RE-EVALUATION OF THE THIAMINE DOSE-RESPONSE RELATIONSHIP FOR LARVAL LAKE TROUT (*SALVELINUS NAMAYCUSH*) USING AN INDIVIDUAL BASED MODEL

By

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ABSTRACT

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Low thiamine levels in lake trout eggs is a condition thought to contribute to widespread recruitment failure in Great Lakes populations. Substantial, natural reproduction has not been achieved by lake trout in the Great Lakes, except Lake Superior, for decades despite continued stocking efforts. Low thiamine levels cause death or abnormal behavior in larval, juvenile and adult lake trout. This condition, known as thiamine deficiency or TD, has been associated with adult lake trout's dietary uptake of the thiaminase enzyme, which is present in prey species at different concentrations. Dose-response curves have been developed to predict mortality based on egg thiamine concentration. These curves may underestimate the response of lake trout to low thiamine because they do not consider sub-lethal effects. To investigate the sub-lethal effects of TD, I developed an individual based model (IBM) for larval lake trout. The IBM simulates the daily activities of fish that survive to the free swimming larval stage and tracks growth and mortality until they become thiamine replete. Low thiamine affects larval traits such as prey capture ability and growth rate, which can be easily adjusted in the model. Simulation results indicate TD in combination with moderate predation or high predation alone can reduce survival to zero. Simulation results also indicate lake trout may be more sensitive to low thiamine than previously reported. The results of this study suggest that an IBM is a promising technique that can be used to develop more accurate dose-response curves for wild populations, and to evaluate the effect of stressors at the population level.

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CHAPTER 1

THIAMINE DEFFICIENCY AND LAKE TROUT POPULATIONS IN THE GREAT LAKES: A RECENT HISTORY

Introduction

Lake trout (*Salvelinus namaycush*) have had limited reproductive success in the Great Lakes over the past 50 years and thiamine deficiency (TD) has been cited as a cause. In all Great Lakes, outside of Lake Superior, lake trout populations have not recovered from collapse caused by both sea lamprey (*Petromyzon marinus*) predation and overfishing pressure, although millions have been stocked annually since the 1960s. Predation on all lake trout life stages, low spawner density, disease, habitat degradation, competition and other unknown stressors have greatly reduced or eliminated natural reproduction in lake trout populations.TD, a condition caused by the dietary uptake of thiaminase enzyme, might be contributing disproportionately to the recruitment failure in lake trout populations of the Great Lakes region in North America. TD is potentially a substantial threat to lake trout populations and, at present, is not well understood (Riley et. al. 2008).

Thiamine (also known as vitamin B_1) is an essential molecule in cellular respiration. It is biosynthesized in bacteria, fungi and plants but other organisms need to acquire thiamine from dietary sources (Wrenger et. al. 2006). Thiamine is essential for catabolic processes in a cell; it is required for the oxidative decarboxylation of pyruvate to form acetyl-coenzyme A (Campbell 2002). Acetyl-coenzyme A is the form of carbon that enters the citric acid cycle. If thiamine is absent or in low concentration, carbohydrate metabolism can be impaired (Wrenger et. al. 2006). Salmonids, like lake

trout, typically require between 10-15 mg of thiamine per kilogram of dry weight in their diet (Moyle and Cech 2004).

Thiaminase is an enzyme that catalyzes the breakdown of thiamine and can be toxic in large concentrations (Tillitt et. al. 2005). When TD has been observed in salmonids, their prey species often have elevated thiaminase levels. In some cases the cause of TD in lake trout and other salmonids has been attributed to the consumption of thiaminase containing prey species like alewife (Tillitt et. al. 2005, Riley et. al. 2008).

The result of TD is death at swim-up if egg thiamine concentrations are below a certain threshold but larval lake trout with egg thiamine concentrations above that threshold can still experience negative effects. The reported threshold thiamine value for death prior to swim-up varies from 0.8 nmol/g (Brown et. al. 1998) to 1-1.5 nmol/g (Honeyfield et. al. 2005). Twenty percent reductions in growth and prey capture ability have been reported in larval lake trout with egg thiamine concentrations of 8.1 nmol/g and 6.9 nmol/g respectively (Fitzsimons et. al. 2009). Sublethal effects of TD may have substantial negative impacts on larval lake trout ability to survive.

Forage fish contain vastly different amounts of thiaminase enzyme. In the Great Lakes system a common native prey item is the bloater (*Coregonus hoyi*). The bloater has an average thiaminase activity of 35 pmol/g*min while the invasive alewife (*Alosa pseudoharengus*) has an average thiaminase activity of 4280 pmol/g*min (Tillitt et. al. 2005; Figure 1.1).

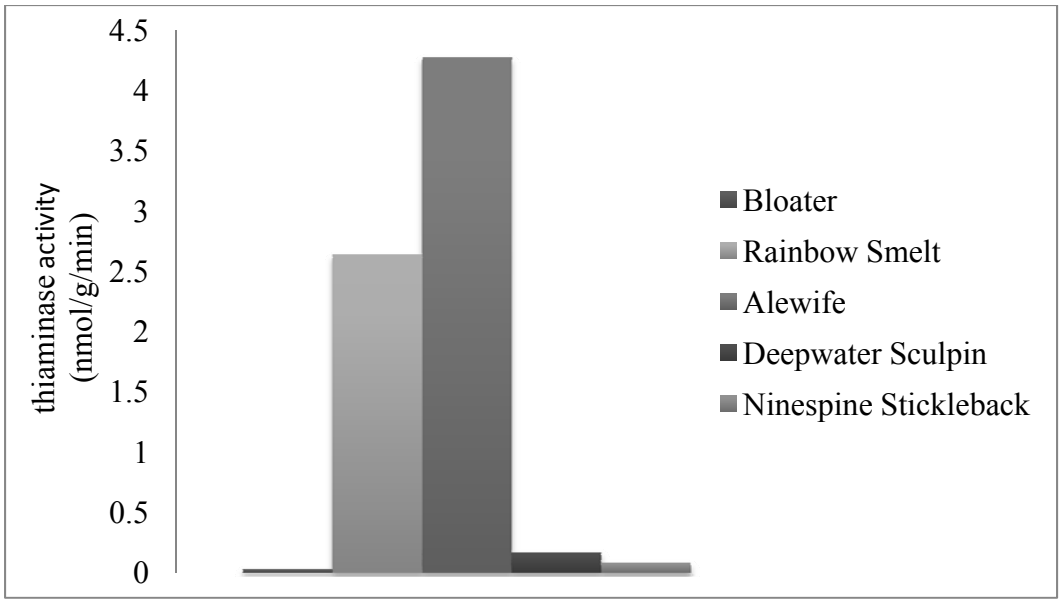

Figure 1.1. Thiaminase Activity of Common Lake Trout Prey in nmol/g/min. From left to right the bars represent Bloater (*Coregonus hoyi*), Rainbow Smelt (*Osmerus mordax*) , Alewife (*Alosa pseudoharengus*), Deepwater Sculpin (*Cottus spp.*) and Ninespine Stickleback(*Pungitius pungitius*) (Tillitt et. al. 2005).

The wide variation in thiaminase activity between species can only be partially explained by phylogeny in the Great Lakes (Riley and Evans 2008) because not only is there great intra-species variation in thiaminase activity but great inter-species variation as well. Experiments were performed by Lepak et. al. (2008) to determine the effect of low food and salt concentration stress on thiaminase activity in alewives. Neither variable could be correlated with thiaminase activity but the captive alewives average thiaminase activity ranged from 6,900 ± 2,800 pmol/g*min at the moment of capture to 16,000 ± 5,900 pmol/g*min, after 1.5 to 2.5 years in captivity (Lepak et. al. 2008). The levels of thiaminase in captive alewife are more than two times higher than levels measured in field caught alewife around the Great Lakes (Fitzsimons et. al. 2005). Some unknown factor, besides food or salt concentration, appears to affect thiaminase activity in captive alewife.

The transmission of thiaminase enzyme through the food web is uncertain, however several hypotheses are suggested. Thiaminase producing bacteria in the gut of prey fishes could be a source, although a study of alewife stomachs revealed that only 25 percent of the fish examined contained thiaminase producing bacteria (Honeyfield et. al. 2002). Thiaminase producing algae might be the source of thiaminase in the food web, although there is less direct evidence to support this connection. Some blue-green algae species show elevated thiaminase activity (Grigor et al. 1977). Although the source of thiaminase is unknown, salmonids of the Great Lakes that consume prey with elevated thiaminase levels tend to have TD.

Occurrence of TD in the Great Lakes Region

TD in wild fish has been observed in the Finger Lakes of New York, Lake Michigan, Lake Ontario and Lake Huron. The disease affects many salmonid species including: lake trout, Coho salmon (*Oncorhynchus kisutch*), Chinook salmon *(Oncorhynchus tshawytscha)* and Atlantic salmon (*Salmo salar*). The following cases represent most of what has been published on the subject of TD in Great Lakes salmonids but the disease may have occurred more often and may have affected more species because thiamine is not often measured.

Some of the earliest reported cases of a TD related disease were found in the Finger Lakes in New York. Around 1974, it was observed that Atlantic salmon larvae were experiencing mortalities of close to 100% (Fisher et. al. 1996). The behavioral symptoms included erratic swimming, and abnormal response to stimuli; such symptoms indicated a neurological disease. At the time the symptoms were attributed to chemical contaminants. Follow up studies using different contaminants tried to replicate the

behavioral symptoms, but these studies were unsuccessful. As a result, scientists began to consider the salmon's diet as a potential cause of the disease (Fisher et. al. 1996). Whole body thiamine measurements were taken from Atlantic salmon larvae affected by the disease and from control lake larvae. The larvae that were affected had thiamine concentrations near the detection limit of the assay at 100 ng/g wet weight. In contrast, the larvae from reference lakes had thiamine concentrations that were five to ten times higher. Lake trout eggs were assayed as well. A significant inverse relationship was found between thiamine concentration of the eggs and mortality characteristic of the disease (Fisher et. al. 1996). Experiments indicated that treatment with thiamine hydrochloride would reduce or eliminate mortality in Atlantic salmon eggs from Cayuga Lake, one of the Finger Lakes (Wooster et. al. 2000). This information indicated that salmon in the Finger Lakes were suffering from TD and that such a deficiency may have been acquired from the environment.

TD occurs in Lake Michigan as well, although the history of fish populations in Lake Michigan is quite different from the Finger Lakes. In the early 1900s, Lake Michigan supported the largest lake trout fishery in the world. However, in only a few decades and not long after the first reports of sea lamprey in the lake, the abundance of lake trout declined to very low levels (Holey et. al. 1995). As the sea lamprey population exploded, alewife populations began to increase. Because sea lamprey had effectively reduced the top predator population, alewife populations were able to grow unchecked. Subsequently, in the 1960s, a control program was instituted to reduce lamprey populations and this program was initially quite successful. To repatriate lake trout, the lake trout stocking program began in Lake Michigan in 1965 (Holey et. al. 1995).

Despite extensive stocking efforts, natural reproduction continues to be minimal and TD may play an important role (Honeyfield et. al. 2005). Between two to three million lake trout have been stocked into Lake Michigan each year since the late 1960s; however, no substantial recruitment of wild fish to the population has been observed (Bronte et. al. 2008). Egg deposition was recorded on the east and south shores of the lake but wild juvenile fish have yet to be found (Marsden et. al. 2005). Thiamine levels were measured in eggs to determine if TD could be a potential cause of poor recruitment. Thiamine levels in lake trout eggs from females captured at Sturgeon Bay in 1996 measured 2.89 nmol/g (Brown et. al. 2005). Thiamine levels of lake trout eggs collected from females in 2006, at two different sites in the lake, were 0.56nmol/g to 3.95nmol/g (Jaroszewska et. al. 2009). The egg thiamine concentrations reported in Lake Michigan are in the range where lethal and sub-lethal effects of TD have been observed in larvae (Fitzsimons et. al. 2009, Honeyfield et. al. 2005, Carvalho et. al. 2009).

In Lake Michigan, other salmonids were experiencing the effects of TD as well. TD associated mortality in Coho salmon ranged from less than 25% to 70%, over a span of six years. Similar patterns of low to very high mortality caused by TD were observed in Chinook salmon as well. The variation in mortality appeared to depend on the location of the spawning river and the spawning year—suggesting that TD is dependent on local prey availability (Wolgamood et. al. 2005). Coho and Chinook are less sensitive to TD than Lake Trout (Brown et. al. 2005, Fitsimons et. al. 2007).

Lake trout in Lake Ontario have also suffered from TD. In Lake Ontario, it is estimated that 77-100% of lake trout suffered from symptoms of TD from 1994-2003 based on routinely measured egg thiamine concentrations (Fitzsimons et. al. 2007). In

1996, lake trout eggs from females captured at Port Weller averaged 2.5 nmol/g thiamine (Brown et. al. 2005). These levels are in the range of concentrations that result in sub-lethal effects of TD in lake trout larvae (Fitzsimons et. al. 2009).

Salmonids in Lake Huron also appear to suffer from TD, and the situation in Lake Huron indicates a correlation between alewife density and lake trout reproduction. Lake Huron, like the other Laurentian Great Lakes, experienced a crash in lake trout populations that was attributed to the combination of sea lamprey invasion and overfishing. The commercial lake trout yield in Lake Huron from the early 1900s to the 1950s went from over 40 kg per km^2 to virtually nothing (Holey et. al. 1995). Following the lake trout population crash, prey fish populations increased including the alewife (Madenjian et. al. 2008).

Low thiamine levels were observed in several salmonid species occupying Lake Huron. Thiamine levels of lake trout eggs collected in 2001 from Lake Huron ranged from 2.9 to 4.9 nmol/g (Fitzsimons et. al. 2010). Thiamine levels in Chinook eggs were also very low, with concentrations ranging from 0.58 to 1.23 nmol/g over the years 1998-2001. Over the same years, the percentage of Chinook that exhibited symptoms of TD ranged from 17% to 82% (Wolgamood et. al. 2005). Although stocking of lake trout has occurred since the 1960s, it is only recently (2004-2006) that natural reproduction of lake trout in Lake Huron was thought to occur. Natural reproduction rose sharply from 2004 to 2006 and this coincided with a decrease in alewife abundance (Riley et al 2007). More evidence is needed to determine whether the increase in natural reproduction was due to increased egg thiamine or to reduced larvae predation, and/or other reasons.

Other Sources of Early Mortality in Lake Trout

Many factors, other than thiamine status, affect recruitment of lake trout. Factors that are important in early survival of lake trout include: 1) High predation pressure, 2) low numbers of spawning adults and consequently low egg deposition, 3) the ability of stocked fish to find and spawn over good habitat and finally there may also be unknown impediments to lake trout recruitment.

Predation on lake trout eggs has been suggested as a potential cause of recruitment failure in the Great Lakes (Jonas et. al. 2005, Clarmunt et. al. 2005, Savino et. al. 1999, Jones et. al. 1995). The round goby (*Neogobius melanostomus*) and rusty crayfish (*Orconectes rusticus*) are two recently introduced species that prey on eggs. The native sculpin (*Cottus spp.*) and crayfish species (*Orconectes propinquus*) also prey on lake trout eggs. The density of egg predators, on some spawning reefs, has been observed as high as 27/m^2 (Jonas et al. 2005). Such high predator densities could lead to reduced egg survival. A model developed by Savino et al. (1999), to simulate predation mortality, predicted no survivors to the swim up stage at lower predator densities than mentioned here.

It is possible that predation on larvae limits recruitment. Lake trout larvae are consumed by a variety of predators (Savino et. al. 1991, Krueger et. al. 1995, Strakosh et. al. 2005, Riley and Marsden 2009). In Lake Champlain, seven different species were captured with lake trout larvae in their stomachs (Riley and Marsden 2009). Alewife predation could be important because their spring habitat overlaps with that of lake trout larvae (Madenjian et. al. 2008). Alewife consume lake trout larvae in the laboratory and

have been captured from the wild with lake trout larvae in their stomachs (Krueger et. al. 1995, Strakosh et. al. 2005).

Low abundance of spawning adults and subsequently low egg deposition rates are also possible reasons for recruitment failure. In Lake Michigan, spawner abundance was found to be too low and of an age structure such that the fish spawning were too young to produce enough viable eggs for successful recruitment (Bronte et. al. 2007). Egg deposition varies widely from lake to lake and within lakes. High egg density could be very important on spawning reefs and may be required for successful recruitment if there are many egg predators in the vicinity. In 2003, lake trout egg density averaged 7.5/m^2 in Lake Michigan, 454.7/m^2 in Parry Sound (Lake Huron), and 2994.1/m^2 in Lake Champlain. In the spring, larvae were observed in Parry Sound and Lake Champlain but not in Lake Michigan. The density of potential egg predators was more than two times higher in Lake Michigan compared to the other lakes (Jonas et. al. 2005).

The failure of stocked lake trout to locate and spawn over good habitat has been discussed as a possible impediment to successful reproduction as well. Lake trout were found on stocking sites in higher densities than on non-stocked sites during the spawning period in Lake Michigan, indicating lake trout returned to their stocking sites to spawn regardless of the habitat quality (Bronte et. al. 2007). Physical disturbance, such as wave action, has the potential to cause high egg mortality (Fitzsimons et. al. 2007). If lake trout select spawning sites that are unprotected or very shallow, physical disturbance may cause mortality.

In the past decades there has been a shift in the community structure in the Great Lakes, which may also impede lake trout recruitment. These changes have been largely

due to introductions of invasive species, in particular dreissenid mussels and *Bythotrephes* (Dobiesz et. al. 2005). These community changes may produce unknown negative impacts on lake trout survival and reproduction. It is possible that important sources of early life stage mortality may, at the present time, still remain unidentified.

CHAPTER 2

SCALING SUBLETHAL EFFECTS OF THIAMINE DEFFICIENCY TO THE POPULATION LEVEL USING AN IBM

Introduction

Lake trout with low levels of thiamine exhibit impaired behavior which could disrupt feeding and predator avoidance ability. Symptoms of thiamine deficiency (TD) can be subtle to severe in fish and can range from impacting only swimming speeds and response times to loss of equilibrium, failure to feed, hyper-excitability, swimming in a spiral pattern, and death (Brown et. al. 2005, Bronte et. al. 2008, Fitzsimons et. al. 1999). Adult fish are less susceptible to TD because their diet generally contains some level of thiamine. However, reproductively mature females that consume prey items with high concentrations of the thiaminase enzyme tend to produce eggs that have low thiamine levels (Honeyfield et. al. 2005). Unfortunately, because larval lake trout experience relatively long developmental periods during which the larval fish depend only on their yolk sac for substance (Balon 1980), larvae with low thiamine levels at the onset of hatching will have no means to replenish their thiamine until they begin feeding regularly. Such low levels of thiamine during a vulnerable life stage can lead to subtle changes in behavior that may affect survival. This situation deserves consideration because direct mortality from low egg thiamine levels alone cannot explain the continued recruitment failure of lake trout in the Great Lakes (Roseman et. al. 2009). Most reported egg thiamine concentrations from the Great Lakes are not low enough to cause direct mortality but can affect behavior (Brown et al. 2005, Fitzsimons et. al. 2007, Jaroszewska et. al. 2009).

Sub-lethal effects of TD may account for some of the observed lake trout recruitment failure and the assessment of indirect mortality was named as the top research need in a Great Lakes Fishery Commission report on mortality from TD (Brown and Honeyfield 1999). Larval lake trout may not experience direct mortality from low thiamine but low thiamine levels may impair their ability to survive by impacting behavior. Low thiamine levels can lead to reduced prey capture efficiency, reduced growth rate, and possibly reduced predator avoidance (Fitzsimons et. al. 2009, Carvalho et. al. 2009). Recent studies have attempted to quantify the sub-lethal effects of TD in age-0 lake trout at the individual level and have found that low thiamine reduces growth, vision, and the ability of larval lake trout to capture prey (Fitzsimons et. al. 2009, Carvalho et. al. 2009).

Toxicity testing is the most common way to assess the effects of a stressor on an organism and involves measuring the response of that organism to various levels, or doses, of a stressor. The resulting data can be plotted to create a dose-response relationship, usually with the dose on the x-axis and the measured response on the y-axis. Dose-response relationships can take many shapes depending on the mechanism of toxicity and the response measured. The response most often measured is death but it is becoming more common to measure behavioral responses (Walker et. al 2006).

Traditionally developed dose-response relationships may underestimate the response of an organism because the experiments are conducted in an environment very different from that organism's natural environment. Organisms may be much more sensitive to stressors in their natural environments than laboratory developed dose-response curves would indicate because of subtle changes in behavior. Behavior, at the

individual level, has enormous consequences for survival. An organism in its natural environment could be rendered ecologically dead from subtle changes in behavior caused by a stressor long before it is physically. Sub-lethal effects of stress are often not measured in acute dose response studies; the response most often measured is death, likely underestimating the true effects of the stressor. This study aims to include sub-lethal behavioral effects of TD in development of a dose-response relationship for larval lake trout.

Dose-response relationships have been developed to relate thiamine concentration to incidence of TD related mortality (Fitzsimons et. al. 2007, Brown et. al. 2005). The published EC50, or median lethal concentration, for lake trout is 1.57 nmol/g egg thiamine (Fitzsimons et. al. 2007). These relationships, although informative, do not represent the complete response of an organism to low thiamine because community interactions, such as predator prey relationships, were not included. Recently, behavioral metrics have been used to investigate sub-lethal effects of low thiamine in lake trout and it was found that the ability to capture prey, vision, and growth in larval lake trout are negatively influenced by low thiamine (Carvalho et. al. 2009, Fitzsimons et. al. 2009). However, the next challenge is to explore the impact of thiamine deficiency at the population level by incorporating that information into a dose response relationship.

Dose-response curves relating lake trout survival and thiamine concentration have yet to incorporate the sub-lethal effects of TD. Low thiamine levels can cause negative changes in individual behavior and can potentially impair their chance of survival. If sub-lethal effects of TD were incorporated into the dose-response curve it might look very different (Figure 2.1) and result in an EC50 that is much higher than previously thought.

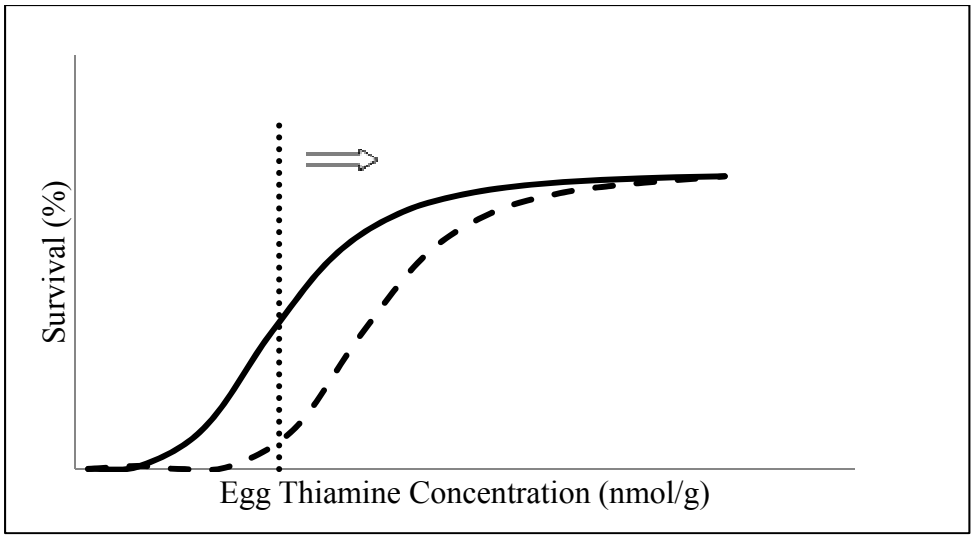

Figure 2.1. Theoretical change in dose-response relationship when the sub-lethal effects of low thiamine are considered. The solid line represents the traditionally developed dose-response relationship, and the dashed line represents the theoretical relation when sub-lethal effects are included. The doted vertical line is the published EC50 at 1.57 nmol/g (Fitzsimons et. al. 2007).

Individual behavior can affect population dynamics, but making the link from individual to population is difficult; often the linkages are indirect and measuring behavior is time consuming. The effects of a stressor on an organism are usually measured at the individual in laboratory studies. However, when species, like lake trout, are managed at the population level it is more useful to know how populations are affected by stressors rather than individuals. Linking organism level responses to population level responses has been a goal for decades but has proved a difficult task (Raimondo and McKenny 2006). Individual based models (IBMs) provide a framework to incorporate behavioral toxicity data and produce more accurate dose-response relationships for wild populations.

The use of an individual based computer simulation provides one avenue to incorporate toxicology data measured on individuals into a population level assessment of the sub-lethal effects of TD. IBMs simulate the interactions of autonomous individuals

and make predictions about the behavior of complex systems. There are many interconnected processes that contribute to early life stage mortality which may vary from year to year and place to place. These complicated interactions often cannot be explained by models like stock recruitment relationships (Letcher et. al. 1996). Individual based models could aid understanding of the sub-lethal effects of TD and the role the disease may play in recruitment of lake trout. IBMs also allow variation in individual traits which is ideal for studying the sub-lethal effects of a stressor like TD. IBMs have been used previously to understand mechanisms controlling larval survival and effects of stressors on early life stages of fish (Murphy et. al. 2008, Letcher et. al. 1996, Rice et. al. 1993). Given the size of the Great Lakes, large lake-wide studies are usually cost prohibitive so computer simulation is an attractive option to study the effects of TD at the population level. Data is now available concerning the sub-lethal effects of low egg thiamine on individual lake trout.

Objectives

My objective for this study is to re-evaluate the thiamine dose-response relationship by incorporating behavioral impairments due to low thiamine and determine if lake trout are more sensitive to low thiamine than previously reported. To accomplish this objective, I modified an IBM that focused on the early life stages of lake trout. The model structure was based on previous IBMs designed to simulate early life stages and evaluate the effects of stress on larval fish (Letcher et. al. 1996, Murphy et. al. 2008). Using the lake trout model, I tested the hypothesis that mortality from low thiamine is underestimated when sub-lethal behavioral effects are not considered. I also examined

how the dose response relationship changes under different amounts of predation pressure and varying predator community structure.

Methods

General Model Description

The IBM was designed to simulate lake trout development from the time of egg deposition in the fall to complete yolk sac absorption in early summer. The simulated physical environment was a shallow (20m), spawning reef composed of rocky substrate. The area simulated was 100m^2 of reef area during the egg predation portion of the model; the volume was increased to 2,000m^3 for the remainder of the simulation. The increase in volume represents the move from the benthos into the water column by larval lake trout at swim-up. The model simulates larval growth over a maximum of 100 days, and each larvae begins the simulation with a mean length of 20mm ± 1.5mm (Krueger et al. 1995) and leaves the simulation when the larvae die or reach 33mm; 33mm is the size at which larval lake trout have absorbed their yolk sac and have begun to feed regularly (Swedberg and Peck 1984). I assumed that once the young trout began to capture prey regularly, thiamine concentration would increase to levels that would not affect their behavior or physiology.

The model is composed of two distinct submodels and includes one submodel related to egg stage development and one submodel that focused on the growth and survival of larval lake trout after hatch. This structure was necessary because the focus of the study was on behavioral effects and behavioral processes had to be isolated. The submodel design allows the egg predation to be bypassed if necessary.

In my model, egg predation occurs first. The eggs that remain after predation are assumed to survive, unless they contain concentrations of thiamine that fall below the threshold for TD induced egg mortality (0.65nmol/g ; Fitzsimons et. al. 2007). Surviving eggs then hatch into free embryos. The larval submodel then follows each larvae through yolk-sac and emergent stages and ends when larvae have fully absorbed their yolk sac and begun feeding regularly. In this second submodel I modified an IBM developed for a general larval fish by Letcher et. al. (1996) where each day the individual larval fish forage, experience predation, and grow or starve based on their success at foraging or utilization of yolk sac. The larval lake trout forage on four types of prey: cladocerans, copepods, chironomids, and mysids. The predators in the simulation are alewife or smelt ranging in size from 60mm to 160mm with maximum consumption rates based on weight. All interactions between organisms in the model are size based (Miller et. al. 1988). After all the surviving individuals have gone through foraging, predator evasion, and growth, the simulation continues to the next day (Figure 2.2) and repeats. Each simulation is set to run for a maximum of 100 days or until all individuals have died or reached the next life stage.

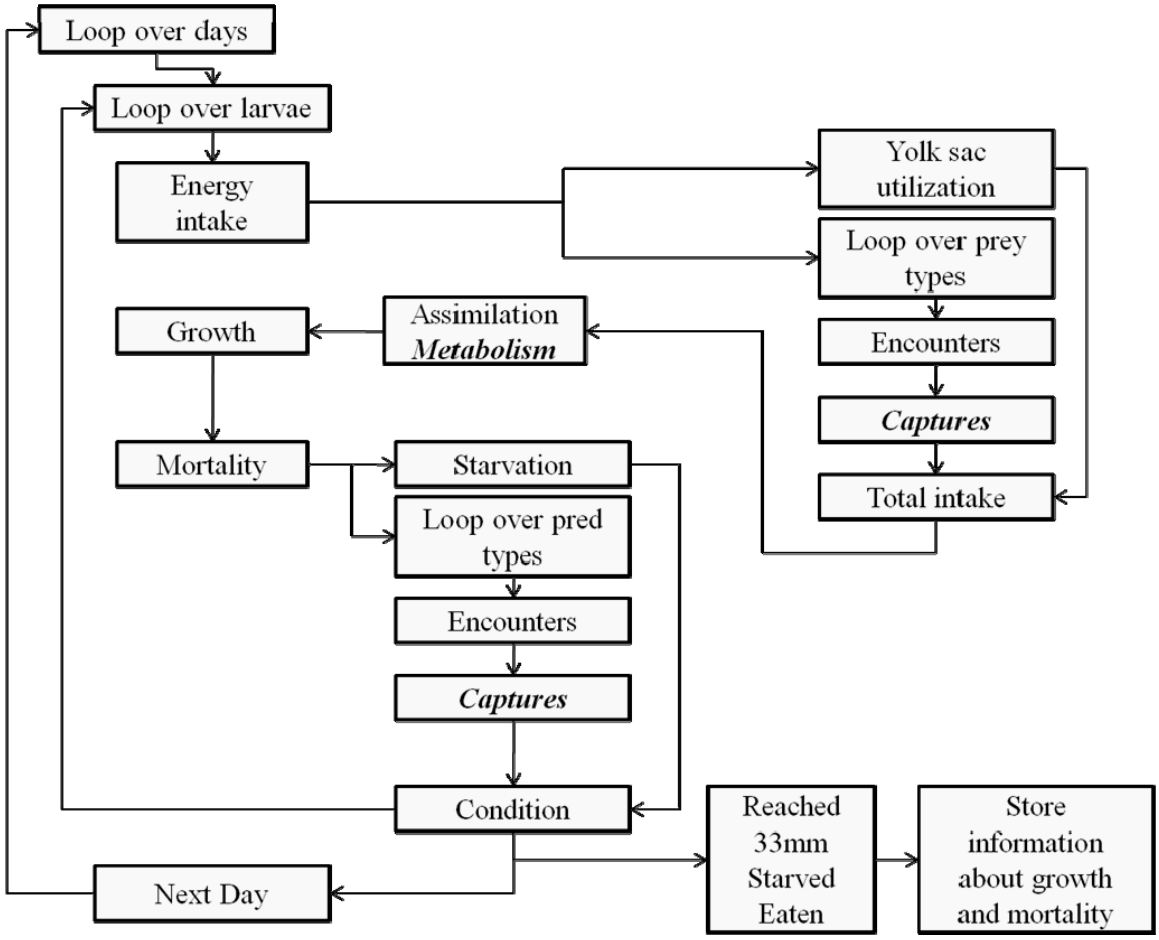

Figure 2.2. Flowchart depicting larval submodel structure. The processes affected by low thiamine are in bold and italics.

Egg Predation Submodel

Egg predation is a potential major source of mortality for lake trout and could lead to recruitment failure (Jonas et. al. 2005, Clarmunt et. al. 2005, Savino et. al. 1999, Jones et. al. 1995). Lake trout typically spawn in October and November across most of their range. The eggs are deposited over rocky substrate where they incubate over winter for a length of time that depends on temperature. The spawning period, for stocks of lake trout, varies in length and can last a week to several months (Balon 1980). The baseline

spawning period in this model was set at 15 days, which is a length that has been observed in onshore areas of Lake Michigan (Ciotti 1973).

In the spring the eggs hatch and lake trout undergo further development as free embryos, where they are dependent on their yolk sacs for sustenance (Balon 1980). Previous studies have modeled egg predation using a deterministic algorithm that estimates survival after predation by interstitial and epibenthic predation with a specified predation duration and egg consumption rate (Savino et al. 1999). I modified the Savino et. al. (1999) model but kept the general framework of a deterministic model with distinct predator guilds.

I included egg predation in my model to understand the effects of different egg predator types and densities on egg predation and to make it a more useful tool in the future. However, the effects of low egg thiamine are most evident in yolk-sac larvae and first feeding lake trout (Fitzsimons et al. 1999, Honeyfield *et al.* 2005) and because my goal was to explore the effects of larval predation in combination with sub-lethal effects of TD, most of the simulations and analysis focused on the period after hatch.

The egg predation subroutine simulates egg consumption by epibenthic and interstitial predators, over the incubation period. Epibenthic predators are those that feed on the bottom but are too large to access small spaces in the substrate, such as white sucker (*Catostomus commersoni*) or round whitefish (*Prosopium cylindraceum*). Interstitial predators are small predator species, like sculpin (*Cottus spp.*), round goby (*Neogobius melanostomus*) or crayfish (*Orconectes spp.*), that can move in substrate spaces and access deposited eggs.

The epibenthic fish predator was modeled as only staying on the reef for the duration of spawning. In Lake Superior, epibenthic predation rates ranged from 0.025 eggs per day to 28.6 eggs per day (Stauffer and Wagner 1979). In my baseline simulations, the epibenthic predation rate was set at a mean value of 1 egg per day per fish for the duration of spawning; this value is equal to the average number of eggs found in round whitefish stomachs (Stauffer and Wagner 1979). The density of epibenthic predators in baseline model runs was 0.5 per meter squared.

The interstitial predator was modeled after round goby, crayfish, and sculpin species; these species are able to move in between the spaces in rocky substrate where lake trout deposit their eggs. Interstitial predator species stay on the reef during and after spawning activity (Jonas et. al. 2005). Egg predation rate, by interstitial predators, has been observed to decrease as the water temperature decreases (Savino et. al. 1999, Ellrott et. al. 2007); therefore, I simulated a slow decline in predation rate to mimic the reduction of predation due to decreased lake temperature. In my simulations, the number of interstitial predators remained constant until day 30 when the predation rate began to decrease slowly to zero at day 80.

Round goby, sculpin, and crayfish exhibit similar maximum larval consumption rates therefore I assumed that all interstitial predator types could be grouped together and use parameters adopted from crayfish studies. Round goby and sculpin predation rates were similar to what was observed for crayfish when foraging over substrate at temperatures less than 10°C; consumption rates ranged from 0.1 eggs per day to 1.7 eggs per day (Fitzsimons et. al. 2006). Native (*Orconectes propinquus*) and exotic (*Orconectes rusticus*) crayfish have average egg consumption rates of 0.1-1.5 eggs per day (Ellrott et.

al. 2007). This rate is lower than the maximum that has been reported in previous studies for mottled sculpin (4.12 eggs per day) and round goby (2.48 eggs per day) that were 60mm in length. Sculpin were able to consume more eggs per day when foraging over level substrate but when foraging over angular or smooth gravel their egg consumption rates did not differ from round goby (Chotkowski and Marsden 1999). Other factors that could influence interstitial predation rates include: temperature, substrate, egg density and competition, however I did not include these factors directly in the model. The knowledge of egg predation over the course of the incubation period is limited due to the difficulties associated with a winter study in the Great Lakes.

In baseline simulations, interstitial predators were simulated at a density of 6 per m^2, which was a moderate estimate based on reported sculpin and crayfish densities in Lake Michigan (Fitzsimons et. al. 2007). In my simulations, mean consumption rate for interstitial predators varied from 0.3eggs/day to 4eggs/day and baseline simulations used a mean consumption rate of 1egg/day. These predation rates are based on laboratory studies of sculpin, crayfish and round goby egg consumption (Chotkowski and Marsden 1999, Savino and Henry 1991, Ellrott et. al. 2007, Fitzsimons et. al. 2006). The rate of predation was kept constant for the first 30 days of egg deposition for interstitial predators. Declining temperatures has been shown to decrease predation (Ellrott et. al. 2007) and although, water temperature was not specifically included in the model, I implicitly included a temperature effect by decreasing predation linearly after the 30 day spawning period with a slope similar to the lake temperature decline (equation 1).

$$(1)\ prate = -0.025 * day + 2$$

Each day of the simulation, each predator is assigned a predation rate that varies randomly with a standard deviation of 0.5, around the assigned mean. Each predator consumes eggs according to its assigned rate and the new number of eggs left remaining at the end of the day is recorded.

Egg predation was examined independent of larval predation so that the sublethal behavioral effects of low thiamine on larval predation could be clearly assessed. The baseline conditions were a starting density of 300 eggs/m^2, 0.5 epibenthic predators per m^2, and 6 interstitial predators per m^2. The average predation rate of the epibenthic predators was 1 egg per day. The average predation rate of interstitial predators was 2 eggs per day but after 30 days the average decreased as described by equation 1.

Larval Submodel

Egg Thiamine and Egg Hatch

To simulate a range of thiamine concentration in eggs, each fish was assigned a random thiamine concentration from a normal distribution with a specified mean and standard deviation of 1 nmol/g. Egg thiamine concentration in each egg is assigned after the incubation period and has no effect on egg predation.

A source of mortality in the model was very low egg thiamine concentration. The published dose response relationship reports 100% mortality at egg thiamine concentrations of 0.65 nmol/g or less (Fitzsimons et. al. 2007). After randomly assigning egg thiamine concentrations, any eggs that had thiamine concentrations of 0.65nmol/g or less were considered dead and were removed from the simulation prior to any larval subroutines. Any eggs remaining after egg predation, with thiamine concentration above 0.65nmol/g, were assumed to hatch into free embryos. The Initial lengths for the hatched

embryos were randomly assigned from a normal distribution with a mean of 20mm and standard deviation of 1.5mm.

Foraging

After absorption of the yolk sac, each individual lake trout larvae performed the daily activities of searching for prey, attacking prey and capturing prey. The foraging subroutine was based on published IBMs described by Letcher et. al. 1996 and Murphy et. al 2008, and all equations came from these studies unless I indicate otherwise. Larval lake trout foraged on four prey types: cladocerans, copepods, chironomids and mysids (Table 2.1). These groups were selected because they comprised most of the larval lake trout diet in Lake Superior (Swedberg and Peck 1984, Hudson et. al. 1995). Copepods were the most abundant organisms, followed by cladocerans, chironomids and mysids respectively. Chironomids and mysids are benthic organisms but the model is not designed to be spatially explicit so prey densities must be entered in meters cubed. To calculate the density of chironomids and mysids, I multiplied published density estimates in meters squared by the total amount of benthic area in the simulation to get number of organisms. I divided the total number of organisms by the total volume of water modeled to get density in number of organisms per liter (Table 2.1.).

Table 2.1. Lake trout prey item density, size, weight and reference.

Prey species	Density (#/l)	Size (mm)	Mass (ug dw)	Reference
copepods	0.25	1	7.7	Dumont et al. 1975, Barbiero et. al. 2001
cladocerans	0.022	1.5	15.81	Barbiero et. al 2001, Bottrell et. al. 1976
chironomids	0.0003	3	31.91	Nalepa et. al. 2007, Benke et. al. 1999
mysids	0.0002	5	324.94	Johannsson et al. 1992

The number of prey encounters was determined by multiplying the area searched each day by prey density. The area searched by individual larval fish is a product of the distance traveled each day and the reactive area. The reactive area, RA, was calculated using the following three equations:

$$(2) \quad 〚RD〛\_(l,i) = \ 〚length〛\_(\ prey\ i)/(2 * \tan(\propto\_l/2)\ )$$

Where RD is the reactive distance and is dependent on prey size and larval fish size. The angle of acuity, α, is calculated using the following equation:

$$〚(3) \quad \propto 〛\_l = 0.0167 \times e\text{^}(\ 〚9.14 - 2.4 \ln(length) + 0.229(\ln(length))〛\text{^}2\ )$$

The reactive area, RA, is defined as the area perceived by the fish and is calculated as a proportion of a circle with a radius of $RD_{l,i}$.

$$〚(4) \quad RA〛\_(l,i) = (\ 〚RD〛\_(l,i)\ )\text{^}2 \times \pi \times 0.4$$

The predicted number of encounters was calculated each day for each prey type. The number of realized encounters was drawn from a Poisson distribution with a mean of the predicted number of encounters.

The ability of larval lake trout to capture prey items depended on the prey size, the larvae size and the individual larvae's thiamine status. As larval fish grew their capture success increased. The equation that describes capture success is as follows:

$$〚(5)\ \ CapSuc〛\_(L,i) = (\ 〚CSnum〛\_i \times\ 〚mass〛\ ^2)/(\ 〚CSden〛\_i +\ 〚mass〛\ ^2\ )$$

Where CapSuc is the probability of a fish, with length= L, successfully capturing a prey item, i. CSnum and CSden are the prey specific capture success numerator and capture success denominator respectively. These values were obtained from Letcher et. al. 1996 or estimated based on McLaughlin et. al. 2000 (Figure 2.3).

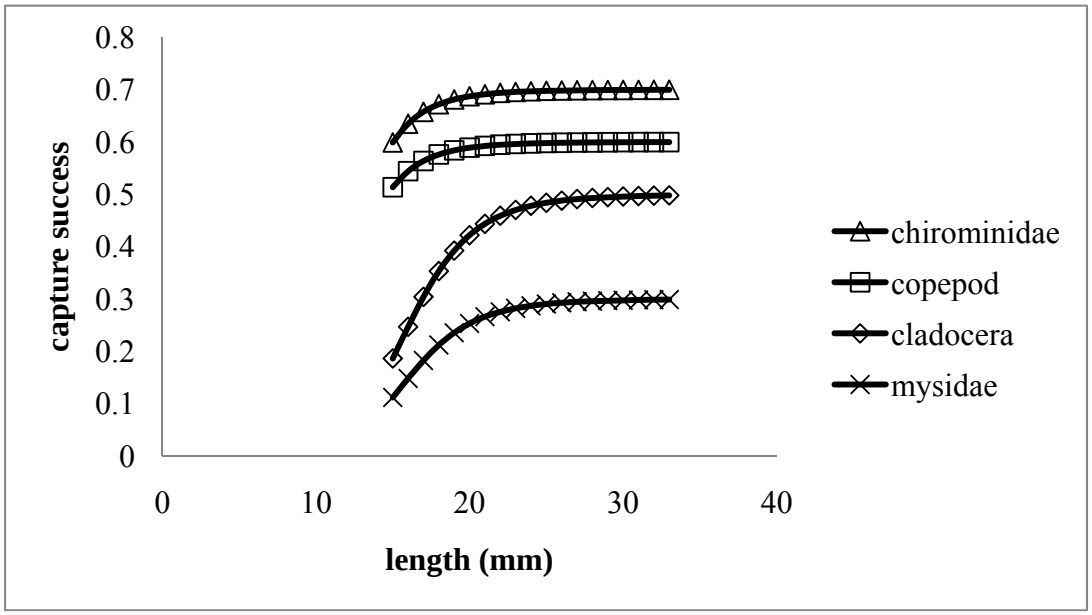

Figure 2.3. The relationship between prey capture success rate of lake trout and lake trout size for all prey types.

In laboratory studies, low egg thiamine concentrations caused a decrease in capture success of prey items, and I simulated this decrease in capture success in the model. Capture success was reduced by 20% and 50% at egg thiamine concentrations of 6.9 nmol/g and 2.9 nmol/g respectively (Fitzsimons et. al. 2009). To include the effects

of reduced capture success in the model, the capture success equation was multiplied by the individual larvae's egg thiamine concentration divided by the no effect concentration (8.5 nmol/g) to produce reductions that were similar to empirical studies (Fitzsimons et. al 2009; Figure 2.4.).

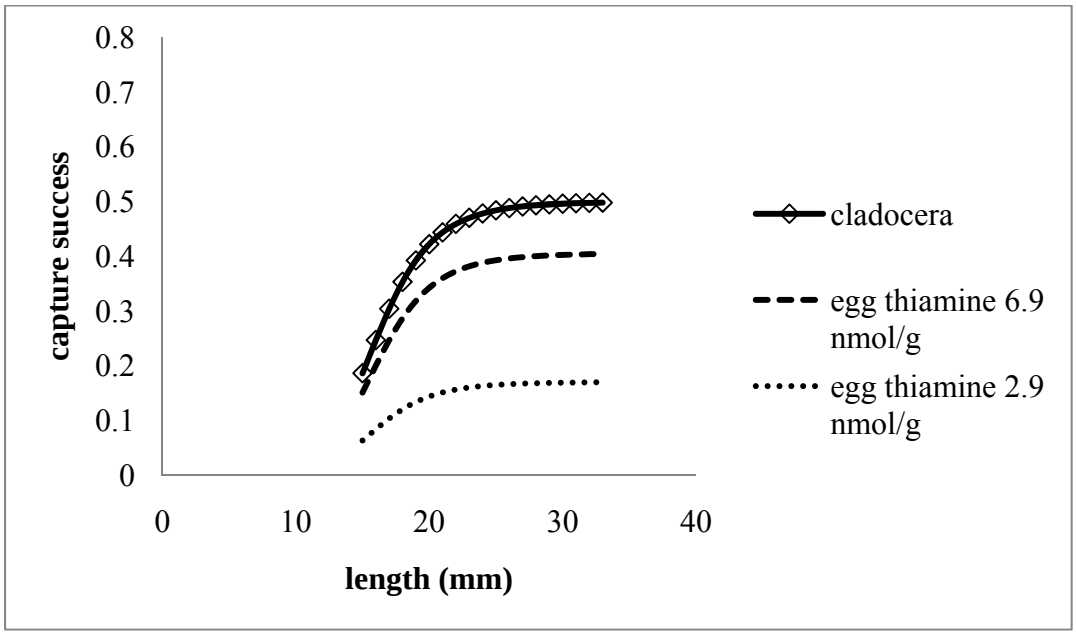

Figure 2.4. Prey capture success rate of lake trout versus lake trout size for cladoceran prey. The line with open diamonds represents capture success of a fish with egg thiamine at or above 8.5nmol/g. The dashed line represents the capture success of a fish with egg thiamine of 6.9 nmol/g and the dotted line represents the capture success of a fish with an egg thiamine concentration of 2.9 nmol/g.

Larval fish in the simulation did not attack all prey they encountered. Optimal foraging theory suggests that fish select prey to optimize their benefit from time and energy expenditure (Charnov 1976, Werner and Hall 1974). To rank prey from most to least profitable we used an algorithm that included mass of the prey, capture success and handling time. To obtain mass per unit time for each prey type the following relation was used:

$$(6) \quad 〚 〚Capture\ sucess〛\_(prey\ i) \times mass〛\_(prey\ i\ )/\ 〚handling\ time〛\_(prey\ i)$$

Handling time was calculated using the formula:

$$〚(7)\ \ HT〛\_(prey\ i\ ) = e\text{^}(0.264 \times\ 〚10〛\ \text{^}(7.0151(preylength/length))\ )$$

The profitability was calculated as the energy gain for a specific prey type divided by the time required to handle it. Prey types were included in the diet of a larval fish until their profitability began to decrease. The number of each prey type captured by a larval fish each day was determined by drawing a random deviate from a binomial distribution.

Growth

Growth before complete yolk-sac absorption was determined by the yolk sac utilization algorithm for larvae under 24mm in length (Jones et.al. 1995), and after yolk-sac absorption, I modeled growth using equations found in Letcher et. al. (1996). There is very little available information on modeling yolk sac utilization. One study by Miller et. al. (1988) developed an equation which predicted time to yolk sac absorption based on size at hatch. However the equation did not adequately represent fish species that inhabit cold water and have long developmental periods like lake trout. The time to yolk absorption, predicted by the equation in Miller et. al. (1988), was nearly half of what has been reported for lake trout (Balon 1980), therefore I modified it as described below. The growth subroutine, after yolk sac utilization, is based on Letcher et. al. 1996, unless otherwise noted.

Energy intake, prior to first feeding, was set as a percentage of maximum consumption. I modeled yolk sac utilization this way because I assumed that the amount of energy used by the larval fish would increase with their size and the amount of yolk

sac metabolized per day would be less than the fish's maximum consumption. If yolk sac utilization is set to maximum consumption then growth rates are higher than reported in the literature for larval lake trout. Once fish grew to 24mm they switched to exogenous feeding. I calibrated maximum consumption to 66% to simulate realistic growth rates for larval lake trout. The maximum amount a larval fish could consume on any given day depended on its weight and was determined by the following relation:

$$(8) \quad Max\ consumption = 2.8275 \times W\text{^}0.8496$$

Where W is the mass of the larvae.

After yolk sac utilization, fish grew according to mass of prey consumed, or ingestion minus maintenance costs. Daily consumption less the maintenance requirement equaled mass of prey consumed that counted towards growth.

$$(9) \quad G = (I \times AE) - TC$$

Where G is growth in ug/day, I is ingestion, AE is assimilation efficiency and TC is total costs.

$$(10) \quad TC = RM + ActvRM \times activp + I(SDA + E)$$

Total costs in ug/day was the sum of resting metabolism in ug/day (RM), active metabolism (ActvRM) times the activity period (activp), and specific dynamic action(SDA) plus egestion (E) multiplied by Ingestion (I). Specific dynamic action and egestion were set as a constant proportion of ingestion with a value of 0.3. The maximum assimilation efficiency was set to 0.6 and was lower than the value of 0.8 used by Letcher et. al. (1996) for the maximum assimilation of a general larval fish, but produced plausible larval growth rates. Assimilation efficiency depended on larval weight and was determined by the function:

$$(11)\quad AE = AE \max\left(1 - 0.25e\text{^}(-0.002(W - 10))\right)$$

All larvae in the simulation began the simulation with an AE=0.6. Resting metabolism was calculated as:

$$(12)\quad rm = (4500 \times W)/(45{,}000 + W)$$

Adequate growth rates were attained using the function above, although the metabolism function was developed for a general larval fish and calibrated to 15° C. Calibrating the metabolism relation to temperatures characteristic of the Great Lakes, between 2°C an 10°C for the period modeled (Beeton et. al. 1999), resulted in daily growth rates much higher than larval lake trout exhibit (Balon 1980). This counterintuitive result was due to the structure of the metabolism equations and the extended period that lake trout rely on their yolk sac for energy. If metabolism is reduced, by lowering temperature, there is more energy available to larval fish for growth, so the growth rates increase. Because the metabolism equation was producing realistic growth rates I left it in its original form. Active metabolism was calculated as 2.5 times the resting metabolism during hours of activity; larvae were active 13 per day, and inactive for the remainder of the day.

Fish with thiamine below the no effect concentration suffered a metabolic cost proportional to their TD. The metabolic cost was determined using the following formula:

$$(13)\quad ((1 - ((thiamine\ ) / (no\ effect\ concentration)))/2 + 1) \times resting\ metabolism$$

The no effect egg thiamine concentration was set at 9 nmol/g. The difference in resting metabolism at egg thiamine concentrations of 5.1 nmol/g, 8.1 nmol/g and 10 nmol/g is relatively small but it does make an impact on individual growth rate (Figure 2.5.).

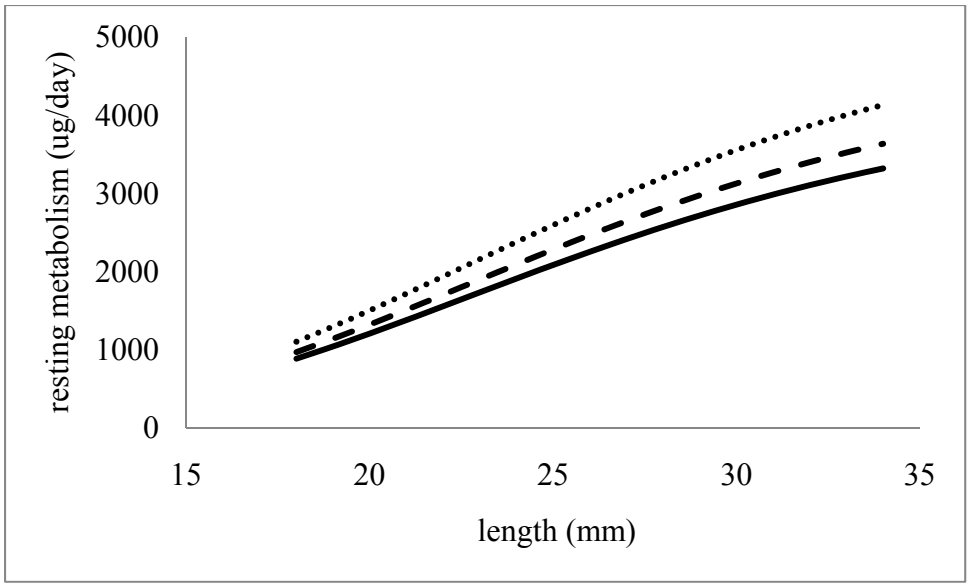

Figure 2.5. Relationship between resting metabolism and larval length. The solid line is resting metabolism of a fish with an egg thiamine concentration above the no effect concentration of 9 nmol/g. The dashed and dotted lines represent the metabolism of larval lake trout with egg thiamine concentrations of 8.1 nmol/g and 5.1 nmol/g respectively.

In laboratory studies a decrease in specific growth rate of 20% occurred at egg thiamine concentrations of 8.1 nmol/g and a decrease in specific growth rate of 50% occurred at an egg thiamine concentrations of 5.1 nmol/g (Fitzsimons et. al. 2009). The reduction in growth rate was attributed to impaired metabolism because the larvae used in the study were not fed during the experiment so the growth rate reduction was not related to prey capture success. Using a no effect concentration of 9 nmol/g in the above equation approximated the reduced growth reported by Fitzsimons et. al. 2009 (Figure 2.6).

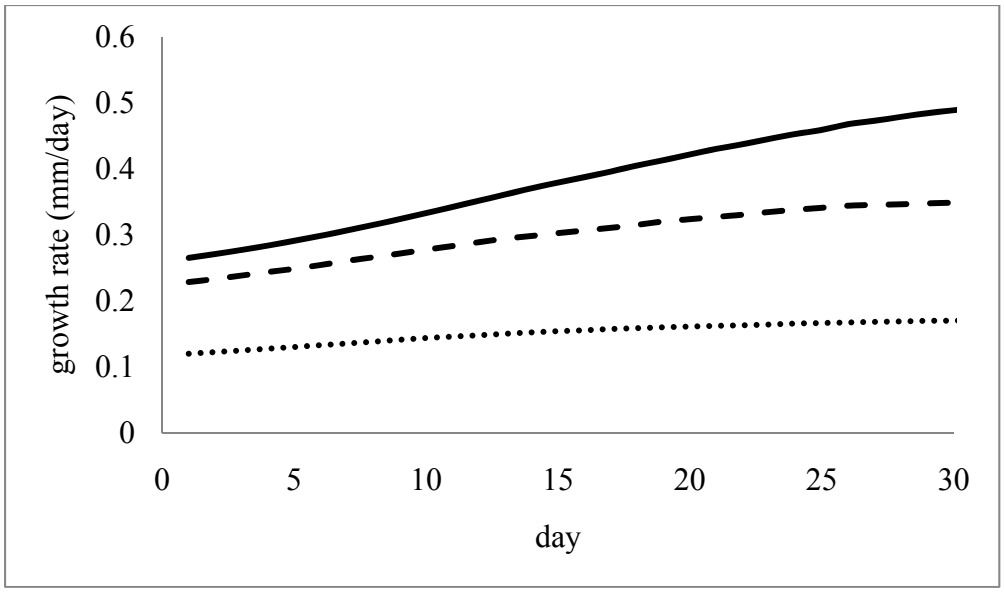

Figure 2.6. Relationship between egg thiamine and growth rate, from one simulation, under baseline and no predation conditions. Each line represents one simulation with all conditions, except egg thiamine concentration, held constant. The solid line is growth rate of a fish with an egg thiamine concentration above the no effect concentration of 9 nmol/g. The dashed and dotted lines represent the growth rate of larval lake trout with egg thiamine concentrations of 8.1 nmol/g and 5.1 nmol/g respectively.

If an individual's weight change was positive, length would be updated using the following length weight relationship:

$$(14)\quad W = 0.1674 \times \llbracket length \rrbracket \text{ ^3.837}$$

This relation was developed as a general relationship for larval fish but described the growth of larval lake trout adequately in the absence of more specific data. Daily ingestion was calculated as total consumption multiplied by assimilation efficiency. Total daily consumption was determined by the foraging subroutine of the model.

Predation

Once larvae hatched, they experienced predation from two types of fish predators, interstitial and pelagic. Predators that occupied interstitial spaces are modeled after a slimy sculpin or round goby. Pelagic predators represent species that feed in the water

column or are unable to access interstitial spaces such as, alewife, burbot (*Lota lota*), yellow perch (*Perca flavescens*) or other fish known to eat lake trout larvae. During the free embryo stage lake trout do not move very much from the rocky substrate thereby reducing pelagic predation. Lake trout in the free embryo stage will also avoid light until just prior to exogenous feeding (Balon 1980). In the month prior to swim up, larval lake trout move off the bottom before they fill their swim bladder, making them susceptible to pelagic fish predation (Kruger et. al. 1995, Baird et. al. 2000). There is experimental data supporting larvae predation from alewife, goby, slimy sculpin, burbot, lake trout and other species during this time (Savino et. al. 1991, Kruger et. al. 1995, Strakosh et. al. 2005). Predation has been an important factor in early life stage survival in other larval fish (Miller et. al. 1988). Other studies suggest that predation on lake trout larvae by alewife could be particularly severe because alewife distribution overlaps with larval lake trout habitat during the first few months after embryos hatch (Medenjian et. al. 2008). In the predation subroutine, all equations, except equation 18, come from Letcher et. al. (1996) or Murphy et. al. (2008).

Successful predation is determined by computing encounter rate and then, if encountered, probability of capture. The realized number of encounters each predator had with larval lake trout was drawn as a random deviate from a Poisson distribution with the calculated encounter rate as the mean. The encounter rate is determined by an equation presented in Bailey and Batty 1983 which is a modified Gerritsen-Strickler formulation where the encounter rate is a function of predator and larval sizes, predator and larval swimming speeds, and the density of predators.

$$(15)\quad ERi = \ \left[\!\left[\pi(R\_L + R\_(P\ ))\right]\!\right]\ \hat{}\ (2\ ) \times (D\_L\hat{}2 + 3D\_P\hat{}2)/\ \left[\!\left[3D\right]\!\right]\ \_P \times (1\hat{}(-9)/Vol)$$

ER_i is the encounter rate of a larvae with a single predator in 24 hours. The realized number of encounters with a predator was drawn from a binomial distribution with probability of success equal to capture success specific to that individual predator and larval lake trout. R_L is the encounter radius for the larval lake trout in mm, R_p is the encounter radius of the predator in mm and is equal to 0.8 times the predator length. Predator lengths were assigned randomly from a normal distribution with a specified mean and standard deviation. The larval encounter radius is calculated as follows:

$$\left[\!\left[(16)\quad R\right]\!\right]\ \_(L =)\ \ (2 \times length)/\pi\hat{}2$$

D_L is distance swum by the larval lake trout in a day and is a product of swimming speed and active period. The distance swum per day depends on lake trout length (Figure 2.7). D_P is the distance swum by a predator in a day and is also a product of swimming speed and active period. Larval lake trout were assumed to be active for 13 hours per day. For a 20 mm larval lake trout, the predicted encounter rate with a 60mm predator is decreased less than one percent if larval activity period is decreased from 13 hours to 1 hour. Increases in predator length had much larger effects on predicted encounter rate. Fish predators were assumed to be active for 12 hours per day throughout the model.

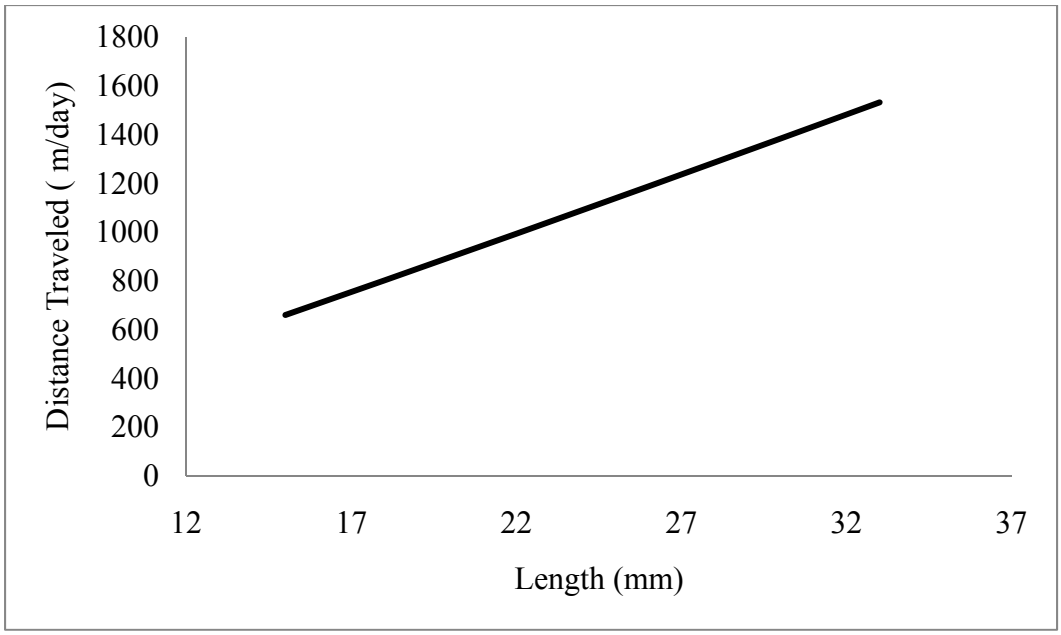

Figure 2.7. Distance traveled by a larval lake trout in a day compared to larva length. Longer distance traveled per day means more encounters with potential prey as well as predators.

Capture success was determined by a relation developed by Miller et. al. 1988. Capture success probability was dependent on the length ratio of predator to prey.

$$(17) \quad CS = 100 - (((pred\ L)/(larval\ L) + 3.37)/44.76)\text{^}(-2.28)$$

The capture success probability was altered by low thiamine. If an individual larval lake trout's initial egg thiamine concentration was below the no effect concentration 8.5 nmol/g then an altered capture success function was employed. The experimental evidence for increased predator capture success is currently very weak. Therefore, I assumed that if lake trout larvae are suffering from neurological symptoms of TD which affect their ability to capture prey, their ability to avoid predators would also be compromised. In one laboratory study, capture success of larval prey by slimy sculpins increased at low egg thiamine concentrations (Fitzsimons et. al. 2009). The altered capture success formula is presented below:

$$(18)\quad CS = 100 - ((((pred\ L)/(larval\ L) * ((1 - \ 〚thicon〛\_larva/(\ 〚thicon〛\_(no\ effect)\ )) + 1)\ ) + 3.37)/44.76)\text{^}(-2.28)$$

Predator capture success decreases with increasing larval size and increases as egg thiamine concentration is reduced (Figure 2.8). If a larvae was encountered, attacked, and captured it was considered eaten and removed from the simulation.

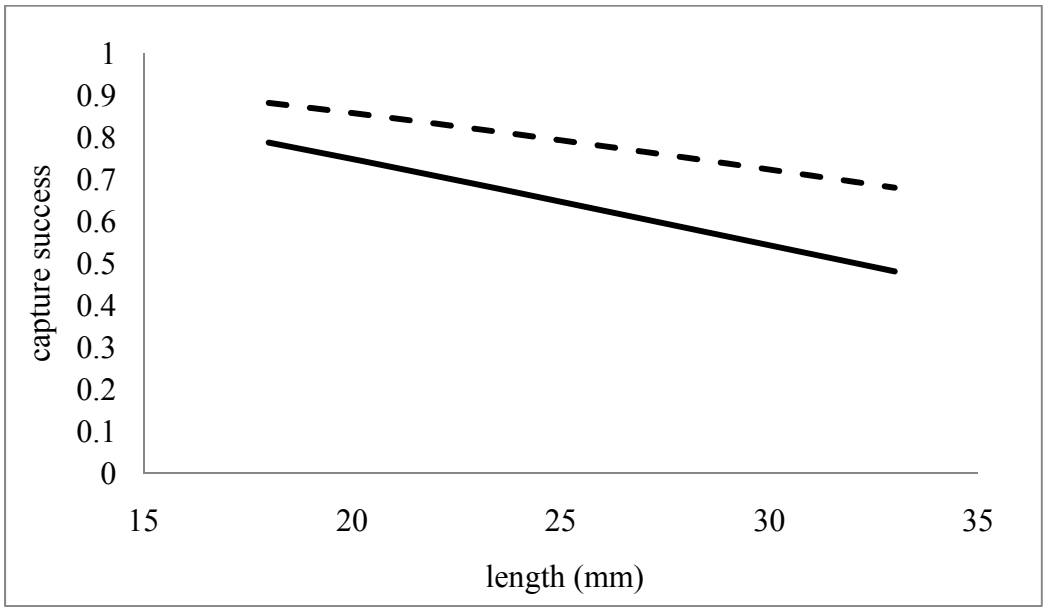

Figure 2.8. Predicted capture success of a 150mm predator versus larval lake trout size. The solid line represents the predator capture success when the larval egg thiamine concentration is above the no effect concentration of 8.5 nmol/g. The dashed line represents predator capture success when egg thiamine is 5nmol/g.

Starvation

If a larval fish lost 25% of the greatest mass it had achieved then it starved to death (Letcher et. al. 1996). If larval fish did not consume enough prey to satisfy their maintenance requirements they lost mass. Fish could only lose weight, not length. Fish that starved were removed from the simulation and the source of mortality and the day of simulation was recorded.

Simulations

The larval stage was the focus of my analysis because the effects of low thiamine are most evident in yolk-sac larvae and first feeding lake trout (Fitzsimons et al. 1999, Honeyfield *et al.* 2005) and a model has been developed previously to determine the effects of egg predation (Savino et al. 1999). Survival, as used here, is defined as lake trout successfully avoiding predation and growing to 33mm within the 100 days of the simulation. A larval lake trout that is still alive after 100 days of simulation but has not yet reached 33mm is not considered a survivor; this particular situation only occurred in the model runs with no predators (Scenario 1). Many fish that had low egg thiamine concentrations in the no predation simulation survived over the 100 day period but did not reach 33mm and therefore were not considered survivors. The effect of predation pressure on the dose-response relationship was also examined. Density of predators and maximum predation rates for each scenario are described in Table 2.2.

Table 2.2. Larval predator simulation information. Pelagic predators were 150mm in length except in the increasing predator size simulations when they were 60, 80, 100, 120, 140 and 160mm in length. Interstitial predator length averaged 60mm.

Scenario Number	Scenario	Pelagic predator density (per/m^3)	Maximum pelagic predation rate (larvae/day)	Interstitial predator density (per m^2)	Maximum interstitial predation rate (larvae/day)
1	Baseline	0	0	0	0
2	Interstitial only	0	0	2	2
3	Pelagic only	.005	18	0	0
4	Both predator types	.005	18	2	2
5	Both predator types	.0025	18	1	2
6	Increasing predator size	.005	2,3,6,10,15,21	0	0
7	Increasing predator density	.0015,.003, .0045,.006,.0075	18	0	0

Predation pressure influences survival and subsequently affects the dose-response relationship. To understand the dynamics of the model without predators, the model was run with no predators through a range of egg thiamine concentrations (Scenario 1). Additional model runs included an interstitial larvae predation scenario (Scenario 2), a pelagic larvae predation scenario (Scenario 3) and a larvae predation

scenario which included both benthic and pelagic predators (Scenario 4 and 5). These different levels of larvae predation generally correspond to different species compositions in the spawning area. Interstitial larvae predation corresponds to predation by round goby or sculpin species. Pelagic larvae predation corresponds to predation by a mix of species, like burbot, alewife and other species known to consume lake trout larvae. The scenario including interstitial and pelagic larvae predation was intended to correspond to predation by a broad range of species including alewife and other pelagic fish predators that may be present on spawning and nursery areas in the spring and early summer. The last two scenarios explored the effect of increasing predator size on larval lake trout survival (Scenario 6) and increasing predator density on larval lake trout survival (Scenario 7).

Each scenario was run 16 times, with an average egg thiamine content starting at 1 and increasing by whole numbers to 16. Predator lengths and initial larval lengths were the same for all 16 simulations. For the first five scenarios, this sequence was repeated three times with a different random number seed each time and I reported survival percentages as an average of the three simulations. The last two scenarios were run through the range of thiamine concentrations only once and with only one initial random number seed. Model parameter values under baseline conditions can be found in Table 2.3.

Baseline – Scenario 1

The baseline (Scenario 1) was simulated to explore the effect of egg thiamine concentration in the absence of predators. Because predation pressure changes the shape of the dose-response relationship; it was important to evaluate the model's performance with no predators.

Table 2.3. Model parameter values under baseline conditions. Low egg thiamine resulted in changes in some parameters including capture success and routine metabolism.

Parameter	Baseline value
Initial number of fish	6000
Number of days in simulation	100
Average initial length (mm)	20
Sd of initial length (mm)	1.5
Length–weight relationship intercept	0.1674
Length–weight relationship exponent	3.837
Predator length (mm)	60-160
Prop. of day available for feeding	13/24
Swimming speed intercept	0.776
Swimming speed exponent	1.07
Prop. of reactive area used for feeding	0.4
Handling time slope	7.0151
Handling time intercept	0.264
Capture success numerator (prey types 1–4)	0.60; 0.50; 0.70; 0.30
Capture success denominator (prey types 1–4)	$5*10^6$; $5*10^7$;$5*10^6$;$5*10^7$
*C*max function intercept	2.8275
*C*max function exponent	0.8496
Maximum assimilation efficiency	0.6
Assimilation efficiency shape parameter	0.002
Routine metabolism numerator	4500
Routine metabolism denominator	45000
Specific dynamic action + egestion	0.3
Activity metabolism multiplier	2.5
Starvation threshold	0.75
Predator's reactive distance multiplier	0.8
Predator's swimming speed multiplier	3.0
Predator's capture success exponent	2.28
Predator's capture success numerator	3.37
Predator's capture success denominator	44.76
Egg death threshold (nmol/g)	<0.65
Egg thiamine concentration no effect (nmol/g)	>9

Interstitial Predation Only – Scenario 2

The interstitial predation simulation (Scenario 2) was based on studies that indicate larvae in rocky substrate are vulnerable to predation by fish that can penetrate those spaces (Chotkowski and Marsden 1999). This interstitial predation only scenario may be representative of some areas in Lake Huron; studies have monitored the decline of many fish populations except the round goby, from 1994-2006 in Lake Huron (Riley et. al.2008). Round gobies are found at higher densities than sculpins on lake trout spawning reefs but begin to consume lake trout eggs at a larger size. The minimum size for egg consumption by round goby was at least 50mm, although sculpin as small as 42mm were found to consume lake trout eggs (Chotkowski and Marsden 1999).

The density of interstitial predators in the natural Great Lakes environment varies depending on the sample site. In Lake Michigan, round gobies were found on spawning reefs at a density of 0.8/m^2 in the fall (Claramunt et. al. 2005). Round gobies have been observed at much higher densities, up to 28 per m^2 in Calumet Harbor, Lake Michigan (Chotkowski and Marsden 1999). The average density of sculpin species on spawning reefs in Lake Michigan ranged from 2.5 to 7.7/m^2 in the fall (Claramunt et. al. 2005). Studies of the benthic fish species composition on lake trout spawning reefs are not often conducted in the spring so, to parameterize my model interstitial predator density estimates were based on studies conducted in the fall. The number of and were set at 200, which corresponds to an interstitial predator density of 2/m^2.

Modeled spring interstitial larvae predator densities were lower than reported in published field studies of interstitial predator densities in the fall (Claramunt et. al.2005, Fitzsimons et. al. 2007, Jonas et. al. 2005). I purposely set these densities low because previous studies suggest that interstitial predators move off lake trout spawning reefs

during the winter. In experimental studies of egg loss over time, most mortality occurred within 2 weeks of egg deposition (Claramunt et. al. 2005). Densities of interstitial predators, capable of consuming lake trout larvae, may be as low or lower than 1-2/m^2 on spawning reefs in the spring.

The interstitial predator's average length and consumption rate were set to imitate a goby or sculpin predator. The average length of an interstitial predator was 60mm with a standard deviation of 15mm which is a size that is larger than the length of round goby and slimy sculpin capable of eating only lake trout eggs (Chotkowski and Marsden 1999). I chose a larger fish size because I assumed that fish of this length would also be able to ingest lake trout larvae. Initial predator lengths were drawn randomly from a normal distribution. If the assigned length was less than 42mm then the length would be reassigned. When predator size was equal to 60mm the capture success of the fish predator dropped to zero when larval lake trout, with thiamine concentrations above the no effect level, reached about 23.5mm.

The maximum consumption for interstitial predators was set at 2 larvae per day, or about 4% of their body weight. Previous studies suggest that lake trout larvae predators would consume about 4% of their body weight per day when feeding continuously over several days (Savino and Henry 1991). Once larval lake trout move off the bottom, inflate their air bladder, and begin to feed they would be much less susceptible to interstitial predators. This change usually occurs when larvae have reached a size of approximately 24mm (Jones et. al. 1995).

Pelagic Predation Only – Scenario 3

The pelagic predation simulation (Scenario 3) was based on the observation that alewife and other pelagic fish predators regularly consume lake trout larvae (Riley and Marsden 2009, Strakosh and Kruger 2005, Krueger et al. 1995). I assumed that as young lake trout move off the substrate, they are consumed by waiting alewife as well as other predators. Scenario 3 was designed to represent the predatory conditions in Lake Michigan from the 1980s and 1990s, before the collapse of alewife populations (Madenjian et. al. 2002, Bunnell et. al. 2006). Simulations for scenario 3 included 10 pelagic fish predators with an average size of 150mm, and a standard deviation of 20mm. These simulated pelagic predators were smaller than the average size of Great Lakes burbot but similar to the average size of alewife captured on or around lake trout spawning reefs (Jonas et. al. 2005, Krueger et. al. 1995). The density of pelagic fish predators is not known with certainty in the spring on spawning reefs but a few predators could consume many larvae. For example, in laboratory experiments, a 250 g burbot was observed to consume 200-300 larvae in a 24 hour period although this feeding rate is not sustained over longer periods. Over extended periods of time, predators consumed about 4% of their body weight per day (Savino and Henry 1991). I arbitrarily assumed that 10 pelagic predators would simulate the predation pressure in the specified volume adequately. The relationship between larvae consumption by predators and predator body weight was used to estimate maximum consumption rates for the pelagic predator. Using length-weight relationships (Carlander 1969) developed for the deep-bodied alewife, and more elongate smelt (*Osmerus mordax*), maximum consumption rates were calculated to fall between 12 and 23, 26mm larvae per day. An average rate, rounded to the nearest

whole number, of 18 larvae per day was used as the maximum daily consumption for pelagic predators averaging 150mm in length.

Both Predator Types – Scenario 4

A simulation that incorporates a combination of both interstitial and pelagic larvae predation (Scenarios 4 and 5) is the most representative of shallow Great Lakes spawning reefs because reefs support a mix of predator types. Simulations for Scenario 4 included interstitial and pelagic predators at densities of 2 per m^2 and .005 per m^3, respectively. The capture success of larval lake trout for a 60mm fish predator is close to zero when larvae successfully grow to 23.5mm in length, but the capture success of larval lake trout for a 150mm predator does not drop below 0.48 for the largest lake trout larvae in simulation. These capture success rates result in a high predation rate throughout the simulation, particularly in the first few days of the simulation when most larval fish are less than 23.5mm in length.

Both Predator Types – Scenario 5

Preliminary simulation runs with both predator types (Scenario 4) resulted in almost no survival at all egg thiamine concentrations. Pelagic predators attained maximum consumption until there was no lake trout left in the simulation, which occurred around day 18. Interstitial type predators consumed larval lake trout at maximum levels until day 6, when their consumption began to drop due to the decreasing number of larvae and the increasing size of larvae. Therefore to identify any interesting trends that may be present when the two predator types were present simultaneously in the simulation, we reduced the number of each predator type by half and ran the simulations, described in Scenario 4, again (Scenario 5).

Predator Size/Larval Survival Relationship – Scenario 6

The relationship between average predator size and larvae survival was explored by varying predator size across the range of thiamine concentrations (Scenario 6). In previous scenarios (Scenarios 2-5), 150mm and 60mm predators were maintained at maximum consumption of 18 and 2 larvae per day respectively. This consumption rate falls within the range of 4% of predator body weight per day. Maximum consumption rates for Scenario 6 simulations were determined by averaging the previously calculated maximum consumption rates for smelt and alewife at lengths of 60, 80, 100, 120, 140 and 160mm. Each set of simulations used a single average predator length, with a standard deviation of 5mm, and maximum consumption rate (Table 2.2). The number of predators was held constant through all the simulations at 10, or a density of .005 per m^3.

Predator Density/Larval Survival Relationship – Scenario 7

The relationship between predator density and larvae survival was explored by varying predator density across the range of thiamine concentrations (Scenario 7). The size of the predator, 150mm, and the maximum daily consumption, 18 larvae, were held constant throughout the simulations. The density of predators was varied from 0 to .0075 per m^3.

Sensitivity and Uncertainty Analysis

To determine which parameters had the most influence on model outputs such as survival, I conducted a Monte Carlo sensitivity and uncertainty analyses. To perform the analyses, model input parameters were drawn from a random distribution for each simulation, 300 times. Parameter values and model outputs were recorded so that, after many simulation runs, the percent variance in model outputs could be correlated to

changes in specific parameters. To ensure that the full range of parameter variance was included in the analyses, I used Latin Hypercube Sampling (LHS). LHS is a stratified sampling technique that divides parameter space into equal sections and ensures samples are included from each (Megrey and Hinckley 2001). The sensitivity analysis involved varying parameters only slightly (coefficient of variation = 1%) to determine local sensitivity to perturbation, The uncertainty analyses selected parameters drawn randomly from uniform distributions (300 times) with mean values equal to values used in the baseline Scenario 1 (Table 2.3, except predation was included as in Scenario 3 and mean thiamine was set to 8 nmol/g) and ranges selected so that the CVs equaled 10% and 25%. I chose to sample from a uniform distribution because the parameters for my model were not well defined (ie. the shape of the distribution for each parameter is unknown).

To determine each parameter's relative influence on survival I performed a correlation analysis using the results from each set of 300 Monte Carlo simulations (SAS version 9.1). The r-squared values indicate how much variance in survival is attributed to each parameter. This technique is commonly used in the development of IBMs to asses which parameters are most important in determining the output of interest (Murphy et. al. 2008, Megrey and Hinckley 2001, Letcher et. al. 1996).

The parameters selected to vary for the sensitivity analyses included previously modeled parameters from Letcher et al, and new additional parameters incorporated to simulate the effects of low thiamine. The IBM described here is very similar to the IBM used by Letcher et. al. (1996) except I calibrated the model to simulate lake trout and I added functions and parameters to relate the larval fishes thiamine status to behavioral and physiological effects. Letcher et. al.(1996) performed a Monte Carlo sensitivity

analysis on key parameters in each submodel of their IBM including: starvation threshold, prey density, total metabolism, larval capture success, predator length, predator density, maximum consumption, assimilation efficiency, search volume and handling time. The parameters selected for my sensitivity and uncertainty analyses include those identified above to ensure consistency between models, and several more that are unique to my model and that affect key processes like larval growth, predation, and foraging. The additional parameters I chose to vary in the sensitivity analysis were: mean thiamine concentration, yolk sac utilization, size at first feeding, and thiamine concentration at which larval lake trout experience no negative effects on prey capture, metabolism, and vulnerability to predators.

Mean thiamine concentration effects survival substantially because the model was designed that way. However, I was interested in precisely how much thiamine concentration influences survival. For my uncertainty analysis, the parameter deviates were randomly drawn from a uniform distribution because lake trout egg thiamine concentrations in the Great Lakes are highly variable with no identified distribution. The range of reported egg thiamine concentrations, over years and lakes, spans from 0.53nmol/g to over 30nmol/g (Brown et. al. 2005, Jaroszewska et. al. 2009, Fitzsimons et. al. 2010).

I suspected that yolk sac utilization had a significant effect on larval survival. Yolk sac utilization determines how fast the fish transition from relying on their yolk sac for energy to capturing prey and, to my knowledge, has not been modeled this way before. There is very little information on yolk sac utilization in lake trout but there is information on growth rate during yolk absorption (Balon 1980). These growth rate data

were used to estimate the baseline yolk sac utilization value of 0.666 percent of maximum consumption. Parameter deviates pertaining to yolk-sac utilization processes in the model were randomly drawn from a uniform distribution.

The size at which lake trout begin capturing prey in the wild occurs across a range of larval sizes, but in my IBM there was only a single, defined size (24 mm) where the lake trout switch from deriving energy from their yolk sac to exogenous feeding. Size at first feeding is important in understanding TD because when larval fish feed they begin to increase their thiamine concentrations. The parameter deviates related to size at first feeding were drawn from a uniform distribution (Balon 1980, Swedberg and Peck 1984, Jones et. al. 1995).

The no effect thiamine concentration was a parameter that determined the magnitude of change in capture success for larval lake trout (Figure 2.4) which may influence survival. The no effect thiamine concentration parameter deviates were also drawn from a uniform distribution. Although there are published data on the thiamine concentration at which lake trout begin to exhibit reduced capture success, there is not enough information to determine if the parameter can be modeled by a normal distribution (Fitzsimons et. al. 2009).

The no effect thiamine concentration parameter that determines the magnitude of change in metabolic cost for larval lake trout (Figure 2.5) is very important when modeling larval growth processes because it affects survival. The no effect thiamine concentration parameter deviates were also drawn randomly from a uniform distribution because there were published data on the concentration at which lake trout begin to

exhibit reduced growth from low thiamine but not enough information to determine if the NOEC follows a normal distribution (Fitzsimons et. al. 2009).

I also chose to vary the no effect thiamine concentration which determines the magnitude of change in vulnerability to predators for larval lake trout (Figure 2.8) in my sensitivity and uncertainty analyses. The parameter deviates associated with vulnerability were randomly drawn from a uniform distribution because there is little published data on the concentration at which lake trout begin to exhibit increased vulnerability to predators due to low thiamine levels.

Results

Egg Predation Results

The simulated egg predation conditions described in the egg predation submodel lead to egg mortality rates that were consistent with field based studies. In my simulations between 76% and 94% of deposited eggs were consumed before hatch, a range that is consistent with what has been observed in the Great Lakes on shallow spawning reefs, although predator densities are much higher than simulated densities in some locations (Jonas et. al. 2005). In my simulations, egg thiamine concentration below 0.65 nmol/g resulted in egg death in the simulation and when the average egg thiamine concentration was set at 3nmol/g, with a standard deviation of 1 nmol/g, there was less than one percent egg mortality from low thiamine.

Simulation Scenario Results

In the baseline simulation (Scenario 1) the only source of mortality was starvation and survival reached 100% after average egg thiamine concentration exceeded 9 nmol/g.

The EC 50 was 6.4 nmol/g and survival showed a typical dose-response curve (Figure 2.9).

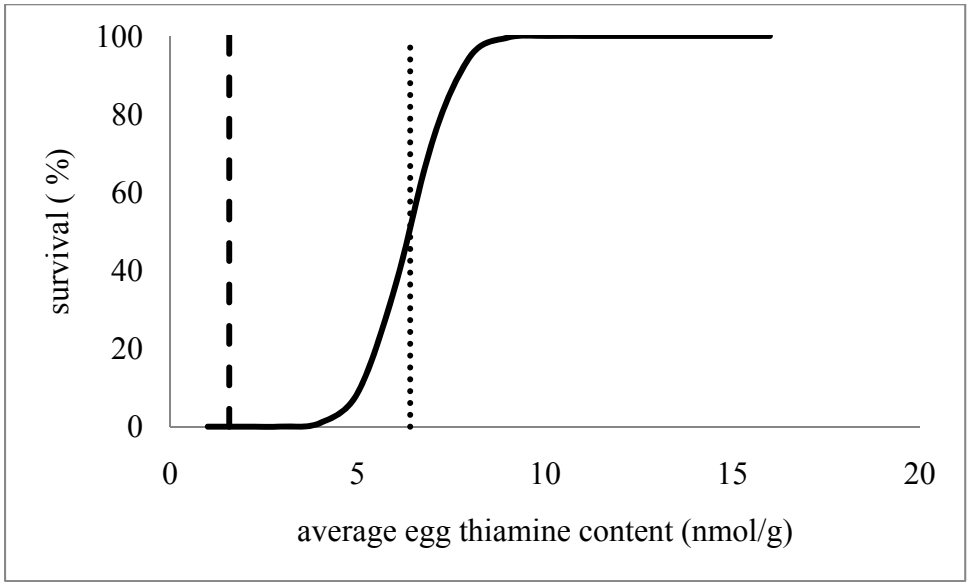

Figure 2.9. Average baseline survival plotted against average egg thiamine concentration. The dashed line represents the published egg thiamine EC50 value of 1.57 nmol/g and the solid line represents the predicted EC50 of 6.4 nmol/g.

In Scenario 1, many larval lake trout with low egg thiamine concentrations did not grow to the endpoint of 33mm. These slow growing larval lake trout avoid starvation but they are not consuming enough prey to grow as fast as other members of their cohort with higher egg thiamine concentrations. As long as the larvae are meeting their basic metabolic needs they will not starve. Starvation mortality begins to occur in simulations with average egg thiamine concentrations of less than 7nmol/g. Starvation mortality greater than 10%, occurs at average egg thiamine concentrations below 5 nmol/g.

Growth rate of larval lake trout showed different patterns that depended on the individual's initial egg thiamine concentration (Figure 2.6) and predation pressure. In simulation runs where average egg thiamine was at or above the no effect concentration, growth rate increased steadily over time. When average egg thiamine levels were below

the no effect concentration, average growth rate started lower and increased only slightly over time. Average growth rate of survivors increased with increasing predation pressure (Table 2.4).

Table 2.4. The average growth rate of simulation survivors with differing amounts of predation pressure and egg thiamine concentrations. Each growth rate represents the results from one simulation. The growth rates increase with increasing predation pressure.

average egg thiamine	growth rate (mm/day) with 5 (150mm) predators	growth rate (mm/day) with 10 (150mm) predators	growth rate (mm/day) with 15 (150mm) predators
12 nmol/g	0.412	0.436	0.466
8 nmol/g	0.374	0.421	0.462
5 nmol/g	0.274	no survivors	no survivors

Mean prey consumption of larval lake trout was dependent on egg thiamine concentration and larval size. In the model, larger fish are able to search more volume and capture prey more successfully compared to the smaller fish. In simulation runs where average egg thiamine was at or above the no effect concentration, mean consumption of prey items increased steadily over time. When average egg thiamine levels were below the no effect concentration, mean consumption started lower and increased only slightly over time (Figure 2.10).

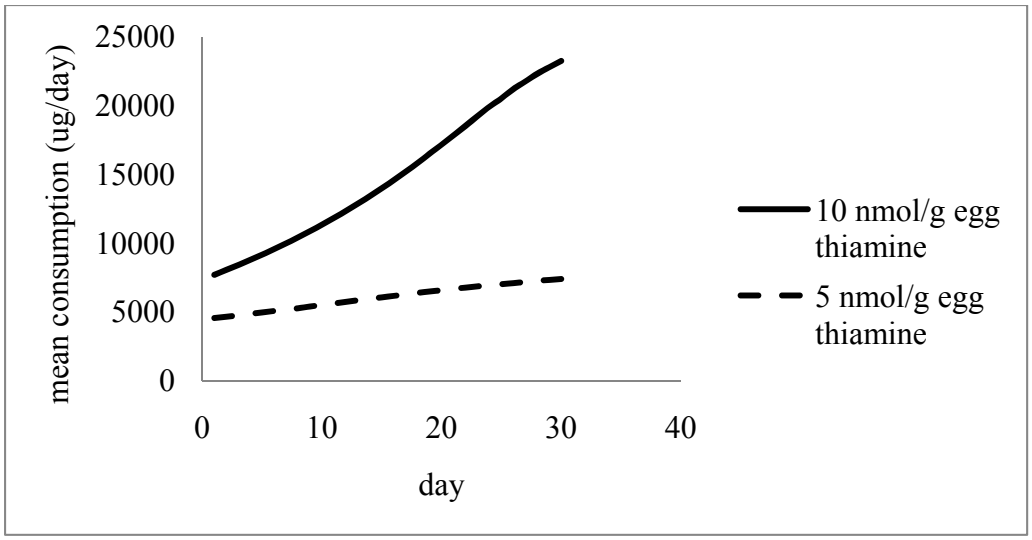

Figure 2.10. Mean consumption of all prey types by larval lake trout over time in 2 runs of the simulation. The solid line is a run with average egg thiamine of 10 nmol/g and the dashed line is a run with the same conditions except an average egg thiamine of 5 nmol/g.

The percent contribution of each prey type to mean consumption changed slightly over time. Trends in mean prey consumption and percent contribution of each prey type were similar for simulations with and without predators (data not shown).

The interstitial predation simulation (Scenario 2) resulted in a maximum survival of 9.7% at egg thiamine concentrations of 12 nmol/g and higher. The EC50 for this scenario was estimated at 8.1 nmol/g (Figure 2.11).

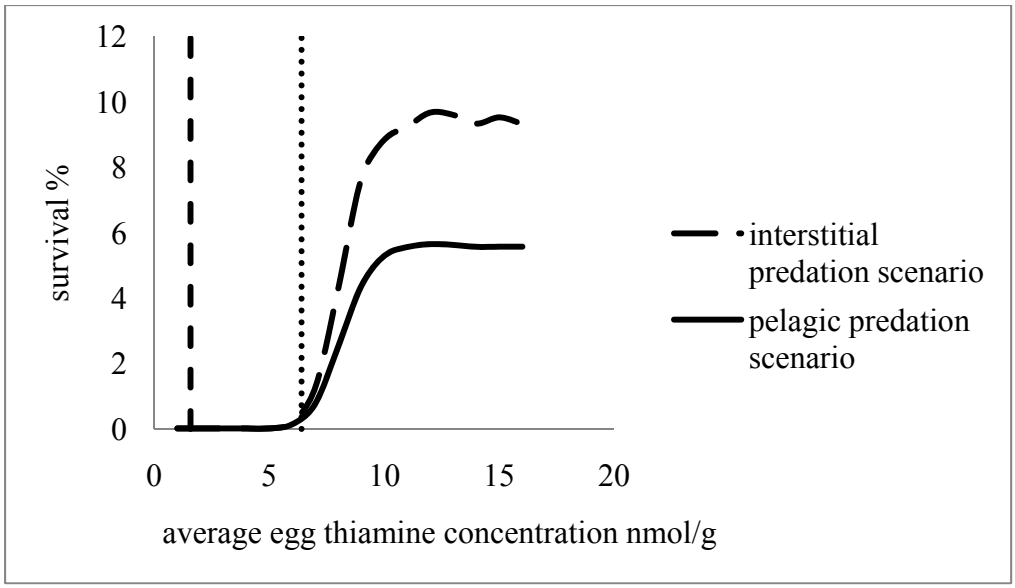

Figure 2.11. Average survival of lake trout in the interstitial and pelagic predation scenarios plotted against average egg thiamine concentration. The dashed line represent the published egg thiamine EC50 value of 1.57 nmol/g. The dotted line represents the EC50 from baseline simulation results with a value of 6.4 nmol/g.

There were no larvae surviving at egg thiamine concentrations of 5 nmol/g or lower. This absence of survival at thiamine concentrations under 5 nmol/g was because larvae experienced such a slow growth rate due to thiamine-impaired behavior that their susceptibility to predators was prolonged. Unlike the no predation scenario, no larval lake trout survived to the end of the simulation (100 days) that were less than 33mm in length. The fluctuating survival percentages, after average thiamine concentrations are above the no effect level, are due to stochastic variations in prey capture and predation processes. Also, as the larvae size increased, the ability of the small interstitial predators to capture larvae declined.

The pelagic predation simulation (Scenario 3) resulted in a maximum survival of 5.6% at egg thiamine concentrations of 11 nmol/g and higher. The EC50 for Scenario 3 was approximately 8.2 nmol/g (Figure 2.11). No larvae survived at egg thiamine

concentrations that were lower than 5 nmol/g. Pelagic predators consumed larvae at maximum levels (18 larvae per day for predators with a length of 150mm) until all the lake trout had been consumed or reached 33mm. Pelagic predators encountered more larval lake trout than they could consume each day. Pelagic predators were better at capturing larval lake trout than interstitial predators, simply because they were bigger.

When both predator types (interstitial and pelagic) were included in the simulation (Scenario 4) the result was 0% survival at egg thiamine concentrations of 8 nmol/g or lower and less than 0.5% to 0% survival at all other thiamine concentrations . Pelagic predators consumed their maximum assigned number of larvae per day until there were until there was no larval lake trout left in the simulation; this point was reached around day 18. Interstitial type predators consumed larval lake trout at their maximum levels until day 6, when their consumption began to drop due to the decreasing number of larvae and the larvae's increased size.

When both predator types were included at half their previous densities (Scenario 5) the result was a maximum survival of 6%, reached at egg thiamine concentrations equal to or greater than 11 nmol/g. No lake trout survived at egg thiamine concentrations less than 6nmol/g. For Scenario 5, the EC 50 was approximately 9.8 nmol/g (Figure 2.12).

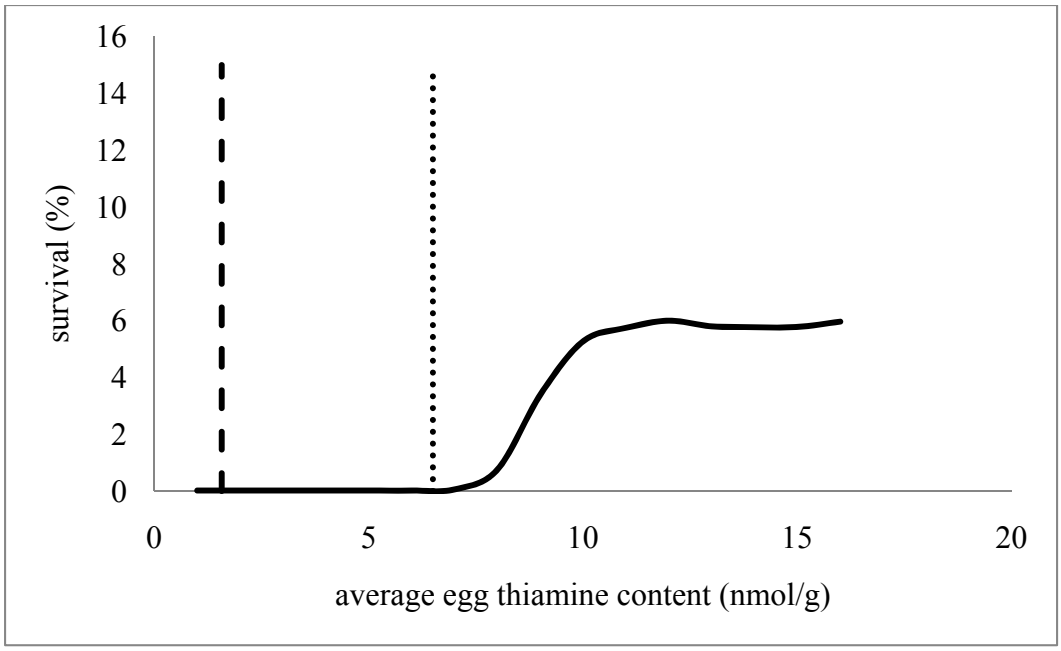

Figure 2.12. Average survival under predation from interstitial and pelagic predators, at half density of previous simulations, plotted against thiamine concentration. The dashed line represent the published egg thiamine EC50 value of 1.57 nmol/g. The dotted line represents the EC50 from baseline simulation results with a value of 6.4 nmol/g.

In Scenario 5, pelagic predators were able to sustain maximum consumption until there were less than 75 lake trout left in the simulation. Interstitial predators ate larvae at maximum rates until approximately day 14, when larvae, on average, were too large for the interstitial predators to capture (at average egg thiamine above no effect concentrations). This period of time where interstitial predators ate at maximum levels was extended when average egg thiamine levels were below the no effect concentration.

As simulated predation pressure increased in my model, the dose-response curve between survival and thiamine concentrations shifted to the right. Simulation results indicate that larval lake trout are more sensitive to low thiamine when predation pressure is high (Figure 2.13). Although these simulations cannot predict actual situations in the Great Lakes, they do indicate that moderate predation in addition to TD can significantly reduce YOY survival. Predators in the model were limited by their maximum

consumption rather than their ability to locate larval lake trout prey; my model concentrated larval lake trout into a small area which made it easy for predators to encounter larval lake trout. Predators, at moderate densities, were able to consume all larval lake trout in the simulations, independent of the average egg thiamine concentration.

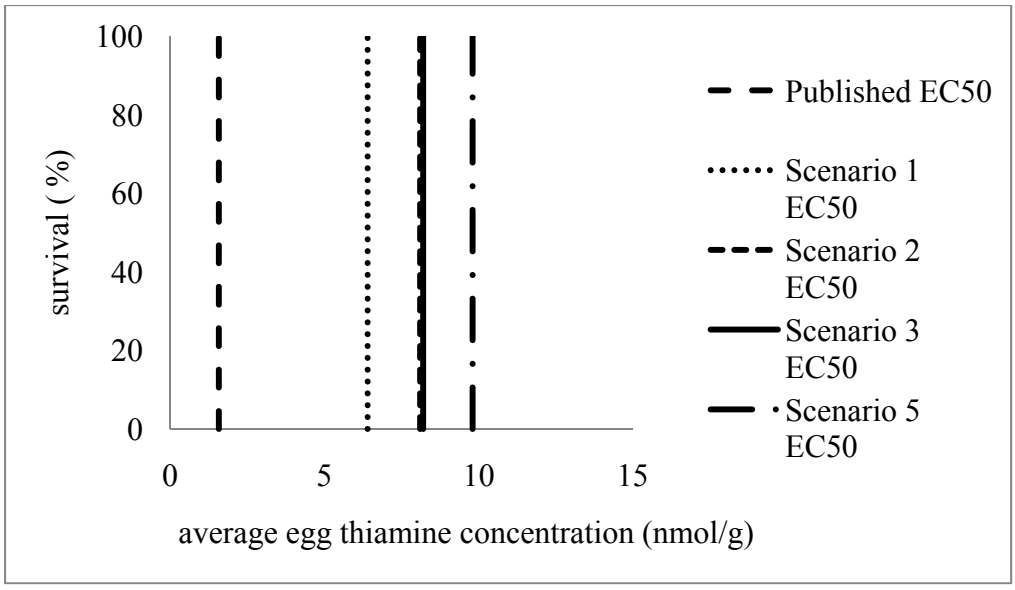

Figure 2.13. A summary of EC50 values from simulation Scenarios 1-5. As predation pressure increases in the simulation larval lake trout become more sensitive to low thiamine.

In simulations where predator size and subsequently the maximum consumption was varied (Scenario 6), I found that average predator size had substantial effects on larval survival and growth rate. The maximum survival decreased as predator size increased (Figure 2.14). For example, when average predator size was 60 mm, maximum survival was almost 93%; however, when average predator size was increased to 160 mm, the maximum survival dropped dramatically to 2% (Figure 2.14). The average growth rate of larval survivors increased with predator size, due to size selective

mortality. The average time for a larval fish to the end length of 33 mm decreased as average predator size increased.

Predator size and egg thiamine concentration interacted to affect survival. When average egg thiamine was 8nmol/g or higher, an increase in predator length of 10mm resulted in a greater reduction in survival than a 1 nmol/g decrease in average egg thiamine, in most cases. At thiamine concentrations of 7 nmol/g and below, reducing average egg thiamine concentration thiamine concentration by 1 nmol/g results in a larger drop in survival than keeping average egg thiamine concentrations the same and increasing predator size by 10mm. For example, when average egg thiamine was 9 nmol/g and predator size was 100 mm predicted survival equals 54.3%; when predator size was increased to 120 mm predicted survival is 30.7% but if predator size remained 100mm and thiamine concentration was reduced to 8 nmol/g predicted survival was 44.2%. Once average egg thiamine was above 7 nmol/g, predator size became more important in determining larval survival than average egg thiamine.

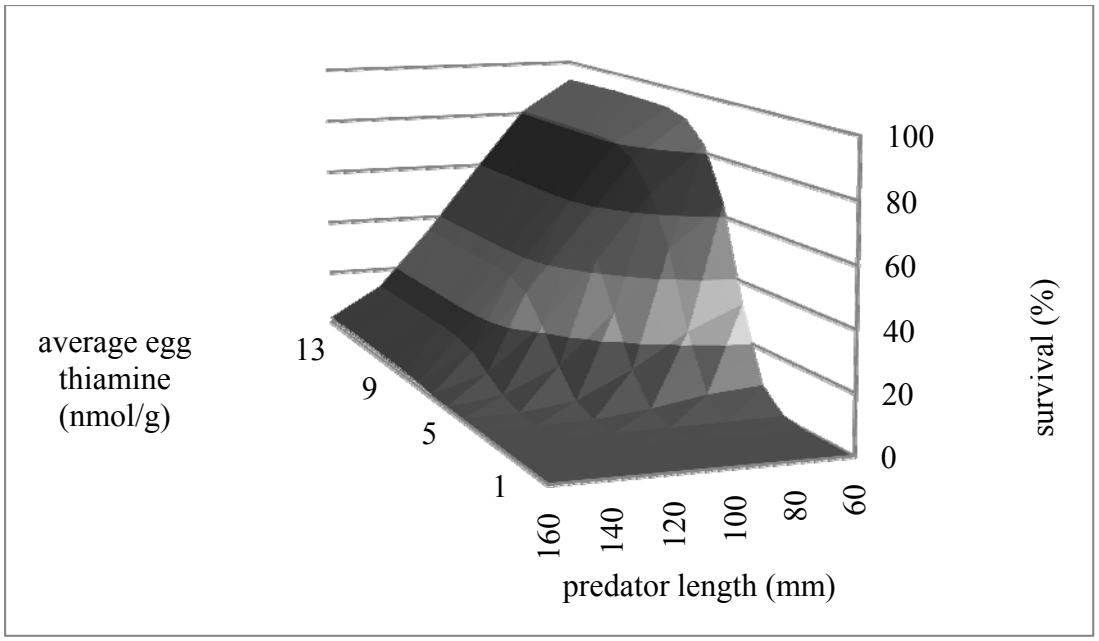

Figure 2.14. Surface plot representing the relationship between average egg thiamine, predator length and survival (Scenario 6).

In Scenario 7, where I varied the predator density along with the thiamine concentration, the predator density and thiamine concentration interacted to have a substantial effect on larval survival (Figure 2.15). When there were no predators in the simulation the maximum larval survival was 100%. When predator density was 0.0075/m^3, survival dropped to a low level of 0.18%. Average growth rate of survivors increased with predator density. The average time it took surviving larval fish to reach 33mm decreased as average predator density increased (data not shown).

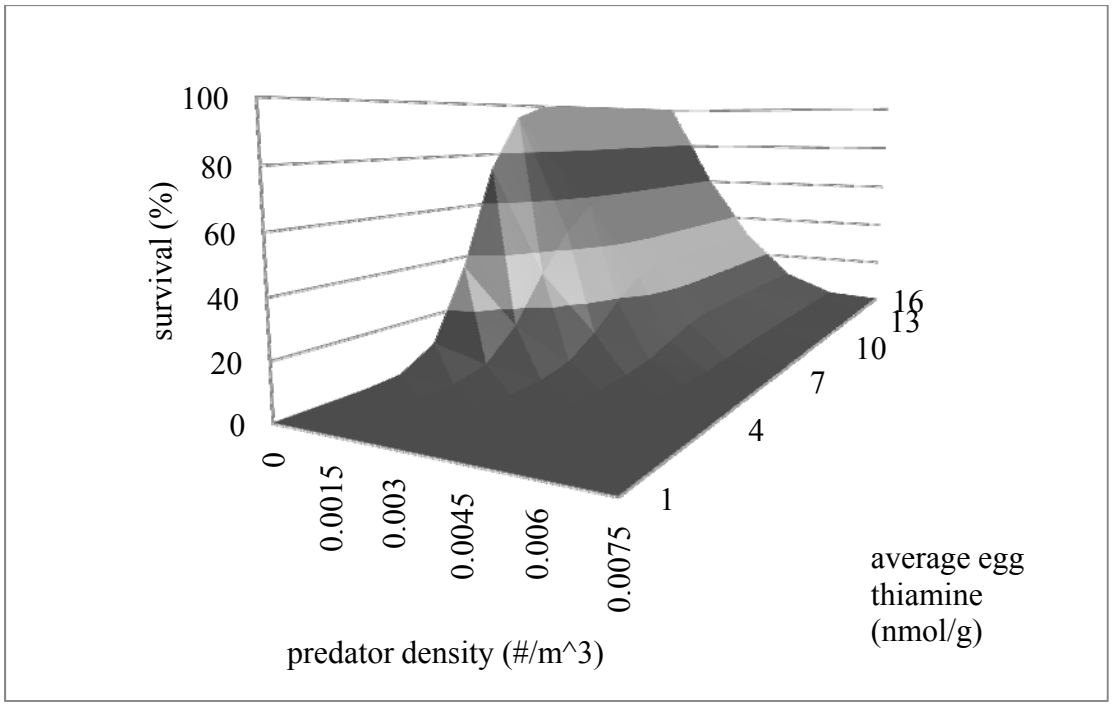

Figure 2.15. Surface plot representing the relationship between average egg thiamine, predator density and survival (Scenario 7).

Predator density and average egg thiamine interacted to affect survival. The relationship between average egg thiamine and predator density was very similar to the relationship between thiamine and predator size. Initial average egg thiamine had a larger effect on predicted survival when thiamine concentrations were around 8 nmol/g and lower; however, when average egg thiamine concentrations were above that threshold predator density had a stronger effect on predicted survival than thiamine concentration. Predator density was increased by multiples of 0.0015 fish/m^3 in Scenario 7. This differs from Scenario 6 where predator size was increased incrementally by the same amount (10mm), although the shape of the surface plots from Scenario 6 and Scenario 7 were very similar (Figures 2.14 and 2.15).

The growth rate of larval lake trout was very important in survival. Fish with high growth rates were vulnerable to predators for shorter lengths of time. Larger larvae had

greater predator escape probabilities which allowed them to avoid predation more often when attacked. Larger larvae were also more likely to capture the prey they encountered. Fish that survived, under predation pressure, tended to have above average initial lengths and consequently had faster growth rates (data not shown).

Sensitivity and Uncertainty Analyses Results

Only a few model parameters accounted for most of the percent variance in predicted survival. Predator length, total metabolism, and assimilation efficiency explained the most variance in survival of all the parameters tested in the sensitivity and uncertainty analysis. Larval prey capture success, search volume, maximum consumption, size at first feeding, and mean thiamine concentration were the other parameters that explained 2% to 6% of the variance in survival (Table 2.5).

Table 2.5. Percent variance in predicted survival explained by model parameters in each of the Monte Carlo simulations. Prey density and larval capture success represent groups of parameters due to a variety of prey and different capture success for each species.

Parameter	CV 1%	CV 10%	CV 25%
Mean thiamine concentration	3.39	4.20	2.02
Yolk sac utilization	0.27	0.36	0.56
Size at first feeding	3.17	2.43	1.00
Prey capture no effect thiamine concentration	0.42	0.62	0.07
Metabolism no effect thiamine concentration	2.22	1.21	0.34
Predator avoidance no effect thiamine concentration	0.31	0.04	0.09
Starvation threshold	0.66	0.22	0.20
Prey density	1.07	0.63	0.35
Total metabolism	19.45	14.29	15.13
Larval prey capture success	5.97	4.52	5.90

Table 2.5 (cont'd)

Predator length	26.01	21.34	19.89
Predator density	0.01	3.50	4.24
Maximum consumption	3.53	3.17	3.61
Assimilation efficiency	19.98	11.29	3.57
Search volume	4.58	1.06	0.56
Handling time	0.09	0.00	0.28

Discussion

TD and Survival

Incorporating the sublethal behavioral effects of TD into a dose response relationship between thiamine and larval lake trout survival using an IBM indicated that lake trout may be more sensitive to thiamine deficiency than a traditionally developed dose-response relation would suggest (Figures 2.9, 2.11, 2.12, 2.13). My IBM also predicted that the shape of the dose response curve will change according to the amount of predation pressure. As predation pressure increased the larval lake trout became more sensitive to low thiamine as indicated by increased EC50 values (Figure 2.13). These findings may suggest alternate reasons for recruitment failure in the Great Lakes; average egg thiamine levels reported around the Great Lakes are not sufficient to overtly kill significant quantities of larval lake trout but low thiamine may intensify the effects of other stressors like predation. My simulations also indicate other important factors in larval survival, including: egg deposition, egg and larvae predation, individual growth rate, and demonstrate that the only comprehensive way to evaluate response to a stressor on a wild population is to consider all the effects that stressor has on survival.

When lake trout suffer the effects of thiamine deficiency in combination with egg and larval predation virtually no larvae survive in model simulations. In the absence of predation, simulated larval lake trout with low thiamine concentrations are still able to survive. Predation is a serious impediment to larval lake trout survival (as indicated by sensitivity and uncertainty analysis) and predation alone could eliminate survival in simulations where average egg thiamine is above the no effect concentration.

Although lake trout recruitment in the Great Lakes is nearly non-existent outside of Lake Superior and Parry Sound and eggs have varying levels of thiamine (Fitzsimons et. al. 2010, Jaroszewska et. al. 2009, Brown et. al. 2005), successful lake trout reproduction has been documented in a Keuka Lake, one of the finger lakes in New York, where measured egg thiamine concentrations are quite low (averaged 3.8 nmol/g in 2000; Fitzsimons et. al. 2005). Alewives are present in the lake and were assumed to be the source of thiaminase in the lake trout diet. The average egg thiamine concentration measured in Keuka Lake is relatively close to my estimated EC50 (6.4 nmol/g) in simulations run with no predation. In these no predation simulations, survival began to occur at average egg thiamine concentrations between 3 and 4 nmol/g. The relatively low abundance of egg predators in this lake may contribute to successful reproduction at such low egg thiamine levels. Additionally, egg deposition occurs later in the season compared to the Great Lakes, which may also improve survival rates by reducing predation. Also, because the model suggested that as egg deposition rates increased, survival increased, egg deposition rates in Keuka Lake, which are over 4 times higher than the rates used in my simulations, may be contributing to successful recruitment in that system (Fitzsimons et. al. 2005).

The Keuka Lake study is encouraging for those interested in lake trout rehabilitation in the Great Lakes. It suggests successful reproduction and recruitment can occur even under low egg thiamine egg conditions, providing that egg predation is low and egg deposition is high. The time between deposition and hatch may also be important in survival and although this was not investigated in our study it could easily be explored using the IBM framework.

Predation and survival

The results of the baseline simulation (Scenario 1, Figure 2.9), indicated that in the absence of predation, survival begins to occur when egg thiamine concentrations are 4 nmol/g or higher. This scenario is difficult to compare to natural conditions because there are almost always predators in natural systems. The previously mentioned study from Keuka Lake determined that lake trout successfully reproduced with egg thiamine concentrations averaging 3.8 nmol/g, under low predation conditions (Fitzsimons et. al. 2005). In other areas of the Great Lakes, successful reproduction has not been observed when egg thiamine concentrations are at similar levels (Brown et. al. 2005, Jaroszewska et. al. 2009). Unfortunately the mortality from predation and TD are impossible to separate in the field.

The interstitial predation only and the pelagic predation only simulation runs had similar EC50s (Scenario 2 and Scenario 3, Figure 2.11) although they had very different potential maximum predation rates. The maximum survival percentage was higher in scenario 2 (interstitial predation only). In the interstitial predation simulations there were a total of 200 predators 60mm in length that could potentially consume 400 larvae per day. In pelagic predation simulations (Scenario 3) there were 10 predators of 150mm that

could potentially consume 180 larvae per day. The pelagic predators searched more area and had higher prey capture success due to their size. The interstitial predators were limited to consuming larval lake trout less than 24mm in length, which probably led to the higher maximum survival in the interstitial predation runs (Scenario 2). Larval lake trout in Scenario 2 experienced high interstitial predation mortality early in the simulation, while lake trout in Scenario 3 experienced a constant level of pelagic predation mortality throughout the simulation.

The size of interstitial predators limited their ability to capture larger lake trout. When predator size was equal to 60mm the capture success of the fish predator dropped to zero when larval lake trout with thiamine concentrations above the no effect level reached about 23.5mm in length. Benthic predators consumed lake trout at maximum levels until day 15 in most simulations; after day 15 is when larval numbers are low and the average size of larval lake trout is over 23.5mm. In the interstitial simulations, as soon as most of the larvae grew past 23.5mm in length, these larger larvae had a higher survival rate. This capture success relationship between a larval predator and larval lake trout is consistent with what might occur in the wild after swim up; the larval lake trout would be much less susceptible to interstitial predators because they are off the bottom. Their susceptibility to pelagic predators may increase after swim up. This increase in susceptibility to pelagic predators after swim up was not reflected in the model specifically. The simulated pelagic predators were larger so their capture success was higher across all sizes of larval prey (Figure 2.16).

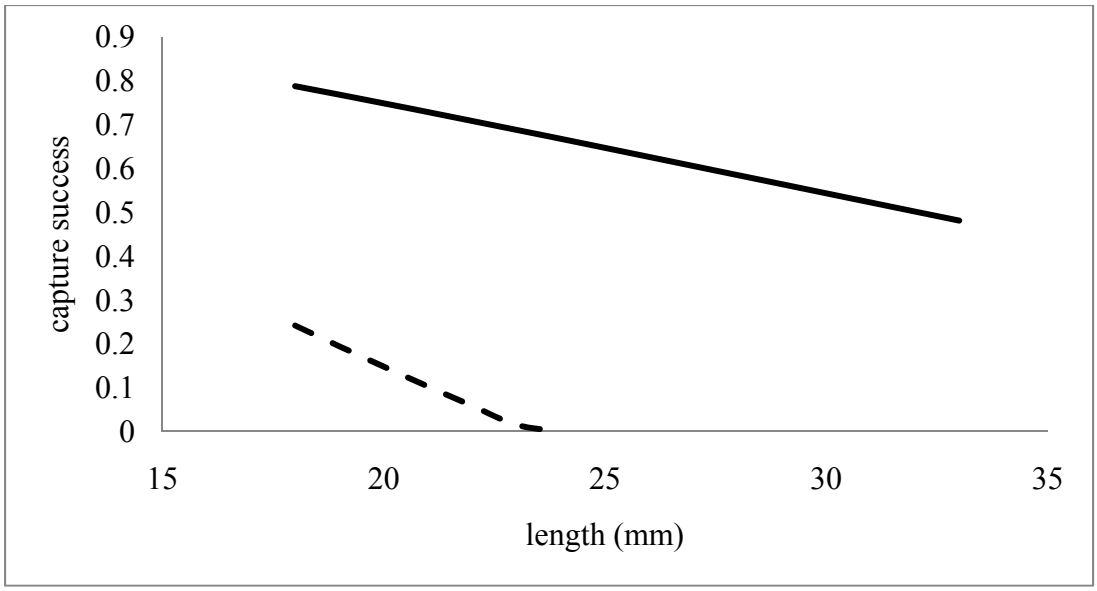

Figure 2.16. This figure illustrates the difference in capture success of a 150mm predator, solid line, and a 60mm predator, dashed line.

When both predator types were present in simulation (Scenario 4), a scenario that was designed to replicate conditions in the Great Lakes, essentially no survival of larval lake trout was observed. In the end, there were only 2 surviving lake trout in all simulations. The potential maximum larvae consumption of 580 per day was too high for survival to occur.

The set of simulations that included both predator types with half the number of each predator compared to previous simulations (Scenario 5) suggests that higher predation pressure renders larval lake trout more sensitive to the effects of low egg thiamine. The scenario with both predators at half density had the highest EC50 value but not the lowest maximum survival (Figure 2.12).

The simulations where the predator size was changed along with thiamine (Scenario 6 and Figure 2.14) were designed to explore the relationship of thiamine to predator size and survival in the model. If egg thiamine was high then predator size could still be high and still ensure survival of lake trout larvae. However, once egg thiamine

dropped below 5 nmol/g, survival dropped substantially regardless of the predator size. Simulation results indicated that if predators larger than 160mm feed on larval lake trout then survival may be reduced to zero at all thiamine concentrations. Predators larger than 160mm are known to consume lake trout larvae and occupy the same habitat (Stauffer and Wagner 1979). It is possible in natural systems that have predators larger than 160mm on and around lake trout spawning reefs, that survival of larval lake trout could be eliminated, particularly if larvae occurred at densities simulated in the model.

The set of simulations where predator density was varied was constructed to explore how the number of predators and egg thiamine concentration interact to influence larval survival (Scenario 7 and Figure 2.15). Predator density influenced survival greatly. Survival dropped from over 30% to less than 10% when predator density was increased from .003 to .0045/m^3. If predator density was above .0075/m^3 survival was reduced to zero at all thiamine concentrations. A predator density of .0075/m^3 means that there were 15 predators over 100m^2 lake area that was 20m deep. These predator densities seem marginal but they were high enough to eliminate nearly all the larval lake trout survival in simulations.

Predator size and density effect survival substantially (Scenarios 6 and 7) and the magnitude of the effect depends on thiamine concentration (Figures 2.14 and 2.15). Egg thiamine concentration drove mortality at predator sizes less than 160mm and densities less than .006/m^3. Once predator size and density were above these points survival was extremely low or zero, regardless of egg thiamine concentration. If predator density and size were low then egg thiamine concentration was very important in survival but its importance decreased as predator density and size increased.

Evaluation of IBM method

Simulations exploring larval survival under a range of egg thiamine concentrations and predation pressure were the focus of this analysis. Simulation results were used to construct dose response relationships to better understand the relationship between egg thiamine concentration and early life stage survival of lake trout. The IBM I developed incorporates laboratory toxicity test data, collected and applied at the individual level. These toxicity experiments measured non-traditional toxicology endpoints like growth rate and prey capture success (Fitzsimons et. al. 2009). IBM techniques have been used to assess the effects of chemical stressors on fish but have not been used to try to develop more accurate dose-response relationships (Murphy et. al. 2007). This is the first time IBMs have been used to develop environmentally relevant dose-response relationships. This technique, though in very early developmental stages, could be very useful in determining the effects of a stressor in wild populations. IBMs could be used to explore a host of other stress response relationships using toxicity data collected at the individual level.

The dose response curves produced by my model are not typical in shape. The curves are inverted because egg thiamine is on the x axis rather than the stressor, thiaminase. This was necessary because thiamine is easier to measure than thiaminase activity and most available data are in thiamine rather than thiaminase activity. The traditional dose response metrics (EC50 and LC50) can still be measured from this curve.

Model Assumptions and Deficiencies

Modeling natural systems invariably involves making assumptions and our understanding of the complex, ever-changing, Great Lakes ecosystem is far from complete. Many aspects of the early life history of lake trout are unknown and what is

known often varies spatially and temporally. In order to represent this environment in computer simulation I had to simplify key processes. Sometimes extensive data were absent and I made assumptions based on the available data. I describe some of these assumptions below and suggest these assumptions be followed up with empirical studies in the future.

In my simulations, lake trout were assumed to be thiamine replete after they had begun to feed regularly and reached 33mm, regardless of their initial egg thiamine concentration. The amount of time it takes lake trout larvae that is suffering TD to acquire adequate thiamine levels through feeding has not been studied. Lake trout and other salmonid larvae have recovered quickly from TD after administration of thiamine in laboratory experiments (Fitzsimons et. al. 2001, Ketola et. al. 2008). I assumed that once lake trout have begun to regularly ingest thiamine containing prey they would no longer exhibit symptoms of TD. If my assumption is incorrect, then the prolonged effects of TD could lead to higher mortality than was predicted by simulations.

Yolk sac utilization was a component of the IBM that has little empirical data to support the modeled processes. One missing piece of information is how much impact low thiamine has on metabolism of the yolk sac, as thiamine is an essential cofactor in metabolism. Future studies on the magnitude of yolk sac metabolic inhibition as a result of TD would help parameterize this lake trout IBM. In my model, yolk sac metabolism is not affected by thiamine concentration; instead, resting metabolism is increased for those individuals with low thiamine. If I incorporated impaired yolk-sac metabolism due to low thiamine in my model, then I predict that more fish would starve before first feeding but after the survivors had begun to feed their growth rates would be much higher.

Another assumption made in the model was that prey availability does not limit the growth of larval lake trout and larval lake trout growth and mortality is essential density independent. The prey densities I used in simulations were obtained from studies conducted in the Great Lakes but it is unrealistic to assume that these densities are representative of conditions everywhere. In addition, prey numbers were not depleted by the successful foraging of lake trout larvae during simulations. In a review of impediments to early life stage survival of lake trout, prey availability is mentioned but predation mortality is the focus of the analysis (Jones et. al 1995). A study conducted in the Apostle Islands region of Lake Superior also indicated that age 0 lake trout were not limited by prey availability. However, the authors suggest that in the other Great Lakes, higher densities of fish on spawning reefs might limit the availability of prey through competition (Hudson et. al. 1995). Adequate prey concentration has not been discussed in assessments of impediments to lake trout recruitment by other researchers (Bronte et. al. 2003, Holey et.al. 1995). Due to the absence of data on prey availability limiting survival of early life stage lake trout and the general consensus that predation mortality is more important I chose not to explore the effects of reduced prey density. I varied prey densities slightly to observe the effects and found that a 20% increase in copepod density resulted in higher survival, shorter stage duration, and an increase in growth rate of over 10%. Based on these preliminary simulations, I suspected that variations in prey density could have large impacts on growth and survival. The results of the uncertainty analyses suggest otherwise. Prey density explains less than 1% of the variance in survival in both uncertainty analyses (Table 2.4).

My model did not incorporate spatial complexity and just simulated a volume of water. The lack of spatial complexity could impact predator consumption rates substantially. Patchiness is common in natural systems, and pelagic predators and prey are not likely to be evenly distributed. In natural habitats, interstitial predators would likely search a smaller area per day (Letcher and Rice 1997). The addition of spatial complexity, if captured accurately, could reveal phenomena not suggested by the present model.

In my model, I ignored potential multi-stressor effects with contaminants. Contaminants were assumed to cause no mortality or behavioral effects in young lake trout. I made this assumption to examine the effects of TD alone but it may or may not be accurate. Each of the Great Lakes receives tens of thousands of kilograms of heavy metals and organic pollutants each year, through atmospheric deposition alone (Hoff 1999). Although the amount of contaminants in lake trout have decreased over the last few decades there are still detectable levels of chemicals in lake trout tissue (USEPA 2007). Chemicals contaminants, like mercury and PCBs, have been shown to cause negative effects in lake trout and other species (Tillitt et. al. 2008, Murphy et. al. 2008). Additional chemical stressors could be included in our model if more information was available on the concentrations found in lake trout and the associated behavioral effects. If the effects of contaminants were added to the model, survival would likely decrease through the range of conditions simulated.

I also did not consider other stressors like climate change; such a stressor may be influencing survival in ways that are currently unknown. Lake Superior summer temperatures have increased 3.5° C in the past century (Austin and Colman 2008).

Changes in lake temperature may influence time of spawning and incubation duration which could influence predator densities and prey availability at time of hatch (Moyle and Cech 2004). Some strains of lake trout may be more suited to longer or shorter incubation times depending on their habitat and climatic conditions under which they evolved. Climate change could have negative effects on lake trout by causing egg to develop too fast leading to underdeveloped fish. In the model smaller fish are less likely to survive because predator avoidance and prey capture depend on size. Conversely, climate change could have positive effects on larval lake trout by increasing prey densities. In the model increased prey densities led to higher growth rates and higher survival. My model could be adapted to include the multi-stressor effects of climate change in the future.

Research Needs and Data Gaps

Construction of my model was useful because it highlighted data gaps. In order to know what is limiting lake trout reproduction in the Great Lakes with certainty, gaps in our current knowledge must be filled. Since information costs money to acquire it is helpful to carefully consider what information would be most useful. IBMs can help identify which pieces of information are most crucial in solving a particular problem. The results of my IBM simulations and review of literature indicated the following information to be crucial in determining the cause or causes of lake trout recruitment failure. Egg and larval predation are very important and reported values are variable. Egg deposition rates and spawning reef quality may also be important. The variables associated with metabolic processes of larval lake trout are important in my simulations, yet specific experimental information is not readily available. The larval thiamine

concentration at which negative effects of TD are no longer apparent is another important piece of information for understanding the role of TD in lake trout recruitment. To date only a few studies have addressed these questions. Additionally, the source of thiaminase in the food web has not yet been identified conclusively.

More information on the density, size, and consumption rates of larval predators would help in determining the role of larval predation in successful lake trout recruitment. The sensitivity and uncertainty analyses indicated that the predator size parameter accounted for most of the variance in survival. Some information is available but predator density and species compositions vary spatially and temporally. Location specific data are essential in understanding predation dynamics.

More investigation of egg predation as well as egg deposition could provide insight into where most of the mortality is occurring during the first year of life for lake trout. Egg predation alone could be a substantial impediment to lake trout recruitment (Jonas et. al. 2005, Clarmunt et. al. 2005, Savino et. al. 1999, Jones et. al. 1995). If lake trout are spawning over poor habitat their eggs could be more susceptible to physical disturbance and predation.

Bioenergetics information specific to larval lake trout would make my IBM results more accurate. The Monte Carlo analysis indicated that a large portion of the variance in survival could be attributed to total metabolism and assimilation efficiency. Simulation results indicate total metabolism explains the second most variance in survival of all the parameters tested in both the sensitivity and uncertainty analysis. If detailed bioenergetics information was available for larval lake trout the model predictions would be more meaningful.

Any information concerning the origin of thiaminase in the food web would also be important for management. Alewife, the species that is the most likely source of thiaminase consumed by lake trout, vary substantially in their thiaminase activity (Lepak et. al. 2008). It is unclear if alewives produce thiaminase, if microorganisms in their gut produce thiaminase, or if they consume prey that contains thiaminase. If the source of thiaminase could be identified then resources could be directed towards mitigating or eliminating the source.

To fully understand why larval lake trout are not surviving to recruitment, I suggest that more empirical data is needed. Useful studies would include those that focus on egg deposition, reef quality, and egg predation. Also, studies that focus on larvae predation rates around the Great Lakes would be helpful to parameterize specific scenarios. Detailed bioenergetics information specific to larval lake trout would also allow for more accurate modeling. Finally, the time at which larval lake trout are no longer influenced by low egg thiamine would be important to identify to understand the full extent of the problem of TD.

General Conclusions

The future of individual based modeling in ecology and lake trout in the Great Lakes will be dependent on model validation, accuracy, and precision. Validation of dose-response relationships developed by IBM methods for wild populations may prove difficult. Mesocosm studies with treatments representative of simulated conditions could be used as a comparison to model results. Mesocosm studies have been performed in conjunction with laboratory studies to clarify predator prey relationships but, to my knowledge, no studies validating the results of an agent based toxicology simulation

model have been performed (Cowan and Houde 1993). The model is relatively precise; the variation in output is a result of drawing some parameter values from specified distributions. Accuracy of the model is much harder to asses. The accuracy of model predictions might be determined by testing the models ability to predict larval survival in a small area in one of the Great Lakes where all inputs come from experimental measurements. If the results of a computer simulation generated dose-response curve could be validated, this method may be very useful in predicting the responses of wild populations to stressors.

Change in the Great Lakes System

TD in lake trout may decline without human interference due to major reductions in alewife populations in the Great Lakes, especially Lake Huron (Riley et. al. 2008, Bunnell et. al. 2006, Madenjian et. al. 2008). Although a study, done by Madenjian et. al. (1998), found that lake trout preferentially feed on alewife when other prey species are more abundant. Larger lake trout show preference for alewife compared with smaller trout (Eck and Wells 1983). These findings suggest that even if alewife population is reduced, lake trout could still ingest enough thiaminase to acquire TD.

TD in other Great Lakes species may persist. According to a recent study, dreissenid mussels are very high in thiaminase activity, with levels 25 times greater than levels measured in alewife from Lake Michigan (Tillitt e. al. 2009). Fish that utilize dreissenid mussels as a prey item, lake whitefish for example, could acquire TD or produce eggs which are thiamine deficient. Fish that utilize dreissenid mussel eating fish as prey could also be affected.

Despite efforts to keep invasive species out of the Great Lakes new species continue to come in. Introduced fish, mussels, plankton, and crayfish have all made substantial impacts on the Great Lakes system and on lake trout in particular. Invasive sea lamprey decimated the lake trout population. Invasive alewives are eaten by lake trout, and are linked to TD in lake trout. Invasive rusty crayfish eat lake trout eggs. Some of the most recent potential invaders are *Hypophthalmichthys molitrix* and *Hypophthalmichthys nobilis*, species that are commonly referred to as Asian carp. Some predict the impact of Asian carp will not be great in pelagic zones of the Great Lakes (Cooke and Hill 2010) but they could have an indirect and unpredictable impact on lake trout populations.

Management Implications

Several strategies, suggested by this investigation, may reduce mortality of lake trout eggs and larvae. Stocking over offshore reefs with fewer egg predators is one that has already been implemented. Stocked lake trout tend to return to their stocking site to spawn regardless of the habitat quality (Bronte et. al. 2007). Increasing stocking rates to increase egg densities is another option to increase survival. In my simulations, increased egg density resulted in higher survival. A similar trend that related lower egg predation with higher egg to predator ratios has been observed experimentally as well (Jonas et. al. 2005). Stocking strains of lake trout with heritably high growth rates could also be beneficial. My simulation results indicated that under high predation conditions the larvae with the highest growth rates survived. Interactions in the model are size-based so that larger fish are able to capture prey and avoid predators more effectively than smaller fish.

In natural systems, where size dependent predation is important, high growth rate may be important in survival (Rice et. al. 1993).

The Great Lakes ecosystem is in a constant state of change due to human induced introduction of non-native species, climate change, and natural biological cycles. It is impossible to predict with certainty the state of any Great Lakes species in the future. Using simulation models parameterized with data from field and laboratory experiments; future scenarios can be explored. The use of an individual based model to re-evaluate a dose-response relationship is a new approach to the study of stress on wild populations. It could prove an effective method if validated experimentally. Ultimately, with further testing and development, these types of models could aid in management of the Great Lakes and their inhabitants under uncertain conditions.

APPENDICES

Appendix A, Table A.1: Complete table of parameters used in uncertainty analysis including descriptions.

Parameter	Description
thicon	Average larval thiamine concentration (nmol/g)
yolkut	Proportion of maximum consumption reached from yolk sac utilization
firstfeed	Size at first feeding (mm)
efeat	Thiamine concentration above which prey capture success was no longer negatively affected (nmol/g)
efmeta	Thiamine concentration above which metabolism was no longer negatively affected (nmol/g)
efpred	Thiamine concentration above which predator escape was no longer negatively affected (nmol/g)
starvenum	Percent of larval weight lost that results in starvation
zoopden1	Density of copepod prey (#/mm^3)
zoopden2	Density of cladoceran prey (#/mm^3)
zoopden3	Density of chironimid prey (#/mm^3)
zoopden4	Density of mysid prey (#/mm^3)
metamult	Total metabolism multiplier
CSnum1	Capture success numerator for copepod prey
CSnum2	Capture success numerator for cladoceran prey
CSnum3	Capture success numerator for chironimid prey
CSnum4	Capture success numerator for mysid prey
CSden1	Capture success denominator for copepod prey
CSden2	Capture success denominator for cladocern prey
CSden3	Capture success denominator for chironimid prey
CSden4	Capture success denominator for mysid prey
predlen2	Predator length (mm)
prednum2	Number of predators in simulation
cmaxnum	Maximum consumption multiplier
assm	Assimilation efficiency multiplier
svmult	Search volume multiplier
htmult	Handling time multiplier

Appendix B, Table B.1: Table of parameters used in sensitivity and uncertainty analysis including means, minimum, and maximum values.

		10%CV		25% CV	
Parameter	Mean	Minimum	Maximum	Minimum	Maximum
thicon	8	6.61	9.385	4.53	11.46
yolkut	0.666	0.55	0.78	0.37	0.94
firstfeed	24	19.84	28.15	13.6	34.39
efeat	8.5	7.02	9.97	4.82	12.18
efmeta	9	7.44	10.55	5.1	12.89
efpred	8.5	7.02	9.97	4.82	12.18
starvenum	0.25	0.206	0.293	0.14	0.358
zoopden1	2.50E-04	2.07E-04	2.90E-04	1.42E-04	3.50E-04
zoopden2	2.20E-05	1.82E-05	2.58E-05	1.25E-05	3.15E-05
zoopden3	3.00E-07	2.48E-07	3.52E-07	1.7E-07	4.3E-07
zoopden4	2.00E-07	1.65E-07	2.35E-07	1.13E-07	2.87E-07
metamult	1	0.826	1.173	0.5669	1.433
CSnum1	0.6	0.496	0.703	0.34	0.859
CSnum2	0.5	0.413	0.586	0.283	0.716
CSnum3	0.7	0.578	0.821	0.396	1.003
CSnum4	0.3	0.248	0.351	0.17	0.429
CSden1	5.00E+06	4.13E+06	5.87E+06	2.83E+06	7.17E+06
CSden2	5.00E+07	4.13E+07	5.87E+07	2.83E+07	7.17E+07
CSden3	5.00E+06	4.13E+06	5.87E+06	2.83E+06	7.17E+06
CSden4	5.00E+07	4.13E+07	5.87E+07	2.83E+07	7.17E+07
predlen2	150	124.01	175.98	85.04	214.95
prednum2	10	8.27	11.73	5.67	14.33
cmaxnum	1	0.83	1.17	0.57	1.43
assm	1	0.83	1.17	0.57	1.43
svmult	1	0.83	1.17	0.57	1.43
htmult	1	0.83	1.17	0.57	1.43

Appendix C, Table C.1: Complete table of parameters used in model runs.

parameter	**baseline value**
Initial number of larvae	6000
Volume of water (m^3)	2000
Number of days in simulation	100
Average initial larval length (mm)	20
Length–weight relationship intercept	0.1674
Length–weight relationship exponent	3.837
Number of predators in simulation	0-210
Predator length (mm)	60-160
Prop. of day available for feeding	13/24
Swimming speed intercept	0.776
Swimming speed exponent	1.07
Prop. of reactive area used for feeding	0.4
Handling time slope	7.0151
Handling time intercept	0.264
Capture success numerator (prey types 1–4)	0.60; 0.50; 0.70; 0.30
Capture success denominator (prey types 1–4)	5*10^6; 5*10^7; 5*10^6;5*10^7
*C*max function intercept	2.8275
*C*max function exponent	0.8496
Maximum assimilation efficiency	0.6
Assimilation efficiency shape parameter	0.002
Routine metabolism numerator	4500
Routine metabolism denominator	45000
Specific dynamic action + egestion	0.3
Activity metabolism multiplier	2.5
Starvation threshold	0.75
Daily yolk sac utilization as percentage of max consumption	66
Predator's reactive distance multiplier	0.8
Predator's swimming speed multiplier	3.0
Predator's capture success exponent	2.28
Predator's capture success numerator	3.37
Predator's capture success denominator	44.76
Egg death threshold nmol/g	<0.65
Metabolism no effect thiamine concentration (nmol/g)	>9
Prey capture no effect thiamine concentration (nmol/g)	>8.5
Predator avoidance no effect thiamine concentration (nmol/g)	>8.5
Average egg thiamine concentrations (nmol/g)	1.0-16.0

Table C.1 (cont'd)

Copepod density (#/liter)	0.25
Copepod size (mm)	1
Copepod mass (ug dw)	7.7
Cladoceran density (#/liter)	0.022
Cladoceran size (mm)	1.5
Cladoceran mass (ug dw)	15.81
Chironomid density (#/liter)	0.0003
Chironomid size (mm)	3
Chironomid mass (ug dw)	31.91
Mysid density (#/liter)	0.0002
Mysid size (mm)	5
Mysid mass (ug dw)	324.94

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