

THE MANY ROLES FOR MODELING IN ECOSYSTEM SCIENCE AND  
MANAGEMENT

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ABSTRACT  
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Sustainability can be defined as maintaining or increasing society's "genuine wealth" or the pool of society's capital. This pool of capital includes financial, physical, natural, and human capital. Sustainable natural resource management science is therefore fundamentally a capital management science. Natural resource management involves making tradeoffs across potential investments to maintain or enhance the pool of capital, with a focus on natural capital. Making such tradeoffs, however, is complicated by the complexities of ecological and economic systems. Models are useful tools for exposing, analyzing, and aiding in making tradeoffs among potential natural resource management strategies or investments. This dissertation uses a variety modeling approaches to expose, analyze, and aid in making tradeoffs associated with three fishery management problems. A common theme among these three problems is they all involve aquatic species introduction; the first two address aquatic infectious pathogens and the third one addresses fish stocking and control of an invasive species. All chapters are motivated by Great Lakes issues involving fish stocking, but they all have broader implications.

Making tradeoffs is a fundamental part of natural resource management. In order to make tradeoffs, information about the ecological and economic systems is needed. Collecting this information involves tradeoffs as well (Chapters 2 and 3). The complex nature of ecological and economic interactions complicates making tradeoffs,

and models can help organize thinking and facilitate analysis. Increasing awareness of the joint production of ecosystem services, multiple equilibria and non-convexities, and the imperfectly targeted nature of management actions (Chapter 4) should be viewed in light of advancing knowledge about ecological and economic systems. It is not that tradeoffs are more complex than in the absence of this knowledge, but rather that there is a growing appreciation and desire to account for potential, unintended effects of management actions.

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# CHAPTER 1

## INTRODUCTION

Sustainability can be defined as maintaining or increasing society's "genuine wealth" (Arrow et al. 2004) or the pool of society's capital. This pool of capital includes financial, physical, natural, and human capital. Sustainable natural resource management science is therefore fundamentally a capital management science. Natural resource management involves making tradeoffs across potential investments to maintain or enhance the pool of capital, with a focus on natural capital. Making such tradeoffs, however, is complicated by the complexities of ecological and economic systems. Models are useful tools for exposing, analyzing, and aiding in making tradeoffs among potential natural resource management strategies or investments. This dissertation uses a variety modeling approaches to expose, analyze, and aid in making tradeoffs associated with three fishery management problems. A common theme among these three problems is they all involve aquatic invasive species, two address aquatic infectious pathogens and one addresses fish stocking and control of an invasive species. All chapters are motivated by Great Lakes issues involving fish stocking, but they all have broader implications as well.

In a number of management situations it can be difficult to know where to begin investing resources in management or what tradeoffs exist. Increased research is one potential solution, but this raises the question of how best to invest resources in research. This is a common problem in the management of aquatic disease systems. There is often little data at the population or system level. The second chapter of this

dissertation develops a multiple-host pathogen model for Lake Michigan focusing on interactions between a pathogen, *Renibacterium salmoninarum* (the etiological agent of bacterial kidney disease) and its hosts. This model is used to organize existing information about the system. The model is analyzed to determine what kinds of information are most likely to have the largest impact on indicators of concern. This can help research funding agencies make tradeoffs among different research proposals. Moreover, the model exposes certain management tradeoffs. For example, the model indicates that there is likely a tradeoff in terms of adult stock sizes between increasing the number of smolts stocked and increasing bio-security measures.

Once managers have an idea of what information is needed to assist in making management decisions, there is a need to design data collection methods that are effective, efficient, and connected to the management objective. Sampling is the result of the need to make tradeoffs. There are never enough resources to collect or analyze all the potential data. Yet, sampling design is often only based on statistical considerations. Chapter 3 presents necessary concepts for developing aquatic pathogen screening programs and connects probability theory to management objectives and the need to make tradeoffs. Chapter 3 emphasizes that estimating only the chance of pathogen detection is not enough; managers also should consider the likelihood and consequences of facilitating pathogen introduction when designing screening programs. Finally, Chapter 3 gives conditions for when a screening program should terminate after a number of successive screening events, during which no infected individuals have been detected. This result is based on the tradeoffs between the costs of screening and the cost of a pathogen introduction.

Mathematical bioeconomic investment models (*sensu* Clark 1990) can aid managers in exploring management tradeoffs. These models explicitly treat natural resources as capital. Chapter 4 presents this type of model for the Chinook salmon (*Oncorhynchus tshawytscha*) stocking program in Lake Michigan. Linear control theory is used to solve for a stocking program that maximizes net present social benefits as a function of angler welfare, averted alewife (*Alosa pseudoharengus*) damages, and stocking costs. The model system includes an open access recreation fishery dependent on salmon. Anglers respond to incentives provided by the salmon stock. Alewives are an invasive species that provide ecosystem disservices by damaging infrastructure, natural amenities, and preying on the eggs of valued native fish. Salmon provide benefits to anglers and control the alewife population, but are also dependent on the alewife population for survival. The model reveals that these interactions result in complex tradeoffs that result in multiple equilibria and non-convexities. These are related to the imperfect nature of controls for targeting alewife management. Policy findings include that *i*) the optimal level of stocking is a function of the current salmon and alewife stock and therefore stocking should be based on “feedback” rules; *ii*) eradication (conservation) of invasive alewife may not be optimal and will depend on initial conditions; *iii*) consideration of the ecosystem disservices caused by alewives alters the nature of the problem in a qualitative way; and *iv*) the maximum catch does not occur at the optimal equilibrium, but rather as part of an approach path as alewife are depleted.

Making tradeoffs is a fundamental part of natural resource management. In order to make tradeoffs, information about the ecological and economic systems is

needed. Collecting this information involves tradeoffs as well (Chapters 2 and 3). The complex nature of ecological and economic interactions complicates making tradeoffs, and models can help organize thinking and facilitate analysis. Increasing awareness of the joint production of ecosystem services, multiple equilibria and non-convexities, and the imperfectly targeted nature of management actions (Chapter 4) should be viewed in light of advancing knowledge about ecological and economic systems. It is not that tradeoffs are more complex than in the absence of this knowledge, but rather that there is a growing appreciation and desire to account for potential, unintended effects of management actions.

This dissertation presents three chapters represented three ways models can be useful in making natural resource management decisions.

**CHAPTER 2**  
**A MODEL OF *RENIBACTERIUM SALMONINARUM* DYNAMICS IN**  
**GREAT LAKES FISH POPULATIONS:**  
**Implications for bacterial kidney disease management and research in Lake**  
**Michigan**

**2.1 Introduction**

Fishery managers are increasingly concerned about fish health, but little is known about the dynamics and effects of pathogens in fish populations outside of the laboratory, aquaculture, and hatchery facilities. Quantitative modeling has provided a useful approach to organize and improve understanding of host-pathogen dynamics in wild populations, but such efforts have been largely applied to terrestrial ecosystems (McCallum et al. 2004), with a few exceptions from marine systems (Murray et al. 2001; Ogut et al. 2005), and have not been applied to managed freshwater ecosystems. The presence of *Renibacterium salmoninarum* (*Rs*), the causative agent for bacterial kidney disease (BKD) is well established in the Great Lakes, a large managed freshwater ecosystem. Benjamin and Bence (2003b) estimated a substantial increase in salmonid mortality in Lake Michigan associated with increased prevalence of *Rs* (Holey et al. 1998). *Rs* is also believed to be “one of the most important bacterial diseases affecting wild and propagated anadromous salmonid stocks [in the Pacific Northwest]” (Wiens and Kaattari 1999), but little is known about the pathogen’s dynamics in the wild.

Here we develop a model of fish-*Rs* dynamics based on free-swimming fish populations to aid fishery managers in two ways. First, a model can help synthesize and interpret complex datasets to draw inferences about the possible consequences of

management strategies. Patterson (1996) uses a complex dataset to estimate disease induced mortality caused by the phycomycete fungal parasite *Ichthyophonus hoferi* in North Sea herring (*Clupea harengus*) from catch-at-age data combined with prevalence data, but does not evaluate how alternate management strategies may affect this parameter. Patterson (1996) notes that failing to account for disease effects can lead to an overestimation of fishing mortality and this may have implications for harvest policy. Barlow (1991; 1996) and Smith and Cheeseman (2002) employ host-pathogen models to evaluate alternative control strategies for bovine tuberculosis (*Mycobacterium bovis*) in New Zealand possums (*Trichosurus vulpecula*) and English badgers (*Meles meles*) respectively.

Second, host-pathogen models also can be used to develop and evaluate hypotheses about system dynamics, thereby exposing critical uncertainties and identifying opportunities for efficient and insightful data collection. A lack of data on disease transmission, however, does not pose a barrier for fulfilling these objectives (Caley and Hone 2004). For example, Murray *et al.* (2001) developed a model of herpesvirus transmission in pilchards to examine assumptions about transmission processes. Ogut *et al.* (2005) demonstrated how host-pathogen models can be used to gain insight into the disease dynamics, explore uncertainties, and estimate parameters for a captive Chinook salmon (*Oncorhynchus tshawytscha*) population infected with *Aeromonas salmonicida* (the etiologic agent of furunculosis).

Following terrestrial investigations, existing fish-pathogen modeling efforts have focused on single fish populations and ignore the influence of management activities on the system. However, the ability of pathogens to influence ecological relationships

among species is receiving increased attention (Hatcher et al. 2006), and it is often necessary to consider pathogen dynamics in the context of a broader host community to understand the effects of a pathogen on a particular host species (Dobson 2004, Tsao 2004). Moreover, many freshwater systems, such as Lake Michigan, are highly influenced by human activities (e.g. stocking of salmonids) that affect host community dynamics.

We incorporate management features into a multiple-host disease model of  $R_s$  in Lake Michigan to qualitatively explore the implications of modeling assumptions, model sensitivity, and management actions on host-pathogen dynamics. Three host stocks were included in the model: two types of non-native Chinook salmon - those that result from spawning in the wild (*wild* Chinook), and those that result from collection of gametes from feral adults and hatchery production (*hatchery* Chinook) - and native, wild spawning lake whitefish, *Coregonus clupeaformis*. The Chinook salmon population was divided into wild and hatchery spawning components to enable analysis of management effects and to help design future experiments. Chinook salmon and lake whitefish usually, but not always, occupy different habitats, do not consume the same prey, and do not eat each other, therefore we have assumed that *direct* pathogen transmission between these two species is negligible. However, pathogen transmission between these two species may be facilitated by an environmental reservoir. A reservoir captures the ability of  $R_s$  to survive in water or sediment and may also represent, to some extent, the presence of infectious pathogen occurring within other currently unknown host species that are not explicitly modeled.



## 2.2 System Background

In 1987, Chinook salmon in Lake Michigan suffered a large increase in mortality (Benjamin and Bence 2003a) associated with a widely observed outbreak of BKD and a high prevalence of *Rs*, first documented in the spring of 1988 (Holey et al. 1998). Researchers subsequently documented *Rs* and clinical signs of BKD in lake whitefish populations (Jonas et al. 2002). Furthermore, *Rs* has been documented in other Lake Michigan salmonines and in sea lampreys (Eissa et al. 2006) and is likely present in other Lake Michigan fishes (M. Faisal, Michigan State University, personal communication).

Knowledge of *Rs*/BKD dynamics in Lake Michigan is extremely limited, despite evidence of the importance of this disease for salmonid dynamics (Holey et al. 1998; Benjamin and Bence 2003a, b). Most knowledge about fish-borne disease comes from observations and studies conducted in hatcheries or fish production facilities (Stephen and Thorburn 2002), and the dynamics of BKD are no exception (Wiens and Kaattari 1999). From such experiments it is known that *Rs* can be transmitted horizontally (i.e., directly) among juvenile and adult salmonids (Mitchum and Sherman 1981; Balfry et al. 1996), as well as vertically from adult females to eggs (summarized in Wiens and Kaattari 1999). Further research has shown that fish may become infected when the pathogen is present in sufficiently high levels in the surrounding water (summarized in Hamel 2005). However, it is unclear which transmission processes drive pathogen dynamics among free-swimming fish populations or are important for pathogen maintenance.

### 2.3 Model Development

Host-pathogen models are population models where the host population is distributed among health classes, and pathogen dynamics are tracked based on the proportion of the population in each health class. We use a susceptible-exposed-infected-exposed (*SEIE*) host-pathogen framework (Fig 1).

The *SEIE* model is a modification of the standard susceptible-infected-recovered (*SIR*) framework for modeling host-pathogen relationships (Anderson and May 1991). The health states *S*, *E*, *I*, and *R* represent four subpopulations - susceptible, exposed, infected, and recovered, respectively. Susceptible individuals do not host the pathogen currently, but may do so in the future. Infected individuals are ones that are *infectious* and subject to disease induced mortality. Recovered individuals have acquired complete immunity (Anderson and May 1991). With respect to BKD, we assume that any immune population is negligible, and thus, we drop the *R* class, as is common for chronic diseases (Barlow 1991). Furthermore, the slow development of BKD motivates the inclusion of a latent/exposed (*E*) stage (Heesterbeek and Roberts 1995 and Murray et al., 2001). Exposed fish in health class *E* harbor *R*s, and host the pathogen; however, they are not yet *infectious*, nor are they subject to disease induced mortality. In our model, infection may abate, and individuals in class *I* may return to class *E*. This can be thought of as an infection in remission.

The number of individuals in a given health class depends on rates of transition among the different classes, including the rates at which individuals become exposed to the pathogen, become infectious, die due to disease, or go into remission. Taking host population dynamics into account is important for a chronic disease like BKD because

pathogen dynamics occur on a comparable timescale with that of hosts. Thus, the number of individuals among different health classes depends on birth and death rates, which are affected by abiotic factors and density-dependence. In these systems, the pathogen can persist in the population for multiple generations (i.e., is endemic, see Barlow 1991 and 1996 for examples). In contrast, for highly transmissible and often virulent pathogens, because pathogen dynamics occur on much shorter timescales than that of hosts, recruitment of new susceptibles through birth is usually ignored. In Murray et al.'s (2001) pilchard example, the pathogen infects a large number of fish and then dies out within one generation (i.e., is epidemic).

Our host-pathogen model is a system of population models for three fish host types - lake whitefish ( $w$ ), wild Chinook, ( $c$ ), and hatchery Chinook, ( $h$ ). In addition to fish population hosts, we consider two pathogen reservoir types, lake ( $l$ ), and river ( $r$ ). At any moment the model tracks five sources of pathogen - wild spawned chinook, hatchery chinook, lake whitefish, the river reservoir, and the lake reservoir. Furthermore, we used an age-structured model for Chinook salmon and a stage-structured (pre-recruit, post-recruit) model for lake whitefish (over much of their life, lake whitefish population dynamics are affected by similar factors in similar ways). Thus there are five age classes for each Chinook type and two stage classes of whitefish. Each age (stage) of each host type is essentially treated as an individual host type. Chinook salmon shift from riverine habitats at age-0 to lake habitats during juvenile life stages, and then back to riverine habitats to reproduce. Only Chinook salmon at age-0 are exposed to the river reservoir. Lake whitefish spend their entire

life in the lake proper and typically do not recruit to the commercial fishery until age-3 or 4.

Given this general framework, we now explain our model in detail. The model operates on an annual time step, and within each year there are five sequential steps (Fig. 2): (1) recruitment and aging, (2) pre-transmission mortality, (3) changes to the pathogen reservoirs, (4) disease transmission (movement of fish among the S, E, and I states), and (5) post-transmission mortality. In the following section, these steps are discussed in detail.

*Step 1.* At the beginning of each time step, the age composition of each host group is updated. For the age-structured Chinook salmon populations this means that all fish of age  $a$  at time  $t$  that survive the year become age  $a+1$  at time  $t+1$ . For the stage-structured lake whitefish, however, only a portion of the surviving stage-0 fish become stage-1 at the start of the next time step.

Recruitment of both wild Chinook salmon and lake whitefish is calculated from a Ricker (1954) stock-recruitment relationship,  $r = \alpha A e^{-\gamma A'}$ , where  $A$  is the mature host abundance (with  $A' = A$  in the standard model),  $r$  is recruitment and  $\alpha, \gamma$  are parameters. For stocked Chinook salmon, recruitment is simply the number of fish stocked. The mature Chinook salmon producing wild recruitment is assumed to include both wild and hatchery fish of all spawning age classes. Lake whitefish recruitment (progeny of fish in stage-1) are added to those that remain in stage 0 from the previous year.

The Ricker function includes a density-independent term ( $\alpha$ ) related to the maximum production of offspring per adult, and a density-dependent term ( $e^{-\gamma A'}$ ), that

accounts for limitations on recruitment due to nest crowding a lack of oxygen or similar processes. We assume that the magnitude of density-dependence is determined by the entire spawning population for both wild Chinook and lake whitefish. For lake whitefish  $A' = \eta_w = N_{w,1}$ , where  $N_{w,1}$  is the population of stage 1 whitefish. For the wild Chinook salmon this  $A' = \eta_c = \sum_i \sum_a \psi_{i,a} N_{i,a}$ , where  $\psi$  is a measure of the relative fecundity and proportion mature for each age class, and where  $N_{i,a}$  is the population of host type  $i$  ( $i=c,h$ ) at age  $a$ .<sup>1</sup> In contrast, we assume that the density-independent term is scaled only by the spawning adults in the specified cohort. Thus, in our model  $A \neq A'$ . This gives the following system of equations:

$$\begin{aligned}
 (2.1) \quad & N_{h,0_t} = s_t \\
 & N_{c,0_t} = \sum_i \sum_a \alpha_{i,a} N_{i,a,t} e^{-\gamma_c \eta_{c,t}} \quad i = \{c,h\} \\
 & N_{w,0_t} = \left( (1-p) N_{w,0_t} + \alpha_w N_{w,1_t} e^{-\gamma_w \eta_{w,t}} \right)
 \end{aligned}$$

The parameter  $p$  is the proportion of whitefish recruited from stage-0 to stage- 1 during the previous year (see below) and  $s_t$  is the number of hatchery salmon that are stocked.

In addition to determining how many progeny are produced, we also must index progeny by infectious status, because their distribution across health classes affects the total mortality rate they experience during the next step of the model.  $R_s$  can be transmitted vertically, i.e., from mother to egg through reproduction (summarized in Wiens and Kaattari 1999). To model vertical transmission we modify the density-

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<sup>1</sup> We assume that  $\psi_a = \alpha_a / \alpha_4$ , where age-4 has the largest value of  $\alpha$ , though more general specifications are possible.

independent component of recruitment in equation (2.1) to distinguish the contribution of infected and uninfected adults to infected and susceptible offspring. We assume that spawners in class  $E$  do not transmit pathogen to their progeny, and that any offspring that contract the pathogen from vertical transmission are placed in class  $I$ . The parameter  $v$  is introduced to account for the proportion of infected progeny produced from class  $I$  spawners (i.e., the vertical transmission rate). We assumed a 10% rate of vertical transmission, which is within the observed range (1%-14%) found in laboratory experiments for coho salmon (*Oncorhynchus kisutch*) (summarized in Hamel 2005). For wild Chinook recruitment the resulting equations are:

$$(2.2) \quad \begin{aligned} S_{i,0} &= \sum_i \sum_a \alpha_{i,a} \left( N_{i,a,t} - I_{i,a,t} + (1-v)I_{i,a,t} \right) e^{-\gamma_i \eta_i} \\ I_{i,0} &= \sum_i \sum_a \alpha_{i,a} \left( v I_{i,a,t} \right) e^{-\gamma_i \eta_i} \end{aligned}$$

where  $S_{i,0}$  and  $I_{i,0}$  represent recruits of susceptible fish from uninfected and infected parents, and infected fish from infected parents, respectively. For lake whitefish the term  $(1-p) S_{i,0}$  or  $(1-p) I_{i,0}$  is added as in equation (2.1) to account for susceptible or infected fish remaining in stage 0.

*Step 2 (and 5).* Instantaneous mortality is applied twice during the year (Figure 2.2), before and after disease transmission. Let the proportion of the year represented by the solid line on the left side of Figure 2.2 be  $\tau$  (in our simulations we assume  $\tau = 0.5$ ). The standard equation for applying instantaneous mortality is

$$(2.3) \quad N_{i,a,t+\tau} = N_{i,a,t} e^{-z_{i,a}\tau},$$

which assumes that the total mortality rate,  $z_{i,a}$  (for host type  $i$  and age (stage)  $a$ ) is constant. In a host-pathogen model even if the component instantaneous mortality rates (i.e., natural, non-BKD mortality ( $m$ ), fishing mortality, ( $f$ ), and BKD-induced mortality ( $d$ )) are constant, the value of  $z$  in this model could not be constant throughout the year because the proportion of  $N$  that experiences disease mortality ( $d$ ) changes as a result of disease transmission (step 3) during the year. Therefore, we model the mortality of each health class separately.

$$\begin{aligned}
 S_{i,a,t+\tau} &= S_{i,a,t} e^{-(m+f)\tau} \\
 E_{i,a,t+\tau} &= E_{i,a,t} e^{-(m+f)\tau} \\
 I_{i,a,t+\tau} &= I_{i,a,t} e^{-(m+f+d)\tau}
 \end{aligned}
 \tag{2.4}$$

For the second application of mortality (right solid line in Figure 2.2),  $\tau$  is replaced with  $1-\tau$  and  $t$  is replaced with  $\tau$ . This approach implicitly assumes that disease transmission occurs over a relatively short period of time during the year. A shorter time step would be required to model a transmission process that occurred throughout the year, but this is only numerically significant to the disease and population dynamics if the changes among classes of host are fairly large and disease mortality is comparable to magnitude of the other mortality sources.

We assumed that the instantaneous rate of BKD-induced mortality,  $d$ , is 0.8 of the rate of natural mortality,  $m$ . This value was derived empirically to allow for pathogen persistence at and interior level in the model – substantially higher values led to pathogen extinction, and substantially lower values led to 100% infection.

*Step 3.* The next event in the model is updating the environmental reservoirs. Reservoir size is defined in terms of infectious units. Environmental reservoirs do not have population growth relationships, as  $R_s$  does not reproduce outside of the host. The source of  $R_s$  to reservoirs is assumed to be from the carcasses of infected individuals that died over the past year. Pathogen loading of the reservoir is modeled as proportional to the number of individuals dying due to natural (non-BKD) or BKD-induced mortality that are in health class  $I$ . We assume the rate of shedding by live fish is negligible (or directly proportionally to dead fish). In the absence of replenishment, the abundance of pathogen in the reservoir is expected to decline and is modeled by an exponential decay function.

*Step 4.* Once they are established in an age (stage) class, susceptible fish move into the exposed class via contact with infected fish through random mixing. During each time step fish move from health class  $S$  to  $E$  at time  $\tau$  according to horizontal transmission function  $H_{i,a}$ , which is specific to host type and age. Horizontal transmission generally follows Dobson (2004)

$$\begin{aligned}
 S_{\tau+\Delta} &= -H_{i,a}(S_{\tau}, I_{\tau}) = S_{\tau} - S_{i,a,\tau} \sum_j \sum_k \beta_{(i,a),(j,k)} I_{j,k,\tau} \\
 (2.5) \quad E_{\tau+\Delta} &= H_{i,a}(S_{\tau}, I_{\tau}) = I_{\tau} + S_{i,a,\tau} \sum_j \sum_k \beta_{(i,a),(j,k)} I_{j,k,\tau}
 \end{aligned}$$

where  $\beta$  is the per capita rate of pathogen transmission from host  $j$  cohort  $k$  to host  $i$  cohort  $a$  (Dobson 2004), where  $i, j$  index all potential host types and  $a, k$  index their age or stage, and  $\Delta$  represents a small change in time.

Immediately following the recruitment of susceptibles to the exposed class, the advancement process between the exposed and infected classes occurs. Traditionally,



exposed individuals are included to model a fixed latency period, and advancement is simply determined by a rate parameter that represents the inverse of the average latency period (Heesterbeek and Roberts 1995). However, researchers do not know what an average latency period is for *Rs/BKD*, and furthermore, the *BKD* epidemic in Lake Michigan coincided with a period of reduced Chinook growth rates, suggesting that nutritional stress may play an important role in triggering clinical disease (Holey et al. 1998). Here, we make advancement a function of nutritional stress. We assume that nutritional stress is a function of intraspecific competition and that increased host densities approximate reduced food resources per individual<sup>2</sup>, thus making advancement a function of  $\sum_i \sum_a N_{i,a,\tau+\Delta}$ . As the density of the host population

increases, *ceteris paribus*, so does the rate at which exposed individuals become infected. Conversely, low host densities result in increased food resources per individual and thus a net recovery from the infected to the exposed class. That is, infected fish return to the exposed class at a greater rate than exposed fish become infected. We define the advancement function  $Q$  as the process by which exposed fish advance to the infected class (negative advancement implies remission to the exposed),

$$(2.6) \quad \begin{aligned} \Delta E_{\tau+2\Delta} &= E_{\tau+\Delta} - Q(Y(\Lambda), E_{i,a,\tau+\Delta}, I_{i,a,\tau+\Delta}) \\ \Delta I_{\tau+2\Delta} &= I_{\tau+\Delta} + Q(Y(\Lambda), E_{i,a,\tau+\Delta}, I_{i,a,\tau+\Delta}) \end{aligned}$$

where  $\Lambda = \sum_i \sum_a N_{i,a,\tau+\Delta}$  and where  $Y$  is a function that defines the rate of movement between classes  $E$  and  $I$  and must have two qualities. First,  $|Y| \leq 1$ , and

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<sup>2</sup> This implicitly assumes that the prey resource is fixed.

second, there must be some  $\Lambda = \tilde{N}$  where  $Y(\tilde{N})=0$ , i.e., where there is no net movement from exposed to infected. Function  $Y$  takes the form

$$(2.7) \quad Y(\Lambda) = \frac{(\Lambda - q_{eq})}{(\Lambda - q_{eq}) + q_{eq} + q_{50\%}}$$

where  $q_{50\%}$  is the population density at which 50% of the exposed (infected) individuals become infected (exposed) and  $q_{eq} = \tilde{N}$ . The parameter  $q_{eq}$  also determines the direction of movement:

$$(2.8) \quad Q(Y(\Lambda), E_{i,a,\tau+\Delta}, I_{i,a,\tau+\Delta}) = \begin{cases} Y(\Lambda)E_{i,a,\tau+\Delta}, & \text{if } \Lambda > q_{eq} \\ -Y(\Lambda)I_{i,a,\tau+\Delta}, & \text{if } \Lambda < q_{eq} \end{cases}$$

### ***Simulations***

Two modeling workshops and numerous correspondences with fish health specialists and Great Lakes fishery biologists were conducted to solicit expert opinion on model processes and parameter values for calibration of the model. There are no estimates for important parameters in the model such as transmission rates. However, using realistic estimates for recruitment, survival, and mortality parameters, it was possible to bound the range of feasible transmission parameters (see Appendix for parameter values used in the simulations). Transmission parameters were chosen to create realistic scenarios, so that meaningful cases could be examined. For most cases, transmission parameters were chosen so that prevalence rates could stabilize at interior levels (i.e., the pathogen persisted but did not result in 100% infection). In other cases,

transmission parameters were set so that infection could not be maintained and the pathogen vanished from the system.

We conducted a series of simulations to identify emergent properties resulting from the combination of population and host-pathogen theories. The model was simulated for a sufficiently long time period so that transient dynamics subsided and the system appeared to arrive at equilibrium (where changes in the values essentially were zero). Most simulations ran for 100 years, but 200 year simulations were used when the system was deliberately perturbed during the first 100 years. The initial source of *R<sub>s</sub>* is assumed to be hatchery fish, though this assumption is explored in the results section.

We investigated how various management activities may influence model outcomes. First, we investigated scenarios that only included hatchery and wild Chinook salmon. This allowed us to explore the model's sensitivity to demographic parameters, the effects of stocking rate and hatchery practices (see Table 2.1 for descriptions of hatchery practice scenarios), and the interactions between stocking rates and hatchery practices. We also characterized conditions that could lead to an outbreak of BKD like that observed on Lake Michigan without the complication of alternative hosts. Next, we included an environmental reservoir in the model to understand how this may impact the results of Chinook salmon only simulations. Finally, we included whitefish and examined interactions with Chinook salmon. We implemented the model in Microsoft Visual Basic interfaced through a Microsoft Excel workbook.

## 2.4 Results

### *Model sensitivity for baseline conditions and the effect of vertical transmission*

We began by exploring the sensitivity of the model to parameter assumptions. The model, even when restricted to Chinook salmon, has a large number of parameters and analysis and presentation of model sensitivity of each one is not feasible. Moreover, in the absence of data, a detailed sensitivity analysis for each parameter may be less illuminating than other sorts of analysis (discussed later in the Results sections). We focus our sensitivity analyses on parameters for which there are the least amount of data in the literature and on parameters that ecological and epidemiological theory predict would have the greatest effect on model outcomes. The first model ignores vertical transmission and disease induced mortality to focus on the impact of horizontal transmission.

To examine sensitivity of a given parameter, we adjusted the value of the parameter by 5% and then compared simulation results to that of the default value. To assess sensitivity, we employed the concept of elasticity (Pindyck and Rubinfeld 2001 p. 30; Coulson and Godfray 2007) and calculated the percent change in the density of wild Chinook salmon at equilibrium in each health class divided by a 5 percent change in the model parameter. If the percent change in the density at equilibrium was  $> 5\%$  (yielding an elasticity  $> |1|$ ), then the model was deemed to be elastic and sensitive to changes in the parameter; likewise, if the percent change was  $< 5\%$  (elasticity  $< |1|$ ), then the model was deemed to be insensitive to changes to the parameter. Parameters explored included the Ricker density-dependence parameter ( $\gamma$ ), the transmission parameter ( $\beta$ ), and the two parameters in the advancement function ( $q_{eq}$  and  $q_{50\%}$ ). The

model showed greatest sensitivity to the Ricker density-dependence parameter but was also sensitive to the transmission parameter and  $q_{50\%}$ , with elasticities  $\gg |1|$  (Table 2.2). These results were consistent across all ages of Chinook salmon and for both the exposed and infected health classes. Wild Chinook salmon prevalence rates varied by health class and age between being inelastic and elastic with respect to changes in the  $q_{eq}$  parameter (Table 2.2).

The sensitivity of wild Chinook salmon prevalence to changes in parameters generally declined when vertical transmission was added to the model (Table 2.3). For all age and health classes except age-4 health class  $E$ , elasticities were largest for  $\gamma$  followed by  $\beta$ . Many elasticities, however, were  $< |1|$  when vertical transmission was added to model, including all age-3 and age-4 exposed elasticities as well as all elasticities with respect to  $q_{50\%}$ . Finally, all elasticities with respect to  $\nu$  were  $< |1|$ . When results were inelastic ( $< |1|$ ) this implies that  $\nu$  does not have a strong direct effect on the outcome.

The presence of vertical transmission does seem to have large effect on the model even though small changes in vertical transmission may not have disproportionately large effects. The inclusion of vertical transmission had two effects that need to be considered. First, vertical transmission increased the prevalence of both exposed and infected individuals of all ages at equilibrium. The second effect was that increased vertical transmission increased the number of age-0 *exposed* fish. This occurs because increased vertical transmission increases the number of age-0 *infected* fish with which susceptible age-0 fish make contact, i.e., additional infected fish provide an additional source for secondary exposures. Disease-induced mortality

reduces the prevalence of disease, *ceteris paribus*. This is because disease induced mortality selects against infected individuals, thereby reducing the probability that these individuals transmit pathogen to susceptible individuals prior to death.

### ***The effects of management***

The sensitivity analyses indicated that parameters that directly influence density-dependent processes had a strong influence on model results. Therefore, it is likely that manipulations to host density due to management practices will substantially influence model outcomes. Managers primarily influence Chinook salmon density in Lake Michigan through stocking. There are two components to a stocking program that could influence  $R_s$  prevalence in the lake: the total number and the infection prevalence of fish stocked. We simulated three different stocking levels: 5, 8, and 10 million fish per year, which correspond to recent levels of Chinook stocking into Lake Michigan (5 million) and 1.6x and 2x increases in numbers stocked. We also examined five hatchery pathogen management scenarios (Table 2.1) that span a plausible range of prevalence of infected, stocked fish. At each stocking level we ran simulations for each of the hatchery practice scenarios (Table 2.4).

When hatchery practices are effective at limiting the prevalence of infection to 5% of that found in the parents that supply the hatcheries with gametes (scenario 1: good, stock-dependent), the resulting prevalence of exposed fish exceeds that of infected fish at all ages (Figure 2.3) when 5 million fish are stocked. A 60% increase in stocking more than doubles the prevalence of fish in both exposed and infected health classes, and at the higher stocking rate, the prevalence of infected fish approaches that

of exposed fish by age 4. This is true for both hatchery and wild Chinook salmon. The prevalence at equilibrium densities varies between hatchery and wild Chinook salmon by  $< 2\%$ .

A temporary increase in stocking (with hatchery scenario 1 still in place) led to an increase in prevalence for an extended period (Figure 2.4). When stocking was increased from 5 million to 8 million fish in years 85 and 86 of a simulation and subsequently returned to 5 million, the prevalence of infection continued to increase and peaked in year 94 (Figure 2.4). Increased host density had a temporary positive feedback effect, as evidenced by the fact that by year 91 all fish stocked in year 86 have been removed from the system so that there was no longer a direct effect from stocking; yet prevalence continued to increase. However, the overall increase in prevalence of infected fish was small. Initially, increasing stocking slightly decreases the prevalence of exposed individuals (Figure 2.4). At a higher density, fish move from the exposed to the infected class at a greater rate, but prevalence of infected fish has not yet increased enough to recruit new fish to the exposed class. More susceptible fish become exposed once the prevalence of infected fish increases.

The other four hatchery management scenarios all led to substantially greater prevalence of exposed and infected Chinook salmon in both wild and hatchery sub-populations (Figure 2.5). The differences are greatest for age-0 fish, particularly for satisfactory or poor practices, and diminish as fish age. More than two-thirds of fish are exposed or infected by age four, except for simulations conducted with good stock-dependent hatchery practices. As hatchery practices deteriorate (i.e., a higher proportion of stocked fish are infected), there is a greater difference in prevalence

between wild and hatchery raised fish, especially for the “poor” scenario, where 95% of hatchery fish were infected while only 45% of wild age 4 fish were infected. Hatchery practices that mimicked what would be expected in wild spawned fish (natural replication) produced fewer exposed and infected fish than hatchery practices that limited infection prevalence to 5%. This is due to our assumption about vertical transmission rates and may not hold if the rate were increased. For example, a 10% vertical transmission rate implies that if <50% of wild spawning fish are infected, then natural replication (scenario 3) will release fewer than 5% infected fish.

A simulated short-term deterioration of hatchery practices (Figure 2.6) had a similar, but much greater effect than increased stocking (Figure 2.4) on prevalence patterns (note the difference in the y-axis between Figures 2.4 and 2.6). A change in hatchery practices in years 85 and 86 from good stock-dependent to poor led to a sudden change in the distribution of fish within health classes. By year 86 there was a large increase in the density of exposed fish, but by year 88, prevalence of exposed fish began to decline. The prevalence of infected fish continued to rise until year 91, a year after fish stocked in year 86 would have died. As with increased stocking, the increase in prevalence of infected fish persisted beyond the lifespan of the directly affected (hatchery) fish, but not for as long as with the stocking rate change. This shorter lasting effect of poorer hatchery practices may be due to the high prevalence rates that are achieved – the system cannot maintain such high prevalence rates without a continued influx of infected fish. Moreover, fish density – the other factor affecting prevalence – declines as more fish experience disease-induced mortality.



Finally, we examined how the interaction of stocking, hatchery practices, and disease dynamics affect fish density (Table 2.4). From these simulations, we can make three important observations about the interaction between population size, stocking level, hatchery practices, and disease dynamics. First, increasing stocking by 100% only increases the population of age-0 fish by 28% - 32%, depending on the hatchery practice scenario. This is a consequence of density dependence in the stock-recruitment relationship - increased stocking in a system where survival from spawning to age-0 decreases with increasing spawning stock. The largest percent gain in age-0 fish was made with good stock-dependent hatchery practices because fewer of these fish are exposed to disease-induced mortality.

Second, the percent increases in age-4 fish with increases in stocking above 5 million fish are also proportionally much less than the commensurate increase in stocking. In contrast to age-0 fish, however, the greatest percent gain realized at age-4 from stocking was achieved when hatchery practices were satisfactory or poor. This counterintuitive result was due to the combined effects of density-dependent transmission and disease-induced mortality. When hatchery practices were relatively good, an increase in density due to stocking led to increased transfer of fish from susceptible to exposed and from exposed to infected states and consequently more fish died due to disease induced mortality. When hatchery practices were poor, most fish start life already infected so increased stocking rates cannot increase the rate of infection much further – the effect of increased density on the rate of infection matters less once most of the fish are infected.

Finally, improving hatchery practices resulted in the greatest number of age-4 fish overall, as would be expected. When hatchery practices were good and stock dependent, and 5 million fish were stocked, the model predicted just fewer than 2 million age-4 fish (Table 2.4). In contrast, when twice as many fish were stocked under poor hatchery practices the model only predicted 1.6 million age-4 fish. Of course this difference will ultimately depend on the actual rate of disease induced mortality, transmission, and other parameters.

We have assumed that infected Chinook salmon experience a disease-induced mortality rate that is only 0.8 of the natural mortality rate. This assumption has allowed *Rs* to persist in the system even under the best hatchery practices and low stocking levels. If the disease induced mortality rate were substantially higher, then *Rs* would die-out under the stock-dependent good hatchery practices scenario because, on average, infected fish die before they infect at least one additional fish. As hatchery practices deteriorate, *Rs* could persist in the system, but prevalence rates would remain low. Furthermore, such low disease induced mortality rates are inadequate to create a major fish-kill as was observed in the late 1980s in Lake Michigan.

In our model increases in stocking alone, and thus increases in Chinook salmon density, cannot create a fish kill. Instead, a temporary lapse in hatchery practices is required. If we were to assume that hatchery practices normally ranged between the satisfactory and good stock-independent scenarios, then a lapse to the poor hatchery practices scenario could create a substantial fish-kill, provided disease-induced mortality rates are sufficiently high. For example, increasing the disease-induced mortality rate to 3.6 times the natural mortality rate (with no change in the fishing

mortality), and assuming satisfactory hatchery practices as the *status quo*, would allow two years of poor hatchery practices to reduce the total Chinook salmon stock to 82%, 73%, 67%, 63%, 59% of the equilibrium stock size for age 0 to 4 fish respectively if 5 million fish were stocked, and 76%, 63%, 57%, 51%, 47% if 8 million fish were stocked. Assuming that hatchery practices were good as opposed to satisfactory to start with results in larger reductions in the stock, but would also result in low equilibrium prevalence of *Rs*. Larger reductions stem from the fact that better hatchery practices produce a greater equilibrium density of fish that can be subject to a fish kill. Stocking more fish also sets up a situation for larger reductions, both due to the disease dynamics and the simple fact that stocking more fish allows more fish to die during a die-off.

### ***Impact of a reservoir and whitefish***

The effect of including a reservoir was to increase the number of newly exposed host individuals per unit time. By definition a reservoir provides a higher force of infection (Caley and Hone 2005) on the target species than the target species directs upon itself at a given point in time (Horan and Fenichel unpublished). The environmental reservoir was initially calibrated so that it had a small but positive effect on the prevalence of exposed individuals at equilibrium (Figure 2.7). We then analyzed the sensitivity, using elasticity calculations, of changes in prevalence with respect to reservoir parameters for each wild Chinook salmon health class. Equilibrium prevalence was relatively insensitive (elasticities  $< |1|$ ) to changes in the reservoir parameters. However, the prevalence in the wild Chinook salmon population at equilibrium was slightly more sensitive to increases in the lake reservoir than the river

reservoir. Prevalence at equilibrium was also more sensitive to the parameter associated with pathogen release than to the parameter associated with pathogen decay.

The addition of a second host population, whitefish, further increased the prevalence of exposed and infected salmon (Figure 2.7). The whitefish population, which was assumed to interact with the Chinook salmon populations only through additions of *Rs* to the lake reservoir, had a large effect on prevalence in Chinook salmon at equilibrium. When whitefish are included, the prevalence of exposed and infected salmon approximately doubles (Figure 2.7), due to dying infected whitefish boosting the size of the lake reservoir.

The source of *Rs* in the Great Lakes is unknown. When *Rs* was discovered in the Great Lakes in 1955, it was assumed that *Rs* was introduced with imported hatchery fish eggs, though this view has come under question (Hnath and Faisal 2004). We used the model to explore the hypothesis that *Rs* historically existed in Lake Michigan at low levels, but that hatcheries and salmon introductions amplified *Rs*. There are insufficient data to formally test this hypothesis, but our model enabled us to attempt to recreate conditions necessary for it to be possible. We began by assuming a minimal, undetectable, and stable prevalence of *Rs* in whitefish (prevalence  $\ll 0.1\%$ ) with no Chinook salmon in the system. This led to a stable environmental reservoir of *Rs*. When uninfected Chinook salmon were introduced into the system two events would occur simultaneously. The stocked fish would be exposed to the existing reservoir and some would become infected. As well, they would begin to establish wild spawning populations. Subsequently, the infected Chinook salmon would begin contributing to the environmental reservoir, resulting in increased prevalence of exposed whitefish.

Under these conditions we were able to develop simulations that resulted in equilibrium levels of infection in salmon which were comparable to levels that occurred in the model when the original source of *Rs* was the hatchery. However, for *Rs* to persist in the whitefish population at undetectably low levels prior to the introduction of Chinook salmon, the whitefish to whitefish transmission rates would have to be quite small. This would result in a low prevalence of *Rs* in whitefish at equilibrium, even in the presence of salmon, which is in contrast to recent findings (M. L. Jones, unpublished data). The density of whitefish in the model is much greater than the density of Chinook salmon, therefore even when a significant number of salmon become infected they have a small impact on the reservoir relative to the whitefish population.<sup>3</sup> It is possible that *Rs* has always been part of the Great Lakes ecosystem, but the scenario we describe above (lake whitefish as a native, historic source of *Rs*) seems unlikely. There are number of alternative hypotheses, however. For example, *Rs* may have been present in whitefish at higher, detectable rates, but they were never tested for the bacterium and clinical signs of BKD were generally absent. Alternatively, *Rs* may have indeed been introduced with hatchery fish or eggs, but at a time prior to the start of modern Chinook stocking program.

## 2.5 Discussion

The results of this modeling exercise are best interpreted qualitatively due to the general lack of quantitative knowledge about parameter estimates. Nevertheless, we

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<sup>3</sup> There are certainly more whitefish than Chinook salmon in Lake Michigan in terms of numbers, but the difference in biomass may not be as great. It is possible that biomass may be more important for contribution to a reservoir. However, our model operates on numbers in a fixed volume of water (density). Whether the amount of *Rs* shed is a function of numbers or biomass is an empirical question that has yet to be answered.

feel that a number of useful inferences can be drawn about relationships among different components of the Lake Michigan *Rs* system. These inferences can help prioritize future research and recommend management experiments. Our modeling results suggest, not surprisingly, that hatcheries may play an important role in Lake Michigan *Rs* dynamics, and that density management of Chinook salmon can be important to pathogen dynamics. The role of hatcheries, however, is not independent of natural recruitment. An understanding of the interaction between wild recruitment and stocking is needed to better understand how the stocking of Chinook salmon affects disease dynamics. Finally, while a multiple species approach will be necessary to fully understand *Rs* dynamics in Lake Michigan, simpler models focusing on Chinook salmon populations alone can provide valuable insights for management.

Both Holey et al. (1998) and Hnath and Faisal (2004) have expressed concern that hatchery practices can affect *Rs* dynamics and outbreaks of BKD in Lake Michigan. Hnath and Faisal (2004) state that practices such as using Heath-type stacking incubators can contribute the spread of *Rs* between eggs. This effectively increases the rate of vertical transmission in hatcheries. The role of vertical transmission (or horizontal transmission among eggs) may not have been fully appreciated. Our results show that there are two effects of increased rates of infection in eggs. The first effect is clear; if there are more infected eggs, this will lead to more infected fish. The less obvious second effect is that age-0 that are born infected (either due to vertical transmission or hatchery induced egg-to-egg transmission), represent an additional source of exposure for susceptible wild and hatchery age-0 fish. Our model also suggests that even short-term failures in hatchery procedures that result in releases

of a high proportion of infected fish can lead to a drastic and prolonged rise in infection of both wild and hatchery salmon. Finally, increasing stocking rates are not likely to overcome poor hatchery practices with respect to density of older Chinook salmon, due to disease induced mortality and density-dependent compensation in wild recruitment (Table 2.4).

Increasing natural production of Chinook salmon in Lake Michigan complicates management of *Rs*. Managers would benefit from a better understanding of the recruitment dynamics of Lake Michigan wild Chinook salmon. Specifically, it would be useful to know if stocking of Chinook salmon leads to compensation in wild production at current abundance levels – as we assumed in our models. The density of Chinook salmon also affects *Rs*-host dynamics. Thus, managers need to account for the response of wild Chinook salmon when making stocking decisions both for disease and production objectives. Because of compensatory processes, it is possible that a reduction in stocking may not reduce the number of fish in the lake and may free resources to improve remaining hatchery conditions. Finally, wild spawning fish may be less likely to create infected eggs than hatcheries. Hamel (2005) reports that it is common for Pacific hatcheries to produce eggs with a 90% prevalence of *Rs*.

A variety of fish species in Lake Michigan may carry *Rs*. Thus, to understand the ecology of pathogens alternative hosts often need to be considered (Dobson 2004). Dividing Chinook salmon into wild and hatchery is the first step in a multiple host approach. The concept of “pathogen spillover” from a reservoir to a host of interest has begun to receive attention (Daszak *et al.* 2000; Power and Mitchell 2004). The inclusion of an environmental reservoir is used in part to capture potential *Rs* dynamics

in other species, but is only a first approximation of how *Rs* dynamics in other species might affect Chinook salmon. To fully understand how Chinook salmon, *Rs*, and other species interact, the dynamics of those other species will need to be considered explicitly.

It would be valuable for managers to understand how stocking contributes to the spread and maintenance of *Rs* in Chinook salmon. Our modeling has highlighted an indicator that may be useful for determining the relative role of density and hatchery practices. If there are significant differences in prevalence between hatchery and wild-reared Chinook salmon, hatchery practices likely have a large affect on *Rs* dynamics, whereas similar prevalence would imply that density has a greater impact on *Rs* dynamics

## **2.6 Conclusion and Research Recommendations**

Models that integrate population and epidemiological theory seldom have been used to improve understanding and management of fish populations or fish health, and pathogen and disease models have not been explicitly considered in the leading fish population dynamics texts (Hilborn and Walters 1992; Quinn and Deriso 1999). These texts acknowledge the importance of disease in shaping mortality, but simply lump disease induced mortality into unmanageable natural mortality. Moreover, such natural mortality estimates are applied to the entire population (combining health classes) and are often assumed constant for older fish. This implies that the proportion of fish in a specific health class does not change. For some populations mortality may change substantially as a result of host-pathogen dynamics. This is especially a concern in



systems where a substantial number of fish are stocked, or where invading species might be bringing with them new pathogens. More careful consideration of the role of host-pathogen dynamics is merited in these cases, and the development of models is critical to “building an ecological approach to fish health management,” (Stephen and Thorburn 2002). In this paper we have introduced a mechanistic approach for modeling the role played by pathogens in shaping fish population dynamics.

Models enable the organization of information that allows formulation of meaningful hypotheses and identification of priority research. In the absence of good empirical data upon which to base parameter estimates, it is best to emphasize the qualitative behavior of the models and their sensitivity to a range of plausible parameter values. Such qualitative conclusions nevertheless provide a strong basis for determining critical areas of uncertainty and thus priorities for future research. Accordingly, we recommend seven priorities for improving understanding of *Rs* dynamics in Lake Michigan. We suspect that these recommendations would be applicable to other fishery-pathogen systems as well, particularly where hatcheries are used to supplement fishing opportunities.

First, from our results we hypothesize that if hatchery practices, *i.e.* the proportion of infected fish being released from hatcheries, play an important role in pathogen dynamics, then prevalence in hatchery reared fish should be substantially higher than prevalence in wild reared fish. A method of discriminating hatchery from wild fish, such as tagging or marking hatchery fish or the use of natural chemical markers, would prove valuable for such comparisons.

Second, it is imperative that researchers measure the stock-recruitment relationship of wild spawning Chinook salmon in Lake Michigan. The prevalence for all but one health-age class was most sensitive to the density-dependence parameter in the Ricker function. It is important for managers to have better idea about the three-way interaction between wild recruitment, stocking, and disease transmission, how the level of stocking influences wild recruitment. Moreover, this is a critical element to understanding vertical transmission – the next research recommendation.

Third, the role of vertical transmission should be further examined. Assessing size of runs of wild spawning Chinook salmon and testing resulting eggs would provide information both about wild recruitment and vertical transmission in the wild. Alternatively, 5- month old wild-spawned smolts could be collected and examined for  $R_s$ , and prevalence rates compared to those being released from hatcheries. Ultimately, prevalence goals for hatcheries will need to be connected to vertical transmission in the wild.

Fourth, the model we used to represent the hypothesized link between nutritional stress and the advancement (function  $Q$ ) of Chinook salmon hosts from exposed to infected states (Holey et al. 1998), needs to be further investigated.  $R_s$  dynamics can be sensitive to parameters in the  $Q$  function, and little is known about this relationship other than the general observation of increased prevalence of BKD when Chinook salmon growth rates were low in the late 1980s. The predator-prey dynamics of Lake Michigan salmonines have been the subject of considerable study (Szalai 2003, Madenjian et al. 2005, also see chapter 4), but this needs to be coupled with a better mechanistic understanding of how nutritional stress affects  $R_s$  dynamics.

Fifth, we urge a more structured approach to assessing fish health at the population level. It is imperative that sampling be designed to estimate epidemiological parameters, which cannot rely on sampling opportunistically nor testing only those fish contributing gametes to hatchery facilities. Stocks that are regularly assessed may have sampling designs adequate for fish health assessment already in place. Additionally, integrating stock assessment models with epidemiological models could facilitate estimation of transmission parameters. Statistical approaches that integrate stock and health assessment should be the subject of future research, but improved health assessment sampling designs can yield a wealth of information prior to the development of methods for full integration of stock and health assessment.

Sixth, the feasibility of testing museum specimens of whitefish and other potential hosts such as lake trout and other Coregonid species for presence of *Rs* (nucleic material) should be investigated. The detection of *Rs* in whitefish that pre-date modern salmon introductions (or even the BKD outbreak in the 1980s) would provide insight into the ecological role of *Rs*. Our model, along with recent data (unpublished) indicates that if *Rs* historically had been present in whitefish, prevalence rates would have been reasonably high.

Finally, although much can be learned from studies focused on Chinook salmon, our model suggests that other species could play an important role in the *Rs* dynamics in Lake Michigan, even if they do not directly interact with Chinook salmon as predators, competitors or prey. We found that *Rs* dynamics in Chinook salmon can depend on the contribution of environmental reservoirs as a source of the pathogen, and other abundant species that harbor the pathogen could play an important role in

determining the dynamics of this reservoir. At a minimum it would be valuable to determine how widespread  $R_s$  is in aquatic species that are either abundant or interact strongly with Chinook salmon, or both. Two obvious candidates would be sea lampreys and alewives.

Alone, models cannot resolve all fishery management questions. However, models are a necessary component of fishery management and fishery research. Models play a major role in stock assessment and harvest policy (Hilborn and Walters 1992; Quinn and Deriso 1999), but have played virtually no role in fish health management. In the future, we hope that the same analytical rigor that has been applied to other areas of fisheries management is applied to fish health. In this paper, we have developed such a model to address the effects of a pathogen on a fishery. We hope that this model can serve as an example for addressing other emerging and chronic fish health issues.

**2.7 Appendix.** Parameters and values used in the model; the same values were used for both wild and hatchery Chinook salmon.

	<b>Parameter values</b>				
	Base case	With disease induced mortality and vertical transmission	Increase disease induced mortality	With whitefish and the reservoir	Alternative whitefish hypothesis
<b>Chinook Salmon Parameters</b>					
Ricker $\alpha$ , age 0	0	0	0	0	0
Ricker $\alpha$ , age 1	0	0	0	0	0
Ricker $\alpha$ , age 2	1	1	1	1	1
Ricker $\alpha$ , age 3	5	5	5	5	5
Ricker $\alpha$ , age 4	10	10	10	10	10
Ricker $\gamma$	0.39	0.39	0.39	0.39	0.39
Instantaneous fishing mortality, age 0	0	0	0	0	0
Instantaneous fishing mortality, age 1	0	0	0	0	0
Instantaneous fishing mortality, age 2	0.1	0.1	0.1	0.1	0.1
Instantaneous fishing mortality, age 3	0.15	0.15	0.15	0.15	0.15
Instantaneous fishing mortality, age 4	0.2	0.2	0.2	0.2	0.2
Instantaneous natural mortality, age 0	0.4	0.4	0.4	0.4	0.4
Instantaneous natural mortality, age 1	0.25	0.25	0.25	0.25	0.25
Instantaneous natural mortality, age 2	0.25	0.25	0.25	0.25	0.25
Instantaneous natural mortality, age 3	0.25	0.25	0.25	0.25	0.25
Instantaneous natural mortality, age 4	0.25	0.25	0.25	0.25	0.25
Instantaneous disease induced mortality	0	0.2	0.9	0.2	0.2
Transmission coefficient, $\beta$ , between Chinook	0.11	0.11	0.11	0.11	0.11
Vertical transmission rate	0	0.1	0.1	0.1	0.1
$q_{eq}$	2.5	2.5	2.5	2.5	2.5
$q_{50\%}$	140	140	140	140	140
Probability of infection from reservoir	0	0.11	0.11	0.11	0.11

**2.7 Appendix continued.** Parameters and values used in the model; the same values were used for both wild and hatchery Chinook salmon.

	Base case	With disease induced mortality and vertical transmission	Increase disease induced mortality	With whitefish and the reservoir	Alternative whitefish hypothesis
<b>Whitefish Parameters</b>					
Ricker $\alpha$ , stage 0	0	0	0	0	0
Ricker $\alpha$ , stage 1	0	0	0	7.25	7.25
Ricker $\gamma$	0	0	0	0.015	0.015
Instantaneous fishing mortality, age 0	0	0	0	0	0
Instantaneous fishing mortality, age 1	0	0	0	0.34	0.34
Instantaneous natural mortality, age 0	0	0	0	0.4	0.4
Instantaneous natural mortality, age 1	0	0	0	0.25	0.25
Instantaneous disease induced mortality	0	0	0	0.2	0.2
Transmission coefficient, $\beta$ , between whitefish	0	0	0	0.01	0.00425
Vertical transmission rate	0	0	0	0.1	0.1
$q_{eq}$	0	0	0	200	200
$q_{50\%}$	0	0	0	800	800
Probability of infection from reservoir	0	0	0	0.0035	0.0035
<b>Reservoir Parameters</b>					
Loading rate (river)	0	0	0	0.05	0.05
Exponential lose rate (river)	0	0	0	0.75	0.75
Loading rate (lake)	0	0	0	0.05	0.05
Exponential lose rate (lake)	0	0	0	0.75	0.75

## 2.8 Tables

Table 2.1. Hatchery practice scenarios used to describe the potential output of infected fish by hatcheries.

Name	Description
1. Good – stock dependent	The prevalence of <i>Rs</i> in stocked fish is 5% of prevalence seen in wild spawned fish. Chosen as an ideal outcome.
2. Good – stock independent	If there is at least one infected fish in the wild population, then the prevalence of <i>Rs</i> in stocked fish is 5%. Chosen based on standard hatchery protocol.
3. Natural Replication	The prevalence of <i>Rs</i> in stocked fish is equal to the prevalence seen in wild spawned fish.
4. Satisfactory	If there is at least one infected fish in the wild population, then the prevalence of <i>Rs</i> in stocked fish is 19%. Chosen based on what is believed to be the historical maximal tolerance for hatchery infection in Michigan.
5. Poor	If there is at least one infected fish in the wild population, then the prevalence of <i>Rs</i> in stocked fish is 95%. Chosen to represent hatcheries with no biosecurity (based on Hamel 2005).

Table 2.2. Elasticity results for wild Chinook salmon without vertical transmission by age and health class. Elasticity is the percent change in the prevalence of a given health class at age divided by 5% (the amount by which the parameter value was increased). If the elasticity  $> |1|$ , then the model is deemed to be elastic and sensitive to changes in the parameter; likewise, if elasticity  $< |1|$ , then the model is deemed to be insensitive to changes to the parameter.

Parameter	Health Class	Age Class				
		0	1	2	3	4
Ricker density-dependence parameter, $\gamma$	<i>E</i>	-12.68	-12.24	-11.77	-11.27	-10.71
	<i>I</i>	-12.95	-12.74	-12.43	-12.14	-11.85
Disease transmission parameter, $\beta$	<i>E</i>	10.24	8.88	7.61	6.39	5.23
	<i>I</i>	10.24	9.44	8.59	7.84	7.16
Advancement parameter, $q_{50\%}$	<i>E</i>	-7.55	-7.10	-6.61	-6.09	-5.54
	<i>I</i>	-8.14	-7.89	-7.59	-7.30	-7.02
Advancement (equilibrium) parameter, $q_{eq}$	<i>E</i>	-1.02	-0.90	-0.82	-0.73	-0.63
	<i>I</i>	-1.11	-1.17	-1.04	-0.96	-0.90



Table 2.3. Elasticity results for wild Chinook salmon with vertical transmission by age and health class. Elasticity is the percent change in the prevalence of a given health class at age divided by 5% (the amount by which the parameter value was increased). If the elasticity  $> |1|$ , then the model is deemed to be elastic and sensitive to changes in the parameter; likewise, if elasticity  $< |1|$ , then the model is deemed to be insensitive to changes to the parameter.

Parameter	Health Class	Age Class				
		0	1	2	3	4
Ricker density-dependence parameter, $\gamma$	<i>E</i>	-3.77	-2.77	-1.81	-0.88	0.03
	<i>I</i>	-3.73	-3.64	-3.24	-2.83	-2.45
Disease transmission parameter, $\beta$	<i>E</i>	3.03	2.01	1.17	0.48	-0.09
	<i>I</i>	2.42	2.24	1.87	1.53	1.23
Advancement parameter, $q_{50\%}$	<i>E</i>	-0.20	-0.10	-0.04	0.01	0.07
	<i>I</i>	-0.23	-0.28	-0.22	-0.18	-0.15
Advancement (equilibrium) parameter, $q_{eq}$	<i>E</i>	-1.78	-1.18	-0.58	-0.01	0.54
	<i>I</i>	-2.25	-2.18	-1.94	-1.70	-1.47
Vertical transmission parameter, $\nu$	<i>E</i>	0.48	0.32	0.18	0.06	-0.04
	<i>I</i>	0.80	0.59	0.45	0.35	0.28

Table 2.4. The equilibrium number of wild + hatchery Chinook salmon, by age, resulting from 15 combinations of between stocking levels and hatchery practices. See Table 2.1 for definitions of each hatchery practice.

Stocking level	Age					% gain at age 4 above stocking 5 million fish
	0	1	2	3	4	
<b>1 - good stock dependent</b>						
5 million	8.926	6.907	4.812	3.171	1.976	
8 million	10.662	8.066	5.421	3.405	2.004	1.4%
10 million	11.765	8.785	5.803	3.578	2.067	4.6%
<b>2 - good stock-independent</b>						
5 million	9.042	6.897	4.705	3.013	1.814	
8 million	10.698	8.011	5.323	3.305	1.925	6.1%
10 million	11.771	8.711	5.707	3.495	2.008	10.7%
<b>3 - natural replication</b>						
5 million	9.007	6.910	4.751	3.073	1.871	
8 million	10.696	8.016	5.330	3.312	1.930	3.2%
10 million	11.771	8.705	5.701	3.489	2.004	7.1%
<b>4 - satisfactory</b>						
5 million	9.078	6.803	4.544	2.846	1.674	
8 million	10.686	7.851	5.128	3.139	1.806	7.9%
10 million	11.722	8.547	5.537	3.361	1.918	14.6%
<b>5 - poor</b>						
5 million	8.853	6.315	4.047	2.448	1.400	
8 million	10.362	7.242	4.550	2.701	1.517	8.3%
10 million	11.347	7.847	4.879	2.868	1.595	13.9%

2.9 Figures

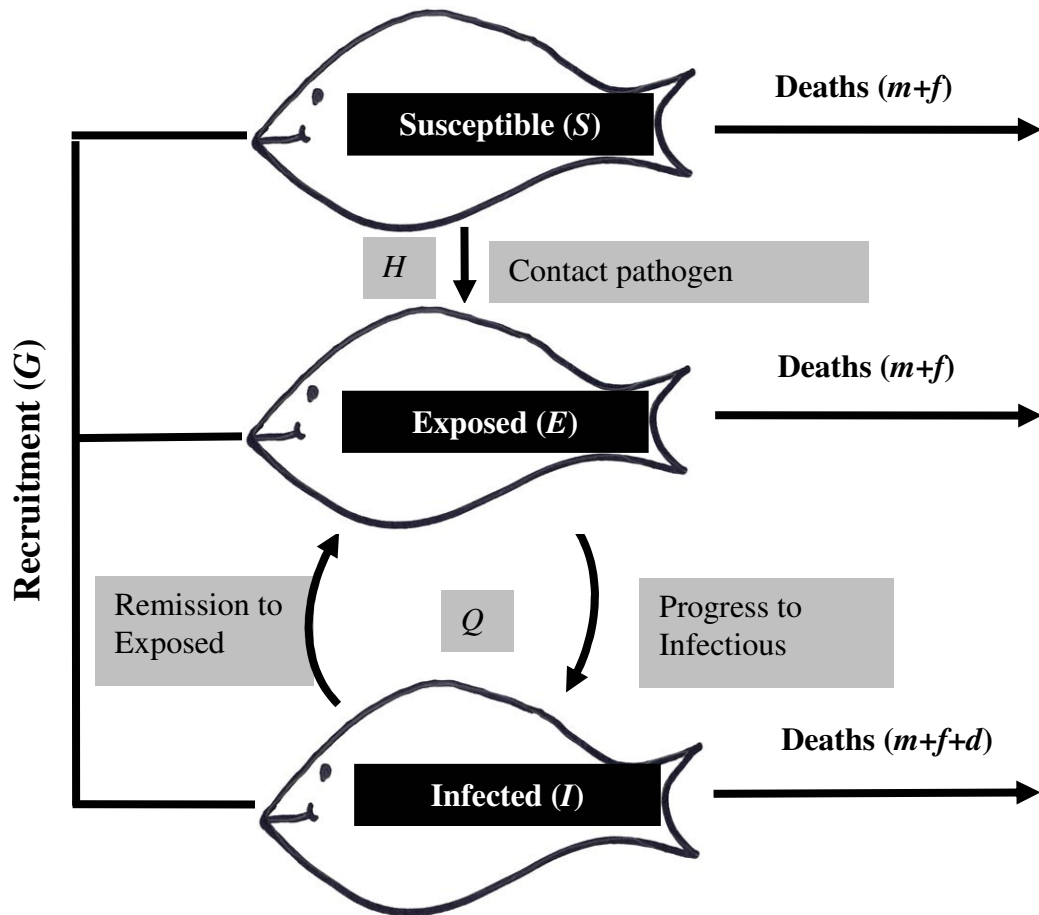


Figure 2.1. A conceptual model of the SEIE system

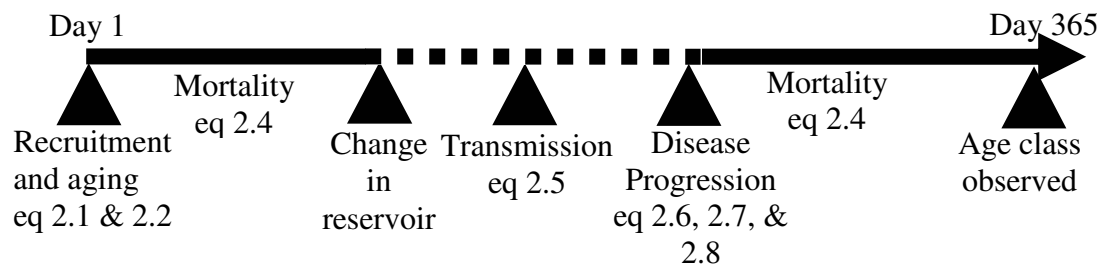


Figure 2.2. The sequence of events throughout a simulated year. Mortality is applied over the solid line. Equations corresponding to processes in the diagram are noted.

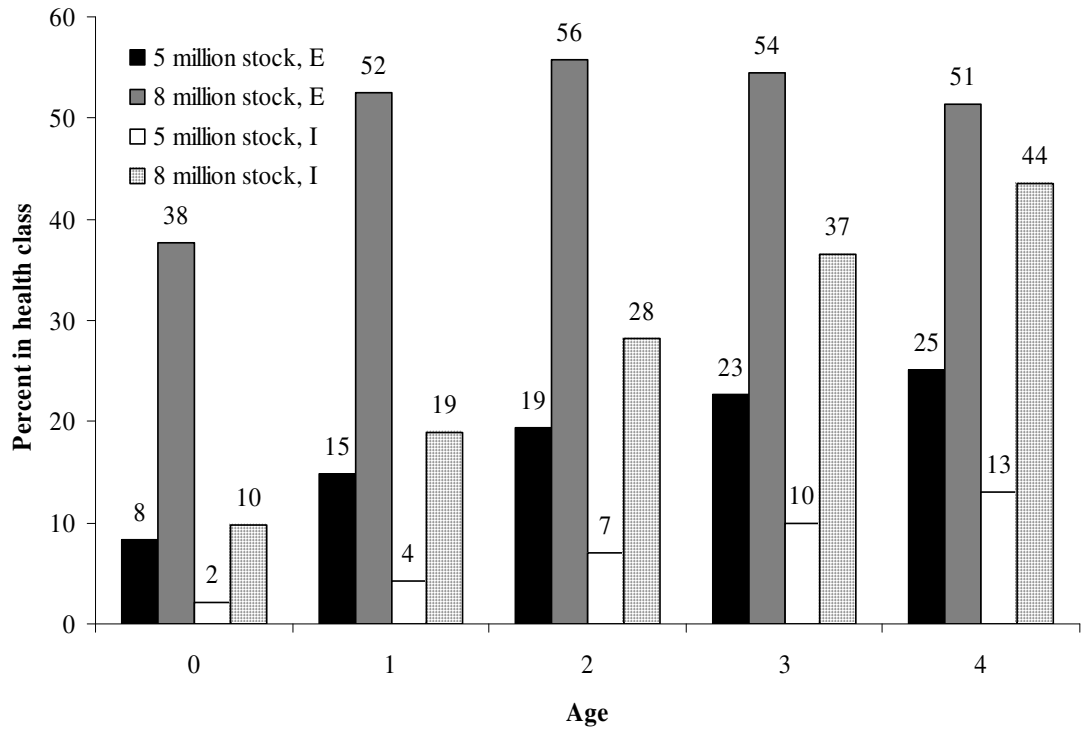


Figure 2.3. The percent of the wild Chinook population in exposed and infected health classes, by age, for two different stocking levels at equilibrium.

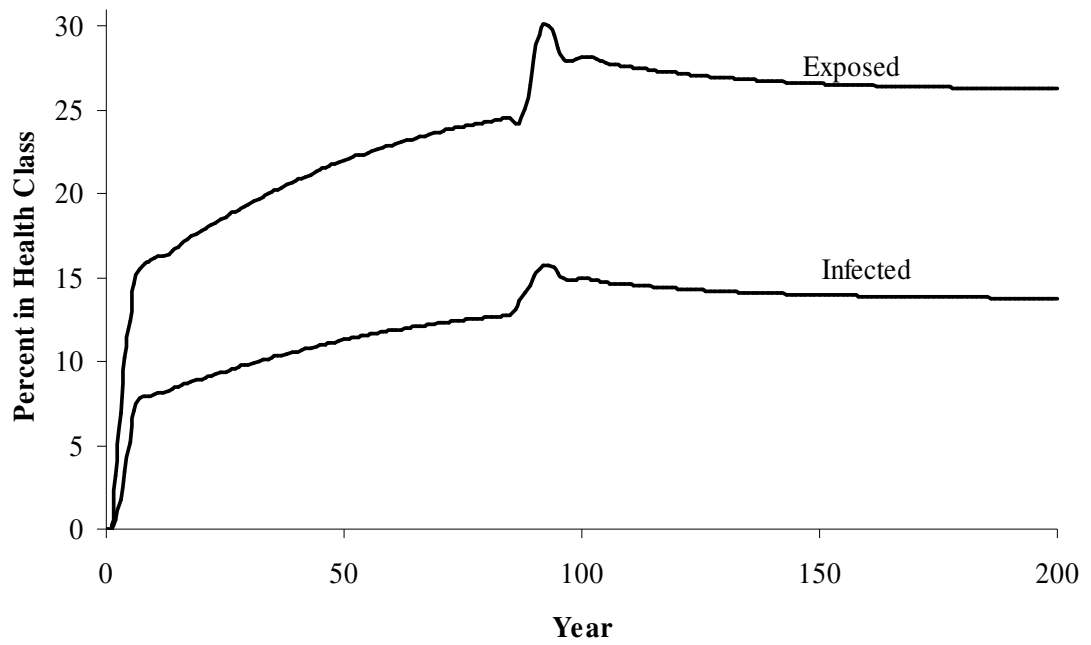
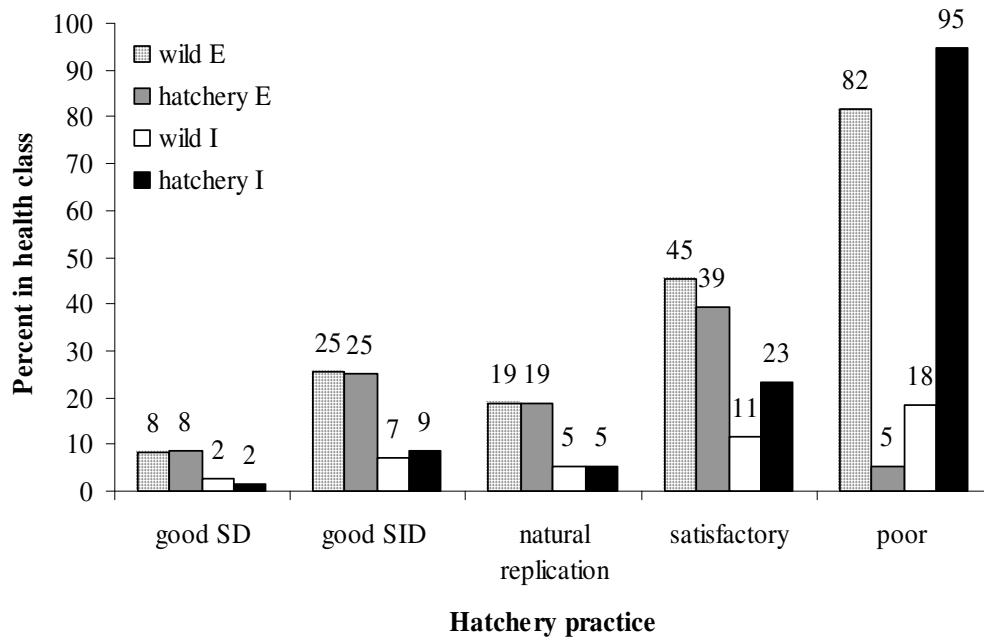
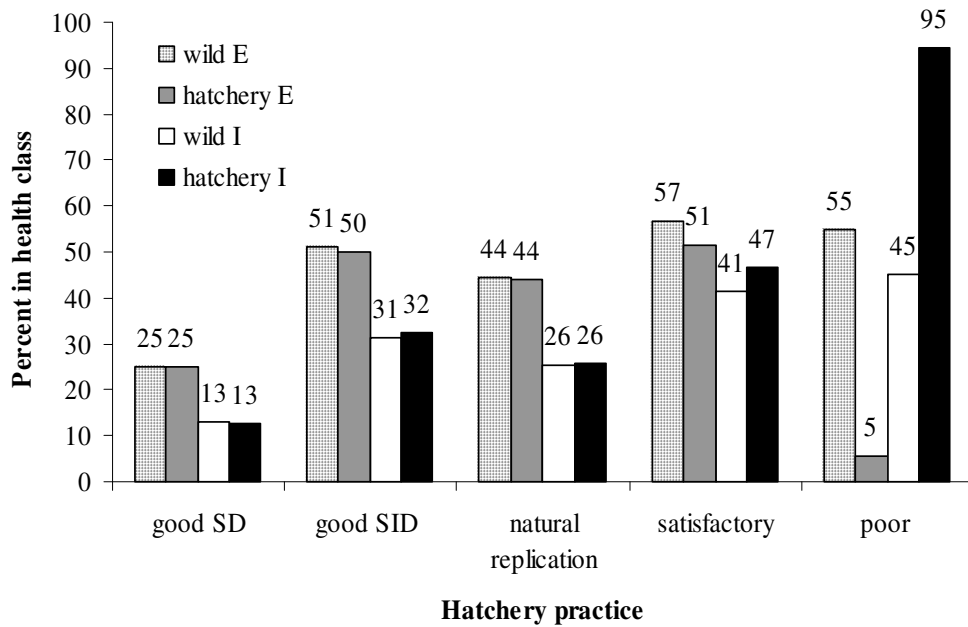


Figure 2.4. A 200-year simulation of wild Chinook exposed and infected prevalence at age-4. Stocking is increased from 5 to 8 million for two years starting in year 85.



Panel A.



Panel B.

Figure 2.5. The percent of the population in the exposed and infected health classes by hatchery practice at equilibrium. Panel A shows age-0 fish and panel B shows age-4 (SID = stock independent, SD = stock dependent).

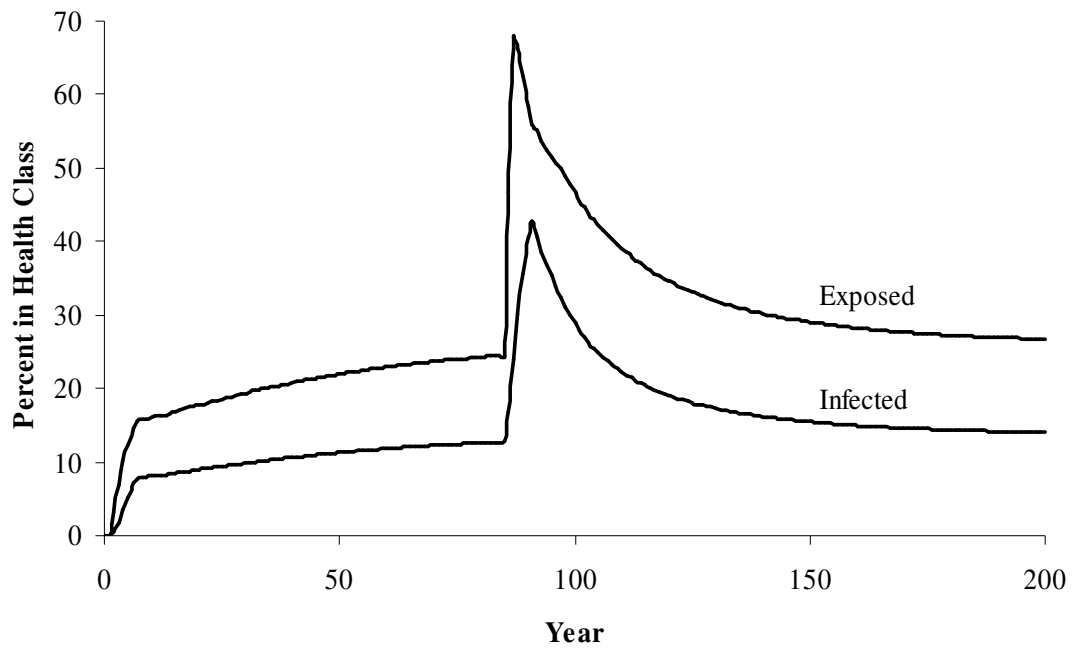


Figure 2.6. A 200-year simulation of wild Chinook exposed and infected prevalence at age 4. Hatchery practices are changed from good stock-dependent to poor for two years, starting in year 85.



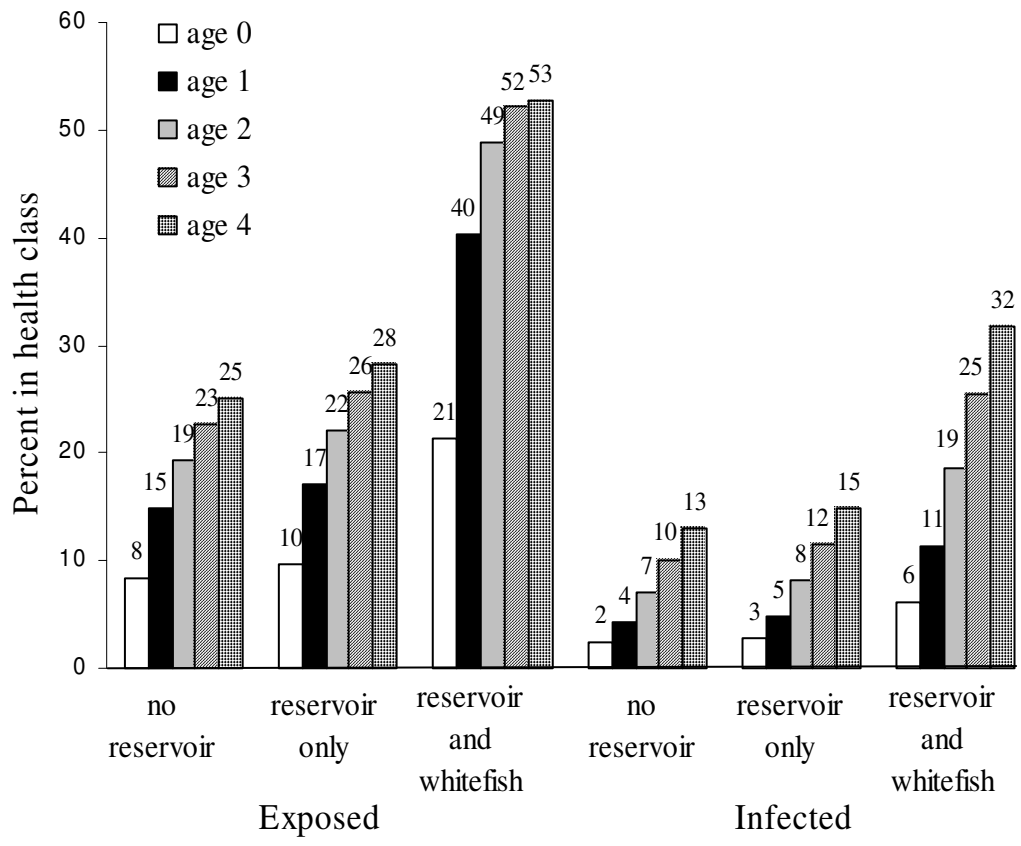


Figure 2.7. The percent of the wild Chinook population in exposed and infected health classes, among ages, and the effects of adding an environmental reservoir and whitefish to the model at equilibrium.

## CHAPTER 3

# FISH PATHOGEN SCREENING AND ITS INFLUENCE ON THE LIKELIHOOD OF ACCIDENTAL PATHOGEN INTRODUCTION DURING FISH TRANSLOCATIONS

### 3.1 Introduction

Fisheries managers often employ fish translocations to help achieve a variety of objectives, including the improvement or rehabilitation of existing stocks, the establishment of populations in new areas, the reintroduction of extirpated stocks, and for use as biological pest control agents.<sup>1</sup> Fish translocations, however, have been implicated in undesirable and costly pathogen translocations (Stewart 1991; Reno 1998; Murray et al. 2002). Consequently, there is increasing interest in conducting risk analyses to evaluate the tradeoffs between stock improvement and disease risk (Stephen 2001; Bartley et al. 2006). Pathogen transfer risk is influenced by human decisions and management actions in addition to biological and environmental factors (Shogren and Crocker 1999; Caley et al. 2006), and therefore key components to managing and evaluating risk are programs that influence managers' decisions, such as pre-translocation screening of fish.

Pathogen screening does not eliminate risk and imposes additional costs on fish translocation programs. First, there is the marginal cost of screening each additional individual fish (e.g., diagnostic tests and opportunity cost of the agency personnel). Second, many aquatic animal screening programs require the sacrifice of the screened individuals, thereby reducing the number of individuals left for transfer. Third, the

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<sup>1</sup> This paper focuses on fish translocation; however, the techniques and conclusions also apply to hatchery management programs.

extra time that fish are held awaiting screening results prior to transfer can reduce their survival. Given the benefits and costs of a screening program, an important question is, “How should a screening program be designed?” Mathews et al. (2006) advocate full veterinary screening of all mammals that are to be translocated, but acknowledge that such screening is unlikely to be feasible, especially in the case where screened individuals must be sacrificed, and is likely to be extremely costly. A common approach in fisheries is to screen subsamples of the groups of organisms slated for transfer (USFWS and AFS-FHS 2005). This often involves sacrificing the screened individuals.

Managers interested in designing screening programs to help evaluate and manage risk need to consider two questions: i) how does the number of fish screened affect the likelihood that a pathogen is translocated; and ii) how long should a screening program continue if no pathogens are found? The literature mostly has focused on statistical approaches for estimating prevalence from increasingly complex sampling designs (Williams and Moffitt 2001; Munoz-Zanai et al. 2006), and less on how such estimates can be used to manage risk.

In this paper, we review screening design and explain the implications of pathogen screening for use in fish health risk analysis (not to be confused with surveillance). We emphasize the relationship between screening assumptions and objectives, while addressing the two questions raised above. We illustrate key concepts using a case study of sea lamprey (*Petromyzon marinus*) translocations in the Great Lakes; these are undertaken as part of the sterile male release method for biological control in the St. Marys River integrated sea lamprey management program (Schleen et

al. 2003; Twohey et al. 2003). We provide a critique of this screening program's implementation.

Ultimately, aquatic pathogen screening programs need to be designed to achieve 'acceptably low' probabilities of pathogen *translocation*. An interdisciplinary approach (e.g., a structured decision analysis *sensu* Peterman and Anderson 1999) is needed to determine the acceptable probability of pathogen translocation and overall risk. Such an analysis is beyond the scope of this paper. We present a narrower analysis that clarifies the linkage among screening program design, assumptions, likelihood of pathogen transfer, and risk analysis.

### **3.2 How Many Fish to Screen for Unwanted Pathogens**

Verifying the absence of a pathogen is all but impossible; given a probability  $> 0$  that the organism is present, the harder one looks, the more likely one is to find an organism, (Regan et al. 2006). Consider the situation in which a batch of fish is collected for the purpose of transfer. A subsample of fish drawn from the batch is first screened for pathogens. Assume that if an infected fish is detected in the subsample, then the remainder of the batch will not be transferred.<sup>2</sup> The transfer proceeds if no infected fish are detected in the subsample. Assume that screened fish are scarified and not transferred. Further, assume that the subsample of fish represents (i.e., is a random sample of) the transfer batch. That is, whether any given fish hosts a pathogen is

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<sup>2</sup> This is a fundamental assumption in our analysis. It is, however, necessary to assume that some probability of pathogen transfer is acceptable. Therefore, it is conceivable that prevalence in the source population would still be acceptably low such that the transfer would proceed when a small number of fish in a large sample test positive. Williams and Moffitt (2001) and Munoz-Zanzi et al. (2006) provide techniques for estimating prevalence and confidence bounds when a proportion of fish in a sample test positive. We believe, however, that our assumption is typical of the way most screening programs operate.

independent of whether any other fish hosts a pathogen, and the probability that any given fish hosts a pathogen is the same, (i.e., identical), regardless of whether the fish comes from the subsample or the batch that is transferred.

A simple decision tree (Figure 3.1) illustrates that the relevant statistic is the probability that a pathogen is present among the fish released, given that the pathogen was not detected within the subsample. Therefore, the probability of transferring a pathogen given screening is

$$(3.1) \quad \Pr(t) = [1 - \Pr(d)] * \Pr(b),$$

where  $\Pr(d)$  is the probability of detecting the pathogen during screening, and  $\Pr(b)$  is the probability that the transfer batch contains at least one infected fish.

To calculate the probability of failing to detect a pathogen (i.e.,  $[1-\Pr(d)]$ ) in a given sample, the Poisson or binomial distribution can be used (Green and Young 1993; Dell’modame and Prati 2005). The Poisson distribution approximates the binomial when the population (batch) is large, but for small batches, representative sampling assumptions for both approaches may be violated (Dell’modame and Prati 2005). We base our analysis on the Poisson distribution, but see endnotes for analogous equations for the binomial distribution. The necessary sample size,  $n$ , for detecting at least one infected fish from a population with prevalence  $m$  at a specified confidence level (probability),  $x$  is:<sup>3</sup>

$$(3.2) \quad n = -\ln(1-x)/m.$$

The true prevalence of pathogens in a wild population is often unknown and must be assumed. This assumption can greatly affect the likelihood of translocating at least one

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<sup>3</sup> The formula using the binomial distribution is  $n = \ln(1-x)/\ln(1-m)$ .

infected fish. As the assumed prevalence level decreases, the number of fish that needed to be tested to achieve a given confidence of detection increases at a rapidly increasing rate, going to infinity as the assumed prevalence declines to zero (Figure 3.2). Similarly, as the desired confidence level increases, the number of fish that need to be tested also increases rapidly.

Equation 3.2 can be rearranged to provide the probability of detecting at least one infected individual in a sample of size  $n$  from a population with a known (or assumed) prevalence  $m$ :<sup>4</sup>

$$(3.3) \quad x = 1 - \exp(-mn) .$$

Thus, the probability of failing to detect a pathogen is  $\Pr(d) = 1 - x$ .

Equations (3.2) and (3.3) implicitly assume that the screening instrument's diagnostic specificity and sensitivity are perfect and equal to 1 (i.e., that infected animals always test positive and uninfected animals always test negative). Equations (3.2) and (3.3) can be generalized to account for the probability of obtaining a false negative,  $p$ , and a false positive,  $q$ . Equation (3.2) provides the necessary sample size if one requires confidence  $x$  given *observable* prevalence  $m$ . If there is a known bias in the observed prevalence, resulting from an imperfect test specificity or sensitivity, then  $m$  in equations (3.2) and (3.3) is replaced with *observable* prevalence

$$(3.4) \quad m' = m(1 - p) + (1 - m)q .$$

If only false negatives are considered ( $q = 0$ ), then observable prevalence decreases, resulting in the need for a larger  $n$ . When only false positives are considered ( $p = 0$ ), the observable prevalence increases reducing  $n$ . Issues regarding sensitivity and

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<sup>4</sup> The formula using the binomial distribution is  $x = 1 - (1 - m)^n$ .

specificity of tests are addressed in more depth by Dell’Omodarme and Prati (2005) and Munoz-Zanzi et al. (2006).

Equation (3.3) also allows us to calculate the probability that a pathogen is present in the translocation batch by replacing the subsample size with the number of (untested) fish in the batch to be transferred (note that  $m$ , and not  $m'$ , should be used for this calculation). Table 3.1 provides calculated probabilities for a range of prevalence levels and sample sizes. For all prevalence levels, as the number of individuals screened or in the batch increases, so does the probability that at least one infected fish will be detected in the screening subsample or will be present in the transfer batch. For example, if prevalence is 5%, then the probability of detecting at least one infected fish in the subsample increases from 0.78 to nearly 1.0 as the sample size increases from 30 to 200 fish.

When the prevalence of a pathogen is high, the situation is straightforward; infected fish will be detected during screening and the translocation will be halted (Figure 3.1, top branch and Figure 3.3). Similarly, when the actual prevalence is near zero, the situation is again straightforward; the pathogen is unlikely to be detected in the screened sample and is unlikely to be present the translocation batch. In such a case, the fish transfer proceeds with a low likelihood of pathogen transfer (Figure 3.1, bottom branch and Figure 3.3). Moderately low prevalence rates (approximately, < 0.05) present a more challenging situation, since the pathogen may go undetected in the subsample, but is likely to be present in the larger transfer batch (e.g., when prevalence = 0.01 and sample size = 60, there is a 45% chance that an infected fish is in the transfer batch – see the middle branch of Figure 3.1, and Figure 3.3).

All else equal, screening smaller subsamples results in higher probabilities of translocating at least one infected individual over a wider range of prevalence. The ability to decrease the likelihood of a translocation event by increasing subsample size, however, decreases as prevalence increases. If prevalence is sufficiently high, then smaller subsamples are nearly as likely to detect a pathogen as are larger subsamples (Figure 3.3). Increasing subsample size generates the greatest effect at moderately low prevalence levels (Figure 3.3).

Figure 3.4 illustrates the interaction between numbers of fish screened and numbers of fish transferred, assuming a given true source prevalence (solid lines, true prevalence = 0.05; dashed lines, true prevalence = 0.01). The figure shows that the probability of transferring an infected fish decreases as the number of fish screened increases, for a given number of fish in the transfer batch. The probability of transferring an infected fish increases, however, as the number of fish transferred increases, for a given number of fish screened. Decreases in true prevalence increase the probability of transferring an infected fish for a given number of fish screened and a given number of fish transferred. This is illustrated by considering the situation where 60 fish are screened and 240 fish are transferred (point A on Figure 3.4). If the true prevalence were 5%, there is a 5% chance of transferring an infected fish, whereas if the true prevalence were 1% then the likelihood of transferring an infected fish climbs to 50% (Figure 3.4).

The utility of a screening program for preventing the transfer of an infected fish depends on pathogen prevalence. In the absence of a screening program, high prevalence is more likely to result in a transfer because at low prevalence there is a



lower probability that infected individuals are in the batch. However, as the number of fish screened increases, the isoclines in Figure 3.4 cross, because at high prevalence screening is likely to detect infected fish and thus stop the translocation.

### 3.3 How Long to Continue Screening for Pathogens

At some point the aquatic pathogen screening program should be terminated if pathogens are not found in multiple successive screening events and there is no evidence that a new (existing) pathogen has been introduced (re-introduced). Regan et al. (2006) develop a framework for evaluating invasive plant search programs that is applicable to this pathogen screening question. The length of time a screening program should persist, given that no infected individuals are found, should be based on three factors: i) the cost of the screening program,  $CS$ ; ii) the expected cost of a pathogen translocation,  $CT$ ; and iii) the probability of facilitating a pathogen translocation ( $\text{Pr}(t) \neq 1$ ). Regan et al. (2006) offer a ‘rule of thumb’ calculation that balances these factors:

$$(3.5) \quad y = \ln\left(\frac{-CS}{CT * \ln(\text{Pr}(t))}\right) / \ln(\text{Pr}(t))$$

where  $y$  is the number of screening events (given a constant interval) that screening should continue when no pathogen is being detected. This criterion can be applied to screening programs and ‘disease free’ certification programs. This approach assumes that there are no other data that conflict with the results of the screening program, i.e., new accounts of pathogen introductions. This rule implies that when the expected damages from translocation are small,  $CT < -CS/\ln(\text{Pr}(t))$ , then no screening should take place.

### 3.4 A Case Study

We now present the case of sea lamprey transfers to illustrate screening program design considerations for risk management and demonstrate the application of the methods discussed above. Sea lampreys are parasitic fish that invaded the North American Great Lakes during the early 20<sup>th</sup> century and have been the object of a pest control program since 1955 (Smith and Tibbles 1980). The sterile male release technique (SMRT) is a component of the sea lamprey control strategy for the St. Marys River, which is a major sea lamprey spawning area and therefore an important source of parasitic sea lamprey for Lake Huron (Schleen et al. 2003). The SMRT provides social and economic benefits through increased production of lake trout (*Salvelinus namaycush*) (Lupi et al. 2003). Sterilized male sea lampreys are released to compete with wild fertile males to attract female mates, thereby reducing female reproductive output. A high ratio of sterile to fertile males is desirable for cost-effective control of sea lampreys (Haeseker et al. in press). Managers go to considerable effort to obtain adult male sea lampreys for sterilization. Greater than 85% of the collection comes from upper Great Lakes sources close to the St. Marys River (Twohey et al. 2003), but some sea lampreys used in the SMRT program are collected in Lake Ontario, which is isolated from the upper Great Lakes by Niagara Falls.

Interest in screening sea lampreys for pathogens before translocation, to prevent unwanted introductions, has intensified due to the discovery of “pathogens of concern” in Lake Ontario. Specifically, a screening program has been initiated to screen for *Heterosporis sp.*, a microsporidian parasite associated with percids (Bergstedt and Twohey 2005) that has not been detected in Lake Huron. *Heterosporis sp.* is known

from Lake Ontario, and sea lampreys there potentially feed on infected fish. Therefore, there is concern that the movement of sea lampreys from Lake Ontario to the St. Marys River could facilitate the spread of *Heterosporis sp.* to valuable fish species in the upper Great Lakes (Bergstedt and Twohey 2005). The sea lamprey's actual disease status and role in pathogen transmission are unknown. Sea lampreys from the Great Lakes have been found to host two other pathogens of concern that are already present in Lake Huron fish: *Aeromonas salmonicida* (causing furunculosis) and *Renibacterium salmoninarum*, (causing bacterial kidney disease) (Eissa et al. 2006). These pathogens are already present in Lake Huron fish, and are not seen as a reason to prevent sea lamprey transfers.

*Screening effort for Lake Ontario sea lamprey and the likelihood of pathogen translocation*

The Great Lakes Fishery Commission (GLFC) has adopted a policy of screening 60 fish prior to transfer (G. Christie, GLFC, personal communication).<sup>5</sup> In 2004, sea lampreys were collected from Lake Ontario tributaries for sterilization and transfer. The Aquatic Animal Health Laboratory at Michigan State University examined a subsample for the presence of emerging and restricted pathogens identified by the Office International des Epizooties (OIE) and the American Fisheries Society (AFS) using the AFS aquatic animal health survey procedures (G. Christie, GLFC, personal communication). All fish tested negative for *Heterosporis sp.*, resulting in the sterilization and transfer of 600 fish to the St. Marys River (Klar and Young 2004).

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<sup>5</sup> In 2004, 119 fish were actually screened (approximately 60 from two different rivers). Since 2004, however, only 60 fish total have been screened annually. We therefore use the 60 fish number in our analyses because it is current policy, and it is a common level of screening effort.

Neither the maximum acceptable probability of *Heterosporis sp.* translocation nor the expected true prevalence of *Heterosporis sp.* in Lake Ontario for *Heterosporis sp.* is documented for the sea lamprey transfer program. Therefore, we assume an initial value of 5% prevalence of *Heterosporis sp.* in Lake Ontario sea lampreys to illustrate the calculations described above. Assuming perfect specificity and sensitivity, screening 60 fish from the batch affords a probability of 0.95 of detecting  $\geq 1$  infected fish (Equation 3.3). If the prevalence of *Heterosporis sp.* in the batch of sea lampreys were exactly 5%, then testing 60 sea lampreys would result in a 5% chance of failing to detect the pathogen in the screened subsample *despite* being present in the batch (Equation 3.3). For a prevalence of 5% and a batch size of 600 fish, the probability of the presence of at least one infected fish in the batch is essentially 1.0 (see Table 3.1). We then use Equation (3.1) to calculate the probability of a pathogen translocation as  $(1-0.95)*1.00 = 0.05$ .

These calculations are sensitive to the assumption made about the assumed prevalence,  $m$ . Had the actual prevalence of *Heterosporis sp.* in Lake Ontario sea lampreys been 1%, then we could be 45.1% confident that at least one infected fish would be detected in a 60-sea lamprey subsample (Equation 3.3, Table 3.1). There would have been a 99.8% chance that at least one infected fish was present in the batch, implying that there would have been a  $(1-0.451)*0.998 = 54.8\%$  chance that at least one infected individual was moved from Lake Ontario to the St. Mary's River. These calculations illustrate the importance of explicitly determining the maximum acceptable likelihood that a pathogen is transferred and assumptions about the prevalence in the

source population. Managers need to be aware of all the factors affecting the risk of a translocation decision and carefully consider their implications.

The likelihood of pathogen translocation given a screening program is also sensitive to assumptions about diagnostic sensitivity and specificity (Figure 3.5), and this issue has received less attention (but see Dell’Omodarme and Prati 2005, and Munoz-Zanzi et al. 2006 for exceptions). The sensitivity and specificity of sea lamprey screening results were not reported to Great Lakes managers (G. Christie, GLFC, personal communication). Therefore, to illustrate the implications of sensitivity assumptions for the sea lamprey example, we assume, hypothetically, that the sensitivity of the test were 0.7 and specificity were 1, implying a probability of a false negative = 0.3. Thus, if the true prevalence were 5%, the observable prevalence would be 3.5% (Equation 3.4). This would increase the chance of failing to detect an infected individual in the subsample from 5% to 12.2%. The corresponding probability of pathogen translocation for a 600- fish batch would be 12.2%. Alternatively, it would be necessary to screen 86 fish, as opposed to 60, to provide a 5% chance of transferring at least one infected fish (see Figure 3.5’s dotted lines). Lower levels of assumed prevalence accentuate the effect of false negatives (in Figure 3.5, notice the increase in distance between the solid lines associated with 5% prevalence and the dashed lines associated with 1% prevalence).

Assumptions about specificity also affect decisions. Specificity < 1 can reduce the probability that pathogen will be transferred because such tests inflate the likelihood of a transfer-blocking positive result. This, too, needs to be accounted for when

designing screening programs, as a cost results when otherwise beneficial transfers are forgone.

*Determining the duration of a Lake Ontario sea lamprey pathogen screening program*

We now demonstrate how to determine how long a screening program should continue given successive failures to detect pathogen in screened samples and assuming that there is no independent evidence for new pathogen introductions. The GLFC pays a direct cost of about \$30 per sea lamprey screened (G. Christie, GLFC, personal communication), which does not include indirect costs such as transportation. The cost of transferring a pathogen has not been estimated, so for this example we compute the duration of the screening program over a range of costs. The probability of pathogen transfer has already been addressed.

Figure 3.6 illustrates how the likelihood of translocation, the cost of screening, and the cost of translocation of at least one infected fish interact to determine the number of years to continue screening, assuming no new pathogen introduction (see Equation 3.5), and leads to four observations. First, the economic impact of a disease event has little effect on the length of time the screening program should continue when zero infected individuals are detected, except in situations when such costs are minor. Second, if the expected damages from a pathogen translocation are small ( $< \$601$  if the true prevalence were 5% and the screening sample size were 60), then no screening should take place. Third, using Table 3.1 and Figure 3.6, we see that the cases where  $n = 60$  and  $m = 5\%$  (solid line) and  $n = 300$  and  $m = 1\%$  (dotted line) have the same probability of pathogen translocation. In the latter case more fish must be screened to

achieve this low probability, the screening program is more costly, and thus should terminate slightly sooner due to higher costs. Finally, in comparing the cases where  $n = 60$ , and  $m = 5\%$  (solid line) versus  $m = 1\%$  (dashed line), the lower source prevalence in the latter case increases the probability of transferring a pathogen (with a 600 sea lamprey transfer) to 54.8%. This implies that screening programs that result in lower confidence of pathogen detection and higher likelihood of pathogen translocation should persist longer.

These calculations are based on the assumption that the pathogen prevalence in the source population is not increasing, but is stable or declining. If there is evidence, independent of the screening program, suggesting that pathogen prevalence is increasing in the source population (e.g. due to the invasion of a new host or detection of the pathogen through other means), then screening efforts should persist. In such cases – where the prevalence is increasing or a pathogen has been introduced – it would be expected that infected individuals would begin to appear in the screening subsample.

### **3.5 Discussion and Conclusion**

Fish translocations create a likelihood of pathogen translocation, which may have adverse consequences for society. This risk can be managed, in part, through pre-translocation screening programs, and the effective and efficient design of these programs is important for two reasons. First, the design will affect the likelihood of pathogen detection and thus translocation decisions. Second, screening programs are not cost-free, and allocating resources to screening diverts them from other beneficial uses including other methods of managing fish pathogen transfer risk (e.g. disinfection

or treatment). This is not to say resources should not be used for pathogen screening, but that tradeoffs and the value of information gained must be considered.

Our analysis demonstrates that the translocation of an infected fish is most likely to occur at moderately low prevalence levels (see Figure 3.3). This is because the probability of failing to detect infected fish through screening declines as pathogen prevalence increases, while the probability that an infected individual would be in a batch for translocation declines as prevalence falls. The probability that the screening program prevents a translocation event is the intersection of these two probabilities. Fish screening is most likely to provide benefits when pathogen prevalence is relatively high, (i.e., > 5%), in which case, relatively small subsamples may be adequate (see Figures 3.3). Increasing subsample size, however, may be beneficial because the probability of a pathogen translocation event decreases as subsample size becomes a substantial fraction of the translocation batch (Figure 3.4). This may, however, be infeasible when screening is lethal. As expected, decreases in diagnostic sensitivity increase the likelihood of transferring at least one infected fish, but sample size and assumed prevalence interact with sensitivity to determine the size of the effect (Figure 3.5). Information about the sensitivity and specificity is vital to decision makers for interpreting results and designing screening programs.

Our analysis also examined the length of time a screening program should continue given no new pathogen detection and no evidence of new pathogen invasions. The probability of transferring a pathogen, which is determined by assumptions about the observable pathogen prevalence ( $m$ ) and management decisions, followed by the cost of screening, have the greatest impacts on the length of the time a program



continues.<sup>6</sup> Pathogen translocation costs are also important to consider but have a lower impact on how long to continue the program, particularly when they are large relative to screening costs, which will typically be the case. We provided a formula for determining the minimum expected costs of a pathogen transfer that justifies screening. These costs are usually modest (<\$601 in the case study).

Aquatic disease and fish health activities must be evaluated within the context of fisheries or ecosystem management objectives (Stephen 2001). Historically, fish health policy has presumed that the consequences of any pathogen translocation are unacceptable (Stephen 2001). Simplistic goals such as estimating and “minimizing disease prevalence” are not appropriate. Such goals do not account for the opportunity cost of using resources to manage disease that could have been used elsewhere in the fisheries management program, and do they adequately address real management tradeoffs.

We have presented a case study where sea lamprey transfers create the potential for pathogen transfer with negative consequences, yet the overall consequences of these translocations may be positive for the fishery, as a consequence of the benefits that the sterile sea lampreys provide to the overall health of Lake Huron fisheries. *Heterosporis sp.* has not been detected in the St. Marys River or Lake Huron, and SMRT related sea lamprey suppression has improved Lake Huron fisheries (Lupi et al. 2003; Klar and Young 2004; G. Christie, GLFC, personal communication). This result prompts one of

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<sup>6</sup> Formally, the marginal impacts are the derivatives of  $y$  with respect to each variable in Equation (5). These are  $\partial y/\partial CS = 1/(CS \ln(\Pr(t)))$ ,  $\partial y/\partial CT = -1/(CT \ln(\Pr(t)))$ , and  $\partial y/\partial \Pr(t) = -1 + \ln(-CS/(CS \ln[\Pr(t)])) \ln[\Pr(t)]^{-2} \Pr(t)^{-1}$ . The marginal effect of a change in either cost is the same in absolute value, but dependent on the cost, with larger costs resulting in effects smaller in absolute value. Given screening should begin and under realistic conditions,  $CT > CS$  (this will always hold if  $\Pr(t) \geq e^{-1}$  and will hold for a range of  $\Pr(t) < e^{-1}$ ). Also,  $|\partial y/\partial \Pr(t)| > |\partial y/\partial CT|$  for given values of  $\Pr(t)$ ,  $CT$ , and  $CS$ .

two possible interpretations. Either the screening program is adequate, or managers have been lucky. Good outcomes do not always imply good past decisions; thus a full review of the screening program for sea lampreys in the context of overall fishery objectives is merited. We found no evidence that managers had documented likely prevalence for *Heterosporis sp.* in Lake Ontario or specified a maximum acceptable likelihood of pathogen transfer - we encourage managers to address this issue explicitly as a part of screening program design. In general, fish health diagnostic professionals should provide, and decision makers should request, information on the performance of the diagnostic tests, specifically diagnostic sensitivity and specificity so that decision makers have a clear context for interpreting screening program results and modifying screening programs.

The concerns raised in the case study are not unique. Managers and researchers seek to understand the issues associated with control of aquatic pathogen spread. For example, Great Lakes fishery managers are grappling currently with disease issues associated with the potential reintroduction of extirpated sculpins (*Cottus ricei* and *Myoxocephalus thompsoni*) and ciscoes (*Coregonus spp.*) (Eshenroder and Krueger 2002). Recent, positive screening results for *R. salmoninarum*, however, may prevent these reintroductions (G. Wright, Chippewa-Ottawa Resource Authority, personal communication). In this case, the likelihood of a pathogen translocation and potential damages must be considered in terms of broader fishery objectives – i.e., what are the translocations likely to achieve for the overall community or ecosystem? Thrush and Peeler's (2006) 'contingency planning' model for the spread of the monogenean parasite *Gyrodactylus salaricus* in Britain may best illustrate the potential for screening

programs to prevent pathogen translocation damages. Although they do not explicitly include screening, they identify the ability to detect and prevent the movement of infected fish as the main component in preventing a “major outbreak.” Including simulated screening programs, based on the principles presented here, in such models could help identify efficient screening policies.

In this paper, we outline the screening design characteristics that affect the likelihood of a pathogen detection event and explain how these can affect pathogen transfer risk. Both over-allocation and under-allocation of resources to screening can have adverse consequences – striking a balance requires a sophisticated consideration of risk.

Table 3.1. The probability that a given pathogen would be present in a sample of a given size (or detected in a subsample with specificity = sensitivity = 1), for different prevalence levels among source fish.

		<b>Number of fish is in the sample</b>						
		<b>30</b>	<b>60</b>	<b>150</b>	<b>200</b>	<b>300</b>	<b>500</b>	<b>1000</b>
<b>Pathogen prevalence</b>	<b>0.001</b>	0.030	0.058	0.139	0.181	0.259	0.393	0.632
	<b>0.01</b>	0.259	0.451	0.777	0.865	0.950	0.993	1.000
	<b>0.02</b>	0.451	0.699	0.950	0.982	0.998	1.000	1.000
	<b>0.05</b>	0.777	0.950	0.999	1.000	1.000	1.000	1.000
	<b>0.1</b>	0.950	0.998	1.000	1.000	1.000	1.000	1.000

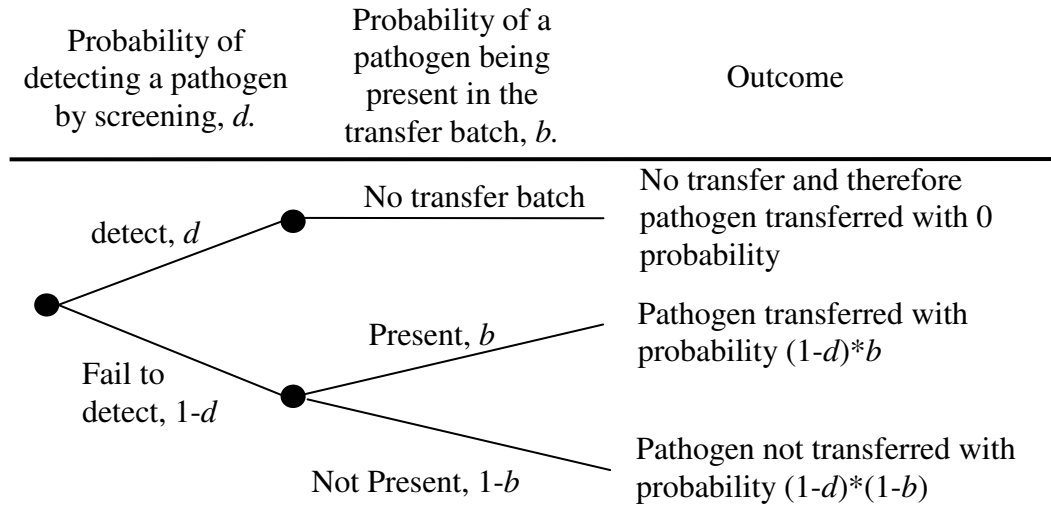


Figure 3.1. Decision tree giving the probability of transferring a pathogen given screening. If a pathogen is discovered upon screening, there is no translocation. Even if no pathogen is detected upon screening, it still may be present in the batch to be transferred. The probability of transferring a pathogen given screening is equal to the joint probabilities of  $\Pr[(1-d)]*\Pr[b]$ .

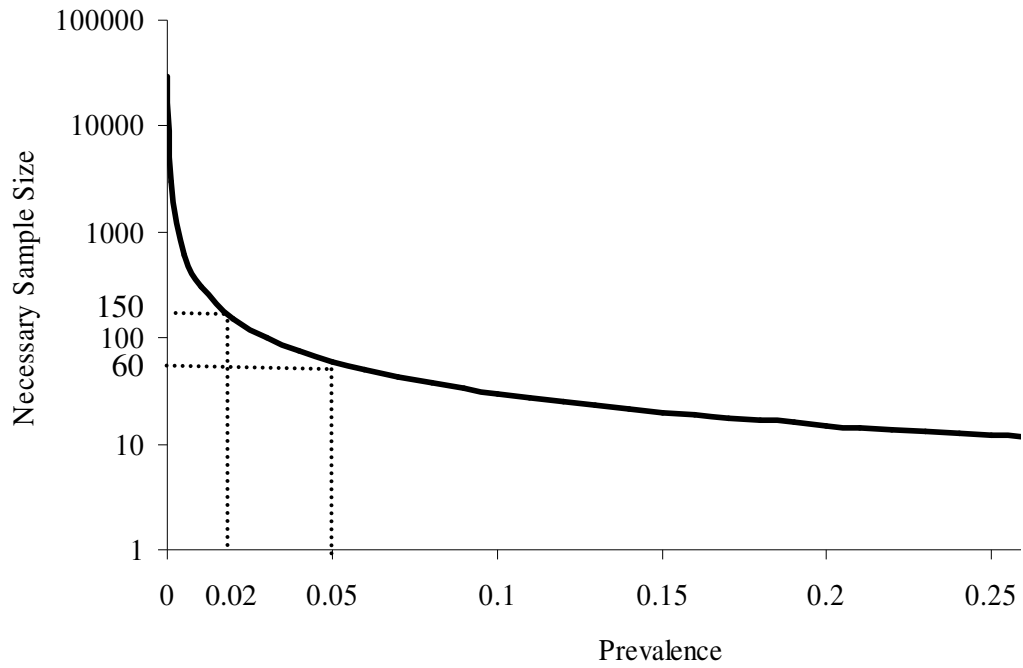


Figure 3.2. Number of fish that need to be tested to achieve 95% confidence that a pathogen is absent from the transfer batch at a given prevalence level (assuming sensitivity = specificity = 1). The numbers of fish required at 2% and 5% prevalence are identified with dotted lines. Note that the y-axis is on a log scale. Equation (3.2) was used to generate this graph.

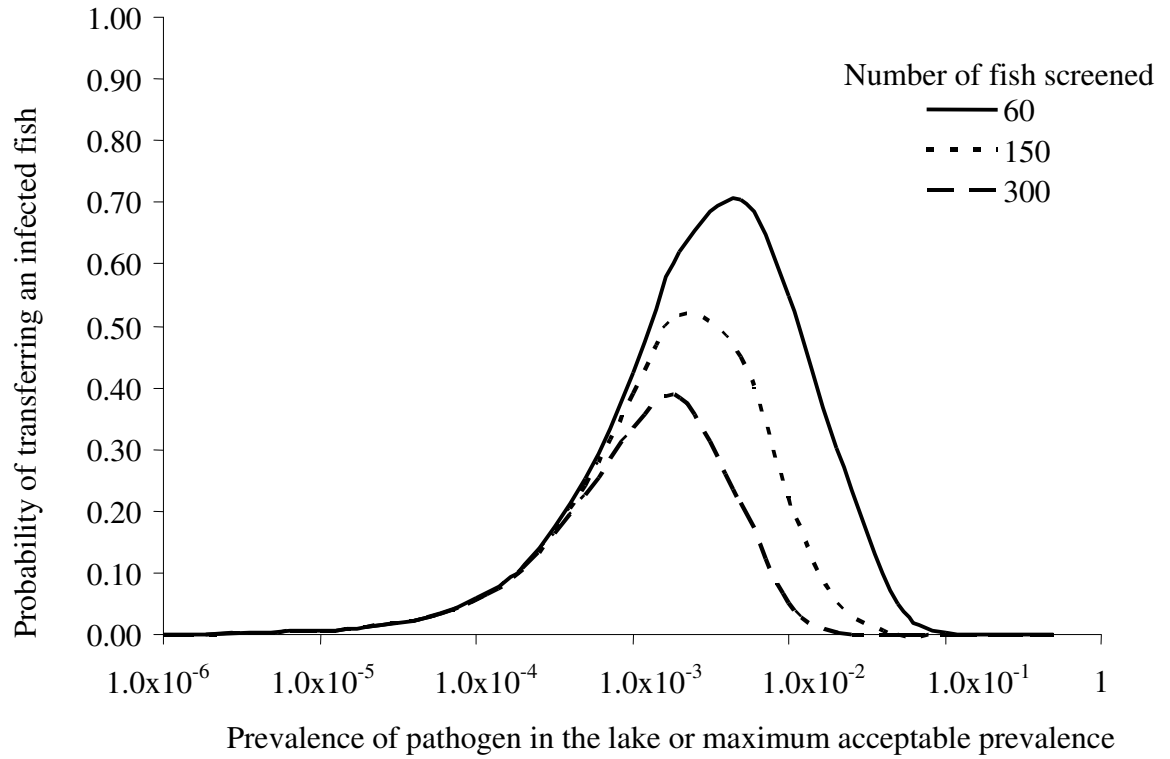


Figure 3.3. Probability that an infected fish would be transferred given different levels of source prevalence and screening effort, assuming translocation of 600 fish. Note that the x-axis is on a log scale. Equations (3.1) and (3.3) were used to generate this graph.

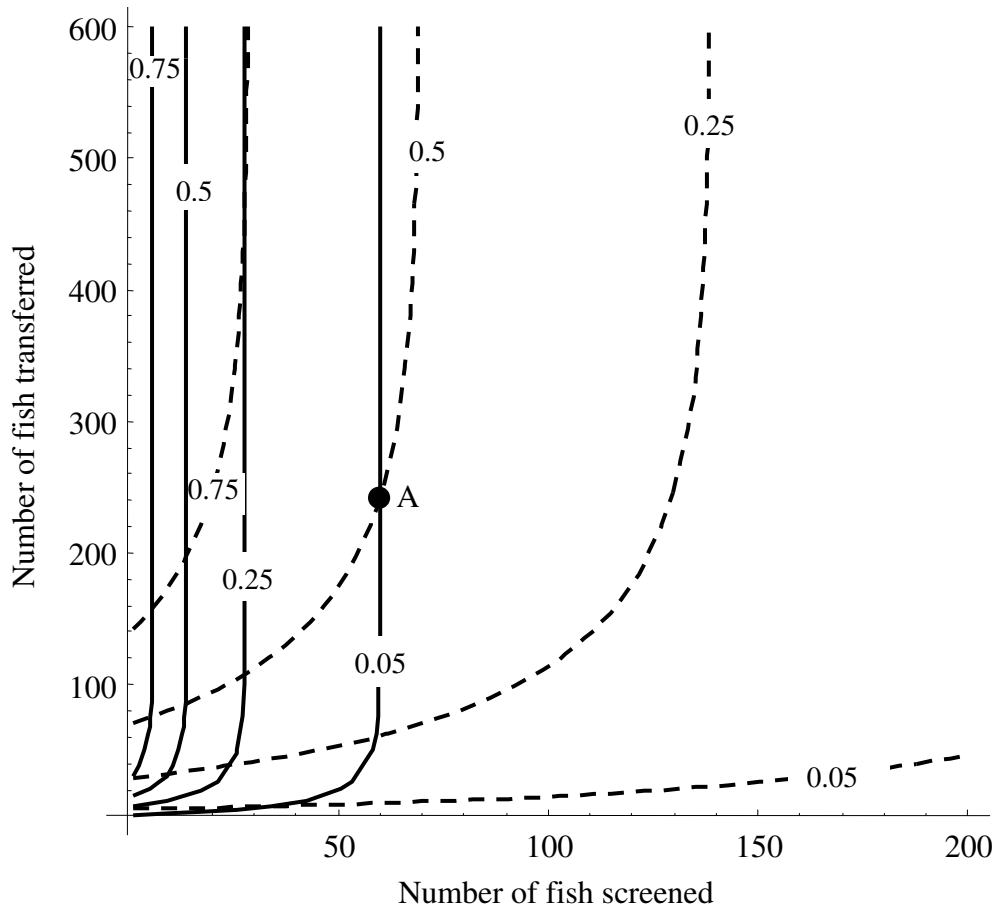


Figure 3.4. Probability (labeled on the contour lines) of translocating at least one infected fish, given the screening and transfer batch numbers. Solid lines represent the cases where the true prevalence in the transfer batch is 5%, and dashed lines represent the cases where the true prevalence in the batch is 1%. Point A indicates how the probability of transferring at least one infected fish increases by 10 times when the true prevalence is 1% rather than 5%. This graph was generated using Equations (3.1) and (3.3).



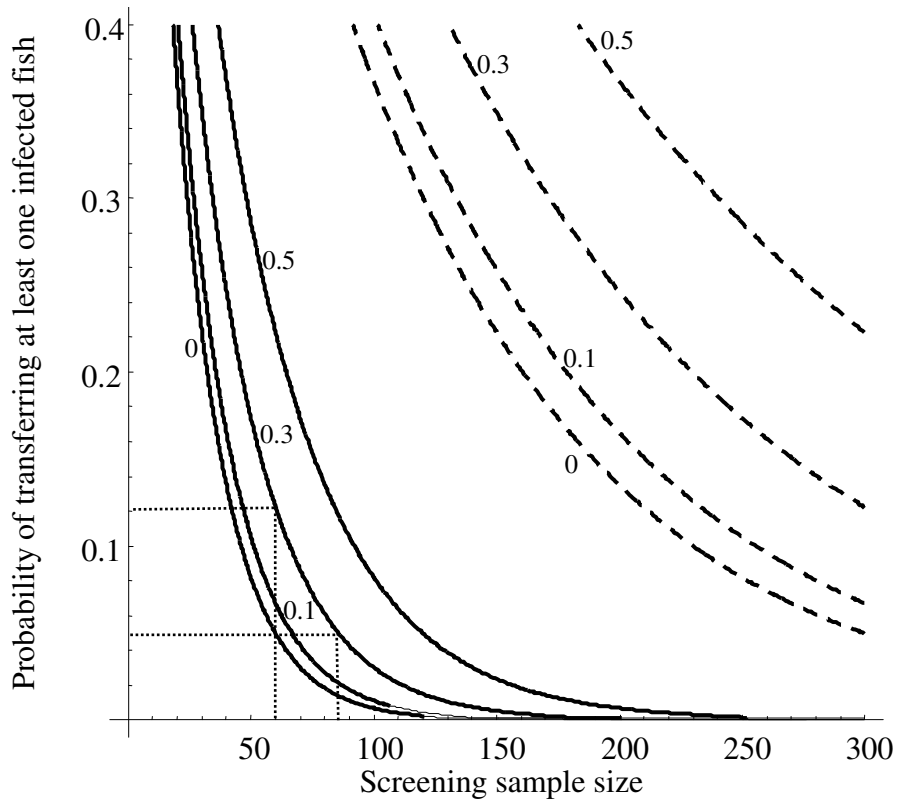


Figure 3.5. Probability of translocating at least one infected fish, given the screening sample size ( $x$ -axis) and the probability of a false negative screening result (labeled contour lines). Solid lines represent the cases where the true prevalence in the transfer batch is 5%, and dashed lines represent the cases where the true prevalence in the batch is 1%. The dotted lines illustrate the adjustment needed in sample size and the increase in the likelihood of a pathogen transfer when the probability of a false negative increases from 0 to 0.3 (representing a sensitivity of 0.7). Figure generated using equations (3.3) and (3.4).

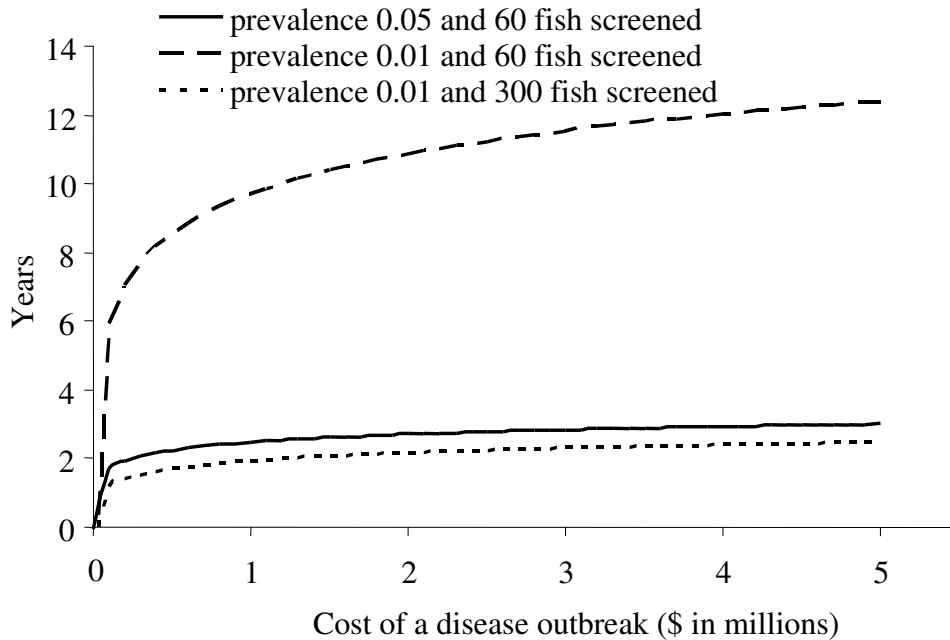


Figure 3.6. Effects of screening costs, the cost of a disease outbreak, and the probability of pathogen introduction on the number of years that a screening program should continue, given that no infected individuals are detected. The cost of the screening program is calculated as \$30 multiplied by the number of fish screened. The probability of transferring at least one infected fish, assuming 600 or more fish are translocated, was determined using Table 3.1. We assume that the translocation of a single infected fish is sufficient to cause damages. Equations (3.3) and (3.5) were used to generate this figure.

## CHAPTER 4

# A BIOECONOMIC MODEL OF CHINOOK SALMON STOCKING AND ALEWIFE CONTROL TO MAXIMIZE ECOSYSTEM BENEFITS THAT ACCOUNTS FOR INSTITUTIONAL CONSTRAINTS

### 4.1 Introduction

Great Lakes fishery managers work within a system that generally seeks to maximize the welfare to society through the management of fishery resources. Formally, the Great Lakes Fishery Commission (2001) states as one of its two major responsibilities, “...to recommend measures which will permit the maximum sustained productivity of stocks of fish of common concern.” We interpret this as to maximize net benefits associated with fish stocks of common concern, because some stocks of common concern may provide non-consumptive values, while others may actually create damages (negative benefits). Two non-native fish stocks of concern are Chinook salmon (*Oncorhynchus tshawytscha*) and alewife (*Alosa pseudoharengus*). Chinook salmon are stocked in the Great Lakes to create a sport fishery and control the invasive alewife population, thereby increasing social welfare. Alewife can be a nuisance species damaging infrastructure, natural amenities, and can negatively affect native fish populations.

Salmon and alewife management is a dominant issue on Lakes Ontario, Huron, and Michigan. Salmon provide fishing benefits and act as a biological control agent. Increasing salmon populations lead to decreases in alewife populations, and this appears to be a “win-win” situation. Alewives, however, comprise the majority of the

Chinook salmon diet (Madenjian *et al.* 2002), and Holey *et al.* (1998) state that the recreational Chinook fishery may depend on sustaining a large alewife forage base. Thus, there is an intertemporal tradeoff whereby efficient management must balance the benefits from the fishery and near-term alewife reductions against the long-term costs associated with the invasive alewives and maintaining a recreational salmon fishery.

Alewives invaded Lake Ontario as early as 1873, but were not detected in Lake Michigan until 1949 (O’Gorman and Stewart 1999). Alewives imposed costs on society by fouling beaches and drainpipes following their invasion (O’Gorman and Stewart 1999). Initially, alewife abundance and expansion was likely curtailed by native predator populations that subsequently succumbed to high fishing levels and mortality caused by the invasive sea lamprey (*Petromyzon marinus*) (O’Gorman and Stewart 1999). Alewives diminished the ability of the Great Lakes to provide ecosystem services. It is believed that alewife predation on lake trout (*Salvelinus namaycush*) fry impedes native lake trout restoration (Krueger *et al.* 1995; Madenjian *et al.* 2002). Moreover, alewife predation has been implicated in the decline of yellow perch (*Perca flavescens*) populations (Shroyer and McComish 2000), perhaps the most widely targeted sport fish in Lake Michigan (Wilberg *et al.* 2005).

Managers began stocking salmon into Lake Michigan in earnest in 1965 in part to control alewife populations (Madenjian *et al.* 2002). Chinook salmon are the main Pacific salmon stocked into Lake Michigan, and today create a valuable sport fishery (Hoehn *et al.* 1996). Alewives provide a benefit to the recreational fishery, because

alewives appear to be a required input, as prey, for Chinook salmon production (Stewart and Ibarra 1991).

Alewives provide both benefits and costs to society that managers concerned with social welfare must balance. Alewives provide benefits in the form of forage for the Chinook salmon. Alewives, however, generate costs (ecosystem disservices) to society through damage to infrastructure, natural amenities, and predation on the eggs and fry of valued native fish populations. The idea that species can generate both benefits and costs depending on the population's size has been explored generally (Rondeau 2001) and specifically in the cases feral pig management (Zivin *et al.* 2000) of urban deer (Rondeau and Conrad 2003) and elephant management (Horan and Bulte 2004). These authors show that this "multiple-use species" problem (Zivin *et al.* 2000) has the potential to result in non-convexities that lead to multiple bioeconomic equilibria. Multiple bioeconomic equilibria imply that the optimal outcome or basin of attraction for the system may depend on the initial conditions. However, in the previously studied cases, managers were assumed to directly control the population size. In the case of alewife management, managers indirectly influence the alewife population through the stocking of salmon.

It is often assumed that, all else equal, more salmon improves social welfare by providing more angling opportunities, but stocking more fish is not always better. This is because there are direct costs of stocking and indirect costs of depleting the prey base. Managers must achieve an intertemporal balance. Lake Michigan Chinook salmon stocking was recently decreased in part due to concern that the prey base had been reduced. However, the idea that the 'correct' stocking level for predator fish must

be connected to the prey fish stock level is not a new idea (Jones *et al.* 1993). Complex stochastic simulation models have been developed to inform stocking decisions in the Great Lakes (Jones *et al.* 1993; Rand 1994; Jackson 1997; Szalai 2003; Jones and Bence in review). These models include multiple species, bioenergetics, age and size-structure, and uncertainty. These efforts have focused on describing the distribution of outcomes that result from stocking strategies. However, such models have not explicitly optimized stocking with respect to an economically or socially defined objective, and they do not consider that angler behavior responds to changes in fish stocks. Morey and Waldman (1998) report that angler behavior is tied to catch rates, which likely correspond to fish stocks. Moreover, the complexity of these simulation models can make understanding and generalizing the results difficult.

Human welfare and the state of ecosystems are jointly determined by ecological and economic processes (Shogren *et al.* 1999; Sanchirico and Wilen 2001). We develop a simple model that captures the relationships between anglers, Chinook salmon, and alewives. We then solve for an optimal stocking program from the agency's perspective – one that maximizes social welfare, defined as the sum of discounted net benefits from the open access salmon sport fishery and averted damages from the alewife stock. In this case the agency is not a true social planner because the agency takes angler behavior as given. This can be thought of as an institutional constraint (Dasgupta and Maler 2003), but makes the nature of the solution “second best.”<sup>1</sup> This paper examines the tradeoffs associated with the stocking program in an analytical fashion, and develops general rules that can help guide stocking decision

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<sup>1</sup> A “first best” solution would require that managers control angler behavior, and therefore could optimally manage salmon and alewife harvests in addition to stocking.

making. The inclusion of human behavior in such rules is important if managers are concerned about human welfare or the effects that humans have on the ecological system (Settle *et al.* 2002). Finally, this paper contributes to the bioeconomic literature by linking the literature on non-convexities (Tahvonen and Salo 1996; Rondeau 2001; Dasguta and Maler 2003) with the literature on imperfectly targeted controls (Clark 1990; Fenichel and Horan 2007a, b; Horan *et al.* in press).

We begin by defining a bioeconomic objective. Then, we model decentralized economic angler behavior and biological processes. Next, we present optimality conditions for a general model, and show that the optimal stocking strategy is a nonlinear feedback rule that is a function of the state of both salmon and alewife populations. We then specify a full model and use phase-plane analysis to present simulation results and sensitivity analyses. We conclude with a discussion of the implications for salmon stocking policy in Lake Michigan and in general, and we discuss the linkage between imperfectly targeted controls and non-convexities.

#### **4.2 A bioeconomic model of salmon stocking**

A fishery management agency's charge can be interpreted as maximizing the discounted social net benefits associated with a fishery resource. Discounted social net benefits (*SNB*) are defined as the present value of the difference between how much salmon anglers gain from the fishery (total angler net benefits, *TAB*) minus how much society invests in the fishery (stocking in kilograms of salmon,  $w$ ) and any damages caused by the fishery.

$$(4.1) \quad SNB = \int_0^{\infty} (TAB(t) - D(a(t)) - vw(t))e^{-\rho t} dt$$

where  $\rho$  is the discount rate,  $v$  is the constant marginal cost of stocking a kilogram of salmon,  $D$  is a damage function, and  $a$  is the stock of alewives.

Alewife damages include ecosystem disservices (predation on the eggs and fry of native fishes) as well as the fouling of beaches, water intake pipes, and other amenities. Assume that alewife damages,  $D(a)$ , increase with increases in the alewife biomass,  $D'(a) > 0$ , and do so at an increasing rate,  $D''(a) > 0$ .<sup>2</sup> This kind of nonlinearity with biomass or abundance is likely a common trait for species that are considered invasive pests.

### ***A model of angler behavior***

Total angler benefits,  $TAB$ , are the sum of the net benefits received by all individual anglers at time  $t$ . Assume that all anglers have unique costs to fishing  $c$ , but all anglers have the same angling preference or “inverse demand” function,  $\Omega(m, z(s))$ , where  $z(s)$  is the quality of the fishery, experienced by all anglers, represented by catch per day, and catch per day increases with increases in salmon stock,  $s$ , and  $m$  is the number of days an individual angler fishes per season.<sup>3</sup> Following Anderson (1983) the inverse demand function is assumed to have the following characteristics. The inverse demand function is downward sloping with increases in days,  $\Omega_m(m, z(s)) < 0$  ( $\Omega_m$

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<sup>2</sup> This seems reasonable at the relevant biomass levels for alewife. There will be a level of alewife at which alewife cease to cause marginal damages, but this level is likely higher than the stock sizes considered.

<sup>3</sup> The current mode captures the general dynamics of angler behavior and we believe that the qualitative results of the model are robust to the assumption that anglers only differ in costs. In reality all anglers also have unique benefits from fishing and skill levels resulting in different catch rates. However, the incorporation of these differences is left for future analysis.



represents the partial derivative of  $\Omega$  with respect to  $m$ ; this notation will be maintained throughout). Increases in quality shift the inverse demand function upwards,  $\Omega_{z(s)}(m, z(s)) > 0$ . An individual angler's net benefits, a money metric of utility, from fishing  $m$  days is written as

$$(4.2) \quad U(m, z(s), c) = \int_0^m \Omega(\xi, z(s)) d\xi - cm$$

where  $\xi$  is the dummy of integration.

In a recreational fishery, the individual angler has two choices *i*) whether or not to fish in a given season, and *ii*) how many days to fish given that he chooses to participate (McConnell and Sutinen 1979; Anderson 1983). An angler enters the fishery if his net benefits,  $U \geq 0$  and will continue to increase the number of days that he fishes as long as his marginal utility from an additional day of fishing is positive,  $U_m \geq 0$ . Given that an angler participates, his individual net benefits is maximized when

$$(4.3) \quad U_m = \Omega(m, z(s)) - c = 0$$

implying that he will fish  $m^* = m[z(s), c]$  days. Thus, we assume that anglers behave to maximize their utilities so that  $U = U(m^*, z(s), c)$ .

To determine the level of effort in the fishery, we recognize that each angler has a unique cost to fishing and think of  $c$  as a cost type. Each cost type is treated as a "micro-unit" (Hochman and Zilberman 1978). Cost types are ordered in increasing order, such that the last cost type to enter the fishery is  $\bar{c}$ . When the distribution of cost types is continuous this is equivalent to the amount the last angler to enter pays for angling. This creates a cost threshold for entry so that only anglers with costs less than

$\bar{c}$  will enter. To solve for  $\bar{c}$ , we identify the cost at which the last angler is indifferent about entry and receives  $U = 0$ . The condition

$$(4.4) \quad U(m^*, z(s), \bar{c}) = \int_0^{m^*} \Omega(m, z(s)) dm - \bar{c}m^* = 0$$

can be solved for  $\bar{c}$ .

Cost types,  $c$ , are distributed over the interval  $[0, \infty]$  with the probability density function  $\psi$ . If  $N$  is the total number of *potential* anglers, then the actual number of anglers in the fishery,  $n(s)$ , depends on salmon biomass.

$$(4.5) \quad n(s) = N \int_0^{\bar{c}(m^*, s)} \psi dc$$

Total angler benefits,  $TAB(s)$  and total catch,  $h(s)$ , in biomass are also functions of salmon biomass and are derived similarly

$$(4.6) \quad TAB(s) = N \int_0^{\bar{c}(m^*, s)} [U(m, z(s), c) \psi] dc$$

$$(4.7) \quad h(s) = N \int_0^{\bar{c}(m^*, s)} (m^* z(s) \psi) dc$$

### ***Fish population models***

The fishery manager must take into account the dynamics of the fish stocks. Indeed, an ecosystem management approach must be concerned with the dynamics of multiple species and the interactions among those species. Let the dynamics of the harvestable Chinook salmon stock be defined in terms of biomass as

$$(4.8) \quad \dot{s} = \theta(a)(w + b) - s\delta(s, a) - h(s)$$

where  $b$  is the total biological recruitment and is independent of the stock size, i.e.,  $b$  represents fish produced in nature rather than by stocking. This is motivated by an assumption that limited spawning habitat will be saturated (implying strong density-dependent mechanisms). In baseline numerical simulations  $b = 0$ , presuming a salmon population entirely dependent on stocking;  $\theta(a)$  is a scaling function that scales biomass at stocking or biological recruitment to harvestable biomass or recruitment to the fishery. The scaling function is included because small non-catchable fish are stocked, and these fish must survive and grow before anglers have an opportunity to catch them. At higher alewife levels more young salmon survive and the average fish is larger. The mortality rate of fish in the fishery,  $\delta(s, a)$ , is a function of salmon and alewife biomass.

General results only require the assumptions that salmon mortality is a decreasing function of alewife biomass,  $\partial\delta(s, a)/\partial a < 0$ , and that as alewife biomass increases, ultimately salmon reach a minimum mortality rate,  $\partial^2\delta(s, a)/(\partial a)^2 \geq 0$ . We also assume that  $\partial\theta(a)/\partial a > 0$  and  $\partial^2\theta(a)/(\partial a)^2 < 0$ . Specific functional forms are specified in the simulation section.

The alewife population is defined in terms of biomass and follows logistic growth.

$$(4.9) \quad \dot{a} = ar\left(1 - \frac{a}{K}\right) - P(s, a)$$

Where  $r$  is the net recruitment rate of biomass in the limit as stock size approaches zero (recruitment minus non-predation mortality) and  $K$  is the alewife carrying capacity. The function  $P(s, a)$  is the Chinook salmon predation rate on alewife. General results

may be reached with a few assumptions. As salmon biomass increases, salmon consume more alewife,  $\partial P(s,a)/\partial s > 0$ . A unit increase in the salmon biomass may lead to a constant rate of increase in the amount of biomass consumed or intra-specific competition may lead to a decline in the amount of alewife consumed per salmon as salmon biomass increases,  $\partial^2 P(s,a)/(\partial s)^2 \leq 0$ . For simplicity assume no intra-specific competition,  $\partial^2 P(s, a)/(\partial s)^2 = 0$ , so that  $P(s, a) = sP(a)$ , where  $P(a)$  is the biomass of alewife consumed by a biomass unit of salmon. Assume salmon consume more alewife as alewife biomass increases such that  $P'(a) > 0$ . However, the rate at which salmon consume more alewife biomass as alewife biomass increases may decline with increasing alewife biomass,  $P''(a) \leq 0$ . That is, the response of salmon to increases in alewife biomass may be saturating, but  $sP(a)$  should be related to  $\alpha(s, a)$ . This is made explicit in the simulation below.

### 4.3 Optimizing social welfare through stocking

The agency is a social decision maker in a “second best” world due to the ecological and institutional constraints. The agency can not manage all aspects of the system, as would be the case in a “first best” world, but only controls the stocking program. The stocking program is therefore not perfectly targeted and leads to joint production of ecosystem services and disservices. When managers alter the salmon stock through stocking, the change in the salmon stock affects the alewife stock and angler behavior. Angler behavior and the alewife stock, however, have feedback effects on the salmon stock. The agency’s problem can be defined as choosing  $w$  to maximize (4.1) subject to equations (4.8) and (4.9). This requires that the agency explicitly consider equations

(4.6) and (4.7). The agency's problem can be solved using the maximum principle (Clark 1990). Write the Hamiltonian as

$$(4.10) \quad H = TAB(s) - D(a) - vw + \lambda \dot{s} + \mu \dot{a}$$

In the Hamiltonian,  $\lambda$  and  $\mu$  are the co-state variables associated with the salmon and alewife stocks respectively. Co-state variables represent the marginal user cost (benefit) associated with the salmon (alewife) stock. This is the cost (benefit) incurred when the salmon (alewife) capital stock is reduced by one unit. An alternative way of thinking of the marginal user cost is as the shadow value, which is the benefit (cost) of increasing the salmon (alewife) stock by one unit.

The marginal impact of stocking salmon is given by

$$(4.11) \quad \frac{\partial H}{\partial w} = -v + \lambda \theta(a)$$

The right-hand-side (RHS) of expression (11) is the coefficient of stocking from the Hamiltonian. The Hamiltonian is linear in the control variable,  $w$ . If  $\partial H/\partial w > 0$ , then stocking always increases the value of the Hamiltonian and stocking should be set at the maximum limit (an exogenously imposed limit that may represent hatchery capacity). On the other hand, if  $\partial H/\partial w < 0$ , then stocking always decreases the value of the Hamiltonian and stocking should be constrained to zero. These are constrained solutions. Another possibility is the case when  $\partial H/\partial w = 0$ , and condition (4.11) vanishes. This is known as the singular solution denoted  $w^*$ . In this case  $\lambda$ , the shadow value for the salmon stock (the value of having an additional salmon *in situ*), is exactly equal to the marginal cost of stocking scaled for growth,  $\lambda = v/\theta(a)$ . Regardless of

whether the solution is constrained or singular, the solution to problem (1) is a feedback rule and a function of the stock of alewife.

$$(4.12) \quad w = \begin{cases} w = 0, & \text{if } \lambda\theta(a) - v < 0 \\ w = w^* & \text{if } \lambda\theta(a) - v = 0 \\ w = w^{\max} & \text{if } \lambda\theta(a) - v > 0 \end{cases}$$

Conrad and Clark (1987, p.76) state that linear control problems guarantee the optimality of constrained solutions. In the linear control problem they describe, a constrained solution is pursued to the equilibrium, and then the singular solution is pursued to maintain the system at equilibrium (in their harvesting model this is a constant escapement policy). This illustrates a case of rapid adjustment and then maintenance at equilibrium. This result relies on the fact the control variable was perfectly targeted at the state variable and had no other side effects. When the control variable does not perfectly target the state variable, as in the case of the agency's salmon stocking program, more complex feedback rules may emerge (Mesterton-Gibbons 1987; Horan and Wolf 2005). In this case, it is optimal to pursue the singular solution when the system is out of equilibrium. This happens due to the nature of joint production and imperfect control. When control perfectly targets the system, adjustment can be rapid as the control completely determines the state of the system, but when control is imperfect, has side-effects, and only partial determines the state of the system, then adjustment must be slower to "smooth out" the non-target effects. This makes nonlinear feedback rule optimal.

Regardless of the type of solution, an optimal program requires that two adjoint equations associated with the co-state variables be satisfied at each point in time

(Conrad and Clark 1987; Clark 1990), where terms with subscripts denote partial derivatives with respect to the subscript

$$(4.13) \quad \dot{\lambda} = \rho\lambda - \frac{\partial H}{\partial s} = \rho\lambda - TAB_s + \lambda[\delta(s, a) + s\delta_s(s, a) + h'(s)] + \mu P(a)$$

$$(4.14) \quad \begin{aligned} \dot{\mu} &= \rho\mu - \frac{\partial H}{\partial a} = \rho\mu + D'(a) - \lambda((b+w)\theta'(a) - s\delta_a(s, a)) \\ &- \mu r \left(1 - \frac{2a}{K}\right) + \mu s P'(a) \end{aligned}$$

These conditions prevent intertemporal arbitrage opportunities: if these conditions are not satisfied, then welfare gains can be made by reallocating stocking across time, in which case the solution would not be intertemporally optimal.

### ***Interpreting the optimality conditions***

Before proceeding to simulation results, we examine the details of the optimality conditions to highlight tradeoffs associated with the stocking program. The first order and arbitrage conditions may be manipulated into two “golden rule” equations that must hold at each point in time (Conrad and Clark 1987; Clark 1990):

$$(4.15) \quad \rho = \frac{\dot{\lambda}}{\lambda} + \frac{1}{\lambda} TAB_s - \frac{\mu}{\lambda} P(a) - [\delta(s, a) + s\delta_s(s, a)] - h'(s)$$

$$(4.16) \quad \rho = \frac{\dot{\mu}}{\mu} - \frac{D'(a)}{\mu} + \frac{\lambda}{\mu} ((b+w)\theta'(a) - s\delta_a(s, a)) + r \left(1 - \frac{2a}{K}\right) - sP'(a)$$

These golden rule equations highlight tradeoffs associated with increases or decreases in salmon and alewife biomass. In equation (4.15)  $\rho$ , the discount rate, is the manager’s rate of time preference and can be thought of as the opportunity cost of providing an

additional unit of biomass to the salmon stock. The RHS of equation (4.15) collectively represents the marginal benefit of stocking an additional unit of salmon biomass. The first term on the RHS is a capital gains term that will be zero at equilibrium. The second RHS term is the total angler benefits gained from a larger salmon stock and is always positive. This term indicates that all else equal anglers are better off with a larger salmon stock because a larger salmon stock increases fishing quality, and thus enables more fishing days and more angler participation. The third RHS term is the marginal impact of an increase in the Chinook population on the alewife resource on which the Chinook population depends. This term is negative. Increasing the Chinook population decreases the alewife population and is costly to Chinook production. The fourth and fifth RHS terms together are the effect of the Chinook stock on its own marginal growth. The fourth term, the term in the square brackets, is the direct effect of the salmon stock on its own growth rate. This term is always negative because recruitment is independent of the stock so increases in stock only increase mortality. This term results because an increase in the salmon population increases the salmon mortality rate, by reducing the prey base. This can be thought of as the cost of natural mortality. The final RHS term represents the change in salmon mortality with an increase in the salmon stock due to changes in angler behavior. This term is negative; an increase in the salmon stock increases the total effort and thus increases the catch in the fishery reducing future fishing opportunities *ceteris paribus*. This can be thought of as the cost of fishing mortality.

Equation (4.16) is also a golden rule equation, but has a different interpretation than equation (4.15). The left-hand-side of the equation is also the discount rate, i.e.,



the manager's rate of time preference, but is interpreted as the marginal cost of diverting resources from elsewhere in the economy to manage the invasive alewife and their associated damages. Alternatively,  $\rho$  is as the rate of return earned by forgoing stocking in order to preserve the prey base, this is appropriate in a case where alewife-induced damages are small. The RHS of equation (4.16) can be thought of collectively as the net gains from alewife management (or the net losses from depleting the prey base). From the perspective of invasive species management, the first RHS term is a capital loss term, but from the perspective of managing the prey base it is a capital gains term, either way it will be zero at equilibrium. The second RHS term represents the marginal social benefits associated with a smaller invasive alewife population (for a native non-pest species this term is zero). The third RHS term is the cost of reducing the prey population to the recreational chinook fishery in terms of lost Chinook salmon production. This is a cost because it reduces the rents to the recreational fishery. The fourth RHS term is the effect of the alewife stock on its own marginal growth as a result of it being a reproducible liability or asset; this may be thought of the base rate of return to controlling alewife. This term can be positive or negative. The final RHS term in equation (4.16) is the marginal benefit (cost) of decreases in the alewife (prey) population on the ability of Chinook salmon to control (deplete) alewife numbers *ceteris paribus*. That is, because for  $P'(a) > 0$ , decreases in the alewife population make chinook salmon more efficient at controlling alewife numbers and provide a benefit (this is cost if the prey does not cause damages), *ceteris paribus*. From a biological perspective, when there are fewer alewife, salmon are not saturated, and a hungry salmon will consume more prey.

### ***Solving for the nonlinear feedback rule***

The approach for finding the nonlinear feedback rule for the singular solution is similar to Fenichel and Horan's (2007a) procedure for finding a partial singular solution (their model has two control variables). First, set equation (4.11) equal to zero and solve for  $\lambda = \lambda(a)$ . Next, take the derivative of  $\lambda$  with respect to time yielding  $\lambda'(t) = \lambda_t(s, a)$  and substitute this into condition (4.13). Solve the resulting equation for  $\mu = \mu(s, a)$  and take the time derivative  $\mu'(t) = \mu_t(s, a, w)$ . Notice that the time derivative of  $\mu$  is a function of  $w$ . Finally, set the time derivative of  $\mu$  equal to condition (4.14) and solve for  $w$ . The solution is a nonlinear feedback rule where  $w = w^*(s, a)$ . A specific feedback rule requires further specification of the general functions used so far (see function specification for simulation section).

This feedback rule contrasts with current stocking policies and with feedback policies under consideration. Management agencies often attempt to identify constant stocking policies that do not respond to the state of the system. Constant stocking policies would be optimal in the case where condition (4.11)  $> 0$ , and thus the maximum stocking level were pursued (or in the case where no stocking takes place because condition (4.11)  $< 0$ ). A constant stocking policy could also be optimal if the system were at the socially optimal equilibrium; however *i*) this is unlikely at present, and *ii*) even if the system were to arrive at the socially optimal equilibrium it may be perturbed away from this point. Moreover, the optimal constant stocking rate in this case would still be a function of the salmon and alewife population. These results support the current efforts underway to develop state dependent stocking rule that are based on more complex and biologically realistic models (Szalia 2003, Jones and Bence

in review). However, to date those efforts have not accounted for the response of anglers to changing stock sizes, and have only consider feedback policies based on alewife *or* salmon stocks.

#### **4.4. Function specification for simulation**

We specify explicit functional forms for the remaining implicit functions in the model in order to examine the optimal solution numerically. A numerical approach is required because the angler behavior and the nonlinear feedback rule for stocking are too complex to analyze analytically. Moreover, when to employ the nonlinear feedback rule and when to pursue a constrained solution inherently requires numerical solutions (Arrow 1968). There are two features of this problem that can lead to non-convexities and multiple equilibria *i*) the multiple-use species problem (Rondeau 2001; Horan and Bulte 2004) and *ii*) the potential for complex human-ecological relationships (Crepin 2003). For problems with non-convexities, “what is required is the sheer brute force of computing welfare along candidate programs and comparing them” (Dasgupta and Maler 2003). The following functional forms were chosen to be as simple as possible while capturing desired aspects of the relationships that are consistent with theory and economic and biological knowledge.

##### ***Economic functions***

The exact nature of the alewife damage function is unknown. However, given the assumptions that  $D'(a) > 0$  and  $D''(a) > 0$ , we use a second order approximation to a

convex function  $D(a) = Da^2$ , where  $D$  is a damage parameter. The angler “inverse demand” function takes the form

$$(4.17) \quad \Omega(m, z(s)) = x_1 - x_2m + x_3qs$$

where  $q$  is the catchability coefficient. The distribution of cost types is assumed to be log-normally distributed (Just and Antle (1990) recommend this distribution for micro-parameter models as a default) with a mean of  $\eta$  and standard deviation  $\sigma$ . Given this specification we derive specific relationships corresponding to the general ones established above.

$$(4.18) \quad m^* = \frac{x_1 + x_3qs - c}{x_2}$$

$$(4.19) \quad \bar{c} = x_1 + x_3qs$$

### *Ecological functions*

We define the function  $\delta(s, a)$  as

$$(4.20) \quad \delta(s, a) = \alpha - \gamma(sP(a)/s).$$

In equation (4.20) salmon mortality declines linearly with increases in the biomass of alewife consumed per biomass unit of salmon. That is,  $\alpha$  is the instantaneous annual mortality rate with zero alewife,  $\alpha - \gamma$  is the instantaneous annual mortality rate at satiation. Assume that salmon have a type-II predator response function with parameters  $\beta$  and  $\omega$ .

$$(4.21) \quad sP(a) = s \frac{\omega a}{1 + \beta a}$$

Equations (4.20) and (4.21) satisfy the condition above in the general model and are coupled, so there is a direct connection between salmon survival and prey consumption.

The alewife population also affects the stocking survival and recruitment to the fishery. We model this with a scaling function,  $\theta(a) = ya^\phi$ , where  $0 < \phi < 1$  and  $y$  are parameters.

Mathematica 6.0 (Wolfram Research) was used to implement the model. The parameter values used are listed in the appendix. We have used the best available data on the salmon-alewife-angler system. Some processes, however have been condensed to simple functional forms to improve tractability. For example, many biological relations are often assumed to depend on size of individuals, and given that fish change many magnitudes order in size, this required that some parameters be rescaled to apply to the average of aggregated individuals represented in our model by the biomass of alewife or salmon. The quantitative rescaling required judgment. Consequently, our specific numerical results should be used as a guide to help navigate the often perplexing outcomes of more complex models. Nonetheless, the model allows us to describe tradeoffs associated with salmon stocking programs and identify important interactions between non-convexities and imperfectly targeted controls.

## **4.5 Optimal management**

### ***The case of no alewife damages***

We begin with the special case when alewives do not cause damages. This case is interesting in its own right because in many systems the prey fish is native and does not have adverse effects. It also serves as a baseline to illustrate the importance of

considering the ecosystem damages (disservices) caused by invasive alewife when developing a stocking strategy. Given an initial state of the world,  $s_0$  and  $a_0$ , the agency planner must choose a stocking program. This program may include constrained or singular values of  $w$  (i.e.,  $w = 0$ ,  $w = w^{\max}$ , or  $w = w^*(s, a)$ ) at different points in time. The choice of when to apply which type of solution is a common problem when multiple populations are managed with imperfectly targeted management measures (Mesterton-Gibbons 1987; Fenichel and Horan 2007a; Horan *et al.* in press). Phase plane analysis is a useful tool for answering this question.

*Dynamics when  $w = w^{\max}$*

Consider the dynamics if stocking were to always occur, i.e.,  $w = w^{\max}$  (Figure 4.1). In this case the biomass of salmon would build up, and the high biomass of salmon would reduce the biomass of alewife. If the system were to start at  $(0, K)$ , then a constant  $w^{\max}$  policy leads to maximum number of salmon that can be in the system (this is the salmon limit). Indeed from any initial condition, pursuit of a maximum stocking strategy will lead to alewife eradication.<sup>4</sup> Recall, that alewife are necessary for the survival of salmon and that the salmon that are stocked are not yet available to the fishery. With no alewife in the system any salmon that are stock are expected to die prior to entering the fishery.

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<sup>4</sup> Eradication results from the assumption of a type-II predator response function.

### *Dynamics when $w = 0$*

Now consider the dynamics if stocking were forgone all together (Figure 4.2). If the system were to start at  $(0, K)$ , then with no stocking, the dynamics are trivial and the system stays at  $(0, K)$ . Given the assumption the salmon do not reproduce natural  $w = 0$  is only possible after some stocking has occurred. The system is divided into two basins of attraction by a separatrix that leads to the origin (Figure 4.2). For starting points above the separatrix the system will ultimately arrive at  $(0, K)$  if stocking is always forgone. If, however, stocking is ended at a point below the separatrix, then the alewife population is eradicated. As with the  $w^{\max}$  case, this means that the salmon fishery can not persist.

### *Dynamics when $w = w^*(s, a)$*

Finally, consider the non-linear feedback rule  $w = w^*(s, a)$  (Figure 4.3). First, we identify the state space where the non-linear feedback rule from the singular solution is feasible. To do this, we identify extreme solutions where the singular solution implies a boundary, i.e.,  $w^*(s, a) = 0$  and  $w^*(s, a) = w^{\max}$  (see Fenichel and Horan 2007a and Horan *et al.* in press for a similar approach). Boundaries are plotted in all figures as dashed lines. Hence, the phase space can be divided into four regions *i*) above the  $w = w^{\max}$  boundary where stocking at the maximum rate is implied, *ii*) an interior region where the non-linear feedback rule is used to determine the amount of stocking, *iii*) the area below the  $w = 0$  boundary in Figure 4.3A (except for a small region close to the origin) where it no stocking is implied, and *iv*) a second interior region between the

$w = 0$  and the  $y$ -axis (Figure 4.3B). The singular solution,  $w = w^*(s, a)$ , can only be pursued in the interior regions between  $w = w^{\max}$  and  $w = 0$  boundaries.

The dynamics when  $w = w^*(s, a)$  are defined by the intersections of the  $\dot{s} = 0$  and  $\dot{a} = 0$  isoclines. These intersect in five places creating five equilibria. There are two trivial equilibria at  $(0, 0)$  and  $(0, K)$ , and three interior equilibria; one is at point A (Figure 4.3A) and two are in the lower left corner near the origin where a second  $\dot{s} = 0$  isocline exists but loops back to the  $y$ -axis. These are labeled points B and C (Figure 4.3B). Multiple interior equilibria are typically associated with non-convexities and come in odd numbers (Tahvonen and Salo 1996). The existence of an odd number of equilibria is verified by taking the limits  $\partial\dot{a}/\partial s = 0$  and  $\partial\dot{s}/\partial s = \infty$  at the origin. Moving from the origin along the  $\dot{s} = 0$  isocline, the  $\dot{s} = 0$  isocline must cross the  $\dot{a} = 0$  isocline an odd number of times (Figure 4.3A and 4.3B). To check the local stability properties of the equilibria we determine the eigenvalues for the linearized system. The eigenvalues associated with equilibria A and C are imaginary with real parts indicating that equilibria A and C are unstable foci (Conrad and Clark 1987). That implies that application of the singular solution leads to a path that spirals away from these equilibria. Therefore, the system can not be maintained in the upper interior region. These equilibria only represent optimal steady states in the case where the initial conditions for the state variables are equal to the state variable values at the equilibrium. One eigenvalue associated with equilibrium B is positive and the other negative, indicating a conditionally stable equilibrium or saddle point (Conrad and Clark 1987). A unique path known as a saddle path or separatrix may be followed to



arrive at this equilibrium point; however, deviations from the saddle path will lead away from the equilibrium (Figure 4.3A and 4.3B).

The saddle path bifurcates the system, and divides alternate basins (Dasgupta and Maler 2003). This saddle path is different from the separatrix in Figure 4.2, but divides the system in a qualitatively similar fashion. Locating the saddle path is useful even though, following the saddle path to the saddle point may not be optimal in the case with multiple equilibria. The saddle path is piece-wise continuous as it approaches equilibrium B from a constrained region. Moving backwards in time from the equilibrium B along the saddle path, initially a singular solution would be pursued until the point where the  $w = 0$  boundary is intersected (Figure 4.3B). Upon crossing the  $w = 0$  boundary the  $w = 0$  solution is pursued. That is, the piece-wise saddle, accounts for the constraint of stocking zero salmon.

### *Optimal stocking strategies*

There are two basins of attraction, above and below the saddle path. Whether the system initially lies above or below the saddle path influences the optimal stocking strategy. Therefore, optimal management from starting points below and above the saddle path must be considered separately. This is the general nature of problems with non-convexities.

First consider the solution for points below the saddle path. Recall that saddle path is piece-wise continuous accounting for the  $w = 0$  constraint. Also, recall that managers do not control harvests, and therefore setting stocking to zero does not lead to a “jump” to the saddle path. This is because the only way for salmon to be removed

from the system is through harvests, which are not controlled and through natural mortality. Therefore, the salmon population responds slowly to management. Below the saddle path, managers optimally stock zero salmon, but this does not reduce the salmon stock fast enough, and the remaining salmon consume all of the alewife.<sup>5</sup>

Now consider starting points above the saddle path. Within this region, there are potentially two ultimate outcomes: (i) movement to the saddle path, which moves the system to equilibrium B, or (ii) cycling around equilibrium A, provided a stable limit cycle exists. These two solutions can not be simultaneously optimal, implying that if a stable limit cycle is encountered the optimality of the saddle point can be ruled out (see Liski *et al.* 2001 for details). Consider a general starting the point,  $(0, K)$ . We consider this to be a general starting point because all other starting points above the separatrix are closer to either potential outcome. From  $(0, K)$ , an interior path that eventually goes to the  $w^{\max}$  boundary is optimal.<sup>6</sup> At this point a  $w = w^{\max}$  is pursued until the dynamics, governed by the phase arrows, push the system back into the interior, prior to hitting the saddle path. In the interior region, the singular solution is pursued until the  $w = 0$  boundary is intersected. Pursuit of the  $w = 0$  rule leads back to the interior region, where the singular solution is again applied. This leads back the  $w = w^{\max}$  boundary, creating a cycle. This cycle can be numerically compared to a path in which  $w^{\max}$  is pursued to separatrix and then followed to equilibrium B. The discounted social net benefits for the stable cycle are greater than those for a program

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<sup>5</sup> Total consumption of alewife is a result of the type II predator response function.

<sup>6</sup> It is possible there to apply the  $w = w^{\max}$  early and move rapidly to the right on the phase plan, but this makes little difference given the high level of stocking implied by the  $w = w^*(s, a)$  rule.

that moves first the separatrix and then to equilibrium B for all initial conditions above the separatrix.

The logic behind the optimality of the stable limit cycle can be described two ways. First, the “second best” nature of the problem requires slower adjustment. In a “first best” world, the planner would have control over salmon stocking and harvesting and alewife harvesting. Hence, control would be perfectly targeted and controls would only have direct effects leading to rapid “jumps” to a saddle path (e.g. Conrad and Clark 1987). Stocking is the only control option and has many indirect effects on the system. These indirect effects require that adjustments take place more slowly. This is akin to convex adjustment costs in investment models (Liski *et al.* 2001).

The second explanation is that the optimal solution is a cycle due to the production effect of alewife. Alewives are important for producing salmon angling benefits. Salmon stocking begins to increase alewife biomass has begun to increase. At this point stocking salmon and increasing alewife biomass are compliments in production. Increasing alewife biomass allows for higher levels of stocking. Over time, salmon biomass builds up because managers do not control the rate which salmon are removed from the system. Managers recognize that increased further increases in stocking will lead to over exploitation of the prey. Managers substitute alewife biomass for stocking at high alewife biomass. This leads to a reduction in stocking. Maximum stocking is achieved prior to the maximum biomass of alewife for the same reason. The long-term planner knows that salmon will reduce the alewife biomass and that salmon will also attract more anglers.

### ***Optimal management with alewife-induced damages***

#### *Describing the phase plane*

We follow the same procedure for the case with alewife-induced damages (ecosystem disservices). We begin by drawing the completed phase plane (Figure 4.4). The individually constrained cases of  $w = 0$  and  $w = w^{\max}$  are the same as before (Figures 4.1 and 4.2). There is a noticeable change in the  $\dot{s} = 0$  isocline. Indeed, there is a single continuous  $\dot{s} = 0$  isocline originating at the origin. This isocline changes because the incentives for stocking salmon have changed. Specifically, managers stock salmon to control alewife related damages in addition to providing angler benefits. The  $\dot{a} = 0$  isocline, however, has not changed because there is no direct control over the alewife population. Furthermore, the additional consideration of alewife related damages causes the stocking boundaries to shift downward relative to the no damage case, and there is only one continuous  $w = 0$  boundary. This occurs because more salmon are stocked, at a given level of alewife, to reduce alewife biomass thereby reducing alewife-induced damages.

In this case there are also three interior equilibria, A, B, C (Figure 4.4A and Figure 4.4B). Linearizing the system at these equilibria and finding the eigenvalues also indicate that the equilibria are an unstable focus, conditionally stable saddle point, and unstable focus respectively. The additional consideration of alewife induced damages causes point A to shift down and to the left relative to the no damage case. This is an intuitive result, because alewife now create damages they are managed at lower population level and can support a smaller population of salmon

### *Optimal stocking strategies*

Damages do not eliminate the need to consider the effect of the initial state of the world on the optimal stocking strategy. There are two possibilities for initial conditions above the saddle path *i*) rapid movement to the piece-wise continuous saddle path and then pursuit of the piece-wise continuous saddle path towards equilibrium B or *ii*) cycling around equilibrium A if a stable cycle exists. Notice that that saddle path leaves the interior region (Figure 4.4B). After this point,  $w = 0$  along the saddle path. When damages are large enough a *stable* cycle does not exist around equilibrium A. Prior to completing even the tightest stable cycle around equilibrium A, applying  $w^*(s, a)$  leads to a path that intersects the piece-wise continuous saddle path. If a  $w^*(s, a)$  were pursued beyond the saddle path, then the system would enter the alternate basin of attraction and could not return. Hence, for all initial conditions above the saddle path, it is optimal to apply the maximum stocking level to move as rapidly as possible to the saddle path (the general dynamics for a  $w = w^{\max}$  are same as in Figure 4.1). Then, the piece-wise continuous saddle path is pursued, initially forgoing stocking until the  $w = 0$  boundary is crossed from below (Figure 4.4B). At that point, stocking commences according to the  $w = w^*(s, a)$ .

Managers face the same problem for all initial states of the world below the saddle path that was described for the no damage case. Specifically, stocking has already been optimally reduced to zero, but salmon mortality is not high enough to rapidly reduce the salmon population. There are no alternative controls to move the

system back to the saddle path. Alewives are eradicated and the salmon fishery can not be sustained.<sup>7</sup>

*Explaining the change in stocking strategy by accounting for ecosystem disservices*

A critical difference from the no damage case is that alewife are a “multiple-use” species (Zivin *et al.* 2000) when damages are included. Alewives jointly produce ecosystem services and disservices. Following Horan and Bulte (2004) we consider *ex situ* and *in situ* marginal net benefits associated with the alewife population at equilibrium. The *ex situ* marginal benefits are equivalent to the shadow value,  $\mu$ , while the *in situ* marginal benefits,  $\Phi$ , represents a suite of tradeoffs and are equal to the RHS of the golden rule equation (4.16) less the base rate of return (Horan and Bulte 2004).<sup>8</sup> Following Horan and Bulte (2004) if  $\mu > 0$ , then alewife are considered to be a commodity, but if  $\mu < 0$  then alewife are considered a nuisance. Alewife may be thought of as a commodity when their net impact on social welfare is beneficial, and this occurs if alewife generate more benefits as input in salmon production than they detract by fouling beaches and drain pipes or preying on eggs and fry. If, however, the net impact of alewife on social welfare is negative, then they are considered a nuisance. Furthermore if  $\Phi > 0$  then alewife are an asset and overall society is better off with more alewife. Conversely, if  $\Phi < 0$ , then alewife are a liability and society is better off limiting the alewife stock. In the case with no damages, we expect alewife to be both an asset and a commodity because they provide benefits in terms of increased salmon

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<sup>7</sup> Eradication is dependent on the type-II predator response.

<sup>8</sup> Recall that at equilibrium the term  $\dot{\mu}/\mu = 0$  and the base rate of return term is  $r(1 - 2a/K)$ .

production. We verified that this is indeed the case at equilibrium A in the no damage case.

In the current case, with damages,  $\mu > 0$  at equilibria A and B. For equilibrium A, however,  $\Phi < 0$ , creating a case where alewives are simultaneously a commodity and liability. Horan and Bulte (2004) point out that this case is unlikely to be stable and optimal because it can often be less costly to convert the liability into an asset. This is achieved by increasing stocking to reduce the alewife stock. At the saddle point equilibrium B,  $\Phi > 0$ . Equilibrium B shifts up and to right, expanding its domain of attraction.

#### **4.6 Sensitivity Analysis**

Sensitivity analyses are commonly used to examine how changes in a parameter affect model results. This model has a large number of parameters and sensitivity analysis could be performed for each. Doing so requires that new a phase plane be drawn and analyzed for each parameter evaluated. In the interest of space we focus on three parameters of interest, the discount rate,  $\rho$ , the amount of natural recruitment,  $b$ , and effect of alewife on salmon recruitment to the fishery,  $\phi$ . The discount rate was chosen in order to understand how time preferences affect optimal management. Chinook salmon naturally reproduce in the Great Lakes (Scott *et al.* 2003), and it therefore is necessary to understand how the natural recruitment rate affects results. Finally, sensitivity analysis was conducted with respect to  $\phi$  because there is little biological data on which to calibrate this parameter. We explore model sensitivity in cases where alewives do and do not cause damages.

The discount rate determines how the planner weighs net benefits in the near term versus the future. Lower discount rates imply that the planner places relatively more weight on the future. To examine changes in time preference, we increase the discount rate from 5% to 10%. Both with and without damages this has no qualitative effect on the results. In both cases equilibrium A shifts down and to the left and equilibrium B shift up and to the right. The shift in B is proportionally larger than the shift in A. This implies that fewer alewife-induced damages are needed to cause the qualitative shift in management strategies at a higher discount rate. This comes about because the future salmon angling is less highly valued and there are fewer incentives to preserve the prey.

There were no wild spawned salmon in the Great Lakes in the 1960s, when the Chinook salmon stocking program began. Moreover, it was suspected that Chinook salmon would not establish wild spawning populations. To test model sensitivity to this assumption, we increased the amount of wild recruitment from 0 kg to 10,000 kg to examine how wild recruitment may affect model outcomes. Wild recruitment has no qualitative effect on the solution. Indeed, the equilibria points are at the same location in  $s$ - $a$  space. The stocking boundaries do move; the  $w = w^{\max}$  and  $w = 0$  boundary shift downward. Without damages, only equilibrium A is achievable; equilibrium B would require negative stocking, but still creates a “saddle path” that bifurcates the system.

Finally, we examine the effect of the importance of alewife biomass to recruitment to the fishery. This is determined by the parameter  $\phi$ . We increase the value of this parameter from 0.1 to 0.2. A large value of  $\phi$  enables greater fishable salmon biomass per unit of salmon biomass stocked. When alewife cause damages the



$w = w^{\max}$  boundary shifts upward, implying that it is optimal to stock fewer salmon at any point  $(s, a)$  because stocking lowers alewife biomass, resulting in a greater cost to salmon production and increasing the fourth term in equation (4.15) in absolute value. Furthermore, equilibrium A shifts up and to the right, while equilibrium B shifts down and to the left. This implies that when recruitment to the fishery is more dependent on alewife, all else equal, alewife-induced damages have to be greater for the system to shift such that pursuit of equilibrium B is optimal. Specifically, in equation (4.16) the term  $(b+w)\theta'(a)$  increases and in order for (16) to continue to hold, damages must increase. The solution itself, however, is qualitatively unchanged.

Increasing the importance of alewife to recruitment to the fishery had a qualitative influence on results when alewife do not cause damages (Figure 4.5). Only equilibrium A continues to exist in the interior region. It is never optimal to stock at the maximum level. Continued pursuit of the singular solution stocking strategy,  $w = w^*(s, a)$ , is a myopic solution and leads to a point on the other side of the saddle path. Starting at  $s = 0$  and  $a = K$  the  $w = w^*(s, a)$  solution is initially pursued. Continued pursuit past point X (Figure 4.5) would lead to eradication of the alewife stock. This is not optimal. At point X the far-sighted planner recognizes that it is optimal to switch “prematurely” relative to the point where the boundary would be imposed (Clark *et al.* 1979; Clark 1990) and impose the  $w = 0$  rule. This rule is applied until the system re-enters the interior region by crossing the  $w = 0$  boundary in the low right quadrant of the phase plane. At that point the  $w = w^*(s, a)$  is applied until the system arrives at point Y. At point Y the manager again recognizes that it is optimal to switch prematurely to the  $w = 0$  stocking rule. The arc X-Z is just above the bifurcating saddle

path; points above this line optimally lead to cycles around point A. For points below this line, managers optimal stock zero salmon and have no other control over the system. Therefore, alewives are eradicated.

## 4.7 Discussion

### *The interaction of imperfectly targeted controls and non-convexities*

The issue of non-convexities and multiple equilibria in natural resource economics is increasingly important (Dasgupta and Maler 2003). Many ecosystem processes include thresholds and multiple basins of attraction. Most previous work on ecologically driven non-convexities (e.g., Toivonen and Salo 1996; Brock and Starrett 2003) assumes convex-concave piecewise differentiable functional forms. In our model, three trophic levels (anglers, salmon, and alewife) interact to create an ecologically driven non-convexity, though none are explicitly modeled with convex-concave functions. The combination of decentralized human behavior (i.e., an open access fishery), ecological processes, and stocking leads to multiple equilibria. We note, however, that the findings of these previous authors are applicable and informative to this case where non-convexities were not imposed *a priori*, but rather emerged due to ecological-economic interactions.

Non-convexities are of interest because they imply that there are multiple candidate equilibria, and the equilibrium that is pursued may depend on initial conditions. In other words, there are multiple basins of attraction. There are two types of bifurcation in this model. First, there is the previously discussed bifurcation *line*, i.e., the saddle path. Below the saddle path there is a region of the state space where the

planner optimal stocks zero salmon, and has no additional control of the system. This can be thought of as a positive feedback process (Brock and Starrett 2003). Below the saddle path the system moves further and further from the region where stocking is not desirable.

The other bifurcation is a bifurcation *point* and has to do with changes in parameters. As alewife damages increase there is a value for  $D$  at which the qualitative nature of the optimal solution changes. This is associated with joint production, the inability to perfectly target controls, and the “multiple-use species” problem (Zivin *et al.* 2000; Rondeau 2001; Horan and Bulte 2004). Specifically, for small levels of damage the cycle illustrated in Figure 4.3a shift down and to the left. Increasing damages further, however, leads to a qualitative change in the nature of the solution. The new solution type is illustrated in Figure 4.4a.

In the current case, there is a single control for managing the angler-salmon-alewife system. Alewives do not provide existence ecosystem disservices (though we model it this way for convenience and for lack of a known intervention opportunity). In reality, alewives cause damages to infrastructure and native fish populations. If managers had other controls available to avert or remedy these damages or there sufficient alternated prey items available to salmon the non-convexity associated with “multiple-use species” problem might vanish. Interdependency and the inability to perfectly target controls, however, are fundamental to the “multiple-use species” non-convexity.

The model presented here focuses on imperfectly targeted controls and non-convexities. The imperfect targeting of controls can be seen as an additional

institutional constraint (Dasgupta and Maler 2003) that is associated with the trouble of managing recreational fisheries (Cox *et al.* 2002) and perhaps all renewable resources. The need to incorporate institutional constraints to develop policy-relevant models can not be overstated. Such institutional constraints will often lead to imperfectly targeted control options that will lead to an increasing importance for ecologists and economists to work together to determine how to optimally allocate natural resources in a world full of non-convexities.

#### *Implications for salmon stocking policy*

The economic-ecological interactions associated with the salmon fishery on Lake Michigan are more complex than any model can fully capture, but the results presented here may provide insight into the tradeoffs associated with the stocking program. Therefore, this analysis can be used to inform stocking policy in concert with more complex simulation models (e.g., Jones and Bence in review). We note four key policy relevant findings *i)* the optimal level of stocking is a function of the current salmon and alewife stock, *ii)* the optimal of alewife eradication depends on current conditions<sup>9</sup>, *iii)* consideration of ecosystem disservices caused by alewife qualitatively alter the optimal program, *iv)* the maximum catch along the optimal management path does not occur at equilibrium.

The optimal stocking level depends on the current state of the system. Often linear control problems result in constrained solutions where a maximum or minimum level of control is applied to rapidly arrive at equilibrium (Conrad and Clark 1987). In

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<sup>9</sup> Alewife eradication in the model depends on the assumption of a type-II predator response. This result however, may be interpreted as extreme alewife suppression. Such extreme suppression may lead to other alternative ecological equilibria.

cases where controls are poorly targeted, as is the case of trying to manage a salmon-alewife fishery (which is effectively a tri-trophic system), adjustment is likely to be sluggish and involve nonlinear feedback rules (see Horan and Wolf 2005; Fenichel and Horan 2007a for other examples). This suggests that stocking policies that respond to the state of the world will provide greater social benefits than state-independent stocking strategies. This supports earlier simulation results by Szalai (2003) and Jones and Bence (in review). The optimal “equilibrium”, however, may be a stable cycle that requires constant adjustment. If there is non-malleable capital involved in generating fishery benefits, and if there are adjustment costs associated with varying catches, then such cycles may be dampened. Finally, we note that the optimal approach path to the equilibrium (or stable cycle) leads to harvest levels that exceed those experienced at equilibrium (or along the stable cycle). This is because it is optimal to “over-invest” in salmon initially to reduce the alewife population and capture benefits associated with the prey stock early. An alternative way to think about this is that it is optimal to experience some higher catch levels early in the fishery that are not sustainable.

Alewife damages create the possibility for a bioeconomic regime shift. Under high alewife-induced damages, management effort optimally shifts to averting these damages and the salmon fishery shrinks drastically, in terms of catch and total net benefit for anglers from the Chinook salmon fishery. It is important to note, however, that averted alewife damages are synonymous with increases in the benefits from other fisheries such as lake trout and yellow perch. This are not modeled explicitly, but are assumed to be part of the alewife damage function.

We find that even though in our model alewife eradication is technically feasible (this may not be the case in reality) the optimality of alewife eradication (conservation) depends on initial conditions. For high initial levels of alewife biomass eradication is never optimal. Conversely, for low initial biomasses of alewife with significant salmon stocks eradication may not only be optimal, it may be inevitable. If alternate prey existed to support the salmon fishery (it does not in our model), then the region where eradication of alewife may be optimal might be larger; we leave this for future work. If salmon have a type-III predator response for alewife, then an alternate equilibrium with low alewife and low salmon may exist. This too is left for future investigation.

Finally, we revisit the notion that salmon stocking creates a win-win situation with regard to angling and invasive alewife management. There is a stocking program that maximizes social welfare, but there are still tradeoffs, as is almost always the case in natural resource management. The use of salmon stocking clearly can provide benefits on both fronts, but the ultimate result may not be perceived as a win-win situation, especially if different stakeholders have different preferences for salmon angling and averting alewife damages.

#### **4.8 Appendix**

This appendix explains the calibration of the model. Unless otherwise noted biological parameters are based on Szalai (2003) or the Lake Michigan Decision Analysis (LMDA) Model (Jones and Bence in review). Most biological parameters for fish are based on size, we use age and weight data to convert these for parameter relevant to a

“representative fish.” The instantaneous net recruitment rate for alewife,  $r$ , was calculated based on per biomass recruitment of alewife minus the natural mortality rate for alewife as 5.41. The carrying capacity for alewife,  $K$ , was based on simulations of the LMMA model with no stocking, and was estimated at  $2.89 \times 10^9$  kilograms of alewife ( $a$ ). The predation parameters  $\beta$  and  $\omega$  were calculated based on bioenergetics, weight, and age data reported in Szalai (2003) as  $\beta = 1.67 \times 10^{-4} a^{-1}$  and  $\omega = 4.27 \times 10^{-8} 1/st$ . The salmon mortality parameters  $\alpha = 5.82 t^{-1}$  and  $\gamma = 0.02 a^{-1}$  were chosen such that salmon had a high instantaneous mortality rate in the absence of alewives and were comparable to the imputed representative instantaneous mortality rates reported by Benjamin and Bence (2003b) associated with comparable alewife biomasses. Salmon recruitment to fishery parameters  $\phi = 0.1$  and  $y = 58 1/at$  were based on bioenergetics parameters in Szalai (2003) and then manipulated to fit historic salmon level. Salmon natural recruitment was assumed to be zero,  $b = 0$  kg.

The recreational angling behavior parameters included  $x_1 = 49.08$ ,  $x_2 = 6.47$ ,  $x_3 = 69.41$ ,  $\eta = 6.66$ , and  $\sigma = 1.74$ . An additional parameter was added to the cost function so that the lognormal distribution was shifted to the right so that it was defined from \$35.93 to infinity. These parameters were estimated by assuming 13% of angling license sold in Michigan resulted in Lake Michigan salmon fishing trips, and fitting a time series of the number of anglers, effort, catch, and salmon biomass (Benjamin and Bence 2003b and Jones personal communication) to the angler response model by minimizing the sum of the squared error between that projected by the model and the observations. The alewife damage function was  $(\$1.78 \times 10^{-11})a^2$ . The daily catchability,  $q = 6.43 \times 10^{-8}$  is based on Benjamin and Bence (2003b) converted for

different time units. The marginal cost of stocking a kilogram of salmon,  $v = \$19.55$  was based on personal communication with Gary Whelan (Michigan Department of Natural Resources) and weight-at-age data (Szalai 2003). The maximum number of anglers was assumed to be the largest value in the number of angler time series. The maximum number of salmon that could be stocked was chosen to be slightly greater than historic high stocking levels at 40,500 kg (Jones personal communication). The discount rate,  $\rho$ , was assumed to be 5%, which is within the range of standard discount rates used in natural resource economics.



#### 4.9 Figures

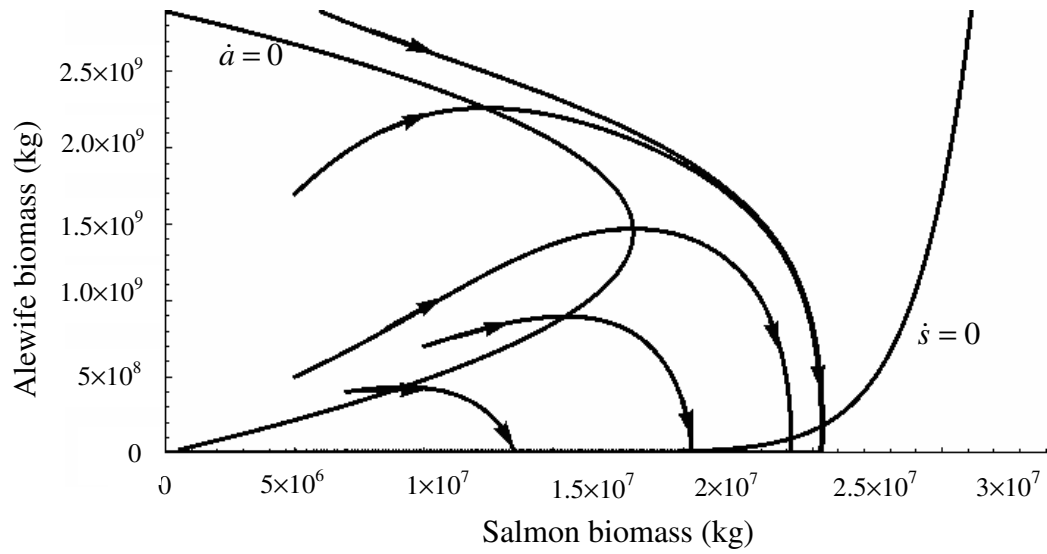


Figure 4.1. Phase plane illustrating the dynamics of stocking salmon at the maximum rate,  $w = w^{\max}$ . Salmon biomass is shown on the  $x$ -axis and alewife biomass is shown on the  $y$ -axis. Dashed lines represent the stocking zero and stocking maximum boundaries (in lower right corner the  $w = 0$  boundary and the  $\dot{s} = 0$  isocline are so close they appear as a single solid line). The salmon axis is extended to 1.5 times the historic high for salmon biomass in Lake Michigan.

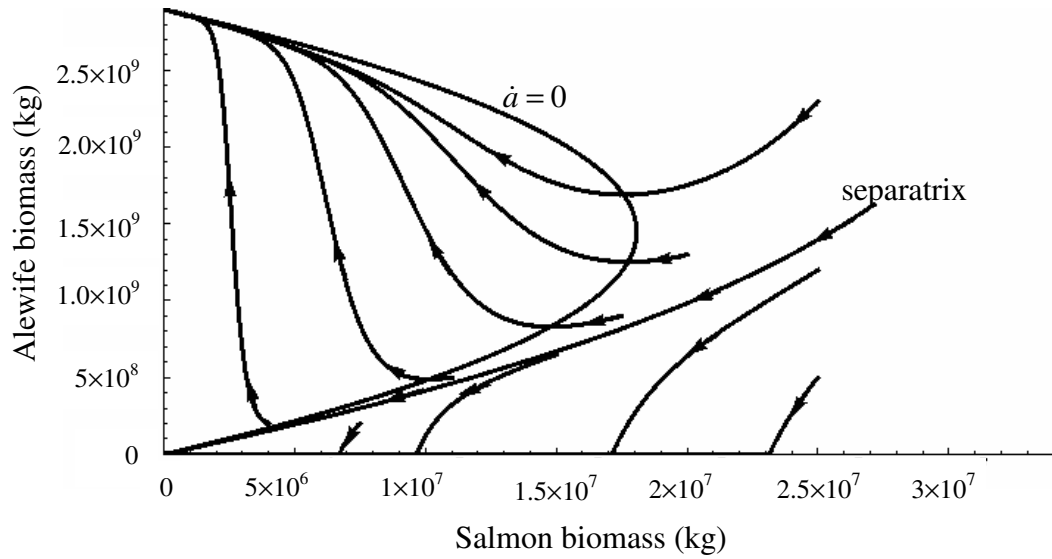


Figure 4.2. Phase plane illustrating the dynamics of stocking zero salmon,  $w = 0$ . Salmon biomass is shown on the  $x$ -axis and alewife biomass is shown on the  $y$ -axis. Dashed lines represent the stocking zero and stocking maximum boundaries (in lower right corner the  $w = 0$  boundary and the  $s = 0$  isocline are so close they appear as a single solid line). The salmon axis is extended to 1.5 times the historic high for salmon biomass in Lake Michigan.

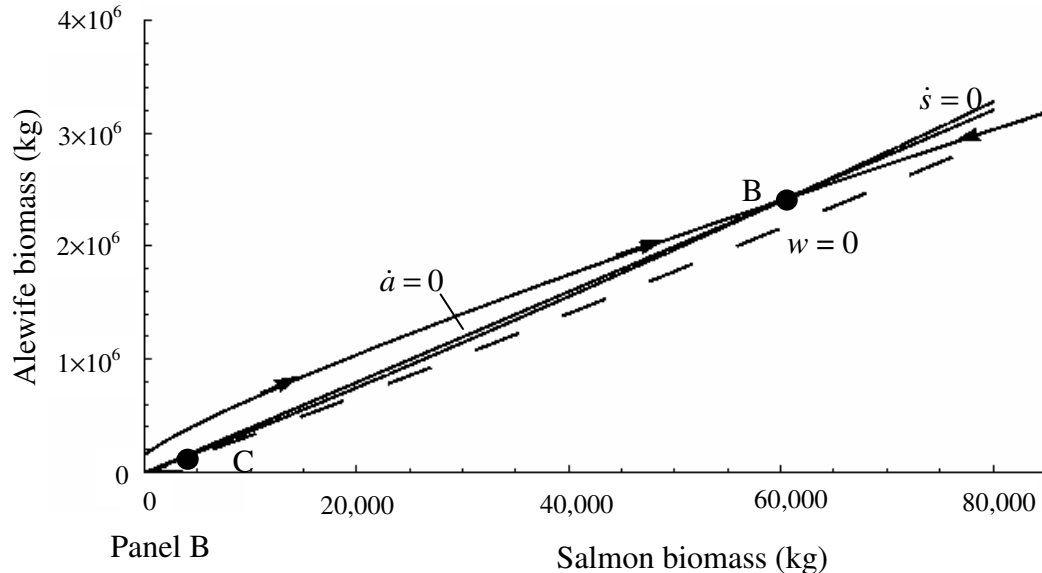
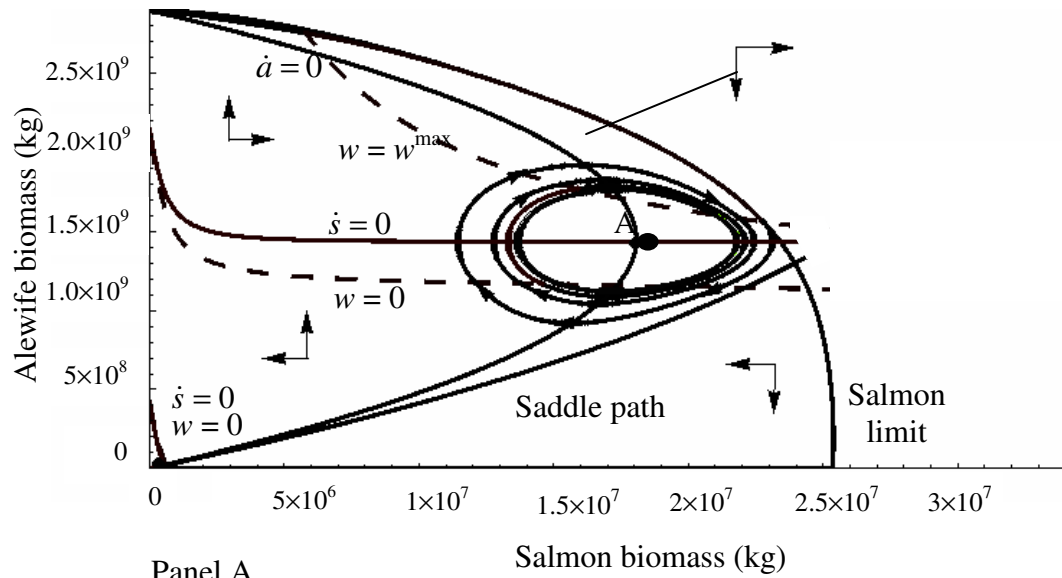


Figure 4.3. Phase plane illustrating the dynamics of the optimal solution in the case with no alewife damages. Salmon biomass is shown on the  $x$ -axis and alewife biomass is shown on the  $y$ -axis. Panel B expands the lower left hand corner of panel A. The salmon limit is the maximum number of salmon that can be put into the system determined by stocking at the maximum allowable level from an initial point of zero salmon and alewife at carrying capacity. Dashed lines represent the stocking zero and stocking maximum boundaries (in lower right corner of panel A the  $w = 0$  boundary and the  $\dot{s} = 0$  isocline are so close they appear as a single solid line). The arcs with arrows represent optimal solutions from the initial point of zero salmon and  $K$  alewives and the separatrices. The small regions in panel B do not permit phase arrows, but local dynamics can be inferred from the arrows on the saddle path. In panel A the salmon axis is extended to 1.5 times the historic high for salmon biomass in Lake Michigan.

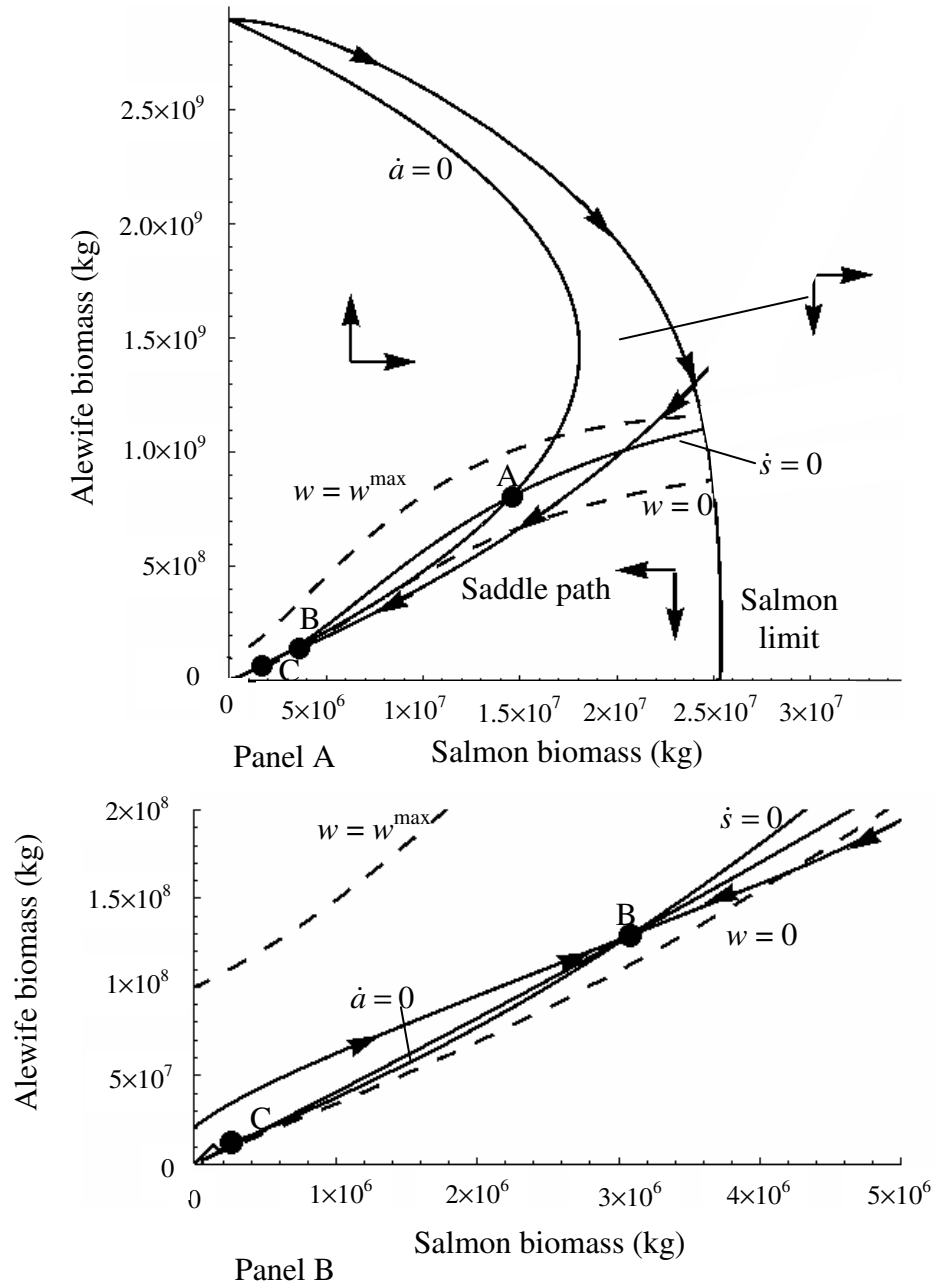


Figure 4.4. Phase plane illustrating the dynamics of the optimal solution in the case with alewife damages. Salmon biomass is shown on the  $x$ -axis and alewife biomass is shown on the  $y$ -axis. Panel B expands the lower left hand corner of panel A. The salmon limit is the maximum number of salmon that can be put into the system determined by stocking at the maximum allowable level from an initial point of zero salmon and alewife at carrying capacity. Dashed lines represent the stocking zero and stocking maximum boundaries. The arcs with arrows represent optimal solutions from the initial point of zero salmon and  $K$  alewives and the separatrices. In panel A the salmon axis is extended to 1.5 times the historic high for salmon biomass in Lake Michigan. The small regions in panel B do not permit phase arrows, but local dynamics can be inferred from the arrows on the saddle path.

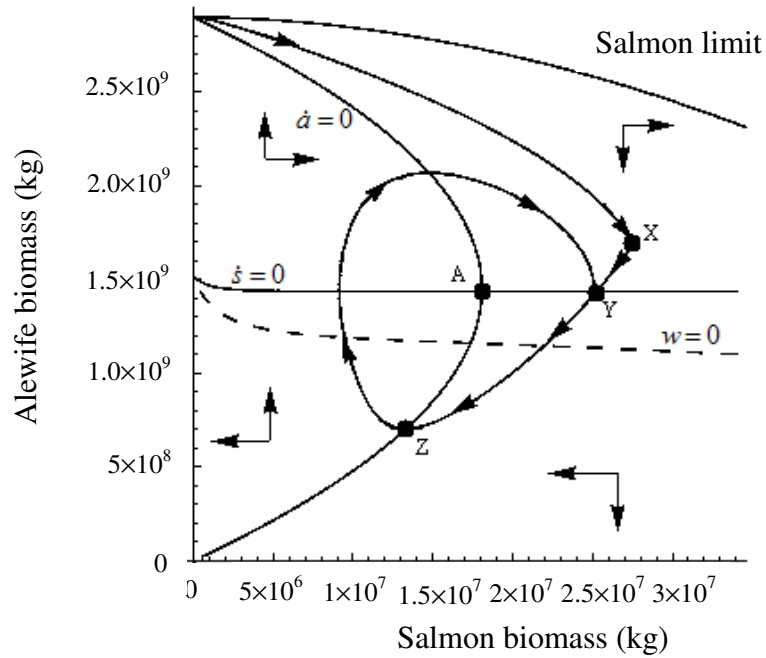


Figure 4.5. Phase plane illustrating the dynamics of the optimal solution in the case with no alewife damages and increased dependence on alewife for recruitment to the fishery;  $\phi$  increased from 0.1 to 0.2. Salmon biomass is shown on the  $x$ -axis and alewife biomass is shown on the  $y$ -axis. The salmon limit is the maximum number of salmon that can be put into the system determined by stocking at the maximum allowable level from an initial point of zero salmon and alewife at carrying capacity. The dashed line represents the stocking zero boundary. The arcs with arrows represent optimal solutions from the initial point of zero salmon and  $K$  alewife. The salmon axis is extended to 1.5 times the historic high for salmon biomass in Lake Michigan.

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