Systemic nematodosis in farmed coho salmon in British Columbia

John R. Brocklebank, David Groman, Gary Conboy

This report describes the clinical signs and pathology associated with a *Philonema oncorhynchi* infection in farmed coho salmon (*Oncorhynchus kisutch*) in British Columbia. The condition was initially diagnosed in February 1994 at a netpen site in the coastal waters of Vancouver Island. At that time, the site contained 27,000 fish (mean weight 0.05 kg) that had been transferred to saltwater pens in January 1994, after initial rearing to 0.03 kg in a freshwater lake during the summer and autumn of 1993. The salmon were distributed evenly between 2 cages, each of which measured 15 m^2 at the surface and extended to a depth of 20 m. Stocking density within the cages averaged 0.3 kg/m^3. Following seawater transfer, the monthly morbidity and mortality rates averaged 0.05% and remained at this level until the fish were destroyed in June 1994. Water quality recorded 3 m below the surface at the time the disease occurred included temperature 6.5°C, 24 ppt salinity, and oxygen ranging from 6.0 to 6.5 mg/L. The cages were regularly flushed free of feces and uneaten feed by tidal movement.

Catchable surface "slowswimmers" or "moribunds" and freshly dead salmon had pale gills and abdominal distension. The abdomen contained a small volume (approximately 0.3 mL) of ascitic fluid. Loosely entwined between the pyloric ceca of affected fish were between 5 and 12, creamy-white, elongated worms that measured 2 mm in diameter and extended to an average length of 10 cm (Figure 1). In the pericardial and retrobulbar cavities, there were 1 to 3 worms, similar in diameter but measuring 1 to 3 cm in length. The stomach was empty and there was little fat between the pyloric ceca.

Samples of liver and kidney from 10 moribund and 10 freshly dead salmon were cultured for bacteria on plain tryptone soy agar (TSA) and on TSA with 5% sheep blood. No bacterial growth was recorded after 10 d aerobic incubation at room temperature. No bacteria were observed in Gram-stained impression smears from liver, kidney, or brain.

Selected tissues from 6 representative moribund salmon were fixed in 10% phosphate buffered formalin (Syndel Laboratories, Vancouver, British Columbia). Microsections (10 μm) were prepared from each fish, stained with hematoxylin and eosin, and examined by light microscopy. Whole parasitic worms from the abdominal cavity from 1 sacrificed fish were forwarded for identification.

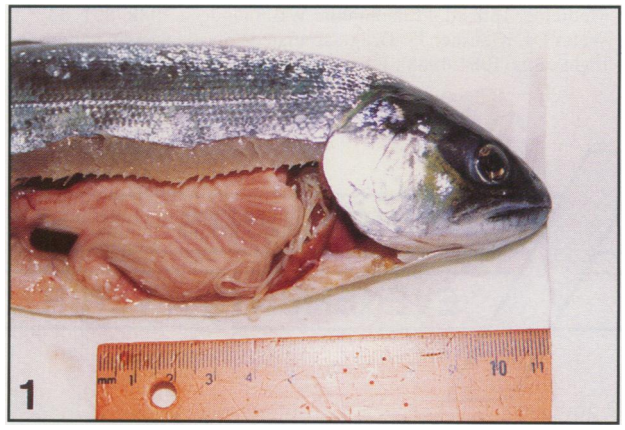

Figure 1. Grossly dissected coho salmon with worms loosely coiled between ceca and liver.

The parasites submitted for identification were nongravid female, dracunculoid nematodes of the genus *Philonema*. Two species have been reported to infect salmonids in North America (*P. oncorhynchi, P. agubernaculum*). Species identification is based on body size, male genitalia, and esophageal morphology (1,2). In this case, the esophageal morphology (ratio of the muscular portion of the esophagus to the glandular portion) of the female worms was consistent with that of *P. oncorhynchi*.

Histological evaluations indicated a fairly long-standing infection (months) of the body cavity by *P. oncorhynchi,* characterized by diffuse fibrogranulomatous (pseudomembranous) perienteritis, perisplenitis, peripancreatitis and steatitis, and loosely attached larval-bearing female worms (Figure 2). Associated mesenteric adipose tissue contained multiple empty pseudocysts, presumably indicating migration tracts. A mixed leucocyte infiltrate was present in the lumen of the pseudocysts, and the peripheral "wall" was composed of fibrogranulomatous tissue. The female worms identified in tissue section appeared to be further advanced in maturation and larval development than those provided for parasite identification. Free immature metazoan larvae, similar in size and microscopic appearance to those noted in female worms, were also present within the vasculature of heart, kidney, and liver (Figure 3). No significant inflammation was found in direct association with these larvae, although the liver of 1 fish contained mononuclear cuffing of bile ducts and increased deposition of interstitial melanin in the kidney. Examination of the body wall, eye, and gill tissues indicated the presence of larvae lodged within hypertrophied dermal scale pockets, loosely associated with ocular musculature, and within interlamellar spaces of the gill, respectively. Adult females, bearing immature larvae, were noted in loose association with filaments.

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640 Haida Street, Comox, British Columbia V9M 2L6 (Brocklebank); Department of Pathology and Microbiology, Atlantic Veterinary College, University of Prince Edward Island, 550 University Avenue, Charlottetown, Prince Edward Island C1A 4P3 (Groman, Conboy).

Address correspondence to Dr. John R. Brocklebank.

Reprint requests to Dr. Gary Conboy.

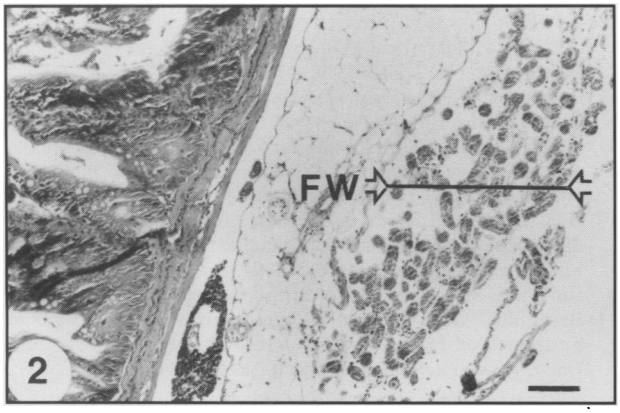

Figure 2. Histological section of gravid female worm adjacent to the serosal surface of the intestine of a coho salmon. FW = female worm. Bar = 100 μm.

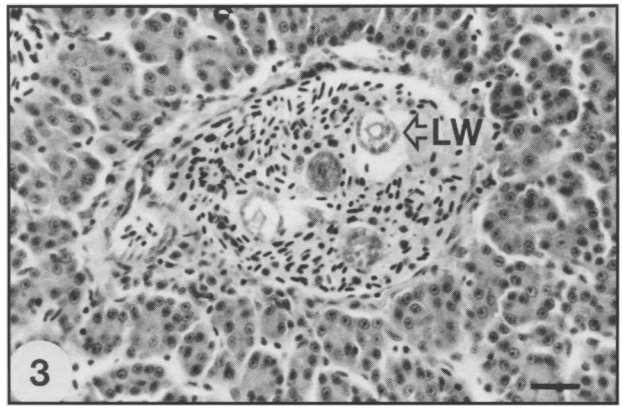

Figure 3. Histological section through liver showing presence of multiple larvae within the lumen of a vessel. LW = larval worm. Bar = 25 μm.

In this case, we believe that the parasitic load produced anorexia that contributed significantly to decreased growth performance of the salmon. In addition, evidence provided from histological examinations indicated further larval development within the uterus of female worms than would normally be expected in similarly infected, yearling wild coho salmon. These findings clearly indicate that *P. oncorhynchi* females can undergo accelerated maturation within the body cavity of farmed yearling coho salmon. It is probable that the development of the parasite in this population was advanced in-tune with the management strategy to produce a fast-growing coho salmon. The coho salmon smolts, in this case, were infected in the freshwater lake pens during early on-rearing and subsequently transferred during the 1st year to seawater.

Due to the lack of tissue damage or inflammation, the larvae associated with gill lamellae, skin, and eye tissue were, most likely, randomly distributed following release from burst females, either during handling procedures from sacrificed moribund coho or on postmortem examination. The finding of larvae within the coho's vascular system is more difficult to explain. There are 4 possible explanations: 1) gravid female worms released larvae in situ, which subsequently entered the circulatory system; 2) large numbers of larvae were released from dead coho and subsequently gained access to the circulatory system; 3) the larvae were from a parasite other than *P. oncorhynchi;* or 4) the larvae represented an arrested developmental stage from the initial infection in freshwater. The latter hypothesis of arrested larval development seems to be the one that is most biologically sound: to the authors' knowledge, no reports exist that detail either premature in situ discharge of larvae or direct access of mature larvae into the circulatory system of salmonids.

Nematodes (*Philonema* spp.) are common parasites of fish, and occasionally infect pen-reared salmon (3). *Philonema oncorhynchi* and *P. agubernaculum* may cause severe visceral adhesions in freshwater, lake-reared, salmon in British Columbia. The clinical significance of these parasites as pathogens is, however, unclear. The prevalence of visceral adhesions decreases with age and maturation in sockeye salmon (*Oncorhynchus nerka*) in the wild, indicating either a transitory condition or, possibly, death loss among severely affected fish (4). Diseases caused by nematodes have not been reported in seawater-reared salmon in the Pacific Northwest (5). In addition, the presence of nematode larvae distributed systemically within the vascular system of salmonids has not been reported previously (3).

The findings reported here indicate a clear potential for early nematode maturation in farmed salmonids, and dictate a prudent approach to accelerated production practices that provide access to infective copepod stages during the freshwater phase of coho production. The life-cycle of *P. oncorhynchi* follows closely that of the host (6–10). Salmon fry become infected by ingesting planktonic cyclopoid copepods (*Cyclops bicuspidatus*) carrying infective 3rd-stage larvae. The parasite is released in the stomach of the fish and migrates to the wall of the swimbladder and, later, to the body cavity, where it increases greatly in size, matures, and mates. Female worms become gravid only in sexually mature fish, from which they are released along with the roe during spawning. The worms burst in water, expelling 1st-stage larvae, which are then ingested by the copepods. The life cycle can take up to 4 y to complete in wild salmonids. Experimentally, premature parasite development has been induced in sockeye salmon treated with pituitary extracts to induce host maturation (11).

Currently, there is no approved chemotherapeutic agent in Canada for the treatment of nematodes of farmed fish, although albendazole has been tried (3). Infection can be minimized by stocking lake netpens in the late spring-early summer months to reduce exposure to infected plankton in the lake. Fish overwintering in netpens could be removed prior to the onset of the spring plankton "bloom" to avoid their becoming infected at this time. Finally, in this outbreak, even though the affected coho salmon were destroyed because of poor growth, the circumstantial evidence linking *P. oncorhynchi* infection with growth performance strongly supports additional studies of the clinical and economic importance of this parasite in aquaculture. CVJ

References

1. Kuitunen-Ekaum E. *Philonema oncorhynchi* nov. gen. et spec. Contrib Can Biol Fish 1993; 8: 71–75.
2. Simon JR, Simon F. *Philonema agubernaculum* spp. nov. (Dracunculidae), a nematode from the body cavity of fishes. Parasitology 1936; 28: 440–442.

3. Kent ML. Diseases of seawater netpen-reared salmonoid fishes in the Pacific Northwest. Canadian Special Publication of Fisheries and Aquatic Sciences, Department of Fisheries and Oceans 1992: 48. Available from Pacific Biological Station, Nanaimo, British Columbia V9R 5K6.
4. Nagasawa K. Prevalence of visceral adhesions in sockeye salmon, *Oncorhyncus nerka* in the central north Pacific Ocean. Fish Pathol 1985; 20: 313–321.
5. Roberts R. Fish Pathology, 2nd ed. Toronto: Baillière Tindall, 1989.
6. Platzer EG, Adams JR. The life history of a Dranunculoid, *Philonema oncorhynchi* in *Oncorhynchus nerka.* Can J Zool 1967; 45: 31–43.
7. Adams JR. Migration route of invasive juvenile *Philonema oncorhynchi* (nematode: Philometridae) in young salmon. J Fish Res Can 1969; 26: 941–946.
8. Ko RC, Adams JR. The development of *Philonema oncorhynchi* (Nematoda: Philometridae) in *Cyclops bicuspidatus* in relation to temperature. Can J. Zool 1969; 47: 307–312.
9. Department of Fisheries and Oceans. Preventing the occurrence of body cavity nematodes (*Philonema*) in salmonids in lake cage culture operations. Canadian Special Publication of Fisheries and Aquatic Sciences, 1994: 66. Available from Pacific Biological Station, Nanaimo, British Columbia V9R 5K6.
10. Department of Fisheries and Oceans. Parasitic worms in trout in lake cage culture operations. Canadian Special Publication of Fisheries and Aquatic Sciences, 1989: 31. Available from Pacific Biological Station, Nanaimo, British Columbia V9R 5K6.
11. Bashirullah AKM, Adams JR. *Philonema oncorhynchi:* Effect of hormones on maturation in anadromous sockeye salmon, *Oncorhynchus nerka.* Int J Parasitol 1983; 13: 261–265.

PRACTITIONERS' CORNER

LE COIN DES PRATICIENS

The economics of using isoflurane in small animal practice

Greg L.G. Harasen

From a strictly clinical perspective, isoflurane has several advantages and few, if any, disadvantages when compared with halothane. However, the much higher price of isoflurane has caused many to restrict its use in private practice.

In considering this agent for our practice, we wondered how the 2 anesthetics would compare on a "cost per minute" or "cost per anesthetic" basis. After converting one of our vaporizers to isoflurane use, we tried to answer this question.

We added a measured amount of isoflurane to our vaporizer and then timed several procedures over the next few weeks. We then drained the vaporizer to determine the total amount used. This gave a cost per minute of anesthetic time. Using our current cost of halothane and assuming a similar rate of consumption, we calculated a comparative cost per minute. Oxygen flow rates ranged from 400–1000 mL/min during these procedures.

Isoflurane used:	70 mL
Total procedure time:	339.35 min
Cost of isoflurane used:	\$48.30 (retail price of \$69.00/100 mL/bottle)
Isoflurane cost per minute:	\$0.142
Cost of same amount of halothane:	\$9.03 (retail price of \$32.25/250 mL)
Halothane cost per minute:	\$0.027

Isoflurane is just over 5 times more costly than halothane by this data. In practice, isoflurane anesthesia lasting 30 min would cost approximately \$4.27, while the same anesthesia using halothane would cost about \$0.80.

This trial does not constitute a controlled study. All or some of the following factors could affect the data: oxygen flow rate; use of rebreathing versus non-rebreathing systems (both were used in this trial); the amount and type of premedication, if any; method of anesthesia induction (all patients in this trial were masked); and individual patient variables. Nevertheless, we feel these figures give a good general idea of the relative costs involved with the use of isoflurane and halothane, and they helped us in establishing fees for the use of isoflurane.

Reference

1. Dohoo SE. Isoflurane as an inhalation anesthetic agent in clinical practice. Can Vet J 1990; 31: 847–850.

Animal Clinic of Regina, 1800 Garnet Street, Regina, Saskatchewan S4T 2Z2.