Epizootics of Infectious Hematopoietic Necrosis Virus in an Enhanced Population of Sockeye Salmon *Oncorhynchus nerka* Smolts at Chenik Lake, Alaska

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ABSTRACT: Infectious hematopoietic necrosis virus (IHNV) epizootics in emigrating sockeye salmon *Oncorhynchus nerka* smolts occurred in the spring of 1991 and 1992 at Chenik Lake in Southcentral Alaska. The lake historically supported a natural run of sockeye salmon, but lake stocking was initiated following a substantial population decline. An estimated 32,000 smolts in 1991 and 42,000 smolts in 1992 emigrated from the lake, which was substantially less than the 1.0–1.4 million smolts expected. IHNV was isolated from both moribund and apparently healthy smolts. Though smolt mortality was attributed to IHNV, other factors also may have contributed to the reduced numbers. Stressful environmental conditions and high fish density may have precipitated the disease outbreaks. In 1993, smolt abundance was less than expected but IHNV was not isolated. Management plans for the Chenik Lake system include maintaining current escapement levels at 10,000 adults and reducing stocking to between 1.0 and 1.5 million fry.

INTRODUCTION

Infectious hematopoietic necrosis virus (IHNV) causes one of the most widespread viral diseases of cultured salmon and trout in North America. Although the disease is best known for its effect upon alevins and young fry in hatcheries, some outbreaks have been documented in feral populations of juvenile (Williams and Amend 1976; Traxler and Rankin 1989) and older fish (Burke and Grischkowsky 1984; Traxler 1986). Mortality due to IHNV has most often been reported from intensive fish culture situations.

In 1991 and 1992, IHNV was isolated from sockeye salmon *Oncorhynchus nerka* smolts emigrating from Chenik Lake, Alaska. This clear-water lake, accessible only by air, is located on the west side of Cook Inlet in the Kamishak Bay area (Figure 1). Chenik Lake covers 290 acres; maximum depth is 57 m and mean depth is 29 m. Historically, the lake supported runs of sockeye salmon exceeding 100,000 fish annually, but a severe population decline of unknown cause occurred in the 1940s (Nick Dudiak, Alaska Department of Fish and Game, Homer, personal communication). This decline effected closure of the commercial fishery in 1952. From 1978 until 1981 Tustumena Lake sockeye salmon fry incubated at an Alaska Department of Fish and Game hatchery were transplanted into Chenik Lake. As a result, the number of adults returning to the lake substantially increased and allowed the commercial fishery in the Chenik Lake harvest area to reopen in 1982.

Lake fertilization, along with additional sockeye salmon fry transplants, were initiated in 1986. Until the IHNV outbreaks in 1991 and 1992, 2.6–3.5 million Tustumena Lake sockeye salmon fry were transplanted each year. Annual escapement into Chenik Lake from 1982 to 1992 averaged 10,000 adults, consisting of both hatchery-incubated and ferally spawned fish (ADF&G 1994). Yearly salmon runs returning to the lake gradually declined from 1988 through 1994 despite stocking, provoking additional efforts to monitor lake production. This paper provides an overview of smolt production at Chenik Lake from 1991 to 1993, with a focus on the IHNV outbreaks.

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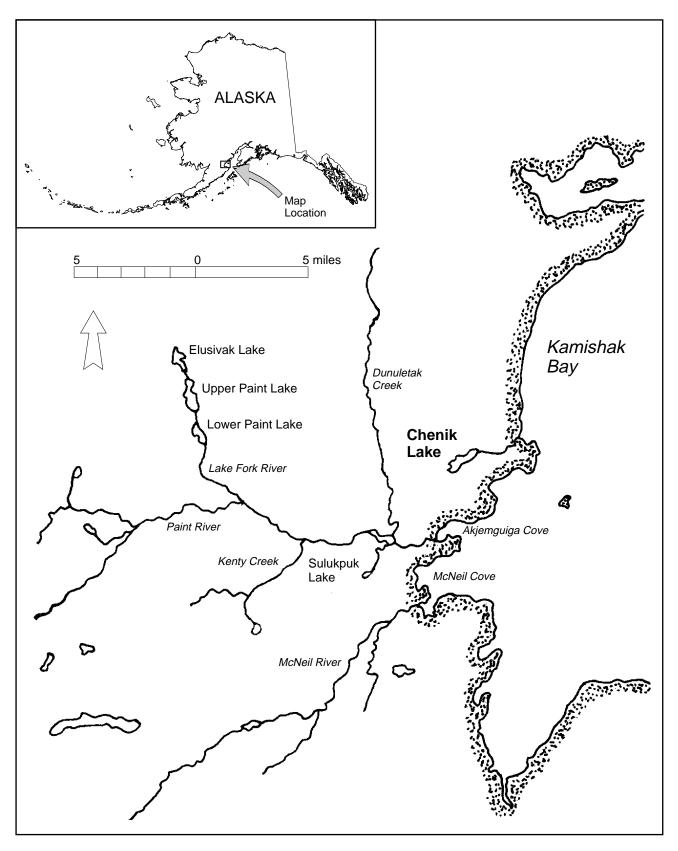


Figure 1. Location of Chenik Lake, Alaska.

EVENTS OF 1991

In July 1990 the Alaska Department of Fish and Game stocked Chenik Lake with 3.2 million Tustumena Lake sockeye salmon fry weighing an average of 0.2 g. The next spring, to enumerate smolt emigrants from the lake, a fyke net leading to a live box was placed in the outlet of the lake to capture emigrating smolts. The lake was almost completely ice-covered when the crew arrived in April 1991 to initiate fyke net monitoring. Smolt mortality was noted around May 1, 1991, when up to 50% of the fish in the collection box were moribund or dead. Dead smolts were also found around the edges of the lake. Moribund smolts exhibited signs typical of IHN disease, including eroded fins with *Saprolegnia* infestation and hemorrhaging of the eye, pectoral fins, and body wall.

Samples of moribund smolts collected several days later on May 3 and 4 were sent to the Alaska Department of Fish and Game Fish Pathology Laboratory in Anchorage for evaluation. Smolts examined at the laboratory averaged 75 mm in fork length and 3.5 g in weight. A total of 20 smolts were processed for virus isolation. Kidney, liver, and spleen tissues were aseptically removed from the smolts and processed in 4 pools of 5 smolts each. The samples were inoculated onto epithelioma papulosum cyprini (EPC) cells using standard viral assay procedures (Fried 1984; Amos 1985). All 4 of the pooled samples tested positive for IHNV, confirmed through a plaque-reduction serum neutralization test using polyclonal rabbit anti-IHNV antiserum. In addition, titers of individual smolts were determined using the plaque-assay technique (Burke and Mulcahy 1980); these ranged from 10^4 to 10^6

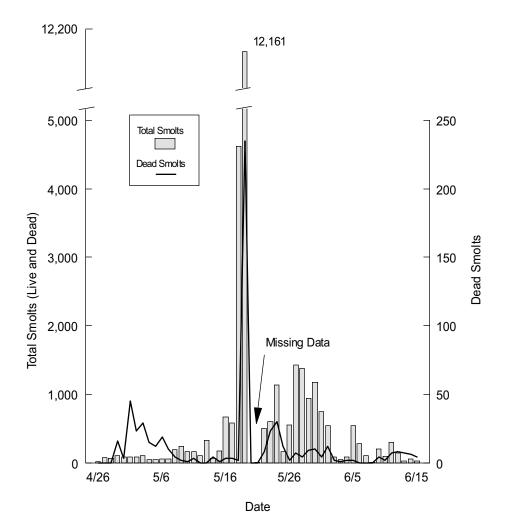


Figure 2. Total daily number of sockeye salmon smolts, migrating and dead, enumerated at the outlet of Chenik Lake, 1991. Arrow indicates 48-h period when counts were not taken due to ice blockage.

plaque-forming units per gram of tissue. No bacterial pathogens were isolated from fish kidney tissues inoculated onto plates of tryptic soy agar. Later, 30 smolts that appeared healthy were also individually tested for the virus and 33.3% were positive for IHNV.

The mortality pattern was typical of a viral outbreak: a rapid onset of high mortality that gradually declined. The highest observed daily mortality, 54% (45/83), was recorded on May 1 (Figure 2). From April 26 to May 7, 172 smolts (21.6%) out of 795 counted were found dead. Starting on May 8 the daily number of smolt emigrants began to substantially increase and reached 4,600 on May 18. Concurrently, the daily occurrence of dead smolts decreased to 0.04%. Both the total number of emigrating and dead smolts peaked on May 19, but the daily percent mortality remained low (1.9%). Daily mortality continued to be low, averaging 1.4% until the middle of June when the smolt emigration ceased.

Daily mortality percentages were greatest just after the lake ice began to break up, at which time numbers of emigrant smolts were relatively low. This high mortality may have been due to moribund smolts near the lake outlet that were unable to resist the current drawing them into the live box, whereas healthy smolts did not leave the lake until conditions were better. During the 2-month monitoring period, an estimated total of 32,000 smolts were caught in the live box, and of these, 599 were found dead. Unfortunately, these counts were considerably below the total number of smolts emigrating because ice at the outlet forced the crew to remove the fyke net for 2 d during peak smolt emigration.

EVENTS OF 1992

In 1992, smolt mortalities with similar clinical signs of disease were again noted in late April (Figure 3). Numerous moribund smolts were observed, and 12 were processed individually for virus isolation. All 12 tested positive for IHNV. Methods used for disease screening were similar to those described for 1991. Daily mortalities of up to 56% continued through the middle of May. Smolt emigration peaked initially on May 19 and secondarily on May 29. While increased numbers of dead smolts were associated with these peaks, daily mortalities were low: 4.4% on May 19 and 2.2% on May 29. Gradually, mortalities tapered off to zero. By June 15 a total of 1,201 dead smolts had been counted out of a total of 42,078 (2.9%) smolts that had been captured by the fyke net and released.

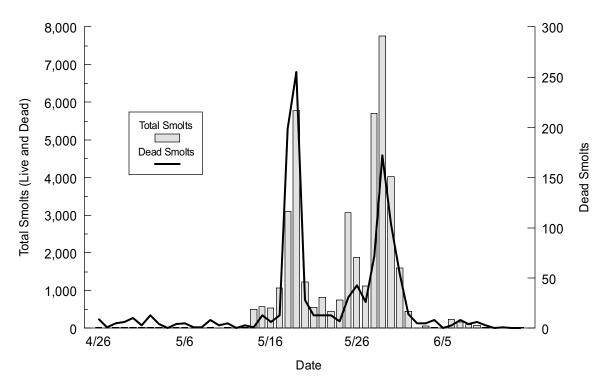


Figure 3. Total daily number of sockeye salmon smolts, migrating and dead, enumerated at the outlet of Chenik Lake, 1992.

EVENTS OF 1993

The total emigration of 14,000 smolts in 1993 was less than half that observed in the previous 2 years. Viral assays of 30 healthy smolts midway through the emigration did not detect IHNV, and there was no evidence that an outbreak had occurred. The lake had been stocked with 2.5 million Tustumena Lake sockeye salmon fry in early July 1992.

A set of 32 ovarian-fluid samples were taken from ripe and post-spawning female sockeye salmon on August 17 and another 30 samples were taken on August 30. The 2 sets of samples were collected on different spawning beds in Chenik Lake. In the first set, 2 of 32 (6.2%) samples were positive for IHNV. In the second set, taken 2 weeks later, 20 of 30 (66.7%) samples were positive for IHNV.

DISCUSSION

Total sockeye salmon runs to Chenik Lake since lake fertilization and stocking began in 1986 averaged over 102,000 sockeye salmon per year until the IHNV outbreak in 1991. After the 1991 outbreak, total sockeye salmon runs decreased significantly to a low of 808 sockeye salmon in 1994. The Chenik Lake commercial fishery, which had provided a significant contribution to the lower Cook Inlet commercial sockeye salmon harvest since 1986, was closed in 1994 and remained closed in 1995. Smolt emigrations based on escapements and transplanted fry numbers for 1991 and 1992 were expected to be 1.0-1.4 million, which was substantially greater than the 32,000 (1991) and 42,000 (1992) smolts counted in the fyke net. How much of this difference was due to mortality from IHN disease is speculative. However, the counts in the fyke net probably do not reflect the full loss due to IHNV because many smolts and transplanted fry probably died prior to emigration, their carcasses remaining in the lake or eaten by scavenging birds and fishes. In addition, smolts that successfully emigrated may have died after leaving Chenik Lake because apparently healthy smolts were also found to be infected with IHNV. Both hatchery and feral smolts, identified through otolith examinations, were infected with the virus.

The source of IHNV in both epizootics was most likely the returning sockeye salmon adults, which in this case was a combination of both hatchery-incubated and ferally spawned fish. The distribution of IHNV in adult sockeye salmon in Alaska is widespread; every anadromous Alaskan sockeye salmon population examined by the Alaska Department of Fish and Game's Fish Pathology Laboratories has had some level of IHNV detected.

The same Tustumena Lake stock of sockeye salmon has been used in Chenik Lake fry transplants for 16 years. Eggs were incubated and fry were reared to the feeding stage each year at Crooked Creek Hatchery. Prior to 1993, IHNV had never been detected in young fry at the hatchery. In 1993, however, fry were held to the presmolt stage for the first time at that facility, and these juveniles did sustain an IHNV outbreak. It is therefore possible that the transplanted sockeye salmon fry were a source of the virus; that is unlikely, however, because no signs of IHN disease were ever reported in these fry. We suggest that adult sockeye salmon returning to the lake were a more likely source for IHNV in Chenik Lake, transmitting the virions through sex products released during spawning and through decomposing carcasses. IHNV can also remain viable for long periods while adsorbed to sediments or the surfaces of spawned eggs (Mulcahy et al. 1983).

The 2 sample groups of adult sockeye salmon screened in 1993 ranged from 6 to 67% positive for IHNV. These returning adults were from feral and transplanted fish leaving the Chenik Lake system before the documented IHNV outbreaks of 1991 and 1992. The difference between the 2 samplings may have been caused by an increase in virus prevalence on the spawning beds over time or by the presence of 2 discrete spawning segments in the run (Mulcahy and Pascho 1986; Mulcahy et al. 1987; Meyers et al. 1990).

Factors involved in precipitating IHNV epizootics are complex and poorly understood. Even in a hatchery situation, where environmental parameters can be easily monitored, epizootics cannot be predicted. Environmental stress often precipitates fish disease outbreaks (Snieszko 1974), but the role of stress in IHNV epizootics is not well documented. Stressful conditions, such as a depleted food supply or a high density of juvenile fish, may have initiated the outbreaks at Chenik Lake. In other documented IHNV outbreaks in Alaskan sockeye salmon smolts, high lake-water temperatures and concurrent infections with other pathogenic agents have been implicated as stressors (Burke and Grischkowsky 1984).

The proximity of the spawning beds to juvenile feeding areas may have exacerbated juvenile exposure to the virus. Also, fry density was relatively high (3.25 million fry were transplanted and 17,000 adults spawned during the summer of 1990), which could

have increased stress. Mulcahy et al. (1983), Mulcahy and Pascho (1986), and our experience have indicated that high densities of mature adults tend to increase virus prevalence by increasing numbers of waterborne virions shed by infected fish.

Outbreaks of IHNV have not been recorded at any site inAlaska where only feral smolts are present, suggesting a causal relationship between hatchery fish and IHNV epizootics. However, mortality in enhanced fish is more likely to be noticed, e.g., when they are monitored for diseases or to evaluate stocking success. Although feral sockeye salmon populations have frequently been enumerated, the focus is not directed at fish health, so epizootics may go largely unnoticed. Since 1993, management plans for Chenik Lake have included disease-related provisions: (1) to maintain escapement levels at or below 10,000 adults per year, thereby reducing virus levels introduced into the lake; and (2) to reduce stocking levels to 1.0–1.5 million fry per year, thereby lessening the chances of fishto-fish virus transmissions. Lower juvenile densities should also produce somewhat larger smolts because of less competition for rearing space and food (Kyle 1994). Increased growth rate is beneficial because IHNV has a greater adverse effect on smaller fingerlings or smolts than on larger ones. Monitoring of Chenik Lake smolts will continue so we can determine if reduced transplants and escapement levels curtail IHNV epizootics.

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