated from the measurements (McLaren, 1993) and the seals were classified as either juveniles (< 2 yr) or adults (> 2 yr) assuming a mean birthday of 25 May for seals on Sable Island (Boulva and McLaren, 1979). Harbor seals from the U.S. Atlantic coast were animals that stranded alive between January 1980 and December 1993 and were sampled on admission to a rehabilitation facility. Records were maintained on all harbor and gray seals that either stranded alive or that were found dead during the same period. Data on stranded harbor seals were analyzed in two annual seasons related to the seals' distribution and behavior. Immature animals aggregate during winter (October through April) at haul-out sites in southern New England (Whitman and Payne, 1990) and migrate northward in May to the breeding range in Maine (USA) and eastern Canada (Payne and Schneider, 1984; Rosenfeld et al., 1988).

Free-ranging harbor seals (n = 80) on the Pacific coast of North America were sampled during March, June, October, and December, 1992 and 1993, at six locations from the Campbell River estuary in British Columbia, Canada, to the Umpqua River in Oregon (USA) (Table 1). Seals on sand-bars were captured by deploying a seine net from a fast-moving boat; those on beaches were approached using four-wheel-drive vehicles. Seals in the water were entangled in 25 cm stretch gill nets. In each case, the seals were caught and restrained in nets and blood was sampled as described above.

Gray seals on the Atlantic coast of North America were sampled for blood from December through February in breeding colonies on Amet and Sable Islands, Nova Scotia, and on the Northumberland Strait pack ice in the southern Gulf of St. Lawrence (Table 1). Whelping areas on the ice were located by helicopter and the seals were approached on foot, captured in nets, and manually restrained for sampling. Gray seals on land were approached on foot or by using all-terrain vehicles and captured in nets as before. Age was determined from body length (McLaren, 1993) and the animals classified as juvenile (< 2 yr) or adult (> 2 yr), assuming a mean birthday of 21 January (Mansfield and Beck, 1977). Molting juveniles (n = 22) were captured by net and sampled for blood on Sable Island in June 1992. In January 1981, blood samples were obtained on Sable Island from 22

culled gray seals and additional samples were collected between May 1993 and June 1994 from 60 culled adult seals (40 females, 20 males). Gray seals (n = 54) sampled along the U.S. northeast coast all were juveniles that had stranded between December 1979 and September 1993.

Virus neutralization tests were carried out as described by Duignan et al. (1994) using PDV obtained from A.D.M.E. Osterhaus, National Institute of Public Health, The Netherlands; canine distemper virus (CDV), Onderstepoort strain and measles virus (MV), Edmonston strain obtained from M. J. G. Appel, Cornell University, New York, peste des petits ruminants (PPRV) Nigeria 75/1 attenuated strain (Diallo et al., 1989); and rinderpest virus (RPV) RBOK vaccine strain obtained from the U.S. Department of Agriculture, Foreign Animal Diseases Diagnostic Laboratory, Greenport, New York). Titration of the sera against all five morbilliviruses was warranted by the fact that there is serologic cross-reactivity and titers are highest against the homologous virus (Liess et al., 1989). Virus neutralizing titers of $\log_2$ 4 (1:16 dilution), or greater, were considered positive. Any serum samples observed to be contaminated by bacteria or to be non-specifically toxic to Vero cells were regarded as negative; therefore, reported prevalences are minimum values. Frequencies of seropositive animals were compared by Yates corrected Chi-square test and Fisher's exact test, and 95% confidence intervals (C.I.) were calculated for annual infection prevalence among harbor seals (Martin et al., 1987). Antibody titers against different viruses and between age classes were compared using Student's t-test. Statistical analyses were carried out using InStat software (Graph Pad Software, Inc. San Diego, California, USA).

Between August 1991 and March 1993, necropsies were performed on 59 harbor seals collected along the U.S. northeast coast from the Canadian border to New York harbor. The seals either were recently dead when found or had died shortly after arrival at a stranding response center. Blood samples were collected from all live seals (n = 35) and included in the serologic survey (Table 1). Clinical signs of disease usually involved emaciation, respiratory distress, subcutaneous emphysema, severe parasite burdens and, in some animals, neurological impairment. Cases with a poor prognosis were euthanized by sodium pentobarbital (0.5 ml/kg intra-venous, Fatal Plus, Vorteck Pharmaceuticals, Dearborn, Michigan, USA). Six juvenile gray seals that died after stranding between 1991 and 1993 also were examined for evidence of morbillivirus infection.

At necropsy, tissue samples from the eyelid, tongue, trachea, lungs, spleen, peripheral lymph nodes, liver, pancreas, stomach, kidneys, urinary bladder, and brain were fixed in 10% buffered formalin, passed through alcohol and xylene, and embedded in paraffin. Sections were cut at 5 μm and stained with hematoxylin and eosin (H&E) for light microscopy (Luna, 1968). Staining for morbillivirus antigen was carried out as described by Duignan et al. (1993) using an avidin-biotin complex (ABC) immunoperoxidase technique and a monoclonal antibody against the nucleoprotein of PDV obtained from C. Lyons and A. Trudgett, The Queen's University, Belfast, Northern Ireland. Tissues from a harp seal with morbillivirus infection and a normal harbor seal were used respectively as positive and negative controls (Daoust et al., 1993). Test and control sections were lightly counterstained in hematoxylin prior to mounting (Luna, 1968). Paraffin tissue blocks from an adult male harbor seal with neurological signs that stranded in 1988 were re-examined by light microscopy and immuno-histochemistry.

RESULTS

The prevalence of morbillivirus neutralizing antibodies in harbor seals from the Atlantic was 37% (n = 387, 95% C.I. = 32 to 42%), while the prevalence in gray seals from the same geographic region was 73% (n = 296, 95% C.I. = 68 to 78%). Based on a Yates corrected chi-square test the association between prevalence and species was significant ($P < 0.0001$) both for the juvenile age class ($P = 0.01$) and for the adults ($P < 0.0001$). Among harbor seals, significantly ($P = 0.02$) more adults were seropositive than juveniles; this was even more pronounced in the gray seals ($P < 0.0001$, Table 2). There was no significant association between antibody prevalence and sex for either species of seal (Table 2). By contrast with the Atlantic population, Pacific harbor seals had no evidence of serum antibodies against morbillivirus.

Using a t-test, the mean antibody titer in adult harbor and gray seals was significantly ($P < 0.0001$) higher against PDV than against any other morbillivirus tested, including CDV (Fig. 1). Mean PDV neutralizing titers were significantly ($P < 0.001$) higher in adult seals than in the juveniles for both species. The mean an-

TABLE 1. Sampling locations and dates for harbor seals from the Atlantic and Pacific coasts and gray seals from the Atlantic.

Location	Coordinates	Year	Juveniles		Adults		Total tested
			Male	Female	Male	Female	
Harbor seals (Atlantic, free-ranging)							
Sable Island, Nova Scotia	43°55′N, 60°00′W	1981	NT[a]	NT	2	NT	2
		1992	7	14	NT	16	37
		1994	4	5	5	12	26
Harbor seals (Atlantic, strandings)							
U.S. Atlantic coast	45°00′N, 67°00′W to 40°30′N, 74°00′W	1980 to 1993	147	160	6	9	322
Totals for Atlantic harbor seals			158	179	13	37	387
Harbor seals (Pacific, free-ranging)							
Boundary Bay, British Columbia	49°00′N, 123°00′W	1992	5	2	10	4	21
Vancouver Is., British Columbia	49°00′N, 123°30′W	1992	1	5	4	2	12
Campbell River, British Columbia	50°00′N, 125°10′W	1993	1	1	1	2	5
Snake Island, British Columbia	49°10′N, 124°00′W	1993	3	2	4	NT	9
Puget Sound, Washington	47°30′N, 123°00′W	1993	NT	2	3	1	6
Umpqua River, Oregon	43°50′N, 124°15′N	1993	7	14	4	2	27
Totals for Pacific harbor seals			17	26	26	11	80
Gray seals (free-ranging)							
Amet Island, Nova Scotia	45°50′N, 63°10′W	1980	NT	4	NT	NT	4
		1983	NT	6	NT	NT	6
Sable Island, Nova Scotia	43°55′N, 63°00′W	1981	4	2	10	6	22
		1992	14	8	NT	NT	22
		1993	12	13	NT	55	80
		1994	NT	NT	20	10	30
Gulf of St. Lawrence, Canada	45°N, 63°W to 46°N, 61′W	1991	NT	NT	7	13	20
		1992	6	6	NT	20	32
		1993	NT	NT	NT	19	19
		1994	NT	NT	6	1	7
Gray seals (stranded)							
U.S. Atlantic coast	45°00′N, 67°00′W to 40°30′N, 74°00′W	1979 to 1993	32	22	NT	NT	54
Totals for gray seals			68	61	43	124	296

[a] Not tested.

tibody titers against PDV were not significantly different between species for either the adult ($P = 0.06$) or the juvenile ($P = 0.74$) age groups.

Among free-ranging seals from Nova Scotia, the prevalence of antibodies against PDV was higher in gray seals (82%, $n = 242$, 95% C.I. = 77 to 87%) than in harbor seals (54%, $n = 65$, 95% C.I. = 42 to 66%). Gray seals were sampled in seven of the years between 1980 and 1994 and, excluding 1980, 1983, and 1992 when most samples were from juveniles, there were no significant differences in prevalence between years (Table 3). Insufficient numbers of free-ranging harbor seals were sampled to allow analysis of trends.

Harbor seals sampled on the U.S. northeast coast were mainly stranded juveniles (307 of 322, 95%). Antibodies to PDV first were detected in four of nine seals that stranded in 1980, the first year from which

TABLE 2. Prevalence of morbillivirus neutralizing antibodies in harbor and gray seals from the Atlantic coast.

Species	Age class		Sex		Total
	Juvenile	Adult	Female	Male	
Harbor seal	118/337 (35)*	27/50 (54)	84/216 (39)	61/171 (36)	145/387 (37)
Gray seal	62/129 (48)	153/167 (92)	138/185 (75)	77/111 (69)	215/296 (73)

* Number positive/number sampled (percent positive).

samples were available for testing. The annual prevalence between 1980 and 1993 for a total of 307 juveniles and 15 adults ranged from 0% to 63% with no significant difference between years. On analysis of the data by season, there was an increase in the prevalence of PDV neutralizing antibodies to 83% (n = 36, 95% C.I. = 71 to 95%) in stranded harbor seals during the winter of 1991 to 1992 (Fig. 2). Using Fisher's exact test, the association between time period and prevalence was significant (P = 0.001). The rise in antibody prevalence corresponded to an increase in the number of live strandings and beached carcasses recorded during the same period (Fig. 2).

Histological changes and positive immunoperoxidase staining consistent with morbillivirus infection were found in six (20%) of 30 (95% C.I. = 6 to 34%) stranded harbor seals (five juveniles and one adult). The seals stranded from 21 November 1991 to 14 April 1992 between southern Maine (41°39′N, 70°07′W) and Fire Island, New York, (40°38′N, 73°08′W). The adult male was comatose when found and the juveniles had signs of dyspnea and depression. Three seals died shortly after arrival at rehabilitation centers and the others were euthanized. Nonsuppurative encephalitis was the primary lesion in five animals while the sixth had broncho-pneumonia. Seals examined during the summer of 1991 (n = 1), 1992 (n = 13) and the winter of 1992 to 1993 (n = 15) had no evident morbillivirus lesions. One adult male (MH88-518 Pv) that stranded in 1988 had progressive neurological impairment for 7 mo. At necropsy, nonsuppurative encephalitis and morbillivirus antigen were present in the cerebrum. The seven affected seals had PDV-neutralizing antibody titers ranging from $\log_2$ 4 (1:16) to $\log_2$ 9 (1:512). Throughout the rehabilitation period, MH88-518 Pv maintained a stable antibody titer.

Neutralizing titers were found in 16 (30%) of 54 (C.I. = 18 to 42%) juvenile gray seals that stranded on the U.S. northeast coast between 1979 and 1993. By contrast with harbor seals, gray seal stranding

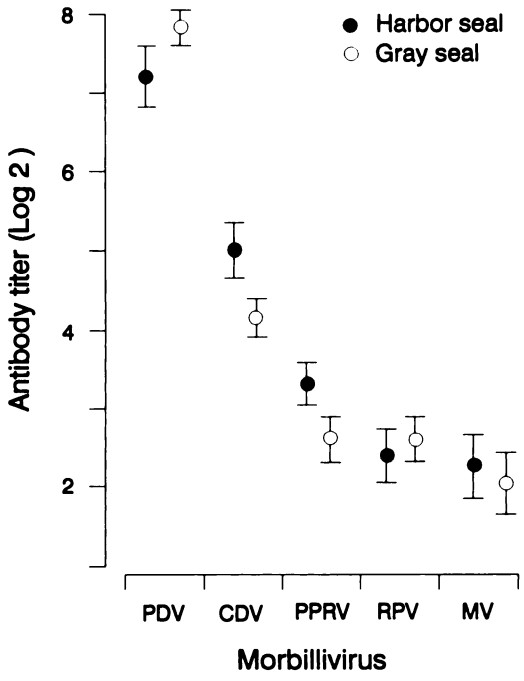

FIGURE 1. Virus neutralizing antibody titers (mean and standard error) against phocine distemper virus (PDV), canine distemper virus (CDV), peste des petits ruminants virus (PPRV), rinderpest virus (RPV) and measles virus (MV) in harbor seals (n = 387) and gray seals (n = 296) from the western Atlantic.

TABLE 3. Prevalence of morbillivirus neutralizing antibodies in free-ranging harbor and gray seals from Maritime Canada.

Year	Location	Age class		Prevalence
		Juvenile	Adult	
Harbor seal				
1981	Sable Island	NT[a]	2/2[b]	100
1992	Sable Island	13/21	13/16	70 (55 to 85)[c]
1994	Sable Island	0/9	7/17	27 (10 to 44)
Gray seal				
1980	Amet Island	0/4	NT	0
1981	Sable Island	6/6	16/16	100
1983	Amet Island	1/6	NT	17 (14 to 20)
1991	Gulf of St. Lawrence	NT	17/20	85 (70 to 100)
1992	Gulf of St. Lawrence and Sable Island	15/34	17/20	59 (46 to 72)
1993	Gulf of St. Lawrence and Sable Island	24/25	68/74	93 (88 to 98)
1994	Gulf of St. Lawrence and Sable Island	NT	35/37	95 (88 to 100)

[a] Not tested.
[b] Number positive/number sampled.
[c] Percent positive (95% confidence interval).

frequency did not increase between 1989 and 1993 (annual mean = 13, range = 11 to 16) and six animals examined after stranding did not have distemper lesions.

DISCUSSION

We found morbillivirus infection in both free-ranging and stranded harbor and gray seals from the western Atlantic as early as 1980. Based on differential virus neutralization tests against five morbilliviruses, we believe that the North American seals were infected by a virus similar, if not identical, to PDV. This kind of assay was previously used to distinguish PDV from CDV infection in European seals (Liess et al., 1989) and the sensitivity and specificity of the assay were further demonstrated using experimentally infected harbor seals (Harder et al., 1992). A morbillivirus has not yet been isolated from North American seals, precluding a direct comparison with PDV. Under similar circumstances, however, differential neutralization tests have been used to identify the most likely cause of infection in crabeater seals (Bengtson et al., 1991), harp seals (Markussen and Have, 1992), and Atlantic walruses (Duignan et al., 1994) from which there were no viral isolates.

The antibody prevalence in adult gray seals (92%) was significantly higher than in adult harbor seals (54%), and similar to that observed in enzootic CDV where the prevalence may be 89% in unvaccinated dogs over 2 yr of age (Rockborn, 1958). For both seal species, the prevalence of antibody in juveniles was significantly lower than in adults. Thompson et al. (1992) reported similar findings in post-epizootic European harbor seals and proposed that an immature immune system may account for the difference. Yet, Ross et al. (1993) found that harbor seal pups have a competent immune response. An alternative explanation for the age disparity, therefore, is the timing of sampling. Antibody titers in seals less than 3-mo old probably are of maternal origin (Harder et al., 1992) and samples taken after this period are likely to have declining or negative titers. Furthermore, seals in their first year tend to disperse away from breeding colonies and either haul out infrequently or in small

groups (Boulva and McLaren, 1979; Harwood et al., 1989). Thus, the potential for virus transmission to, and between, juvenile seals is much less than between older animals.

Based on our data, we propose that a PDV-like morbillivirus is enzootic in gray seals and that the virus constantly circulates within the population resulting in a level of herd immunity sufficient to prevent an epizootic (Gorham, 1966; Martin et al., 1987). This hypothesis is based on the presumption that the host population is large enough to maintain infection and that recruitment of non-immune animals is above a certain threshold (Martin et al., 1987). Although these absolute requirements have not been defined for pinnipeds, we believe that they are fulfilled by gray seals. Decimated by over-hunting since the early 1800's the gray seal population in eastern Canada has grown exponentially to more than 120,000 animals (Zwanenburg and Bowen, 1990) and small breeding colonies have recently become established in Maine (Kenney and Gilbert, 1994). The principal colonies also are expanding (Stobo and Zwanenburg, 1990) and provide an environment where pups are likely exposed to morbillivirus early in life while protected from clinical disease by maternal antibodies, and later by their own induced immunity. Although an epizootic is unlikely, individuals may succumb if the balance between the challenge dose and the level of immunity is altered.

The lower prevalence of morbillivirus infection among sympatric harbor seals on the Atlantic coast is intriguing. We propose three possible explanations for this finding. First, there are fewer than 50,000 harbor seals (Kenney and Gilbert, 1994; Stobo and Fowler, 1994) distributed between several, apparently isolated, groups from Labrador to southern New England (Boulva and McLaren, 1979; Payne and Schneider, 1984). Thus, the smaller, more fragmented, harbor seal population may not be sufficient to maintain morbillivirus infection. Second, based on mortality data

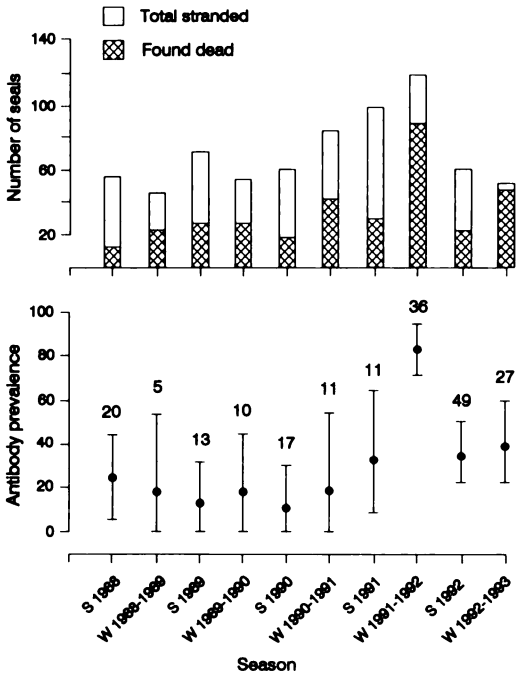

FIGURE 2. Number of harbor seals stranded, and number found dead, on the northeast coast of the United States by season (top). Summer (S) is the period from May through September and winter (W) is the period from October through April. Seasonal prevalence (percent seropositive), and 95% confidence intervals, of PDV neutralizing antibody titers in harbor seals stranded on the northeast coast over the same period (bottom). The number above each bar is the sample size.

from the epizootic in Europe, harbor seals may be inherently more susceptible to PDV than gray seals (Kennedy et al., 1989). If so, clinical disease may occur more frequently among harbor seals, thereby removing infected animals from the population. By contrast, gray seals are more likely to have a sub-clinical infection, develop antibodies, and transfer passive immunity to their pups. In this study we observed a significant difference in antibody prevalence between juvenile harbor and gray seals. Third, behavioral differences between the species may play a role in disease transmission and maintenance of enzootic infection. Harbor seals tend to form small groups during the breeding season (Kovacs et al., 1990) and larger ca-

sual aggregations at haul-out sites during winter (Boulva and McLaren, 1979). However, group size probably is determined as much by the size and density of the population as by social constraints. By contrast, gray seals form large breeding colonies in winter (Boness and James, 1979), large dense molting aggregations during spring and early summer, and casual aggregations at other times of the year (Mansfield and Beck, 1977). Outside the breeding and molting seasons, gray seals from the Gulf of St Lawrence and Sable Island are known to disperse widely along the coast from Labrador to Cape Cod, Massachusetts (USA) (Stobo et al., 1990; Lavigueur and Hammill, 1993). Juvenile harbor seals also disperse away from breeding areas but to a lesser degree than gray seals (Boulva and McLaren, 1979). Thus, the opportunity for density-driven virus transmission potentially is greater between gray seals (Harwood, 1989; Harwood and Grenfell, 1990).

Enzootic infection appears to be present in both seal species, although with a different average frequency of disease (Martin et al., 1987). The lower proportion of immune adult harbor seals in the population likely results in an increase in the proportion of susceptible animals with each successive whelping season. Furthermore, the number of harbor seals over-wintering in southern New England has increased in recent years (Payne and Selzer, 1989), and perhaps of greater significance is that these aggregations are composed almost exclusively of immature animals (Whitman and Payne, 1990). The combination of these host factors might have promoted the epizootic that occurred during the winter of 1991 to 1992.

The extent of this epizootic is difficult to quantify. Some animals certainly died from pneumonia and encephalitis (Duignan et al., 1993), but the more indirect effects of morbillivirus infection, leading to immunosuppression and secondary infections, are difficult to assess. An additional confounding factor is the role that influenza played in the harbor seal strandings (Callan et al., 1995). In fact, one of the stranded seals examined in January 1992 for this study had necrotizing pneumonitis characteristic of influenza infection (Geraci et al., 1982), and influenza A (H_3N_3) was isolated from its lungs (Callan et al., 1995).

The contrast between the epizootic reported here and the European event is striking. Infection progressed rapidly through European seal colonies causing pneumonia in its victims (Kennedy et al., 1989; Heide-Jørgensen et al., 1992). By contrast, the epizootic on the U.S. northeast coast smoldered along for at least 5 mo over a wide geographic area. The predominant lesion was encephalitis, which in dogs with CDV signals a more chronic manifestation of disease (Appel et al., 1981). Enzootic infection in pinnipeds appears to take this chronic form. A low level of immunity in the affected seals may have been sufficient to prevent acute fulminant infection but insufficient to prevent chronic infection of the central nervous system. It is interesting that encephalitis also was the principal finding in the only documented case of clinical distemper in a harp seal (Daoust et al., 1993).

Despite the elevation of herd immunity among harbor seals during the epizootic, we observed a substantial drop in antibody prevalence following recruitment of the 1992 cohort. Thus, we believe recurrence of disease is not unlikely on the U.S northeast coast. Similar predictions, dependant on re-introduction of PDV, were made following the European epizootic (Harwood et al., 1989; Harder et al., 1993). The diagnosis of morbillivirus encephalitis in a harbor seal that stranded in 1988 lends credence to the proposal that epizootics may occur periodically.

Assuming that the morbillivirus infecting harbor and gray seals is the same, where did it originate and how is it maintained on the western Atlantic? We might infer circumstantially that, since the virus was present in both species in the early 1980's, long before its appearance in Europe, it

may have originated in North America. Harp seals, dispersing from the large breeding colonies in Canada, possibly carried the virus to the eastern Atlantic (Sergeant, 1973). Alternatively, phocine distemper is a disease of Arctic phocids, and harp seals may have seeded first the western, and then the eastern, Atlantic (Markussen and Have, 1992). Gray seals probably are an important reservoir host in the western Atlantic and casual contact between them and harbor seals at shared haulout sites (Davis and Renouf, 1987; Stobo and Fowler, 1994) may provide a means of inter-species transmission. Confirmation of the identity and relationships of the viruses infecting different hosts should help resolve these questions.

Further studies also must be done to evaluate the potential risk to the harbor seals along the Pacific coast. The exponential population increase in this region following legislative protection in the early 1970's (Calambokidis et al., 1979; Olesiuk et al., 1990) parallels the increase in the Kattegat and Skaggerak prior to the PDV epizootic in Europe (Heide-Jørgensen and Härkönen, 1988). The only thing protecting Pacific harbor seals at present is the barrier formed by the Arctic, and as yet, there is no evidence that a morbillivirus has penetrated that far west (Osterhaus et al., 1988; Steiger et al., 1989). Yet, hooded seals (*Cystophora cristata*) from the eastern North Atlantic recently appeared as far from their normal range as the western Beaufort Sea (Burns and Gavin, 1980) and California (Dudley, 1992), and it might only take a single such event to produce an epizootic as devastating as the recent one in Europe.

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