

SNAKE RIVER FALL CHINOOK SALMON
BROOD-STOCK PROGRAM (1981-1986)

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ABSTRACT

The objective of the Snake River Fall Chinook Salmon Brood-stock Program was to hatch eggs from upriver stocks, rear the fish to spawning maturity, and use the resulting eggs for stock restoration in the Snake River. Approximately 15,000 eyed Snake River fall chinook salmon eggs were obtained each winter in 1981, 1982, 1983, and 1984 from various Columbia River hatcheries. Fish from these eggs were reared in dechlorinated City of Seattle water at the Northwest and Alaska Fisheries Center or in constant 10.5°C groundwater at the University of Washington's Big Beef Creek Research Station. Seawater tolerance trials of 0+ age (3-5 months) juveniles in all four brood stocks were strongly suggestive of the 1+ age smoltification pattern of spring chinook salmon. Attempts to transfer 0+ age fish to marine net-pens at the Manchester Marine Experimental Station were unsuccessful during the four brood years. The only Snake River fall chinook salmon that demonstrated acceptable survival after 4 months residence in seawater were fish that were transferred as 1+ age smolts. After smolts were successfully transferred to seawater, losses were minimal for several months. However, in all Snake River chinook salmon stocks, mortality due to bacterial kidney disease (BKD) and a previously undescribed "rosette disease" resulted in very few maturing fish at 4 or 5 years of age.

Results of this experimental program indicate that at this time the rearing of Snake River chinook salmon to maturity in marine net-pens would not lead to a viable egg-bank program.

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INTRODUCTION

The objective of the Snake River Fall Chinook Salmon (SRFC) Brood-stock Program was the enhancement of upriver (bright) stocks of chinook salmon through research and development of an egg bank. These stocks historically made significant contributions to ocean and lower Columbia River fisheries and are uniquely adapted to the upper Snake River environment. However, as a result of habitat destruction and over-fishing, the formerly abundant wild-spawning stocks of fall chinook salmon in the Snake River diminished to only about 2,000 fish per year by 1977 (Ortmann 1978). The low natural returns during the late 1970s and early 1980s prompted the National Marine Fisheries Service (NMFS) and the Bonneville Power Administration (BPA) to cooperatively explore alternatives to traditional rear/release brood-stock strategies.

The NMFS program utilized a captive rearing concept, with fish reared to maturity in seawater net-pens. With other salmonids, captive brood stocks have shown the potential for production of large numbers of eggs for enhancement purposes (Harrell et al. 1984). Throughout the production program, research was also conducted on disease diagnosis and control, nutrition, acclimation to seawater, and spawning strategies.

The program began in February 1981 when approximately 15,000 eyed eggs (1980 brood) were obtained from the Dworshak National Fish Hatchery. Resulting fry were reared to smolts, transferred to seawater net-pens, and grown to maturity. This was repeated for three consecutive brood years. During the 3 years residence in seawater net-pens, before maturity at 4 years of age, mortality due to bacterial kidney disease (BKD) (Fryer 1985) and a new pathogen ("rosette disease") (Harrell et al. 1986) approached 99% and

completely compromised attempts to produce viable egg-bank gametes. Attempts to control BKD and the rosette disease with chemotherapeutants and dietary supplementation were unsuccessful. The authors offer strong presumptive evidence that BKD was readily transmitted between various stocks of chinook salmon in marine net-pens. A description of the previously undescribed rosette disease, as well as results of attempts to culture and control it in vitro, were published by Elston et al. (1986) and Harrell et al. (1986); these reports are considered complementary to this final report.

DESCRIPTION OF STUDIES

Brood-stock Program

The NMFS brood-stock program was intended to provide a stable egg-bank of Snake River fall chinook salmon through a captive marine rearing concept. During the program, NMFS had stocks from 1980, 1981, 1982, and 1983 brood under production. These fish were reared through their freshwater cycle and transferred to seawater net-pens at the NMFS' Manchester Marine Experimental Station near Manchester, Washington.

A parallel program goal was an understanding of the seawater phase of the life-cycle of chinook salmon. Captive rearing in seawater offers the unique opportunity to document factors affecting growth and survival. Currently, management models consider the seawater period of the chinook salmon life-cycle to be a "black box" with perhaps the greatest mortality occurring as predation on young fish. Once the fish reach about 3 to 4 lbs, it is assumed that survival to adult is good. Our research, however, revealed that BKD and the previously unreported rosette disease killed up to 90% of our brood stock between 2 years of age and maturity. NMFS researchers, in

conjunction with the Battelle Marine Laboratories at Sequim, Washington, described these diseases and investigated methods of control.

Special Disease Investigations

Experiments, since August 1983, attempted to understand the source, occurrence, and pathogenesis of the previously unreported infectious rosette organism that killed most of the 1980, 1981, and 1982 brood Snake River chinook salmon during their final year before maturation.

Methods for control of the rosette disease and BKD in the marine environment were also investigated during the brood-stock program. Isolation and transmission experiments, as well as attempts to characterize the rosette pathogen, were conducted at the Center for Marine Disease Control, Battelle Marine Research Laboratory, Sequim, Washington.

MATERIALS AND METHODS

Freshwater Husbandry

Approximately 15,000 eggs from Snake River fall chinook salmon were received from egg-bank facilities on the Snake and Columbia Rivers for the brood years 1981, 1982, 1982, and 1983. Freshwater rearing was conducted at the Northwest and Alaska Fisheries Center (NWAFC), in ambient-temperature (dechlorinated) City of Seattle water, or (for the 1983 brood) at the NMFS experimental hatchery at the University of Washington's Big Beef Creek Research Station near Seabeck, Washington. At the Big Beef Creek Hatchery, fish were reared in large (13-ft diameter) fiberglass tanks supplied with constant-temperature (10° C) pathogen-free groundwater and fed via automatic feeder. Smolted fish were transferred to the Manchester Marine Experimental Station.

Transfer to Seawater

Both 0+ and 1+ age fish transferred to Manchester were acclimated to full-strength seawater (28 ppt) over a period of several days using a freshwater lens system developed for this program. Fresh water was introduced at the surface of a vinyl-skirted seawater net-pen, creating a freshwater/seawater interchange that allowed the fish to choose their salinity. This lens system provided a modified-volitional transition to seawater (Harrell et al. 1984).

Marine Husbandry

Brood stocks were held in 24 16-x16-x12-ft deep net-pens at a density of 0.5 lb/ft³ or less. Seawater temperatures ranged from 7° to 13°C during the year, and mean salinity was 28 ppt. Fish were fed pelleted rations from several commercial manufacturers supplemented with fresh frozen herring, Clupea harengus, and whole krill, Euphausia pacifica. Most fish were injected intraperitoneally with a vibrio bacterin/oxytetracycline mixture at 6- to 8-month intervals during their seawater residence. The salmon were also fed antibacterial drugs during epizootics of bacterial disease. Dead and moribund fish were removed from the population daily, weighed, measured, and necropsied.

Spawning Strategies

Maturing 4-year-old 1980 Snake River chinook salmon were spawned in fall 1984. These fish were sorted for maturity in September and October 1984 and, under Washington Department of Fisheries (WDF) provisions, moved to quarantine facilities at the Battelle Marine Laboratory. This was necessary because of the unknown aspects of the newly-observed rosette pathogen. The Battelle

facility was supplied with constant-temperature (11.3°C) pathogen-free groundwater, and the effluent was chlorinated. Mature fish were subsequently spawned, and each female's eggs were placed in isolated incubation systems.

No fish were successfully spawned in 1985.

Bacterial Kidney Disease Investigations

Snake River chinook salmon eggs from the 1983 spawning were incubated and cultured at the Big Beef Creek Research Station. A portion of these fish were transferred to seawater at Manchester as 0+ age smolts (n=6,608, at 8.2 g average) and some as 1+ age smolts (n=1,258, at 61.4 g average) in 1984 and 1985. Another group of these fish was transferred to the U.S. Fish and Wildlife Service (USFWS) seawater facility on Marrowstone Island, Washington, as 2+ age smolts (n=150, at 252.0 g average) in February 1986. The incidence of BKD-related mortality in these three separate lots of chinook salmon was compared to the 1983-brood fish (n=530) which were retained on groundwater at the Big Beef Creek Research Station.

RESULTS AND DISCUSSION

Freshwater Growth and Survival

The NMFS cultured four brood years of Snake River fall chinook salmon (1980, 1981, 1982, and 1983 stocks) in fresh water prior to transfer to marine net-pens. Freshwater growth and survival were variable in the 1980, 1981, and 1982 broods. The Big Beef Creek facility was operational in late 1982, and both growth and survival of the 1983 brood reared at Big Beef Creek were excellent (Figs. 1 and 2).

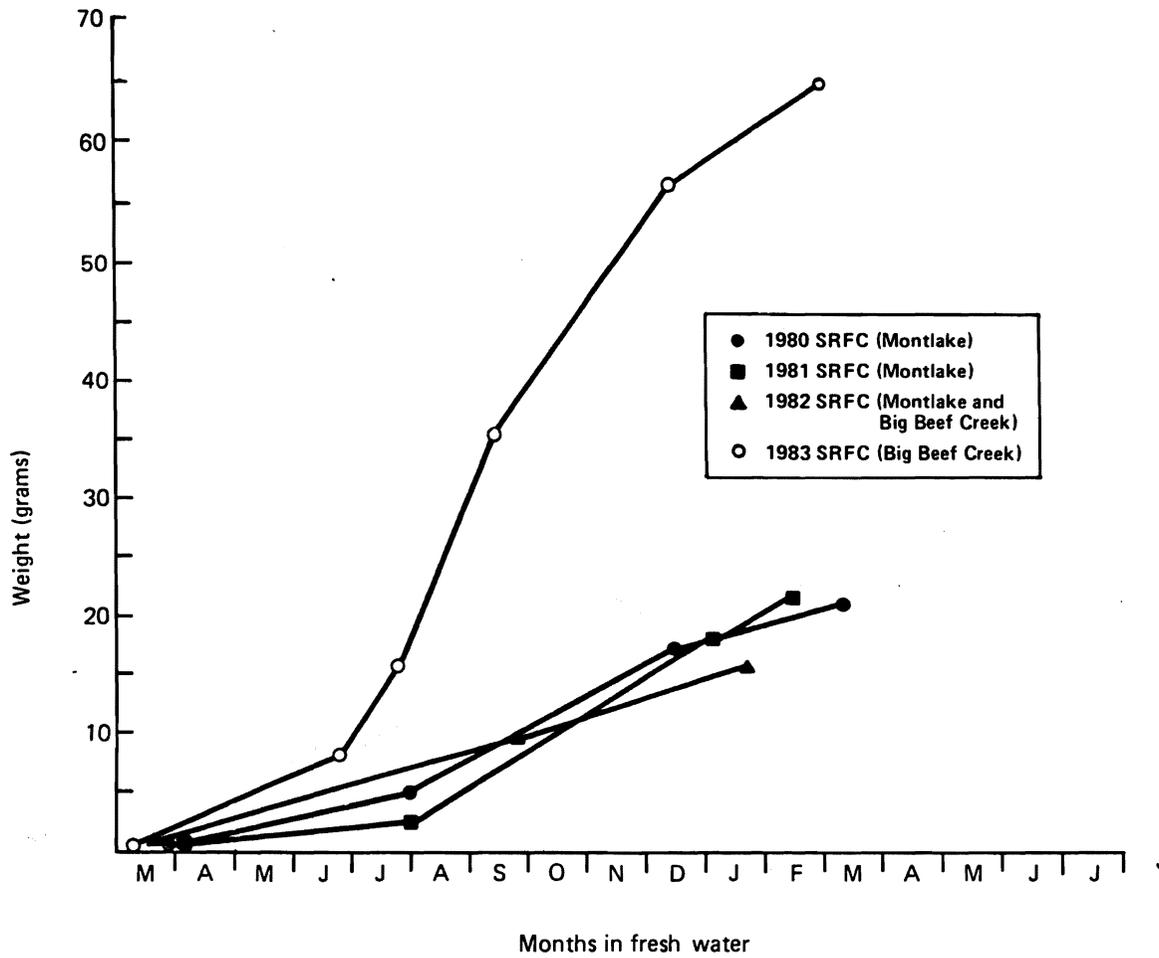


Figure 1.--Freshwater growth for Snake River fall chinook salmon (SRFC) broods. Freshwater rearing was conducted at NMFS Montlake laboratory or NMFS Big Beef Creek Hatchery as indicated.

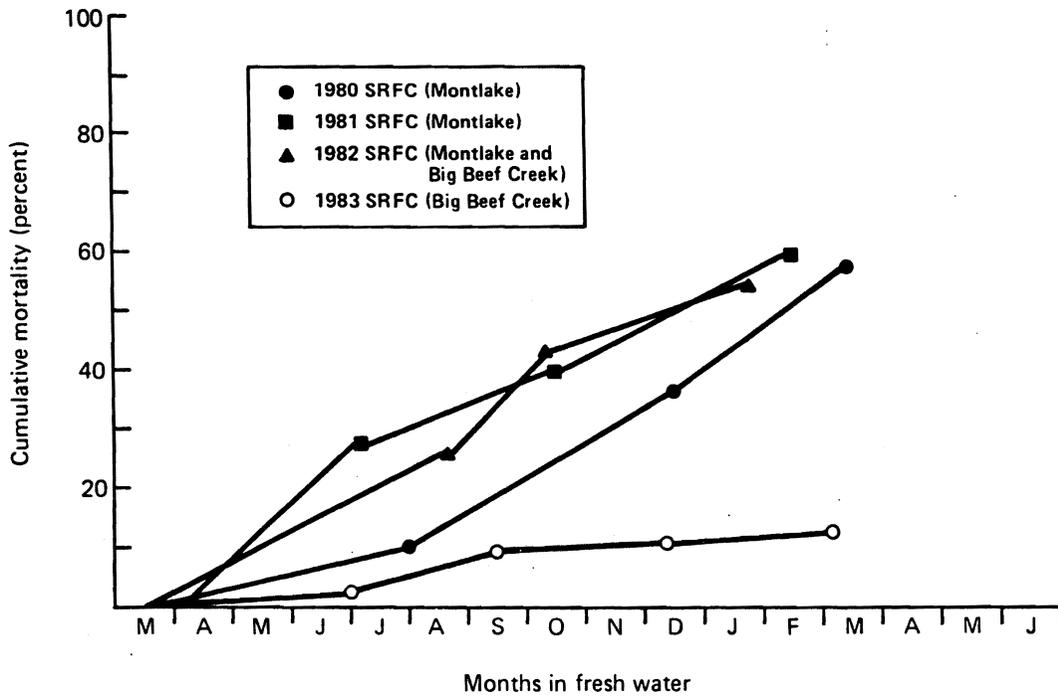


Figure 2.--Mortality of four successive Snake River fall chinook salmon (SRFC) brood years in freshwater facilities at Montlake (dechlorinated City of Seattle water) and Big Beef Creek (groundwater).

Transfer to Seawater

Management practices for Snake River fall chinook salmon include hatchery release as 0+ age fish. However, our research suggested that this technique may not be as conducive to seawater survival as 1+ age smolt releases. Initial freshwater growth for the 1980, 1981, and 1982 stocks was poor, therefore, only experimental lots (n=200-400) were transferred to seawater at 0+ age. These tests were not entirely successful; fish experienced osmoregulatory-related mortalities, and the few survivors required up to 6 months to fully adapt to seawater. For these same broods, the main production lots were transferred as 1+ age fish. These yearlings experienced fewer problems at seawater entry, and survival during the first year of seawater residence was acceptable (Fig. 3). This pattern suggested a strong 1+ age smolting component for this stock. However, the small number of 0+ age fish transferred to seawater during these years did not allow adequate testing of this hypothesis.

The increased growth and survival of the 1983 stock allowed significant numbers (n = 6,608) of 8.2-g average 0+ age fish to be transferred to seawater in spring 1984. This transfer had a high mortality during the first 4 months of marine residence (Fig. 4), and the survivors were lethargic and did not feed or grow well during this period. Mortalities from this group were examined, and losses could not be attributed to infectious agents. Mean serum levels of Ca^{++} and Mg^{++} were extremely high (85 and 10 mg%, respectively) and were indicative of osmoregulatory problems. All evidence confirmed that these 0+ age fish were not complete smolts when transferred to seawater.

Conversely, 1983 stocks transferred to marine net-pens a year later as 1+ age smolts experienced excellent seawater survival for the first 4 months of

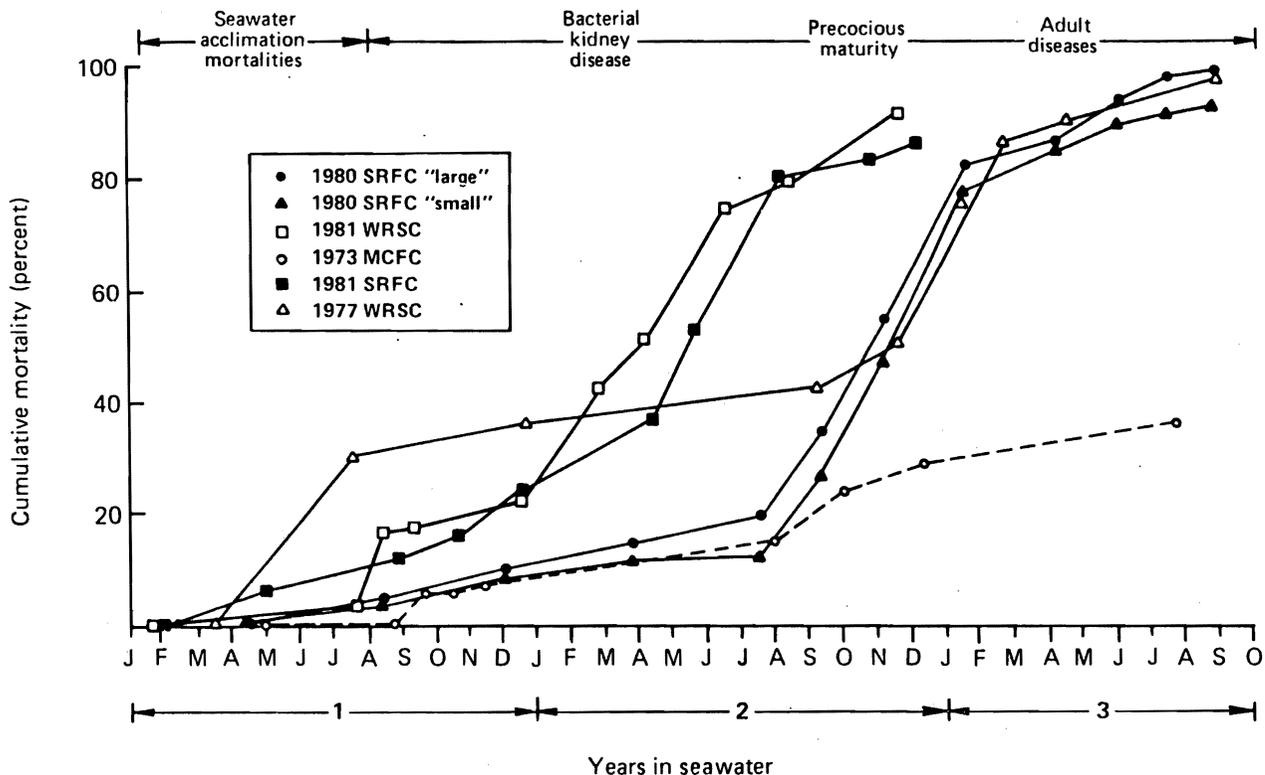


Figure 3.--Cumulative mortality in seawater for chinook salmon that entered seawater as yearling smolts and were reared at the NMFS Manchester Marine Experimental Station. Mortality profiles for "large" 1980 Snake River fall chinook salmon (●), which entered seawater averaging 29 g; "small" 1980 Snake River fall chinook salmon (▲), which entered seawater averaging 15 g; 1981 White River spring chinook salmon (◻); 1973 Minter Creek fall chinook salmon (○); 1981 Snake River fall chinook salmon (■); and 1977 White River spring chinook salmon (△).

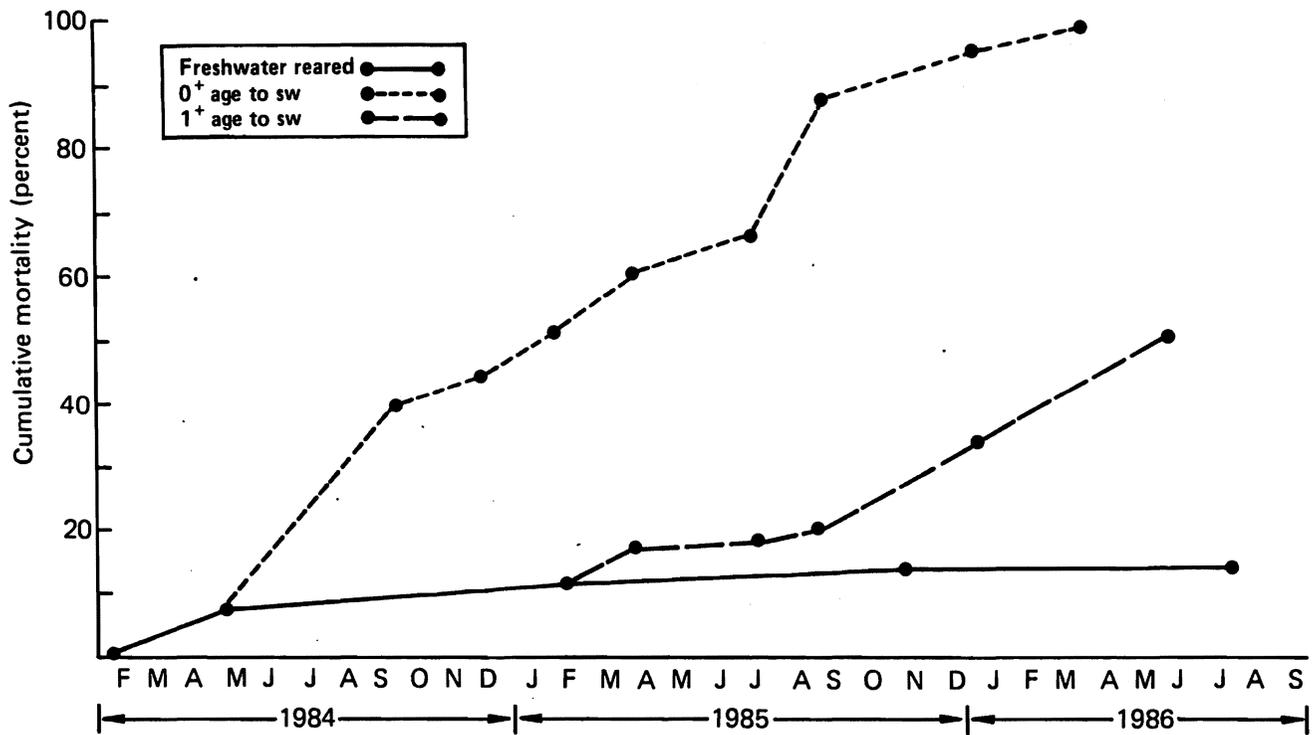


Figure 4.--Mortality of 1983-brood Snake River fall chinook salmon retained in groundwater and after transfers to seawater at 0+ age and 1+ age.

seawater residence (Fig. 4). This pattern demonstrated that, for marine net-pen culture, the upriver bright stock should only be transferred to seawater as yearlings.

Seawater Growth and Survival

After Snake River fall chinook salmon were transferred and fully adapted to seawater, growth was excellent and similar for three broods (Fig. 5). During the 3 years of culture in marine net-pens, mortality patterns in the three brood stocks were also nearly identical (Fig. 6). The cause of these losses, BKD, precocious male development, and the rosette disease, occurred at approximately the same time for each brood year and resulted in nearly identical losses (Figs. 3 and 6).

The first Snake River fall chinook salmon smolts (approximately 6,500 1980-brood) were successfully transferred to full-strength seawater in April 1982. During the following 15 months of seawater residence, losses to vibriosis (Vibrio anguillarum) were prevented by vaccination, and mortality due to BKD was less than subsequently occurred in 1981 and 1982 brood years. In August 1983, however, a sudden increase in mortality could not be attributed to typical pathogens. During the following 10 months, over 4,500 3-year-old fish (1,200-1,500 g) succumbed to a previously-unreported systemic infection. This pattern of losses was repeated during autumn of the following 2 years with the 1981 and 1982 brood-stock adults (Figs. 3 and 6). Because of the seriousness of this pathogen and its implication to overall marine survival, a cooperative study was initiated between NMFS and the Battelle Marine Laboratory. Study of the pathogenesis of this organism included pathology, hematology, and electron microscopy.

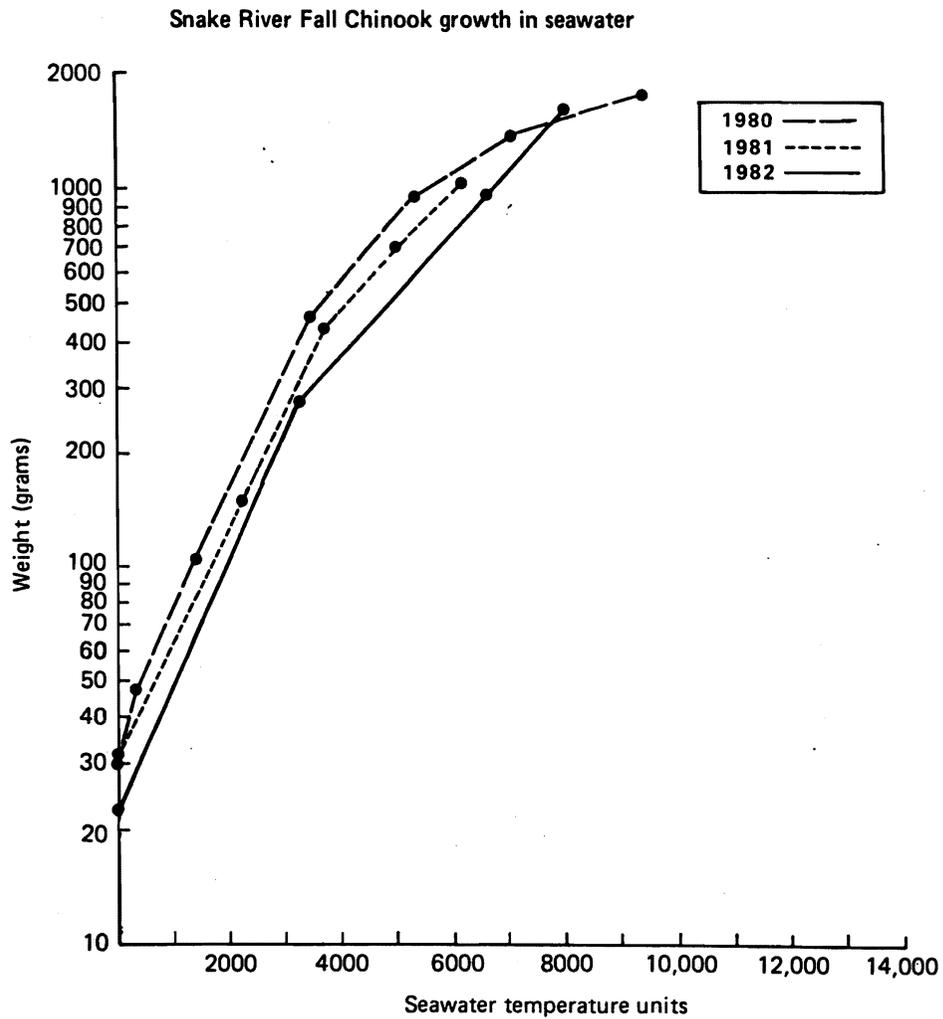


Figure 5.--Growth of three successive brood-years of Snake River fall chinook salmon in seawater net-pens.

Snake River Fall Chinook mortality in seawater

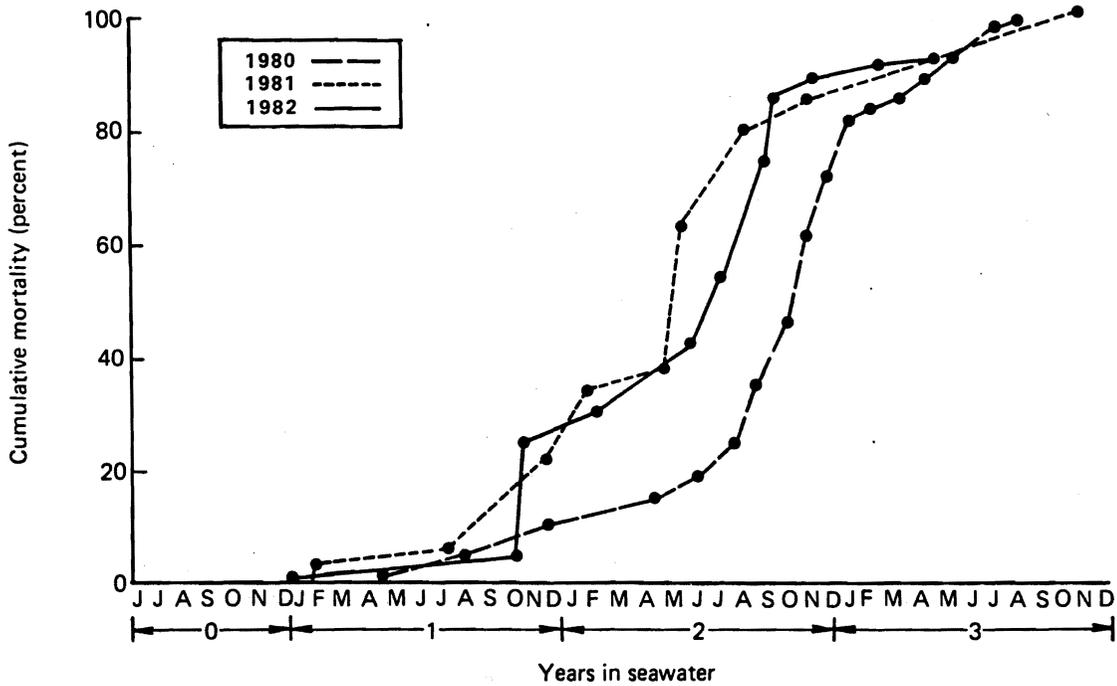


Figure 6.--Mortality of three successive brood-years of Snake River fall chinook salmon in seawater net-pens.

Diagnosis of Rosette Disease

Necropsy of moribund and dead fish revealed swollen kidneys and spleens. Fish were severely anemic, with packed cell volumes averaging 12% and hemoglobin values of 4.9 mg/dl. Whole blood smears stained with DIFF-Quik^{1/} indicated a pronounced lymphocytosis. Kidney and spleen tissue streaked on typticase soy and Sabouraud's agar plates were negative for bacterial or fungal growth. Gram-stained smears of these tissues were negative for Renibacterium salmoninarum, the causative bacterium of BKD. However, clusters of gram-positive staining, variable-sized (3-7 μ m in diameter), spherical organisms were observed. The organisms were intensely birefringent in wet-mount preparations observed with Nomarski interference contrast microscopy. The organisms appeared to initially accumulate and replicate within fixed macrophages of the spleen and kidney. In more severe infections, the pathogen was seen in peripheral blood and the vascular spaces of liver, gonad, heart, brain, and intestinal mucosae. Variable-sized free organisms were formed within the interstitium of spleen and kidney parenchyma and were associated with areas of edema and focal necrosis. There was little evidence of inflammatory change or fibroblastic proliferation associated with this disease. Staining reactions of the organism in tissue sections indicated they were positive to both PAS and GMS and stained brown with Lugol's iodine solution.

Transmission electron microscopy of the spherical organisms demonstrated the existence of intracellular clusters. Detailed ultrastructural examination

^{1/} Reference to trade names does not imply endorsement by the National Marine Fisheries Service, NOAA.

of the causative organisms revealed a cell wall composed of a single-layered outer membranous structure (possibly of host cell origin), a second moderately electrondense layer, and a thin, inner electronlucent zone that separated the second layer from the plasma membrane. The organisms contained peripherally-oriented mitochondria within a ribosomal matrix and both membrane-bound and non-membrane-bound vacuoles of varying density. Nuclei were relatively indistinct.

The cause of mortality in the adult-size brood stock was unquestionably a result of the invasive nature of the pathogen and the associated anemia. Except for limited macrophage activity and edema, there was negligible tissue reaction in the form of inflammation or granulation. This minimal host response could be attributed to the immunologically inert cellulose cell wall of the pathogen. The lymphocytosis appeared to be a limited inflammatory response to the new pathogen, although the infection appeared to overwhelm the salmon before an effective defense mechanism was established. The cell wall of the causative organism appeared to be composed of cellulose based on its positive staining with PAS, GMS, and Lugol's and its marked degree of birefringence. Organisms considered to have fungal affinities are known to have cell walls composed of chitin, cellulose, or a combination.

Tissue from fish infected with the new organism was inoculated on chinook salmon embryo cell lines (CHSE-214 cells). After 30 days incubation, the cells were infected with the obligate intracellular parasite. The organisms were subsequently harvested from the cell culture and used in attempts to infect chinook salmon. Naive fish were either injected intraperitoneally or force-fed the tissue culture isolate. The disease and mortality were reproduced using the tissue culture isolate, and, in addition, the organisms

were reisolated in CHSE-214 cells 25 days after inoculation with tissue from moribund fish. Identification of the reisolated organism was confirmed with the tissue culture isolate by morphological and antigenic methods. Antigenic identification was confirmed between tissue culture isolate and field isolated organisms. These laboratory results demonstrated the fungal affinities of the causative organism of the marine mortality, and that it had been isolated.

Laboratory (tissue culture) experimentation was also used to demonstrate a positive, in vitro, effect of several antifungal and antibacterial drugs. Oxytetracycline and amphotericin-B both showed promise in these tests. However, limited trials testing these drugs as preventives with fish in seawater have shown no positive demonstrable effect.

Other systemic fungal infections of marine-reared salmonids have been attributed to the feeding of raw marine fish. Both 1980- and 1981-brood Snake River fall chinook salmon were fed supplements of raw herring and krill. No evidence of the chinook salmon pathogen was observed during microscopic examination of either supplement. To investigate the possibility that the rosette disease was diet-related, a feeding trial was conducted during early 1984 in an attempt to induce the disease in naive chinook salmon (1982 SRFC 1+ age smolts). Approximately 200 fish each were fed either chopped herring or Oregon moist pellets as a sole ration for 60 days. Subsequently, the new disease was observed in both experimental groups in marine net-pens during 1985. Therefore, it is probable that the transmission of the rosette disease can not be attributed to the feeding of raw marine fish.

Detailed results of the diagnosis and pathogenesis of the rosette organism are discussed in Elston et al. (1986) and Harrell et al. (1986). To date, the chinook salmon rosette disease has only been isolated in its

infective form at Manchester. However, we are attempting to locate carriers of this disease in the wild.

It is inconceivable that this is a freshwater pathogen. Routine gram-staining of kidney and spleen tissues is a common practice of fishery biologists, and it would not be difficult to recognize the organism in gram-stained smears. We have also examined more than 300 pre-smolt Snake River fall chinook salmon and have seen no evidence of the pathogen in these fish.

Bacterial Kidney Disease

All brood years of Snake River fall chinook salmon at Manchester have experienced serious losses due to BKD (Fig. 3). Attempts to control BKD by oral administration of antibiotics (erythromycin) were not successful. This was probably due to a continuous bacterial challenge between different year-class brood stock in adjacent marine net-pens which negated chemotherapeutic efforts. Comparisons of BKD incidence within and between year-classes at Manchester suggest that the disease was contracted during seawater residence.

In an effort to document horizontal seawater transmission, a study was initiated in 1984 that compared BKD incidence for different treatments of 1983-brood Snake River fall chinook salmon reared at the Big Beef Creek Hatchery on pathogen-free groundwater. Prior to seawater transfer in June 1984, a subsample of 176 fish was sacrificed and surveyed for presence of BKD by the fluorescent-antibody technique (FAT). No BKD organisms were documented in this sample, and the population was diagnosed as BKD-free. On 20 June 1984, 6,608 (8.2 g, 0+ age) fish from this population were transferred to

seawater at Manchester. The remaining fish were retained in fresh water at Big Beef Creek.

The 0+ age fish transferred to seawater experienced approximately 40% mortality during the first 4 months in marine net-pens. High serum levels of divalent cations in these fish, and no evidence of mortality due to BKD or other pathogens, indicated osmoregulatory dysfunction as a cause of these early losses. Beginning in October 1984, BKD was diagnosed as the continuing cause of mortality that eventually resulted in nearly 99% cumulative mortality (Fig. 4). During this same period, mortality of cohorts in fresh water was only 3.9%. All freshwater mortalities were examined, and BKD was not documented as the cause of death in any case. It is possible that osmoregulatory stress during early seawater residence allowed the fish in seawater to become infected with BKD from other carrier stocks.

On 28 February 1985, a group of 1+ age smolts (n=1,258) of the 1983-brood SRFC were transferred to seawater at Manchester. Prior to transfer, 30 fish were sacrificed and FAT analyses determined that the stock was still BKD-free. An additional 530 fish from this stock were retained at the hatchery to allow freshwater/seawater comparisons. The fish transferred to seawater as 1+ smolts experienced little osmoregulatory difficulty and had good survival for the first 7 months of seawater residence. However, in October 1985, BKD began to be responsible for losses, and the mortality rate and severity were ultimately similar to the 0+ age entry (Fig. 4). By March 1986, the overall seawater mortality for this 1+ smolt entry approached 50%, and BKD was responsible for the majority of deaths. During this same 24-month period (March 1984 to March 1986), only 4% of the cohorts in fresh water had died. Most mortalities in fresh water were due to precocity (jacks); all

freshwater mortalities were examined, and BKD was not documented as the cause of death in any case.

Our experiments revealed a repetitive pattern of apparently BKD-free freshwater stock transferred to seawater ultimately contracting BKD after 6 to 9 months residence, whereas cohorts in fresh water remained BKD-free. This pattern suggested horizontal infection from carrier stocks in seawater. Fryer (1985) reported the potential for BKD transfer in seawater, however, the present experiments are the first large-scale, multi-year documentation of this phenomenon. It is probable that the confinement of other carrier stocks of chinook salmon in close proximity in the net-pen complex induced the high level of BKD infection seen in this experiment. Managers should be aware of the potential for BKD cross-contamination in seawater.

In February 1986, a group of 150 2-year-old 1983-stock SRFC were transferred from fresh water at Big Beef Creek to the USFWS facility at Marrowstone Island, Washington. These fish are being held in a pumped seawater raceway system. The intake for this system is in a natural area with only native fish populations. After 7 months in seawater at Marrowstone Island, only three fish have died, and the cause of death was not BKD. Similarly, the few fish that died in the 1983-brood SRFC retained on groundwater at Big Beef Creek were negative for BKD. In previous years, BKD incidence in seawater has accelerated at 6 to 9 months residence. Continued observation of this stock should help define (presumptive) horizontal seawater BKD transmission in other than high-density aquaculture situations.

Freshwater Brood-stock Potential

Comparison of growth and survival in fresh water and seawater indicates a strong potential for a freshwater brood-stock concept with chinook salmon.

Survival to 2.5 years of age has been almost 90% for 1983-brood SRFC retained in fresh water, whereas less than 2% of the 0+ age and only about 50% of the 1+ seawater entry remain (Fig. 4). In addition, growth has been comparable between these three treatments, indicating that captive freshwater culture has the potential to produce approximately the same size fish as does seawater culture (Fig. 7). Observations continue on these test groups, and fecundity and viability will be compared at spawning (fall 1987).

SUMMARY AND CONCLUSIONS

1. Results of NMFS seawater acclimation trials indicate that Snake River fall chinook salmon should not be transferred to seawater until they are 1+ age smolting fish.
2. Maintaining chinook salmon to maturity in net-pens affords a unique opportunity to observe marine growth and survival during an otherwise inaccessible phase of their life cycle.
3. Our investigations showed there are many factors affecting marine survival of chinook salmon. Marine mortalities first occurred during the osmoregulatory adaptation to seawater. During the first winter of seawater residence, Snake River fall chinook salmon mortality increased markedly due to BKD. Losses to this disease continued until maturity, and mortality can then exceed 30%. During the fall of the following year, chinook salmon were infected with previously undocumented diseases. We have recently identified a pathogen with probable fungal affinities that is responsible for catastrophic losses (95+) in Snake River fall chinook salmon at 3 to 4 years of age in seawater. Another serious adult disease, an infectious anemia, has been observed at the Manchester Marine Experimental Station in captive spring

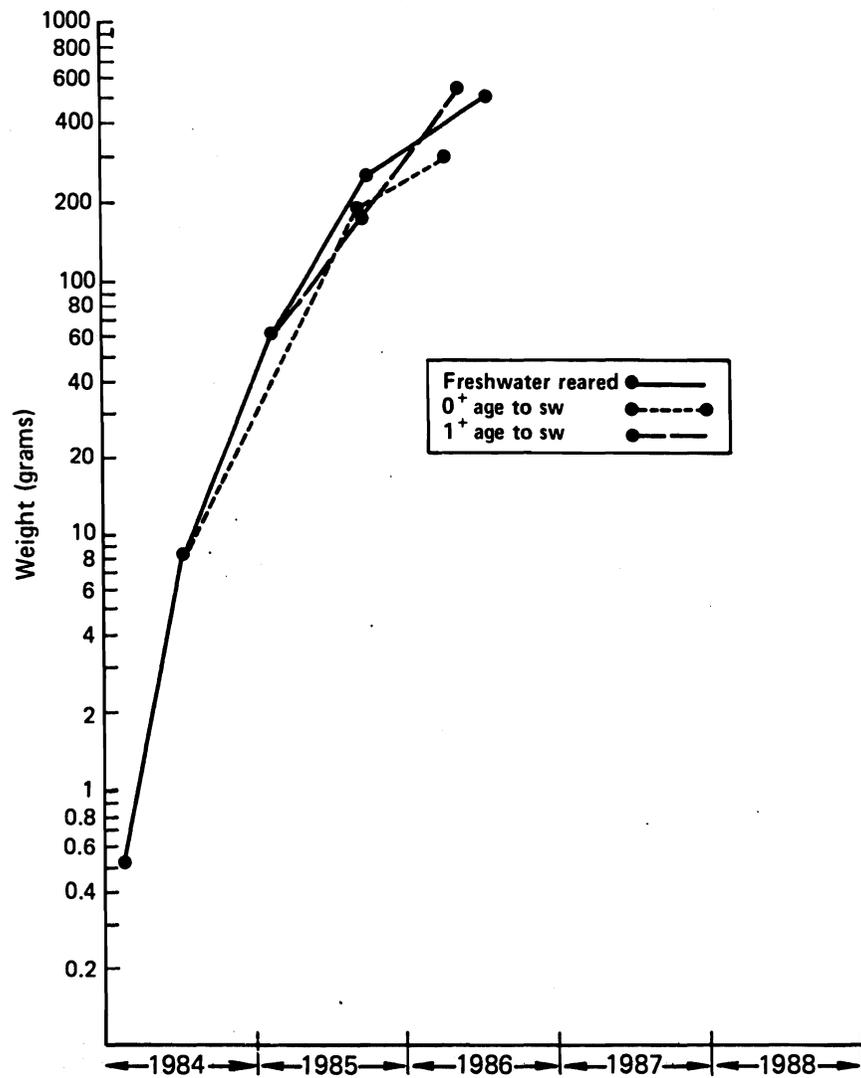


Figure 7.--Growth of 1983-brood Snake River fall chinook salmon in groundwater and after transfers to seawater at 0+ age and 1+ age.

chinook salmon. A better understanding of these diseases may provide insight on problems of high-seas survival.

4. The Snake River fall chinook salmon brood-stock program's egg production goals were not reached because of very low survival of adults to maturity in seawater net-pens. Seven 1980-brood females matured in 1984, and only two of these fish produced viable eggs. Only two females from the 1981-brood reached maturity in 1985, and none of their eggs was viable. These figures would undoubtedly have been repeated in 1986 and 1987 had the program continued. Until an effective method of prevention or control of both BKD and the rosette organism is developed, a Snake River chinook salmon brood-stock program in marine net-pens at Manchester is not feasible.

ACKNOWLEDGMENT

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