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PREVALENCE AND DISTRIBUTION OF SERUM NEUTRALIZING ANTIBODIES TO TILLAMOOK (BOVINE) CALICIVIRUS IN SELECTED POPULATIONS OF MARINE MAMMALS

Jeffrey E. Barlough,[1,2] Eugene S. Berry,[1] Alvin W. Smith,[1] and Douglas E. Skilling[1]

ABSTRACT: Neutralizing antibodies to Tillamook calicivirus (TCV) were found in sera collected from California sea lions (*Zalophus c. californianus* Lesson) in 1983 and 1984 and in sera collected from Steller sea lions (*Eumetopias jubatus* Schreber) in 1976 and 1985. The combined prevalence of antibodies for these two species was 10/228 = 4.38%. Titers ranged from 1:20 (five animals), to 1:40 (four animals), to 1:80 (one animal) by standard microtiter neutralization assay. The seropositive pinnipeds were dispersed widely along the margins of the eastern Pacific rim, from the Bering Sea to the Santa Barbara Channel. Antibodies to TCV were not found in sera collected from northern fur seals (*Callorhinus ursinus* L.), Pacific walruses (*Odobenus rosmarus divergens* Illiger), seals of the family Phocidae, or several cetacean species. Tillamook calicivirus was isolated originally in 1981 from dairy calves in Oregon; the finding of neutralizing antibodies in two widely distributed species of sea lions suggests the possibility of a marine origin for this agent.

INTRODUCTION

Reports published over the past 14 yr indicate that caliciviruses are circulating among a number of animal species of the Pacific Ocean basin, including pinnipeds, cetaceans and fish (Smith and Akers, 1976; Smith, 1981; Barlough et al., 1986a). These marine viruses are of considerable interest because they are physicochemically and morphologically indistinguishable from the exotic disease agent, vesicular exanthema of swine virus (VESV), and are capable of producing vesicular lesions in experimentally exposed pigs (Bachrach and Hess, 1973; Schaffer and Soergel, 1973; Smith et al., 1973, 1977, 1980b; Breese and Dardiri, 1977; Wilder et al., 1977; Wilder and Dardiri, 1978; Berry et al., 1985). Such findings have served to support the hypothesis that the costly outbreaks of vesicular exanthema of swine (VES) that spread throughout California and eventually to the remainder of the United States between 1932 and 1956 originated from calicivirus reservoirs in the sea (Madin, 1973; Smith et al., 1973; Sawyer, 1976; Smith and Akers, 1976; Smith, 1981). The feeding of virus-contaminated fish scraps and marine mammal remains to domestic swine has been proposed as the major means by which these "marine caliciviruses" gained access to terrestrial populations (Madin, 1973; Smith and Akers, 1976; Sawyer et al., 1978; Smith et al., 1980b; Smith, 1981).

In 1981 a calicivirus was recovered from calves in a dairy herd in Oregon with persistent respiratory problems (Smith et al., 1983a). This agent, the Tillamook calicivirus (TCV), has been shown to produce small cutaneous lesions in experimentally inoculated calves, and mild clinical VES in inoculated swine (Smith et al., 1983a). Considering our present knowledge of the natural history of VESV, and of the marine caliciviruses in general, the question of a possible marine origin for TCV has been raised. In this paper we report the results of a serologic study to search for serum neutralizing (SN) antibodies to TCV in Pacific populations of marine mammals.

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TABLE 1. Results of serologic testing for SN antibodies to Tillamook calicivirus in selected populations of marine mammals.

Species	Location and year of sample collection	Serologic results: No. positive/ no. sampled	Serologic results: Percent positive
Otariid pinnipeds			
California sea lion (*Zalophus c. californianus*)	San Miguel Island, California (1977)	0/43[a]	0
	Fort Cronkhite, California (1983)	4/10	40
	San Miguel Island, California (1984)	2/29[a]	6.9
	Total	6/82	7.3
Steller sea lion (*Eumetopias jubatus*)	Eastern Bering Sea (1976)	1/5	20
	Fort Cronkhite, California (1983)	0/1	0
	Near Kodiak Island, Alaska (1985)	2/74	2.7
	Near Kodiak Island, Alaska (1985)	0/29[b]	0
	Rogue Reef, Oregon (1985)	1/37[a]	2.7
	Total	4/146	2.7
Northern fur seal (*Callorhinus ursinus*)	San Miguel Island, California (1976)	0/13[a]	0
	St. Paul Island, Alaska (1978)	0/40	0
	Fort Cronkhite, California (1983)	0/2	0
	San Miguel Island, California (1984)	0/1	0
	San Miguel Island, California (1984)	0/13[a]	0
	Total	0/69	0
Pacific walrus (*Odobenus rosmarus divergens*)	South-central Chukchi Sea (1983)	0/154	0
	South-central Chukchi Sea (1983)	0/1[c]	0
	Western Bering Sea (1984)	0/68	0
	Total	0/223	0
Phocid pinnipeds			
Northern elephant seal (*Mirounga angustirostris*)	Fort Cronkhite, California (1983)	0/10	0
	San Miguel Island, California (1985)	0/56	0
	Total	0/66	0
Hawaiian monk seal (*Monachus schauinslandi*)	Hawaiian leeward islands (1985)	0/4	0
	Hawaiian leeward islands (1985)	0/1[a]	0
	Total	0/5	0
Ringed seal (*Phoca hispida*)	Barrow, Alaska (1975)	0/1	0
Spotted seal (*Phoca largha*)	Eastern Bering Sea (1976)	0/10	0
Ribbon seal (*Histriophoca fasciata*)	Eastern Bering Sea (1976)	0/4	0
Pacific harbor seal (*Phoca vitulina richardsi*)	Eastern Bering Sea (1976)	0/2	0
	Grays Harbor, Washington (1979)	0/30	0
	Fort Cronkhite, California (1983)	0/17	0
	Total	0/49	0
Bearded seal (*Erignathus barbatus*)	Eastern Bering Sea (1976)	0/1	0
Cetaceans			
Killer whale (*Orcinus orca*)	Victoria, British Columbia (1985)	0/3	0
Atlantic bottlenosed dolphin (*Tursiops truncatus*)	San Diego, California (1979)	0/16	0
Pacific bottlenosed dolphin (*Tursiops gilli*)	San Diego, California (1979)	0/3	0
	Grand total	10/678	1.47

MATERIALS AND METHODS

Viruses

Plaque-purified TCV was propagated in Vero cell cultures (CCL 81, American Type Culture Collection, Rockville, Maryland 20852, USA) grown in 75-cm² flasks, as described previously (Smith et al., 1983a). Virus was harvested by freeze-thawing culture flasks, followed by low-speed centrifugation of culture fluids to remove cell debris. Supernatants were dispensed in 0.2-ml aliquots into screw-cap glass vials and stored at −70 C. Stock aliquots contained approximately $10^{6.75}$ $TCID_{50}$/0.025 ml.

Serology

A microtiter (96-well) SN procedure using Vero cells was performed, first to screen sera at a dilution of 1:20, and then to titrate positive samples (Monto and Bryan, 1974; Smith et al., 1976, 1983a; Barlough et al., 1986c). Serum-virus mixtures were incubated for 60 min at room temperature prior to addition of cells. The antibody titer (as judged by cytopathic effect after incubation for 72 hr at 37 C) was defined as the highest dilution of serum completely neutralizing 100 $TCID_{50}$ of virus in all four replicate test wells (100% end-point). Rabbit antiserum to TCV was used as a positive antibody control, and type specificities were checked in parallel SN tests during end-point titrations. The specificity of this antiserum for TCV has been described elsewhere (Smith et al., 1983a).

Serum samples

Blood samples were obtained from a total of 678 marine mammals (Table 1): 82 California sea lions (*Zalophus c. californianus* Lesson), 146 Steller sea lions (*Eumetopias jubatus* Schreber), 69 northern fur seals (*Callorhinus ursinus* Linné), 223 Pacific walruses (*Odobenus rosmarus divergens* Illiger), 66 northern elephant seals (*Mirounga angustirostris* Gill), five Hawaiian monk seals (*Monachus schauinslandi* Matschie), one ringed seal (*Phoca hispida* Schreber), 10 spotted seals (*Phoca largha* Pallas), four ribbon seals (*Histriophoca fasciata* Zimmerman), 49 Pacific harbor seals (*Phoca vitulina richardsi* Gray), one bearded seal (*Erignathus barbatus* Erxleben), three killer whales (*Orcinus orca* Linné), 16 Atlantic bottlenosed dolphins (*Tursiops truncatus* Montagu), and three Pacific bottlenosed dolphins (*Tursiops gilli* Dall).

Blood cells were removed from samples by low-speed centrifugation. The sera were than heat-inactivated (57 C for 30 min) in preparation for SN testing. All samples were stored at −20 C.

RESULTS AND DISCUSSION

The prevalence and distribution of SN antibodies to TCV in the populations of marine mammals under study are presented in Table 1. Neutralizing antibodies were found only in the two species of sea lions sampled (California sea lion and Steller sea lion), in both pups and adult animals. The seropositive individuals showed a wide geographical and temporal distribution, ranging from the coast of Alaska (1976, 1985), to Oregon (1985), to California (1983, 1984).

End-point titration results for the seropositive animals are shown in Table 2. Nine of the ten positive samples were of low to moderate titer (1:20 to 1:40). One animal, an adult female Steller sea lion sampled near Kodiak Island, Alaska, had a neutralizing antibody titer of 1:80 to TCV (animal no. EJ 452).

The overall prevalence of TCV antibodies in the entire collection of sera from marine mammals was 10/678 = 1.47%; among pinnipeds of the family Otariidae, 10/520 = 1.92%; and among sea lion species (subfamily Otariinae), 10/228 = 4.38%. Northern fur seals, Pacific walruses, phocid seals and cetaceans were negative for SN antibodies to TCV at a 1:20 serum dilution.

These data are the first to suggest the possibility of a marine origin for TCV. Although isolated from a dairy herd in Tillamook County, Oregon, in 1981, TCV would appear to have infected sea lions of the North Pacific realm at least as early as 1976. Evidence such as this of possible translittoral movement is a characteristic finding for marine caliciviruses, including

←

[a] Pups.

[b] Fetuses.

[c] Calf.

TABLE 2. Tillamook calicivirus-neutralizing antibody titers in seropositive marine mammals.

Species	Animal no.	Age status	Sex	Location and year of sample collection	Antibody titer
California sea lion	CSL 082	Adult	Male	Fort Cronkhite, California (1983)	1:40
(*Zalophus c. californianus*)	CSL 148	Subadult	Male	Fort Cronkhite, California (1983)	1:20
	CSL 150	Juvenile	Female	Fort Cronkhite, California (1983)	1:40
	CSL 153	Subadult	Male	Fort Cronkhite, California (1983)	1:20
	ZC 658*	Pup	Male	San Miguel Island, California (1984)	1:40
	ZC 894	Pup	Male	San Miguel Island, California (1984)	1:20
Steller sea lion	SUV-25-76	Adult	Female	Eastern Bering Sea (1976)	1:40
(*Eumetopias jubatus*)	EJ 412	Adult	Female	Near Kodiak Island, Alaska (1985)	1:20
	EJ 452	Adult	Female	Near Kodiak Island, Alaska (1985)	1:80
	EJ 33M	Pup	Male	Rogue Reef, Oregon (1985)	1:20

* Eroded flipper vesicle present.

both VESV and San Miguel sea lion virus (SMSV). These agents are believed to be maintained in nature by complex enzootic/epizootic cycles that in some cases may involve phylogenetically diverse marine species (Akers et al., 1974; Smith and Akers, 1976; Smith et al., 1976, 1980b; Smith and Latham, 1978; Smith, 1981; Barlough et al., 1986a, b).

Recently, SN antibodies to two established marine caliciviruses, SMSV-5 and SMSV-13, were found in cattle in Oregon (Berry et al., 1985, and unpubl. data). Both of these caliciviruses have been isolated only from diseased pinnipeds: SMSV-5 from vesicular fluid collected from a northern fur seal in Alaska in 1973 (Smith et al., 1977), and SMSV-13 from an outbreak of vesicular disease among California sea lions near San Francisco in 1984 (Berry et al., 1985). These data, together with those of the present report, suggest the possible existence, either now or in the very recent past, of a mechanism for translittoral movement of caliciviruses from the sea to domestic cattle. While the precise mode of virus transmission (if, indeed, such transmission has occurred) remains speculative, one obvious possibility, reminiscent of the VES outbreaks, might be certain Pacific fisheries products prepared for use as livestock feed or feed supplements, either for cattle directly, or for other, co-resident species (e.g., swine) (Madin, 1973; Smith and Akers, 1976; Smith et al., 1978, 1980a, b, 1981; Smith, 1981). The recognized resistance of caliciviruses to environmental conditions—including an ability to survive, on certain occasions, in supposedly cooked organic matter—is of particular significance in this regard (Bankowski, 1965; Madin, 1970; Smith and Akers, 1976; Schaffer et al., 1980). Once introduced into populations of cattle, viral infection cycles might be maintained by persistent infection of carrier animals—a relatively common sequela to many calicivirus infections

(Wardley, 1976; Studdert, 1978; Smith et al., 1981, 1983b, 1986).

The confinement of TCV antibodies to sea lion sera was not a totally unexpected finding. The vast majority of caliciviruses isolated to date from marine mammals have been obtained from members of the subfamily Otariinae (see Barlough et al., 1986a, for review). Seroepizootiologic studies indicate that California sea lions and Steller sea lions are significant constituents of calicivirus transmission cycles (Akers et al., 1974; Prato et al., 1974; Smith et al., 1976; Smith and Latham, 1978; Berry et al., 1985; Barlough et al., 1987). The northern fur seal, by contrast, in both Alaska and southern California, appears to be involved characteristically at the epizootic level, with little evidence of participation in enzootic cycling of the viruses. Phocid seals (only distantly related to otariids) for the most part are infected only rarely (Akers et al., 1974; Smith and Latham, 1978; Smith et al., 1980b). Such interspecies variation, which has been observed consistently for many years, is believed to reflect dissimilar feeding habits and differences in prey selection—e.g., pelagic feeding of northern fur seals and benthic feeding of northern elephant seals vs. near-shore feeding of California and Steller sea lions (Fiscus and Baines, 1966; Smith et al., 1976, 1980a, b; Jameson and Kenyon, 1977; Smith and Latham, 1978; Pitcher, 1981; Kajimura, 1984). Hence, if certain near-shore sea lion prey species are indeed primary reservoirs for marine caliciviruses, as is currently suspected (see Barlough et al., 1986b for review), then the rather extensive involvement of their sea lion predators in calicivirus transmission cycles is relatively easy to understand. Phylogenetic differences (as between phocid and otariid seals) also are undoubtedly of importance in susceptibility to infection, although much recent work indicates that the host-range spectrum of some marine caliciviruses is unusually broad (see Barlough et al., 1986b for review).

Although isolated from cattle with a history of persistent respiratory problems, TCV has not been linked etiologically to respiratory disease (Smith et al., 1983a). However, it has been shown to produce small cutaneous lesions in experimentally exposed calves, and vesicular disease in inoculated swine. Considering in addition the presence of SMSV-5 and SMSV-13 antibodies in Oregon cattle, some questions present themselves: Are caliciviruses now producing disease of some kind in bovine populations? Might they be involved in some disease outbreaks of unresolved etiology? Recently, caliciviruses not yet propagated in cell culture have been visualized by electron microscopy in fecal specimens from scouring calves in the Pacific Northwest (Mattson et al., unpubl. data). In Britain, caliciviruses (the Newbury agents SRV-1 and SRV-2) have been collected from scouring calves and shown by experimental inoculation to produce anorexia, atrophy of intestinal villi, malabsorption, and diarrhea in gnotobiotic calves (Woode and Bridger, 1978; Bridger et al., 1984; Hall et al., 1984). Indeed, caliciviruses have been reported to be common in calves in Great Britain (Bridger et al., 1984). Newbury agents SRV-1 and SRV-2 appear to be distinct calicivirus serotypes, and their presence in an island nation, with a large fishing industry and an established pinniped population, remains especially intriguing.

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