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Isolation of North American Viral Hemorrhagic Septicemia Virus (VHSV) from Alaskan Pacific Herring, *Clupea harengus pallasi.*

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The North American strain of viral hemorrhagic septicemia virus (VHSV) was first isolated in Alaska from skin lesion material of two Pacific cod, *Gadus macrocephalus*. These fish were sport-caught in Prince William Sound (PWS), Alaska during the summers of 1990 and 1991 (Meyers et al. 1992). During April of 1993, two thirds (about 100,000 tons) of the 5 yr-old 1988 year class of Pacific herring, *Clupea harengus pallasi*, expected to return to spawn in PWS failed to appear. Hence, the commercially important seine fishery was never opened for harvest. Among the herring that did return, 15% to 43% had varying degrees of external ulceration or hemorrhage beneath the skin, at the bases of fins and around the vent accompanied by lethargic swimming behavior. A rhabdovirus identified as VHSV by serum neutralization and further identified as the North American strain of VHSV by cDNA probe methods was isolated from kidney and spleen pools from two groups of 10 of these herring, and from skin and organ samples of a single Pacific cod sport-caught nearby. Also during late April, herring with similar skin and fin hemorrhages were observed near Kodiak Island, Alaska. Subsequently, the same North American VHSV was isolated from one pool of 4 of these fish, although herring returns and the commercial fishery there had been successful. In mid-May, the same VHSV was isolated from over 43% of 46 juvenile herring from Auke Bay, Alaska near Juneau. These fish had a concomitant VEN infection and were captured for use in a VHSV susceptibility study.

This report documents the Pacific herring as a new host species for North American VHSV which may have been responsible for the high prevalences of external skin and fin lesions and lethargic behavior observed in herring returning to PWS. VHSV was present in high titers indicative of active replication in host tissues while other likely fish pathogens were not isolated or observed. Histological lesions observed including massive passive congestion of the liver and kidney, subdermal and kidney hemorrhages, kidney tubule degeneration and active RE cell foci in the livers and kidneys of PWS herring were suggestive of a systemic viral agent.

Whether VHSV played any role in the reduced number of herring returning to PWS has not been established nor has there been verification of major herring mortality except for unconfirmed reports. As indicated by isolation of the virus from herring over wide geographic areas, VHSV may be indigenous to Pacific herring throughout Alaska and possibly the Pacific Northwest. There appears to be no relationship of VHSV isolation with the Exxon Valdez oil spill of 1989 since the virus has been isolated from areas other than PWS. The virus may be an opportunistic pathogen causing periodic occurrences of external and internal lesions in herring following stress from various factors including, VEN infections, spawning, commercial fishing or nutritional deficiency through lack of forage. The latter condition was suggested this year by the smaller size of herring returning to PWS. Some unknown amount of

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herring mortality must occur during these epizootics primarily from the progressive ulcerating skin lesions that would result in osmoregulatory failure and/or act as portals of entry for secondary microbial infections. Such osmoregulatory collapse would result in shock and vasodilation, which may have contributed to some of the passive congestion we observed in tissues.

Our discovery of VHSV in Pacific herring strongly suggests that this fish species may be a major marine reservoir of the virus. A marine reservoir for VHSV has been suggested previously and is more likely now as an explanation for the isolations of VHSV from adult coho, *Oncorhynchus kisutch* ,and chinook, *O. tshawytscha*, salmon returning to Washington State during 1988, 1989 and 1991 (Brunson et al. 1989, Hopper 1989, Winton et al. 1989 & 1991, Eaton & Hulett 1990, Stewart et al. 1990). Should North American VHSV be indigenous in Pacific herring populations then it is possible that the virus will be detected again in salmonids. Additional studies need to be conducted to determine the distribution of the virus in Pacific herring in other Pacific Northwest waters.

Rhabdoviruses are noted for their potential of rapid evolution. Hence, North American VHSV should be a concern to salmonid aquaculturists in the Pacific Northwest and Canada due to its potential to become a significant pathogen of trout and salmon. Currently, North American VHSV appears to be largely adapted to marine fish species but this could be altered if the virus is subjected to strong selective pressures as might occur during the course of intensive fish culture practices. It is very plausible that the European strain of VHSV also came from the marine environment where it became a virulent pathogen of rainbow trout after unpasteurized marine fish were used as a food source for hatchery fish. This exact route of adaption is not as likely today in North America with the use of high quality processed fish foods, but nonetheless conservative methods should be employed to eradicate the virus whenever detected in salmonids.

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ANOUNCEMENTS and MEETINGS

Uses and Effects of Cultured Fishes in Aquatic Ecosystems. March 12-17, 1994. Albuquerque, NM. Contact: Gary Carmichael, 213 Bryn Mawr Drive SE, Albuquerque, NM 87106.

Pathology in Marine Aquaculture, 6th International Colloquium. April 28-30, 1994. Palais des Congres, Montpellier, France. Contact: Conference Secretariat, BIOCIM/IMIM, Parc Euromedicine, 34198, Montpelier Cedex 5, France.

International Association of Aquatic Animal medicine. May 11-14, 1994. Napa, CA. Contact: Brad Fenwick, Department of Veterinary Pathology, College of Veterinary Medicine, Kansas State University, Manhattan, KS 66506; (913) 532-4412.

Eastern Fish Health Workshop. May 25-28, 1994. Blacksburg, VA. Contact: Steve Smith, Department of Pathobiology, VA/MD Regional College of Veterinary Medicine, VPI, Blacksburg, VA 24601; (703) 231-5131.

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