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Memorandum

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From: Scott VanderKooi, Grand Canyon Monitoring and Research Center, Acting Deputy Chief

CC: Shane Capron, GCDAMP Technical Work Group, Chair

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Subject: Whirling disease in Glen Canyon, Arizona and implications for resource management in the Colorado River

The confirmation of the presence of whirling disease in Glen Canyon has repercussions for the management of fisheries and other resources in the Colorado River below Glen Canyon Dam. The risk of spreading the disease must be taken into consideration as the recently completed Environmental Assessments (EA) for Nonnative Fish Control and High-Flow Experiments are implemented. Of the actions proposed, live removal and relocation of rainbow trout (*Oncorhynchus mykiss)* represents the greatest risk for spreading whirling disease, as these fish are highly susceptible to infection and can carry large numbers of the parasite. Risks associated with experimental floods further spreading the disease are low as the downstream movement of infected fish is already occurring. Higher flows may actually decrease the prevalence of whirling disease through disruption of the parasite's life cycle by displacing its alternate host and reducing its preferred habitat.

Whirling disease was initially detected in Glen Canyon in 2007 (Makinster and others, 2008) and re-detected in 2011 (B. Stewart, AZGFD, pers. comm.). The 2011 results showed 22 percent of samples (90 fish pooled into batches of five fish each) tested positive for the disease with positive groups collected from both upstream and downstream reaches. The presence of whirling disease has implications for a number of proposed management actions related to Glen Canyon Dam and the Colorado River in Glen, Marble, and Grand Canyons. Two potential actions that have raised concerns due to their perceived potential to spread whirling disease are: 1) live removal and relocation of rainbow trout associated with the Nonnative Fish Control EA (Bureau of Reclamation, 2011a); and 2) experimental floods conducted as part of the High-Flow Experiment EA (Bureau of Reclamation, 2011b). Below, we summarize available literature concerning the risk of spreading whirling disease through these potential management actions.

Background: Whirling disease biology and life cycle

Whirling disease only infects salmon and trout species, and is caused by *Myxobolus cerebralis*, a myxozoan parasite introduced to North America from Europe in the 1950s (Bartholomew and Reno. 2002). Elwell and others (2009) provide a thorough description of the parasite and summarize the disease and its effects on fish in the United States in a white paper prepared as part of the Whirling Disease Initiative. Myxozoan parasites exhibit complex life histories requiring both an invertebrate and vertebrate host to complete their life cycle (Figure 1). In the case of *M. cerebralis*, the invertebrate host is the oligochaete worm *Tubifex tubifex* and the vertebrate host is a salmonid fish (e.g., salmon, trout and whitefish).

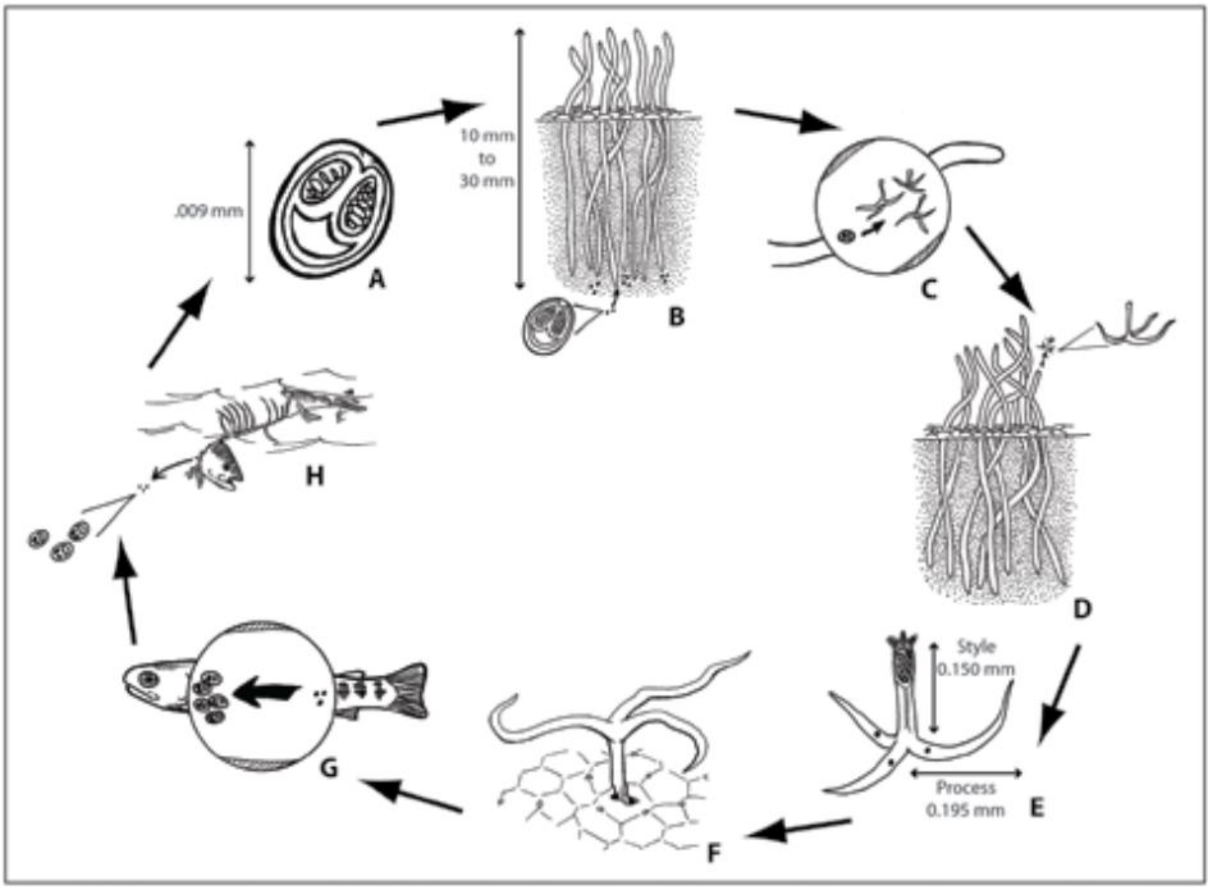

Figure 1. The life cycle of *Myxobolus cerebralis*, the parasite that causes whirling disease. Myxospores (A) are ingested by the oligochaete worm *Tubifex tubfex* (B) which are infected and subsequently produce (C) and release (D) triactinomyxons (E). Triactinomyxons infect a salmonid fish through their skin (F) which then produce myxospores (G) that are released following the death of the fish (H) completing the life cycle (Figure from Elwelland others,. 2009).

No other worm or fish species can be infected, but susceptibility to whirling disease does vary by strain of *T. tubifex* as well as species and stock of salmonid. For example, rainbow trout are quite susceptible to the disease although some stocks are resistant, while brown trout (*Salmo trutta)* can carry the parasite and show few clinical signs of infection. Susceptibility in fish also varies by life stage and size with young and small fish most vulnerable, because the parasite targets cartilage as the infection develops. Resistance to whirling disease increases in developing fish as cartilage is replaced with bone. Infection is also influenced directly and indirectly by a number of environmental factors including water temperature, substrate, and flow. Water temperatures between 10 and 15 °C are most suitable for triactinomyxon (TAM) production (the life-stage of whirling disease produced in *T. tubifex* that subsequently infects fish) and are associated with the highest levels of infection and disease severity. Substrates composed of finer materials like silt and clay are more favorable for *T. tubifex* reproduction and TAM production in those worms. Higher levels of organic material in streams lead to higher worm abundance which, in turn, may be related to an elevated risk of infection in fish. In the short term, flows high enough to scour substrates can disadvantage *T. tubifex* by displacing fine sediment and the worms residing in it. Scouring high flows can also disadvantage *T. tubifex* in the long term by limiting amounts of suitable habitat. Anthropogenic changes in watersheds can also affect whirling disease prevalence. The tailwaters downstream from dams often have conditions favorable to *T. tubifex*, which could increase *M. cerebralis* infection risk in fish.

Risk of spread through relocation of trout

Live removal and relocation of rainbow trout or brown trout from the Colorado River to other waters poses a substantial risk of unintentionally spreading whirling disease as both species can carry the parasite. An infected fish can release millions of myxospores (Hallet and Bartholomew, 2008), which can then infect *T. tubifex*, thus completing the life cycle of the parasite. There is no effective treatment of whirling disease in fish once a population has become infected (Gilbert and Granath, 2003). Because of this, possessing or transporting fish from whirling disease infected waters is explicitly forbidden by state law (see Arizona's Aquatic Invasive Species Interdiction Act; A.R.S. 17-255.02). Absent human intervention, the natural movement of infected fish appears to be a likely mechanism by which *M. cerebralis* is spread within rivers and watersheds (Zielinkski, 2008).

Risk of whirling disease spread through High-Flow Experiments

Controlled floods, administratively called High-Flow Experiments (HFEs), appear to pose a minor risk of spreading whirling disease, particularly since there are several mechanisms that allow the disease to spread to downstream reaches in the absence of controlled floods. Triactinomyxons are neutrally buoyant (Gilbert and Granath, 2003) and are easily dispersed downstream in rivers and streams. In addition, TAMs have been shown to be viable for as long as 15 days at temperatures ranging from 7 to 15 °C (El-Matbouli and others, 1999). These findings are particularly relevant to the current situation in the Colorado River downstream from Glen Canyon Dam. Triactinomyxons released from infected worms in Glen Canyon clearly have the potential to infect fish throughout the length of Marble and Grand Canyons during normal dam operations given that water transport times are short (e.g., 4.5 d to travel 235 miles at 15,000 ft^3/s; Graf, 1997) and water temperatures throughout Grand Canyon are usually between 7 and 15 °C (Voichick and Wright, 2007). This is why many agencies consider the length of the Colorado River between Glen Canyon Dam and Lake Mead to be infected by whirling disease.

Higher flows are thought to limit whirling disease (Hallet and Bartholomew 2008). In fact, flushing flows have been identified as a potential management tool for decreasing whirling disease infections (Elwell and others, 2009). Higher flows scour fine sediments and associated organic material from larger substrates and displace *T. tubifex* living there, thus reducing sources of TAMs that can infect fish, as well as limit suitable habitat for worm recolonization. A flushing flow with a peak approximately 6 times higher than base flows on the San Juan River in New Mexico temporarily reduced organic matter and *T. tubifex* densities in downstream deep water habitats, which may have decreased the prevalence and severity of whirling disease there (DuBey and Caldwell, 2004). The last controlled flood on the Colorado River in March2008 resulted in a temporary decrease in the density of tubificid worms in Glen Canyon (Cross and others, 2011). Thus, future HFEs are likely to result in a decrease of whirling disease prevalence and severity in Glen Canyon.

Controlled floods on the Colorado River could facilitate the spread of whirling disease to downstream reaches by temporarily increasing *M. cerebralis* myxospore and TAM abundance in the water column, but an increase in infection risk due to these floods seems unlikely. Floods of the magnitude proposed result in short travel times through Grand Canyon (e.g., 2.4 d to travel 235 miles at 45,000 ft^3/s; Graf, 1997) and Hallett and Bartholomew (2008) found higher flow rates resulted in lower *M. cerebralis* infection prevalence in both *T. tubifex* and rainbow trout, and reduced disease severity in rainbow trout. Another factor that makes it unlikely that future HFEs will facilitate the downstream spread of whirling disease, is that most rainbow trout in downstream reaches are relatively large (Yard and others, 2011). Fish are most susceptible to infection when they are young and small, thus the large rainbow trout that are common in Grand Canyon naturally have low susceptibility to whirling disease.

Conclusions

The implications of the presence of whirling disease in Glen Canyon for proposed management actions in the Colorado River vary by action. Live removal and relocation of trout from the Colorado River represents, by far, the greatest risk of spreading the disease. Relocation of trout from an infected population without any risk of fish escapement or spread of myxospores is virtually impossible. There is a low risk of spreading whirling disease as a consequence of conducting experimental floods. The disease is already present downstream from Glen Canyon Dam, and infected fish are already moving into Marble and Grand Canyons. It is likely that HFEs will result in a decrease in the prevalence and severity of the disease through reductions in the abundance of the intermediate host *T. tubifex* and its preferred habitat of fine sediment and organic matter.

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