

# **Understanding the relationships between fish health and ecosystem dysfunction in the Great Lakes**

## **Research Theme Paper**

### **Fishery Research Program**

#### **Great Lakes Fishery Commission**

Stephen C. Riley<sup>1</sup>, Kelly R. Munkittrick<sup>2</sup>, Allison M. Niggemyer<sup>3</sup>,  
and Charles C. Krueger<sup>3</sup>

<sup>1</sup>U. S. Geological Survey  
Great Lakes Science Center  
1451 Green Road  
Ann Arbor, MI 48105

<sup>2</sup>Canadian Rivers Institute  
University of New Brunswick  
St. John, New Brunswick, Canada

<sup>3</sup>Great Lakes Fishery Commission  
2100 Commonwealth Blvd., Suite 100  
Ann Arbor, MI 48105

## **Executive Summary**

The Great Lakes Fishery Commission's Strategic Vision seeks to achieve sustainable fisheries as part of its Vision Statement on Health Ecosystems. To achieve this objective, the Great Lakes Fishery Commission requires the Board of Technical Experts, through the Fishery Research Program, to initiate research that identifies the degree to which diseases have influenced fish populations. This theme paper provides a conceptual framework for conducting research that addresses the inter-relationships among fish health, fish populations, and ecosystem dysfunction in the Great Lakes. The conceptual framework describes one way in which disease processes and the population-level impacts of disease may relate to ecosystem function. In summary, the framework suggests that functioning ecosystems are more likely to be resilient with respect to disease events than dysfunctional ecosystems. Two overarching research questions structure this theme: 1) what is the relationship between ecosystem function and fish health (how is fish health related to ecosystem dysfunction or its symptoms?); and how can we improve prediction of population-level effects of diseases on wild fish populations? Research conducted under this theme will either support or refute the conceptual framework. Examples of how the framework can be used to generate research questions are provided using three disease models of interest in the Great Lakes; thiamine deficiency complex, botulism, and bacterial kidney disease. At the conclusion of this theme, the results from research projects addressing the questions in this theme paper will be used to update the conceptual framework and to inform the decadal revision of the Great Lakes Fishery Commission's Vision Statement on Healthy Ecosystems.

## **Introduction**

The Great Lakes Fishery Commission (GLFC) adopts and advocates an ecosystem approach to fishery management and research in its Strategic Vision (GLFC 2001). The strategic vision is organized into three vision statements, one of which is the Vision Statement on Healthy Ecosystems. This vision statement seeks to achieve sustainable fisheries in the Great Lakes and, to that end, requires the Board of Technical Experts to initiate research that identifies the degree to which diseases have influenced fish populations. The Board of Technical Experts has organized the core of its Fishery Research Program into broad themes each described in a research theme paper. Theme papers review literature to establish the state of the science on a topic and describe central research questions and hypotheses for the Board and the Commission to focus on within their research program.

This theme paper specifically addresses inter-relationships among fish health, fish populations, and ecosystem dysfunction in the Great Lakes. This theme is related to another on-going theme “Ecosystem Health of Large Lakes: Identifying Utility and Metrics” (Hecky 2006) available for review at [www.glfc.org](http://www.glfc.org).

Understanding the ecology of emerging disease is crucial to long-term sustainability of wildlife populations (Friend et al. 2001), including diverse natural fish populations in the Great Lakes. Here we describe an ecosystem-level conceptual framework for fish health research in the Great Lakes and illustrate how it may be applied using three examples of fish health concerns in the Great Lakes: thiamine deficiency complex, botulism, and bacterial kidney disease. This approach recognizes that managing risks to fish health in the Great Lakes requires an understanding of the complex interactions among hosts, pathogens, and the environment (Snieszko 1973; Hedrick 1998).

The objectives of this research theme are to 1) propose an ecosystem-level conceptual framework that will focus research on the relationship between ecosystem dysfunction and fish health in the Great Lakes, 2) encourage research that will ultimately improve the understanding and prediction of the population-level effects of diseases on wild fish populations in the Great Lakes, and 3) provide a forum for coordinated and collaborative research in support of understanding the relationship between ecosystem function and fish health in the Great Lakes. We adopt a broad definition of fish health as a state characterized by anatomical integrity, the ability to perform individual-, population- and community-level roles, the ability to deal with physical, biological and social stresses, and the freedom from risk of disease and untimely death (Stephen and Thorburn 2002). We define diseases as threats to fish health which may include communicable diseases, parasites, nutrient deficiencies, and trophically accumulated toxins (e.g., botulism), but not environmental contaminants.

## **Fundamental assumptions**

The conceptual framework proposed below is built on several fundamental assumptions about the nature of Great Lakes ecosystems and disease processes:

- The biosphere is always changing; these changes have affected and will continue to affect Great Lakes aquatic ecosystems.
- Pathogens are natural components of ecosystems.
- Pathogens and hosts evolve in response to each other and to ecosystem conditions.
- Pathogens occur in the absence of disease.
- The prevalence and severity of disease are controlled by interactions among hosts, pathogens, and the environment, all of which are controlled by a combination of forcing by natural and anthropogenic variables.
- The prevalence and severity of disease vary temporally and spatially, within which a normative condition can be defined.
- Although diseases may have population-level effects and can exert a regulating force under some conditions, most pathogens do not exert long-term population-level effects, although individuals may be severely affected.
- Epidemiological techniques and tools from other disciplines (e.g., human health, conservation biology) can be applied to understanding diseases in aquatic ecosystems.
- Fish health and disease in wild fish populations can be better understood if evaluated in an ecosystem framework than in pathogen-centric or host-centric frameworks.
- Functional ecosystems are more likely to be resilient to disease events than ecosystems disrupted in structure and function.

### **Conceptual framework**

We present a conceptual model that describes one way in which disease processes may relate to ecosystem function. The model is a hypothetical framework that research will either support or reject, and which may lead to specific, process-related hypotheses that may be tested using a variety of disease models. A central premise is that functioning ecosystems are more likely to be resilient to disease events than dysfunctional ecosystems (e.g., Folke et al. 2004). The importance of ecosystem stability and function is consistent with epidemiological principles which suggest that the frequency and severity of disease events are governed in part by the state of the ecosystem (Hedrick 1998). For example, variables influencing disease transmission include temperature, host-immune response, and host-population density. These variables are further influenced by many ecosystem elements including climate warming, pollution (toxicants influence host susceptibility and infectivity of pathogens), fishery management actions (harvest and stocking alter host population density), and exotic species (which have the potential to alter host population density and may carry new pathogens that are frequently more pathogenic in naïve hosts).

Definitions of ecosystem health and ecosystem dysfunction are currently under debate by ecologists (Rapport et al. 1998; Hecky 2006), and a complete discussion of these topics is beyond the scope of this theme paper. We offer a broad definition of ecosystem dysfunction as a state in which ecosystems have been sufficiently disrupted to reduce vigor, organization, or resilience (cf. Rapport et al. 1998). For example, dysfunctional ecosystems may be characterized by major changes in community structure or in pathways of energy flow which may result in diminished ecosystem services. We hypothesize that functional healthy ecosystems with high resilience (McCann 2000) and few disruptions will experience relatively few or small changes to variables that influence the expression of disease. A functional ecosystem should have a

narrower natural range of temporal and spatial variability surrounding the variables which influence disease transmission than a dysfunctional ecosystem. Likewise, variables that influence disease transmission should exhibit a wider range of variability in dysfunctional ecosystems, thereby increasing the likelihood that the threshold required for development of epizootics will be exceeded.

The prevalence and severity of disease in wild populations are functions of interactions among pathogens, hosts, and the environment, and the latter is the least understood (Hedrick 1998). Most pathogens and other disease agents are natural components of ecosystems, but may only cause disease events under certain environmental conditions. We present a diagram (Fig. 1) based on Hedrick (1998) that represents one potential conceptual model linking environmental factors to fish disease. This conceptual framework suggests that disease processes must be considered in an ecosystem context to predict the impact of disease on wild fish populations, and that the maintenance of ecosystem function may be one way to minimize disease events in natural systems. Readers who disagree with our assumptions and framework are invited to submit research projects to disprove the conceptual model under this theme.

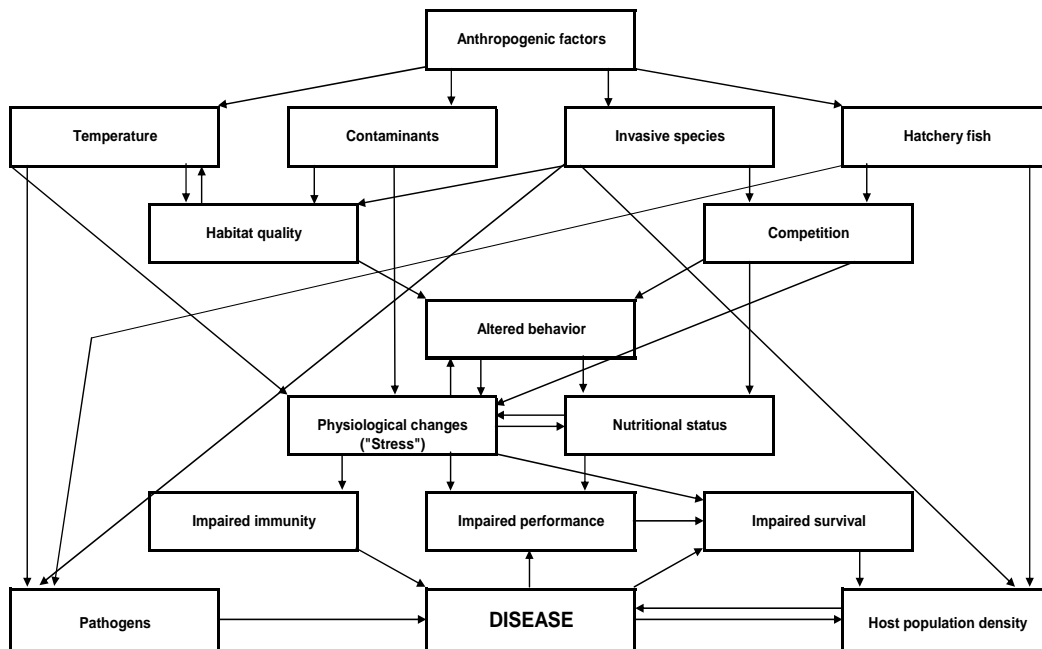


Figure 1. Conceptual framework representing a hypothetical web of causation for the manifestation of disease in a population of fish. Diagram modified after Hedrick (1998).

## Research goals

- To understand the relationship between ecosystem dysfunction and the frequency and severity of disease events in Great Lakes fish populations.
- To improve the understanding and prediction of population-level effects of diseases on wild fish populations in the Great Lakes.

## Overarching questions

- What is the relationship between ecosystem function and fish health (how is fish health related to ecosystem dysfunction or its symptoms?)
  - Which variables affect fish health and disease transmission and how and why do these variables differ between functional and dysfunctional ecosystems?
  - What are the mechanisms by which environmental variables affect disease processes at the population-level?
  - How do environmental variables interact to affect fish health?
  - What is a normative condition for fish pathogens in functional ecosystems?
- How can we improve prediction of population-level effects of diseases on wild fish populations?
  - What ecosystem components must be characterized to predict and understand population-level effects of diseases?
  - How can we best predict why specific diseases or types of diseases are more likely than other types of diseases to manifest themselves in the Great Lakes?
  - How can epidemiological and ecological models be used to predict the population-level effects of disease on fish populations?

## Understanding fish health in the Great Lakes from an ecosystem perspective

Many diseases have been reported from Great Lakes fish populations during the past few decades, including epizootic epitheliotropic disease (EED; Bradley et al. 1989), bacterial kidney disease (BKD; Beyerle and Hnath 2002), whirling disease (Beyerle and Hnath 2002), infectious pancreatic necrosis (IPN; Beyerle and Hnath 2002), viral hemorrhagic septicemia (VHS; NYSDEC 2006), heterosporis (Duggan et al. 2003), furunculosis (Beyerle and Hnath 2002), largemouth bass virus (SALBRC 2004), spring viremia of carp (SVC; Greg Wright, Chippewa-Ottawa Resource Authority, personal communication), piscirickettsia (MDNR 2004), thiamine deficiency complex (TDC, also known as EMS; Honeyfield et al. 1998) and botulism (Klindt and Town 2005; Getchell et al. 2006). The incidence of several diseases of Great Lakes fish populations appears to have increased in recent decades, including TDC (Honeyfield et al. 1998) and botulism (NYSDEC 2006), while emerging diseases such as VHS have recently resulted in large fish kills in the Great Lakes (NYSDEC 2006; USDA 2006).

Although the frequency of disease events in aquatic ecosystems has seemingly increased in recent decades (Harvell et al. 1999; Ward and Lafferty 2004), a lack of baseline data makes an explicit test of this hypothesis difficult (Lafferty et al. 2004). Disease detection ability has improved with advancing technology, which can lead to the appearance of an increasing rate of disease events (Lafferty et al. 2004). Given the lack of adequate baseline data, determining whether an apparent increase in frequency of disease events is real or due to a combination of better detection abilities and increased observational intensity is difficult (Lafferty et al. 2004). In the Great Lakes, baseline data do not exist and whether the incidence of disease is increasing or not in Great Lakes fish populations is unknown.

Although a lack of information exists on the ecology of disease in Great Lakes fish populations, how to best collect information describing the ecology of fish diseases is unclear (Stephen and Thorburn 2002). Monitoring programs cannot feasibly be developed for all potential diseases in all species in all of the Great Lakes. The focus of much fish health work has been at the individual level (Stephen and Thorburn 2002), but examining the effects of disease at population, community, or ecosystem levels may be more appropriate because these are the levels at which management and public concern are focused (Power 2002). Fish population status provides an integrative indicator of environmental conditions (Munkittrick and Dixon 1989) and may provide a relatively cost-effective way of assessing fish health. Population-level indices of the health of fish populations are based on the assessment of population characteristics such as size and age distributions, fecundity, age at maturity, abundance, or average body condition (Munkittrick and Dixon 1989; Shuter 1990; Power 2002). Stephen and Thorburn (2002) suggest that a population-based approach to fish health is necessary to understand fish disease ecology in the Great Lakes.

Epidemiology and disease dynamics have been studied extensively in human and some wild animal populations (Anderson and May 1979), but not in wild fish populations (Gozlan et al. 2006). Ecological investigations of natural populations have paid relatively little attention to disease (Real 1996; Lafferty and Gerber 2002), and ecological aspects of disease in wild fish populations have received particularly little study (Hedrick 1998; Reno 1998). Even for Atlantic salmon, one of the best-studied fish species, disease in wild populations has been little studied (Bakke and Harris 1998). Although population models show that the abundance and recruitment of herring can be affected by disease (Patterson 1996; Marty et al. 2003), little related work has been conducted in the Great Lakes, and therefore limited understanding exists of how disease affects the dynamics of Great Lakes fish populations. Integrated epidemiological and ecological models may prove useful in studying the ecology of fish disease in the Great Lakes.

Disease in wild fish populations results from complex interactions among pathogens, hosts, and the environment (Snieszko 1973; Hedrick 1998; Reno 1998; see Fig. 1). Many variables affect disease transmissibility, including temperature, host-immune response, and host-population density. Because these and other variables interact to determine the effect of a pathogen at the population level, understanding fish disease ecology and population-level impacts on fishes in the Great Lakes requires an integrated ecosystem perspective that accounts for variables affecting host resistance, pathogens, and the environment. Understanding the population-level impact of diseases and the conditions under which diseases are most likely to regulate fish populations is essential because this is the level at which managers and the public are concerned. For example, management biologists are less concerned about a disease that affects a few individuals severely but not the population as a whole than they are with a disease affecting individuals less severely but with population-level effects.

Whether or not diseases commonly have regulating effects on wild fish populations is unknown (Stephen and Thorburn 2002) and little field data are available to quantify the effects of disease on fish populations, particularly over large temporal scales (Bakke and Harris 1998; Stephen and Thorburn 2002). Although there are a number of examples of reductions in vertebrate abundance being directly linked to the prevalence of a pathogen (e.g., Sinderman 1987; Whittington et al. 1997; Hochachka and Dhondt 2000; Lafferty and Gerber 2002; Gozlan et al. 2005), disease mortality may invoke compensatory mechanisms and hence be non-regulating.

Indeed, some pathogens may play positive roles in promoting biodiversity and maintaining ecosystems (Lafferty et al. 2004). Some, perhaps most, infectious agents have no detectable effects on host populations, whereas others cause mortality, reduce growth, lower fecundity, alter behavior, or change social status (Lafferty et al. 2004). Moreover, the effects of disease on wild fish populations may be difficult to detect because diseased or dead animals may not be noticed (Schneider 1998), are difficult to directly observe, and because sub-clinical carriers of a pathogen may not be systematically surveyed (Gozlan et al. 2006).

We hypothesize that population-level effects of disease will be fewer in functional than in dysfunctional ecosystems. Recent environmental changes in the Great Lakes may be affecting the relative importance of disease as a factor regulating fish populations. The Great Lakes have been substantially disrupted in the past century, and with the exception of Lake Superior, largely have not achieved a stable equilibrium. Great Lakes ecosystems are currently dysfunctional due to altered land-use, increasing human populations, introduced species, pollution, and climate change, among other factors, and the effects of these factors on the prevalence, severity, and population-level impact of disease are unknown. A major goal of research and management in the Great Lakes is to understand how these variables affect ecosystems, including consideration of fish health and the ecology of disease.

Although anthropogenic factors may have profound effects on disease in Great Lakes ecosystems, relatively little research has focused on the effects of anthropogenic changes to the environment on the incidence of disease in natural populations (Daszak et al. 2001). The ecology of disease in Great Lakes ecosystems is complex due to the large number of potential disease pathogens and vectors and the variety of environmental factors that affect the incidence of disease. The incidence and impacts of disease in natural populations may be affected by pollution (Arkoosh et al. 1998; Lafferty and Gerber 2002), habitat alteration (Real 1996; Lafferty and Gerber 2002), food availability/host condition (Marty et al. 2003), outbreeding (Goldberg et al. 2005), temperature or climate warming (Harvell et al. 1999; Cairns et al. 2005), water quality (Inendino et al. 2005), introduced species (Lafferty et al. 2004), and the size, age, or life stage of the host (LaPatra 1998). These variables, many of which are affected by human activities, interact to affect fish health in the Great Lakes.

The rate of introduction of new pathogens to ecosystems has increased in recent decades (Daszak et al. 2000; Lafferty et al. 2004; Gozlan et al. 2006), and some may have devastating effects on recipient ecosystems (e.g., Gozlan et al. 2005). When a well-established population is severely affected by an epizootic, the cause is often a newly introduced pathogen (Lafferty and Gerber 2002). Introduced pathogens that are successful typically have a broad host range and are more pathogenic in new naïve hosts than in the original host (Lafferty et al. 2004).

Increases in anthropogenic stressors (e.g., contaminants, climate warming, habitat change) are often assumed to lead to increased disease in populations (Lafferty et al. 2004) because stressors typically increase an individual's susceptibility to disease. However, an increase in susceptibility at the individual level does not always translate directly to an increased effect at the population level. For example, some stressors may have more negative impacts on parasites than on hosts, thereby resulting in increased recovery rates of infected individuals and mitigation of the impacts of disease on the host population. If this effect is compounded by differential mortality of

diseased individuals, the spread of an epizootic through the population may actually be decreased by the stressor, despite an increased impact of the disease on infected individuals (Lafferty et al. 2004). Thus, the population-level effect of a disease is difficult to predict from the effect of the disease on individuals (Lafferty and Holt 2003).

Not all pathogens respond to stressors or environmental change in the same way, and recognizing that stressors can increase or decrease disease is important for a full understanding of the population-level effects of diseases (Lafferty et al. 2004). For example, stressors tend to have mitigating effects on infectious diseases when transmission is mainly between members of the same population and host specificity is high. Stressors are more likely to aggravate the effects of infectious diseases when pathogens are generalists or persist in a resistant environmental reservoir population (Lafferty and Holt 2003).

Some stressors affect population dynamics through mechanisms that decrease host density or through behavioral effects that decrease the probability of contact among hosts, and in these cases, stressors will reduce the chance of an epizootic through decreased probability of transmission (Lafferty et al. 2004). Population density may have significant effects on disease transmission in wild populations, because denser populations are more likely to promote transmission (Reno 1998; Ögüt 2001). Relatively small populations are less likely to transmit disease between individuals, because epidemiological principles suggest that the spread of infectious disease increases with host density (Lafferty and Gerber 2002). Because dense populations tend to have more parasites (Lafferty et al. 2004), epizootics could result from increases in host density rather than increased susceptibility due to external stressors. Environmental factors and fishery management practices that affect fish population size may therefore also affect disease transmission and fish health.

### **Application of the conceptual framework: three examples**

Invasion of the Great Lakes by multiple exotic fish and invertebrate species caused perturbations that may cascade through the entire ecosystem. For example, the invasion of the Great Lakes by zebra and quagga mussels (*Dreissena* spp.) has been implicated in changes in water chemistry (Nicholls et al. 1999), water clarity (Fahnenstiel et al. 1995), nutrient and energy flow (Hecky et al. 2004), benthic invertebrate density (Lozano et al. 2001; Mayer et al. 2002), algal community composition (Pillsbury et al. 2002), and the proliferation of blue-green algae (Bierman et al. 2005). The combined effects of invasive species and other anthropogenic stressors on Great Lakes ecosystems may be great but are difficult to predict (Vanderploeg et al. 2002), and may include serious impacts on fish health, which have the potential to undermine restoration efforts for native species.

We propose that an integrated, multi-species, ecosystem-level approach is necessary to understand the ecology and potential population-level effects of fish disease in the Great Lakes. An integrated research program should attempt to determine how environmental variables interact to affect fish health and develop a better understanding of the population-level impacts of disease on fish health in the Great Lakes. Here we provide three examples of fish health concerns that may be related to one ecological factor, exotic species, along with examples of specific research questions. Although the examples presented focus on three specific fish health

concerns, research proposed on other disease models that address the overarching research questions are welcome under this theme.

### **Thiamine Deficiency Complex**

Thiamine Deficiency Complex (TDC – also known as Early Mortality Syndrome, or EMS) results in high levels of early life stage mortality of salmonines in the Great Lakes and may affect adult behavior and survival (Brown et al. 2005b). TDC may be a significant impediment to restoration of lake trout and has the potential to severely restrict salmonine production in the Great Lakes (Fisher et al. 1996). TDC results from low egg thiamine concentrations that are thought to be caused by thiaminolytic enzymes (i.e., thiaminase) present in the diet (Tillitt et al. 2005) rather than low thiamine levels in the diet (Fitzsimons et al. 1998). TDC was present in the Great Lakes as early as the 1960s, but mortality rates from TDC rose dramatically in the 1990s (Honeyfield et al. 1998), coincident with the most recent wave of invasive species.

The sources and pathways of thiaminase in Great Lakes food webs have not been identified. Thiaminase may be produced by bacteria in the gut of forage fishes, although thiaminase-producing bacteria were found in only 25% of alewife stomachs sampled in one study (Honeyfield et al. 2002). Blue-green algae may also be a source of thiaminase (Honeyfield et al. 2002; Tillitt et al. 2005), but little evidence currently exists to identify sources of thiaminase in Great Lakes fishes. Identifying sources of thiaminase is an important step in determining how it moves through Great Lakes food webs.

Thiaminase activity of prey-fish species varies widely among sampling locations (Ji and Adelman 1998; Fitzsimons et al. 2005; Tillitt et al. 2005), so environmental variables may affect the incidence of TDC. Analysis of historical data suggests that the occurrence of TDC may be correlated with the presence of certain zooplankton and phytoplankton species (Hinterkopf et al. 1999), and thiaminase activity has been found in zooplankton (Zajicek et al. 2005). Thiaminase levels in salmonids are correlated with dietary factors (Brown et al. 2005a), so nutritional status may be related to thiaminase activity. Recent changes in the species composition of Great Lakes food webs may have resulted in increases in the abundance of lower trophic species that contain thiaminase, which may have affected the diet of fish species and may have increased the incidence or severity of TDC in some areas.

Key research questions for TDC:

1. What are the sources of thiaminase in Great Lakes food webs and how are they affected by environmental variables?
2. What are the trophic pathways by which fish predators acquire thiaminase from Great Lakes food webs and how is trophic transfer affected by environmental variables and food-web structure?
3. By what mechanisms do environmental variables control spatial and temporal variation in TDC?

4. How and why do the incidence and severity of TDC vary between functional and dysfunctional ecosystems?
5. What are the effects of TDC at the population level?
6. What are potential management strategies to mitigate TDC?

## **Botulism**

Botulism is caused by a potent neurotoxin produced by the bacterium *Clostridium botulinum*. Botulism toxin impacts neuromuscular function and may cause paralysis and death of infected animals. *C. botulinum* is classified into seven types (A – G) based on characteristics of the toxin produced; types C and E are most often associated with outbreaks of disease in fish and fish-eating birds. Periodic outbreaks of botulism have resulted in die-offs of fish and fish-eating birds in the Great Lakes since at least the 1960s (Brand et al. 1983), but outbreaks may have become more common and widespread in recent years, particularly in lakes Michigan, Huron, and Erie. Botulism has been implicated in the recent deaths of lake sturgeon in lakes Ontario and Erie coincident with bird die-offs (Klindt and Town 2005; Getchell et al. 2006) and may therefore be a serious impediment to lake sturgeon restoration.

Spores of *C. botulinum* are widely distributed in the environment and may occur in soil, surface waters, aquatic sediments, and the tissues of fish and other animals (Jensen and Allen 1960; Bott et al. 1968; Sayler et al. 1976; Reed and Rocke 1992; Kadlec 2002). *C. botulinum* was found in the digestive tracts of less than 10% of fish from the open waters of the Great Lakes, but was present in over 50% of fish from Green Bay (Lake Michigan), which suggests that the relative occurrence of bacterial spores varies among locations and may be related to local environmental conditions (Bott et al 1966).

Although *C. botulinum* spores are ubiquitous in the environment, toxin is produced only when the bacteria encounter suitable conditions for growth. The bacteria require anoxic conditions within specific temperature (>10°C) and pH (>5) ranges for germination, but the environmental factors required for toxins to accumulate to levels sufficient to cause die-offs are unclear (Rocke and Samuel 1999). Decaying organic matter, particularly plants or animal carcasses, may provide anaerobic conditions necessary for *C. botulinum* to grow (Reed and Rocke 1992; Barras and Kadlec 2000), and abundant populations of invertebrates that consume bacteria, or their predators, may also contribute to bioaccumulation of botulism toxin (Jensen and Allen 1960; Rocke et al. 1999; Kadlec 2002).

The presence of invasive species, particularly Dreissenid mussels and round gobies, has been hypothesized to play a role in the increased occurrence of botulism in Great Lakes fish and birds (Klindt and Town 2005; Perez-Fuentetaja et al. 2006). The invasion of the Great Lakes by Dreissenid mussels has been implicated in enhanced nearshore benthic algal growth (Hecky et al. 2004), which may help create the anoxic conditions required by *C. botulinum*. Dreissenid mussels may accumulate botulism toxin, which may be passed on to fish predators (e.g., round gobies, lake sturgeon).

Key research questions for botulism:

1. What are the trophic pathways by which fish and birds acquire botulism toxin from Great Lakes food webs and how is trophic transfer affected by environmental variables and food web structure? Is the occurrence of botulism outbreaks associated with the presence of certain species?
2. By what mechanisms do environmental variables control spatial and temporal variation in botulism toxin production and accumulation and the occurrence of botulism outbreaks?
3. What are the population-level effects of botulism? Are there sub-lethal effects of botulism that have population-level effects?
4. What are potential management strategies to mitigate botulism outbreaks?

### **Bacterial Kidney Disease**

Bacterial kidney disease (BKD) is a serious and often fatal disease caused by the bacterium *Renibacterium salmoninarum*, and is common in wild and hatchery-reared salmonids worldwide (Fryer and Sanders 1981; Arkoosh et al. 2004). BKD was first recognized in Atlantic salmon in Scotland in the 1930s (Fryer and Bartholomew 1996). Pacific salmon in the Great Lakes have been infected with *R. salmoninarum* since they were first introduced in the late 1960s (Holey et al. 1998), and the bacterium continues to be found in wild and hatchery-reared salmon in the Great Lakes (Beyerle and Hnath 2002).

BKD is characterized by kidney lesions, or in advanced stages, enlarged and necrotic kidneys (Beacham and Evelyn 1992). Although *R. salmoninarum* occurs naturally in wild salmonids, many infected fish show no clinical signs of disease (Jónsdóttir et al. 1998; Arkoosh et al. 2004). BKD may affect the behavior and survival of salmonids (Sanders et al. 1992; Price and Schreck 2003) and may increase susceptibility to predation (Mesa et al. 1998).

The epidemiology of BKD may be affected by ecological features of the environment such as the abundance of food (Jónsdóttir et al. 1998). The occurrence of a BKD epizootic in Lake Michigan Chinook salmon in the late 1980s and early 1990s may have been due to a decline in the abundance of alewives, which may have caused sufficient nutritional stress to make the salmon susceptible to *R. salmoninarum* (Holey et al. 1998). Such rapid changes in the abundance of a fish species may be a symptom of ecosystem dysfunction. Because *R. salmoninarum* continues to be routinely isolated from wild and hatchery salmon in the Great Lakes (Beyerle and Hnath 2002), the potential for future outbreaks of BKD exists.

Key research questions for BKD:

1. By what mechanisms do environmental variables control spatial and temporal variation in the occurrence and severity of BKD epizootics?

2. What are the population-level effects of BKD and how can we improve our ability to predict the occurrence of epizootics?
3. How does the incidence and severity of BKD vary between functional and dysfunctional ecosystems?
5. What are potential management strategies to mitigate BKD epizootics?

## References

- Anderson, R. M., and R. M. May. 1979. Population biology of infectious diseases. *Nature* 280: 361-367.
- Arkoosh, M. R., E. Casillas, E. Clemons, A. Kagley, R. Olson, R. Reno, and J. E. Stein. 1998. Effect of pollution on fish diseases: potential impacts on salmonid populations. *J. Aquat. Anim. Health* 10: 182-190.
- Arkoosh, M. R., and 11 coauthors. 2004. Survey of pathogens in juvenile salmon *Oncorhynchus* spp. migrating through Pacific Northwest estuaries. *J. Aquat. Anim. Health* 16: 186-196.
- Barras, S. C., and J. A. Kadlec. 2000. Abiotic predictors of avian botulism outbreaks in Utah. *Wild. Soc. Bull.* 28: 724-729.
- Beacham, T. D., and T. P. T. Evelyn. 1992. Genetic variation in disease resistance and growth of Chinook, coho, and chum salmon with respect to vibriosis, furunculosis, and bacterial kidney disease. *Trans. Am. Fish. Soc.* 121: 456-485.
- Beyerle, J., and J. G. Hnath. 2002. History of fish health inspections, State of Michigan, 1970-1999. Michigan DNR Fisheries Technical Report 2002-2.
- Bierman, V. J., J. Kaur, J. V. DePinto, T. J. Feist, and D. W. Dilks. 2005. Modeling the role of zebra mussels in the proliferation of blue-green algae in Saginaw Bay, Lake Huron. *J. Great Lakes Res.* 31: 32-55.
- Bott, T. L., J. S. Deffner, E. McCoy, and E. M. Foster. 1966. *Clostridium botulinum* type E in fish from the Great Lakes. *J. Bacteriol.* 91: 919-924.
- Bott, T. L., J. Johnson, E. M. Foster, and H. Sugiyama. 1968. Possible origin of the high incidence of *Clostridium botulinum* type E in an inland bay (Green Bay of Lake Michigan). *J. Bacteriol.* 95: 1542-1547.
- Bradley, T. M., D. J. Medina, P. W. Chang, and J. R. McClain. 1989. Epizootic epitheliotropic disease of lake trout (*Salvelinus namaycush*): history and viral etiology. *Diseases of Aquatic Organisms* 7:195-201.

- Brand, C. J., R. M. Duncan, S. P Garrow, D. Olson, and L. E. Schumann. 1983. Waterbird mortality from botulism type E in Lake Michigan: an update. *Wilson Bull.* 95: 269-275.
- Breitholtz, M., C. Hill and B. Bengtsson. 2001. Toxic substances and reproductive disorders in Baltic fish and crustaceans. *Ambio* 30: 210-216.
- Brown, S. B., M. T. Arts, L. R. Brown, M. Brown, J. D. Fitzsimons, D. C. Honeyfield, D. E. Tillit, J. L. Zajicek, M. Wolgamood, and J. G. Hnath. 2005a. Can diet-dependent factors help explain fish-to-fish variation in thiamine-dependent early mortality syndrome? *J. Aquat. Anim. Health* 17: 36-47.
- Brown, S. B., D. C. Honeyfield, J. G. Hnath, M. Wolgamood, S. V. Marquenski, J. D. Fitzsimons, and D. E. Tillit. 2005b. Thiamine status in adult salmonines in the Great Lakes. *J. Aquat. Anim. Health* 17: 59-64.
- Cairns, M. A., J. L. Ebersole, J. P Baker, P. J. Wigington, H. R. Lavigne, and S. M. Davis. 2005. Influence of summer stream temperatures on black spot infestation of juvenile coho salmon in the Oregon Coast Range. *Trans. Am. Fish. Soc.* 134: 1471-1479.
- Daszak, P., A. A. Cunningham, and A. D. Hyatt. 2000. Emerging infectious disease of wildlife – threats to biodiversity and human health. *Science* 287: 443-449.
- Daszak, P., A. A. Cunningham, and A. D. Hyatt. 2001. Anthropogenic environmental change and the emergence of infectious diseases in wildlife. *Acta Tropica* 78: 103-116.
- Duggan, I. C., S. A. Bailey, R. I. Colautti, D. K. Gray, J. C. Makarewicz, and H. J. MacIsaac. 2003. Biological invasions in Lake Ontario: past, present and future. In M. Munawar, Ed. *State of Lake Ontario – Past, Present and Future*. Ecovision World Monograph Series, Aquatic Ecosystem Health and Management Society.
- Fahnenstiel, G. L., G. A. Land, T. F. Nalepa, and T. H. Johengen. 1995. Effects of zebra mussel (*Dreissena polymorpha*) colonization on water quality parameters in Saginaw Bay, Lake Huron. *J. Great Lakes Res.* 21: 435-448.
- Fisher, J. P., J. D. Fitzsimons, G. F. Combs, and J. M. Spitsbergen. 1996. Naturally occurring thiamine deficiency causing reproductive failure in Finger Lakes Atlantic salmon and Great Lakes lake trout. *Trans. Am Fish. Soc.* 125: 167-178.
- Fitzsimons, J. D., S. B. Brown, and L. Vandenbyllaardt. 1998. Thiamine levels in food chains of the Great Lakes. *American Fisheries Society Symposium* 21: 90-98.
- Fitzsimons, J. D., B. Williston, J. L. Zajicek, D. E. Tillitt, S. B. Brown, L R. Brown, D. C. Honeyfield, D. M. Warner, L. G. Rudstam, and W. Pearsall. 2005. Thiamine content and thiaminase activity of ten freshwater stocks and one marine stock of alewives. *J. Aquat. Anim. Health* 17: 26-35.

- Folke, C., S. Carpenter, B. Walker, M. Scheffer, T. Elmqvist, L. Gunderson, and C. S. Holling. 2004. Regime shifts, resilience, and biodiversity in ecosystem management. *Annu. Rev. Ecol. Evol. Syst.* 35: 557-581.
- Friend, M., R. G. McLean, and F. J. Dein. 2001. Disease emergence in birds: challenges for the twenty-first century. *Auk* 118: 290-303.
- Fryer, J. L., and J. E. Sanders. 1981. Bacterial kidney disease of salmonid fish. *Annual Review of Microbiology.* 35: 273-298.
- Getchell, R. G., W. J. Culligan, M. Kirchgessner, C. A. Sutton, R. N. Casey, and P. R. Bowser. 2006. Quantitative polymerase chain reaction assay used to measure the prevalence of *Clostridium botulinum* type E in fish in the lower Great Lakes. *J. Aquat. Anim Health* 18: 39-50.
- GLFC (Great Lakes Fishery Commission). 2001. Strategic vision of the Great Lakes Fishery Commission for the first decade of the new millennium. Great Lakes Fishery Commission, Ann Arbor, MI.
- Goldberg, T. L., E. C. Grant, K. R. Inendino, T. W. Kassler, J. E. Claussen, and D. P. Philipp. 2005. Increased infectious disease susceptibility resulting from outbreeding depression. *Cons. Biol.* 19: 455-462.
- Gozlan, R. E., S. St-Hilaire, S. W. Feist, P. Martin, and M L. Kent. 2005. Disease threat to European fish. *Nature* 435: 1046.
- Gozlan, R. E., E. J. Peeler, M. Longshaw, S. St-Hilaire, and S. W. Feist. 2006. Effect of microbial pathogens on the diversity of aquatic populations, notably in Europe. *Microbes and Infection* 8: 1358-1364.
- Harvell, C. D., and 12 coauthors. 1999. Emerging marine diseases – climate links and anthropogenic factors. *Science* 285:1505-1510.
- Hecky, R. E. 2006. Ecosystem Health of Large Lakes: Identifying Utility and Metrics. Research Theme Paper, Fishery Research Program, Great Lakes Fishery Commission. [http://www.glfc.org/research/Hecky\\_Health\\_of\\_large\\_systems.pdf](http://www.glfc.org/research/Hecky_Health_of_large_systems.pdf)
- Hecky, R. E., R. E. H. Smith, D. R. Barton, S. J. Guilford, and W. D. Taylor. 2004. The nearshore phosphorus shunt: a consequence of ecosystem engineering by dreissenids in the Laurentian Great Lakes. *Can. J. Fish. Aquat. Sci.* 61: 1285-1293.
- Hedrick, R. P. 1998. Relationships of the host, pathogen, and environment: implications for diseases of cultured and wild fish populations. *J. Aquat. Anim. Health* 10: 107-111.
- Hinterkopf, J. P., D. C. Honeyfield, J. Mardarewicz, and T. Lewis. 1999. Abundance of plankton species in Lake Michigan and incidence of early mortality syndrome from 1983

- to 1992. Great Lakes Fishery Commission Board of Technical Experts Report on Early Mortality Syndrome Workshop. Great Lakes Fishery Commission.  
<http://www.glfrc.org/staff/emsmeet7.pdf>
- Hochachka, W. M. and A. A. Dhondt. 2000. Density-dependent decline of host abundance resulting from a new infectious disease. *Proc. Nat. Acad. Sci.* 97: 5303-5306.
- Holey, M. E., R. F. Elliott, S. V. Marcquenski, J. G. Hnath, and K. D. Smith. 1998. Chinook salmon epizootics in Lake Michigan: possible contributing factors and management implications. *J. Aquat. Anim. Health* 10: 202-210.
- Honeyfield, D. C., J. D. Fitzsimons, S. B. Brown, S. V. Marcquenski, and G. McDonald. 1998. Introduction and overview of early life stage mortality. *American Fisheries Society Symposium* 21: 1-7.
- Honeyfield, D. C., J. P. Hinterkopf, and S. B. Brown. 2002. Isolation of thiaminase-positive bacteria from alewife. *Trans. Am. Fish. Soc.* 131: 171-175.
- Inendino, K. R., E. C. Grant, D. P. Philipp, and T. L. Goldberg. 2005. Effects of factors related to water quality and population density on the sensitivity of juvenile largemouth bass to mortality induced by viral infection. *J. Aquat. Anim. Health* 17: 304-314.
- Jensen, W. I., and J. P. Allen. 1960. A possible relationship between aquatic invertebrates and avian botulism. *Trans. N. Am. Wildl. Nat. Res. Conf.* 25: 171-180.
- Ji, Y. Q., and I. R. Adelman. 1998. Thiaminase activity in alewives and smelt in Lakes Huron, Michigan, and Superior. *American Fisheries Society Symposium* 21: 154-159.
- Jónsdóttir, H., H. J. Malmquist, S. S. Snorrason, G. Gudbergsson, and S. Gudmundsdóttir. 1998. Epidemiology of *Renibacterium salmoninarum* in wild Arctic charr and brown trout in Iceland. *J. Fish Biol.* 53: 322-339.
- Kadlec, J. A. 2002. Avian botulism in Great Salt Lake marshes: perspectives and possible mechanisms. *Wildl. Soc. Bull.* 30: 983-989.
- Klindt, R. M., and B. Town. 2005. Lake sturgeon restoration and botulism E, 2004. NYSDEC Lake Ontario Annual Report 2004. New York State Department of Environmental Conservation. Presented at the Lake Ontario Committee meeting, Niagara Falls, Ontario, 29-30 March, 2005.
- Lafferty, K. D., and L. Gerber. 2002. Good medicine for conservation biology: the intersection of epidemiology and conservation theory. *Conserv. Biol.* 16: 593-604.
- Lafferty, K., and R. D. Holt. 2003. How should environmental stress affect the population dynamics of disease? *Ecol. Lett.* 6: 654-664.

- Lafferty, K. D., J. W. Porter, and S. E. Ford. 2004. Are diseases increasing in the ocean? *Annu. Rec. Ecol. Evol. Syst.* 35: 31-54.
- LaPatra, S. E. 1998. Factors affecting pathogenicity of infectious hematopoietic necrosis virus (IHNV) for salmonid fish. *J. Aquat. Anim. Health* 10: 121-131.
- Lozano, S. J., J. V. Sharold, and T. F. Nalepa. 2001. Recent declines in benthic macroinvertebrate densities in Lake Ontario. *Can. J. Fish. Aquat. Sci.* 58: 518-529.
- Marty, G. D., T. J. Quinn, G. Carpenter, T. R. Myers, and N. H. Willits. 2003. Role of disease in abundance of a Pacific herring (*Clupea pallasii*) population. *Can. J. Fish. Aquat. Sci.* 60: 1258-1265.
- Mayer, C. M., R. A., Keats, L. G. Rudstam, and E. L. Mills. 2002. Scale-dependent effects of zebra mussels on benthic invertebrates in a large eutrophic lake. *J. N. Am. Benth. Soc.* 21: 616-633.
- McCann, K. W. 2000. The diversity-stability debate. *Nature* 405:228-233.
- MDNR (Michigan Department of Natural Resources). 2004. Muskie Piscirickettsia (Muskie Pox) Update. MDNR Fisheries Division Public Update, 11 May 2004.
- Mesa, M. G., T. P. Poe, A. G. Maule, and C. B. Schreck. 1998. Vulnerability to predation and physiological stress response in juvenile Chinook salmon (*Oncorhynchus tshawytscha*) experimentally infected with *Renibacterium salmoninarum*. *Can. J. Fish. Aquat. Sci.* 55L: 1599-1606.
- Munkittrick, K. R., and D. G. Dixon. 1989. A holistic approach to ecosystem health using fish population characteristics. *Hydrobiologia* 188/189: 123-135.
- Nicholls, K. H., G. J. Hopkins, and S. J. Standke. 1999. Reduced chlorophyll to phosphorus ratios in nearshore Great Lakes waters coincide with the establishment of dreissenid mussels. *Can. J. Fish. Aquat. Sci.* 56: 153-161.
- NYSDEC (New York State Department of Environmental Conservation). 2006. DEC continues investigation of death of Lake Ontario shore birds and fish. NYSDEC press release, 20 July 2006.
- Ögüt, H. 2001. Modeling of fish disease dynamics: a new approach to an old problem. *Turk. J. Fish. Aquat. Sci.* 1: 67-74.
- Patterson, K. R. 1996. Modelling the impact of disease-induced mortality in an exploited population: the outbreak of the fungal parasite *Ichthyophonus hoferi* in the North Sea herring (*Clupea harengus*). *Can. J. Fish. Aquat. Sci.* 53: 2870-2887.

- Perez-Fuentetaja, A., M. D. Clapsadl, D. Einhouse, P. R. Bowser, R. G. Getchell, and W. T. Lee. 2006. Influence of limnological conditions on *Clostridium botulinum* type E presence in Eastern Lake Erie sediments (Great Lakes, USA). *Hydrobiologia* 563: 189-200.
- Pillsbury, R. W., R. L. Lowe, Y. D. Pan, and J. L Greenwood. 2002. Changes in the benthic algal community and nutrient limitation in Saginaw Bay, Lake Huron, during the invasion of the zebra mussel (*Dreissena polymorpha*). *J. N. Am. Benth. Soc.* 21: 238-252.
- Power, M. 2002. Assessing fish population responses to stress. Pages 379 - 430 in S. M. Adams, ed. *Biological indicators of aquatic ecosystem stress*. American Fisheries Society, Bethesda, MD.
- Price, C. S., and C. B. Schreck. 2003. Effects of bacterial kidney disease on saltwater preference of juvenile spring chinook salmon, *Oncorhynchus tshawytscha*. *Aquaculture* 222: 331-341.
- Rapport, D. J., R. Constanza, and A. J. McMichael. 1998. Assessing ecosystem health. *Trends Eco. Evol.* 13: 397-402.
- Real, L. A. 1996. Sustainability and the ecology of infectious disease. *BioScience* 46: 88-97.
- Reed, T. M., and T. E. Rocke. 1992. The role of avian carcasses in botulism epizootics. *Wildl. Soc. Bull.* 20: 175-182.
- Reno, P. W. 1998. Factors involved in the dissemination of disease in fish populations. *J. Aquat. Anim. Health* 10: 160-171.
- Rocke, T. E., and M. D. Samuel. 1999. Water and sediment characteristics associated with avian botulism outbreaks in wetlands. *J. Wildl. Manage.* 63: 1249-1260.
- Rocke, T. E., N. H. Euliss, and M. D. Samuel. 1999. Environmental characteristics associated with the occurrence of avian botulism in wetlands of a northern California refuge. *J. Wildl. Manage.* 63: 358-368.
- SALBRC (Smallmouth and Largemouth Bass Regulations Committee). 2004. Black bass fishing seasons in Michigan: background, research review, and recommendations. Michigan DNR, Fisheries Division.
- Sanders, J. E., J. J. Long, DC. K. Arakawa, J. L. Bartholomew, and J. S. Rohovec. 1992. Prevalence of *Renibacterium salmoninarum* among downstream-migrating salmonids in the Columbia River. *J. Aquat. Anim. Health* 4: 72-75.
- Sayler, G. S., J. D. Nelson, A. Justice, and R. R. Colwell. 1976. Incidence of *Salmonella* spp., *Clostridium botulinum*, and *Vibrio parahaemolyticus* in an estuary. *App. Env. Microbiol.* 31: 723-730.

- Schneider, J. C. 1998. Fate of dead fish in a small lake. *Am. Midl. Nat.* 140: 192-196.
- Shuter, B. J. 1990. Population-level indicators of stress. *Am. Fish. Soc. Symp.* 8: 145-166.
- Sinderman, C. J. 1987. Effects of parasites on fish populations: practical considerations. *Int. J. Parasit.* 17: 371-382.
- Snieszko, S. F. 1973. Recent advances in scientific knowledge and developments pertaining to diseases of fishes. *In*: C. A. Brandly and C. E. Cornelius, eds, *Advances in Veterinary Science and Comparative Medicine*. Academic Press, New York.
- Stephen, C., and M. Thorburn. 2002. Formulating a vision for fish health research in the Great Lakes. Great Lakes Fishery Commission Project Completion Report.
- Tillitt, D. E., J. L. Zajicek, S. B. Brown, L. R. Brown, J. D. Fitzsimons, D. C. Honeyfield, M. E. Holey, and G. M. Wright. 2005. Thiamine and thiaminase status in forage fish of salmonines from Lake Michigan. *J. Aquat. Anim. Health* 17: 13-25.
- USDA (U. S. Department of Agriculture). 2006. Viral Hemorrhagic Septicemia in the Great Lakes. Animal and Plant Health Inspection Service Veterinary Services, Centers for Epidemiology and Animal Health. Emerging Disease Notice, July 2006. [http://www.aphis.usda.gov/vs/ceah/cei/taf/emergingdiseasenotice\\_files/vhsgreatlakes.htm](http://www.aphis.usda.gov/vs/ceah/cei/taf/emergingdiseasenotice_files/vhsgreatlakes.htm)
- Vanderploeg, H. A., T. F. Nalepa, D. J. Jude, E. L. Mills, K. T. Holeck, J. R. Liebig, I. A. Grigorovich, and H. Ojaveer. 2002. Dispersal and emerging ecological impacts of Ponto-Caspian species in the Laurentian Great Lakes. *Can. J. Fish. Aquat. Sci.* 59: 1209-1228.
- Ward, J. R., and K. D. Lafferty. 2004. The elusive baseline of marine disease: are diseases in ocean ecosystems increasing? *PloS Biology* 2: 542-547. <http://biology.plosjournals.org>
- Whittington, R. J., J. B. Jones, P. H. Hine, and A. D. Hyatt. 1997. Epizootic mortality in the pilchard *Sardinops sagax neopilchardus* in Australia and New Zealand in 1995. I. Pathology and epizootiology. *Dis. Aquat. Org.* 28: 1-16.
- Zajicek, J. L., D. E. Tillitt, D. C. Honeyfield, S. B. Brown, and J. D. Fitzsimons. 2005. A method for measuring total thiaminase activity in fish tissues. *Journal of Aquatic Animal Health* 17:82-94.

## **Products**

**Objective 1** (Propose an ecosystem-level conceptual framework that will focus future research on the relationship between ecosystem dysfunction and fish health in the Great Lakes):

- An approved theme paper available through the internet under the GLFC's Fishery Research Program (by December 2006)
- Publication of a variation of the theme paper as a perspectives-type article in a peer-reviewed journal (by May 2007)

**Objective 2** (Encourage research that will ultimately improve the understanding and prediction of the population-level effects of diseases on wild fish populations in the Great Lakes):

- A suite of research projects (including completion reports and peer-reviewed publications) that address the questions presented in the theme paper (by December 2012)

**Objective 3** (Provide a forum for a coordinated and collaborative approach to research in support of understanding the relationship between ecosystem function and fish health in the Great Lakes):

- Three biennial research coordination meetings in which PIs funded under this theme and other researchers participating in similar research present findings and discuss research needs (one each in 2008, 2010, and 2012). Workshop summaries will be available via the internet.
- A completion report summarizing progress towards achieving an understanding of the relationship between ecosystem function and fish health in the Great Lakes and a revised conceptual framework with revised research questions (by December 2012)

## **Schedule**

This research theme will begin on 1 January 2007 and continue through 31 December 2012.

Theme research coordination meetings will be held in summer/fall 2008, 2010 and 2012.

Progress reports will be submitted to the Board of Technical Experts at spring and fall meetings.