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Whirling Disease Prevention, Control, and Management: A Review

ERIC J. WAGNER
Fisheries Experiment Station, 1465 West 200 North, Logan, Utah 84321, USA
ewagner@sisna.com

ABSTRACT. Whirling disease occurs in salmonid fishes infected by the myxosporean parasite *Myxobolus cerebralis*. This review summarizes the literature regarding control and management of the disease. Suggested methods for killing the myxospore stage include thorough drying, heating for 10 min at 90°C, calcium hydroxide at more than 0.5% for 24 h, and calcium oxide or KOH at more than 0.25% for 24 h. Chlorine was also effective at 1600 ppm in 24-h exposure or 5,000 ppm in a 10-min exposure. Roccal (alkyl dimethylbenzylammonium chloride) at more than 200 ppm active ingredient was also effective. Calcium cyanide at 4,000 kg/ha has been used effectively for control in infected ponds. Treating incoming water with 2537 Å of ultraviolet (UV) light at dosages greater than or equal to 35,000 microwatt-sec/cm^2 was effective in preventing infection of rainbow trout fry. Filtration of water through a 25 µm commercial filter cartridge did not reduce or eliminate the disease, but sand-charcoal filters have been used successfully in France. Prophylactic treatment of incoming water with 0.5 ppm chlorine for 2 h once a week over a 4 month period reduced infection by 63–73%. Electrical charges of 1–3 kV pulsed for 1–25 times at 99 µsec per pulse have killed a high proportion of the triactinomyxon stage. Triactinomyxons were also inactivated by 260 ppm chlorine as sodium hypochlorite, 10% hydrogen peroxide for 10 min, or 50% povidone-iodine for 10 min. Various drugs have been tested, some of which reduced the level of infection but failed to eliminate the parasite. Management strategies to control the disease include control of the worm host and its habitat, stocking larger fish into infected waters, not stocking infected fish, education, enforcement of disease regulations, and stocking less susceptible species.

Whirling disease, caused by the myxosporean *Myxobolus cerebralis*, has been associated with significant declines in wild rainbow trout *Oncorhynchus mykiss* populations in Montana and Colorado. In both states, yearling rainbow trout densities indicated a 90% drop in young-of-the-year abundance in infected reaches of important fisheries, such as the Madison, Gunnison, and Colorado Rivers (Vincent 1996; Nehring and Walker 1996). The parasite has a widespread distribution in the United States in regions where trout are reared, and it is recognized as a prohibitive pathogen by many states and countries. Clearly, whirling disease is a significant problem.

In its life cycle, the parasite infects two hosts, alternating between a salmonid host and the oligochaete worm *Tubifex tubifex* (Markiw and Wolf 1983). The triactinomyxon is the developmental stage of the parasite released by the worm and is the stage that infects the fish host. Consumption of infected worms may also result in infection (Wolf and Markiw 1984). The triactinomyxon is a type of actinosporean, a group of organisms once thought to be separate species that parasitized worms (Janiszewska 1955) but now are considered alternate stages of myxosporean parasites (Wolf and Markiw 1984; Kent et al.1994; Brinkhurst 1996). After entering the fish host, the sporoplasm cells of the triactinomyxon replicate and become trophozoites, feeding on cartilage tissue (Hedrick et al. 1998). This presporogonic phase then shifts to the sporogonic phase, which culminates in the formation of the mature myxospore (Hedrick et al. 1998). Upon death of the fish host, the myxospore is released into the environment, where its consumption by the worm host renews the cycle. Control of the parasite is aimed at these various stages, which differ greatly in susceptibility to control measures.

Prior to understanding the complete life cycle, research was primarily aimed at the resistant myxospore stage. The myxospores can tolerate freezing at –20°C for at least 3 months and 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 Hoffmann 1991a). There have been reports from Europe of myxospores remaining viable in dry pond beds for twelve years (Bauer 1962).

This article summarizes whirling disease control measures attempted to date. These measures include methods for the control of the worm host and its habitat, physical and chemical control of both the myxospore and triactinomyxon stages, and oral drugs aimed at the trophozoite stage. These measures are largely applicable to aquaculture. Also discussed are some general guidelines for management of the disease in the wild and the scientific rationale behind them.

CONTROL STRATEGIES

Control of Worm Host

One control strategy is to break the life cycle by removal of the worm host that produces the triactinomyxon stage. This approach is more applicable to aquaculture, but recent experimentation with electricity suggested that treatment of 'hot spots' in the wild may soon be practical (R. Ingraham and T. Claxton, Hamilton High School, Montana, personal communication). Using an electrophoresis chamber and worms from a commercial supplier, these students found that after 1,000-s exposure to low-level DC voltage, almost all the worms were dead within 48 h.

Thorough drying of ponds may be effective in certain climates. However, *Tubifex tubifex* has the ability to form resistant cysts, living for up to 14 d in dried mud (Kaster and Bushnell 1981). If the encysted worms were wetted periodically, they survived up to 70 d. Worms may also burrow down to moist depths that never dry. No work has been conducted, to date, that evaluates the ability of an infected worm to produce actinospores after encysting.

The lampricide TFM (3-trifluoromethyl-4-nitrophenol) has been used to control sea lamprey *Petromyzon marinus* in the Great Lakes, without harming fish. In an evaluation of the chemical's effect on nontarget organisms (Lieffers 1990), a reduction in the abundance of oligochaetes and other invertebrates was observed after treatment (4.2–14.0 mg/L). The widespread killing of nontarget organisms makes this chemical and others inappropriate for treatment of field ecosystems. However, it may be applicable for aquaculture ponds. In ponds, treatment without fish present would be advisable, since TFM can reduce oxygen and increase ammonia, apparently through its effect on photosynthesis (Dawson et al. 1992). Toxicity of TFM to fish is pH-dependent, increasing at pH 7.25 or lower (Bills and Johnson 1992). Further testing may provide appropriate dosages and application techniques for controlling oligochaete worms with fish present, as well as without.

A number of researchers have examined the effects of water quality variables and pollutants on the survival of *T. tubifex* (Chapman et al. 1982; Whitley 1968; Whitten and Goodnight 1966). For aquaculture applications, manipulation of water quality variables would be more practical for control of the worms than chemicals potentially toxic to fish. For pH, Whitley (1968) reported 72-h LC_{50} values of 5.8 and 9.7 at 20°C for a mix of *T. tubifex* and *Limnodrilus hoffmeisteri* without sediment. Chapman et al. (1982) reported that 96-h LC_{50} values for the acid and basic ends of the pH spectrum were pH 3.5 and 10.5, respectively, for *T. tubifex* held at 10°C without sediment. In tests on the effects of salinity, *T. tubifex* was more tolerant than many other freshwater species, with a 96-h LC_{50} of 14‰ in sediment (Chapman et al. 1982). A mixed culture of *T. tubifex* and *L. hoffmeisteri* was tolerant of shorter exposures in 2 cm of fine silt; salinities of 95‰ for 100 min or 24 h resulted in mortalities of only 18% and 60.6%, respectively (E. Wagner, unpublished data).

High temperature may be more effective in eliminating worms than salinity or application of other chemicals that cannot penetrate the sediment, but achieving the high temperatures presents challenges as well. The 96-h lethal temperature (LT_{50}) was 34°C in a study by Birtwell and Arthur (1980) and 35°C in another (Chapman et al. 1982). Shorter exposures to high temperatures on a mix of *T. tubifex* and *Limnodrilus hoffmeisteri* have recently been tested; LT_{50} values ranged from 38.1°C to 39.2°C in 10-min exposures and from 33.8°C to 36.6°C in 100-min exposures of worms acclimated to 6°C, 9°C, 13°C, or 22°C (Wagner and coauthors, Utah Division of Wildlife, unpublished data). Sublethal high temperature may be effective in controlling triactinomyxon release (El-Matbouli et al. 1999). These authors noted that infected worms transferred from 15°C to 25°C or 30°C ceased triactinomyxon production after 4 d, and developmental stages had degenerated, effectively purging the worm of infection.

Chemical and Physical Control Methods for Myxospore and Actinospore Stages

A variety of physical and chemical control measures have been tested. Many of these studies were conducted before the role of *T. tubifex* in the life cycle was known, so the measured result was a decrease in infection without a determination of where the life cycle was broken. For example, Hoffman and O'Grodnick (1977) noted that contaminated mud dried for 13–19 months was incapable of inducing infection, possibly indicating that the myxospore stage is susceptible to desiccation. Alternatively, the lack of the worm host in the test tank may have prevented infection of the fish. Similar observations are noted for the study of Hoffman and Hoffman (1972), in which quicklime (CaO) applied to simulated ponds at concentrations of 380 g/m^2 or greater, for two weeks, prevented infection of rainbow trout (Table 1). Treatment of simulated earthen ponds (2 cm infected mud in aquaria) with either 4,550 g/m^2 hydrated lime (CaOH) or up to 1,200 ppm chlorine, for 18–24 h, did not destroy all the myxospores (Hoffman and O'Grodnick 1977).

Various chemicals applied to aquaculture ponds in field tests have been reported to reduce infection. Calcium cyanide was effective in disinfecting ponds, whereas CaO was less effective (Table 1; Bauer 1962). Calcium cyanamide (488 g/m^2) applied to dirt ponds, combined with chlorine gas (300 ppm) disinfection of incoming spring water, prevented recurrence of whirling disease the following year in a Pennsylvania trout hatchery (Hoffman and Dunbar 1961). However, some German culturists prefer CaO because it does not have the undesirable nutrient-fertilizing effect of calcium cyanamide (Hoffman and Hoffman 1972).

Chlorine has been one of the most effective chemicals for disinfection. For supernatant from infected mud, 10 ppm chlorine for 30 min was sufficient to prevent infection of rainbow trout fry (Hoffman and O'Grodnick 1977). Likely, the chlorine killed triactinomyxons in the supernatant water, rather than the myxospore, which would have needed the worm host to complete the parasite's life cycle. Research by Wagner and his coauthors (Utah Division of Wildlife, unpublished data) also indicated that triactinomyxons are sensitive to chlorine, succumbing to concentrations as low as 13 ppm in 10-min treatments. However, because low temperatures reduced the efficacy, 130–260 ppm chlorine for 10 min was recommended for 100% kill of triactinomyxons. For treatment of the myxospore, 200 ppm chlorine, for 18–24 h, gave variable results (Hoffman and Putz 1969); 400 ppm killed 36–90% of myxospores (Hoffman and Hoffman 1972). Recent data from E. MacConnell and her coauthors (U.S. Fish and Wildlife Service, Bozeman, Montana, personal communication), based on methylene blue vital staining (dead spores take the stain, live spores do not), suggested that 5,000 ppm chlorine, for 10 min, was sufficient for destruction of myxospores.

Another control strategy in hatcheries is to disinfect incoming water. Ozonation has also shown promise in preliminary tests for treatment of the actinospore stage (Hedrick et al. 1998). Markiw (1992a) noted that a hatchery water supply treated for four months with 0.5 ppm chlorine, for 2 h once a week, reduced infection by 63% and

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

Chemical	Concentration	Duration	Citation
Calcium hydroxide	0.5 and 2.0%	24 h	Hoffman and Putz 1969
Calcium oxide (quicklime)	0.25, 0.5, and 1.0%	6, 2, & 2 d	Hoffman and Hoffman 1972
	380 g/m2 (3360 lb/acre)	2 weeks	
Potassium hydroxide	0.25, 0.5, and 1.0%	2 d	Hoffman and Hoffman 1972
Sodium hydroxide	0.5%	24 h	Hoffman et al. 1962
Available chlorine as	1,600 ppm	24 h	Hoffman and Putz 1969
sodium hypochlorite	5,000 ppm	10 min	MacConnell et al., personal communication
Alkyl dimethylbenzylammonium chloride (Roccal®)	200 and 800 ppm	24 h	Hoffman and Putz 1969
Calcium cyanide	4,000 kg/ha	no data	Bauer 1962

73% in two lots of trout. This prophylactic treatment did not harm the fish. However, the use of chlorine may be prohibited 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.

Filtration has not been effective when using membranes to prevent passage of triactinomyxons, but three-dimensional material has been a better filter. For example, filtration of water through a 25 µm commercial filter cartridge did not reduce or eliminate the disease (Hoffman 1974). Research by the author has shown that triactinomyxon recovery from 10 or 20 µm filters decreased with increasing water volume filtered. In these tests, water spiked with a known number of *M. cerebralis* triactinomyxons was filtered through either 10 or 20 µm mesh Nitex cloth. Recovery rates of 80 ± 26 (SD) and 62 ± 6% for 10 and 20 µm filters, respectively, were recorded when 2 L were filtered through, but this rate dropped to 54 ± 17 (10 µm) and 40 ± 32% (20 µm) when 8 L was filtered. The filtrate was not examined for triactinomyxons due to the large volume, so it is not clear if the triactinomyxons that were not recovered had passed through or were somehow destroyed in the filtration process. Hoffman et al. (1962) noted that sand-charcoal filters had been used successfully in France.

Hoffman (1974) found that treating water with 2537 Å of ultraviolet (UV) light, at dosages of more than 35 milliwatt-sec/cm^2 (mWs/cm^2), was effective in preventing infection of rainbow trout fry. At lower doses, 18 and 27.65 mWs/cm^2, infectivity was reduced by 31–86% and 86–100%, respectively, in comparison to control fish in untreated water (Hoffman 1975). Hoffman (1975) noted that soviet researchers have also had some success using UV treatment for whirling disease control (Ivanov et al. 1968). O'Grodnick and Gustafson (1974) successfully treated a flow of 31 L/min with 35 mWs/cm^2 of UV after filtration through a 25 µm mesh filter. Hedrick et al. (2000) noted that a dose of 1,300 mWs/cm^2 was required to inactivate 100% of triactinomyxons held under a static collimated beam of UV. Ultraviolet treatment systems have been developed recently for decontaminating water infected with another pernicious parasite, *Cryptosporidium parvum* (Clancy et al. 1998). The Cryptosporidium Inactivation Device (CID), tested by Clancy et al. (1998), relies on trapping pathogens on 2 mm sintered stainless steel filters and irradiating them with UV light. Hedrick et al. (2000) tested the device at UV dosages of 4,000–8,000 mWs/cm^2 and found no evidence of infection among juvenile rainbow trout dosed with 7,539 or 64,200 irradiated triactinomyxons/fish.

In work with the triactinomyxon stage, using vital stains, Wagner and coauthors (Utah Division of Wildlife, unpublished data) evaluated the effects of various physical and chemical treatments (Table 2). Thorough drying (>1 h) was effective in killing the actinospore stage. They also found that hydrostatic pressure up to 6.2×10^7 Pa (9,000 psi) for 5 min significantly reduced viability to 43–60%, compared with 70–74% viability in controls. Recent research (E. Wagner et al., this volume) has indicated that electrical charges of 1–3 kV, pulsed for 1–25 times at 99 µsec/pulse, killed a high proportion of triactinomyxons. Drying or freezing triactinomyxons for at least an hour was effective in killing 100%. Lethal concentrations to triactinomyxons were high when using either hydrogen peroxide (10% H_2O_2 for 10 min) or povidone-iodine

Table 2. Summary of effective physical and chemical treatments for killing the triactinomyxon stage of *Myxobolus cerebralis*. CID = *Cryptosporidium* inactivation device.

Treatment	Concentration	Duration	Citation
Chlorine as sodium Hypochlorite	260 ppm	10 min	Wagner et al., personal communication
	10 ppm	30 min	Hoffman and O'Grodnick 1977
Povidone-iodine	50%	60 min	Wagner et al., personal communication
Hydrogen peroxide	10%	10 min	Wagner et al., personal communication
UV light-static	1300 mWs/cm^2		Hedrick et al. 2000
UV light-CID	4,000–8,000 mWs/cm^2		Hedrick et al. 2000
Temperature	75°C	5 min	Wagner et al., personal communication
Drying		>1 h	Wagner et al., personal communication
Freezing		>1 h	Wagner et al., personal communication
Electricity	25 99-µsec pulses of 3 kV each		Wagner et al., this volume

(50% for 60 min). Temperatures above 75°C, for at least 5 min, were lethal. Markiw (1992b) noted that triactinomyxon survival was temperature dependent; only 2 d at 23–24°C, but 7–8 d at 7°C.

Heat has been effective in causing the distortion and probable death of myxospores. Hoffman and Putz (1969) examined spores after heating in 0.85% saline to 60°C, 80°C, and 100°C. These temperatures were effective in killing myxospores, 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. Heating at lower temperatures progressively reduced the mortality percentage (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 dead spores but still did not reach 100% (Hoffman and Markiw 1977). Despite the lower temperature, smoking fish at 66°C for 40 min was effective in killing spores (Wolf and Markiw 1982).

Drug Treatment

Various drugs have been tested for their ability to reduce the severity of clinical whirling disease, with limited success (Table 3). Medicated pellets, containing 0.1% fumagillin (dicyclohexylamine), fed to rainbow trout reduced clinical infections in two tests (El-Matbouli and Hoffmann 1991b). In the first test, fish were fed from day 30 to day 160 post infection and, in the second, from day 14 to day 64 post infection; 73–100% of nonmedicated fish had severe infections, whereas only 10–20% of medicated fish harbored spores (El-Matbouli and Hoffmann 1991b). Staton et al. (this volume) also tested fumagillin at several hatcheries, using either natural exposure or controlled exposure to triactinomyxons. At doses of 3.7 or 7.5 mg/kg body weight per day, for 10 d, fumagillin was not effective in controlling the disease. Furthermore, higher mortality in some treatment groups indicated possible toxicity from the drug, suggesting that higher doses hold little promise. For another myxosporean *Thelohanellus hovorkai*, a 0.1% fumagillin diet (10 mg/kg fish) fed to koi carp *Cyprinus carpio*, for 3–7 weeks, significantly reduced prevalence rates and eliminated mortality and morbidity (Yokoyama et al. 1999). Molnar et al. (1987) also noted significantly reduced infections of *Sphaerospora renicola* after feeding common carp *Cyprinus carpio* 0.1% fumagillin in the feed. However, control of *Myxobolus cyprini* or *Thelohanellus nikolskii* in the same fish was not achieved with fumagillin treatment (Molnar et al. 1987), indicating differences among myxosporeans in susceptibility to the drug.

In drug efficacy tests with rainbow × cutthroat trout hybrids fed medicated feed, Taylor et al. (1973) found that furazolidone inhibited spore formation. However, the drug affected feed palatability, and growth in this group was half that of con-

Table 3. A list of drugs tested for use against *Myxobolus cerebralis*.

Drug	Dosage	Reduction of incidence (%) Lot 1	Lot 2	Citation
Acetarsone (Stovarsol)	10–1000 mg/kg fish/d (3 d/week for 6 months)	suppression[1]		Hoffman et al. 1962
Amprolium	13–18 mg/kg fish	17	0	Taylor et al. 1973
	24–44 mg/kg fish	50	0	Taylor et al. 1973
Fumagillin	1 g/kg feed fed at 1% of body weight	73		El-Matbouli and Hoffman 1991
	3.7 or 7.5 mg/kg fish for 10 d	0		Erdahl and Staton, personal communication
Furazolidone	152–194 mg/kg fish	100	39	Taylor et al. 1973
Merck 930	8–15 mg/kg fish	0	—	Taylor et al. 1973
	33–64 mg/kg fish	0	0	Taylor et al. 1973
Nicarbazine	6–14 mg/kg fish	17	0	Taylor et al. 1973
	30–60 mg/kg fish	22	0	Taylor et al. 1973
Oxytetracycline	68–152 mg/kg fish	39	—	Taylor et al. 1973
Sulfamerazin	15–36 mg/kg fish	0	0	Taylor et al. 1973

[1]No percentages given. This data was cited from Scolari (1954). Additional trials by Hoffman et al. (1962), using concentrations up to 100 times that recommended by Scolari, (1954) indicated this was not a promising drug.

trols. 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, but no concentrations or treatment regimen was given. Acetarsone (Stovarsol) used at 10 mg/kg body weight for 3 d suppressed the disease but did not eliminate it (Hoffman et al. 1962). Alderman (1986) tested the same drug and found no reduction in pathology, spore frequency, and severity. However, the drug proguanil reduced the frequency of pathology and spores, as well as severity of the disease (Alderman 1986). Alderman (1986) also found that clamoxyquin reduced the frequency of clinical signs, though to a lesser extent than proguanil, but was more effective at reducing the frequency and severity of sporulation. Similarly, O'Grodnick and Gustafson (1974) noted that furoxone and benomyl reduced losses and infection of young salmonids, but none prevented or eliminated the disease. Even if these drugs were effective, registration of these drugs through the FDA for use with fish would require many years of testing and millions of dollars of funding.

MANAGEMENT STRATEGIES

Reduce or Eliminate Habitat of Alternate Host

Areas of high sediment loading have been observed to be 'hot spots' of parasite infection, producing higher numbers of triactinomyxons, resulting in higher prevalence rates and more severe infections (Thompson and Nehring 2000; Allen 1999). For wild fish, efforts to reduce introduction of sediment into the streams should help reduce the amount of habitat for the parasite's alternate host. This means controlling excess sediment from sources such as road construction, logging, grazing, mining, and recreation. Benefits would not only include reduced worm habitat, but improved egg and fish survival, less gill damage, better growth rates, and reduced stress as well (Newcombe and MacDonald 1991; Lake and Hinch 1999). For aquaculturists, avoiding earthen ponds for culturing fish and keeping concrete systems free of organic waste and sediment are good management strategies (Markiw 1992a). Infected hatcheries in Colorado have applied this strategy, in concert with disinfection of incoming water, and have been able to avoid reinfection (Nickum 2000). The Bellevue-Watson Fish Hatchery of Colorado has implemented a strategy of lining earthen ponds with 50-mil white plastic sheets, then alternating culture between ponds every 2 months (Nickum 2000). The pond not in use is drained and cleaned, removing any habitat for worms as well as any worms that may have been infected. Since incubation of the spore in the worm host takes about 3 months before triactinomyxons are produced (Markiw 1992a), infection of fish is avoided.

Stocking Strategies

Stocking larger fish may be a practical alternative for aquaculturists and state wildlife agencies. Fish are less susceptible to the disease as they grow older (Markiw 1992c), since the cartilage attacked by the trophozoite is largely converted to bone in older fish. Rasmussen (1965) reported significant improvements in survival of rainbow trout in Danish trout farms, by rearing fry to 5 cm total length in concrete tanks before stocking into infected dirt ponds. Hoffman (1990) recommended using fish that are at least 6 cm long. E. Wagner and coauthors (unpublished data, Utah Division of Wildlife Resources) noted that fingerling rainbow trout stocked at a size of 10 g, or about 100 mm, into an irrigation reservoir, survived well and provided an excellent fishery, despite infection levels reaching 100% after stocking. E. K. N. Ryce and her coauthors at the Montana Cooperative Fisheries Research Unit (Montana State University, personal communication) have noted in laboratory tests that mortality dropped significantly if rainbow trout were exposed after 9 weeks post hatching. Thompson et al. (1999) also noted that rainbow and cutthroat trout naturally exposed to *M. cerebralis* infection at larger mean weights survived better than smaller counterparts.

Stocking of infected fish into parasite-endemic areas, as well as disease-free areas, is not recommended. This practice may exacerbate problems by increasing the dose of triactinomyxons. Markiw (1992c) demonstrated that rainbow trout exposed to low numbers (1 or 10) of triactinomyxons did not develop spores. Higher doses of triactinomyxons resulted in proportionately more spores being recovered from infected fish, presumably overwhelming the immune system. Nehring and Thompson (2001) observed a similar relationship between the triactinomyxon numbers and infection severity in the wild. Fish should not be transferred from positive sites (Hoffman et al. 1962). The disease is not considered egg-transmissible

(O'Grodnick 1975), so expansion programs for sensitive species, such as cutthroat trout, may benefit from egg transfer, if disease-free sources are not available.

For management of naturally reproducing populations in positive waters, selection of resistant species or strains for stocking is one of the few management options currently available. Salmonids vary in susceptibility (see MacConnell and Vincent, this volume; O'Grodnick 1979; Markiw 1992a). Selection for resistance may take many generations, as Ryce et al. (2001) noted no difference in susceptibility to *M. cerebralis* infection between rainbow trout recruited during the early years of infestation and those recruited before. Grayling appeared to be resistant to infection at doses of 1,000–2,000 triactinomyxons/fish, whereas this dose caused infection in bull trout *Salvelinus confluentus* (Hedrick et al. 1999). Cutthroat trout are more resistant to the disease than rainbow trout and produce fewer myxospores per fish (Walker and Nehring 1995; Hedrick et al. 1999; Markiw 1992a). In contrast, Thompson et al. (1999) found that three subspecies of cutthroat trout native to Colorado suffered higher mortality than rainbow trout exposed to the same ambient levels of triactinomyxons within the Colorado River over a period of 9 months. In that study, chronic stress, resulting from the use of cages (Strange et al. 1978), may have been a factor in the difference. With these susceptibility differences and lower spore load in mind, cutthroat trout may be better candidates than rainbow trout for stocking or wildfish management in infected waters, especially in the West. The lower spore load should help reduce the number of triactinomyxons being produced in an area and, in turn, reduce disease severity.

Among the geographic variants of some species, there may be slight differences in susceptibility. For example, E. Wagner and coworkers (unpublished data, Utah Division of Wildlife) found that after exposure to 1,000 triactinomyxons/fish, the Bear Lake Bonneville cutthroat trout *O. clarki utah* had significantly lower prevalence rates than a southern Bonneville form, Snake River cutthroat trout or Yellowstone cutthroat trout *O. clarki bouvieri*.

Other Strategies and Future Research Needs

The best management is to avoid infecting negative waters, containing the infection through enforcement of disease regulations, public education, and disinfection. Another management option is the use of different life history strategies of salmonids that reduce the risk of infection. For example, rainbow trout in the Madison River in Montana spawn either in mainstem sites or in the tributaries (Downing 2000). Fry emerging from tributary redds may experience lower triactinomyxon doses or not be exposed at all until entry into the mainstem portion of the river. Differences in spawning times may also be a factor in infection. Fry of early spawners may emerge sooner, possibly reducing exposure to infection until a later age at which they are less vulnerable. These life history differences will likely be selected naturally by the populations in the river, where other variables that affect mortality, such as food availability, flow, and predation risk, will guide selection for the most appropriate survival strategy. Monitoring of these natural selection variables, in relation to whirling disease, is a daunting task. Nonetheless, it should provide insight into the role of *M. cerebralis* and other parasites in the natural selection process.

Future research into control of the disease is needed. Further exploration of electrical charge effects and designing these into affordable and practical devices for disinfection is needed. Also, research into the mechanisms of polar filament discharge may provide a means of controlling the parasite. Premature discharge, induced artificially, could prevent later infection of fish.

Enhancement of the immune response may be one avenue of research. 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). Low levels of actinospore exposure over time need to be tested to see if this results in increased antibody titers and reduced susceptibility to subsequent infection. Benign species of *Myxobolus* may induce immunity if used as vaccines, either used as a whole or by focusing on certain immunogenic antigens. A greater understanding of the environmental determinants influencing the severity of the disease should lend greater insight into control measures and management strategies that minimize mortality. Until future research provides additional approaches to controlling whirling disease, the data summarized above should be helpful in efforts directed at control and eradication of the parasite.

REFERENCES

Alderman, D. J. 1986. Whirling disease chemotherapy. Bulletin of the European Association of Fish Pathologists 6(2):38–40.

Allen, M. B. 1999. Factors influencing the distribution of *Myxobolus cerebralis*, the causative agent of whirling disease, in the Cache LaPoudre River, Colorado. Master's thesis, Colorado State University, Fort Collins, Colorado.

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).

Bills, T. D., and D. A. Johnson. 1992. Effects of pH on the toxicity of TFM to sea lamprey larvae and nontarget species during a stream treatment. Great Lakes Fishery Commission Technical Report 57:7–19.

Birtwell, I. K., and D. R. Arthur. 1980. The ecology of tubificids in the Thames estuary with particular reference to *Tubifex costatus* (Claparéde) and *Tubifex tubifex* (Müller). Pages 331–381 *in* R. O. Brinkhurst and D. G. Cook, editors. Aquatic oligochaete biology. Plenum, New York.

Brinkhurst, R. O. 1996. On the role of tubificid oligochaetes in relation to fish disease with special reference to the Myxozoa. Annual Review of Fish Diseases 6:29–40.

Chapman, P. M., M. A. Farrell, and R. O. Brinkhurst. 1982. Relative tolerances of selected aquatic oligochaetes to individual pollutants and environmental factors. Aquatic Toxicology 2:47–67.

Clancy, J. L., T. M. Hargy, M. M. Marshall, and J. E. Dyksen. 1998. Inactivation of *Cryptosporidium parvum* oocysts in water using ultraviolet light. Journal of the American Water Works Association 90:92–102.

Dawson, V. K., D. A. Johnson, and J. F. Sullivan. 1992. Effects of the lampricide 3-trifluoromethyl-4-nitrophenol on dissolved oxygen in aquatic systems. Great Lakes Fishery Commission Technical Report 57:21–33.

Downing, D. C. 2000. Spawning, and rearing ecology of Madison River rainbow trout in relation to whirling disease infection risk. Master's thesis, Montana State University, Bozeman, Montana.

El-Matbouli, M., and R. W. Hoffmann. 1991a. 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. Hoffmann. 1991b. Prevention of experimentally induced whirling disease in rainbow trout *Oncorhynchus mykiss* by Fumagillin. Diseases of Aquatic Organisms 10:109–113.

El-Matbouli, M., T. S. McDowell, D. B. Antonio, K. B. Andree, and R. P. Hedrick. 1999. Effect of water temperature on the development, release and survival of the triactinomyxon stage of *Myxobolus cerebralis* in its oligochaete host. International Journal for Parasitology 29:627–641.

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.

Hedrick, R. P., M. El-Matbouli, M. A. Adkison, and E. MacConnell. 1998. Whirling disease: re-emergence among wild trout. Immunological Reviews 166:365–376.

Hedrick, R. P., T. S. McDowell, K. Mukkatira, M. P. Georgiadis, and E. MacConnell. 1999. Susceptibility of selected inland salmonids to experimentally induced infections with *Myxobolus cerebralis*, the causative agent of whirling disease. Journal of Aquatic Animal Health 11:330–339.

Hedrick, R. P., and seven coauthors. 2000. Ultraviolet irradiation inactivates the waterborne infective stages of *Myxobolus cerebralis*: a treatment for hatchery water supplies. Diseases of Aquatic Organisms 42:53–59.

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. 1975. Whirling disease (*Myxosoma cerebralis*): control with ultraviolet irradiation and effect on fish. Journal of Wildlife Diseases 11:505–507.

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, Indiana. Journal of Parasitology 47 (4, section II):29.

Hoffman, G. L., C. E. Dunbar, and A. Bradford. 1962. Whirling disease of trout caused by *Myxosoma cerebralis* in the United States. Special Scientific Report No. 427. U.S. Fish and Wildlife Service, Washington, D.C.

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.

Ivanov, A. P., R. Y. Kosyreva, and N. L. Nechaeva. 1968. Implementation for health of salmonids in fish culture in southern U.S.S.R. (in Russian). 1972. Food and Agriculture Organization of the United Nations, Symposium on the major communicable fish diseases in Europe and their control. FI:EIFAC 72/SC II-Symp 18.

Janiszewska, J. 1955. Actinomyxidia: morphology, ecology, history of investigations, systematics, development. Acta Parasitologica Polonica 2:405–443.

Kaster, J. L., and J. H. Bushnell. 1981. Cyst formation by *Tubifex tubifex* (Tubificidae). Transactions of the American Microscopy Society 100:34–41.

Kent, M. L., L. Margolis, and J. O. Corliss. 1994. The demise of a class of protists: taxonomic and nomenclatural revisions proposed for the protist phylum Myxozoa Grassé, 1870. Canadian Journal of Zoology 72:932–937.

Lake, R. L., and S. G. Hinch. 1999. Acute effects of suspended sediment angularity on juvenile coho salmon (*Oncorhynchus kisutch*). Canadian Journal of Fisheries and Aquatic Sciences 56:862–867.

Lieffers, H. J. 1990. Effects of the lampricide 3-trifluoromethyl-4-nitrophenol on macroinvertebrate populations in a small stream. Great Lakes Fisheries Commission Technical Report 55, Ann Arbor, Michigan.

Markiw, M. E. 1992a. Salmonid whirling disease. Fish and Wildlife Service Leaflet 17. Washington, D.C.

Markiw, M. E. 1992b. Experimentally induced whirling disease II. Determination of longevity of the infective triactinomyxon stage of *Myxobolus cerebralis* by vital staining. Journal of Aquatic Animal Health 4:44–47.

Markiw, M. E. 1992.c. 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.

Markiw, M. E., and K. Wolf. 1983. *Myxosoma cerebralis* (Myxozoa: Myxosporea) etiological agent of salmonid whirling disease requires tubificid worm (Annelida: Oligocheata) in its life cycle. Journal of Protozoology 30:561–564.

Molnar, K., F. Baska, and C. Székely. 1987. Fumagillin, an efficacious drug against renal sphaerosporosis of the common carp *Cyprinus carpio*. Diseases of Aquatic Organisms 2:187–190.

Nehring, R. B., and K. G. Thompson. 2001. Impact assessment of some physical, and biological factors in the whirling disease epizootic among wild trout in Colorado. Colorado Division of Wildlife, Special Report No. 76, Fort Collins, Colorado.

Nehring, R. B., and P. G. Walker. 1996. Whirling disease in the wild: the new reality in the Intermountain West. Fisheries 21(6):28–30.

Newcombe, C. P., and D. D. MacDonald. 1991. Effects of suspended sediments on aquatic ecosystems. North American Journal of Fisheries Management 11:72–82.

Nickum, M. J. 2000. Whirling disease forces hatchery modernization in Colorado. Hatchery Magazine 1(2):10–12,25.

O'Grodnick, J. J. 1975. Egg transmission of whirling disease. Progressive Fish-Culturist 37:153–154.

O'Grodnick, J. J. 1979. Susceptibility of various salmonids to whirling disease (*Myxosoma cerebralis*). Transactions of the American Fisheries Society 108:187–190.

O'Grodnick, J. J., and C. C. Gustafson. 1974. A study of the transmission, life history, and control of whirling disease of trout. Federal Aid to Fish Restoration Progress Report F-35-R-6. U.S. Fish and Wildlife Service, Washington, D.C.

Rasmussen, C. J. 1965. Control of whirling disease in Danish trout farms. European Inland Fisheries Advisory Commission Technical Paper No. 2:14–15.

Ryce, E. K. N., A. V. Zale, and R. B. Nehring. 2001. Lack of selection for resistance to whirling disease among progeny of Colorado River rainbow trout. Journal of Aquatic Animal Health 12:63–68.

Scolari, C. 1954. Sull'impiego dello Stovarsolo nella profilassi del "capostorno" O "lentosporiasi" delle trote d'allevamento. Clinica Veterinaria 77:50–53.

Strange, R. J., C. B. Schreck, and R. D. Ewing. 1978. Cortisol concentrations in confined juvenile chinook salmon (*Oncorhynchus tshawytscha*). Transactions of the American Fisheries Society 107:812–819.

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.

Thompson, K. G., R. B. Nehring, D. C. Bowden, and T. Wygant. 1999. Field studies of seven species or subspecies of salmonids to *Myxobolus cerebralis* in the Colorado River, Middle Park, Colorado. Journal of Aquatic Animal Health 11:312–329.

Thompson, K. G., and R. B. Nehring. 2000. A simple technique used to filter and quantify the actinospore of *Myxobolus cerebralis* and determine its seasonal abundance in the Colorado River. Journal of Aquatic Animal Health 12:316–323.

Vincent, E. R. 1996. Whirling disease and wild trout: the Montana experience. Fisheries 21(6):32–33.

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 Report, Denver, Colorado.

Whitley, L. S. 1968. The resistance of tubificid worms to three common pollutants. Hydrobiologia 32:193–205.

Whitten, B. K., and C. J. Goodnight. 1966. Toxicity of some common insecticides to tubificids. Journal of the Water Pollution Control Federation 38:227–235.

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.

Wolf, K., and M. E. Markiw. 1984. Biology contravenes taxonomy in the Myxozoa: new discoveries show alternation of invertebrate and vertebrate hosts. Science 225:1449–1452.

Yokoyama, H., Y. S. Liyanage, A. Sugai, and H. Wakabayashi. 1999. Efficacy of fumagillin against haemorrhagic thelohanellosis caused by *Thelohanellus horvorkai* (Myxosporea:Myxozoa) in colored carp, *Cyprinus carpio* L. Journal of Fish Diseases 22:243–245.