

WHIRLING DISEASE PREVENTION AND CONTROL:

A REVIEW

Eric J. Wagner
Fisheries Experiment Station,
1465 West 200 North Street
Logan, UT 84321
Pphone: 801-752-1066 Fax: 801-752-6977
e-mail: ewagner@mail.sisna.com

Whirling disease is caused by the myxosporean protozoan *Myxobolus cerebralis*. The disease has been associated with significant declines in rainbow trout *Oncorhynchus mykiss* populations that have been closely monitored on the Madison River in Montana and in Colorado. In the Middle Park, Colorado reach of the Colorado River, Walker and Nehring (1995) observed high mortality of young-of-the-year rainbow trout, reducing age 1+, 2+, and 3+ cohorts to 0.7, 0.5, and 9.7% of the 1994 population, respectively. Subsequent live-cage studies showed that mortality varied among the species tested: brown trout *Salmo trutta*, 2%; Colorado River cutthroat trout *Oncorhynchus clarki pleuriticus*, 10%; Colorado River rainbow trout, 23%; Tasmanian strain rainbow trout 50%. The caged fish exhibited typical signs of the disease, including whirling behavior, skeletal deformities, and black tails. According to an account by H. Novick in the 18 August 1994 "The River Reporter", 95 to 97% of all wild rainbow trout from 1991 to 1993 had been lost in the upper Colorado River. In Montana, rainbow trout densities have dropped 90% in infected reaches of the Madison River (6 April 1995, Missoulian).

Clearly, whirling disease is a significant problem. Its control is difficult given the tenacity of the spore and its longevity in wet muds. The spores can tolerate freezing at -20°C for at least 3 months and the spores are still viable after passage through the guts of predators such as northern pike *Esox lucius*, black-crested night herons *Nycticorax nycticorax*, or mallard ducks *Anas platyrhynchos* (Taylor and Lott 1978; El-Matbouli and Hoffman 1991). There have been reports from Europe of spores remaining viable in dry pond beds for as many as twelve years (Bauer 1962). This article summarizes control methods attempted to date.

For the fish culturist, there are a variety of ways of dealing with the parasite. Since the alternate host for *M. cerebralis* is the oligochaete worm *Tubifex tubifex*, avoiding earthen ponds for culturing fish and keeping concrete systems free of organic waste and sediment are good common sense solutions (Markiw 1992a). Fish are less susceptible to the disease as they grow older, since the cartilage attacked by the trophozoite is largely converted to bone in older fish. Therefore, stocking dirt ponds with larger fish is an option for fish not destined to be stocked in the wild; Hoffman (1990) recommends using fish that are at least 6 cm long. Rasmussen (1965) reported success in Danish trout farms by rearing rainbow trout to 5 cm in concrete tanks before stocking into infected dirt ponds.

Disinfection of in-coming water is possible with ultraviolet radiation. Hoffman (1974) found that 2537 Angstrom units of UV light at dosages of 35,000, 43,000, and 112,000 microwatt sec/cm² were effective in controlling infection of rainbow trout fry. Filtration of water through a 25 µm commercial filter cartridge did not reduce or eliminate the disease (Hoffman 1974). However, Hoffman et al. (1962) noted that sand-charcoal filters had been used successfully in France.

Disinfection of hatcheries and ponds is feasible with chemicals (Table 1). Calcium cyanide was effective in disinfecting ponds, whereas quicklime was less effective (Bauer 1962). Calcium cyanamide (488 g/m²) used for disinfection of dirt ponds and chlorine gas (300 ppm) for disinfection of incoming spring water were effective in preventing the recurrence of whirling disease the following year in a Pennsylvania trout hatchery (Hoffman and Dunbar 1961). Tests with quicklime in simulated tests by Hoffman and Hoffman (1972) were effective in preventing infection of rainbow trout. Treatment of simulated earthen ponds with either 4550 g/m² hydrated lime (CaOH) or 1200 ppm chlorine on the wet mud did not destroy all the spores (Hoffman and O'Grodnick 1977).

However, when the spores were not protected by the mud, 10 ppm chlorine was sufficient to kill the spores (Hoffman and O'Grodnick 1977). Chlorine at 200 ppm gave variable results (Hoffman and Putz 1969). Chlorine at 400 ppm killed 36-90% of spores (Hoffman and Hoffman 1972).

Table 1. A list of chemicals causing distortion and probable death of *Myxobolus cerebralis* spores.

Chemical	Concentration	Citation
Calcium hydroxide	0.5 and 2.0%	Hoffman and Putz (1969)
Calcium oxide (quicklime)	0.25, 0.5, and 1.0% 380 g/m ² (3360 lb/acre)	Hoffman and Hoffman (1972)
Potassium hydroxide	0.25, 0.5, and 1.0%	Hoffman and Hoffman (1972) Uspenskaya (1957)
Sodium hydroxide	1.0%	Uspenskaya (1957)
Available chlorine as sodium hypochlorite	1,600 ppm	Hoffman and Putz (1969)
Alkyl dimethylbenzylammonium chloride (Roccal)	200 and 800 ppm	Hoffman and Putz (1969)
Calcium cyanide	4,000 kg/ha	Bauer (1962)

Prophylactic treatment with chlorine can be effective. Chlorine was effective in reducing infection by 73% in one group and 63% in another group of trout treated weekly with 0.5 ppm for 2 hr over a 4 mo period. Presumably, the triactinomyxon stage of whirling disease and tubeficids were killed by the chlorine, whereas the treatment was not toxic to the fish (Markiw 1992a). The use of chlorine may be hampered by the U.S. Food and Drug Administration (FDA) and state water quality regulations, as it is not an approved compound for discharging from hatcheries.

Table 2. A list of drugs tested for use against *Myxobolus cerebralis*.

Drug	Concentration	Results (% reduction of incidence)		Citation
		Lot 1	Lot 2	
Acetarsone (Stovarsol)	10-1000 mg/kg fish/d (3 d/wk for 6 mo)	suppression		Hoffman et al. 1962
Amprolium	13-18 mg/kg B.Wt	17	0	Taylor et al. 1973
	24-44 mg/kg B.Wt.	50	0	Taylor et al. 1973
Fumagillin DCH	1 g/kg feed fed at 1%B.W.	73		El-Matbouli and Hoffman 1991
Furazolidone	152-194 mg/kg B.Wt.	100	39	Taylor et al. 1973
Merck 930	8-15 mg/kg B.Wt.	0	---	Taylor et al. 1973
	33-64 mg/kg B.Wt.	0	0	Taylor et al. 1973
Nicarbazin	6-14 mg/kg B.Wt.	17	0	Taylor et al. 1973
	30-60 mg/kg B.Wt.	22	0	Taylor et al. 1973
Oxytetracycline	68-152 mg/kg B.Wt.	39	---	Taylor et al. 1973
Sulfamerazine	15-36 mg/kg B.Wt.	0	0	Taylor et al. 1973

Various drugs have also been tested, with limited success (Table 2). The antibiotic Fumagillin (dicyclohexylamine) fed to rainbow trout (medicated pellets contained 0.1% Fumagillin) reduced clinical infections of whirling disease; 73-100% of non-medicated fish had severe infections whereas only 10-20% of medicated fish harbored spores (El-Matbouli and Hoffman 1991). In drug efficacy tests with rainbow X cutthroat trout hybrids fed medicated feed, Taylor et al. (1973) found that furazolidone inhibited spore formation. However, the drug affected palatability and growth in this group was half that of controls. Also, some fish on furazolidone still had trophozoites and granulomas. Russian literature (Bauer 1962) suggested that osarsol added to feed was effective in controlling the disease. Acetarsone (Stovarsol) suppressed the disease, but did not eliminate it (Hoffman et al. 1962). Similarly, Markiw (1992a) noted that furoxone, benomyl, proguanil and clamoxiquin reduced losses and infection of young

salmonids, but none prevented or totally eliminated the disease. Even if these drugs were effective, registration of the drug through the FDA generally requires many years of testing and millions of dollars.

For those trying to manage whirling disease in natural waters, the options are fewer. For programs relying upon stocked fish, stocking larger fish (> 6 cm) should be evaluated. Fish should not be transferred from positive sites. The disease is not considered egg-transmissible, so expansion programs for sensitive species such as cutthroat trout may benefit from egg transfer if no other disease-free sources are available. Stocking of infected fish into infected areas is not recommended. This practice may exacerbate problems by increasing the dose of triactinomyxons. This hypothesis needs to be tested in the wild, but Markiw (1992b) demonstrated that rainbow trout exposed to low numbers (1 or 10) of triactinomyxon did not develop spores. Higher doses of triactinomyxon resulted in proportionately more spores being recovered from infected fish, presumably overwhelming the immune system. In Utah, removal of trout from the upper Fremont River drainage to break the life cycle of the parasite is being tried, but the experiment is still in progress.

For management of naturally reproducing populations in positive waters, selection of resistant species or strains is one of the few options currently available. Salmonids vary in susceptibility, with the following list ranking the most common in order of decreasing susceptibility: rainbow trout, sockeye salmon *Oncorhynchus nerka*, golden trout *O. aguabonita*, cutthroat trout *O. clarki*, brook trout *Salvelinus fontinalis*, steelhead *O. mykiss*, chinook salmon *O. tshawytscha*, Atlantic salmon *Salmo salar*, brown trout, coho salmon *O. kisutch*, lake trout *Salvelinus namaycush*, and splake (lake X brook trout hybrid) (O'Grodnick 1979; Markiw 1992a). Walker and Nehring (1995) reported that the kokanee in Colorado are more resistant to whirling disease than previous literature indicates.

Cutthroat trout are more resistant to the disease than rainbow trout. Walker and Nehring (1995) noted that Snake River finespotted cutthroat trout in a state hatchery that shared a similar lot history with similar-sized rainbow trout were negative ($n = 20$) whereas rainbow trout had infection rates of 65-70%. In a single trial with greenback cutthroat trout *O. clarki stomias*, M. Markiw noted that rainbow trout yielded 15.6 times more spores than the cutthroat trout (Walker and Nehring 1995). With these susceptibility differences in mind, cutthroat trout may be better candidates for stocking or wild-fish management in infected waters, especially in the West.

The best management is to avoid infecting negative waters, containing the infection through enforcement of disease regulations, public education, and disinfection. Thorough drying of contaminated mud can kill spores (Hoffman and O'Grodnick 1977). Heat has been effective in causing the distortion and probable death of spores. Hoffman and Putz (1969) examined spores after heating in 0.85% saline to 60°, 80°, and 100°C. These temperatures were effective in killing spores whereas temperatures of 40°C or room temperature were not. Later tests by Hoffman and Markiw (1977) indicated that heating spores for 10 min at 90°C was effective in killing the spores as determined by methylene blue staining (killed spores take the stain, live spores do not). Heating at lower temperatures progressively reduced the percentage killed (80°C, 98%; 70°C, 60 %; 60°C, 34%; 50°C, 24%) in five trials. Heating for longer periods (up to 100 min) at 70°C increased the percentage of spores that were stainable, but still did not reach 100% (Hoffman and Markiw 1977). Smoking fish at 66°C for 40 min was effective in killing spores (Wolf and Markiw 1982)

Future research into control of the disease is needed. Immunological studies have indicated that rainbow trout produce antibodies against *M. cerebralis*, but protection against infection has not been demonstrated (Griffin and Davis 1978; Markiw 1992a). Enhancement of the immune response may be one avenue of research. Hybrids of resistant salmonid hybrids are being evaluated in Utah for use in infected reservoirs. Resistance of various strains of rainbow trout need to be determined. A greater understanding of the environmental determinants influencing the severity of the disease should lend greater insight in control measures that minimize mortality. Until future research provides additional approaches to controlling whirling disease, the data summarized above should be helpful in the control and eradication efforts.

References

- Bauer, O. N. 1962. The ecology of parasites of freshwater fish. Bulletin of the State Scientific Research Institute of Lake and River Fisheries 49:3-189. (Translated from Russian for the National Science Foundation, Washington, DC by the Israel Program for Scientific Translations, Jerusalem).
- El-Matbouli, M., and R. W. Hoffman. 1991. Effects of freezing, aging, and passage through the alimentary canal of predatory animals on the viability of *Myxobolus cerebralis* spores. Journal of Aquatic Animal Health 3:260-262.
- El-Matbouli, M., and R. W. Hoffman. 1991. Prevention of experimentally induced whirling disease in rainbow trout *Oncorhynchus mykiss* by Fumagillin. Diseases of Aquatic Organisms 10:109-113.
- Griffin, B. R., and E. M. Davis. 1978. *Myxosoma cerebralis*: Detection of circulating antibodies in infected rainbow trout (*Salmo gairdneri*). Journal of the Fisheries Research Board of Canada 35:1186-1190.
- Hoffman, G. L. 1974. Disinfection of contaminated water by ultraviolet irradiation, with emphasis on whirling disease (*Myxosoma cerebralis*) and its effect on fish. Transactions of the American Fisheries Society 103:541-550.
- Hoffman, G. L. 1990. *Myxobolus cerebralis*, a worldwide cause of salmonid whirling disease. Journal of Aquatic Animal Health 2:30-37.
- Hoffman, G. L., and C. E. Dunbar. 1961. Studies on *Myxosoma cerebralis* (Hofer) Plehn (Protozoa: Myxosporidea) the cause of whirling disease of trout. Annual meeting of the American Society of Parasitologists, Abstract 53, August 27-31, Lafayette, IN. Journal of Parasitology 47 (4, section II): 29.
- Hoffman, G. L., C. E. Dunbar, and A. Bradford. 1962. Whirling disease of trouts caused by *Myxosoma cerebralis* in the United States. Special Scientific Report No. 427, U.S. Fish and Wildlife Service, Washington, DC.
- Hoffman, G. L. Sr., and G. L. Hoffman, Jr. 1972. Studies on the control of whirling disease (*Myxosoma cerebralis*). Journal of Wildlife Diseases 8:49-53.
- Hoffman, G. L., and M. E. Markiw. 1977. Control of whirling disease (*Myxosoma cerebralis*): use of methylene blue staining as a possible indicator of effect of heat on spores. Journal of Fish Biology 10:181-183.
- Hoffman, G.L., and J. J. O'Grodnick. 1977. Control of whirling disease (*Myxosoma cerebralis*): effects of drying, and disinfection with hydrated lime or chlorine. Journal of Fish Biology 10:175-179.
- Hoffman, G. L., and R. E. Putz. 1969. Host susceptibility and the effect of aging, freezing, heat, and chemicals on spores of *Myxosoma cerebralis*. Progressive Fish-Culturist 31:35-37.
- Markiw, M. E. 1992a. Salmonid whirling disease. Fish and Wildlife Service Leaflet 17. Washington, D.C.
- Markiw, M. E. 1992b. Experimentally induced whirling disease I. Dose response of fry and adults of rainbow trout exposed to the triactinomyxon stage of *Myxobolus cerebralis*. Journal of Aquatic Animal Health 4:40-43.
- O'Grodnick, J. J. 1979. Susceptibility of various salmonids to whirling disease (*Myxosoma cerebralis*). Transactions of the American Fisheries Society 108:187-190.
- Rasmussen, C. J. 1965. Control of whirling disease in Danish trout farms. European Inland Fisheries Advisory Commission Technical Paper No. 2: 14-15.

- Taylor, R. E. L., S. J. Coli, and D. R. Junell. 1973. Attempts to control whirling disease by continuous drug feeding. *Journal of Wildlife Diseases* 9:302-305.
- Taylor, R. L., and M. Lott. 1978. Transmission of salmonid whirling disease by birds fed trout infected with *Myxosoma cerebralis*. *Journal of Protozoology* 25:105-106.
- Uspenskaya, A. V. 1957. The ecology and spreading of the pathogen of trout whirling disease- *Myxosoma cerebralis* (Hofer, 1903, Plehn, 1905) in the fish ponds of the Soviet Union. *Bulletin of the All-Union Scientific Research Institute Fresh-water Fisheries* 42: 47-55 .
- Walker, P. G., and R. B. Nehring. 1995. An investigation to determine the cause(s) of the disappearance of young wild rainbow trout in the Upper Colorado River, in Middle Park, Colorado. Colorado Division of Wildlife, Brush, Colorado.
- Wolf, K., and M. E. Markiw. 1982. *Myxosoma cerebralis*: inactivation of spores by hot smoking of infected trout. *Canadian Journal of Fisheries and Aquatic Sciences* 39:926-928.