Effect of Pollution on Fish Diseases: Potential Impacts on Salmonid Populations

MARY R. ARKOOSH*
National Marine Fisheries Service, Northwest Fisheries Science Center
Environmental Conservation Division, Hatfield Marine Science Center
2030 South Marine Science Drive, Newport, Oregon 97365, USA

EDMUNDO CASILLAS
National Marine Fisheries Service, Northwest Fisheries Science Center
Environmental Conservation Division
2725 Montlake Boulevard East, Seattle, Washington 98112, USA

ETHAN CLEMONS AND ANNA N. KAGLEY
National Marine Fisheries Service, Northwest Fisheries Science Center
Environmental Conservation Division, Hatfield Marine Science Center

ROBERT OLSON AND PAUL RENO
Oregon State University, Hatfield Marine Science Center
2030 South Marine Science Drive, Newport, Oregon 97365, USA

JOHN E. STEIN
National Marine Fisheries Service, Northwest Fisheries Science Center
Environmental Conservation Division

Abstract.—Anthropogenic factors have contributed to the precipitous decline of wild Pacific salmon stocks, although the mechanisms and processes at work are largely unknown. Pollution may be one of these factors. Sediments in estuaries are known to act as repositories for contaminants, and estuaries are important habitats for ocean- and river-migrating salmon. We have shown that juvenile salmon *Oncorhynchus* spp. and their prey bioaccumulate chlorinated hydrocarbons and aromatic hydrocarbons—important classes of toxic xenobiotics. Furthermore, we have shown that exposure to these pollutants can lead to immunosuppression and increased disease susceptibility in juvenile salmon. Whether pollution influences natural disease outbreaks in host populations, including salmon, is currently unknown. It is postulated that the occurrence of disease depends on the interaction of the host, the environment, and the pathogen. Absence of pathogens would reduce the potential for adverse environments to influence disease outbreaks. However, a recent reconnaissance survey of juvenile chinook salmon *Oncorhynchus tshawytscha* from Oregon coastal rivers revealed that pathogens were an integral component in all systems studied, although the prevalence of the pathogens varied. Furthermore, recent studies of natural fish populations have demonstrated that infectious-disease-induced mortality can significantly reduce the size of the host population. By creating adverse environments (e.g., polluted estuaries) which alter the susceptibility of the host to pathogens that are integral and ubiquitous components of the habitat, pollution increases the probability of disease-related impacts on fish populations.

Disease agents in fish populations are thought to exert important effects on host population dynamics through endo- or epizootic events. Enzootic disease can influence host abundance through long-term impacts on physiological processes affecting growth and reproduction, as well as survival, whereas epizootic diseases typically affect population dynamics by reducing populations in short-term events, which if sufficient might result in stochastic processes causing extinction (Gulland 1995). The extent to which disease organisms typically affect the population dynamics of fish is, however, largely unknown, as are the spatial and temporal scales of these events. Disease occurrence in fish and in fish populations is thought to depend on the interaction of three variables (Śnieszko 1973). These variables, controlled by abiotic, biotic, and genetic factors, in-
clude the quality of the environment, differential susceptibility of individuals to the pathogen as a result of genetic predisposition or the physiological health of individual members of the host population, and the presence and virulence of the pathogen. Modulation of any of these factors can alter the dynamics of the interaction between host and pathogen, which may in turn alter the susceptibility of fish populations to disease. As a result, disease may affect recruitment potential. Here we review our studies and examine how pollution, representing an alteration in the quality of the environment, may influence the physiological health of juvenile salmon by affecting their susceptibility to disease, which in turn may potentially alter population structure.

**Habitat Quality**

Wild Pacific salmonids have disappeared from about 40% of their historical breeding ranges (Wilderness Society 1993). Many of the remaining stocks that were once abundant have now declined precipitously (Quinn 1994). Their current status is attributed to the cumulative effects of both natural and anthropogenic factors, creating adverse environmental conditions for salmon survival. Although our understanding of how individual factors contribute to this outcome is poor, anthropogenic factors are considered to have contributed significantly to declines in various salmon populations (Nehlsen et al. 1991; NRC 1996). Pollution, a well-recognized anthropogenic factor known to adversely affect environmental quality, has the potential to affect critical life-history stages of salmon.

Estuaries are a very important habitat for various life history stages of salmon as they serve as the natural linkage for salmon migrating between freshwater and ocean environments. Because of the urbanization of many coastal regions, estuaries located near urban centers receive chemical contaminants via direct pipeline discharges from coastal communities and from ships, rivers, atmospheric deposition, and nonpoint source runoff (Kennish 1992). Many of the chemicals from these sources accumulate in estuarine sediments (McCain et al. 1988). A large number of these chemicals are also bioaccumulated by sediment-dwelling organisms (McCain et al. 1990), including amphipods and copepods, which are important prey organisms for downstream migrant juveniles of certain salmonid species (J. W. Nicholas and H. V. Lorz, Oregon State University, unpublished data). We have demonstrated that juvenile fall chinook salmon Oncorhynchus tshawytscha bioaccumulate significant concentrations of chemical contaminants during their relatively short residence time in the estuary, primarily through exposure from their diet. In studies of juvenile chinook salmon from urban estuaries in Puget Sound, we found high levels of polycyclic aromatic hydrocarbons (PAHs) and polychlorinated biphenyls (PCBs) or their metabolites in stomach contents and tissues of these fish (McCain et al. 1990; Stein et al. 1995). These findings revealed that pollution in urban estuaries has the potential to adversely influence the health, and ultimately the survival, of juvenile salmon in the estuary or subsequently in the ocean. Determining if contaminants can alter the health and survival of salmon became the focus of our studies.

**Host Susceptibility**

Immune dysfunction in mammals has been recognized as a sublethal effect of chemical contaminant exposure, affecting both cellular and humoral aspects of the immune system (Dean et al. 1990). When we began our research on immune dysfunction in Puget Sound, laboratory studies had extended these findings by demonstrating that the fish immune system is susceptible to specific contaminants, often resulting in a wide array of immunosuppressive effects (McLeay and Gordon 1977; Arkoosh and Kaattari 1987; Rice and Weeks 1989; Thuvander 1989). Field studies focusing on the effects of environmental exposure to chemical contaminants on the primary and secondary humoral responses of fish had, however, not been previously performed.

A number of immune parameters can be examined in fish (Sindermann 1990). In our studies with juvenile salmon, immunocompetence was evaluated by analyzing the functional ability of B-leukocytes to produce an in vitro primary and secondary plaque-forming cell (PFC) response to a T-independent antigen, trinitrophenyl–lipopolysaccharide (TNP–LPS), and to a T-dependent antigen, TNP–keyhole limpet hemocyanin (TNP–KLH). This technique provides a very sensitive approach to evaluating the chronic effects of short-term contaminant exposure (Arkoosh and Kaattari 1987). We showed that leukocytes of juvenile salmon collected from hatcheries and from a non-urban estuary were able to generate a significantly higher secondary PFC response to a foreign antigen than primary PFC response, which is the normal and expected response (Arkoosh et al. 1991). However, an enhanced secondary PFC response
did not occur with leukocytes of juvenile salmon exposed to pollution from an urban estuary (Figure 1). Although immunosuppression was observed in individuals, projection of this effect through modeling has demonstrated that the ability to mount an immune response not only affects the individual but also has varying and depressing impacts on population dynamics (Norman et al. 1994).

Describing pollution-induced immunosuppression lays a foundation for understanding how an anthropogenically altered environment creates the potential for reduced survival of salmon populations. As illustrated by Snieszko (1973), host susceptibility, presence and virulence of the pathogen, and environmental conditions all influence the occurrence of disease (Figure 2A). Accordingly, given the suppressive effect of adverse environmental conditions (e.g., chemical contaminants) on the immune system, juvenile salmon from polluted environments may be more susceptible to disease than those from nonpolluted environments (Figure 2B). This increased potential for disease may reduce the size of a chronically exposed fish population. To demonstrate that salmon from a contaminated environment may be more susceptible to disease, we collected juvenile fall chinook salmon from urban and nonurban estuaries and from the respective releasing hatcheries. We then exposed them in the laboratory to the marine pathogen *Vibrio anguillarum*, a facultative fish pathogen that is known to cause disease when fish are under stress (Warren 1991). We found that juvenile chinook salmon from the contaminated estuary were more susceptible to mortality due to *V. anguillarum* than fish from the corresponding hatchery (Figure 3). In contrast, juvenile fall chinook salmon from the nonurban estuary showed no greater susceptibility to *V. anguillarum*-induced mortality than the fish from the corresponding hatchery. The results of these initial disease challenges indicate that, in juvenile chinook salmon from an urban estuary, contaminant-associated immunodysfunction appears to be associated with an increased susceptibility to pathogenesis by a virulent marine bacterium (Arkoosh et al., in press).

Both PAHs and PCBs are known to induce immunosuppression as well as to increase disease susceptibility in mammals (Ward et al. 1985; Dean et al. 1990). This suggests that either PCBs, PAHs, or both may be responsible for a suppressed PFC response to an antigen as well as increasing mortality over time due to *V. anguillarum* in juvenile salmon from the contaminated urban estuary. This hypothesis is supported by the results of recent laboratory studies. Juvenile chinook salmon were administered sublethal doses of PAHs and PCBs. The exposed fish exhibited suppression of their primary and secondary PFC response to the antigen, TNP-LPS, (Arkoosh et al. 1994) as well as an increase in disease susceptibility (Arkoosh, unpublished data). Thus, the linkage between an impaired secondary PFC response (Figures 1, 4) and an increase in disease susceptibility (Figures 3, 5) has been demonstrated in both field and laboratory studies, supporting a putative causal relationship between impaired immunity, increased disease susceptibility, and chemical contaminant exposure in juvenile fall chinook salmon from polluted urban estuaries. Conceptually, documenting the relationship between anthropogenically created adverse environmental conditions (polluted estuaries) and altered host health (immunosuppressed and increased disease susceptibility) fulfills modulation of two of the three of the factors proposed by Snieszko (1973) that can influence (increase) the development of disease in wild populations. Contributions from the third factor, the presence and influence of pathogens, became the focus of our research in the past year.

**Pathogen Distribution and Host Infection**

Snieszko (1973) suggested that perturbations in the environment may increase the potential for disease in fish populations if pathogens are present. From a pragmatic point of view, although changing
environmental quality and altering host health and susceptibility are potential concerns, the absence of pathogens dramatically decreases the probability of debilitating epizootic- or enzootic-related reductions in population size. Therefore, we have initiated studies to document the occurrence of salmonid pathogens in various watersheds in the Pacific Northwest. To gain a better understanding of the distributional patterns of salmon pathogens, a reconnaissance survey was conducted on outmigrating juvenile chinook salmon in coastal rivers and estuaries of Oregon. Outmigrating juvenile chinook salmon were collected from the Salmon River, Alsea Bay, Elk River, Coos Bay, and Coquille River during August through October 1996 (Figure 6). Salmon were collected from the estuaries with a 37-m beach seine net. Once the fish were collected they were transported in aerated tanks to the Hatfield Marine Science Center in Newport, Oregon. The number of fish examined from each of the estuaries for the presence of specific pathogens ranged from 39 to 110 individuals.

The presence of specific disease organisms of interest was determined by use of diagnostic procedures (Thoesen 1994; Noga 1996). Briefly, these tests included using a fluorescent antibody test to detect *Renibacterium salmoninarum*, isolating *Aeromonas salmonicida* and *V. anguillarum* from the kidney by plating onto trypticase soy agar (TSA) and thiosulfate–citrate–bile salts–sucrose (TCBS) agar, respectively, and detecting infectious hematopoietic necrosis virus (IHNV), infectious pancreatic necrosis virus (IPNV), and viral hemorrhagic septicemia virus (VHSV) in cell culture and erythrocytic inclusion body syndrome (EIBS) virus and erythrocytic necrosis virus (ENV) in blood smears. The presence or absence of the parasites *Ceratomyxa shasta, Nanophyetus salmincola,*
Sanguinicola sp., and Cryptobia salmositica was determined by microscopic examination. Certain pathogens were present in fish from all the Oregon coastal watershed sampled, although the prevalences of the pathogens varied (Figure 7). The three most prevalent pathogens infecting juvenile chinook salmon from Oregon coastal rivers were *N. salmincola*, *R. salmoninarum*, and an erythrocyte cytoplasmic virus, which could have been either ENV or EIBS (Arkoosh, unpublished data). *Renibacterium salmoninarum* is the causative agent of bacterial kidney disease (BKD), which is a chronic and often lethal disease in salmon. *Nanophyetus salmincola* is a digenetic trematode that can cause exophthalmia, erratic swimming, blockage of blood vessels, kidney damage, and death when fish become heavily infected (Warren 1991). The erythrocyte cytoplasmic virus (either ENV or EIBS) can debilitate the fish so that other pathogens or adverse environmental conditions can cause death (Thoesen 1994). The prevalence of *N. salmincola* was 70% or greater and ranged as high as 100% in juveniles from the various watersheds of Oregon’s coastal rivers. The intensity of infection ranged from 1 to 175 metacercariae per kidney. Similarly, *R. salmoninarum* was detected in salmon from all sites examined except the Elk River estuary, although it was found to occur in juveniles from the hatchery that release salmon into this watershed. The intensity of infection ranged from 1 to 12 positive microscopic fields per 50 microscopic fields examined. In contrast to the prevalence of *N. salmincola* infection, the prevalence of *R. salmoninarum* in juvenile chinook salmon was lower and ranged from 0% to 12%. This lower prevalence does not inherently minimize the potential contribution of BKD to affect salmon populations relative to the high prevalence for *N. salmincola*, because BKD is a well-recognized debilitating disease of salmon (Evelyn et al. 1973; Fryer and Sanders 1981; Fryer and Lannan 1993). The prevalence of the erythrocyte cytoplasmic virus in the salmon ranged from 0%
to 21%. The virus was not detected in fish from the Elk River estuary. Although the prevalence and intensity of pathogens varied, results of this reconnaissance survey revealed that pathogens are integral and ubiquitous components of the river and estuarine habitat and, if environmental conditions are suitable, there is a potential for disease to occur within the population.

The presence or absence of pathogens in various watersheds has been confirmed by other investigators, although the variety of watersheds evaluated has typically been more limited. For example, Bartholomew et al. (1990) detected C. shasta in up to 24% of salmon sampled in the Columbia River. Similarly, D. G. Elliott and R. J. Pascho (U.S. Geological Survey, unpublished data) determined the prevalence of R. salmoninarum to be between 13% and 100% in Snake River salmon with 64% of the samples having a prevalence greater than 50%. As in our study, Sanders et al. (1992) determined that approximately 20% of migrating hatchery and wild salmonids (Oncorhynchus spp.) collected in the Columbia River were infected with R. salmoninarum. Also, H. M. Engelking and J. Kaufman (Oregon Department of Fish and Wildlife, unpublished data) found that wild fall chinook salmon from several areas of Oregon had a high prevalence of IHNV. These studies confirm that pathogens are present in salmon populations from various watersheds and that some salmon populations have a higher prevalence of pathogens than do other populations.

The presence of pathogens, as previously stated, is not sufficient to lead to disease or dysfunction. When assessing the effects of contaminants on salmon health, it is important to understand the other factors that regulate the transition from simply harboring a pathogen to actual disease or dysfunction. What regulates this transition in a salmon population may vary with different pathogens present in a contaminated environment and with individual fish within a population (reviewed in Schreck 1996). Adverse environmental conditions that weaken the host defenses in the presence of pathogens, however, allow increased opportunities for enzootic or epizootic disease processes to affect populations.

Population Impacts of Disease

The objective of our investigations is to identify mechanisms and processes that can cause disease to have a critical influence on the size and structure of fish populations. The possibility that disease can affect fish populations is gaining acceptance and visibility worldwide. Gulland (1995) described the
available evidence that infectious disease influences viability of wild populations. Although this process is often discussed and recognized, thorough documentation of these events is minimal in the literature. The reality, though, is that disease can significantly affect population numbers in many species, including fish. Patterson (1996) recently described a fungal parasite *Ichthyophonus hoferi*, that significantly reduced the survival of an exploited population of Atlantic herring *Clupea harengus*. Furthermore, Sanders et al. (1996) described a natural epizootic due to VHSV that dramatically reduced the size of a population of Pacific herring *C. pallasi* in Prince William Sound, Alaska. In this latter case, the epizootic was speculated to result from exposure of the population to oil spilled from the *Exxon Valdez*, which is consistent with the premise that altered environmental conditions may influence disease epizootics.

**Conclusion**

The findings reviewed here indicate that juvenile fall chinook salmon from polluted estuaries (altered environments) are immunosuppressed (altered hosts) and are more susceptible to disease than those from less-polluted waters. Evidence is provided linking the presence of elevated levels of complex mixtures of chemical contaminants in polluted estuaries to the reduced health and survival of juvenile chinook salmon. The potential for disease in contaminant-exposed salmonid populations may be great if the fish are exposed to pathogens. Studies have shown that pathogens are a ubiquitous and integral component of most wa-
tersheds. This suggests that disease is a natural occurrence contributing to the regulation of populations of fish, including salmon, and that anthropogenically induced factors may significantly shift the balance between salmon survival and mortality due to disease. Our observations highlight the role that contaminant-induced disease may have on salmon stocks. The challenge is documenting such occurrences in natural populations. Further evaluation of the potential impact of various abiotic and biotic factors, including contamination, on disease processes could provide additional insights into estuarine and early ocean survival of juvenile salmon.

References


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