

## Submission 20

### BKD Extra Mortality Hypothesis (EM2)

C. Petrosky

State and Tribal Fisheries Agencies, August 11, 1998

---

The “BKD” or Stock Viability Hypothesis proposes that 1) the viability of Snake River stocks declined as a direct or indirect result of the hydropower system in the 1970s; 2) current extra mortality is not related to either the hydrosystem or climate conditions; and 3) extra mortality is here to stay, even if hydrosystem extra mortality is reduced and/or the climate improves. One hypothesis to account for decreased extra mortality is that hatchery programs implemented after construction of Snake River dams increased either the incidence in the level of bacterial kidney disease (BKD) within the wild population or its severity. In both cases, mortality increased in juvenile fish after they exited the hydropower system as compared to earlier years (or as compared to downstream stocks for the same time period).

Note that this hypothesis implies some *selective mortality specific to Snake River stocks*, without supporting empirical evidence. The draft WOE report states (Section 4.2.3, page 92) that “it makes sense to replace this hypothesis with the much better elaborated hatchery hypothesis EM4”. Even so, many of the elements of this hypothesis have been carried into the hatchery hypothesis. Our comments on this extra mortality hypothesis are included in the more recently submitted hatchery extra mortality hypothesis (Appendix 19).

Fish health staff at the Idaho Department of Fish and Game (IDFG) addressed the fish health and stress aspects of this hypothesis, with particular reference to spatial patterns hypothesized above and stressors associated with the hydropower system. Their issue paper (IDFG 1998) is duplicated below in this PATH appendix. The authors intend to develop this issue paper for journal publication. Copies of the Report to the Director and appendices can be obtained from IDFG, Fisheries Bureau, Box 25 600 S. Walnut St, Boise, Idaho 83707.

### Literature Cited

IDFG (Idaho Department of Fish and Game). 1998. Issue Paper: Fish health and stress related to transportation. Appendix 3.3 in: IDFG (1998). Idaho’s anadromous fish stocks: Their status and recovery options. Report to the Director. May 1, 1998. IDFG 98-13. Idaho Department of Fish and Game, 600 S. Walnut, Boise, Idaho 83707.

Issue Paper: Fish Health and Stress Related to transportation  
Fisheries Bureau, IDFG, May 1, 1998

*Does fish health suffer from the stress related to transportation?*

CONCLUSION: Collection and barging through the upper Snake and Columbia River corridor represents the largest fish health danger to upper Snake River stocks of anadromous fish by exposing chinook to overwhelming stress and to horizontal transmission of etiologic agents, particularly *Renibacterium* (BKD).

Several aspects of fish health and stress issues are related to transportation. These are dealt within the context of the differences and similarities of spring/summer chinook stocks which PATH identified as being Snake River drainage, Upper Columbia River drainage, and Lower Columbia River drainages as follows:

Snake River Stocks:

- Upper Salmon River-Lemhi, Salmon River upstream of Yankee Fork
- Upper Middle Fork-Marsh Creek/Cape Horn Creek
- South Fork-Poverty Flats and Johnson Creek
- Minam River of NE Oregon
- Lostine River of NE Oregon

Upper Columbia River Stocks:

- Entiat River
- Methow River
- Wenatchee River

Lower Columbia River Stocks:

- Deschutes River
- John Day River
- Klickitat River
- White Salmon and Wind Rivers

Data on fish health status of hatchery and wild, juvenile and adult Spring/Summer Chinook was sought from ODFW, WDFW, and USFWS. The fish health database of the Eagle Fish Health Lab was also queried for the same information. The primary salmonid disease issue related to transportation, identified by NMFS and the Independent Scientific Review Group, has been Bacterial Kidney Disease (BKD) caused by *Renibacterium salmoninarum* (RS). Consequently, our primary focus was to collect data on juvenile and adult chinook related to RS and BKD. Excellent cooperation was obtained from all the agencies. Most of the agencies provided detection data from enzyme-linked immunosorbent assay (ELISA) analyses conducted since 1991 when the application of ELISA became standard in the Columbia River Basin.

### **FISH HEALTH ASPECTS OF COLLECTION AND TRANSPORTATION**

There are several premises which relate to this issue: BKD has been implicated to be the cause of delayed mortality following release of transported chinook; BKD occurs in Snake River Basin hatchery chinook at a greater rate and level (prevalence and intensity of infection) than in chinook stocks from the two other locations identified by PATH; BKD is a problem of hatchery chinook and not of wild chinook; that IDFG

has not taken steps to control BKD in its chinook hatchery programs; and that diseases other than BKD need to be addressed.

It is very important to emphasize the difference between detection of the pathogen (RS) which causes BKD and the clinical disease itself. This was the subject of a symposium sponsored by the Pacific Northwest Fish Health Protection Committee (PNFHPC, 1997). This point is often lost in discussions of this nature but is the cornerstone of fish health. There is ample data indicating that detection of the disease agent does not always constitute disease. All managing agencies differentiate between detection and disease. Clinical BKD (the disease state) is associated with ELISA values greater than an optical density (OD) value of 1.0 and correlates well with observing RS from kidney smears by optical methods as well as the progression to the death of the host (Pascho et al. 1993). Environmental factors are key to whether the disease agent becomes problematic. These environmental factors include both those external and internal to the fish host. Most factors are stressors and some will be identified in the section on Stress to follow.

### *DELAYED MORTALITY*

Bacterial kidney disease is a chronic, debilitating disease, meaning it causes mortality within a population over a long period of time following a protracted incubation period which is inversely related to water temperature (Sanders et al. 1978). Post-challenge incubation periods usually exceed 100 days at water temperatures experienced during transportation and in the Columbia River estuary (Murray et al. 1992). Infections with RS have been shown to inhibit smoltification and delay salt water entry. Consequently, the mortality rate is exacerbated in salt water compared with freshwater (Banner et al. 1983). Therefore, BKD has been implicated to be a cause of delayed mortality. Infection rates in hatchery and wild chinook smolts captured at Jones Beach in the Columbia River estuary have been about 25% (Sanders et al. 1992).

Exceptions to this “rule” have been seen with Idaho and Oregon wild chinook taken as parr and reared in either fresh or salt water in the captive propagation programs. Mortality attributed to BKD has been more severe in freshwater than in salt water. Chinook demonstrated to be infected with RS were able to resolve the infection when reared under optimal conditions in salt water (Thorarinsson et al. 1994; Elliott et al. 1995). This provides a link between culture conditions, stress, and mortality to BKD (Congleton et al. 1984). These exceptions indicated that only those fish which had relatively high ELISA values subsequently succumbed to the infection and that those with low values were able to cope with and resolve the infection.

**CONCLUSION:** BKD can be the cause of delayed mortality which may be amplified after entry into salt water particularly when initial infection rates are high and when environmental conditions which are not optimal.

### *BKD IS A HATCHERY DISEASE*

Research by Pascho and Elliott (1991) on the effect of BKD and the transportation process examined ELISA values of wild and hatchery chinook captured at Lower Granite Dam. They concluded that both wild and hatchery chinook from the Snake River had detectable levels of RS by ELISA. Some years, the prevalence of RS in wild chinook exceeded that of hatchery fish. However, ELISA optical density values were generally low in wild fish. ELISA values of hatchery and wild chinook collected from the three sections of the Columbia River Basin considered by the PATH process will follow. A topic for which there is not much data is the correlation of ELISA value at collection with the subsequent mortality. This

mortality data must be partitioned between those fish which were initially infected from those subsequently infected by cohabitation during the holding period.

Observations from IDFG's captive propagation programs which originate from collections of wild parr and smolt sockeye and chinook from three river systems illustrate that BKD was present in a small percentage of the wild fish. Mortality attributed to BKD which occurred within the first five months following collection was low indicating that these wild fish may have had RS in the carrier state but that progression to the disease state was rare. For Redfish Lake sockeye and Salmon River chinook, it appears that about 0.2% of over 1,000 sockeye smolts and 0.7% of the chinook parr carried RS. These rates of infection are similar to those found as "highs" within the Snake and Columbia River Basin areas shown in Table 1.

CONCLUSION: Both wild and hatchery chinook of the Snake River have detectable, but low intensity levels of RS.

*INFECTION RATES ARE MUCH HIGHER IN SNAKE RIVER CHINOOK COMPARED TO RATES OBSERVED IN CHINOOK FROM THE UPPER COLUMBIA AND LOWER COLUMBIA RIVER AREAS*

Survey data for juvenile and adult chinook from the areas of the Columbia River Basin areas identified by PATH are summarized in Table 1. Most of these data are from hatchery populations except for the ID/NE OR area. Some of these data originate from transportation studies on wild and hatchery chinook of the Snake River by Pascho and Elliott (1991). They demonstrated that RS could be detected in water of the post-collection holding raceways and barge holds and that brook trout contracted BKD when exposed to these environments. RS was not in sufficient quantity to be detected in waters of the Snake River outside of the barge hold. They also determined that chinook with ELISA values exceeding 2.0 (very high level) were those shown to shed RS into the water.

Chinook of all three areas have been demonstrated to harbor antigens of RS regardless of which area of the Snake River and Columbia River they originated from. Both the prevalence and intensity of infection of those originating from the Snake River Basin were frequently lower than those of either the Upper and Lower Columbia River Basin area. Detections of smolts with clinical BKD by ELISA are rare. These data illustrate the need to avoid the crowded conditions, such as barging, where horizontal transmission from the few "highs" could occur. The actual transmission of BKD from hatchery to wild chinook (or vice versa) through smolt collection and subsequent barging has not been demonstrated (Steward and Bjornn 1990).

CONCLUSION: BKD is no more prevalent in Snake River systems than elsewhere and infectious diseases would not constrain recovery of these stocks if migration conditions in the mainstem are corrected.

*IDFG HAS NOT TAKEN MEASURES TO CONTROL BKD WITHIN ITS HATCHERIES*

The evolution of fish culture methods for successful control of BKD in chinook has emphasized injection of returning adults with Erythromycin (Klontz. 1982), feeding the same antibiotic to juveniles (Moffitt and Schreck. 1988), and segregation rearing (with or without culling) of progeny from females demonstrated to be clinically infected with BKD (Pascho et al. 1991). IDFG has been proactive in the application of these practices over the last two decades. Application of the most effective combination of these practices has resulted in marked reduction in the occurrence of clinical BKD at IDFG facilities (Figure 1; Munson

1998). These improvements have been observed by reduced detection of clinically infected chinook at collection facilities of the dams on the lower Snake River (Maule et al. 1996; VanderKooi and Maule. in press).

**CONCLUSION:** Dramatic reduction in clinical BKD has occurred at IDFG facilities over the last decade.

Table 1. Comparison of adult and juvenile spring/summer chinook BKD ELISA categories for Snake River, Upper Columbia River, and Lower Columbia River for the years 1991-1997.

<b>Idaho/Northeast Oregon</b>					
	Number	BL	L	M	H
Adult	10,450	48%	29%	14%	9%
Juvenile	1987	66%	30%	3%	1%
<b>Upper/Mid Columbia River</b>					
	Number	BL	L	M	H
Adult	7452	43%	41%	7%	9%
Juvenile	1905	60%	29%	2%	9%
<b>Lower Columbia River</b>					
	Number	BL	L	M	H
Adult	5132	58%	18%	6%	18%
Juvenile	1330	52%	32%	8%	8%

Legend of ELISA Cut-off Categories:

- BL = Below Low, ELISA OD <0.1
- L= Low, ELISA OD 0.1 to 0.2
- M= Moderate, ELISA OD 0.2 to 0.45
- H= High, ELISA OD > 0.45

## *DISEASES OTHER THAN BKD*

*Myxobolus cerebralis*, the myxozoan parasite which can cause whirling disease was demonstrated to infect chinook at Sawtooth and Pahsimeroi Hatcheries in 1987. Facility management actions were implemented to limit exposure of small fish to the infective river water at both sites. This action has been demonstrated to be effective in Europe and has limited infections to the asymptomatic carrier state. Approximately 4.6% of wild chinook parr collected for the chinook captive propagation program have been shown to be infected with *M. cerebralis*, but whirling disease has not caused any mortality after 2.5 years of rearing. Infections of returning adults have also been at the carrier level. It is doubtful that this parasite exerts a limiting effect on chinook populations in the Salmon River.

Erythrocytic Inclusion Body Syndrome (EIBS) occurs at a higher rate in hatchery chinook (70%) than those produced naturally (50%) in the Snake River Basin (PNFHPC 1997). This viral condition, deemed “fuzzy-tail,” has been shown to be debilitating to chinook at Rapid River Hatchery. It was especially prevalent in the late 1980's but has diminished dramatically once BKD was controlled with Erythromycin therapy (Munson 1998).

## **STRESS ASPECTS OF COLLECTION AND TRANSPORTATION**

A variety of biological, physical, and chemical factors are capable of challenging the physiological systems of fish, forcing the fish to adapt to survive. Selye (1950) described stress and the general adaptation syndrome (GAS) which states that if an animal is stressed long enough, the physiologic systems will adapt to accommodate the stress and to maintain or reestablish a normal metabolism. Once the accommodating systems are exhausted, the animal will die. It is well established that acute and chronic stress that approaches physiological tolerance limits of fish will impair reproductive success, growth, resistance to infectious disease, and survival (Wedemeyer et al. 1990). The cumulative effects of stress may reduce recruitment to successive life stages and eventually cause populations to decline (Vaughn et al. 1984; Adams et al. 1985).

Several authors have described the stressors and the cascade of events that follow stress related events during collection, barging and release of anadromous fish (Congleton 1984; Bjornn 1984, 1986, and 1987; Wedemeyer 1985; Pascho and Elliott 1993; Schreck 1998). Taken individually, these stressors may not be directly lethal, but the cumulative effect does change a group of individual stressors into a deadly gauntlet which poses the overwhelming threat at any life stage to wild and hatchery anadromous fish.

Location/actions which appear to be most stressful in the collection and transportation process vary in fish response. The separation step and loading the barges were found to be high point sources of stress for chinook. Lesser reactions were noticed during holding the fish in raceways (exposed to direct sunlight) and unloading the barges during these studies. Trucking fish exacted more stress from chinook than barging, because recovery from stress was achieved during barging. Transporting chinook with steelhead was more stressful to chinook than if chinook were transported without steelhead. Barge loads at near maximum densities created higher levels of stress than loads well below maximum density. Decreased adult return rates have been demonstrated from chinook with elevated stress levels during transportation. Stress and delayed mortality are related topics especially when dealing with BKD (Schreck, 1998; Pascho & Elliott, 1993). Cohabitation of high titer RS individual fish with low titer fish, exacerbates horizontal transmission, with the prolonged time of infection to death (up to 104 days). Thus, most deaths would occur in the salt water stage. Chinook which are disoriented, diseased, and/or with diminished

smoltification are predisposed to predation after release from the barges and passage through dams (Schreck, 1998).

Smoltification and migration of chinook are normal, but chronically stressful events (Steward and Bjornn 1990). The success of regional anadromous fish restoration efforts rests on immediate improvements in the quality of the mainstem corridor (Bisbal 1998). Linkage of Idaho spawning habitats (and hatcheries) to the ocean is crucial to Idaho anadromous stocks. Mainstem alterations have slowed down migration times, supersaturated the water with nitrogen, and provided a beneficial habitat for predators (squawfish). Collection and barging have been implemented to circumvent these adverse migration conditions but are themselves inherently stressful. These additional acute and chronic stressors diminish and drain the anadromous populations' ability to survive. The cumulative effects of all factors natural and manmade, need to be considered. The unnecessary stressors should be eliminated from this process. IDFG feels that the choice of a normative river is the best way to achieve this result.

**CONCLUSION:** Collection and barging through the upper Snake and Columbia River corridor represents the largest fish health danger to upper Snake River stocks of anadromous fish by exposing chinook to overwhelming stress and to horizontal transmission of etiologic agents, particularly *Renibacterium*.

## LITERATURE CITED

### INFECTIOUS DISEASES

Banner, C.R., J.S.Rohovec, and J.L.Fryer. 1983. *Renibacterium salmoninarum* as a cause of mortality among chinook salmon in salt water. *Journal of the World Mariculture Society*. 14:236-239.

Congleton, J.L., T.C.Bjornn, C.A.Robertson, J.L.Irving, and R.R.Ringe. 1984. Evaluating the effects of stress on the viability of chinook salmon smolts transported from the Snake River to the Columbia River Estuary. Final Report, U.S.Army Corps of Engineers, Contract DACW68-83-C-0029.

Elliott, D.G. and R.J.Pascho. 1993. Juvenile fish transportation: Impact of bacterial kidney disease on survival of spring/summer chinook salmon stocks. U.S.Army Corps of Engineers, Walla Walla, WA. November, 1995.

Elliott, D.G., R.J.Pascho, 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.

Klontz, G.W. 1982. Bacterial kidney disease in salmonids: an overview. Symposium International de Tallories: Antigenes of Fish Pathogens. Anderson, Dorson, and Dubourget, Editors.

Maule, A.G., D.W.Rondorf, J.Beeman, and P.Haner. 1996. Incidence of *Renibacterium salmoninarum* infections in juvenile hatchery spring chinook in the Columbia and Snake Rivers. *Journal of Aquatic Animal Health*. 8:37-46.

Moffitt, C.M. and J.A.Schreck. 1988. Accumulation and depletion of orally administered Erythromycin Thiocyanate in tissues of chinook salmon. *Transactions of the American Fisheries Society*. 117:394-400.

- Munson, A.D. 1998. Integrated management of bacterial kidney disease at IDFG Lower Snake River Compensation Fish Hatcheries. Lower Snake River Compensation Plan Status Review Symposium. 2-4 February 1998, Boise Idaho.
- Murray, C.B., T.P.T.Evelyn, T.D.Beacham, L.W.Barner, J.E.Ketcheson, and L.Prospieri-Porta. 1992. Experimental induction of bacterial kidney disease in chinook salmon by immersion and cohabitation challenges. *Diseases of Aquatic Organisms*. 12(2):91-96.
- PNFHPC Symposium. 1997. Pathogens and diseases of fish in aquatic ecosystems: Implications for fisheries management. *Journal of Aquatic Animal Health*. *In press*.
- Pascho, R.J., D.G.Elliott, and S.Achord. 1993. Monitoring of the in-river migration of smolts from two groups of spring chinook salmon, *Oncorhynchus tshawytscha* (Walbaum), with different profiles of *Renibacterium salmoninarum* infection. *Aquaculture and Fisheries Management*. 24:163-169.
- Pascho, R.J., D.G.Elliott, and J.M.Streufert. 1991. Brood stock segregation of spring chinook salmon *Oncorhynchus tshawytscha* by the use of enzyme-linked immunosorbent assay (ELISA) and the fluorescent antibody technique (FAT) affects the prevalence and levels of *Renibacterium salmoninarum* infection in progeny. *Diseases of Aquatic Organisms*. 12:25-40.
- Sanders, J.E., J.J.Long, C.K.Arakawa, J.L.Bartholomew, and J.S.Rohovec. 1992. Prevalence of *Renibacterium salmoninarum* among downstream migrating salmonids in the Columbia River. *Journal of Aquatic Animal Health*.4:72-75.
- Sanders, J.E., K.S.Pilcher, and J.L.Fryer. 1978. Relation of water temperature to bacterial kidney disease in coho salmon (*Oncorhynchus kisutch*), sockeye salmon (*O. nerka*), and steelhead trout (*Salmo gairdneri*). *Journal of the Fisheries Research Board of Canada*. 35:8-11.
- Steward,C.R. and T.C.Bjornn. 1990. Supplementation of salmon and steelhead stocks with hatchery fish: synthesis of published literature. Part 2. In W.H.Miller (ed). *Analysis of Salmon and Steelhead Supplementation, Parts 1-3*. Technical Report, Proj. No.93-013, Contr.No.DE-AM79-93BP99954, Task Order DE-AT79-93BP00121.
- Thorarisson, R, M.L.Landolt, D.G.Elliott, R.J.Pascho, and R.W.Hardy. 1994. Effect of dietary vitamin E and Selenium on growth, survival and the prevalence of *Renibacterium salmoninarum* infection in chinook salmon (*Oncorhynchus tshawytscha*). *Aquaculture*. 121:343-358.
- VanderKooi, S.P. and A.G.Maule. Incidence of *Renibacterium salmoninarum* infections in juvenile spring chinook salmon (*Oncorhynchus tshawytscha*) at Columbia and Snake River Hatcheries. *Journal of Aquatic Animal Health*. *In Press*.

## **STRESS**

- Adams, S. M., Breck, J. E., and McLean, R. B., 1985. Cumulative stress-induced mortality of gizzard shad in a southeastern U. S. Reservoir. *Environmental Biology of Fishes* 13:103- 112.

- Barton, B.A. and Iwama, G.K., 1991. Physiological changes in fish from stress in aquaculture with emphasis on the response and effects of corticosteroids. *Ann. Rev of Fish Diseases* 3-26.
- Bisbal, 1998. Northwest Power Planning Counsel Decision Memorandum. Captive Brood Stock
- Bjornn, T.C., J.L.Congleton, R.R.Ringe, and C.M.Moffitt. 1984-1987. Survival of chinook salmon smolts as related to stress at dams and smolt quality. Idaho Cooperative Fish and Wildlife Research Unit. Technical Reports 85-1, 87-4, and 88-1. Moscow, ID
- Pascho, R. J. and Elliott, D. G., 1993. Juvenile fish transportation: Impact of bacterial kidney disease on survival of spring/summer chinook salmon stocks. Annual Report for US Army Corps of Engineers.
- Selye, H. 1950. Stress and the general adaptation syndrome. *British Medical Journal* (1)1383-1392.
- Selye, H. 1973. The evolution of the stress concept. *American Scientist* (61) 692-699.
- Schreck, C. B. 1981. Physiological, behavioral, and performance indicators of stress. *American Fisheries Society Symposium* (8) 29-37.
- Schreck, C. B. 1998. Personal communications with Keith Johnson, 14 April 1998.
- Vaughn, D.S., Yoshiyama, R. M., Breck, J. E. and DeAngelis, D. L., 1984. Modeling approaches for assessing the effects of stress on fish populations. Pg. 259-278 in Cairns et al., *Contaminant Effects on Fisheries*. Wiley, Toronto.
- Wedemeyer, G. A., Palisimo, A. N., and Salsbury, L. E., 1985. Development and evaluation of transport media to mitigate stress and improve juvenile salmon survival in Columbia River barging and trucking operations. Final Report (Contract DE-A179-82BP-35460) to Bonneville Power Administration, Portland Oregon.
- Wedemeyer, G. A.; Barton, B. A.; and Mcleay, D. J. 1990. Stress and acclimation. *Methods For Fish Biology*. 451-489.