Interactive Effects of Hypoxia and Mercury Contamination in Great Lakes Fish

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Overview

The broad goal of this research is to evaluate impacts of hypoxia and mercury on long-term sustainability of fish populations. Mercury and hypoxia are two stressors of concern in the Great Lakes ecosystem. Although they commonly co-occur in aquatic systems, it is not known how effects of these two stressors might interact to impair survival, growth, and reproduction. The hypothesis of this study is that mercury-polluted fish have a reduced ability to respond to hypoxic stress. We will address this hypothesis by conducting a laboratory study to expose yellow perch to environmentally relevant levels of hypoxia and mercury. Health effects will be determined using molecular and physiological techniques. This study serves the dual purpose of 1) increasing our understanding of mechanisms underlying responses to hypoxia and mercury in fish, and 2) identifying novel molecular/physiological markers than can be used to forecast early changes to animal health. Future planned studies will include applying these novel molecular/physiological markers to evaluate and predict sustainability of fish populations.

Objective

Increase number of protected species that reach stable or increasing population levels.

Proposed Work

The hypothesis of this research is that mercury-polluted fish have a reduced ability to respond to hypoxic stress. This hypothesis will be addressed by conducting a laboratory study to:

1. Determine if hypoxia affects the accumulation and distribution of mercury in fish tissues
2. Determine if mercury polluted fish have impaired abilities to mount a response (both cellular and physiological) when confronted with hypoxic challenge
3. Validate molecular biomarkers of hypoxia and mercury for use in diagnosing fish health and managing wild fish populations.

Scientific Rationale

Hypoxia

Low dissolved oxygen resulting from eutrophication of aquatic systems is a common problem in both marine (Diaz & Rosenberg 2008) and freshwater (Hawley et al. 2006) environments. Extreme hypoxia can lead to massive fish kills, while moderate hypoxia causes alterations to community structure, predation, and behavior (Pollock et al. 2007). Compensatory mechanisms of exposure to hypoxia are fairly well described in fish (e.g. increases in gill surface area,

increased haemoglobin, altered metabolic pathways, reduced neurotransmission (Wu 2002)), but less is known about mechanisms by which episodic and sub-lethal exposure might affect ecologically relevant parameters such as survival, growth and reproduction. Moreover, interactive effects between the response to low oxygen and other environmental stressors have seldom been studied in fish.

Mercury

Water bodies that experience seasonal hypoxia can also be heavily contaminated with environmental pollutants such as the heavy metal mercury (Hg). Mercury is found in all Great Lakes fish, often at levels exceeding the EPA threshold for human consumption (0.3 ppm, U.S. EPA 2007). Nearly 40% of U.S. water bodies are under a mercury advisory. Beyond the human health risk, growing evidence suggests that current levels of mercury have the potential to negatively impact the sustainability of wild fish populations. Sub-lethal concentrations of mercury impair behavior, reproduction, and stress responses in fish (Wiener et al. 2003; Hammerschmidt et al. 2002; Sandheinrich et al. 2006; Hontela et al. 1992).

Hypoxia x Mercury

Hypoxia and mercury contamination commonly co-occur in nature, but little is known about the interactive effects of these two stressors on fish populations. Molecular level responses to multiple stressors can be informative for assessing population health in that they can serve as early warning for, and establish causal linkages to, physiological responses. In the brain, hypoxia and methylmercury both disrupt signaling between the neurotransmitter glutamate and its NMDA receptor (Walsh et al. 2007, Basu et al. 2007). Downregulation of NMDA receptors has been seen in multiple species and can have serious impacts on learning and motor function (Ozawa et al. 1998). In spite of this molecular link, effects of hypoxia on NMDA receptor levels in mercury-exposed animals have not been explored.

Interactions between molecular responses to hypoxia and mercury may also occur in other tissues. Hypoxia inducible factor (HIF) is a widely expressed protein that coordinates the compensatory response to hypoxia. Divalent metals such as cobalt and nickel have also been shown to stabilize HIF proteins and promote transcription of HIF-mediated genes. Mercury, another divalent metal, may also be involved in HIF signaling (Li et al. 2006; Vengellur & LaPres 2004). The relationship between divalent metals and HIF signaling has not yet been examined in fish.

Figure 1: *Mercury x Hypoxia experiment in Dr. Michael Carvan's lab at the Great Lakes WATER Institute, Milwaukee, WI*

Governmental/Societal Relevance

Hypoxia and mercury are two stressors of concern in the Great Lakes ecosystem. Both have the potential to affect the sustainability of Great Lakes commercial and recreational fisheries. We are not aware of any studies (lab or field based) that address the combined and interactive effects of hypoxia and mercury on fish. The data generated from this project will provide new tools to assess interactive effects of two stressors of concern to Great Lakes fish. This will be useful to government agencies and benefit the public by contributing to the sustainability and quality of Great Lakes fish populations.

Relevance to Ecosystem Forecasting

Ecosystem forecasting depends on a basic understanding of the underlying causes of the phenomenon being studied. Little work has been done to understand how stressors in the Great Lakes ecosystem might interact to produce effects at a molecular, cellular, or physiological level. Our laboratory-based work will provide a basis for understanding and evaluating interactive effects of multiple stressors. These data will contribute to more realistic assessments of Great Lakes ecosystem health. Follow-up studies using these same endpoints in a field setting will allow causal linkages to be established. Integrated knowledge of physiological and molecular effects of multiple stressors will lead to better forecasting at an ecosystem level.

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