The Laurentian Great Lakes compose the largest freshwater ecosystem on earth, which supports a number of economically and ecologically valuable sport and commercial fisheries. A combination of habitat destruction, invasions by exotic species, and the overexploitation of the fishery decimated stocks of native fish species in the Great Lakes basin (GLB). As a result, large numbers of Chinook (Oncorhynchus tshawytscha) and Coho (O. kisutch) Salmon were introduced into the GLB in 1966 to rebalance the energy flow of the system and to maintain commercially-viable sport fisheries. Unfortunately, the intentional and non-intentional introduction of non-native aquatic species facilitated the invasion of exotic fish pathogens and diseases into the GLB, to which native fish species were very vulnerable.

In the GLB, most conservation and restoration efforts depend primarily on manipulating stock sizes by the stocking of hatchery-propagated fingerlings or yearlings. This practice requires collecting gametes from wild, feral, and domesticated broodstocks and raising the offspring to a stage that would yield a high survival rate when released into the GLB. Maintaining the health of these hatchery-propagated fish is instrumental to the success of these efforts. This can only be achieved by implementing strict biosecurity measures and meticulous husbandry techniques in fish production facilities to minimize the spread of disease-causing organisms. In the same context, effective disease control measures in wild fish stocks are essential to prevent the spread of pathogens and reduce their effects on these stocks. Developing such disease management strategies depends on thorough knowledge of the biological properties of both the pathogen and host, the hydrobiological characteristics of the waterbody, as well as prevailing environmental conditions. In the presence of limited knowledge on these three main factors, including their interactions, developing an effective disease control strategy has been extremely challenging.

For example, in the last three decades, an ecological catastrophe has occurred within the GLB with the invasion by non-native dreissenids, such as the Zebra Mussel (Dreissena polymorpha) and the Quagga Mussel (D. rostriformis bugensis). Due to their high water-filtering rates, dreissenids have been able to alter the composition and structure of the food web at the lower trophic level in the most of the Great Lakes, leading to decline in abundance of important prey items (e.g., Diporeia spp.) and decline in the abundance and condition of the fish species they support (Nalepa et al. 1998; Nalepa et al. 2000; McNickle...
et al. 2006). The effects of the dreissenid invasion on the flourishing of fish pathogens or the susceptibility of resident fish have not been determined; however, concomitant with this invasion, a number of serious, novel diseases of fish have emerged, often in an epidemic manner that can threaten the success of the Great Lakes fisheries conservation efforts.

The list of emerging diseases in the GLB continues to increase with the most recent emergence of the Viral Hemorrhagic Septicemia Virus (Rhabdovirus, Novoviridae); the Largemouth Bass Virus (Ranavirus, Iridoviridae); a Piscirickettsia-like infection in the Muskelunge (Esox masquinongy) population of Lake St. Clair; visceral mycosis in propagated Chinook Salmon fingerlings; and the Heterosporis sp. infection. The situation has been further exacerbated by the inability to eliminate endemic diseases that continue to affect important fish stocks, such as the Bacterial Kidney Disease caused by Renibacterium salmoninarum, furunculosis caused by Aeromonas salmonicida, and flavobacteriosis caused by Flavobacterium and Chryseobacterium spp. What causes these pathogens to emerge, retract, or resurge and why the control efforts are far from eliminating these serious pathogens are questions currently unanswered. To this end, it is obvious that the severe lack of knowledge on fish diseases threatening Great Lakes fisheries constitutes a major impediment toward developing an effective comprehensive disease control management strategies in the GLB.

The objectives of this chapter are (1) to provide fishery biologists and managers with a synopsis of what is known about the major pathogens of potential threat to important fish stocks in the GLB and (2) to shed light on the efforts of the federal, state, and tribal agencies in developing and implementing a regional disease control program. This review is not intended to cover or even list each reported parasite, pathogen, or disease condition; rather, it focuses on emerging and widespread diseases of importance to conservation efforts.

Fish Diseases in the GLB with Emphasis on Michigan

As previously mentioned, reports that describe diseases and their impacts on fish populations in the GLB have been relatively scarce. The majority of the GLB disease information is found in the grey literatures or interagency correspondence and often focuses on microorganisms and parasites that cause negligible harm to their hosts. However, because of some major fish mortality events, a number of fish diseases in the GLB have made appearances on the first page of national newspapers. The Viral Hemorrhagic Septicemia Virus is one such example in the 2000s, whereas Bacterial Kidney Disease raised public concerns in the 1980s about the future of sport fisheries in Lake Michigan.

Bacterial Diseases

Diseases caused by bacteria are a serious problem in both wild and propagated fish stocks. Numerous mortality events in economically and ecologically important fish stocks have been found associated with the presence of fish-pathogenic bacteria (Holey et al. 1998; Klindt and Town 2005; Monette et al. 2006). This section discusses some examples of widespread bacterial infections in Great Lakes fish species.
**RENBACTERIUM SALMONINARUM**

*Renibacterium salmoninarum* is a Gram-positive diplobacillus that causes a granulomatous, chronic, and frequently occurring disease of salmonids, known as Bacterial Kidney Disease (BKD). Bacterial kidney disease was first reported in North America in 1935 in captive Brook (*Salvelinus fontinalis*), Brown (*Salmo trutta*), and Rainbow Trout (*Oncorhyncus mykiss*) stocks in Massachusetts (Rucker et al. 1951). By 1956, BKD spread west to Iowa, Michigan, Minnesota, Nebraska, South Dakota, Wisconsin, and Wyoming. The first published report of BKD in the GLB was made by Allison (1958), who described two cases of BKD in Michigan fish hatcheries in 1955. From 1955 to 1964, there were many cases of BKD in propagated fish lots from several Great Lakes states. MacLean and Yoder (1970) reported a high prevalence of the disease (more than 50 percent) in Coho, Chinook, and Kokanee (*O. nerka*) Salmon from several sites in Lakes Michigan and Superior, as well as from a Michigan fish hatchery and state research stations. In 1972, BKD became widespread and the Great Lakes Fish Health Committee (GLFHC, a body of the Great Lakes Fishery Commission) declared it enzootic within the basin. The GLFHC further recommended that member agencies should not import or stock fish with overt signs of the disease (Hnath 1993).

In 1986–1987, there were widespread reports of Chinook Salmon kills (estimated at between ten thousand and twenty thousand fish) washed up onto the beaches of southern Lake Michigan. *Renibacterium salmoninarum* was detected in these clinically diseased adult fish (Johnson and Hnath 1991). Holey et al. (1998) attributed the Chinook BKD epizootics in Lake Michigan to a reduced abundance of Alewives (*Alosa pseudoharengus*) and other prey species. To control BKD in hatcheries, the states of Michigan and Wisconsin fed erythromycin medicated diets to all Coho and Chinook Salmon reared from eggs collected from Lakes Michigan and Huron. By 1993, Michigan started to cull adult salmon with BKD at spawning weirs based on clinical signs and field-enzyme linked immunosorbent assays. Two years later, the GLFHC recommended that fisheries agencies not stock hatchery-reared lots in which seven or more of sixty randomly sampled fish tested positive for *R. salmoninarum* by the direct fluorescent antibody test (DFAT) conducted at no more than eight weeks and not less than two weeks before they were stocked (Stark 1995).

Such strategies improved fry and fingerling survival within state and federal hatcheries and after stocking. For example, after broodstock culling was initiated at Michigan’s Platte River egg take weir, the average hatchery mortality rate of Coho Salmon dropped from 12.3 percent in 1982–1991 to 3.7 percent in 1992–1998 (Beyerle and Hnath 2002). The recreational catch per one hundred angler hours in Lake Michigan also recovered from a low of less than two fish to more than four fish by 1996. Furthermore, the prevalence of clinical disease among Lake Michigan salmon returns at harvest weirs dropped from 12.2 percent in 1991 to 2.6 percent in 1999 for Chinook Salmon adults and from 14.7 percent in 1991 to 2.8 percent in 1999 among Coho Salmon (Beyerle and Hnath 2002).

By 1996, the salmon fishery in Lake Michigan had rebounded and few reports were made of dead and/or dying salmon; however, the disease remained a concern. Coho Salmon, Chinook Salmon, Rainbow Trout, Lake Trout (*Salvelinus namaycush*), Lake Whitefish (*Coregonus clupeiformis*), and Bloater (*C. hoyi*) collected in Lakes Michigan and Huron were still found to harbor *R. salmoninarum*, and some fish exhibited clinical signs of BKD (Beyerle and Hnath 2002; Jonas et al. 2002; Faisal et al. 2010b). Interestingly, BKD appears to have also emerged in other non-salmonid fishes, as *R. salmoninarum* has been detected in Sea Lamprey (*Petromyzon marinus*) collected from the Humber River and Duffins Creek, Ontario, Canada, at a prevalence of 31.5 percent (Eissa et al. 2006).

In a 2000–2003 survey of feral salmonid fish populations, Michigan Department of Natural Resources
FIG. 1. The prevalence and intensity of *R. salmoninarum* in feral Chinook Salmon (*Oncorhynchus tschawytscha*) returning to spawn at the Little Manistee River Weir, Lake Michigan watershed, from 2001 to 2009. Prevalence and intensity of *R. salmoninarum* was based on results of a quantitative Enzyme Linked Immunosorbent Assay (Q-ELISA) conducted on kidney homogenates. According to the intensity of the color reaction, positive samples could be stratified into low, medium, or high, depending on *R. salmoninarum* antigen concentration in fish tissues.

(MDNR) and the U.S. Fish and Wildlife Service (USFWS) detected *R. salmoninarum* in fifty-one of sixty-seven watersheds (76.1 percent) within the Great Lakes. Additional long-term (2001–2010), state-wide monitoring and analysis conducted by the MDNRE and the Michigan State University Aquatic Animal Health Laboratory (MSU-AAHL) demonstrated the prevalence and intensity of *R. salmoninarum* among Chinook Salmon returning to spawn to weirs in Lakes Michigan and Huron has been as high as 81.6 percent and 45.7 percent, respectively, in the early 2000s (figs. 1 and 2). However, from 2005 to 2009, the prevalence of *R. salmoninarum* in Lakes Michigan and Huron Chinook Salmon has decreased dramatically, with the current overall prevalence less than 10 percent in both lakes (figs. 1 and 2). Although there may be other contributing factors, the decline of BKD is also concomitant with the implementation of more stringent biosecurity measures in the state of Michigan and other state hatcheries in the basin. It should be noted that the prevalence of *R. salmoninarum* has been consistently greater in Lake Michigan Chinook Salmon than in Lake Huron Chinook Salmon.

Because of the nature of vertical transmission, managers have attempted to minimize the occurrence and intensity of *R. salmoninarum* in the pre-stocking fingerlings. As a result, the prevalence of *R. salmoninarum* in pre-stocking fingerlings from Lake Michigan Chinook Salmon decreased from 96 percent in 2003.
FIG. 2. The prevalence and intensity of *R. salmoninarum* in feral Chinook Salmon (*Oncorhynchus tschawytscha*) returning to spawn at the Swan River Weir, Lake Huron watershed, from 2002 to 2009. Prevalence and intensity of *R. salmoninarum* was based on results of a quantitative Enzyme Linked Immunosorbent Assay (Q-ELISA) conducted on kidney homogenates. According to the intensity of the color reaction, positive samples could be stratified into low, medium, or high, depending on *R. salmoninarum* antigen concentration in fish tissues.

To a low of 0 percent in 2010, reflecting the efficiency of the implemented biosecurity program at Michigan state fish hatcheries (fig. 3). The prevalence of *R. salmoninarum* has also experienced a decline in progeny from Lake Huron Chinook Salmon, from 46 percent in 2003 to 0 percent in 2010 (fig. 4). Interestingly, the declines in prevalence in the pre-stocking fingerlings correspond to the decline of *R. salmoninarum* in the parent broodstock as well, demonstrating that the BKD epizootic in Chinook Salmon has diminished during the last decade.

Similarly, there has been a steady decline in the prevalence of *R. salmoninarum* in strains of feral Coho Salmon from Lake Michigan. There are two strains of Coho Salmon that return from Lake Michigan to spawn, the Hinchinbrook (HB) strain, which was imported to Michigan from New York in the late 1980s (abandoned as a broodstock strain in 2007), and the Michigan-adapted strain, which was originally imported from the Pacific Northwest and has been in Michigan since late 1960. The HB strain, which was sampled from 2001 to 2007, had a prevalence as high as 100 percent in 2001 and declined dramatically to 1.7 percent in 2007 (fig. 5). The Michigan-adapted strain has experienced a similar decline in prevalence of *R. salmoninarum*, from 84 percent in 2001 to a low of 0 percent in 2009 (fig. 6). Additionally, the decline in the prevalence of *R. salmoninarum* in the HB and Michigan-adapted parental broodstock coincided...
FIG. 3. The prevalence and intensity of *R. salmoninarum* in hatchery-raised Little Manistee River Chinook Salmon (*Oncorhynchus tschawytscha*) progeny from 2002 to 2010.

FIG. 4. The prevalence and intensity of *R. salmoninarum* in hatchery-raised Swan River Chinook Salmon (*Oncorhynchus tschawytscha*) progeny from 2003 to 2010. (*No progeny were produced this year.*)
FIG. 5. The prevalence and intensity of *R. salmoninarum* in the Hinchenbrook strain of Coho Salmon (*Oncorhynchus kisutch*) returning to spawn at the Platte River Weir, Lake Michigan watershed, from 2001 to 2007.

FIG. 6. The prevalence and intensity of *R. salmoninarum* in the Michigan-adapted strain of Coho Salmon (*Oncorhynchus kisutch*) returning to spawn at the Platte River Weir, Lake Michigan watershed, from 2001 to 2009.

FIG. 8. The prevalence and intensity of *R. salmoninarum* in hatchery-raised Michigan-adapted Coho Salmon (*Oncorhynchus kisutch*) progeny (MI) from 2003 to 2010.
with a decline in the progeny as well, resulting in a prevalence of 0 percent in 2007 and 2010, respectively (figs. 7 and 8). A similar trend was observed in the prevalence of *R. salmoninarum* in Lake Michigan wild Steelhead (Rainbow Trout), returning spawners to the Little Manistee Weir, which have remained very low compared to other fish species and further declined over the years. As a result, Steelhead Trout progeny have also maintained a low prevalence of the disease.

Furthermore, a recent study found that the prevalence of *R. salmoninarum* in Lake Whitefish from Lakes Huron and Michigan was about 60 percent, which is higher than in other salmonids sampled during the same time (Faisal et al. 2010b). The prevalence, however, fluctuated between adjacent sampling periods, sites, and seasons (Faisal et al. 2010b). The sum of these findings demonstrates that *Renibacterium salmoninarum* continues in the GLB wild stocks, thus, remaining a pathogen that must be actively managed around by fishery managers.

*AEROMONAS* SPP.

Members of the genus *Aeromonas* are a major cause of diseases in fish worldwide and are ubiquitous to freshwater, brackish, and marine environments. These Gram-negative bacteria are associated with diseases in both poikilothermic and homeothermic organisms. At present, there are twenty-four described species within this ever growing genus; among these are a number of motile species, such as members of the *A. hydrophila*, *A. sobria*, and *A. caviae* complexes, as well as the non-motile bacterium, *A. salmonicida*, frequently associated with diseases in fish (Cipriano and Bullock 2001).

*AEROMONAS SALMONICIDA*

In the Great Lakes, this bacterium has been associated with numerous disease and mortality events in cultured, feral, and wild Great Lakes fish stocks and was such a serious problem that *A. salmonicida* became reportable for the eight U.S. Great Lake states and the two Canadian provinces under the Great Lakes fish disease control policy and model program (Hnath 1993). Until recently, published reports describing disease prevalence in the Great Lakes did not exist; however, successful treatment of furunculosis using antibiotics is documented from as early as the 1950s in the records of the MDNR Fish Health Laboratory. Records also document the use of commercial anti-furunculosis vaccines with remarkable success in Michigan state fish hatcheries. For example, historical accounts of furunculosis outbreaks, involving broodstock and production fishes, show they were common within all Michigan state fish hatcheries (Beyerle and Hnath 2002); however, through the implementation of increased biosecurity measures and commercially available vaccines, no outbreaks of furunculosis have occurred since 2005.

In contrast, *A. salmonicida* infections have been detected in a number of feral and wild Great Lakes fish stocks, particularly salmonids. For example, Cipriano et al. (1996a) found an 18–38 percent prevalence of *A. salmonicida* infections in Coho Salmon returning to spawn in the Salmon River, Altmar, New York, whereas Bruneau et al. (1999) found that *A. salmonicida* infections were common in wild salmonid stocks throughout Ontario. Beyerle and Hnath (2002) detected *A. salmonicida* in spawning Chinook Salmon returning to the Boardman River Weir, the Little Manistee River Weir (LMRW), the Platte River Weir (PRW), and the Swan River Weir (SRW). Infections with this bacterium were also detected in Coho Salmon from LMRW and PRW, whereas steelhead trout returning to the LMRW were also found infected with *A. salmonicida* (Beyerle and Hnath 2002). Most recently, *A. salmonicida* infections in salmonid broodstock returning to gamete collecting weirs have varied among fish species, location, and year of sampling (fig.

and have also coincided with clinical disease in the form of furuncles (fig. 10a) that can progress into deep cavitating ulcerations (fig. 10b). For example, furunculosis prevalence in Chinook Salmon returning to the LMRW to spawn fluctuated between approximately 3 percent in 2008 and 60 percent in 2009, but consistently shows the highest infection prevalence compared to all other sampled weirs in Michigan (Records of MSU-AAHL). *A. salmonicida* infections are also enzootic in Coho Salmon returning to the PRW and Atlantic salmon (*Salmo salar*) returning to the St. Mary’s River, albeit at a much lower prevalence than the LMRW (fig. 9).

In addition, furunculosis has been observed in the HB strain of Coho Salmon and the Alaskan strain of Coho Salmon collected from Thompson Creek, as well as in other non-*Oncorhynchus* salmonids in Michigan, including Brook Trout from the Pigeon River Research Lakes (Beyerle and Hnath 2002). More recent studies have also shown that Lake Whitefish from Lakes Michigan and Huron are susceptible to infections by *A. salmonicida* subsp. *salmonicida* that can result in damage to the host (Loch and Faisal 2010a). Although these infections were detected at a relatively low prevalence (0.3 percent in 635 fish from Lake Huron, 0.3 percent in 651 fish from Lake Michigan), it is possible that Lake Whitefish serve as a reservoir for other more susceptible Great Lakes fish species. *A. salmonicida* has also been recently
recovered from Bluegill (*Lepomis macrochirus*) and Yellow Perch (*Perca flavescens*) reared in private hatcheries within Michigan (Records of MSU-AAHL). Additionally, clinical furunculosis has been observed in Sea Lamprey inhabiting Lake Ontario, further illustrating the wide host range of this bacterium (Faisal et al. 2007a). This endemic pathogen will continue to be actively managed in the Great Lakes Basin and be of concern to fisheries managers.

**MOTILE *AEROMONAS***

Motile members of the genus *Aeromonas* are pathogenic to fish (Cipriano 2001) and cause multiple disease syndromes, collectively referred to as motile aeromonad septicemia (MAS). These include members of the *A. hydrophila*, *A. sobria*, and *A. caviae* complexes, as well as a number of other species not falling into these three complexes, and are commonly associated with mortality events in hatchery and wild fish stocks alike.

Within the Great Lakes system, a mass mortality event involving Common Carp (*Cyprinus carpio*) residing in the St. Lawrence River was partially attributed to motile aeromonads (Monette et al. 2006). These bacteria are commonly recovered from salmon and trout returning to Michigan gamete-collecting weirs. A recent study of bacterial pathogens infecting spawning Chinook Salmon returning to weirs within the Lake Michigan watershed found an infection prevalence of approximately 10 percent (420 fish; Loch et al. 2012). A significant association between the probability of *R. salmoninarum* and motile aeromonad infections was found, suggesting a possible synergism between these two bacterial fish pathogens (Loch et al. 2012). In a recent study, Loch and Faisal (2010b) detected motile aeromonad infections in Lake Whitefish collected from Lake Michigan at an overall prevalence of 13.3 percent and from Lake Huron at an overall prevalence of 15.7 percent. This study also found that the majority of the detected motile

**FIG. 10.** Lesions in Chinook Salmon (*Oncorhynchus tshawytscha*) caused by *Aeromonas salmonicida*: (A) swelling in the dorsolateral musculature (termed furuncle) and (B) hemorrhagic deep ulceration penetrating into the musculature of the infected fish.
aeromonad infections occurred in Lake Whitefish that also harbored other infectious agents, such as *R. salmoninarum*, *Cystidicola farionis*, *Neoechinorhynchus tumidus*, and *Echinorhyncus salmonis*, further suggesting a synergism between motile *Aeromonas* spp. and other fish pathogens.

**FLAVOBACTERIUM SPP.**

Since the first report in the early 1920s, flavobacteriosis has posed a serious threat to wild and propagated fish stocks. Traditionally, the disease has been attributed to three bacterial species within a family now known as Flavobacteriaceae. As a group, *Flavobacterium* spp. have recently accounted for more fish mortality in the state of Michigan and its associated hatcheries than all other pathogens combined (Records of MSU-AAHL). Acute flavobacteriosis can greatly reduce infected fish populations, whereas survivors suffer poor growth and spinal abnormalities. In sub-acute and chronic infections, flavobacteriosis can cause lingering mortalities, thereby causing significant economic losses and negatively impacting post-stocking performance (reviewed in Shotts and Starliper 1999).

For decades, three *Flavobacterium* spp. have dominated fish disease literature: *F. columnare*, the causative agent of columnaris disease; *F. psychrophilum*, the etiological agent of bacterial coldwater disease and Rainbow Trout fry syndrome, both of which are serious diseases of salmonids; and *F. branchiophilum*, the agent of bacterial gill disease. *Flavobacterium branchiophilum* has caused perennial problems in some fish production facilities within the GLB, thus, necessitating diligent applications of antimicrobial agents ranging from the now-banned pyridylmercuric acetate in the 1950s and 1960s (Allison 1957), through quaternary ammonia compounds in the 1970s and 1980s (Hnath 1975), and more recently Chloramine-T, sodium para-toulene-sulphonchloramide (Bowker and Erdahl 1998) and hydrogen peroxide (Rach et al. 2000). We expect this ubiquitous bacteria group will continue to dominate fish health concerns into the future. The following two species of *Flavobacterium* and the emergence of novel Flavobacterium deserve specific discussions because of their documented effects on hatchery populations of salmonids.

**FLAVOBACTERIUM PSYCHROPHILUM**

*Flavobacterium psychrophilum* infections are regularly detected in feral salmonids returning to spawn at Michigan gamete collecting weirs during annual fish health examinations, including Chinook, Coho, and Atlantic salmon, as well as Steelhead (Rainbow Trout). For instance, during the spring spawning run of Steelhead (Rainbow Trout) returning to the LMRW in April of 2010, *F. psychrophilum* was isolated from more than 25 percent of the fish sampled.

As this bacterium is vertically transmitted from infected parent to progeny, stringent egg disinfection procedures are pivotal in preventing the spread of infection to the production fish destined to be raised within fish hatcheries. Despite the employment of rigorous egg disinfection procedures, bacterial coldwater disease annually causes high losses of production of Coho Salmon, Brown Trout, Lake Trout, Rainbow Trout, and other species in facilities using open water sources that become quite cold (near 0°C) during winter months, particularly when there are high flows in the source water that create a lot of turbidity. Although this bacterium responds to oral antibiotic therapy, treatment is usually ineffective, when the disease is most severe, as the fish do not feed well during the cold water periods.

Disease signs associated with *F. psychrophilum* infection often include severe erosion of the caudal peduncle that can lead to exposure of vertebrae/dorsal spines (fig. 11a), thus, creating a portal on entry for secondary invaders (e.g., *Saprolegnia* spp.; fig. 11b), and severely swollen spleens (fig. 11c). This disease
FIG. 11. (A) Hatchery-raised Rainbow Trout (*Oncorhynchus mykiss*) with bacterial cold water disease (BCWD) exhibiting ulceration of the caudal peduncle with the vertebral column exposed (arrow); (B) Rainbow Trout with BCWD, where peduncular ulceration has been invaded by fungi of the *Saprolegnia* spp.; and (C) severely swollen spleen of a Rainbow Trout with BCWD.

can generate extremely high losses in affected lots of production fish, exceeding 85 percent in Lake Trout (Schachte 1988). Interestingly, *F. psychrophilum*, known to affect salmonids primarily, was recently recovered from infected Sea Lamprey inhabiting Lake Ontario (Elsayed et al. 2006). This again shows that many of the salmonid diseases are likely to have a much wider host range as additional information on other species is collected.

**FLAVOBACTERIUM COLUMNARE**

*F. columnare* is one of the most well-known species within its genus because of its ability to cause serious disease events, especially in the extensive culture of cool and warm-water fish species. Hatchery mortalities associated with this bacterium in the GLB were seldom published; however, Pecor (1978) reported 6
FIG. 12. (a) Extensive erosion of the anal fin of a spawning Chinook Salmon (*Oncorhynchus tschawytscha*) systemically infected with *Flavobacterium columnare* and (B) focal erosion of gill lamellae of a spawning Chinook Salmon (*Oncorhynchus tschawytscha*) systemically infected with *F. columnare*.

percent mortality within a seven day period among cultured tiger Muskellunge (male *Esox lucius* x female *Esox masquinongy*), whereas severe fin erosions in cultured Walleye (*Stizostedion vitreum vitreum*) was attributed to *F. columnare* infection by Clayton et al. (1998). Most recently, an outbreak of columnaris disease in production Muskellunge at Michigan’s Wolf Lake State Fish Hatchery resulted in heavy losses for the 2009 year class and almost wiped out all infected lots before Chloramine-T and florfenicol treatments, eventually proved effective. Affected fish showed shallow epidermal and dermal ulcerations along the dorsal musculature that sometimes also involved the dorsal fins. High mortality rates have been observed in extensively-reared Walleye fingerlings (Records of the MSU-AAHL). Chloramine-T has been used to effectively control columnaris infections among cultured fish (Bowker and Erdahl 1998).

*F. columnare* is commonly recovered from infected salmonids returning to Michigan weirs to spawn. For example, Chinook Salmon returning to the LMRW had an *F. columnare* infection prevalence of about 28 percent in 2006, about 35 percent in 2007, about 12 percent in 2008, and about 54 percent in 2009 (Records of MSU-AAHL). The prevalence of *F. columnare* infections in Michigan Coho Salmon returning to the PRW from 2006 to 2009 fell somewhere in between, ranging from about ~4 percent to about 22 percent (Records of MSU-AAHL). The most predominant pathology associated with infections was extensive erosion and necrosis of the fins (fig. 12a) and gills (fig. 12b).
NOVEL FLAVOBACTERIA
The period from 2000 to 2009 has witnessed the emergence of multiple novel flavobacterial species that have caused substantial damage to fish stocks worldwide. In this context, the MDNRE and MSU-AAHL undertook extensive field and laboratory studies to identify emerging and resurging pathogens threatening conservation efforts in wild and propagated fish stocks. The multiyear studies identified flavobacteria and chryseobacteria as a major cause of fry and fingerling mortalities. When 144 Michigan flavobacteria isolates were analyzed by ribosomal RNA partial gene sequencing, numerous novel flavobacterial strains were identified (Records of MSU-AAHL). Members of the genera *Flavobacterium* and *Chryseobacterium* were associated with deadly septicemias in Brown Trout, Rainbow Trout, Coho Salmon, and Chinook Salmon fry in propagated and wild fishes. These bacterial strains, never before reported from North America, shared similarities with strains found in Europe and South Africa, a matter that implies the emergence of these bacteria in the GLB.

Other novel flavobacteria isolated from Michigan were associated with mortality episodes in which disease signs and tissue alterations mimicked those associated with *F. columnare, F. psychrophilum, and F. branchiophilum* (fig. 13a-d). What effects these novel flavobacteria will have on Great Lakes fishes remains to be determined and is the focus of a number of ongoing detailed studies by the MSU-AAHL and others in the GLB.

FIG. 13. (A) A *Chryseobacterium*-infected Steelhead (Rainbow Trout; *Oncorhynchus mykiss*) spawning at the Little Manistee River Weir (LMRW) with extensive erosion of the soft rays of the dorsal fin; (B) ulceration on the trunk of a *Chryseobacterium*-infected LMRW Steelhead; (C) ulceration on the dorsal surface of a flavobacteria-infected brown trout (arrow); and (D) flavobacteria infected Rainbow Trout exhibiting erosion of the epidermal and dermal layers of the caudal peduncle penetrating into the musculature and exposing vertebrae (arrow).
The genus *Carnobacterium* currently contains nine Gram-positive, atypical lactobacilli species, including *C. maltaromaticum*, which has been reported in association with a number of fish disease outbreaks. Disease outbreaks associated with *Carnobacterium* spp., often referred to as pseudokidney disease, cause post-spawning morbidity and mortality. Recently, a *Carnobacterium* sp. that closely resembled *C. maltaromaticum*, yet also showed distinct phenotypic and genotypic differences according to 16S rRNA gene sequencing and phylogenetic analysis, was recovered from Lake Whitefish inhabiting Lakes Michigan and Huron (Loch et al. 2008) at a prevalence of 2.2 percent (651 fish total) and 1.4 percent (635 fish total), respectively. Clinical signs in infected Lake Whitefish included splenomegaly, renal hyperemia and friability, hepatic motting and pallor, and copious amounts of a mucoid exudate within the lumen of opaque and thickened swimbladders (Loch et al. 2008). The prevalence of these infections varied statistically by season, with Lake Whitefish collected during the winter months over a three year period having the highest prevalence (Loch et al. 2008). Interestingly, these winter samples correspond to a post-spawning period for Lake Whitefish, which is a time commonly associated with *Carnobacterium* epizootics in other salmonid species. As such, spawning stress may play an important role in *Carnobacterium*-derived infections in Great Lakes Lake Whitefish (Loch et al. 2008).

Most recently, members of the genus *Carnobacterium* have also been detected in feral and captive *Oncorhyncus* spp. broodstock and in association with mortalities. Out of 1,564 Rainbow Trout (Steelhead), Coho Salmon, and Chinook Salmon sampled from egg-take weirs throughout Michigan during 2005–2008, 57 *Carnobacterium* isolates were recovered from the kidneys, spleens, swimbladders, and/or external ulcerations of 51 infected fish (Records of MSU-AAHL). Analyses of representative isolates via partial 16S rRNA gene sequencing and phylogenetic analyses showed greater than 99 percent similarity to *C. maltaromaticum*, whereas one isolate was most similar to *C. divergens*. *Carnobacterium* spp. have also recently been recovered from disease and/or mortality events in captive Lake Trout, Brook Trout, and Brown Trout, as well as from wild Mottled Sculpin (*Cottus bairdii*) and hatchery reared Muskellunge fingerlings and Channel Catfish (*Ictalurus punctatus*) without signs of disease. These data indicate this bacteria group likely has a wide host range and must be considered as a possible epizootic factor in fish kills by fisheries managers.

*Yersinia ruckeri*

*Yersinia ruckeri*, a causative agent of enteric red mouth disease, is intensively screened for in Great Lakes fish stocks, as it is known to be enzootic in localized areas throughout the basin. Historically, this bacterium was detected in Steelhead (Rainbow Trout) collected from Lake Michigan in 1987 and from spawning Chinook Salmon returning to SRW in 1998 and 1999 (Beyerle and Hnath 2002). Interestingly, *Y. ruckeri* was also isolated from the kidney of one Chinook Salmon (*N* = 60) returning to the SRW in 2007, an occurrence not observed since 1999. *Yersinia ruckeri* has not been detected in any other salmonids returning to the egg-take in Michigan (Records of MSU-AAHL). The most recent detections of this bacterium from Michigan fishes occurred in wild Northern Pike (*Esox lucius*) broodstock collected from Sanford Lake in 2007 (about 3 percent prevalence). *Yersinia ruckeri* was also isolated from wild Northern Pike broodstock collected from Little Bay de Noc in 2008 (about 2 percent prevalence, *N* = 60 fish) and in 2009 (about 3 percent prevalence, *N* = 60). Infected fish did not display any overt signs of disease and most likely represented carriers of this bacterium (Records of MSU-AAHL).
Piscirickettsia-like infections have been observed since 2002 among adult Muskellunge (Esox masquinongy) in Lake St. Clair (LSC; Records of MSU-AAHL) and have been attributed to Muskellunge mortality events in LSC from 2000 to 2003. Affected fish have exhibited red skin rashes with red raised ulcers and subcutaneous edema within the Anchor Bay area of LSC, near Detroit, Michigan. The rounded rashes may reach one inch in diameter. Laboratory analyses and electron microscopy have revealed the association of these lesions with a Gram-negative, curved, intracellular bacterium that occurs as rings or curved rods. Based on morphological characteristics, the agent was determined to be a Piscirickettsia-like organism (fig. 14a-b). Disease is characterized with granulomatous skin ulcers and glomerular degeneration in the kidney (Records of MSU-AAHL). A similar, yet not identical, organism, Piscirickettsia salmonis, was the first “rickettsia-like” bacterium recognized as a fish pathogen. This species infects a wide range of salmonid fishes and has caused systemic infections associated with high mortalities in Chile, Norway, Ireland, and Canada. Piscirickettsia salmonis has only been found in salmonid fish species. This bacterial species, which has very distinct chronic symptoms, is likely a relatively recent invader to the GLB and will need to be watched as a potential epizootic factor for cool-water species by fisheries managers.
**TYPE C BOTULISM**

Caused by the bacterium *Clostridium botulinum*, type C botulism is commonly associated with aquatic environments and has been responsible for numerous waterfowl and fish mortalities throughout the world. In the Great Lakes region, type C botulism has been reported since 1936, and, in Michigan specifically, it has been reported from both shoreline areas of Lakes Michigan and Huron, as well as some inland lakes. Type E botulism is commonly associated with fish, birds, or mammals that feed on sick or dead fish. *Clostridium botulinum* is commonly found in the sediment and resides in invertebrates, such as amphipods, mussels, oligochaetes, and chironomids (Pérez-Fuentetaja et al. 2006). The bacterium toxin is transmitted to fish and birds via contaminated food; therefore, bird and mammal die-offs often coincide with fish kills. In 1963 and 1964, an outbreak was responsible for the deaths of at least 6,870 loons, 5,110 gulls, and 660 birds of miscellaneous species along the Michigan shoreline of Lake Michigan (Fay 1966). Moreover, in 1999, massive die-offs of millions of Alewives (*Alosa pseudoharengus*) and Gizzard Shad (*Dorosoma cepedianum*), as well as over 6,000 waterbirds on the U.S. and Canadian shores of Lake Erie were caused by the toxin from type E botulism (Domske and Obert 2001). This was followed in 2000 by additional waterbird mortalities, which were also attributed to the toxin from type E botulism (Domske and Obert 2001). In 2006, approximately 2,900 waterbirds were found dead in northern Lake Michigan (near the Sleeping Bear Dunes National Lakeshore), with type E botulism once again determined as the cause of death (Byappanahalli and Whitman 2009). Reports of type E botulism causing fish and bird mortalities are on the rise and may be attributable to the benthification of nutrients by dressinids that have, in turn, produced large amounts of *Cladophora* sp. When the thick mats of this algae die, it creates anaerobic conditions, leading to type E botulism. This will be a continuing issue for the GLB.

**OTHER BACTERIAL INFECTIONS**

Numerous other bacterial species have been recovered from diseased fish and/or mortality events in Great Lakes fish stocks. For example, *Proteus* sp., *Salmonella* sp., and *Seratia* sp. have been recovered from Common Carp and White Suckers (*Catostomus commersoni*) residing within the Great Lakes (Souter et al. 1976). These same three bacterial species, all of which belong to the family *Enterobacteriaceae*, were also recently recovered from the kidneys of Chinook Salmon returning to the LMRW during the last few years (Records of MSU-AAHL) and are occasionally associated with mixed bacterial infections in production hatchery fishes. Other members of the family *Enterobacteriaceae*, such as *Hafnia* spp., *Morganella* spp., *Escherichia* spp., and *Providencia* spp., are also occasionally isolated from wild and hatchery Great Lakes fish stocks with mixed bacterial infections. *Pantoea agglomerans*, a ubiquitous Gram-negative epiphytic bacterium commonly associated with plants, was recently recovered from a systemically infected Brown Trout residing within Gilchrist Creek (Lake Huron watershed; Loch and Faisal 2007). This was the first report of this bacterium causing infections within Great Lakes fishes and was of major interest, as *P. agglomerans* is an emergent cause of localized and systemic infections in humans (Loch and Faisal 2007). *Plesiomonas shigelloides* has also been recovered from a number of diseased Great Lakes fishes, particularly Largemouth Bass (*Micropterus salmoides*), Smallmouth Bass (*M. dolomieu*), and other centrarchids, during the summer months, from several Michigan inland lakes, including Camp 8 Lake (Luce County), Gilchrist Creek (Montmorency County), and Gourdneck Lake (Kalamazoo County; Records of MSU-AAHL). This bacterium has also occasionally been isolated from Muskellunge fingerlings reared at Michigan’s Wolf Lake State Fish Hatchery in association with mixed bacterial septicemia (Records of MSU-AAHL). *Shewanella*
**FIG. 15.** Juvenile Muskellunge (*Esox masquinongy*) infected with viral hemorrhagic septicemia virus-genotype IVb exhibiting: (A) severe gill palor (asterisk) with hemorrhaging at the dorso-lateral aspect of the branchial chamber (arrow), (B) dermal petechial hemorrhages (arrows), and (C) and hemorrhaging within the musculature (arrows).

*putrifaciens* is also occasionally recovered from a number of wild and aquacultured fish stocks experiencing mixed bacterial infections, including Sea Lamprey, captive Muskellunge fingerlings, and many different salmonid production stocks found throughout the State of Michigan. Determination of the reservoir of these bacteria is being studied.

**Viral Diseases**

**VIRAL HEMORRHAGIC SEPTICEMIA VIRUS**

In 2003, a virus was isolated from tissues of Muskellunge collected from LSC, Michigan. Characterization, including gene sequence analysis of the isolate, identified it as belonging to the North American genotype IV of the viral hemorrhagic septicemia virus (VHSV, Genus *Novirhabdovirus*, family *Rhabdoviridae*). The LSC isolate was, however, amply unique from other VHSV-genotype IV isolates and, therefore, was considered a separate sublineage (designated VHSV-genotype IVb). During 2005–2009, VHSV-IVb was isolated from twenty-eight fish species residing in all of the five Great Lakes and a number of inland water bodies in Michigan (two), New York (four), Ohio (one), and Wisconsin (one). The isolation was sometimes associated with outbreaks of wild fish kills (Gagné et al. 2007; Groocock et al. 2007; Lumsden et al. 2007; and...
FIG. 16. Histologic sections from juvenile Muskellunge (Esox masquinongy) infected with viral hemorrhagic septicemia virus-genotype IVb showing: (A) hemorrhaging between the dermis and muscle (asterisk) and infiltration of erythrocytes into the muscle (arrows) (scale bar = 100 µm), (B) intramuscular hemorrhage between muscle bundles and fibers (scale bar = 100 µm), and (C) severe hematopoietic necrosis and hemorrhaging into the interstitium (scale bar = 50 µm).
MSU-AAHL unpublished data). Infected fish often exhibited exophthalmia, dermal petechial hemorrhages, darkened body coloration, severe gill pallor, and death (fig. 15a-c). Histopathologic alterations were often dramatic and appeared in the form of hemorrhaging in the muscle and skin and widespread necrosis throughout the liver and kidneys (fig. 16a–c). Interspecies variability in susceptibility to VHSV-IVb has been demonstrated with Muskellunge, Lake Herring (*Coregonus artedi*), and Largemouth Bass being the most susceptible, whereas salmonids of the *Oncorhynchus* spp. are the least (Kim and Faisal 2010). The wide host range of VHSV has been demonstrated in its expansion throughout the Great Lakes (fig. 17).

More recently, VHSV-IVb was isolated from non-teleost hosts, such as *Diporeia* sp., a deep water amphipod, collected in Lakes Huron and Michigan (Faisal and Winters 2011), and from the gut of leeches (*Myzobdella lugubris*) from LSC (Faisal and Schulz 2009). Sequence analysis of the isolates revealed they were closely related to VHSV genotype IVb. Although these isolations prove that macroinvertebrates may play a role in transmitting VHSV, future studies are necessitated to fully understand the involvement of these hosts and whether successful transmission could be duplicated in a laboratory setting.

The emergence of a reportable viral infection in the Great Lakes raised major concerns among federal and state agencies. In response to OIE requirements for reportable diseases, such as VHSV, and to slow the spread of this virus to other parts of the United States, the U.S. Department of Agriculture—Animal and Plant Health Inspection Service issued an emergency order in 2006 that will soon be followed up by a
formal rule to limit the movement of fish, unless certified disease-free, from the Great Lakes to other parts of the United States. To slow the spread of this pathogen within the Great Lakes, fisheries agencies in the Great Lakes with infected waters have implemented the following measures: (1) regulations that restrict the use and movement of live bait that often used zonation approaches to reduce the inadvertent movement of VHSV; (2) greatly increased testing of broodstock and hatchery fish to ensure fisheries agencies and private hatcheries are not a vector of transmission; (3) greatly increased surveillance to determine the dispersal rate of the virus and the current locations of the virus that directly influences angling regulations; (4) increased biosecurity measures in all areas of fisheries survey work; (5) restricted stocking of cool-water species, until biosecurity issues are fully satisfied; (6) requirement that anglers not be able to move live sportfish overland; and (7) requirement that boaters and anglers empty live wells and binges upon leaving waters. These measures are supported by similar regulations for fish importation and private aquacultural operations promulgated by state agricultural agencies. Given the now endemic nature of this virulent pathogen, it is likely these measures are permanent and will likely spread to other states, as this virus continues to spread in the GLB.

LARGEMOUTH BASS VIRUS
Another emerging viral infection that arrived in the GLB in 2000 is the Largemouth Bass virus (LMBV, genus *Ranavirus*, family *Iridoviridae*). Originally, the first isolation of LMBV in the United States occurred in Lake Weir, Florida, in 1991, but the first mass mortality associated with LMBV was reported from the Santee-Cooper Reservoir of South Carolina (Grizzle et al. 2002), which suggested the virus’s range was limited to the southeastern United States in the 1990s. However, in the summer of 2000, a mass mortality
event involving Largemouth Bass was reported in Lake George, a lake bordering Michigan and Indiana (MDNR-Fisheries Division unpublished data). Diseased Largemouth Bass were collected from this event and LMBV was believed to be the cause of these mortalities (Grizzle and Brunner 2003). Subsequent to this isolation, mortality events were documented in a number of southern Michigan lakes from 2001 to 2003. Sampling in 2001 and 2003 in Michigan inland lakes revealed the presence of LMBV in fifteen of thirty sampled lakes, including Lake St. Clair (fig. 18; Records of MSU-AAHL). Surveys and modeling of the isolations demonstrated that the virus was likely progressing northward, westward, and eastward in southern Michigan. Given the jump of this virus to Michigan from the southern United States, it is suspected that angler transport of infected fish is the likely vector to bring this pathogen to the GLB.

Subsequent to these initial detections in the GLB, LMBV has also been isolated in Illinois, the upper Mississippi River, and Lake Champlain, which borders Vermont, New York, and Quebec, thus, extending the distribution of LMBV to the Midwest and northeastern United States. (Grizzle and Brunner 2003). Since the last sampling efforts of the MDNRE, mass mortality episodes of Largemouth Bass in Michigan have not been reported; however, the typical internal lesions of the virus, such as hemorrhages and yellow crusts in the swimbladders, have often been observed (records of MSU-AAHL). The fate of this virus is unclear, as the epizootic events have not been observed for the last four to five years, but it is a pathogen on the watch list for fisheries agencies.

INFECTIOUS PANCREATIC NECROSIS VIRUS
Infectious pancreatic necrosis virus (IPNV, genus *Aquabirnavirus*, family *Birnaviridae*) is often associated with fry mortality in hatchery settings. The virus has been isolated worldwide from a number of salmonid and non-salmonid fish species, as well as invertebrates, such as crayfish, prawns, and mussels (Bovo et al. 1984; Halder and Ahne 1988; Mortensen 1993; Gregory et al. 2007). Although several isolations occurred throughout the GLB (Beyerle and Hnath 2002), pathogenicity and genotyping of the various isolates have not been completed. In 2003, a study was completed by McAllister, using both non- and endemic IPNV isolates of the Great Lakes and demonstrated the virus caused the highest number of mortality in Brook Trout, when immersion challenged.

In November 2005, IPNV was detected in fish from the Allegheny National Fish Hatchery in Warren, Pennsylvania (USFWS unpublished data). As a result, all Brook and Lake Trout were culled and the hatchery was cleaned and sanitized. Another outbreak of IPNV was discovered in the fall of 2006 in Brook, Brown, and Rainbow Trout at the Conнетquot River Hatchery (NYDEC unpublished data). During the period from 1998 to 2009, repeated detections of IPNV have also occurred from private aquaculture facilities within Michigan and Wisconsin (MDNRE and MSU-AAHL unpublished data).

Surveys of Michigan salmonid streams from 1995 to 2003 documented IPNV from 5 of 115 sites sampled for Brook Trout ($N = 1940$), 3 of 95 sites sampled for Brown Trout ($N = 1753$), and 3 of 113 sites sampled for Rainbow Trout. A total of three watersheds from Lake Michigan and one watershed in Lake Huron have been documented to have IPNV. These data indicate that IPNV is present in Michigan waters, and likely in the GLB, and warrants continued close management to ensure it is not spread to additional waters.

LYMPHOCYSTIS DISEASE
A chronic, mostly non-fatal, disease caused by the lymphocystis virus (family *Iridoviridae*) has been noted in more than two hundred species of fish and most often appears as individual or clustered pink and white
nodules that range from pin to pearl sized on the skin or fin. The disease has been described from a number of fish species in the GLB (Johnson 1971; Amin 1979; Bowser et al. 1988; Smith et al. 1992; Muzzall and Haas 1998). Although the outgrowths arising from the disease are generally benign, a study conducted by Margenau et al. (1988) showed that mortality of Walleye during spawning season may be associated with lymphocystis disease. Although a broadly found viral disease, it is a disease on the fisheries agencies watch list for changes in prevalence or as a factor in mortality events during spawning periods.

**DERMAL SARCOMA VIRUS OF WALLEYE**

Walleye, particularly during the spawning season, exhibit a number of nodular, firm, vascularized, and white to pink in color dermal outgrowths. Some of these lesions have been associated with the dermal sarcoma virus of Walleye (family *Retroviridae*, Walker 1969). The disease was first recognized by Walker (1947) from adult Walleye from Lake Oneida, New York. Subsequent descriptions have been noted in Lakes Erie and Ontario, as well as throughout the Great Lakes region (LaPierre et al. 1998). Signs of this viral infection can often be confused with lymphocystis virus but can be differentiated by histology. Little is known about the pathogenity of this virus, and it is one worth watching by fisheries agencies in the GLB.

**EPIZOOTIC EPITHELIOTROPIC DISEASE VIRUS**

In the early to mid-1980s, the epizootic epitheliotropic disease virus (EEDV, family *Herpesviridae*) was responsible for high rates of mortality in Lake Trout fingerlings reared in a few federal and state fish hatcheries throughout the Great Lakes region. Morbidity associated with the disease has been described as erratic swimming, lethargy, and hyper excitability (Bradley et al. 1989). Clinical manifestations of disease often appear as intraocular hemorrhages and gray to white skin discolorations, whereas internal signs of disease are not described (McAllister and Herman 1989). Histologic changes have been described as hyperplasia of the epidermal cells, with enlarged nuclei and swollen cytoplasm (Bradley et al. 1989; McAllister and Herman 1989). Although the etiology of the disease was initially suspected to be gram-negative bacteria, studies by Bradley et al. (1989) and McAllister and Herman (1989) revealed the etiologic agent was a herpesvirus. Although it could not be isolated in vitro, experimental infections revealed the virus was pathogenic to Lake Trout by waterborne immersion challenge (Bradley et al. 1989). These findings led to depopulation and disinfection of fish at several state and federal hatcheries in Michigan and Wisconsin and resulted in the destruction of over fifteen million Lake Trout and salmon (Bradley et al. 1989; McAllister and Herman 1989; Kurobe et al. 2009). Additionally, movement and transport of fish from facilities in which EEDV was diagnosed was restricted. After two years, in which the fish from disinfected hatcheries showed no signs of EEDV infection even after stress, stocking of fish into waters without a known history of EEDV was allowed (Hnath 1993).

The persistence of EEDV in the Great Lakes has not been determined, in part because of the lack of a specific diagnostic tool (McAllister and Herman 1989). Kurobe et al. (2009) described an improved diagnostic method, which involved polymerase chain reaction, whereby specific regions of the herpesvirus genome can be amplified from extracts obtained from skin tissues. Efforts are currently ongoing to determine the distribution of this virus in the GLB and in other areas of the United States, as well as whether it is an endemic herpesvirus that causes mortality events during stress episodes.
Parasitic Diseases

As with all natural aquatic systems, freshwater fish in the Laurentian Great Lakes are host to a multitude of parasites. A few can cause serious problems that concern fisheries agencies. Other parasites are common and often occur in high abundance, yet appear to coexist with their hosts in a rather innocuous fashion; however, the implications of most on natural mortality rates are unknown at this time. A comprehensive list of parasites that infect Great Lakes fish can be found in Nepszy (1988), Hoffman (1999), and Muzzall and Whelan (In press). This section provides examples of parasitic diseases that are well known to cause harm to Great Lakes fishes.

PROTOZOA

Protozoa, such as *Ichthyophthirius multifiliis*, *Trichodina* spp., *Chilodonella* spp., and *Costia* spp., are often found on wild and propagated salmonid, centrarchids, and cyprinid fish species throughout the GLB. Propagated Brook Trout suffering from subclinical or chronic BKD often die from heavy infections with sessile protozoa (*Epistylis* spp. and *Apisoma* spp.). Many genera of myxozoans and microsporidia are found on feral fishes, sometimes causing unsightly external or bulging internal cysts. Among the more common myxosporidia are the genera *Myxobolus* and *Thelohanelus*, which produce external cysts on various species of *Cyprinidae* and *Percidae*.

MYXOBOLUS CEREBRALIS

The most notorious myxosporidian parasite is *Myxobolus cerebralis*, the causative agent of whirling disease (WD) of trout. The disease was first detected in Michigan in August 1966, among Rainbow Trout at three commercial hatcheries, all of which were components of the same business. Fish in these facilities were imported from the same infected commercial hatchery in Ohio. Despite strict quarantine measures imposed on the three farms and all state and federal hatcheries in Michigan, the disease spread to other sites (Yoder 1972). Through voluntary agreements, affected fish in all 159 secondary farms were destroyed. Those from which *M. cerebralis* were detected were disinfected with chlorine at 1600 mgL\(^{-1}\). In 1970, fish were found infected within a six mile length of the north branch of the Tobacco River that received effluent from one of the primary WD facilities. This river was disinfected several times from 1970 to 1975, using alternating rotenone and chlorine treatments with chlorine concentrations in excess of 1100 mgL\(^{-1}\) for a minimum of one hour. Despite this intensive management, *M. cerebralis* can still be found in this system today. Unrelated to this event, *M. cerebralis* was also detected in fish in the Sturgeon River, two hundred miles from the initial site of infection. The source of this infection is also speculated to be the stocking of infected fish from private hatcheries. In 1975, *M. cerebralis* was identified in Rainbow Trout collected from the MDNRE Sturgeon River Rearing Station and all of the fish at this station (1.5 million Coho Salmon and 600,000 Rainbow Trout) were eventually destroyed. In 1994, *M. cerebralis* was detected in several state fish hatcheries in New York, which led to the destruction of 570,000 Rainbow Trout (Hulbert 1996).

Despite these control efforts in Michigan, the parasite has spread to rivers, such as the Manistee and Au Sable Rivers. Sampling from 1995 to 2003 in Michigan trout waters found *M. cerebralis* in 12 of 158 sampling sites in six of fifty-two watersheds for Brook Trout (\(N = 2,579\)), 13 of 139 sampling sites in three of fifty-two watersheds for Brown Trout (\(N = 2,710\)), and 13 of 158 sampling sites in seven of fifty-two watersheds for Rainbow Trout (\(N = 2,217\); MDNRE Fisheries Division unpublished data). Despite the occasional presence
of *M. cerebralis* spores in fish tissue digests from approximately 10 percent of the rivers and sites sampled in Michigan (MDNRE Fisheries Division unpublished data), the clinical form of whirling disease has never been observed in the wild and the disease seems to be running a subclinical course, as the spore numbers and prevalence observed are generally low.

*M. cerebralis* can be found throughout a number of the Great Lakes states, including Michigan, New York, Pennsylvania, and Ohio, as well as several other states in the northeastern United States (Crosier et al. 2003; Steinbach-Elwell et al. 2009). Currently, this parasite is relatively rare in trout streams in the GLB but, once established, is impossible to eliminate, thus, the fisheries agencies in the GLB are using containment strategies to prevent the establishment of this parasite in waters with susceptible species. A key component of this containment strategy is the prohibition of the stocking or importation of *M. cerebralis*-infected fish in the GLB, which is likely to continue into the future.

**HETEROSPORIS SP.**

Organisms in the genus *Heterosporis* belong to a unique phylum, known as Microsporidia. Microsporidians were traditionally classified belonging to the kingdom Protista, although recent studies have shown microsporidians are much more similar to the kingdom Fungi (Lee et al. 2010). Among microsporidians, *Heterosporis* spp. (phylum Microsporida: family Pleistophoridae) are intracellular parasites of skeletal muscle cells. Fillets of infected fish exhibit opaque areas in the dorsal and lateral musculature and around the anus. Affected muscles are stuffed with mature sporophorous vesicles containing either eight or sixteen spores.

Between 2000 and 2002, *Heterosporis* spp. infections emerged in feral populations of Yellow Perch in three geographically separated regions of North America: northeastern Lake Ontario and the Bay of Quinte (5 percent), Eagle Lake in Vilas County, Wisconsin (more than 50 percent), and several inland lakes in central Minnesota (prevalence unavailable; MN DNR, WI DNR and MSU-AAHL unpublished data). It is currently unclear whether *Heterosporis* spp. were present in North America before these reports, albeit at much lower prevalence, but this is unlikely given the readily detectable symptoms. Although *Heterosporis* is not always fatal, the damage done to the fillet often prevents the fish from being commercially valuable. *Heterosporis* spp. have also been found in other species, including Walleye, Burbot, and Northern Pike, but at much lower prevalences than observed in the Yellow Perch. Given the loss of economic value of key sport and commercial fish, fisheries agencies in the Great Lakes are implementing containment measures, including importation certifications for this parasite.

**GLUGEA SPP.**

Another group of microsporidians that cause numerous large internal cysts in a number of fish species is *Glugea* spp. *Glugea hertwigi* has infected Rainbow Smelt (*Osmerus mordax*) in Lakes Erie and Ontario (Chen and Power 1972) with a prevalence attaining 90 percent. This parasite has reduced fecundity and caused mass mortalities among smelt populations (Nepszy et al. 1978). Additionally, an epizootic of *G. anomala* erupted in a Three-Spine Stickleback population in Michigan’s Upper Peninsula (fig. 19) in which a massive infection completely occluded the intestinal tract (Records of MSU-AAHL). A 2009 survey of Mottled Sculpin from Seven Mile Creek in Battle Creek, Michigan, revealed numerous cysts in the peritoneal cavity of several fish (Records of MSU-AAHL). Phylogenetic testing determined these cysts are a new, previously undiscovered *Glugea* species. Additional research is needed to determine the effects of
FIG. 19. Three-spined Stickleback (Gasterosteus aculeatus) showing a large cyst of Glugea anomala. The fish was caught from a stocking pond in Michigan’s Upper Peninsula.

these protozoa at the population level, and, given the potential population implications of this parasite, future management actions to contain this parasite are likely.

MONOGENEANS
There are many species of monogeneans that parasitize gills and skin of Great Lakes fish; however, the species that cause direct harm to their hosts are relatively rare in this parasite group. Gyrodactylus salmonis is known to heavily parasitize Rainbow Trout and often causes skin lesions associated with massive mortalities in hatchery fingerlings (Cone and Odense 1984; Cusack and Cone 1986). Monogeneans have caused problems in nearly every hatchery with open water supplies in the basin. In most instances, their presence was associates with reduced growth and survival. Recent data indicates a relationship may exist between BKD and monogenean-associated mortalities in Brook Trout (Records of MSU-AAHL), and this may be the case for other pathogens and stress-causing parasites in this group.

TREMATODES
Freshwater fish serve as final and intermediate hosts for numerous species of trematodes, which use snails and fish eating birds or mammals as hosts to complete their life cycles. Most common among these are the metacercarial parasites of the yellow grub (Clinostomum complanatum) and black spot (Uvulifer ambloplites and Crassiphiala bulboglossa). Clinostomum complanatum is very common in Michigan and has been found in Round Gobies (Neogobius melanostomus) from the Saginaw and St. Clair Rivers and LSC (Szalai and Dick 1988; Pronin et al. 1997; Kvach and Stepieen 2008), as well as throughout North America.
Yellow grubs are particularly common in inland lakes that have the appropriate hosts, in particular large snail and great blue heron populations. Black spot metacercaria are easily discerned by their obvious black cysts (the size of a pinhead) that are slightly raised from the skin or fins and sometimes found in the mouth or flesh. The parasites commonly infest a range of cool- and warm-water fish, including Rock Bass (Ambloplites rupestris) and other sunfish, Largemouth Bass, minnows, and numerous other fishes (Steedman 1991). Various species of black spot are found in practically all parts of the world and are common throughout the inland waters of the Great Lakes region (Hoffman 1999; Records of MSU-AAHL), as well as the Great Lakes themselves (Dechtiar 1972; Nepsy 1988).

There are also many species of non-pigmented metacercarial parasites (Neascus spp.) in the region, notably Posthodiplostomum minimum and Ornithodiplostomum ptychocheilus. Metacercarial parasites start as free-living eggs in the water and are eaten by an intermediate host, such as a snail or mollusk (Hoffman 1999). The snails or mollusks are then eaten by a fish (the second intermediate host), and the metacercariae are passed on to the final host, which can be either fish or mammals, but is most commonly fish-eating birds (Hoffman 1999). Another of these, Metorchis conjunctus (Distoma conjunctum), can infect humans, although no human cases have been reported from Michigan. Additionally, these parasites have been found in several fish species, such as Bluegill, Arctic Grayling (Thymallus arcticus), and Longnose Dace (Rhinichthys cataractae) in various inland Michigan lakes and rivers, as well as Lake Michigan (Radabaugh 1980; Muzzall 1990; Muzzall et al. 1992; Wilson et al. 1996). Larval O. ptychocheilus are also known to encyst in the brain tissue of host fish and can cause abnormal behavior (Radabaugh 1980). Although the erratic behavior often enables the affected hosts to be more easily preyed upon, no large-scale mortalities have occurred because of these metacercarial parasites, and, although of a concern to anglers who wish to consume infected fish, they are of a low concern from a population perspective to GLB fisheries agencies.

**Cestodes**

Cestodes are widespread in wildfish stocks in the GLB, where fish can be intermediate or final hosts. Most of these infections are harmless; however, pathological lesions have been reported from some cestode infections.

**Trienurophorus spp.**

Intermediate stage cysts of the *Trienurophorus crassus* have been observed in the flesh of Cisco and Lake Whitefish in Michigan and Canada (Bangham 1955; Boily and Curtis 1992). The parasite has had significant economic effects in Canada, where heavy infestations of the larval worms in the flesh of Cisco and Lake Whitefish have reduced the commercial value of infected fish (Boily and Curtis 1992). In addition, the intermediate cysts of *T. nodulosus* have been observed in the liver and, to a lesser extent, in other internal organs of Yellow Perch in Michigan. The cysts can occupy a major portion of the liver of affected fish. As a result of the known economic effects of this group of parasites, this group of parasites is one that GLB fisheries agencies need to monitor for and potentially consider control measures to prevent large outbreaks.

**Proteocephalus ambloplitis**

The adult bass tapeworm *Proteocephalus ambloplitis* is found in the intestines of Largemouth and Small-mouth Bass and Bowfin (*Amia calva*; Amin 1989; Hoffman 1999). The plerocercoid larvae, however, are
found in the body cavity and internal organs of many species of fish, especially Rock Bass and Largemouth and Smallmouth Bass, in many lakes and streams throughout the United States and Canada (Sparks 1951; Hoffman 1999; Kvach and Stepien 2008). The plerocercoids develop in the body cavity and internal organs, especially the liver and ovaries. Because they do not encyst and continue to move around, they destroy tissue and cause multiple hemorrhages, which produce a brownish color, and multiple adhesions in the body cavity. Heavy infestations of the gonads may sterilize the fish (Sparks 1951; Hoffman 1999; Gillilland and Muzzall 2004). Mortalities of intensively cultured Muskellunge fingerlings have been caused by heavy intestinal parasitism of the adult tapeworms *P. ambloplites* and *Triaenophorus* sp. (Schachte and Hoffman 1986). Although large-scale mortalities in the wild have not been associated with *P. ambloplites*, heavy infestations and high prevalences of the cestode in wild populations are common (Gillilland and Muzzall 2004). Currently, GLB fisheries agencies are not consistently monitoring for this parasite because of funding constraints, but should be to ensure significant resource losses are not occurring.

**Nematodes**

Nematodes are also widespread, particularly among wildfish stocks in the GLB. Except for a few examples, the effects of nematodes on their fish hosts are generally unknown, although it is likely their population effects are negligible except for rare cases.

**Eustongylides spp. and Philometra spp.**

These parasites are commonly seen as red roundworms in the flesh and/or body cavity of fish in the GLB. *Eustongylides* is abundant in Yellow Perch of Lake Huron (particularly Saginaw Bay) and western Lake Erie but has also been found in Round Gobies and Yellow Perch in LSC (Crites 1979; Thomas and Haas 2004; Fielder and Thomas 2006; Kvach and Stepien 2008). As high as 86 percent of fish examined from areas of Lake Huron have been found infected, with as many as seventy-eight worms per fish (Allison 1966), although it is usually found in lesser abundance. These parasites usually cause little harm to the fish; however, mortalities often occur in affected birds, particularly hatchlings and nestlings of herons and egrets (Cole 1999). These nematodes are zoonotic, especially when infected raw fish are ingested (Wittner et al. 1989). *Philometra* sp. is common in the body cavity or tissues of various fish, including Lake Whitefish, Muskellunge, Brook Trout, Yellow Perch, Black Bass, Rock Bass, White Sucker, and Walleye, and has been documented in several of the Great Lakes, as well as in other lakes and streams of Michigan, Ohio, Wisconsin, and Canada (Hare 1943; Fantham and Porter 1947; Fischthal 1947; Fischthal 1950; Bangham 1955; Dechtiar and Nepsky 1988; Dechtiar et al. 1988; Thomas and Haas 2004; Fielder and Thomas 2006). Although it is not of population concerns to GLB fisheries agencies, effort is usually needed to educate anglers on their potential consumption effects of infected fish.

**Cystidicola spp.**

*Cystidicola* spp. are common parasites of trouts, salmons, Rainbow Smelt, Cisco, and Lake Whitefish. As an adult, the worm is found in the swim bladder of these fish and causes thickening and irritation of the swim bladder membrane (Willers et al. 1991; Faisal et al. 2010a). Anthony (1963) found more than two hundred worms per fish, and Warren (1952) found an infection rate of 72 percent in Lake Superior Cisco. A recent study of 1,281 Lake Whitefish in northern Lakes Huron and Michigan revealed the presence of *C. farionis* in the swimbladder of affected fish, with a prevalence of 27 percent (Faisal et al. 2010a).
farionis had a higher prevalence in fish from Lake Huron (41.44–50 percent) than from Lake Michigan (2.24–14.68 percent; Faisal et al. 2010a). Additionally, the walls of the swimbladders in Lake Whitefish with C. farionis were thickened, and the mucosal lining was deteriorated (Faisal et al. 2010a). The effect of these worms on their hosts is unknown, and these worms are a parasite that GLB fisheries agencies should be routinely monitoring.

ACANTHOCEPHALANS
Acanthocephalans are easily recognizable because of the proboscis used for attachment and the shortened body, and they commonly attach to the intestinal wall of fishes. They are widely dispersed throughout North America, including the GLB and Canada, and infect a variety of hosts (Hoffman 1999). Eggs of acanthocephalans are ingested by a crustacean (typically a copepod, amphipod, etc.), in whom the eggs develop into a larval stage (Hoffman 1999). The crustacean and, in turn, the larval stage parasite are then consumed by a fish, in whom the adult parasite then attaches itself to the intestinal wall (Hoffman 1999).

One of the most common acanthocephalans present in the GLB is Echinorhynchus salmonis. Salmons, trouts, Cisco, Lake Whitefish, and Rainbow Smelt in the Great Lakes are commonly infected with E. salmonis, as well as Lake Sturgeon (Acipenser fulvescens), Rock Bass, Black Bass, sunfishes, and Yellow Perch (Bangham 1955; Amin and Burrows 1977; Amin 1985; Muzzall and Peebles 1986; Muzzall 1995). Additionally, Applegate (1950) found E. salmonis in the Sea Lamprey. In high numbers, these worms cause hemorrhagic enteritis at the site of attachment, affect intestinal functions, and often penetrate the intestinal wall, creating portals for secondary bacterial infections (Bullock 1963). Interestingly, high intensities (as many as 785 parasites per fish) of E. salmonis were found in the same Chinook Salmon from the late 1980s that were part of the BKD epizootic (Holey et al. 1998). The overall population effects of this parasite group are not known, and additional information is needed in this area. Given some of the potential interactions between this parasite group and other pathogens, the routine monitoring of their prevalence and abundance should be part of the fish health plans of GLB fisheries agencies.

LEECHES
Leeches are also known to parasitize many fish species in the GLB. Multiple reports by Klemm (1972, 1977, 1982, 1991) identified more than forty-five leech species throughout the Great Lakes region, with the highest number of leech species recorded in Michigan. Because of their invasive method of attachment, leeches can cause extensive damage to their fish host, including erosion of the epithelium, thickening of the dermis, hyperplasia, focal and widespread hemmorhages, and edema (Paperna and Zwerner 1974; Appy and Cone 1982; Roubal 1986; Volonterio et al. 2004). In addition to the damage to the underlying musculature, they are also known as vectors for several pathogens and have been found associated with bacteria, trypanosome blood parasites, and viruses, including VHSV (Burreson 1982; Ahne 1985; Hemmingsen et al. 2005; Kikuchi and Fukatsu 2005; Faisal and Schultz 2009). The feeding and attachment behaviors of leeches have been shown to cause emaciation, anemia, melanosis, frayed fins, and abnormal swimming behavior and have the potential to allow entry for secondary invaders (Meyer 1946; Kua et al. 2010). Additionally, Wolf et al. (2008) found extremely large specimens of A. pediculata attached to Freshwater Drum (Aplodinotus grunniens) in Lake Ontario and noted that the leeches could prevent closing of the operculum. Infestations such as this may reduce the efficiency of the respiratory system, which can affect the health of the fish (Wolf et al. 2008).
A recent study determined there are three prominent leech species, *Actinobdella pediculata* Hemingway 1908 (Rhynchobdellida: Glossiphonida); *Myzobdella lugubris* Leidy 1851 (Rhynchobdellida: Piscicolida); and *Placobdella montifera* Moore 1906 (Rhynchobdellida: Glossiphonida), parasitizing fish hosts in LSC, Michigan, which connects Lake Erie to Lake Huron (Schulz et al. 2011). *Myzobdella lugubris* is an intermittent feeder and has a very wide host range in freshwater and marine waters throughout North America and was the most commonly occurring leech in LSC (92 percent prevalence; Hoffman 1999; Schulz et al. 2011). Additionally, the intensity of *M. lugubris* attached to a host varied from one leech up to seventy-nine leeches (fig. 20). Moreover, VHSV was detected from within pooled leech samples of *M. lugubris* from LSC, with a prevalence of 73 percent (Faisal and Schulz 2009). This is the first record of VHSV detected from within a leech, specifically *M. lugubris*, and suggests the potential of *M. lugubris* involved in VHSV transmission. Further studies are required to determine if *M. lugubris* could be a vector for the virus. The potential of leeches to be direct mortality factors and indirect carriers for other pathogens makes it important for GLB fisheries agencies to have surveillance systems in place to examine infection trends and for research conducted to understand the interactions between this group of parasites and other pathogens to see if any interventions are needed to control disease outbreaks.
COPEPODS
Several parasitic copepods are common to the GLB, including *Lernaeu* spp., *Ergasilus* spp., *Argulus* spp., and *Salmincola* spp. The skin of several species of fish, especially Lake Trout, Brook Trout, and coregonids, is often infested with *Salmincola siscowett* (Anderson 1993; Hudson et al. 1994), and *Argulus* spp. have been found in Lake Sturgeon from the St. Marys River (MDNR Fisheries Division unpublished data). Brook Trout from some Michigan waters are often infested with *Salmincola edwardsii*, which is particularly common in northern Michigan streams that sustain populations of Brook Trout. Studies of *S. edwardsii* on Brook Trout have indicated that rather heavy infestations, up to 125 parasites per fish, do not appear to affect growth (Allison and Latta 1969). However, mortalities have occurred among hatchery Brook Trout (5–7 cm long), when the fish are first attacked by these parasites, and extensive bleeding has been documented from the gills (Allison and Latta 1969). *Salmincola edwardsii* is found throughout the GLB, as well as the Pacific Northwest, Canada, and Russia. Although a common group of parasites frequently found during fish health surveys, they do not appear to be significant causes of or vectors for disease at this time and are of low concern to GLB fisheries agencies.

POLYPodium HYDRIFORMe
*Polypodium hydriforme* is the only coelenterate parasitic in freshwater fishes in North America. This parasite was believed to exist only in rivers of the former Soviet Union, until 1974, when it was found in Lake Sturgeon from the Black River, in northern Michigan (Hoffman et al. 1974). Since then, it has been found in feral Paddlefish (*Polyodon spathula*) in Missouri and in several other locations around North America (Hoffman 1999). Although the parasite feeds on and effectively kills individual parasitized eggs, there have been no studies done on Great Lakes Lake Sturgeon to determine what effect the parasite has on natural reproduction. Although there is little information regarding *P. hydriforme* in the GLB, it is an economically significant parasite in Russia, an area that accounts for approximately 90 percent of the world production of caviar harvested from several species of sturgeon (Raikova 2002). Given the potential economic implications for a species of concern in the GLB, GLB fisheries agencies need to obtain additional information on the ecology of this parasite to determine if it should be considered a pathogen of concern.

Fungal Diseases
Fungal diseases are widespread in the GLB. *Saprolegnia* spp. are particularly problematic at cold temperatures and in sexually mature fish prior to spawning. *Phoma herbarum* has also been isolated from propagated Chinook Salmon fingerlings, causing significant mortalities during an eight week period, ranging from 500 to 3,400 mortalities per week (Faisal et al. 2007b). The fungus localizes in the swim bladder and often infiltrates the body cavity and other visceral organs (Faisal et al. 2007b). In released Muskellunge, *Branchomyces* spp. can cause reduced growth and survival. These pathogens are key disease factors in GLB fish hatcheries and control is focused on that area by GLB fisheries agencies, as little information has been documented on any specific threats to feral or wild fish populations in the Great Lakes.
Noninfectious Diseases

In the early 1990s, serious problems arose in Great Lakes salmon hatcheries, using eggs from feral Lake Michigan salmon stocks. Losses of pre-feeding fry reached levels of nearly 100 percent in progeny of some female fish, and general losses averaged more than 50 percent (Honeyfield et al. 1998). This was named Early Mortality Syndrome (EMS; now known as Thiamine Deficiency Complex [TDC]), because, initially, no pathogens were found, nor was the cause determined. The syndrome was characterized by loss of equilibrium, hyper-excitability, lethargy, anorexia, and high mortality rates. Similar clinical signs were seen in Atlantic Salmon fry from the Baltic Sea, where the condition was labeled M-74 Syndrome (Bengtsson et al. 1999). Fisher et al. (1995) also noted similar clinical signs and mortality in Atlantic Salmon in the Finger Lakes of New York and determined that application of thiamine (vitamin B1) reversed the symptoms and cured the affected fish. Thiamine applications were tried on affected eggs and fry from Lake Michigan in 1996, which also resulted in the reversal of clinical signs and increased fry survival (MDNRE Fisheries Division unpublished data). The use of thiamine baths on affected fry is now a routine practice in the GLB and has mitigated the initially low thiamine levels in eggs, allowing for the production of apparently healthy fish for stocking.

Various contaminants were considered possible causes of EMS, but none have yet been linked to EMS (Honeyfield et al. 1998). Fitzsimons et al. (1995) did not rule out the possibility that contaminant interactions with other factors might contribute to mortalities. In the late 1990s, it was soon determined that experimentally-induced thiamine deficiency alone produced clinical EMS in captive Lake Trout (Honeyfield et al. 2005). A linkage was determined between low concentrations of thiamine in the egg with subsequent EMS in progeny. Although research has not yet identified a specific cause for EMS, in virtually all instances in which the problem occurs naturally, there is a link with thiaminase containing prey species (Alewives and Rainbow Smelt in the Great Lakes and Finger Lakes, and herrings and sprat in the Baltic Sea). Thus, a high consumption rate of Alewives and Rainbow Smelt by predators in the GLB appears to lead to low levels of thiamine in salmonid eggs, and this effect varies by species. There is also a correlation in the Great Lakes between increased EMS and declines of the important benthic macro-invertebrate Diporeia (Fitzsimons et al. 1999). Diporeia declines are associated with the invasion of and huge population growths of Zebra and Quagga Mussels, which have substantially reduced phytoplankton biomass (Mills et al. 2003) and, ultimately, affected the nutritional base of Alewives, perhaps resulting in increased production of thiaminase.

Although the problem is currently under control in fish hatcheries through the routine use of thiamine baths, there remains serious concern about the long-term effects of EMS on natural reproduction and development of self-sustaining populations of Great Lakes stocks of salmonids, especially Lake Trout, Coho Salmon, Chinook Salmon, Steelhead (Rainbow Trout), Atlantic Salmon, and Brown Trout. Thiamine deficiency is still a widespread issue of concern to all of the GLB fisheries agencies, and all have put into place mitigation measures to control this disease, now and likely well into the future.

In addition to EMS, several other non-infectious pathologies and tumors have been described from a number of Great Lakes fishes (Black 2000). Baumann (1984) diagnosed thyroid hyperplasia, gonadal neoplasm, papillomas, skin tumors, and hepatocellular carcinoma from a number of fish species, including Coho Salmon, Common Carp, Freshwater Drum, Walleye, and Brown Bullhead (Ameiurus nebulosus), in all of the Great Lakes, as well as several rivers and an inland lake (Baumann 1984). Baumann (1984)
attributed the high frequency of tumors to the highly industrialized nature of the GLB and proposed tumor incidence as a sensitive indicator of the level of carcinogenic compounds in the environment. The tumors listed above are not known to have negative large-scale effects on infected fish; although, there was a high occurrence of gonadal tumors in several of the fish species, which may inhibit the reproductive capabilities (Baumann 1984). The concerns with tumors on fish populations are not clear, but public concerns with visible tumors remain high, so GLB fisheries agencies need to continue to develop information to understand how they form, what population effects they have, and what, if any, these mean as environmental monitors of general Great Lakes health.

Disease Control Management Strategies in the Great Lakes Basin

In 1973, fisheries agencies in the United States and Canada formed the Great Lakes Fish Health Committee (GLFHC) under the Great Lakes Fishery Commission with the mandate to coordinate regional efforts in the GLB, aiming at preventing the introduction and dissemination of communicable fish diseases by (1) recommending and fostering research and studies related to fish health and disease control, (2) recommending and coordinating measures within the GLB that minimize risk of introduction and dissemination of communicable fish diseases, and (3) preparing for publication of scientific and other information related to fish health protection. The Committee consists of two representatives appointed by each fisheries management agency that is a signatory to the Joint Strategic Plan for the Great Lakes. Currently, the GLFHC has agency membership from the eight Great Lakes states, the Province of Ontario, the two federal governments (U.S. Fish and Wildlife Service and the Canadian Department of Fisheries and Oceans), and the United States Native American tribes, represented by the Great Lakes Fish and Wildlife Commission and the Chippewa-Ottawa Resource Authority.

The GLFHC develops and recommends fish health policies and protocols to the Council of Lake Committee, which have been generally adopted and have reduced the risk of introducing or transferring serious disease agents into or within the GLB. One such policy and procedure document is the Great Lakes Fish Disease Control Policy and Model Program (Model Program; Hnath 1993). The Model Program calls on member agencies to classify salmonid fish hatcheries, based on disease history of all lots of fish on the station, so all know the status of a hatchery prior to moving fish from hatchery to hatchery and the general status of each hatchery system is known to all GLFHC members. The Model Program established infectious disease categories, based on their presence (restricted) or absence (emergency) in the GLB. The list of emergency diseases includes viral hemorrhagic septicemia, infectious hematopoietic necrosis, Ceratomyxosis, and proliferative kidney disease. The list of restricted diseases includes whirling disease, infectious pancreatic necrosis, bacterial kidney disease, furunculosis, enteric redmouth, and epizootic epitheliotropic disease. The Model Program encourages each fisheries agency to control fish diseases in the GLB, by developing legislative authority and regulations to (1) control and, where possible, eradicate fish diseases; (2) prevent the release of seriously infected fish; (3) discourage the rearing and stocking of diseased fish; (4) prevent the importation into the GLB of fish infected with emergency diseases; and (5) prevent the transfer within the GLB of fish infected with restricted diseases. At the time of this publication, the Model Program is undergoing a substantial revision and will likely include both cold- and cool-water fish diseases, along with a companion risk assessment document.

As a result of GLFHC actions, a fish health inspection program has been adopted basin-wide for all
member hatcheries and egg-take weirs. The implemented fish health inspection program complies with recommendations and requirements in the Fish Health Section of the American Fishery Society Blue Book and the Canadian Fish Health Protection Regulations Manual of Compliance. This program sets guidelines and procedures for the inspection of salmonid fish stocks prior to their release into waters of the Great Lakes. The committee has increased the awareness of the importance of fish health in both wild and cultured fish through the development of educational publications, such as “A Guide to Integrated Fish Health Management in the Great Lakes Basin” (Meyer et al. 1983), “Gross Signs of Tumors in Great Lakes Fish: A Manual for Field Biologists” (Black 2000), and the “Protocol to Minimize the Risk of Introducing Emergency Disease Agents with Importation of Salmonid Fishes from Enzootic Areas” (Horner and Eshenroder 1993).

Finally, the GLFHC provides a key communications forum for fish health issues in the GLB and has an association group of technical experts that ensures the best possible information is available to all member agencies. A recent example of the effectiveness of this forum was the coordinated response of all of the member agencies to the discovery of VHSV, which has, in part, slowed the spread of this pathogen and likely reduced mortalities basin-wide.

**Fish Diseases and Management**

The GLB has experience significant epizootic events that clearly had population efforts that directly impaired the ability of fisheries manager to protect the public trust resources in the basin. The mortalities that occurred in Lake Michigan Chinook Salmon in the late 1980s and early 1990s in association with *R. salmoninarum* infections definitively demonstrate the population level effects that infections can have on Great Lakes fish populations. This event also demonstrated how a successful, well-implemented, and coordinated fisheries management plan was able to remediate the effects of a *R. salmoninarum* infection. Other similar examples, such as the New England Atlantic Salmon Restoration Program and its battle with *A. salmonicida* infections (Barbash et al. 1991; Cipriano et al. 1996b; Cipriano 1997) also serve as reminders that some fish pathogens can severely impair conservation and restoration efforts.

Management strategies for combating the negative effects of these infections are of the utmost importance but really need to be implemented basin-wide for multi-jurisdictional waters to be effective. For example, the broad implementation of screening broodstock and gametes for those bacterial pathogens known to be transmitted vertically (i.e., *R. salmoninarum* and *F. psychrophilum*) before being utilized in hatchery conservation programs has greatly reduced the prevalence of infection in the resultant progeny and reduced their likelihood of being immediate pathogen vectors on release. Moreover, egg disinfection procedures and antibiotic treatments are another way to reduce and/or negate some effects of certain bacterial infections of hatchery-produced fish, again removing additional vectors from the environment. In the case of bacterial involvement in hatchery mortality events, antibiotic susceptibility testing must be conducted on the recovered isolates to ensure the most efficacious treatment and reduce the likelihood for antibiotic resistance. Additionally, enhanced biosecurity measures, such as foot baths, tool disinfections, and exclusion of wildlife through barriers, can also drastically reduce the chance of disease transmission. All of these, in turn, can result in both a higher likelihood of survival and a lower prevalence of disease in those fish destined to be stocked into the Great Lakes and its tributaries, thus, potentially translating into a reduced risk of disease transmission to valuable feral and wild fish stocks.
Summary

Fish residing in the GLB exhibit a number of diseases, although assessing their effect at the population level has received little attention. As a consequence, the understanding of disease dynamics, spread, and proper control measures is not currently sufficient. It is important to fill this gap to develop effective, regional fish disease control strategies and to begin to determine how fisheries managers can change natural mortality rates. This is also true for the physiological diseases, and continued research is needed to understand and minimize the effects of such diseases as thiamine deficiency syndrome in important Great Lakes fish stocks.

As previously stated, many new diseases and pathogens have recently emerged. Thus, it is critical to expedite the development of sensitive assays for their detection (preferably non-lethal) along with the development of effective means to control or minimize their impacts. Additionally, there is still insufficient information about the geographic ranges of many important enzootic fish pathogens or reservoirs of infection for these pathogens, although some recent work synthesizing the literature on parasites will help fill a few of the gaps. Multidisciplinary research efforts are needed to determine the relationship between energy pathways and disease resistance, to determine and evaluate effects of anthropogenic stressors on the health of Great Lakes fish populations, and, most importantly, to accurately estimate the contribution of disease to natural mortality rates in feral fish populations. Finally, it is critical that the fish disease control and fishery management practices be reviewed and evaluated on a fixed time interval to incorporate the latest information into practice.

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