The History and Dissemination of Whirling Disease

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ABSTRACT. The explosion of information on the distribution and impacts of whirling disease in the United States during the last decade has changed the way in which we view *Myxobolus cerebralis*. However, even a cursory review of whirling disease literature reveals that many of our concerns today have been expressed at some previous time in the history of our experience with this parasite. From the first description of *M. cerebralis* in Germany in 1893, it was recognized that whirling disease could severely affect the growing trout farming industry. During the first half of this century *M. cerebralis* was disseminated throughout Europe, especially following WWII when live rainbow trout *Oncorhynchus mykiss* were transferred freely. Between 1950 and 1970, the parasite began to appear at trout farms on other continents, and it was in the late 1950s that whirling disease first emerged in the United States. Nearly all reports of detection, both here and in Europe, were associated with artificial rearing facilities. Until the 1980s, the only references reporting infections in natural populations of salmonids are from Finland, Russia, and Michigan, and the reported infections were usually light. Clinical whirling disease was largely associated with culture of trout in earthen ponds, where the infective agent concentrated. In the period between 1970 and 1990, there were increasing reports of the parasite in hatcheries throughout Europe and the United States. In Europe, the perspective after many years of living with whirling disease was that eradication was not possible in most cases, but that we knew enough to reduce infection levels below the point where clinical disease occurs. In the United States, reports of whirling disease in hatcheries were often followed by destruction of any fish on the facility, but as it became apparent that proper management could reduce infection levels, and as there appeared to be no effects outside the bounds of the hatchery, these standards were relaxed. However, in the 1990s, clinical whirling disease was reported in free-ranging trout populations in Colorado and Montana, causing us, once again, to rethink how this disease can be controlled and managed.

When *Myxobolus cerebralis* was first detected in 1893 (Höfer 1903), the myxosporea were already well known fish pathogens. The first members of this taxon were described as early as 1838, from the retina of a carp (Müller 194, in Shul'man 1966). During the past 150 years, there has been considerable scientific interest in these parasites; however, because of its importance to the trout farming industry, *M. cerebralis* became a focal point upon which much of our knowledge of this group has evolved.

This review traces the history and distribution of whirling disease and will discuss prevailing perspectives on the disease, its effects, and attempts at control during different periods. Much of the information gathered here was gleaned from primary literature, previous reviews and reports, both formal and informal. In addition, personal correspondence between the authors and other scientists helped confirm previous information and update current distribution and effects. A caveat is warranted, however: while the collected data were approached conservatively, some inaccuracies with the historic data are inevitable, given the disparate nature of the sampling, monitoring, and identification of this pathogen. Thus, we have attempted to remain strictly prudent in our presentation. The information is presented chronologically, and within each timeframe, by distribution, epizootiology, and a general perspective on the disease. The information outlined below is depicted in Figure 1, for world data. Figures 2 and 3 depict parasite distribution in the United States, with Figure 2 characterizing the data by decade and Figure 3 by location and whether infected fish were cultured or wild/feral. Table 1 contains details of information on individual states.

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1890–1910

Distribution and Epizootiology

Germany—Höfer 1903

The first report of whirling disease, (Drehkrankheit) occurred in Germany. The infection was detected in rainbow trout Oncorhynchus mykiss and brook trout Salvelinus fontinalis. These species are not indigenous to Europe and eggs had been imported from the United States into Germany beginning in the late 1870s (Höfer 1903) to supplement the culture of the native brown trout Salmo trutta. The disease had a devastating effect on cultured rainbow and brook trout in the fish farms in which it was found.

Perspectives on Whirling Disease

The original report by Bruno Höfer (1903) described a myxosporidian of trout that infected the brain—hence the original designation Myxobolus "cerebralis". With the recognition that this was instead a parasite of skeletal tissue, a proposal was made by Marianne Plehn to change the name to Myxobolus chondrophagus (Höfer 1904). This revision was not accepted due to zoological nomenclatural rules and the name "Lentospora cerebralis" was introduced (Höfer 1904), with the new genus name describing a species similar to Myxobolus, but not possessing an iodinophilic vacuole within the spore.

The first detailed descriptions of whirling disease and Lentospora cerebralis were by Plehn (1905). Stages of infection including initial invasion and erosion of the cartilage by the trophozoites and the subsequent formation of large lesions containing parasites and debris from cartilage degradation were described. She also speculated that because the cartilaginous region around the auditory capsule was usually invaded, either toxins released by the parasite, or simply the weakening of the capsule itself, destroyed the equilibrium of the fish and precipitated the tail-chasing behavior which is observed when fish are disturbed. Weakening of the vertebral column posterior to the 26th vertebra was proposed to exert pressure on the caudal nerves and result in loss of melanocyte control, causing black tail. The most commonly observed cranial deformities were sunken areas behind the eyes and the permanently open or twisted lower jaw, both caused by loss of cartilage during bone formation. In fish that survived to the second year, she described the formation of epitheloid granulomas, which may result in secondary damage. It was suggested that when the cartilage ossifies, the spores become encapsulated and new infections could not occur.

Infection of fish was presumed to occur by ingestion of spores, as in other myxosporidian diseases known at that time. The resilience of the spores to the effects of drying and freezing, and their ability to survive for long periods were noted (Plehn 1905), and the implications of spore stability for the eradication of the parasite were recognized.

1911–1930

Distribution and Epizootiology

Denmark—Bruhl 1926

Perspectives on Whirling Disease

The continuing spread of whirling disease throughout trout-rearing facilities in Germany caused concern and speculation on how introduction had occurred. Several reports suggested that whirling disease originated from feeding rainbow trout with raw marine fish (cod) infected with the parasite (Bergman 1922; Plehn 1924), although later investigators pointed out that introduction by this route was unlikely (Schäperclaus 1931).

Since it was recognized that the clinical signs exhibited in whirling disease were common to other fish diseases, accurate identification of the spores was necessary for confirmation. It was also recognized that because spore formation required several months to occur, diagnosis of early stages of infection was difficult. Therefore, selection of diagnostic methods depended on the age or life stage of the fish. Infection in age 0+ trout was best detected by observing external signs. In fish larger than 10 cm and beyond their first winter, spores could be detected in histological preparations or from dissection of the vestibular organ. (Plehn 1924; Schäperclaus 1931).

1931–1950

Distribution and Epizootiology

Finland (southern)—Dogel (1932, in Uspenskaya 1957)
France—Vanco (1952, in Hoffman 1970)
Italy—1950 (Scolari 1954)
Figure 1. Geographic distribution and dispersal of *Myxobolus cerebralis* infections from its initial detection until present. Figures 1A–E represent accumulative detection for individual countries in 20-year increments. Countries in which *M. cerebralis* was detected during the period are shaded in dark grey; those in which the pathogen had been detected prior to the period are shaded in light grey. Figures 1A: 1890–1910, 1B: 1911–1930, 1C: 1931–1950, 1D: 1951–1970, 1E: 1971–2000.
In Germany, transfer of infected fish among breeding installations had led to the occurrence of whirling disease in most facilities rearing rainbow trout. The extent of the problem was evident from descriptions by Schäperclaus (1931) who indicated that trout breeders were less than forthcoming about the occurrence of whirling disease, often hiding affected fish from visitors. In areas where the parasite was established, whirling disease caused severe losses in trout pond farms and severely limited trout production.

Perspectives on Whirling Disease

Schäperclaus (1931) expanded his earlier descriptions of whirling disease. He recognized four classes of disease signs: 1) whirling movements caused by impairment of the vestibular apparatus; 2) premature death without significant signs other than loss of weight; 3) chronic disease with darkening of the tail, purportedly as a result of damage to the sympathetic nerves and; 4) chronic infection without external signs. This latter condition was particularly significant as it was recognized that these fish were likely the primary source of dissemination of the pathogen.

Other explanations for routes of parasite dissemination were proposed. Refuting earlier conclusions that raw marine fish were the primary source of infection (Plehn 1924), Schäperclaus (1931) suggested that this was unlikely because fish in hatcheries where saltwater fish were not fed also developed whirling disease. He believed that dissemination was largely a result of transfer of infected fish. The brown trout was particularly suspected as the primary source, because this species often became infected but did not show significant evidence of whirling disease. Reports of transfer of infection via shipments of eggs were noted, and were explained by contamination of the eggs with the pathogen adhered to the egg surface or on fomites such as shipping material and equipment. These observations resulted in guidelines for insuring that eggs were incubated on well water, and for disinfecting equipment. The nature of transfer in some cases suggested that eyed eggs could become infected but this was not demonstrated. Schäperclaus also investigated invertebrates as potential carriers and was especially interested in snails because of their presence in large numbers in many affected ponds. However, examination of these animals failed to support this hypothesis; other species of invertebrates were not examined.

Schäperclaus (1931) suggested guidelines for control of the disease which included setting low flow rates to avoid stirring up bottom sediment thereby making the spores available for ingestion, and keeping breeding and rearing ponds separated. After incorporating these precautions, it was recommended to disinfect unused ponds with caustic lime and calcium cyanimide, remove mud from the pond bottom and to discard the sickest fish. However, trout farmers were cautioned against seeking a panacea against whirling disease.

Continued revision of the taxonomy of the agent established the genus Lentospora (Plehn 1905) as synonymous with Myxosoma (Kudo 1933), hence the change to “Myxosoma cerebralis”. The spores of the Myxosoma were differentiated from those of Myxobolus by their lack of an iodophorous vacuole.

1951–1970

Distribution and Epizootiology

- USSR
  - Leningrad region—1952 (Uspenskaya 1955)
  - Abkhazian district—1954 (Uspenskaya 1957)
  - Black River—Uspenskaya (1957)
  - Sakhalin Island—1959 (Bogdanova 1960)
  - Luvenga River, White Sea tributary—1962 (Bogdanova 1964)
  - Lake Issyk-Kul, South-Central Russia—1964 (Bogdanova 1968)
  - Black Sea region—1964 (Bogdanova 1968)

- Europe
  - Czechoslovakia—1954 (Dyk 1954)
  - Poland—Kocylowski (1953)
  - Bulgaria—Margaritov (1960)
  - Yugoslavia—Tomasec (1960)
  - Sweden—Johansson (1966)
  - Scotland—Elson (1969); Roberts and Elson (1970)
  - Norway—1970 (Hastein 1971)
Figure 2. Geographic distribution and dispersal of *Myxobolus cerebralis* infections within the United States, from its initial detection in 1956 until present. Figures 2A–D represent accumulative detection for individual states in 10-year increments. States in which *M. cerebralis* was detected during the period are shaded in dark grey; those in which the pathogen had been detected prior to the period are shaded in light grey. The pathogen was not detected in any new states between 1971 and 1980. Figures 2A: 1951–1960, 2B: 1961–1970, 2C: 1981–1990, 2D: 1991–2000.
In his thorough examination of dissemination and transfaunation of *M. cerebralis*, Hoffman (1970) suggests that the original range of *M. cerebralis* covered an area from Central Europe to Northeast Asia. This conclusion was based on the resistance of the native brown trout to the disease and the hypothesis that this resistance is specific and acquired. From its first recognition in 1903 until the mid-1930s, the disease had been reported only from Germany, Denmark (Bruhl 1926) and Finland (Dogel 1932, in Uspenskaya 1957). However, widespread detection occurred following WWII. Hoffman (1970) associated this apparent dissemination to unrestricted transfers of live rainbow trout throughout Europe. However, he also pointed out that some of the fish farms might have become infected with spores from wild native brown trout. If these subsequent reports are interpreted on this basis, then transmission was from natural sources within this region. This mechanism of dissemination would imply multiple initiation sites by "natural" routes, followed by efficient establishment in the majority of the fish farms in Europe where earthen ponds were the norm. This explanation may be viable for Europe where the parasite was enzootic in native salmonids, but would not explain the spread to regions outside Europe. In these instances, the dissemination was certain to have occurred by transport of live fish.

In Europe, the impact of the disease on wild trout populations appeared to be negligible (Christensen 1972). The hypothesis for this lack of effect is that resident species (primarily brown trout) may have acquired resistance to *M. cerebralis* and rainbow trout had not been stocked extensively. Reports of infections in natural populations of salmonids are primarily from southern Finland (Dogel 1932, in Uspenskaya 1957) and western Russia (Uspenskaya 1957). In the Sakhalin Islands, off the coast of eastern Russia, there appeared to be an enzootic focus of parasitism and the parasite apparently had a wide distribution by the late 1950s. In this region not only cultured trout, but also cultured and wild anadromous Pacific salmon were infected, enhancing the likelihood of disseminating spores. Bogdanova (1960, 1966, 1968) documented infections in salmonids from four rivers on Sakhalin Island. However, clinical disease signs were largely associated with rearing facilities using earthen ponds; clinical disease was unknown in natural populations where infections were usually light (Bogdanova 1970).

Although there are many confirmed reports documenting the worldwide dissemination of *M. cerebralis* during this time, reports from certain regions remain unconfirmed and are not generally accepted in the fish health community; for their historic value, they are mentioned below. These events are likely a result of misinterpretation because of diagnosis based on clinical signs alone or confusion of *M. cerebralis* spores with those of other myxobolid species. They may also reflect cases of parasite detection in fish that were imported for culture but where subsequent establishment of the parasite failed to occur. However, they are mentioned here as a reminder of the importance of confirming infections.

Detection of *M. cerebralis* in Korea (Tinkina 1962) and Japan (Halliday 1976) was not unexpected because of their geographic proximity to the focus...
Figure 3. Locations within the United States from which *M. cerebralis* has been isolated between 1956 and present. Data were accumulated from literature citations, unpublished historical data, and personal communications; as much as possible, confirmation was acquired from telephone or electronic means. The data are grouped by type of sample collected: ⚫ = wild or feral fish; ○ = fish in public culture/holding facilities; ● = fish in private culture/ holding facilities.
Table 1. Detection of *Myxobolus cerebralis* in hatcheries, rivers, and standing waters in states where the parasite has been detected.

<table>
<thead>
<tr>
<th>State</th>
<th><em>Myxobolus cerebralis</em> detected</th>
<th>Detection in culture facilities current and historical</th>
<th>Detection in rivers/standing waters</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arizona</td>
<td>2000 (P. Lopez, Arizona Game and Fish Department, personal communication)</td>
<td>None</td>
<td>Confirmed introduction in one private pond</td>
</tr>
<tr>
<td>California</td>
<td>1966 (Yasutake and Wolf 1970)</td>
<td>2 state hatcheries and 3 private facilities currently positive</td>
<td>South Fork American and Trinity Rivers; Wolf, Battle, Deer, Garrivanta, and Big Creeks; Carmel River; Aptos and LaHonda Creeks and multiple points in the Owens, Lahontan, Stanislaus, Calaveras, Mokelumne, Feather, and San Lorenzo River drainages</td>
</tr>
<tr>
<td>Colorado</td>
<td>1987 (Barney et al. 1988; Walker and Nehring 1995)</td>
<td>9 state hatcheries, 1 federal hatchery and numerous private facilities positive</td>
<td>Enzootic in all coldwater drainages except the Animas and North Republican Rivers; populations impacted in the following rivers: Cache la Poudre, Colorado, Dolores, Fryingpan, Gunnison, Middle Fork of South Platte, South Platte, Rio Grande, Roaring Fork Rivers, as well as some smaller streams</td>
</tr>
<tr>
<td>Connecticut</td>
<td>1961 (Hoffman 1962; Hnath 1970)</td>
<td>No recent data; 2 state hatcheries historically positive</td>
<td>None reported</td>
</tr>
<tr>
<td>Idaho</td>
<td>1987 (Hauck et al. 1988)</td>
<td>2 of 22 state hatcheries currently positive</td>
<td>Coeur D’ Alene, St. Joe, American, Middle Fork Salmon, Yankee Fork, East Fork, S. Fork Boise, Lemhi, Pahsimeroi, Big Lost, Big Wood, Little Lost, Henry’s Fork, Teton, and Bruneau Rivers; Loving, Salmon Falls, and Jackknife Creeks and Henry’s Lake</td>
</tr>
<tr>
<td>Maryland</td>
<td>1995 (C. Geseker, Department of Natural Resources, personal communication)</td>
<td>2 of 6 hatcheries positive in 1995</td>
<td>Jennings Reservoir on north branch of the Potomac River</td>
</tr>
<tr>
<td>Massachusetts</td>
<td>1966 (Hoffman 1990)</td>
<td>No state hatcheries currently positive; 1 commercial hatchery historically positive</td>
<td>Not currently detected</td>
</tr>
<tr>
<td>Michigan</td>
<td>1968 (Yoder 1972)</td>
<td>No public hatcheries currently positive, several private hatcheries positive; one state hatchery historically positive</td>
<td>Sturgeon, Big Manistee, Little Manistee, AuSable, Jordan, Black, and Pigeon Rivers</td>
</tr>
<tr>
<td>State</td>
<td>Myxobolus cerebralis detected</td>
<td>Detection in culture facilities current and historical</td>
<td>Detection in rivers/standing waters</td>
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<tr>
<td>Montana</td>
<td>1994 (Vincent 1996)</td>
<td>None</td>
<td>87 waters in the following River drainages: Beaverhead, Gallatin, Madison, Bighole, Bitterroot, Blackfoot, Jefferson, Swan, Clark Fork, Missouri, Sun, Flathead, and Yellowstone</td>
</tr>
<tr>
<td>Nevada</td>
<td>1966 (Yasutake and Wolf 1970); 1957 (Taylor et al. 1973)</td>
<td>1 of 4 state facilities currently positive; 4 public hatcheries historically positive (2 now closed)</td>
<td>Truckee, Carson, Owyhee, East Walker, and Little Humboldt Rivers; Dorsey Creek, Dry Creek, Lahontan, Wildhorse, and Wilson Sink Reservoirs; Topaz, Pyramid, and Walker Lakes, Lake Tahoe; Bilk, Clear, and Duck Creeks</td>
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<tr>
<td>New Hampshire</td>
<td>1981 (Jay Hendee, New Hampshire Fish and Game Department, personal communication)</td>
<td>No recent data; 1 private hatchery historically positive</td>
<td>South Merrimack River basin</td>
</tr>
<tr>
<td>New Jersey</td>
<td>1968 (Halliday 1976)</td>
<td>2 state hatcheries historically positive, no longer in operation</td>
<td>Not currently detected, previously stocked rivers surveyed negative</td>
</tr>
<tr>
<td>New Mexico</td>
<td>1987 (R. Goede, Utah Division of Wildlife Resources, personal communication)</td>
<td>3 of 7 state hatcheries positive</td>
<td>Pecos, Cebolla, San Juan, Cimarron, Red, and Canones Rivers</td>
</tr>
<tr>
<td>New York</td>
<td>1984 (Hoffman 1990)</td>
<td>1 state hatchery and 1 private facility currently positive; 6 public facilities historically positive</td>
<td>At least one positive population or facility in all watersheds except on Long Island</td>
</tr>
<tr>
<td>Ohio</td>
<td>Late 1960s (Tidd and Tubb 1970)</td>
<td>No recent data; 1 private hatchery in 1960s</td>
<td>Not currently detected</td>
</tr>
<tr>
<td>Oregon</td>
<td>1986 (Holt et al. 1987)</td>
<td>1 private facility currently positive; exposure at 2 public adult facilities from infected adult salmon; 1 state facility historically positive</td>
<td>Grande Ronde and Imnaha River basins</td>
</tr>
<tr>
<td>Pennsylvania</td>
<td>1956 (Hoffman 1962)</td>
<td>3 of 13 state facilities currently positive; 7 state hatcheries historically positive; no information on private hatcheries</td>
<td>Consider all watersheds to potentially have infected fish; infected fish detected in Lake Erie and its tributaries</td>
</tr>
<tr>
<td>State</td>
<td>Myxobolus cerebralis detected(^2)</td>
<td>Detection in culture facilities current and historical(^3)</td>
<td>Detection in rivers/standing waters(^4)</td>
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<tr>
<td>Utah</td>
<td>1991 (Heckman 1992)</td>
<td>1 state hatchery previously positive, 10–12 private facilities positive</td>
<td>Logan, Little Bear, South Fork Ogden, Weber, Provo, Beaver, Otter, Sevier, and Fremont drainage and in some isolated ponds</td>
</tr>
<tr>
<td>Virginia</td>
<td>1965 (Hoffman 1970)</td>
<td>2 state hatcheries positive and 4 private hatcheries historically positive</td>
<td>Tye River, Staley Creek, Whitetop fork of Laurel River</td>
</tr>
<tr>
<td>Washington</td>
<td>1996 (K. Amos, Washington Department of Fish and Wildlife, personal communication)</td>
<td>None</td>
<td>Cottonwood Creek and Grande Ronde River</td>
</tr>
<tr>
<td>West Virginia</td>
<td>1969 (Meyers 1969)</td>
<td>No recent data; 1 state hatchery, 1 federal hatchery and several private facilities historically positive</td>
<td>Not currently detected</td>
</tr>
<tr>
<td>Wyoming</td>
<td>1988 (D. Money, Wyoming Game and Fish Department, personal communication)</td>
<td>5 of 11 state facilities positive</td>
<td>North Platte, S. Fork Shoshone, Salt, Yellowstone, Fire Hole, Big and Little Laramie, N. Fork Popo Agie, Jakey’s Fork, E. Fork, New Fork, and Green Rivers; Fontenelle Reservoir; Yellowstone Lake</td>
</tr>
</tbody>
</table>

\(^1\)Only states where *M. cerebralis* has been confirmed are included. Information from several states included in this table report that the parasite has not been recently detected.

\(^2\)Dates are the first confirmed detection of the parasite in the state.

\(^3\)Data for detection in hatcheries was acquired through a survey conducted by the Whirling Disease Foundation, personal communications with state managers, and use of historical records. It must be acknowledged that monitoring varies among states and may not have been done for private facilities. Identification of facilities as historically positive indicates that either the facility has been disinfected and not subsequently detected positive or that it has been decommissioned.

\(^4\)Data for detection in rivers and standing waters was acquired similarly to that for hatchery data. The extent of, and methods for, monitoring of waters varies among states. The waters listed are those where *M. cerebralis* is known to have become established, although in most cases it was not possible to determine how widely or to what degree the parasite was established.

\(^5\)In Nevada, the parasite was first detected in 1966; however, examination of archived samples confirmed its earlier presence.
of infection in the Sakhalin Islands. However, the original report of *M. cerebralis* in Korea (Tinkina 1962) only states that the usual preventative measures are adopted against whirling disease, and not that the parasite was detected (Halliday 1976), and subsequent detection of *M. cerebralis* has not been confirmed in Japan (Egusa 1992). Similarly, reports of clinical whirling disease from several locations in South America were not confirmed. An account of whirling disease in Venezuela was based on a misinterpretation by Bogdanova (1968, 1970) of a report by Martinez (1966) in which lentosporidiasis (whirling disease) was described as a serious problem in trout culture. However, the report did not state that the disease occurred in Venezuela. Although succeeding reports continued to describe rainbow trout with signs of whirling disease (Espinoza and Diaz-Ungria 1970; Diaz-Ungria 1970), there was no laboratory confirmation of the infection. Reports of whirling disease also appeared from Columbia (Anonymous 1972), Ecuador (Meschkat 1971) and Chile (Anonymous 1974). Conroy (1975, 1981) and Margolis et al. (1996) attempted to sort through these conflicting and unconfirmed reports. Their conclusion, based on their own observations as well as on the failure of any of the authors who observed “whirling disease” to detect *M. cerebralis* spores from the affected fish, was that the myxosporian is absent in South America. Reports of *M. cerebralis* from Mexico (Halliday 1976) and Canada (Bogdanova 1969, in Halliday 1976) are also unconfirmed. Bogdanova (1969) reported spores in wild *Salvelinus malma* from British Columbia, Canada but a study by Margolis et al. (1981) did not support this finding and there has been no subsequent evidence that the parasite is present.

In the United States, whirling disease was first diagnosed in 1956 from brook trout at the Benner Springs Fish Research Station in Bellefonte, Pennsylvania (Hoffman 1962). Imported frozen European table trout were circumstantially implicated as the source of infection as samples of these food fish were found to contain spores. It was suspected that infected fish tissues were fed to the hatchery trout, or that infected viscera were discarded in the stream (G. L. Hoffman, U. S. Fish and Wildlife Service, retired, personal communication). Another possible source of infection was live brown trout imported from Europe in the 1950s by governmental, commercial and private hatcheries. As some outbreaks in commercial hatcheries were observed in the absence of direct transfer of fish from contaminated hatcheries, this raises the possibility that *M. cerebralis* may have been introduced through infected fish or material at more than one site (Hnath 1996). Infected fish from the affected hatcheries were stocked throughout Pennsylvania until the early 1970s, disseminating the parasite among many hatcheries and in natural waters in the state.

In Nevada, the first reported detection was in 1966 at the Verdi State Fish Hatchery (SFH) on the Truckee River by Yasutake and Wolf (1970), but examination of preserved fish showed the parasite had been present in the state by 1957 (Taylor et al. 1973). In 1968, *M. cerebralis* was detected in wild rainbow and brown trout from the Truckee River in the Lahontan basin. Because *M. cerebralis* had been present, but undetected, since at least 1957, many of the state’s waters had been stocked with infected fish. By 1969 infections had been confirmed in three state hatcheries and one federal hatchery in Nevada. Rivers that had been stocked with infected hatchery fish included the Truckee, Carson, East Fork Walker, Humboldt, and Owyhee.

In 1965 whirling disease was diagnosed in California from fish at a private hatchery on Garrapata Creek, in Monterey County (Yasutake and Wolf 1970). The hatchery had reported a history of receiving only trout eggs, and this was not believed to be the source of the infection. Imported frozen fish were again implicated (Hoffman 1990), as the affected fish had reportedly been fed frozen trout product from a Danish merchant vessel. However, because the life cycle of *M. cerebralis* was, as yet, unknown, other more likely sources of infection may not have been considered. The report of the outbreak was especially vivid. The behavior of the fish was described as “an extremely nervous twisting action as though the fish were hooked in the mouth and then got off the hook. They spiral around and around as though they were tied by the nose. This becomes so violent at times that some of them come out onto the bank.” (Yasutake and Wolf 1970).

Once established at these locations in the eastern and western United States, subsequent spread of the whirling disease parasite has been attributed to transfers of live fish (Hoffman 1970, 1990). In Connecticut, whirling disease was diagnosed in 1961 from rainbow trout fingerlings at the Kensington SFH and the parasite was detected in 1966 from fish at the Burlington SFH. It was suspected that the source of the first infection was fish transferred from
the infected Benner Springs facility in 1959 (Hoffman 1962). Between 1965 and 1968, infections were detected in fish from hatcheries in Virginia (Hoffman 1970), Massachusetts (Halliday 1976), West Virginia (Meyers 1969), New Jersey (Hoffman 1968: In Halliday 1976), Ohio (Tidd and Tubb 1970), and Michigan (Yoder 1972), and from additional sites in California, Pennsylvania and Nevada (Hoffman 1990). In West Virginia, introduction occurred at three separate locations in 1966, 1972, and 1984. Clinical whirling disease was diagnosed in Ohio at one location in 1967 and M. cerebralis-positive fish were reported from two additional sites (Tidd and Tubb 1970).

In Michigan, M. cerebralis was detected in 1968 at three facilities belonging to a commercial trout grower. Records showed that infection was likely a result of an importation of infected fish the previous year from a private facility in Ohio, and fish had been subsequently distributed to 159 smaller hatcheries, fee fishing ponds and private recreational ponds. The state quarantined these sites, established a permit system to prevent further spread and through voluntary agreement, treated all 159 sites to kill the fish. Subsequent monitoring at these secondary sites indicated that these actions were successful. However, action was delayed at the primary sources and the parasite became established in populations of wild brook and brown trout from the North Branch Tobacco River (Yoder 1972). The time between suspected introduction and detection in the wild populations in 1970 was 28 months. A second introduction occurred in the Sturgeon River, possibly by legal stocking of fish by a private club. The parasite became established and was eventually detected at the state hatchery on that river in 1975. All the fish at the hatchery were destroyed, and the facility was disinfected and decommissioned (J. G. Hnath, Michigan Department of Natural Resources, personal communication).

In the United States, although the pathogen was found in several regions, clinical whirling disease was only reported in hatcheries. However, there were few published studies during this time that examined infection in wild fish and low-level infections in wild fish would probably not have been evident. Exceptions to this were the survey data in Michigan demonstrating establishment of the parasite in native brook and brown trout populations below the hatchery where it had been introduced (Yoder 1972) and studies on the Truckee River and other sites in Nevada demonstrating establishment of the pathogen in wild fish (D. Junell, Nevada Department of Conservation, Division of Wildlife, personal communication).

**Perspectives on Whirling Disease**

During this period whirling disease was still considered a serious problem throughout trout farming areas in Europe. Methods for control included destruction of all fish from ponds containing infected fish. It was suggested that concrete and earthen ponds should be drained and treated with calcium cyanamide, quicklime or sodium hypochlorite, with several treatments required for earthen ponds. Only fish from a known uninfected source should be used, and during the period of maximum susceptibility (the first 8 months), the young fish should be reared in spring or well water (Schäperclaus 1954; Hoffman 1962). Denmark went a step further to insure that young fish were reared on clean water supplies (Rasmussen 1967). There, they established separate facilities to rear fry to the fingerling stage, at which point they were transferred to rearing farms. This method proved successful and was implemented in other countries (Ghittino 1970). With adoption of these methods, as well as the construction of new ponds, the shift toward raising fry in troughs of concrete or synthetic fiber, and assuring that young fish were not exposed to the pathogen until their skeletons had achieved some degree of ossification, the impact of infection was reduced. However, this did not necessarily lead to a reduction of the prevalence of the pathogen in cultured or wild populations in enzootic areas.

In the United States, the discovery of M. cerebralis was considered a resource disaster because of the susceptibility of rainbow trout that are indigenous to the western United States and were widely dispersed here in the late 19th century. Efforts to restrict establishment and movement of the parasite in Michigan and in other areas where the parasite was detected were extreme, requiring destruction of fish from contaminated facilities and no transfer or stocking of fish from any infected facility.

Concerns about the effects of introduced pathogens led to development of the first national fish disease law in the United States. This legislation, a 1968 amendment to Title 50 Wildlife and Fisheries Act, attempts to exclude certain fish diseases from importation to the United States. The regulations, which became effective 1 July, 1968 (Code of Federal Regulations, Title 50, Section
require that salmonids and salmonid eggs be certified free from the hemorrhagic septicemia virus (VHSV) and *M. cerebralis* before they may be imported into the United States. Suggested methods for detection of the parasite (Hoffman 1968) included 1) maceration of the head in a mortar and pestle, 2) splitting the head sagitally and scraping the cut surface with a scalpel, 3) homogenizing the head in a tissue grinder, and 4) cutting the head lengthwise and examining under a dissection microscope for granulomas or cysts, then squashing these cysts for examination. To be certified free of the pathogen, eggs and sperm were required to be from sources free of infection for at least one year before spawning, and incubation prior to shipment must have been in water from a source free of fish carrying the disease. Fish for importation were required to be from sources certified to be free of the disease during the lifetime of the fish.

Researchers continued to examine modes of transmission and the parasite’s life cycle. Transovarian transmission was not documented, but it was considered possible that viable spores may be transferred with eggs (Schäperclaus 1931), although by 1954, Schäperclaus indicated that this was an unlikely means of dissemination. Schäperclaus (1954), who detected spores in the posterior gut of kingfishers, proposed the potential for transmission by avian vectors. However, he emphasized that the most common route for transmission between hatcheries was the introduction of apparently healthy carriers. It was generally accepted that spores were released when fish die and decompose in ponds; however, Uspenskaya (1955, 1957) offered evidence for live fish releasing spores. Detecting parasite spores in other tissue and organs, she hypothesized that they had been released from the destroyed cartilage and made their way via the blood or lymphatic vessels to the liver. From the liver, spores could be passed through the bile ducts to the intestinal lumen and be expelled. Rydlo (1971) supported the observations of Uspenskaya, demonstrating the presence of spores in the intestinal tract and skin of infected rainbow trout by digesting these tissues separate from cartilage.

Although it was still generally accepted that fish become infected by ingesting *M. cerebralis* spores, only Uspenskaya (1982) describes establishing infection by feeding spores. Most evidence indicated it was likely that either an intermediate host and/or certain water conditions were necessary for infections to occur. The widely accepted hypothesis was that spores are infective after ageing in the presence of mud for 3–6 months (Hoffman and Putz 1969; Putz 1969).

1971–1990

**Distribution and Epizootiology**

- **Europe**
  - Austria—FAO 1972 (In Halliday 1976)
  - Belgium—FAO 1972 (In Halliday 1976)
  - Ireland—1971 (southern Ireland) (Halliday 1974)
  - Liechtenstein—1973 (Halliday 1976)
  - Netherlands—FAO 1972 (In Halliday 1976)
  - Spain—Cordero-del-Campillo et al. 1975
  - Lebanon—1973 (Halliday 1976)
  - New Zealand—1971 (Hewitt and Little 1972)

- **USA**
  - New Hampshire—1980 (Hoffman 1990)
  - Oregon—1986 (Holt et al. 1987; Lorz et al. 1989)
  - Idaho—1987 (Hauck et al. 1988)
  - Colorado—1987 (Barney et al. 1988; Walker and Nehring 1995)
  - Wyoming—1988, (D. Money, Wyoming Game and Fish Department, personal communication)
New Mexico—1987 (D. Eib, New Mexico Department of Game and Fish, personal communication).

In 1976, Halliday reviewed the status of whirling disease in Europe. He concluded that differences in monitoring and reporting and inconsistencies in the original range of *M. cerebralis* or how rapidly the parasite was disseminated. The presence of fish rearing facilities on the same rivers where the parasite was detected in natural populations made it difficult to ascertain if the infection was introduced or enzootic. By the early 1970s, *M. cerebralis* had been reported from most European countries, although it is certain that it existed much earlier in some areas.

In 1971, *M. cerebralis* was reported from New Zealand (Hewitt and Little 1972). The report noted that heavy mortality accompanied by violent whirling motion, a condition known locally as “whirly-gig” disease, had occurred periodically at the affected hatchery since 1955 and perhaps as early as 1952. No diagnostic tests had been performed during these outbreaks; however, when an episode occurred in 1971, spores with morphologic characteristics that corresponded with those of *M. cerebralis* were isolated from the head region of rainbow trout. Examination of preserved specimens demonstrated that the parasite had been present at least five years before it was identified (Boustead 1993). At the time of the detection in 1971, there was a complete ban on importation of salmonid fish in any form, unless heat-treated. However, if the earlier episodes were a result of *M. cerebralis* infection, then introduction may have occurred prior to the enactment of these measures in 1952. Other suspected routes of introduction were ova and the packing material that they are shipped in, and live dried food for tropical fish, which may have included infected tubificids. Import of the latter product was not prohibited until 1971.

In the northeast United States, *M. cerebralis* was detected in New Hampshire at a private hatchery in 1983 (J. Schachte, New York State Department of Environmental Conservation, personal communication). The outbreak occurred when the small rainbow trout were moved to a pond receiving effluent from an earthen pond where adult fish were reared, evidence that the parasite had become established some time earlier.

The immediacy and degree of active control measures taken varied among states. In Pennsylvania, where the parasite was first detected, infected fish were stocked until the early 1970s and the parasite became established in public and private hatcheries and in wild populations statewide. During the 1970s earthen ponds at many state facilities were replaced with concrete raceways, and this resulted in disappearance of clinical disease. Infected fish were also restricted from being stocked into waters of concern; however, monitoring of wild populations has been limited. Other states took more active measures and required quarantine of affected facilities, destruction of fish, and treatment of ponds and streams with chlorine and quicklime. In New Hampshire, West Virginia and Ohio, where introduction was limited to a few facilities, these measures were reported to have been effective and the parasite has not been detected/reported since the initial introductions.

In Nevada, the perennial presence of the pathogen, and the disease, in the two main facilities that were sources of fish for stocking in the state led to widespread contamination of stocked waters. Surveys undertaken statewide during the 1970s indicated a wide distribution of the pathogen, but no evidence of disease outside the hatcheries. During this time no extraordinary efforts were made to rid the facilities of the pathogen.

In California, between 1965 and 1985 five commercial growers, one state fish hatchery and one federal hatchery had fish diagnosed with clinical whirling disease. In each case prior to 1984, the infected stocks at these facilities were destroyed (Horsch 1987; Modin 1998). In response to the control efforts, intensive monitoring was conducted during the ensuing 12 years. Results of these surveys demonstrated that the parasite had been unknowingly dispersed, likely with transfers of subclinically infected fish or infected processed products. Infected fish were detected from the San Lorenzo River on the central California coast in 1973 and from the Licking Fork and Middle Fork of the Mokelumne River on the West slope of the Sierra Nevada in 1982 and 1983, respectively. In 1984, infected fish were detected from the Owens River in the Eastern Sierra, and in 1985 from trib-
utaries of the Sacramento River, in Northeastern California. In each of these locations, infections were detected in fish both from culture facilities and wild fish. Infections in culture facilities ranged from subclinical to epizootic, and in the wild from subclinical to observance of occasional minor skeletal anomalies (Modin 1998).

Similar surveys were conducted in Oregon, where *M. cerebralis* was detected in a private trout hatchery in 1986. Infected resident trout and anadromous salmonids were found in the Lostine River, in northeastern Oregon, the source of the facility's water supply (Lorz et al. 1989). Subsequent monitoring demonstrated that the parasite was established in naturally reproducing resident and anadromous salmonid populations within the Grande Ronde and Imnaha River basins. These rivers are tributaries of the Snake River, and are part of the Columbia River basin. Fish from this hatchery were distributed to more than 75 private ponds throughout the state, and fish infected with *M. cerebralis* were found in at least 35 of these ponds. However, subsequent surveys of wild fish from watersheds that had received infected fish failed to detect a spread of infection outside of the infected ponds (R. Holt, Oregon Department of Fish and Wildlife, personal communication). In Idaho, *M. cerebralis* was detected in 1987 from juvenile chinook salmon *Oncorhynchus tshawytscha* at two state hatcheries on the Salmon and Pahsimeri Rivers and from rainbow trout at a private hatchery on the Lost River (Hauck et al. 1988). *Mycobacterium cerebralis* was confirmed in rainbow trout from a state hatchery on a tributary in the Snake River system the following year. The subsequent discovery of infection in returning 3- and 4-year-old chinook salmon indicated that the parasite had likely been established in the Salmon and Pahsimeri Rivers for some time.

Elsewhere in the west, *M. cerebralis* was detected in Colorado, Wyoming, and New Mexico between 1987 and 1988. In Colorado, the parasite was first detected in 1987 at four facilities (one state and three private) and fish at all four locations displayed clinical signs of the disease (Barney et al. 1988). Review of shipment records indicated that the parasite had been disseminated within the state as a result of transfers and planting of infected fish from the affected state and private hatcheries. In Wyoming, *M. cerebralis* was first detected in 1988 from rainbow and brown trout collected from the North and South Platte River drainages; these fish had been stocked the preceding year by a commercial grower (D. Money, Wyoming Game and Fish Department, personal communication). Introduction into New Mexico probably occurred as a result of illegal importation of trout to a private facility. Since a single focus of infection was present, an extensive effort was made to eradicate the infected fish. Subsequent sampling of these waters revealed no signs of whirling disease and monitoring was discontinued. (D. Eib, New Mexico Department of Game and Fish, personal communication).

For many states, *M. cerebralis* was detected only after the parasite had been introduced and had become established in populations. In these cases, control measures came too late and the parasite had already become dispersed through transfers and stocking of subclinically infected fish.

**Perspectives on Whirling Disease**

Legislation to control *M. cerebralis* was adopted in Europe in 1970 when the International Office of Epizootics accepted a proposal to include whirling disease in the “International Zoo-Sanitary Code” along with four other diseases of fish (Ghittino 1970). However, the impetus for this legislation was the fear of U.S. legislation that could effectively close U.S. markets to any country that could not guarantee absence of the parasite in exported trout/eggs. By the 1970s, whirling disease appeared to be under control in Europe and attention was turned to other pathogens. However, it was recognized that since this parasite cannot be easily eradicated, relaxing the diligence of control standards could readily lead to epizootics (Schäperclaus 1986). “Accommodation,” an approach used to minimize mortality and clinical signs in facilities where the parasite is enzootic, is widely practiced in Europe where trout are used for food, and stocking of waters with rainbow trout for anglers is not a major endeavor. Although technology is available to decontaminate water supplies, the excessive cost/benefit ratio of the equipment and operation was considered counterproductive in most situations.

In the United States, methods for certification of fish for importation and transfer were updated to include the pepsin-trypsin-dextrose centrifugation method and a modified plankton centrifuge method (O’Grodnick 1975; Markiw and Wolf 1974, 1980). Although additional restrictions were placed on importation of eggs, this was because of concerns with importation of the VHS virus rather than *M. cerebralis*, as O’Grodnick (1975) had demonstrated...
that *M. cerebralis* was not transmitted vertically. However, changes in the federal regulations did result in requirements for holding sentinel fish with brood fish for detection of *M. cerebralis*.

During the 1970s and into the mid-1980s, *M. cerebralis* was not reported from any “new” states. Then, in the late 1980s, *M. cerebralis* was detected from the western states of Idaho, Oregon, Colorado, and Wyoming. Based on the experience of states that had lived with the parasite for two decades, managers looked for an approach that was less extreme, with emphasis on avoiding spread of the pathogen while allowing for management in enzootic areas. Consequently, in 1988, the Colorado River Wildlife Council, Fish Disease Subcommittee recommended that *M. cerebralis* be reclassified from prohibited to notifiable status. This reclassification continued to require inspection but did not demand depopulation and disinfection of facilities. It was felt that this gave states the flexibility to regulate the disease. Some states would remain closed; others would have more complex regulations, allowing stocking of infected fish where the parasite was already established, or where it was unlikely to become established.

Dramatic changes also occurred in our knowledge of the parasite itself. Lom and Noble (1984) synonymized *Myxosoma* with *Myxobolus* because the separating characteristic, the glycogen (iodinophilous) vacuole, is not a reliable taxonomic characteristic. Thus, after 80 years, the parasite was once again recognized as *Myxobolus cerebralis*. In the same year, Wolf and Markiw (1984) announced the discovery that a species of *Triactinomyxon* and *M. cerebralis* are alternating life stages of a single life cycle, one stage undergoing development in an aquatic worm and the other in the fish. The stunning pronouncement that the aquatic oligochaete *Tubifex tubifex* was required to complete the parasite life cycle (Markiw and Wolf 1983) lent a sense of clarity to the success of control measures like replacement of earthen bottom ponds with concrete raceways. However, at the same time, evidence of experimental infection using spores aged in mud continued to be reported (Uspenskaya 1982) and the findings of Wolf and Markiw were met with reservation. It was not until the end of the 1980s that transmission of *M. cerebralis* was independently confirmed (El-Matbouli and Hoffmann 1989). This confirmation insured that our concepts of the life cycle and taxonomy of the Myxosporea would undergo radical change. It also opened new avenues for research on detection techniques and control methods.

**1991–PRESENT**

**Distribution and Epizootiology**

- **USA**
  - Washington—1996 (K. Amos, Washington Dept. of Fish and Wildlife, personal communication)
  - Montana—1994 (Vincent 1996)
  - Maryland—1995 (C. Geseker, Department of Natural Resources, personal communication)
  - Arizona—2000 (Patricia Lopez, Arizona Game and Fish Department, personal communication)

In the eastern United States, *M. cerebralis* was detected for the first time in Maryland (1995) at a state facility that had received fish from several sources, and in a second facility that had been stocked with fish from the first facility. The parasite was detected again in New York at the Rome SFH (1994), and subsequently at two other hatcheries. It was determined that the 1994 infection originated at the Caledonia SFH, from which it was transferred to the other facilities. Collections of wild fish from natural waters demonstrated infected fish were present, although not common, in most watersheds in the state except for Long Island. Voluntary inspections of private hatcheries demonstrated that *M. cerebralis* was present at a number of facilities (P. Hulbert, New York State Department of Environmental Conservation, personal communication). *Myxobolus cerebralis*-infected fish were removed from the four public hatcheries that were positive in 1994 and these hatcheries remain negative with the exception of the Chateaugay SFH, which tested positive in 2000 after receiving a group of infected fish from the previously uninfected Randolph SFH (J. H. Schachte, personal communication).

Re-emergence of *M. cerebralis* was also reported in Connecticut from an earthen pond at the Kensington fish hatchery, which had been contaminated in 1959. The pond had been free of fish for more than 30 yrs and was restocked with *M.*


Mi. cerebralis-free trout in 1994 (J. H. Schachte, personal communication). In Virginia, the parasite was detected in the early 1990s at two state fish hatcheries and four private hatcheries and is considered established in that state (J. G. Hnath, personal communication).

In California, where the range of the parasite has been monitored since its detection in 1966, Mi. cerebralis was reported in wild rainbow and brown trout from the Santa Ana River in the southern part of the state in 1995. It is likely that infection had been present, but undetected for a number of years, as there are records of a local trout grower making frequent purchases from parasite-positive facilities (Modin 1998). Although Mi. cerebralis is now enzootic in a number of rivers in California, in some waters the prevalence has declined in wild fish following closure of facilities with positive populations. The parasite can no longer be detected in wild populations from Garapata and Tularcitos Creeks and the Carmel River, or from the Coleman National Fish Hatchery (Modin 1998).

On the heels of the accumulating evidence from California and other states that wild trout populations can exist with Mi. cerebralis came the first observations of overt whirling disease in free-ranging populations from Colorado and Montana in the mid-1990s. In Colorado, where Mi. cerebralis was first detected in 1987, whirling disease was reported in the upper Colorado River, from an area suffering recruitment failure of wild rainbow trout in 1993, without a concomitant decrease in brown trout populations (Walker and Nehring 1995). At present, the parasite is considered enzootic in all coldwater drainages of Colorado except the North Republican River. Population level reductions among wild rainbow trout have been associated with Mi. cerebralis in the South Platte, Dolores, Roaring Fork, Fryingpan, Gunnison, Cache la Poudre and Rio Grande as well as the Colorado River. In most of these systems, brown trout populations have been unaffected, or have increased, resulting in a stable trout biomass. However, overt whirling disease has been reported even in this resistant species (Walker and Nehring 1995).

In 1994, Montana reported whirling disease in sections of the Madison River where a precipitous decline in estimated rainbow trout numbers had occurred beginning in 1991 (Vincent 1996). In contrast to Colorado, where heavy stocking of rainbow trout had occurred, the Madison River had not been stocked with hatchery fish since the late 1970s. The origin and route of dissemination of the parasite in Montana is unclear, although it had likely been present for some time prior to its detection. Infected fish have never been detected in state, federal or private hatcheries within the state, and many affected waters are managed for wild populations. Detection of the parasite, sometimes at a high prevalence, in nine drainages in the western part of the state (Baldwin et al. 1998) indicates that the parasite has been present for a long period.

Mi. cerebralis was detected in Utah in 1991 at a private facility on the Fremont River, within the Colorado River drainage (Wilson 1991). The parasite was subsequently detected in fish from a pond and from two additional private facilities in the adjacent Sevier River. In 1993, Mi. cerebralis was detected at three private hatcheries on the Bear River drainage; in 1994, infected rainbow trout and kokanee (land-locked form of sockeye salmon O. nerka) with significant skeletal deformities were reported in Porcupine Reservoir and in 1995 the parasite was demonstrated to have extended its range within the Little Bear and Ogden Rivers. Following an extensive survey in 1996, it became clear that although the parasite distribution had expanded within the drainages where it was originally identified, it was not widespread in the state. One state hatchery and at least twelve private hatcheries have tested positive for Mi. cerebralis at various times. Infection at the state hatchery was detected only using molecular techniques and the facility was subsequently depopulated (C. Wilson, Utah Division of Wildlife Resources, personal communication). Population surveys have demonstrated a decrease in rainbow trout populations from 1988 levels in the Beaver River, although the causal relationship between the decline and presence of the parasite has not been established (Eric Wagner, personal communication).

Surveys conducted in Wyoming following detection of the parasite in 1988 have documented Mi. cerebralis from wild fish in the following river drainages: Snake, Wind/Bighorn, Madison, Yellowstone, Green, South Platte, and North Platte. Fish with gross clinical signs of whirling disease have been observed, especially from the Salt River (Snake River drainage). Infected cutthroat trout gillnetted from Yellowstone Lake in 1998 also tested positive for Mi. cerebralis. Five of the 11 state facilities have had infected fish on site, some the result of transfer from other facilities (D. Money, personal communication).
In New Mexico, infected fish have recently been detected in the Pecos, Cebolla, San Juan, Cimarron, Red, and Canones Rivers, and in three of the state’s seven hatcheries. Coldwater streams and reservoirs in the state were inadvertently stocked with infected fish, although it is not yet known to what extent *M. cerebralis* has become established. Surveys of the state’s trout waters for *M. cerebralis* are currently in progress (D. Eib, personal communication). In 2000, Arizona reported that fish had been imported to a private pond from a hatchery in Colorado where *M. cerebralis* was subsequently detected. These suspect fish were stocked at five locations, some consisting of multiple ponds. Fish were recovered and tested from three of the five locations, and infection was confirmed at one site. However, it is too early to determine if the parasite will become established at any of these locations (P. Lopez, Arizona Game and Fish Department, personal communication).

In the Northwest, detection of *M. cerebralis* infections in returning adult salmon illustrates the increased opportunities for dissemination of the parasite as these fish migrate hundreds of miles or more to and from their natal streams. *Myxobolus cerebralis* was confirmed in adult steelhead and chinook salmon returning to two adult capture facilities on the Deschutes River, Oregon, a tributary of the mid-Columbia River. The first record of detection was 1987, and monitoring by Oregon Department of Fish and Wildlife between 1997 and 2000 resulted in detection of infections in stray hatchery steelhead (anadromous rainbow trout), chinook and sockeye salmon. However, several years of intense monitoring of resident fish in the Deschutes River have failed to detect *M. cerebralis* (H. M. Engelking, this volume), and there is currently no evidence that the parasite has become established in any tributaries of the lower Columbia River basin.

**Perspectives on Whirling Disease**

Following the trend in the late 1980s to modify the extreme management reactions to whirling disease, measures short of eradicating the parasite were recommended. These closely followed hatchery guidelines developed in Europe. *Myxobolus cerebralis* was removed from the OIE list of injurious pathogens in 1993 and its presence is no longer being used in regulating imports into the United States.

This de-emphasis on *M. cerebralis* immediately preceded the accumulating evidence of effects on wild populations in certain states. The events that occurred in Montana and Colorado precipitated a renewed interest in whirling disease with a shift in the focus from hatcheries to impacts on wild populations. These reports also resulted in the adoption of more aggressive control policies and increased funding for surveys and research in some states. Colorado initiated an aggressive program to clean up its contaminated state hatcheries and to stop, or severely limit, stocking of infected fish. However, with the establishment of the parasite in most drainages and in a large number of private aquaculture facilities, it may be some time before the effects of these measures are evident. Montana also adopted a plan to mitigate the effects of whirling disease. Because infection has not occurred in any state or private hatcheries, this plan is directed toward educating the public and learning how to manage rivers for reduction or ultimately elimination of the parasite. It is interesting that two states with such markedly different fish management programs should share the distinction of being the most severely affected. In contrast, in California, where the parasite has been established since 1966, there are decades of data and experience to support the view that the parasite has had little impact on wild populations (Modin 1998).

**SUMMARY**

Although breakthroughs on the life cycle of *M. cerebralis* have increased our understanding of parasite biology, they have made little difference in how the disease is controlled. It is worth noting that most control methods recommended today were in effect before tubificids were implicated in the life cycle. In reviewing these historical data it has become evident that many of our hypotheses of how *M. cerebralis* became so broadly distributed are based on the life cycle that was known to exist prior to the implication of the alternate host. We have presented some of these “pre-triactinomyxon” views here because they were the prevailing perspectives at those times. However, it is likely that some, more probable and now obvious, routes of dissemination were dismissed at the time these hypotheses were developed.

A great deal of confusion over the present distribution of *M. cerebralis* in the United States stems from 1) the lack of, and inconsistencies in, survey data, and 2) the fact that many reports are a result of sampling fish known to have been infected when
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Myxosoma cerebralis and peculiarities of its ecology
released. The act of stocking infected fish or the
detection of infected migratory fish in a water body
does not necessarily imply the establishment of the
parasite life cycle capable of infecting susceptible
fry or fingerlings. Confusion has also resulted from
the manner in which data have been presented.
Because the presence of parasite infection does not
necessarily mean that disease will occur, it is impor-
tant to include any data on clinical signs when
sampling fish. Since clinical signs of whirling dis-
ease are not pathognomonic, and may be subtle,
they might not be noted except when the disease
reaches epizootic levels. This could explain why
there is no differentiation between parasite pres-
ence and clinical disease in most reports. Furth-
more, the presence of the pathogen and even frank
disease in wild populations would be unlikely to be
noticed since dead fish would be rapidly scavenge-
d and moribund fish could be subjected to enhanced
predation. In addition, most surveys have present-
ed the distribution of M. cerebralis in a cumulative
manner, so that locations which have tested posi-
tive at even a single point in time remain listed as
such, although subsequent testing has indicated
that the parasite may not have become established
or may be present below detectable levels. In this
review we attempted to address these considera-
tions and portray the status of the parasite as accu-
trately as possible. However, we acknowledge that
data are continually being collected and that dis-
tribution data are constantly in flux.

Strides are being made to develop risk assess-
ment models to determine under what circum-
stances the introduction of a cohort of infected
fish will result in establishment of enzootic or epi-
zootic whirling disease in wild populations. In fact,
in many instances where known planting of infect-
ed fish has occurred there has been no detection of
M. cerebralis in wild fish during subsequent sur-
veys. This difficulty was encountered by us when
drawing up Table 1, in which the simple detection
of the pathogen was reported as positive or nega-
tive, depending on whether the representative
considered detection or establishment as the
prime criterion for reporting a positive outcome.
Thus, one of the primary conclusions (and recom-
mendations) of this manuscript is the compiling
and dissemination of accurate information about
this onerous pathogen and the disease it causes.
This is beginning to be recognized as an important
endeavor and one necessary for proper manage-
ment of wild fisheries.

THE HISTORY AND DISSEMINATION OF WHIRLING DISEASE

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THE HISTORY AND DISSEMINATION OF WHIRLING DISEASE


