

POTENTIAL IMPACT OF *RENIBACTERIUM SALMONINARUM* ON ADULT RETURNS OF SNAKE RIVER SPRING/SUMMER CHINOOK SALMON

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ABSTRACT

Snake River spring/summer chinook salmon populations declined rapidly during the mid- to late 1970s after construction of four lower Snake River dams. Considerable efforts followed to improve the direct survival of downstream-migrating juvenile smolts through these four dams, and through four additional dams on the lower Columbia River. Presently, although survival of downstream migrants is now as high or higher than it was prior to construction of the Snake River dams, adult return rates remain low. Therefore, other factors that have increased mortality compared to pre-dam conditions must act on the fish in the lower Columbia River estuary or in the ocean. One hypothesis is that *Renibacterium salmoninarum*, a bacterium found in nearly all Snake River chinook salmon and the causative agent of bacterial kidney disease (BKD), is responsible for decreased adult returns. This hypothesis assumes that the disease is triggered by stress associated with bypass systems at dams or during the smolt transportation process. In a related hypothesis, hatchery fish with high levels of *R. salmoninarum* intermingle with wild fish and spread infections, which result in increased mortality. A final hypothesis is that decreased ocean productivity may account for the low chinook salmon returns.

Key words: chinook salmon, disease, survival, Snake River

The Columbia River watershed historically produced more chinook salmon (*Oncorhynchus tshawytscha*) than any other river system in the world (Netboy, 1980), with the majority of spring chinook salmon originating in the Snake River basin (Fulton, 1968) (Figure 1). In the early 1880s, spring and summer chinook salmon provided commercial fisheries in the lower Columbia River with average annual catches of 17.7 million kg (Craig and Hacker, 1940). Heavy exploitation by these fisheries, however, caused a substantial depletion of the dominant summer stock; the fisheries then concentrated on the spring and fall stocks (Craig and Hacker, 1940; Gangmark, 1957). Summer chinook salmon populations from the mid- and upper Columbia River continued to decline such that by 1964, the inriver commercial fishery for all summer fish was closed. By this time, Snake River basin spring/summer chinook salmon accounted for approximately 78% of the remaining upper river populations (Fulton, 1968).

In addition to overfishing, further causes of stock de-

clines in the early years were habitat destruction and damming of tributaries for water withdrawal and small-scale hydropower (Craig and Hacker, 1940). Populations of wild Snake River spring/summer chinook salmon continued to decrease coincident with construction of hydroelectric dams on the lower Snake and Columbia rivers (Raymond, 1988; Williams, 1989). Smolt-to-adult return rates of these populations fell from greater than 4% in the mid- to late 1960s, when only four dams were in place, to generally less than 2% during the 1970s after seven or eight dams were in operation (Figure 2). This large additional decrease in adult return rates coincided with substantially reduced juvenile survival (Raymond, 1979) with concurrent high levels of descaling and injury to fish (Williams and Matthews, 1995). As a consequence of low returns, harvest rates of 40-60% were reduced to less than 10%.

To address the low adult returns, two major directions were pursued: (1) changes to hydropower system operation

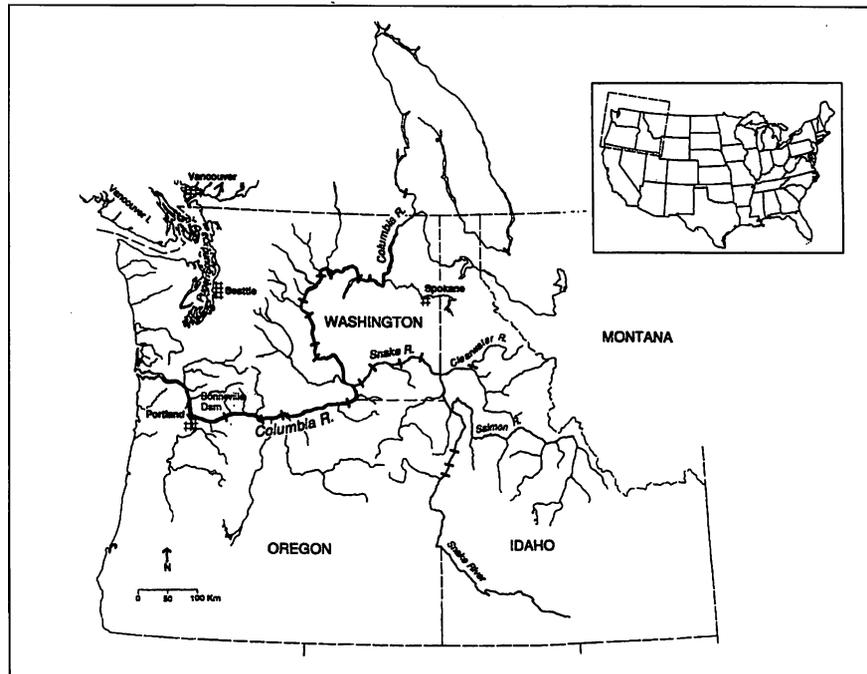


Figure 1. Map of the Columbia River basin.

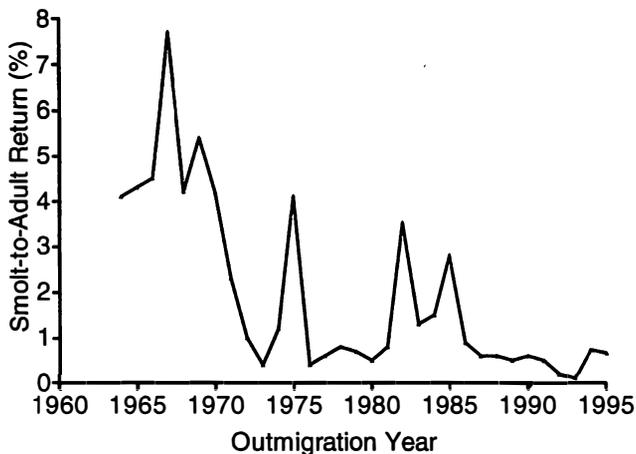


Figure 2. Smolt to adult returns of wild Snake River spring/summer chinook salmon.

and the construction of fish-handling and bypass structures at dams to increase survival of juvenile migrant fish, and (2) construction of fish hatcheries to increase smolt production. Major changes in hydropower system operation included increases in river flow, removal of debris from forebays at dams, more efficient and continuous turbine operation, and installation of "flip-lips" on spillways to decrease atmospheric gas supersaturation. Considerable resources were invested in the construction of facilities to bypass juvenile fish away from turbines, and this construction was followed by extensive evaluation and modifications to bypass facilities that were found inadequate (Williams, 1989; Williams and Matthews, 1995). Further,

barges were constructed to transport fish collected at upper dams to a release site below Bonneville Dam. As a result of these efforts, survival of juvenile fish that migrate downstream through the hydropower system is now as high as it was prior to construction of the dams (Figure 3) (Williams *et al.*, 2001) (Figure 3), and direct survival of fish transported around the hydropower system is nearly 100%. Additionally, Schreck and Stahl (1998) found that survival was greater than 90% for fish migrating between the tailrace of Bonneville Dam to near the mouth of the Columbia River. Surprisingly, these substantial improvements in downstream migrant smolt survival have not led to concurrent increases in adult return rates of chinook salmon.

The continued low adult return rates, but relatively high freshwater smolt survival, indicate that factor (s) limiting survival to adulthood are those that act on fish during ocean residence. Tremendous controversy exists over the possible factor (s) and the mechanism (s) that limit adult returns (Schaller *et al.*, 1999; Anderson, 2000; Zabel and Williams, 2000). The following different factors are hypothesized to have caused increased ocean mortality: (1) increased rates of bacterial kidney disease (BKD) caused by *Renibacterium salmoninarum* infections contracted during hydropower system passage, or exacerbated by stress from passage; (2) hatchery overproduction which has increased competition for food, stress levels, or transmission of *R. salmoninarum*; and (3) changed ocean conditions leading to changes in predator/prey dynamics. However, empirical evidence to support these hypotheses is lacking.

In the late 1980s, Raymond (1988) and Williams (1989)

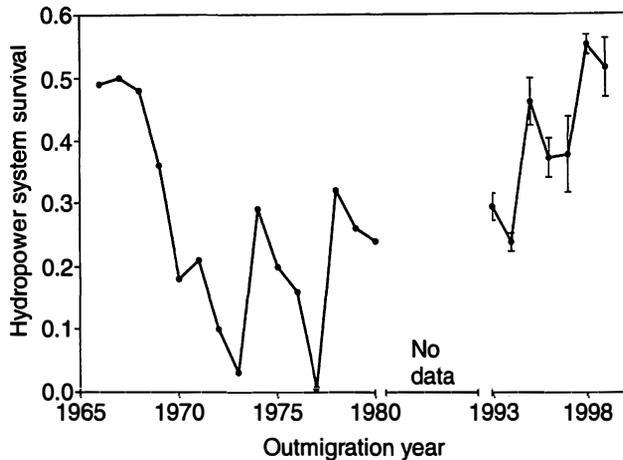


Figure 3. Estimated survival of juvenile Snake River spring/summer chinook salmon through the lower Snake and lower Columbia river hydropower system. Four dams were in place in 1966-67, five in 1968, six in 1969, seven in 1970-74, and eight from 1975 to present.

hypothesized that a likely cause of decreased adult returns was an increased loss of fish from BKD. Bacterial kidney disease is a chronic, systemic disease present in salmonid stocks from virtually every river basin draining to the Northeast Pacific Ocean and is considered more severe in spring/summer chinook salmon than in other salmonids (Bullock and Wolf, 1986). Kent *et al.* (1998) in a survey of salmonid pathogens in catch from ocean fisheries in British Columbia found that 44 of 77 chinook salmon tested positive for *R. salmoninarum*, and this infection rate was substantially greater than that of any other salmonid species.

Most Snake River chinook salmon harbor *R. salmoninarum*. Using the enzyme immunoassay (ELISA), Maule *et al.* (1996) found an *R. salmoninarum* infection rate of nearly 100% at some Snake River chinook salmon hatcheries in 1988, with a mean infection rate of 68% measured between 1988 and 1992. In contrast, the mean infection rate of migrant fish monitored at Lower Granite Dam was 91%, suggesting that either *R. salmoninarum* infections progressed during migration, or that wild fish with higher infection levels increased the average infection rate in samples taken at Lower Granite Dam. From 1988-1991, Elliot *et al.* (1997) sampled migrant smolts at Lower Granite Dam and, based on ELISA techniques, determined that 86-100% of the fish were infected with *R. salmoninarum*, with a 100% infection rate in all wild fish samples. Although a high percentage of the fish were infected, the infection levels were considered low. Vanderkooi and Maule (1999) continued hatchery monitoring from 1993-1996 and found that *R. salmoninarum* infections fluctuated between high and low levels, but levels of infections were considered low in greater than 90% of the fish sampled.

Although *R. salmoninarum* infection rates are high in

chinook salmon throughout the Columbia River basin, clinical BKD does not necessarily follow. Bakke and Harris (1998) suggested *R. salmoninarum* exists normally in the gut of all wild and hatchery Atlantic salmon, but at times of stress migrates to the kidneys and causes BKD. In chinook salmon, increases in stress are presumptively necessary for most cases of BKD to occur, as susceptibility to disease in fish increases under stressful conditions (Maule *et al.*, 1989; Sunyer *et al.*, 1995; Bakke and Harris, 1998; Van Muiswinkel *et al.*, 1999).

Yearling chinook salmon smolts from the Snake River experience elevated levels of stress, as measured by blood chemistry, when they are routed through juvenile bypass systems or loaded into barges for transportation (Bjornn *et al.*, 1985; Bjornn and Congleton, 1987). Some of the increased stress may result from passing through flumes or pipes in small volumes of water. Wild fish may experience elevated stress levels from interactions with high concentrations of larger hatchery fish. To evaluate the potential impacts of stress on the longterm survival of yearling chinook salmon, Park *et al.* (1986) conducted an extended seawater holding experiment in 1985. They sampled juveniles from several areas in a juvenile bypass system and from barges after loading, subsequently holding the fish for 141 days. Subsamples from the groups indicated a range of stress response. The major cause of mortality (range 60-75% from the least to the most stressed groups, respectively) was BKD. High levels of *R. salmoninarum* were found in all of the dead fish, but survivors had relatively low levels of infection.

In addition to experiencing direct mortality, Mesa *et al.* (1998) found that fish experimentally infected with *R. salmoninarum* were more susceptible to predation. In the latter stages of BKD, Mesa *et al.* (1999) observed higher levels of stress (measured through blood chemistry) which they attributed to the disease itself. They also suggested that the smoltification process may have triggered BKD. In both instances, they caution that their results were derived from laboratory experiments and may not apply in the wild. Further, BKD infections acquired in freshwater may impair the ability of salmonid smolts to acclimate to seawater, and entry into seawater may actually accelerate mortality among infected fish (Fryer and Sanders, 1981; Banner *et al.*, 1983; Moles, 1997).

R. salmoninarum is slow-acting and, therefore, mortalities due to BKD are likely to occur after fish reach the ocean. Banner *et al.* (1983) found that for chinook salmon, the percentage of mortality attributable to BKD increased the longer fish were held in seawater tanks. Spring chinook salmon held 200 days had mortalities ranging from 45-81%. Thus, it is not surprising that few mortalities during downstream migration are attributable to BKD. Further, since most chinook salmon juveniles (wild and hatchery) apparently harbor *R. salmoninarum* and the disease is transmitted both vertically and horizontally, removal of

the organism from the population appears unlikely. In fact, past attempts to treat or control hatchery infection levels through the use of antibiotics may have caused more harm than good, as *R. salmoninarum* is found intracellularly and antibiotics only inhibit its growth extracellularly (Bullock and Wolf, 1986). Keeping fish alive in the hatchery with antibiotics may not decrease subsequent BKD-related mortality. On the contrary, antibiotics may allow highly infected fish to live, at least temporarily after release from the hatchery, which may allow them to shed more bacterium and increase the probability of keeping a high disease incidence in the population.

A better solution appears related to segregation of adult spawners by disease level. Maule *et al.* (1996) attributed the decreased infection rates at hatcheries between 1988-1992 to hatchery practices that culled eggs from adult female spawners with high *R. salmoninarum* infections, segregation of eggs by severity of infection for the remainder of the brood, and reduced rearing densities. Segregation of eggs by severity of infection was implemented based on research by Elliot *et al.* (1995). Elliot *et al.* (1995) held juvenile spring chinook salmon in seawater net pens for 98 days and found a 12% mortality for fish with low infection levels (based on optical densities and ELISA techniques) and a 44% mortality for fish with high infection levels. Nearly 85% of fish in the high infection group were determined to have *R. salmoninarum* infections based on FAT.

Since most Snake River chinook salmon likely carry *R. salmoninarum* and experience increased levels of stress during their downstream migration, this increased stress may decrease immune system response. Thus, the hypothesis that BKD likely is responsible for decreased adult returns is viable. Nonetheless, direct empirical evidence linking hydropower-system passage to increased BKD mortality is lacking. But because wild Snake River chinook salmon stocks have not rebounded since changes were made to improve hydropower system passage, continued efforts are needed to address the problem. Eliminating all stressful conditions encountered by migrants within the hydropower system does not appear feasible as most fish will experience some stresses at bypass systems or during the transportation process. Further, periodic regime shifts in the ocean will continue to occur. The impact of hatchery fish on wild fish is unknown, but it is important to limit the extent to which disease transmission might play a role in decreased adult returns. It is unlikely that elimination of *R. salmoninarum* from hatchery or wild fish is possible. Thus, it is critical that hatcheries take all measures possible to eliminate releases of individual fish with high infection levels.

Data to resolve the controversy about causes for continued low chinook salmon returns will not likely become available soon. Meanwhile, it seems prudent to continue evaluating impacts of the hydropower system and

following hatchery rearing practices that reduce horizontal transmission of *R. salmoninarum* to the greatest extent possible, both within hatcheries and after fish are released.

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REFERENCES

- Anderson, J. J. (2000): Decadal climate cycles and declining Columbia River salmon, In "Sustainable fisheries management: Pacific salmon" (ed. By E. E. Knudson, C. R. Steward, D. D. MacDonald, J. E. Williams, and D. W. Reiser). CRC Press, Boca Raton, FL. pp. 467-484.
- Bakke, T. A. and P. D. Harris (1998): Diseases and parasites in wild Atlantic salmon (*Salmo salar*) populations. *Can. J. Fish. Aquat. Sci.*, 55 (Suppl. 1), 247-266.
- Banner, C. R., J. S. Rehovec, and J. L. Fryer (1983): *Renibacterium salmoninarum* as a cause of mortality among chinook salmon in salt water. *J. World Maricult. Soc.*, 14, 236-239.
- Bjornn, T. C. and J. L. Congleton (1987): Survival of chinook salmon smolts as related to stress at dams and smolt quality. Tech. Rep. 87-4, Idaho Cooperative Fish and Wildlife Research Unit, Moscow, ID. 23 p.
- Bjornn, T. C., J. L. Congleton, R. R. Ringe, and C. M. Moffitt (1985): Survival of chinook salmon smolts as related to stress at dams and smolt quality. Tech. Rep. 85-1, Idaho Cooperative Fish and Wildlife Research Unit, Moscow, ID. 25 p.
- Bullock, G. L. and K. Wolf (1986): Infectious diseases of cultured fishes: current perspec U.S. Fish Wildl. Serv., Wildl. Leaflet No.5.
- Craig, J. A. and R. L. Hacker (1940): The history and development of the fisheries of the Columbia River. *U.S. Fish Wildl. Serv., Fish. Bull.*, 32, 133-216.
- Elliot, D. G., R. J. Pascho, and A. N. Palmisano (1995): Brood stock segregation for the control of bacterial kidney disease can affect mortality of progeny chinook salmon (*Oncorhynchus tshawytscha*) in seawater. *Aquaculture*, 132, 133-144.
- Elliot, D. G., R. J. Pascho, L. M. Jackson, G. M. Matthews, and S. Achord (1997): *Renibacterium salmoninarum* in spring/summer chinook salmon smolts at dams on the Columbia and Snake Rivers. *J. Aquat. Anim. Health*, 9, 114-126.
- Fryer, J. L. and J. E. Sanders. (1981): Bacterial kidney disease of salmonid fish. *Annu. Rev. Microbiol.*, 35, 273-298.
- Fulton, L. A. (1968): Spawning areas and abundances of chinook salmon (*Oncorhynchus tshawytscha*) in the Columbia River basin—past and present. U. S. Fish Wildl. Serv., Spec. Sci. Rep. No.571.
- Gangmark, H. A. (1957): Fluctuations in abundance of Columbia River chinook salmon 1928 U.S. Fish Wildl. Serv., Spec. Sci. Rep. No.189.
- Kent, M. L., G. S. Traxler, D. Kieser, J. Richard, S. C. Dawe, R. W. Shaw, G. Prospero-Porta, J. Ketcheson, and T. P. T. Evelyn (1998): Survey of salmonid pathogens in ocean-caught fishes in British Columbia, Canada. *J. Aquat. Anim. Health*, 10, 211-219.
- Maule, A. G., R. A. Tripp, S. L. Kaattari, and C. B. Schreck (1989): Stress alters immune function and disease resistance in chinook salmon (*Oncorhynchus tshawytscha*). *J. Endocrinol.*, 120, 135-142.
- Maule, A. G., D. W. Rondorf, J. Beeman, and P. Haner (1996):

- Incidence of *Renibacterium salmoninarum* in infections in juvenile hatchery spring chinook salmon in the Columbia and Snake rivers. *J. Aquat. Anim. Health*, **8**, 37-46.
- Mesa, M. G., T. P. Poe, A. G. Maule, and C. B. Schreck (1998): Vulnerability to predation and physiological stress responses in juvenile chinook salmon (*Oncorhynchus tshawytscha*) experimentally infected with *Renibacterium salmoninarum*. *Can. J. Fish. Aquat. Sci.*, **55**, 1599-1606.
- Mesa, M. G., A. G. Maule, T. P. Poe, and C. B. Schreck (1999): The influence of bacterial kidney disease on smoltification in salmonids: is it a case of double jeopardy? *Aquaculture*, **174**, 25-41.
- Moles, A. (1997): Effect of bacterial kidney disease on saltwater adaptation of coho salmon smolts. *J. Aquat. Anim. Health*, **9**, 230-233.
- Netboy, A. (1980): The Columbia River salmon and steelhead trout. Univ. Washington Press, Seattle. 180 p.
- Park, D. L., G. M. Matthews, T. E. Ruehle, J. R. Harmon, E. Slatick, and F. J. Ossiander (1986): Evaluation of transportation of juvenile salmonids and related research on the Columbia and Snake rivers, 1985. Report to Walla Walla District Corps of Engineers, Walla Walla, WA, 52 p. (Available from U.S. National Marine Fisheries Service, Northwest Fisheries Science Center, 2725 Montlake Blvd. E., Seattle, WA 98112.)
- Raymond, H. L. (1979): Effects of dams and impoundments on migrations of juvenile chinook salmon and steelhead from the Snake River, 1966 to 1975. *Trans. Am. Fish. Soc.*, **108**, 505-529.
- Raymond, H. L. (1988): Effects of hydroelectric development and fisheries enhancement on spring and summer chinook salmon and steelhead in the Columbia River basin. *N. Am. J. Fish. Manage.*, **8**, 1-24.
- Schaller, H. A., C. E. Petrosky, and O. P. Langness. (1999): Contrasting patterns of productivity and survival rates for stream-type chinook salmon (*Oncorhynchus tshawytscha*) populations of the Snake and Columbia rivers. *Can. J. Fish. Aquat. Sci.*, **56**, 1031-1045.
- Schreck, C. B. and T. P. Stahl (1998): Evaluation of migration and survival of juvenile salmonids following transportation. Report to Walla Walla District Corps of Engineers, Walla Walla, WA, 49p. (Available from Oregon Cooperative Fish and Wildlife Research Unit, 104 Nash Hall, Oregon State Univ., Corvallis, OR 97331-3803.)
- Sunyer, J. O., E. Gómez, V. Navarro, J. Quesada, and L. Tort. (1995): Physiological responses and depression of humoral components of the immune system in gilthead sea bream (*Sparus aurata*) following acute stress. *Can. J. Fish. Aquat. Sci.*, **52**, 2339-2346.
- Van Muiswinkel, W. B., G. F. Wiegertjes, and R. J. M. Stet (1999): The influence of environmental and genetic factors on the disease resistance of fish. *Aquaculture*, **172**, 103-110.
- Vanderkooi, S. P. and A. G. Maule (1999): Prevalence of *Renibacterium salmoninarum* in juvenile spring chinook salmon of Columbia and Snake River hatcheries, 1993-1996. *J. Aquat. Anim. Health*, **11**, 162-169.
- Williams, J. G. 1989. Snake River spring and summer chinook salmon: can they be saved? *Regul. Rivers Res. & Manage.*, **4**, 17-26.
- Williams, J. G. and G. M. Matthews (1995): A review of flow and survival relationships for spring and summer chinook salmon, *Oncorhynchus tshawytscha*, from the Snake River basin. *U. S. Fish Wildl. Serv., Fish. Bull.*, **93**, 732-740.
- Williams, J. G., S. G. Smith, and W. D. Muir (2001): Survival estimates of downstream migrant yearling juvenile salmonids through the Snake and Columbia river hydropower system, 1966-1980 and 1993-1999. *N. Am. J. Fish. Manage.*, **21**, 310-317.
- Zabel, R. W. and J. G. Williams (2000): Comments on "Contrasting patterns of productivity and survival rates for stream-type chinook salmon (*Oncorhynchus tshawytscha*) populations of the Snake and Columbia rivers by Schaller et al. (1999)." *Can. J. Fish. Aquat. Sci.*, **57**, 1739-1741.

