Disease interactions between wild and cultured fish: Observations and lessons learned in the Pacific Northwest

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Abstract
This paper examines the interactions of selected endemic pathogens in fish stocks in the Pacific Northwest. In particular, case histories involving infectious hematopoietic necrosis virus (IHNV), and viral hemorrhagic septicemia virus (VHSV) will be discussed. Field observations and epidemiological studies indicate the natural hosts and reservoir of infection are wild fish populations, both salmonid and non-salmonid marine species. Salmon recovery and restoration initiatives which re-introduce or significantly increase the number of returning adult salmon into streams and rivers supplying water for hatcheries increase the threat of exposure to the natural pathogen reservoirs. Additionally, spawned-out salmon carcasses obtained at hatcheries are being utilized for nutrient enhancement of aquatic ecosystems. For hatcheries to continue their success as a tool for salmon enhancement and restoration, strategies to maintain/improve the pathogen status of hatchery water supplies need to be pursued.

Introduction
The adage “familiarity breeds contempt” seems appropriate when examining the phenomena of disease epizootics in cultured fish. Because clinical disease and mortality are easily observed and analyzed in fish populations in captivity, and not so easily observed in wild stocks, fishery managers have speculated, without supporting evidence in many cases, that cultured fish are the sources of “disease” which contaminate the natural ecosystem and contribute to the depletion of wild stock fisheries (White et al, 1995).

Over 140 state, federal, tribal, and private fish culture facilities are operated in Washington State. These hatcheries are the cornerstones for the production of salmonid smolts released into the wild for eventual harvest by commercial and recreational fishers. Salmonid hatcheries in Washington are also being successfully used as a tool to recover wild fish stocks listed as endangered or threatened under the Endangered Species Act of 1973 (ESA - 16 U.S.C. 1531 et seq.). The fish health management programs for these hatcheries are administered by the Washington Department of Fish and Wildlife (WDFW), in cooperation with Native American Treaty Tribes, National Marine Fisheries Service, United States Fish and Wildlife Service, the Washington State Department of Agriculture, and the private growers.

The salmon produced for harvest or restoration programs are reared primarily on surface water supplies - the same streams and rivers inhabited by wild salmon which are listed
under ESA. It is unknown whether these hatchery stocks amplify pathogens to the point of impacting wild stocks. Pathogens that are detected in adult salmon at spawning in the hatcheries are known to be present in wild adult salmonids. In many cases, we have evidence that naturally spawning fish impact the hatchery fish. Further complicating the task of maintaining the health status of the cultured fish is the strategy to restore the aquatic habitat to a "native" condition, regardless of the impact on an existing hatchery. Anadromous fish passage is being allowed and/or increased into hatchery water supplies that were previously free of adult salmon. Spawned-out salmon carcasses from hatcheries are being distributed into streams, which are also hatchery water supplies, for the purpose of nutrient enhancement of the aquatic ecosystem. These restoration strategies, which appear to increase the pathogen load in the hatchery water supply, pose many challenges to the fish health practitioner. In spite of unsecured water supplies, hatchery staff do have opportunities to implement fish health management strategies that are not possible for wild fish. These techniques include egg sanitation, pond cleaning and sanitation, and therapy for fish undergoing infectious disease events. Given the location of WDFW hatcheries, their water supplies, and salmon restoration initiatives, the following questions need to be answered:

1) Is there compelling evidence that disease episodes in hatcheries are the result of pathogen shedding by wild fish?
2) Is there evidence that isolations of IHNV or VHSV at hatcheries in Washington can be linked to a source of infection other than wild adult salmonids?
3) Are the disease events that have been observed in the wild the result of pathogens shed by hatchery or wild stocks?
4) Does the rearing and release of fish infected with IHNV pose an unacceptable risk to the wild fish stocks?

By examining these selected pathogens and their case histories, these questions will be answered.

The pathogens:
Infectious hematopoietic necrosis virus (IHNV) and viral hemorrhagic septicemia virus (VHSV) are both members of the rhabdovirus family. They produce similar disease signs in clinically affected fish (hemorrhage, necrosis, anemia) and exhibit similar cytopathic effect in tissue culture (Wolf, 1988). All Pacific salmon are susceptible to infection by IHNV; however, pathogenicity is highly variable between salmon species and between strains of virus. Sockeye salmon (Oncorhynchus nerka) in particular are highly susceptible to infection and all wild populations of sockeye salmon in Washington have been demonstrated to be carriers of IHNV at one or more life stages.

VHSV was not known to exist in North America prior to 1987. In that year, the virus was isolated from chinook (O. tshawytscha) and coho (O. kisutch) salmon in Washington and later confirmed as VHSV in 1988. (Winton et al., 1991). Due to the existence of this pathogen in Europe, and the import of live Atlantic salmon (Salmo salar) eggs, some presumed that the VHSV was imported to Washington via Atlantic salmon eggs. Data has since clearly demonstrated this is not the case. The VHSV most likely comes from marine baitfish such as herring. However, interesting inter-
actions have subsequently been observed with this enzootic pathogen between cultured and wild fish.

**Question 1**
Is there compelling evidence that disease episodes in hatcheries are the result of pathogen shedding by wild fish? Yes, at least in the cases of IHNV and VHSV, it appears that wild/feral adult salmon and steelhead are the reservoirs. Presented below are a few, select case histories.

**Case # 1 - Seward Park Hatchery.**
The Seward Park Hatchery is operated by the University of Washington (UW) as a research station. One of its water supplies is Lake Washington, a lake inhabited by a variety of fish species including a large population of sockeye salmon. In 1980, UW staff obtained Atlantic salmon eggs from an out-of-state source for research purposes. After hatching and some rearing, the Atlantic salmon juveniles at Seward Park underwent a disease outbreak caused by IHNV. There was no history of IHNV in the Atlantic stock prior to arrival at Seward Park nor was there IHN in any other fish on station at that time. This was the first recorded epizootic of IHN in Atlantic salmon.

**Case # 2 - Skamania Steelhead Hatchery**
The Skamania Hatchery, located on a tributary to the Columbia River, experienced a massive outbreak of IHN in steelhead trout fry (O. mykiss) in the spring of 1981. Steelhead are an anadromous form of rainbow trout. Historic testing of the brood had been negative for virus. Wild adult steelhead were passed above the hatchery weir into the water supply for the hatchery and were spawning at the time of the episode in the juveniles. In subsequent years, incubation and early rearing of fry occurred at another locale or exclusively in the pathogen-free creek supply at the hatchery. Since 1981, as long as the young fry are maintained in a water supply free of virus, epizootics do not occur.

**Case # 3 - Hoodsport chinook salmon and chum salmon with IHNV.**
The Hoodsport Hatchery is a freshwater salmon production facility with a water supply secure from adult salmon. The hatchery is located immediately adjacent to marine waters in Puget Sound. Chum, coho, and chinook salmon smolts are released each spring and adults return to the hatchery from the Pacific Ocean each fall. Prior to 1996, IHNV had never been found on the facility in juvenile or adult salmon, as evidenced by monthly health monitoring and annual viral testing of all salmon broodstocks returning to the hatchery. In 1996, “fall” chinook salmon adults tested positive at spawning. The prevalence was high - approaching 100% in five-fish pooled samples. The fall chinook salmon adults were held together in adult holding ponds. Spring chinook salmon adults spawned at the hatchery prior to the arrival of the fall chinook were negative for IHNV. A large portion of the coho (80 kidney/spleen) and chum (O. keta) salmon which spawned subsequent to the fall chinook were negative for IHNV. The chum salmon adults were sampled weekly for five weeks with 60 to 90 ovarian fluid samples tested each week. Juvenile salmon in the raceways above the adult holding pond were tested and found to be IHNV-negative. Subsequent viral testing of offspring
from positive adults were negative. No clinical disease was associated with this isolation in adult or juvenile fish. The “suspect” offspring were released in the spring of 1997. From 1997 to 1999, adult salmon returning to Hoodsport Hatchery tested negative for virus. Last October, 2000, the fall chinook adults were again found to be positive for IHNV. Last November, the chum salmon adults also tested positive. Characterization of the isolates using ribonuclease protection assay and nucleotide sequencing indicated that the two chinook isolates, 1996 and 2000, were identical to each other, but distinct from other isolates in Washington (Emmenegger and Kurath, in review). Curiously, the chum adult salmon isolate was dissimilar to the chinook isolates, but was more similar to the typical sockeye salmon isolate as observed in Lake Washington (Case #1 above). Again, no clinical IHN was observed at the site nor did other juveniles on station test positive for IHNV.

Case #4 - Adult salmon infected with VHSV.
There are multiple instances, similar to the Hoodsport Hatchery scenario, where adult salmon returning from the ocean (primarily coho salmon) to state, federal, or tribal hatcheries have been infected with VHSV (Amos et al, 1998). Typically, the prevalence of infection is very low - one or two individuals from hundreds to thousands of fish tested. The only VHSV isolation in juvenile salmon in freshwater was recorded at the Makah National Fish Hatchery in 1988. This hatchery re-circulated water which had been exposed to VHSV-infected adult salmon in hatchery raceways through juveniles in other raceways on the station. Clinical disease caused by VHSV has not been observed in Pacific salmon in a hatchery or in the wild.

Case # 5 - Atlantic salmon in marine net pens in Puget Sound, Washington
Atlantic salmon are commercially reared in marine net pens at seven sites in Washington. In 1998, fish at one site were off their feed, though abnormal mortality levels were not observed. Clinical signs in some of the moribund fish suggested an infectious agent and specimens were submitted by the attending veterinarian for histopathology and tissue culture. One of the fish was positive for VHSV. WDFW personnel were notified and conducted an investigation. Initial sampling and viral screening was conducted on moribund fish from all pens and healthy fish from the site in question. The initial testing was followed by weekly testing of all pens at the site for a four week period. No VHSV was found. All brood fish which had originated from the site had been moved to a freshwater facility prior to the VHSV isolation. All fish spawned were tested for virus and were negative. The Atlantic salmon in marine waters are continually exposed to herring and other non-salmonid marine fish which have been demonstrated to be carriers of VHSV. This observation in Washington was similar to an isolation of VHSV from Atlantic salmon in marine pens in British Columbia, Canada (Margolis, 1995).

Question 2
Is there evidence that isolations of IHNV or VHSV at WDFW or tribal hatcheries in Washington can be linked directly to a source of infection other wild adult salmonids? No.
Question 3
Are the disease events that have been observed in the wild the result of pathogens shed by hatchery or wild stocks? All the disease events observed due to IHNV or VHSV can be linked to wild stocks. There are no recorded IHN or VHS episodes in wild fish populations in Washington that can be directly linked to pathogen shedding by hatcheries. Case histories which are examples of natural episodes are listed below.

Case # 6 - Cedar River Sockeye
A naturally spawning population of sockeye salmon in the Cedar River (tributary to Lake Washington and mentioned above in Case #1) are used as a source for eggs for a traditional hatchery program. Extensive IHN studies have been conducted over the years on this salmon population (Mulcahy et al., 1983; Mulcahy et al., 1984; Amos et al., 1989; Winton, 1991). Prevalence of IHNV in the spawning adults is high towards the end of the run and has approached 100%. Up to 10 million eggs for the hatchery program are taken annually. The egg incubation/hatching water is a fish-free spring. Only one isolation of IHNV in the spawning adults is high towards the end of the run and has approached 100%. Up to 10 million eggs for the hatchery program are taken annually. The egg incubation/hatching water is a fish-free spring. Only one isolation of IHNV in hatchery-produced fry has been made in the ten years of operation of the hatchery. The unfed fry, prior to actual release into Cedar River, are transferred from the incubation units to an acclimation pond for a period of 24 hours prior to release. Water supply for the acclimation pond is Cedar River. As fry are released from the acclimation pond into Cedar River they are sub-sampled and tested for virus. So in the one instance where IHNV was isolated from hatchery fry, they very well could have been infected via the water supply from wild sockeye fry in the river. All other testing of fry lots released into Cedar River each year from the hatchery have been negative. Simultaneously, wild sockeye fry have been captured and tested as they emigrated down Cedar River to Lake Washington for rearing. Repeatedly, samples of these wild fry test positive for IHNV.

Case #6 - Pilchard in British Columbia, Canada
Massive mortality caused by VHS occurred in wild pilchard (Sardinos sagax) in 1999 near the northern portion of Vancouver Island (Traxler et al, 1999). The population of pilchard was larger than normally observed in this area. Infected herring (Clupea pallasi) were also observed in the area. Numerous non-salmonid marine species with a wide geographic range in the Pacific Northwest have been demonstrated to be natural reservoirs for VHSV.

Question 4
Does the rearing and release of salmon/steelhead infected with IHNV pose an unacceptable risk to the wild fish stocks? While the data is limited, apparently not.

Two studies recently completed suggest that IHNV shedding from a hatchery or the release of infected smolts do not pose an unacceptable risk to wild fish populations. LaPatra et al (2001) reported at the American Fisheries Society, Fish Health Section meeting this past June, that he measured a 99.99% reduction in virus titer after IHNV shed by infected fish was suspended in river water for 24 hours. Fish exposed to seeded river water following a 30 hour incubation period had negligible mortality.
Researchers in California (Foote et al, 2000) attempted to simulate the exposure a wild chinook salmon smolt would receive from an infected hatchery smolt in the process of migrating together down the Sacramento River to the Pacific Ocean. IHNV-infected hatchery fish were co-mingled with captured wild smolt in tanks which mimicked the riverine conditions. Regardless of the duration of exposure, they were unable to detect virus in the tissues of the exposed natural fish.

**Discussion:**
IHNV and VHSV are important pathogens in the Pacific Northwest because of their impact on salmonids. IHNV is important because of the mortality it causes in hatchery fish and wild stocks, alike. VHSV is important because of the mortality it causes in marine bait fish stocks and because of the concern that VHSV in the Pacific Northwest could evolve into a virulent pathogen for salmonids as it has in Europe.

While this paper has described a limited number of case histories, a common thread is found in all recorded viral-caused episodes in WDFW hatcheries. The recipe for an IHN episode is rather simple: to a densely populated susceptible fish stock, hatchery or wild, add a water supply which contains infected wild/feral adults, mix in a little stress (though not always required), then stand back and watch the disease process evolve. In virtually all the episodes of IHN in our salmon and trout hatcheries, there is the common link of exposed fish in the culture facility to a water supply which contains infected wild/feral adults. In a few, rare cases, outbreaks of IHN have occurred in recently hatched fry whose eggs were incubated in pathogen-free water but were obtained from infected adults, i.e., egg-associated transmission of IHNV.

Disease events in wild fish may not be easily observed. River currents rapidly flush mortalities downstream and aquatic and terrestrial predators quickly consume moribund fish. Observed disease outbreaks in wild salmon in Washington are typically caused by enzootic bacteria or parasites which thrive during extreme water temperatures or other stressful environmental conditions. Natural outbreaks due to IHNV and VHSV do occur and have been documented in this paper. The fact that disease events can be detected in the wild as demonstrated by these case histories is evidence they exist. In the past twenty years, numerous outbreaks of IHN have occurred in salmon and steelhead hatcheries, yet during these outbreaks, there has not been a documented case or observation of a concurrent outbreak of IHN in wild fish below the hatchery. Failure to observe this event does not mean that it hasn’t occurred, however, there is no evidence to prove that it has. The studies conducted by LaPatra (2001) and Foote (2000) give some indication that virus shed in hatchery effluent or from infected fish in a natural setting seem to rapidly decompose and pose a low risk to their wild cohorts. In the case of the adult fall chinook salmon at Hoodsport Hatchery, there was no disease associated with the IHNV isolations. It is not well understood what the reservoir of infection was for the initial isolation in 1996. It is possible that the repeat finding of IHNV in the adult fall chinook in 2000 was linked to the 1996 finding. Perhaps the IHNV persisted in the population via vertical transmission that was not detectable. Given that the juvenile chinook salmon were negative prior to
release in 1997, it is possible that the adult chinook in 2000 encountered the same IHNV marine reservoir that the adult chinook did in 1996. Clearly, as the analyses indicate (Emmenegger and Kurath, in review), the adult fall chinook were not responsible for infecting the chum salmon as they were infected with different viral genotypes. While the chum salmon adults arrive at Hoodsporo Hatchery en masse two to four weeks after the peak of chinook spawning, it is unexpected, based on conventional thinking, that the IHNV in the chum salmon was unrelated to the one in the fall chinook. This example gives further strength to the argument that there are apparently many reservoirs for IHNV in the marine environment and most of them are not well understood.

Our case histories suggest that WDFW hatcheries are not likely to cause IHN or VHS disease episodes in wild stocks in the streams and rivers. However, as management strategies for ecosystems change, risk factors may increase. The passage of large numbers of adult salmon into previously secure hatchery water supplies and/or the use of spent hatchery adults in the aquatic ecosystem for nutrient enhancement are cause for concern. It is ironic that hatcheries being used as a tool to restore endangered stocks may be jeopardizing their ability to be successful - as hatcheries increase the number of adults returning to the wild to spawn the risk level to the fish in the culture facility further increases.

Given the strategies being implemented for salmon restoration (fish passage and nutrient enhancement), what actions need to be taken for hatcheries to be successful in completing their missions? Since WDFW has a legal mandate to restore wild salmon and there is an expectation that harvest fisheries will continue in the future, WDFW needs to implement a comprehensive plan to protect hatchery water supplies from pathogens. This translates to significant capital investments to sanitize hatchery inflow and/or install intake systems which limit or prevent the entry of pathogens. By protecting the security of the hatchery water supplies, the agency can predict that disease events by viral pathogens (and other horizontally transferred pathogens) will be reduced significantly in frequency such that state and tribes will be successful in their management schemes of providing fish for harvest and restoring the depleted naturally-reproducing salmon populations.

References:


