PEOPLE, PREDATORS AND PARASITES: UNEXPECTED DYNAMICS OF SEA LICE IN PACIFIC CANADA

by

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Abstract

The current rate and extent of human-induced changes to the environment are unprecedented. There is an urgent need to understand and predict the dynamics of coupled human and natural systems so that we can maintain the ecosystem services on which we depend. Temperate coastal regions have experienced a rapid expansion of open-net salmon farming in recent decades. The success of aquaculture depends on maintaining a healthy coastal environment in which to grow fish, but the transition to aquaculture has changed the structure of coastal fish populations and lead to the unexpected emergence of disease in both farmed and wild populations. In particular, sea louse parasites are a persisting problem that threaten the long-term sustainability of salmon farming. In this thesis, I consider the reciprocal interactions between people and parasites and between parasites and predators that mediate sea louse dynamics on farmed salmon and impacts on wild fish populations. Analysis of a simple model for parasites on adjacent salmon farms coupled by dispersal suggested that strict thresholds for parasite abundance requiring management intervention may actually hinder efforts to eradicate sea lice at the regional scale. Model analysis and examination of long-term data on sea lice of farmed and wild salmon from the Broughton Archipelago of British Columbia show that coordinated timing of treatments among farms is more effective at reduc-

ing sea lice on farmed salmon for the betterment of wild salmon populations. But assessing the effects of sea lice on migrating wild juvenile salmon is much more complex than accounting for transmission from farms as wild salmon are embedded in an ecosystem and subject to interacting pressure from predation and competition. In the second part of my thesis, I consider how predators may mediate the effect of parasites in multi-host systems. Model analysis and field-based experiments suggest that selective predation on pink salmon and on parasitized prey may result in a parasite-mediated release from predation for chum salmon, a less-desirable prey species. This is contrary to previous research suggesting that parasites increase the predation susceptibility of both juvenile pink and chum salmon and may explain why chum salmon population appear unaffected by sea louse epizootics in the early 2000s while pink salmon populations declined. Throughout this work, I have found that unexpected behaviour can emerge from a combination of factors – people, predators and parasites – even when each part seems well understood in isolation. The complexity ecosystems and our role in them is important for ecologists and policy makers to consider as we enter an era of unprecedented human growth and impacts on natural ecosystems.

Preface

Some of the research conducted for this thesis was the result of collaborations with partners from academia, industry, government, and non-government organizations. My supervisors, Martin Krkošek and Mark Lewis, contributed to the ideas, analyses and gave feedback on the writing of this thesis and associated publications. Other contributions are noted below.

Chapter 2 has been submitted to *Theoretical Ecology* and is currently under review. Coauthor Andrew Bateman contributed to the ideas and model analysis of this chapter.

Chapter 3 has been published in *Ecological Applications*. Coauthors Craig Orr and Stan Proboszcz of Watershed Watch Salmon Society contributed to the conceptual design and writing of the manuscript.

Chapter 4 has been published in *Proceedings of the Royal Society B: Biological Sciences*. Coauthor Brendan Connors had considerable input into the design of the study, data acquisition, and data analysis. Coauthor Jim Irvine contributed to the analysis of chum salmon spawner-recruit data and writing.

Chapter 5 is in press, to appear in *Ecosphere*. Coauthor Andrew Bateman contributed ideas, participated in data collection, and gave feedback on the writing of the manuscript.

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Chapter 1

General introduction

1.1 Emerging infectious diseases in wildlife

Wildlife populations globally are under pressure due to human-induced changes to their environment (Ceballos et al., 2015; Hautier et al., 2015). Emerging infectious diseases caused by bacterial, viral and parasitic pathogens are one challenge to the conservation of wildlife (Daszak et al., 2000; Deem et al., 2001; McCallum and Dobson, 1995; Pedersen et al., 2007), and have recently been increasing in incidence (Jones et al., 2008a; Tompkins et al., 2015). Humans have facilitated this increase in wildlife disease outbreaks via habitat degradation (Allan et al., 2003), climate change (Harvell et al., 2002), the introduction of invasive species (Tourchin et al., 2002), and establishment of domesticated host populations (Woodroffe, 1999).

The threat of disease is perhaps greatest for wildlife populations in contact with domesticated animals that can act as reservoir hosts (Pedersen et al., 2007; Woodroffe, 1999). In the absence of domesticated animals, density dependent transmission dictates that epizootics will fade-out as the host population declines (e.g., Hochachka and Dhondt, 2000). However, domesticated

animal populations can continue to transmit to even small wildlife populations, potentially driving them to extinction (De Castro and Bolker, 2005). Domestic dogs as reservoir hosts has been cited as a cause in the decline of critically-endangered Ethiopian wolves (Haydon et al., 2002) and spillover of rinderpest from cattle to wild ungulates has been well-documented (Plowright, 1982). However, the reverse scenario can also occur, where wildlife harbour disease and transmit to domestic animals, hindering efforts to eradicate the disease. For example, badgers have been implicated in recurrent outbreaks of tuberculosis in cattle in the UK, resulting in large-scale efforts to reduce badger populations (Donnelly et al., 2003).

The disease impact of domesticated animals on wildlife is perhaps greater than the reverse because farmed animals are not subject to the natural mechanisms that rid populations of diseased individuals - e.g., predation (Hudson et al., 1992; Murray et al., 1997; Ostfeld and Holt, 2004), competition (Godwin et al., 2015), and migration (Satterfield et al., 2015). Thus, domesticated animals can sustain much higher infection intensities than natural populations. Pathogens can be transmitted from wild to domesticated hosts, bioamplify in the farm environment, and then spill-back to infect wild populations. This process of spill-over and spill-back not only intensifies infection pressure on wild hosts, but can disrupt the natural mechanisms that reduce disease transmission in wildlife. For example, migratory escape from infection hotspots may reduce disease prevalence in wildlife (Altizer et al., 2011), but a stationary population of domesticated hosts can reinfect wildlife upon their return and undermine the disease-related benefits of migration (Krkošek et al., 2007b).

The transition from hunter/gatherer to modern agricultural practices in terrestrial systems was accompanied by a rise in infectious diseases of humans (Pearce-Duvet, 2006; Wolfe et al., 2007) and wildlife, but that transition happened over 10 000 years ago. We are currently witnessing a second agricultural

revolution - the blue revolution - characterized by a decline in capture fisheries (Dulvy et al., 2003; Myers and Worm, 2003) and a sharp increase in aquaculture (FAO, 2014). This transition to aquaculture has altered transmission dynamics for native pathogens and facilitated the emergence of novel pathogens (Kent, 2000; Walker and Winton, 2010). The impact of infectious diseases may be even more devastating in aquatic systems due to higher connectivity among populations in the aquatic environment relative to terrestrial systems (Gaughan, 2001; Walker and Winton, 2010). In this thesis, I focus on marine net pen salmon farming, which has expanded rapidly over recent decades (FAO, 2014) and resulted in dramatic changes to the distribution of hosts and disease dynamics in coastal ecosystems. I consider the reciprocal interactions between humans and parasites of farmed and wild fish, and the implications for wild salmon survival in Pacific Canada.

1.2 Study system

1.2.1 Ecology of sea lice

Lepeophtheirus salmonis, commonly known as sea lice or salmon lice, are marine copepods that infest salmon and trout. Sea lice reproduce sexually while attached to a host (Costello, 2006; Tully et al., 2002). Mated adult females extrude egg strings from which free-swimming and non-feeding nauplii hatch. Nauplii moult into copepodites, which become infectious and seek a host fish. Once attached to a host, copepodites develop through two chalimus stages and then motile pre-adult and adult stages (figure 1.1; Hamre et al., 2013; Johnson and Albright, 1991b). The motile stages are mobile over the surface of their host and can also move among host fish (Connors et al., 2008b, 2011; Ritche, 1997).

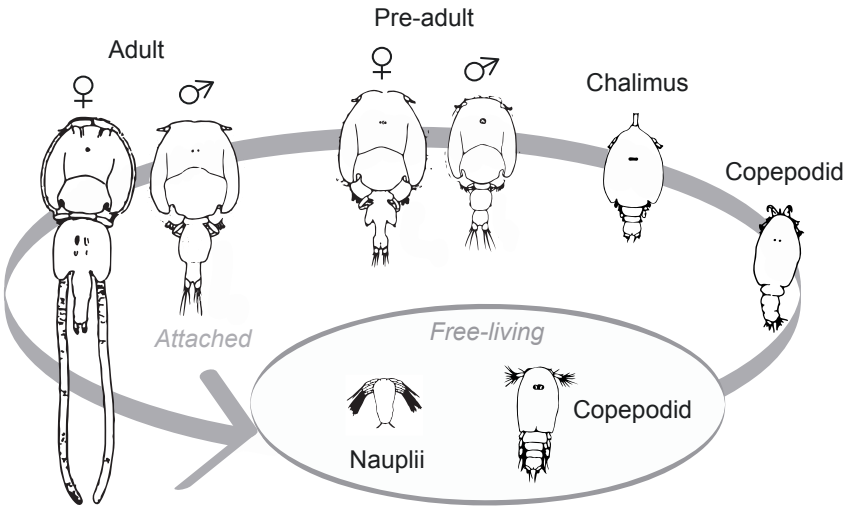

Figure 1.1: The lifecycle of the sea louse, *Lepeophtheirus salmonis*. Free-living nauplii moult into infectious copepodites, which attach to a host and develop through two chalimus stages, and pre-adult and adult stages. Adults reproduce sexually on the host, and gravid females extrude egg strings from which nauplii hatch, completing the lifecycle. Adapted from Schram (1993).

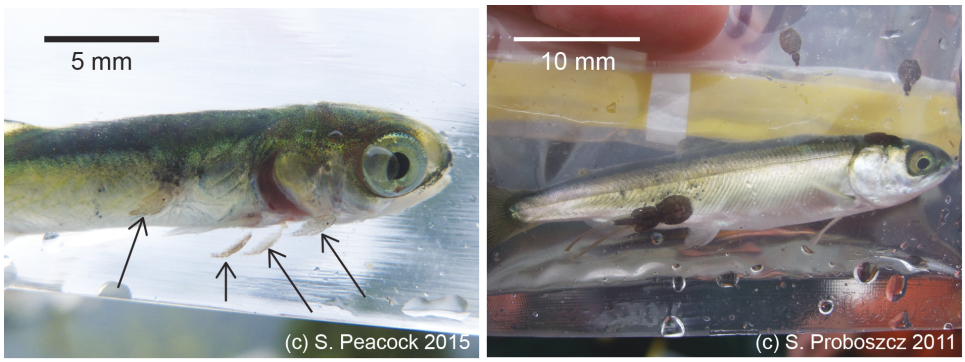

Figure 1.2: Chalimus-stage sea lice are securely attached by a frontal filament (left, arrows), while adult sea lice are mobile on the host (right, gravid female *L. salmonis* on flank).

Sea lice feed on host epidermis, muscle and blood, causing damage to host surface tissues (figure 1.2; Costello, 2006). For adult salmon, the impact on overall fish health may be minimal. However, for smaller juvenile fish, infestations can lead to osmoregulatory stress (Brauner et al., 2012), expose hosts to secondary infections (Johnson et al., 2004), and cause host behavioural changes (Krkošek et al., 2011a) or death (Krkošek et al., 2009; Morton and Routledge, 2005). Sea louse infestations may also have ecological impacts on juvenile wild salmon as infested individuals have compromised schooling (Krkošek et al., 2011a) and swimming abilities (Mages and Dill, 2010; Nendick et al., 2011) and may be unable to complete migrations or evade predators (Krkošek et al., 2011a). Juvenile salmon experience very high predation rates during early marine life (Parker, 1968, 1971), suggesting that the effects of parasitism on predator-prey interactions may be important for evaluating the consequences of sea lice infestations on salmon population dynamics. I explore this idea further in chapter 4 and chapter 5.

1.2.2 Ecology of Pacific salmon

Pacific salmon (*Oncorhynchus* spp.) are anadromous and semelparous species; they hatch in freshwater, migrate to sea to spend the majority of their lives and then return to freshwater to spawn and die (figure 1.3). The different species of Pacific salmon display variations on this general theme. There are five salmon species and two trout species from the genus *Oncorhynchus* that inhabit British Columbia waters (Quinn, 2005). Of particular interest for this work are the life histories of pink, chum and coho salmon. Pink salmon (*O. gorbuscha*) and chum salmon (*O. keta*) migrate immediately to sea after hatching, entering the marine environment when they are small and vulnerable to both parasites and predators (Groot and Margolis, 1991). Pink salmon return to spawn exactly

a year and a half later, resulting in a two year lifecycle that separates even- and odd-year populations. Chum salmon, on the other hand, spend three to six years at sea. Coho salmon (*O. kisutch*) have an alternative strategy as juveniles, spending a year in freshwater before heading seaward, and generally return as three to five year olds. Due to these differences, yearling coho salmon are actually a primary predator of juvenile pink and chum fry in the coastal marine environment (Parker, 1968).

Juvenile pink and chum salmon are often observed in mixed-species schools when migrating seaward (Groot and Margolis, 1991). At this stage, pink and chum salmon are small in size ($\sim$ 30 mm body length and 0.2 g weight) and have underdeveloped epidermal, immune and osmoregulatory systems (Sackville et al., 2011). Further, they do not develop scales until several weeks after marine entry and therefore lack the mechanical resistance to sea louse attachment and feeding that scales may confer (Jones et al., 2008b). Because of their similar early life histories and comparable rates of direct parasite-induced mortality (Krkošek et al., 2009), both juvenile pink and chum salmon may succumb to even low levels of parasitism (figure 1.2; Krkošek et al., 2006a). Research on sea lice and juvenile salmon has focused on direct physiological effects of sea lice (e.g., Brauner et al., 2012), but ecological effects including parasite-induced changes to predation vulnerability may be important (Krkošek et al., 2011a), especially given the high levels of predation on juvenile salmon in the wild (Parker, 1968).

1.2.3 Transmission between farmed and wild salmon

In regions without salmon aquaculture, the vulnerability of juvenile salmon to sea lice may be of little consequence. Outwardly-migrating juvenile salmon are relatively free of sea lice, which cannot survive in freshwater (Gottesfeld

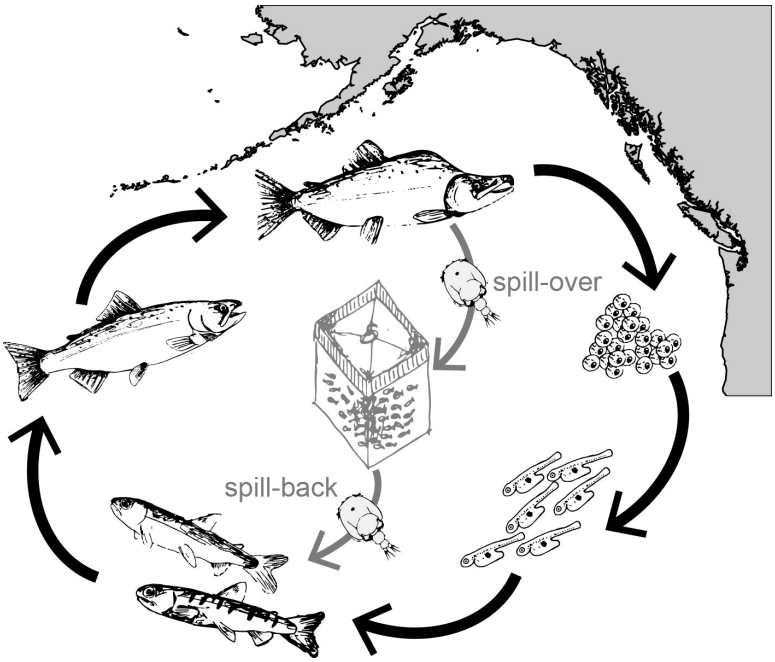

Figure 1.3: In the absence of salmon farms, migratory allopatry separates vulnerable juvenile salmon from pathogens of adult salmon (Krkošek et al., 2007b). The introduction of salmon farms provides a novel host population for parasites and pathogens, such as sea lice, that can be infected by returning adult salmon (spillover; Daszak et al., 2000). Pathogens are then amplified, and can then spill-back to infect juvenile wild salmon in coastal ecosystems.

et al., 2009). Juvenile salmon are not exposed to substantial numbers of sea lice until several months into their migration when they encounter returning adult salmon (Krkošek et al., 2007b). However in recent decades, salmon farms have provided a host reservoir population for sea lice that persists year-round in close proximity to salmon-bearing rivers (figure 1.3). The high density of hosts on salmon farms can amplify natural infestations and sea lice can spill back from farmed salmon to infest juvenile wild salmon very early in the juvenile salmon migration (Krkošek et al., 2006a). Epizootics of sea lice on farmed salmon have been implicated in the decline of wild salmon in Pacific Canada (Krkošek et al., 2011b) and Europe (Bjørn et al., 2001; Butler and Watt, 2003; Costello, 2009; Krkošek et al., 2013). The expansion of salmon aquaculture (FAO, 2012) has therefore raised conservation concerns in regions where the narrow inlets occupied by salmon farms are important migratory corridors for wild salmon.

One region where wild salmon share the inlets and bays with an increasing number of salmon farms is the Broughton Archipelago of British Columbia, Canada (figure 1.4). The survival of Broughton coho and pink salmon declined concurrent with sea louse infestations on salmon farms in the early 2000s (Krkošek et al., 2011b). There remains uncertainty about the magnitude of these effects due to the potential for unidentified confounding factors affecting salmon survival as well as both process and observation error. Conflicting reports (e.g., Krkošek et al., 2011b; Marty et al., 2010) highlight the sensitivity of these results to model assumptions and error. Despite these uncertainties, or perhaps because of them, the evidence linking sea louse infestations to declines in pink salmon has triggered flurry of scientific activity and public debate centred around the Broughton Archipelago.

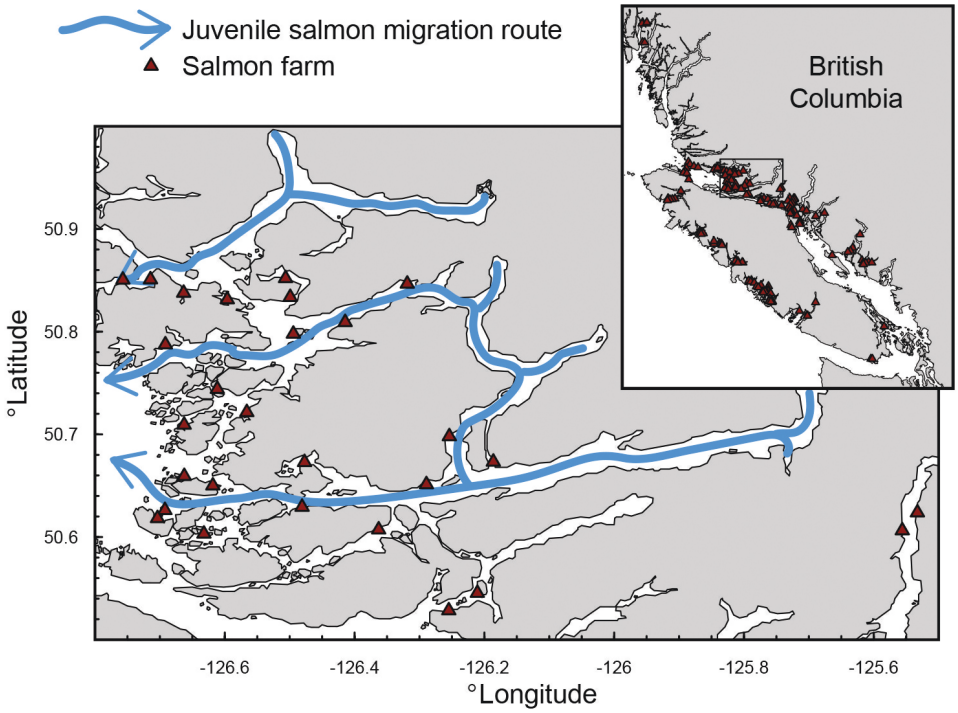

Figure 1.4: The Broughton Archipelago of British Columbia, Canada is home to over 30 open-net salmon farms (triangles), sited along on the migration route of juvenile wild salmon (blue line).

1.2.4 Sea louse control

Salmon farms employ a variety of methods to manage sea lice including fallowing of farms following harvest, regularly removing moribund fish from pens, using cleaner fish such as wrasse that will eat sea lice, and administering parasiticide treatments in fish feed or by immersion of the net pens (reviewed by Igboeli et al., 2014; Rae, 2002). In Pacific Canada, the parasiticide emamectin benzoate (SLICE®, Merck Animal Health) is the only chemical treatment for sea lice that has been approved for the past decade (Saksida et al., 2013). SLICE® is given to farmed salmon in feed, which has the benefit of being easy and safe to administer but can result in variable dosages among fish depending on their feeding rates, less effective treatments, and promote the evolution of resistance to parasiticide (Igboeli et al., 2014). Sea louse resistance to drugs is a major threat to sea louse management on salmon farms (Aaen et al., 2015; Igboeli et al., 2014), but has yet to develop in Pacific Canada (Saksida et al., 2013).

Optimization of parasite management on salmon farms on the Pacific coast of Canada has involved formal policy development and management changes (Brooks, 2009; Krkošek et al., 2010). In the Broughton Archipelago, outbreaks of sea lice on wild juvenile pink salmon in 2001 and 2002 and accompanying population collapse of pink salmon stocks (Morton et al., 2004; Morton and Williams, 2003; PFRCC, 2002) triggered media attention, policy development, management changes and scientific investigation (Krkošek, 2010b). Government regulators initiated guidelines for systematic monthly monitoring of sea lice on farms in 2003, and added that treatment with SLICE® or harvest of a farm should occur if the average abundance of motile-stage lice exceeds three lice per farm fish during the months March-June when most juvenile wild salmon migrate through the area. During the remainder of the

year, it was initially suggested farms treat or harvest if lice levels exceeded six motile lice per farm fish, but in 2006, this was changed to an increased monitoring frequency of twice per month and optional harvest or treatment at the discretion of the farming company (`www.agf.gov.bc.ca/ahc/fish_health/sealice_MS.htm`).

1.3 Models as tools

Throughout my thesis, I use a variety of complementary approaches, including mathematical modelling and simulation analysis, fitting models to long-term observational datasets, and field-based experimentation. Here, I describe the general modelling approaches I use and introduce some of the basic models I will apply in the proceeding chapters.

The use of models as tools to understand the significance of biological observations is common in ecology. Statistical models such as the t-test are used to determine if biologically interesting trends underlie our apparently random observations. I apply statistical regression models in chapter 3 and chapter 5, in trying to understand what factors are important in explaining my observations of sea lice on wild juvenile salmon and the prey-preference of coho salmon predators.

Mechanistic models do more than just tell us whether a factor can explain some of the variance in our observations. Additionally, they attempt to uncover the processes that connect those factors and give rise to the observed patterns. Mechanistic models describe processes at a more detailed level of biological organization than that of the observations, and the use the outcome to understand the observed patterns at a broader scale. Whether a model is considered mechanistic therefore depends on the scale of inference. For ex-

ample, the processes of birth, death, predation and disease may all affect the dynamics of a salmon population. A mechanistic model explicitly incorporating the dynamics of these processes can be used to predict population-level survival from one year to the next. Parameters in mechanistic models can be estimated from data using likelihood, but mechanistic models can also be useful in the absence of data. Model simulations drawing on parameters from previous studies or simulations over a range of parameter values can elucidate the conditions under which we might expect certain behaviour to arise and allow us to make predictions that can then be tested in the field. I take this simulation approach in chapter 4, which leads to the experimental work of chapter 5 to test the assumptions of the model.

In assessing the influence of sea lice on wild salmon populations, it is necessary to account for the natural dynamics of those populations including density dependence. The Ricker model (Ricker, 1954) is one of the most widely applied models in fisheries science, and describes the discrete-time dynamics of a population as $R_t = S_t e^{r-bS_t}$, where R_t are the recruits from spawners S_t in year t, r is the growth rate at low spawner spawner abundance and b is the inverse of the carrying capacity, describing negative density dependence (figure 1.5a). A characteristic of the Ricker model is overcompensation, where a large spawner abundance can lead to a crash in the number of recruits. Overcompensation results from "scramble competition", and may occur in salmon due to, for example, interference among spawners excavating redds, oxygen limitation in redds, and/or the depletion of food resources for juveniles (Ricker, 1954). The inclusion of additional factors in the exponent of the Ricker model is a common approach to assess the importance of various factors thought to influence survival (Connors et al., 2012; Krkošek et al., 2011b). In chapter 3 and chapter 4, I apply a modified version of the Ricker model that includes the effect of sea lice on the number of recruits to determine the importance of parasitism to

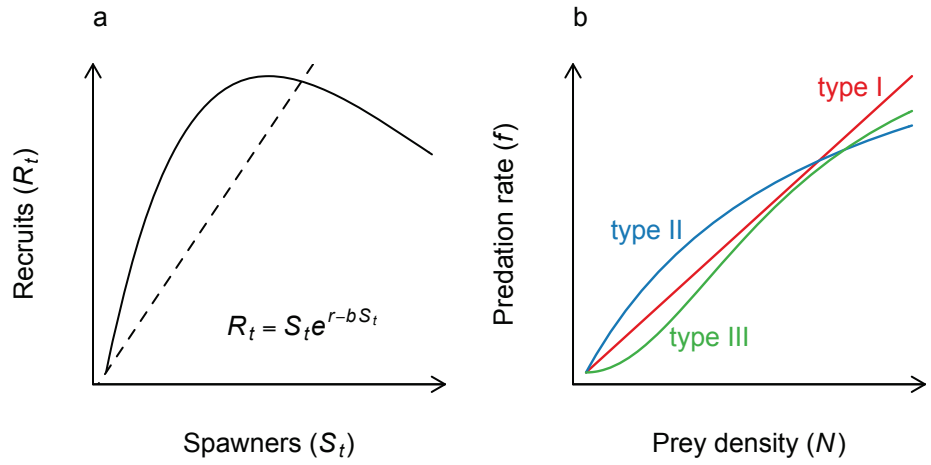

Figure 1.5: Illustration of (a) the Ricker model (Ricker, 1954) and (b) the three Holling functional response types (Holling, 1959).

population-level survival of wild pink and chum salmon. I also include random effects in the Ricker equation to account for shared variation among salmon populations at various spatial scales (Krkošek and Hilborn, 2011).

Around the same time that Bill Ricker was developing his model for stock and recruitment (Ricker, 1954), another Canadian ecologist was also investigating the nonlinear dynamics of populations. Crawford Stanley (Buzz) Holling developed what would become a widely applied model for how the consumption rate of a predator changes with prey density, known as the functional response (Holling, 1959). The general equation for the functional response is $f(N) = aN^n/(1 + aTN^n)$, where f is the instantaneous rate of consumption per predator, N is the prey density, T is the handling time and a is the attack rate and n is the number times a predator must encounter a prey item before feeding on that prey at the maximum rate a (representing some learning behaviour Real, 1977). This general equation is often referred to as a type III functional response. The type II functional response is the nested model $n = 1$ and the type I functional response is the model with $T = 0$ and $n = 1$ (figure

1.5b). The functional response can also be adapted for generalist predators to include multiple prey types in the denominator, so that the predation rate on any one prey type declines as predators spend time consuming other prey types (Lawton et al., 1974). I use a two-prey type II functional response to describe predation by coho salmon smolts on juvenile pink and chum salmon in chapter 4 and chapter 5, but adapt the functional response to include a linear increase in the attack rate with the number of parasites per host such that prey with high numbers of parasite are consumed at a higher rate (Krkošek et al., 2011a).

To investigate how this parasite-mediated predation and direct parasite-induced mortality affect the survival of hosts/prey, it is necessary to consider how both host/prey density and parasite load change over time as prey and parasites are consumed. A system of coupled ordinary differential equations describing the rate of change in host and macroparasite populations as a function of natural and parasite-induced mortality was described by Anderson and May (1978) and quickly became influential in the field. The Anderson-May model considers host mortality as a linear function of the parasite load, and accounts for overdispersion of parasites among hosts in a population, which occurs for sea lice (Murray, 2002) and is common for macroparasites in general (Shaw et al., 1998). As with the previous model I have introduced, I adapted the Anderson-May host-parasite model to include an additional factor - host mortality due to predation, where predation rates change with prey/host abundance according to Holling's type II functional response but also depends on the number of parasites per host. I use this adapted model to understand how parasitism and parasite-mediated predation interact to affect the survival of two host species that interact through a shared predator.

Mechanistic models have been key to our understanding of how sea louse parasites transmit and influence farmed and wild salmon hosts (e.g., Frazer et al.,

2012; Krkošek et al., 2006a, 2005a), and play a central role in my thesis. The models described above make appearances throughout this thesis, where they are described in greater detail.

1.4 Disseration outline

A decade of research has greatly improved our understanding about the interactions between farmed and wild salmon in Pacific Canada. It is now widely accepted that sea lice transmit between farmed and wild salmon in British Columbia (Krkošek et al., 2011b; Marty et al., 2010), Norway (Bjørn et al., 2001), Ireland (Gargan et al., 2003) and Scotland (Butler and Watt, 2003), but the subsequent effects on wild salmon populations in Canada in particular have been debated (e.g., Brooks and Jones, 2008; Krkošek et al., 2007a, 2011b; Marty et al., 2010). Correlative studies have been complicated by multiple interacting sources of wild salmon mortality and various spatial scales of synchrony in salmon population dynamics and sea louse abundances. The gradual changes in management of sea lice on salmon farms in the Broughton Archipelago have likely altered the relationship between salmon farms and wild salmon productivity. The management of sea lice and implications for wild salmon form the initial part of my thesis, in which I ask (1) how are parasite population dynamics influenced by threshold control treatments and the dispersal of parasites among farms within a region, and (2) how have adaptive changes in sea louse management over the past decade affected sea lice on farmed and wild salmon, and what are the implications for wild pink salmon populations?

I begin in chapter 2 with a theoretical investigation of how the dispersal of larvae between two farms can affect the dynamics of treatment on a given farm. A relatively simple model reveals a rich variety of dynamical behaviour, and

provides an example of the complexity of coupled human and natural systems (Liu et al., 2007b). In chapter 3, I connect trends in treatment timing, sea lice on farmed salmon, and sea lice on wild salmon in the Broughton Archipelago to determine if improved treatment timing has resulting in lower numbers of sea lice on farmed and wild salmon, and a cessation of the pink salmon decline. The results confirmed previous analyses of pink salmon, finding a negative relationship between sea louse abundances and pink salmon survival, but the effect of sea lice on chum salmon survival was yet to be determined.

In the second part of my thesis, I investigate how chum salmon populations are affected by sea lice, which leads me to consider the effect of parasites in multi-host systems. Chapter 4 investigates the effect of sea lice on chum salmon populations, and unexpectedly I did not find a significant relationship. Are there ecological reasons why chum salmon populations may be less affected? I follow up with a theoretical model investigating the conditions under which predation might offset negative effects of sea lice on chum salmon. In chapter 5, I present a series of field-based experiments designed to test whether predation mortality of pink and chum salmon is different and the impact that sea lice have on predation of pink an chum in mixed species schools. Finally, in chapter 6, I discuss how our understanding of sea lice and salmon in Pacific Canada has evolved, and the lessons learned for management and conservation in other systems moving forward.

Chapter 2

Complex dynamics of coupled populations subject to control

2.1 Introduction

As the global human population grows, there is an increasing need to understand how interactions between human and natural systems alter ecosystems and the services they provide (Millennium Ecosystem Assessment, 2005). Social and ecological systems have traditionally been studied separately, but their integration as coupled human and natural systems (CHANS) can reveal unexpected dynamics due to nonlinearities and thresholds in the way that humans and ecosystems interact (Liu et al., 2007a). Emergent properties are characteristics of CHANS that are not present in human or natural systems in isolation, but emerge from the interactions between them. For example, overfishing has

A version of this chapter has been submitted and is in review at *Theoretical Ecology*. Used with permission of coauthors.

resulted in fundamental changes to the structure of fish communities that have hindered the recovery of collapsed stocks even after the cessation of fishing pressure (Savenkoff et al., 2007). There is a need to integrate studies of human actions with the natural dynamics of populations and communities to understand such feedbacks and develop effective policy that reduces human degradation of essential ecosystems services.

The unexpected appearance of new and re-emerging diseases is one consequence of humans altering their environment (Finucane et al., 2014). The onset of devastating infectious diseases in humans coincided with the agricultural revolution (Wolfe et al., 2007), with increasing population sizes and the transmission of zoonotic disease from domesticated animals being cited as causes (Pearce-Duvet, 2006). We are now undergoing a second agricultural revolution - the blue revolution - that is characterized by the rapid expansion of aquaculture to fill the growing gap between depleted ocean fisheries and a growing global population (FAO, 2014). The growth of aquaculture has resulted in changes to coastal ecosystems including the emergence of disease (Gaughan, 2001; Kent, 2000) and transmission of pathogens between farmed and wild fish (Heggberget et al., 1993; Krkošek et al., 2006b).

In regions where farmed and wild fish coexist, the health of the system depends on effective management of disease in farmed fish (Peacock et al., 2013; Tompkins et al., 2015), which can be complicated by the natural dynamics of growth and dispersal of pathogens in the marine environment. Connectivity among populations in the marine environment is typically higher than their terrestrial counterparts (McCallum et al., 2003), and dispersal of pathogens among host populations may be especially important in predicting disease dynamics for marine aquaculture. If pathogen populations on nearby farms fluctuate out of phase, such that high abundances at one farm correspond to low abundances at another, dispersal can increase the probability of persis-

tence via the rescue effect (Brown and Kodric-Brown, 1977; Kendall and Fox, 1998). The rescue effect is often thought of as beneficial in the context of population viability of endangered species, but in the context of disease, dispersal amongst local populations with asynchronous dynamics may hinder efforts to eradicate disease (e.g., Bolker and Grenfell, 1996). Interestingly, mathematical models (Hastings, 1993; Holt and McPeek, 1996; Liebhold et al., 2004) and observational data (e.g., Ranta et al., 1995; Steen et al., 1996) have suggested that dispersal will tend to synchronize local populations. Local populations with synchronized dynamics may be more susceptible to extinction becuase stochasticity or human intervention can cause catastrophic losses across synchronized populations when they are all at low abundance, with little opportunity for recolonization by neighbouring populations. Paradoxically, dispersal could therefore help or hinder efforts to control disease in metapopulations (Abbott, 2011).

Sea lice are parasitic marine copepods that have been a persisting problem for the aquaculture industry, costing millions of dollars in treatment and lower feed conversion ratios, negatively impacting fish health, and damaging public perception of farmed salmon (Costello, 2009). Sea louse populations on different salmon farms within a region are likely connected via the dispersal of free-living larvae (Adams et al., 2012; Stucchi et al., 2011). Studies have shown that critical host density thresholds for sea lice exist at regional scales (Frazer et al., 2012; Jansen et al., 2012; Kristoffersen et al., 2013), and it has been estimated that 28% of infections are due to the influx of larvae from neighbouring farms (Aldrin et al., 2013). This connectivity among farms affects the growth of sea louse populations on any given farm and the efficacy of treatments. There is also growing concern that more frequent and less effective treatments facilitate the evolution of resistance to chemotherapeutants in sea lice (Aaen et al., 2015; Igboeli et al., 2014). Coordination among farms may be key in

managing sea lice and the evolution of resistance (Kristoffersen et al., 2013), as well as the spread of other pathogens. Many studies have focused on statistical analyses of monitoring data to uncover the relationships among farms (e.g., Aldrin et al., 2013; Jansen et al., 2012; Revie et al., 2002; Rogers et al., 2013) but much can be learned from applying more general theoretical models of population and disease dynamics (e.g., Frazer, 2009; Frazer et al., 2012).

In this chapter, I develop a simple model for the dynamics of two populations connected by dispersal, where both populations are subject to external control when they reach a threshold value. The model complements previous work examining sea louse populations on individual salmon farms (Krkošek et al., 2010; Rogers et al., 2013) and within a region (Aldrin et al., 2013; Jansen et al., 2012) to explicitly examine how dispersal of larvae between two farms can alter the timing and frequency of treatments. This work also builds on our general theoretical understanding of how coupling (Dey et al., 2015; Goldwyn and Hastings, 2011; Hastings, 1993; Kendall and Fox, 1998) and intervention (Chau, 2000) affect the dynamics of metapopulations. My relatively simple model resulted in complex behaviour, including synchrony between populations or seemingly chaotic dynamics depending on the relative strength of internal growth and connectivity between the populations. The results provide motivation to further explore how natural population dynamics are changed by human intervention within metapopulations, and may help inform management of sea louse populations on salmon farms and other parasites or pathogens of spatially structured host populations.

2.2 Methods

2.2.1 A simple model for growth and control

Analyses of sea louse population dynamics on isolated salmon farms suggest that parasite populations grow exponentially in the absence of treatment (Krkošek et al., 2010; Rogers et al., 2013). This pattern of exponential growth is not unique to sea lice; exponential growth has been observed in birds (Van Bael and Pruett-Jones, 1996), mammals (Silva, 2003), and insects (Birch, 1948), and has been used to describe dynamics of forest and agricultural pests (e.g., Samways, 1979). Although negative density dependence will regulate populations at some point, management intervention in the case of pests and parasites may prevent populations from reaching such high densities. Thus, although the following model was motivated by sea louse parasites on salmon farms, it likely has broad applicability and may inform management of other pests and parasites. In developing the model, I refer to populations on adjacent patches rather than parasites on adjacent salmon farms to maintain this generality.

First, I consider a simple model of exponential population growth that includes dispersal between two populations in separate patches. I call these populations continuously coupled because dispersal happens continuously, as opposed to pulse coupled systems that are common in the study of coupled oscillators, where connectivity occurs only when a certain threshold abundance is reached (e.g., Mirollo and Strogatz, 1990). These two continuously-coupled populations are described by,

$$\begin{bmatrix} u \\ v \end{bmatrix}' = \begin{bmatrix} r_{uu} & r_{uv} \\ r_{vu} & r_{vv} \end{bmatrix} \begin{bmatrix} u \\ v \end{bmatrix}, \tag{2.1}$$

where u is the population density in patch one, v is the population density

in patch two, r_{ii} is the growth rate within population i where $i = u$ or v and r_{ij} is the dispersal rate of individuals from population j to population i. The solutions for $u(t) = f_u$ and $v(t) = f_v$ are given in Appendix 2.A.

I included control treatments in my model by forcing a reduction in a population when it reached a threshold abundance of $N_{\max}$. Many countries, including Norway, Ireland, the United States, and Canada, require salmon farms to treat their fish with chemotherapeutants when a threshold louse abundance is reached, but this threshold may vary among regions (Brooks, 2009). For my simulations, I chose $N_{\max} = 3$, based on guidelines in Pacific Canada that recommend treatment when farmed salmon have an average of three lice per fish (British Columbia Ministry of Agriculture and Lands, 2005), but the value of the threshold is arbitrary for the qualitative analysis I perform here. Observations suggest that chemotherapeutants may kill up to 95% of motile sea lice on treated farmed salmon (Lees et al., 2008). Therefore, when either u or v exceeded $N_{\max}$, I modelled a treatment of that patch by forcing the dynamics to reset with the initial condition for the treated patch being a 95% reduction from the threshold (i.e., $N_{\min} = (1 - 0.95)N_{\max}$), and the initial condition for the untreated patch being the population density in that patch when the other patch was treated. For example, if u reached the threshold $N_{\max}$ at time $t = T_u$, the system would be reset with $t = 0$ and initial conditions $u_0 = N_{\min}$ and $v_0 = f_v(T_u)$.

2.2.2 Discrete-time treatment dynamics

To understand the conditions under which the populations will become synchronized, settle into a regular pattern of alternating treatments, or have unpredictable treatment timing, from equation 2.1 I derived a discrete-time map of the population density at one patch when the other patch is treated. This

approach reduced the dimensionality of the system while retaining key properties (Schaffer, 1985). It is related to "peak to peak" dynamics of time series data in which past maxima are used to predict future peaks in time-series oscillations (e.g., Rinaldi et al., 2001). A similar approach is also often used to reduce the dimensionality of a system of three or more differential equations by plotting successive points where the three-dimensional phase dynamics pass through a two-dimensional plane, called a Poincaré section (e.g., Hastings and Powell, 1991; Schaffer, 1985).

I begin with a discrete-time dynamical system that gives the two population densities when either patch is treated. The independent variable is no longer continuous time, but increments with each treatment, $k = 1, 2, 3$, where treatment number k is counted across both patches. One of u_k or v_k will always be $N_{\min}$, because a treatment has always just occurred when $k \to k + 1$.

Given the initial population densities in the two patches, I rearranged the solution to equation 2.1 to give the time until population u reaches the treatment threshold, which I denote T_u (Appendix 2.A). Similarly, I obtained the time until population v reaches the treatment threshold, T_v. I then calculated $\mathbf{T} = T_u - T_v$, where $\mathbf{T} < 0$ indicates that the treatment of patch one happens next, and $\mathbf{T} > 0$ indicates that the treatment of patch two happens next. The solution for the parasite abundances after the next treatment $k + 1$ is

$$\begin{bmatrix} u \\ v \end{bmatrix}_{k+1} = \underbrace{\Big(1 - H(\mathbf{T})\Big) \begin{bmatrix} N_{\min} \\ f_v\Big(T_u, u_k, v_k\Big) \end{bmatrix}}_{u \text{ is treated}} + \underbrace{H(\mathbf{T}) \begin{bmatrix} f_u\Big(T_v, u_k, v_k\Big) \\ N_{\min} \end{bmatrix}}_{v \text{ is treated}}, \quad (2.2)$$

where $H(\mathbf{T})$ is the Heaviside step function that equals zero when $\mathbf{T} < 0$ and one otherwise.

I used this dynamical system to construct a return map that takes the popula-

tion density in patch one when patch two is treated and returns the population density in patch one the next time patch two is treated. I first considered the scenario where patch one is not treated in between consecutive treatments of patch two. I denote the time to the next treatment of patch two as T_{v0}. In this case, the resulting population density in patch one at the next treatment of patch two is

$$\phi(u) = f_u\big(T_{v0},\ u,\ N_{\min}\big), \tag{2.3}$$

where f_u is the solutions to equation 2.1, given in Appendix 2.A. Next, I considered the case where patch one is treated once in between treatments of patch two. This leads to a return map of the form,

$$\phi(u) = f_u\big(T_{v1},\ N_{\min},\ f_v(T_{u0}, u, N_{\min})\big), \tag{2.4}$$

where T_{u0} is the time from the initial treatment of patch two to the treatment of patch one and T_{v1} is the subsequent time from the treatment of patch one to the next treatment of patch two. These two cases can be combined into a single equation as,

$$\begin{aligned}\phi(u) = &\underbrace{H(\mathbf{T}_0)\ f_u\big(T_{v0},\ u,\ N_{\min}\big)}_{u \text{ not treated}} \\ &+ \underbrace{H(\mathbf{T}_1)\ [1 - H(\mathbf{T}_0)]\ f_u\big(T_{v1},\ N_{\min},\ f_v(T_{u0}, u, N_{\min})\big)}_{u \text{ treated once}}.\end{aligned} \tag{2.5}$$

Following this pattern, I arrived at the equation that includes the possibility for patch one being treated twice in between treatments of patch two,

$$\phi(u) = \underbrace{H(\mathbf{T}_0)\ f_u\big(T_{v0},\ u,\ N_{\min}\big)}_{u \text{ not treated}}$$

$$+ \underbrace{H(\mathbf{T}_1)\ [1 - H(\mathbf{T}_0)]\ f_u\big(T_{v1},\ N_{\min},\ f_v(T_{u0}, u, N_{\min})\big)}_{u \text{ treated once}} \tag{2.6}$$

$$+ \underbrace{H(\mathbf{T}_2)\ [1 - H(\mathbf{T}_1)]\ f_u\big(T_{v2},\ N_{\min},\ f_v(T_{u1}, N_{\min}, f_v(T_{u0}, u, N_{\min}))\big)}_{u \text{ treated twice}}.$$

By induction, the general equation for the return map is.

$$\phi(u) = \left[\sum_{m=0}^{\infty} H(\mathbf{T}_m) \prod_{n=0}^{m-1} [1 - H(\mathbf{T}_n)]\right] f_u\Big(T_{vm},\ N_{\min},\ v_{m-1}\Big), \tag{2.7}$$

where treatment on patch one occurs m times before patch two is treated again. The value of m will depend on the relative growth rates of the two populations and their connectivity. Because there is no explicit solution for T_u and T_v (Appendix 2.A), I had to simulate the dynamics using a recursive algorithm to obtain the shape of ϕ (Appendix 2.B).

2.2.3 Parameter sensitivity

The return map given by equation 2.7 can be used to determine the conditions under which the two populations will become synchronized, phase locked, or have more complex dynamics. I had to simulate the map numerically because it could only be written as a recursive algorithm (see Appendix 2.B), making a complete description of the dynamics under all parameter combinations impossible. I therefore constrained my investigation to four scenarios that may represent realistic parameter changes with application to sea louse populations

Table 2.1: Summary of scenarios for how changing connectivity affects dynamics.

Scenario	Growth rates			
	u internal	v internal	from u to v	from v to u
	r_{uu}	r_{vv}	r_{vu}	r_{uv}
A	1.0	1.0	$0.01 \rightarrow 1$	$0.01 \rightarrow 1$
B	1.0	1.0	$0.01 \rightarrow 1$	0.01
C	1.0	0.5	$0.01 \rightarrow 1$	$0.01 \rightarrow 1$
D	1.0	0.5	$0.01 \rightarrow 1$	0.01

in networks of salmon farms (table 2.1).

First, I considered a scenario where the internal growth rates are constant and equal at $r_{uu} = r_{vv} = 1.0$ and connectivity increased from 0.01 to 1.0 in increments of 0.01 ($r_{uv} = r_{vu} = r_{ij}$, scenario A). This scenario could represent two salmon farms being brought closer together, increasing exchange of parasites between them. Second, I considered increasing r_{vu} from 0.01 to 1.0, but connectivity in the other direction constant at $r_{uv} = 0.01$ (scenario B). This scenario could represent an increase in the advection of larvae from one farm to another. The third scenario had connectivity equal and increasing as in scenario A, but patch one had twice the internal growth rate as patch two ($r_{uu} = 1.0, r_{vv} = 0.5$, scenario C). Similarly, in scenario D, patch one had twice the internal growth rate as patch two but r_{vu} increasing from 0 to 1. Different internal growth rates could represent different host population densities or environmental conditions affecting growth on the two farms.

In each scenario, for each value of the control parameter (i.e., r_{vu} in scenarios A-D and r_{vu} and r_{uv} in scenarios A & C; table 2.1), I simulated the return map over 2000 iterations starting at $u_0 = 2.7$. I constructed a bifurcation diagram by plotting the values of $\phi(u)$ for the last 500 iterations, over the value of the control parameter. I present the results for $u_0 = 2.7$, but I examined the bifurcation diagrams starting from several values of u_0 and found that the long-

term dynamics were not dependent on the initial conditions. I also calculated the time between treatments for each patch over the last 500 iterations, and report the inverse of the mean time between treatments as the frequency of treatments for each value of the control parameter.

2.2.4 Testing for chaos

Under certain parameter values, the numerically-calculated return map given by equation 2.7 had a discontinuity at the point where patch one was treated m times or $m+1$ times, depending on the population density u at the first treatment of patch two (see Results). This discontinuity resulted in cyclic behaviour that was difficult to classify by numerical simulations as periodic or chaotic (Galvanetto, 2000). To determine if the resulting population dynamics were chaotic, I numerically calculated the Lyapunov exponent of the return map (Hastings et al., 1993). The Lyapunov exponent is the rate of divergence between two trajectories that are initially close, $\epsilon_n = \epsilon_0\, e^{\lambda n}$ where $\epsilon_0 << 1$ and ϵ_n is the difference between two trajectories after n iterations (Earnshaw, 1993). Positive exponents indicate that two trajectories will diverge and therefore the dynamics are sensitive to the initial condition, characteristic of chaos (Hastings et al., 1993; Sprott, 2003).

The algorithm I used to calculate the Lyapunov exponent is given in Sprott (2003, p. 116-117). Briefly, I began with two trajectories: a fiducial trajectory started at u_0 and a perturbed trajectory started at $u_0 + \epsilon_0$. I calculated the difference between these trajectories over 10 000 iterations of the return map, with the difference after the n^{th} iteration denoted ϵ_n. The Lypanuov exponent was then calculated as

$$\lambda = \sum_{n=1}^{1000} \log\left(\frac{|\epsilon_n|}{\epsilon_0}\right). \tag{2.8}$$

For discontinuous return maps such as the one here, the above numerical calculation of the Lyapunov exponent is not valid if the fiducial and perturbed trajectories project onto different pieces of the return map (Galvanetto, 2000). To avoid this problem, I chose a small initial difference between the trajectories of $\epsilon_0 = 10^{-8}$. At every iteration n, I readjusted the two trajectories bringing them back together along the line of separation such that the difference between them was ϵ_0, with the sign of the difference equal to the sign of ϵ_{n-1} (Sprott, 2003). This correction made it very unlikely that the two trajectories would project onto different pieces of the return map, as the difference between them remained relatively small. I also checked that $\epsilon_n < 1$, as larger values of ϵ_n would suggest that the two trajectories had projected on to different pieces of the return map.

In the numerical calculation, the value of the Lyapunov exponent may depend on the choice of u_0 (Earnshaw, 1993), so I repeated the calculation of equation 2.8 for three randomly-chosen values between $N_{\min}$ and $N_{\max}$ of $u_{0,j} =$ 1.187465, 1.995080 and 1.328498. For each starting value, I iterated the map 200 times, and then used the subsequent 10 000 iterations in the calculation of λ_j (Sprott, 2003). I report the mean value of λ_j for each value of connectivity described in section 2.3.

2.2.5 Stochasticity

In reality, there may be considerable environmental stochasticity influencing the growth of populations, as is the case for sea louse populations on salmon farms (Aldrin et al., 2013; Rogers et al., 2013). I added a small amount of stochasticity to the return map and evaluated its influence on the long-term dynamics for parameters that corresponded to a Lyapunov exponent close to zero versus those that produced a Lyapunov exponent that was significantly

negative. Stochasticity was added to the return map by multiplying $\phi(u)$ at each iteration by e^z, where $z \sim \text{Normal}(\mu = -s^2/2,\ \sigma^2 = s^2)$. The standard deviation in the stochastic term was set at $s = 10^{-2}$, and the mean of the normal distribution was adjusted so that the expected value of e^z was one. I examined 200 iterations of the return map for two trajectories: one fiducial trajectory starting at $u_0 = 2.7$ and a second perturbed trajectory initially separated by a small distance $\epsilon_0 = 10^{-8}$ from the fiducial trajectory. I compared the difference between these trajectories over increasing iterations, with and without stochasticity in the model.

2.3 Results

2.3.1 Simulations of simple model

Simulations of the model predicted that for two isolated populations (i.e., $r_{ij} = 0\ \forall\ i \neq j$), each population will oscillate with treatments occurring at regular intervals. The frequency of treatments was dictated by the internal population growth rate r_{ii}, with higher growth rates resulting in more rapid resurgence of the population after treatment and therefore less time until the treatment threshold was reached again.

When I included dispersal by coupling the two populations, the results were less trivial. Simulations displayed a range of behaviour from alternating treatments (i.e., phase locking; figure 2.1a), synchrony between the populations (figure 2.1b), or seemingly chaotic dynamics (figure 2.1c; table 2.2). To better understand this complex behaviour, I considered a one-dimensional discrete-time return map describing the change in the population in patch one in between treatments of patch two.

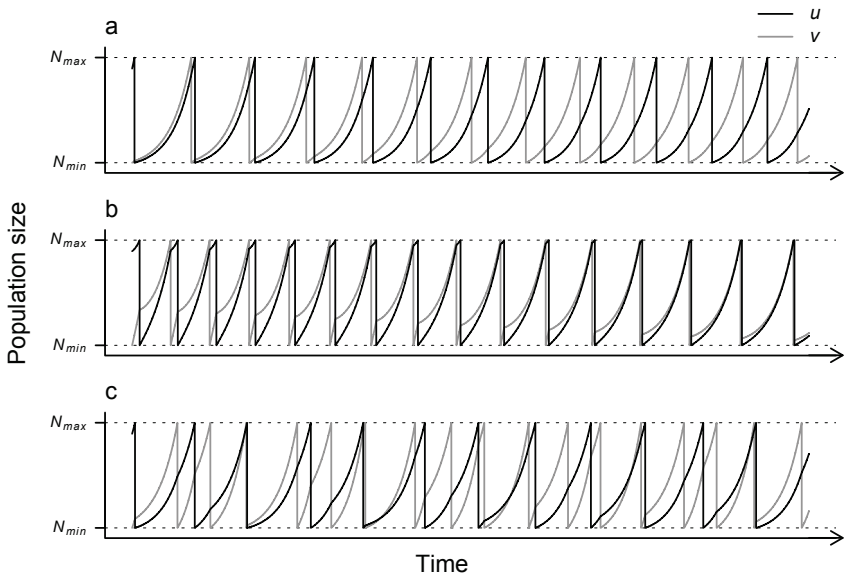

Figure 2.1: Three types of behaviour observed were observed for connected populations subject to control: (a) alternating treatments of patches for $\mathbf{r} = \begin{pmatrix} 1.0 & 0.1 \\ 0.1 & 1.0 \end{pmatrix}$, (b) synchrony in the population dynamics between patches for $\mathbf{r} = \begin{pmatrix} 0.1 & 1.0 \\ 1.0 & 0.1 \end{pmatrix}$ and (c) apparently chaotic dynamics where the treatment timing was unpredictable for $\mathbf{r} = \begin{pmatrix} 0.8 & 0.2 \\ 0.6 & 0.8 \end{pmatrix}$. Initial conditions were $u_0 = 2.7$ (black line) and $v_0 = N_{\min}$ (grey line). The upper and lower horizontal dashed lines indicate the treatment threshold and abundance of parasite immediately after treatment, respectively.

2.3.2 Discrete-time treatment dynamics

For two populations that have identical growth rates but lower connectivity, the return map had a stable equilibrium in the open interval $(N_{\min}, N_{\max})$ (the exact value depended on the level of connectivity) and unstable equilibria at $N_{\min}$ and at $N_{\max}$. This is termed phase locking because the two populations had the same period but their dynamics were shifted out of phase (Becks and Arndt, 2013). The result was alternating treatments of u and v, with a stable equilibrium for the abundance of parasites in patch one whenever patch two was treated (figure 2.1a & 2.2a). If both populations were treated at the same time, u was exactly at the unstable equilibrium. In this case, the two populations remained synchronized because the period of their oscillations was identical.

If the stable equilibria were at the treatment threshold $N_{\min}$ or $N_{\max}$, then the dynamics of the two populations tended towards synchrony. This was observed when connectivity between the patches was equal and greater than the internal growth rates of the populations (i.e., $r_{ij} = r_{ji} > r_{ii} = r_{jj}$; table 2.2). Synchrony also occurred if the internal growth rates were unequal, but the total growth rates of the two populations were equal (i.e., $r_{uu} + r_{uv} = r_{vv} + r_{vu}$) and one population had a lower growth rate and higher connectivity to the other population. In this case, the population with higher connectivity became entrained by the dynamics of the "source" population.

A third type of behaviour occurred when the total growth rates of the populations were not equal. In this case, the two populations oscillated with different periods. There was a discontinuity in the return map where patch one went from being treated once to twice (or two to three times, depending on the relative growth rates) before patch two was treated (figure 2.2c). This discontinuity resulted in periodic or seemingly chaotic behaviour. Unlike in phase

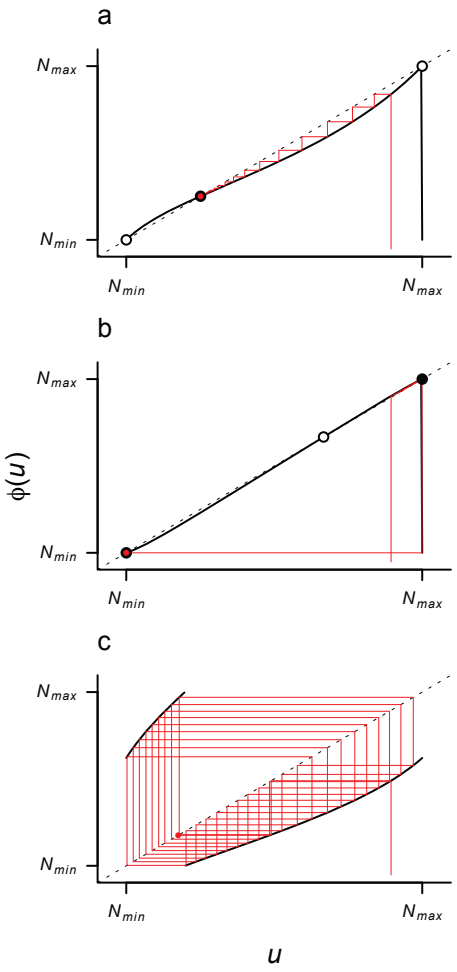

Figure 2.2: Return maps for the population density in patch one the next time patch two was treated (y-axis) over increasing population density in patch one when patch two was treated initially (x-axis). (a) For low connectivity, there was a stable equilibrium in $(N_{\min}, N_{\max})$ (black point) and unstable equilibria at $N_{\min}$ and $N_{\max}$ (white points). (b) When connectivity was higher than internal growth, there was an unstable equilibrium in $(N_{\min}, N_{\max})$ and stable equilibria at $N_{\min}$ and $N_{\max}$, and the two populations become synchronized. (c) For unequal connectivity, patch one may have been treated m or $m+1$ times before patch two was treated, yielding a discontinuity in the return map that resulted in cycles. The growth rates correspond to those in figure 2.1. The red line shows 30 iterations of the return map (i.e., cobwebbing) from $u = 2.7$, ending at the red point.).

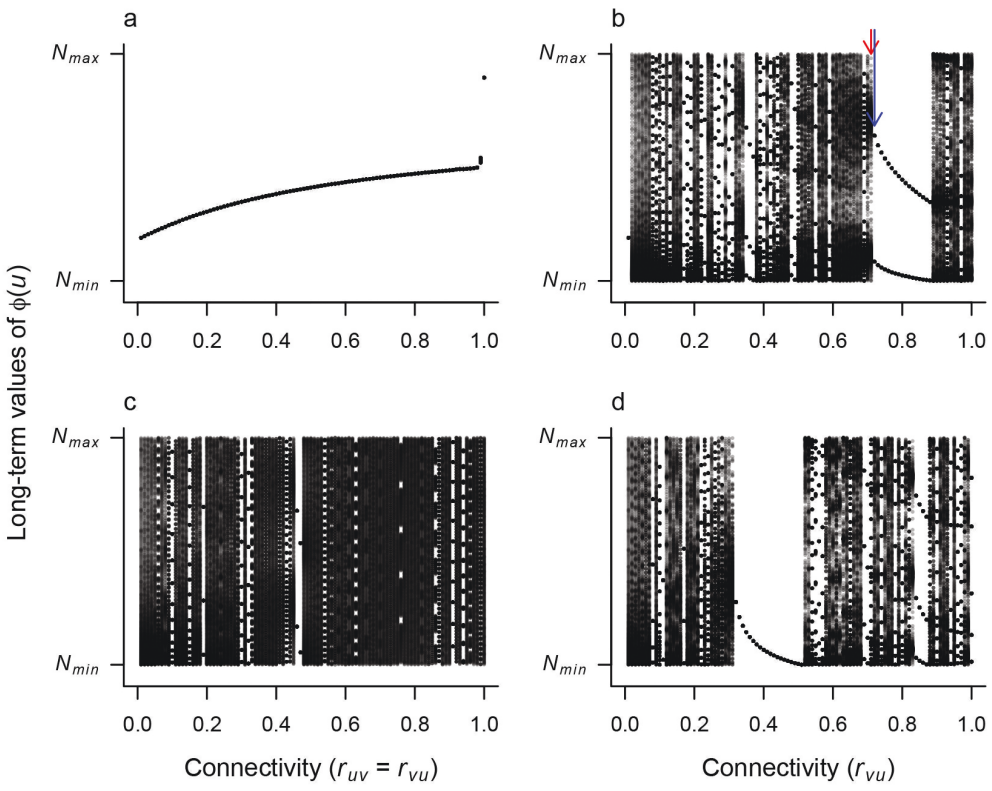

Figure 2.3: Long-term values of $\phi(u)$ under four different scenarios for changing connectivity (scenarios A-D in table 2.1). For each value of connectivity, I plotted the last 500 of 2000 iterations starting at $u_0 = 2.7$ (other starting values produced similar results). Blue and red arrows in (b) indicate the parameter values for stochastic simulations in figure 2.6.

locking or synchrony, the population density in patch one was not the same each time patch two was treated (figure 2.1c).

2.3.3 Parameter sensitivity

Increasing the connectivity between two patches resulted in changes to the long-term values of $\phi(u)$ and the stability (figure 2.3). Some of these changes happened abruptly when the connectivity crossed a threshold (figure 2.3b,d) while others happened gradually (figure 2.3a). When the two populations had equal internal growth rates and equal connectivity, increasing the connectivity

Table 2.2: Summary of parameter values under which different dynamics were observed.

Internal growth rate	**Connectivity**	**Behaviour**	**Figure**
$r_{uu} = r_{vv}$	$(r_{uv} = r_{vu}) \leq (r_{uu} = r_{vv})$	Phase locking	Fig. 2.2a
	$r_{uv} = r_{vu} = 0$	Phase locking	
	$(r_{uv} = r_{vu}) > (r_{uu} = r_{vv})$	Synchrony	Fig. 2.2b
	$r_{uv} \neq r_{vu}$	Cycles	
$r_{uu} \neq r_{vv}$	$r_{uv} = r_{vu}$; incl. $r_{uv} = r_{vu} = 0$	Cycles	Fig. 2.2d
	$(r_{uu} + r_{uv}) = (r_{vu} + r_{vv})$	Synchrony or phase locking	Fig. S10
	Else	Phase locking or cycles	Fig. 2.2c

resulted in an increase in the population density in patch one when patch two was treated until the level of connectivity approached the internal growth rates (scenario A in table 2.1; figure 2.3a). At that point, the return map was simply a 1:1 line and $(\phi(u) = u)\ \forall\, u$.

When connectivity was increased from patch one to patch two only (i.e., scenario B in table 2.1), the return map had a discontinuity because the total growth rate in patch two was higher than in patch one. Therefore, the dynamics underwent cycles, the simplest being a two-point cycles which occurred near $r_{vu} = 0.35$ and $r_{vu} = 0.8$ (figure 2.3b). The interpretation of the two-point cycle is that after the initial treatment of patch two, patch one will be treated once, then after the next treatment of patch two, patch one will be treated twice. This cycle repeats itself exactly so that the treatment order is 2, 1, 2, 1, 1, 2, 1, 2, 1, 1, 2, etc., with patch one having a lower population density at the treatment of patch two if patch one has been treated twice.

When the internal growth rates of the populations were not equal (i.e., scenarios C and D in table 2.1), the dynamics tended to be cyclic (figure 2.3c-

d). However, abrupt changes from cyclic dynamics to stable points occurred as connectivity was increased to the point where the return map touched or crossed the 1:1 line. For example, when r_{vu} neared 0.51, the dynamics tended towards phase locking. When the total growth rates were exactly equal (i.e., $r_{vu} = 0.51$ such that $(r_{uu} + r_{uv}) = (r_{vu} + r_{vv})$), the two populations became synchronized (figure 2.3d; table 2.2).

Increasing the connectivity between the patches did not necessarily result in a smooth increase in the frequency of treatments. For scenario A, the frequency of treatments increased with connectivity until the point where the dynamics tended towards synchrony (i.e., $r_{ij} = r_{ii}$). Once synchronized, the frequency of treatments dropped abruptly. As connectivity was increased further, the frequency of treatments started to increase again (not shown). The highest frequency of treatments occurred when connectivity was just below the level required to synchronize the populations and the total growth rates were balanced between the patches (figure 2.4a). This scenario resulted in phase locking of the return map, where that the dynamics of the two populations were exactly out of phase. In this case, the growth rate due to dispersal from the adjacent patch was maximized after treatment resulting in rapid resurgence of the populations after treatment (figure 2.1a).

In scenario D, the frequency of treatments on patch two plateaued with increasing connectivity (figure 2.4d) at the point where the two populations became phase locked (figure 2.3d). This plateau was also observed in scenario B (figure 2.4b), where the dynamics changed from a many point to a two point cycle (figure 2.3b). In these regions, phase locking or exactly periodic dynamics persist over a range of connectivities, and thus the frequency of treatments does not change.

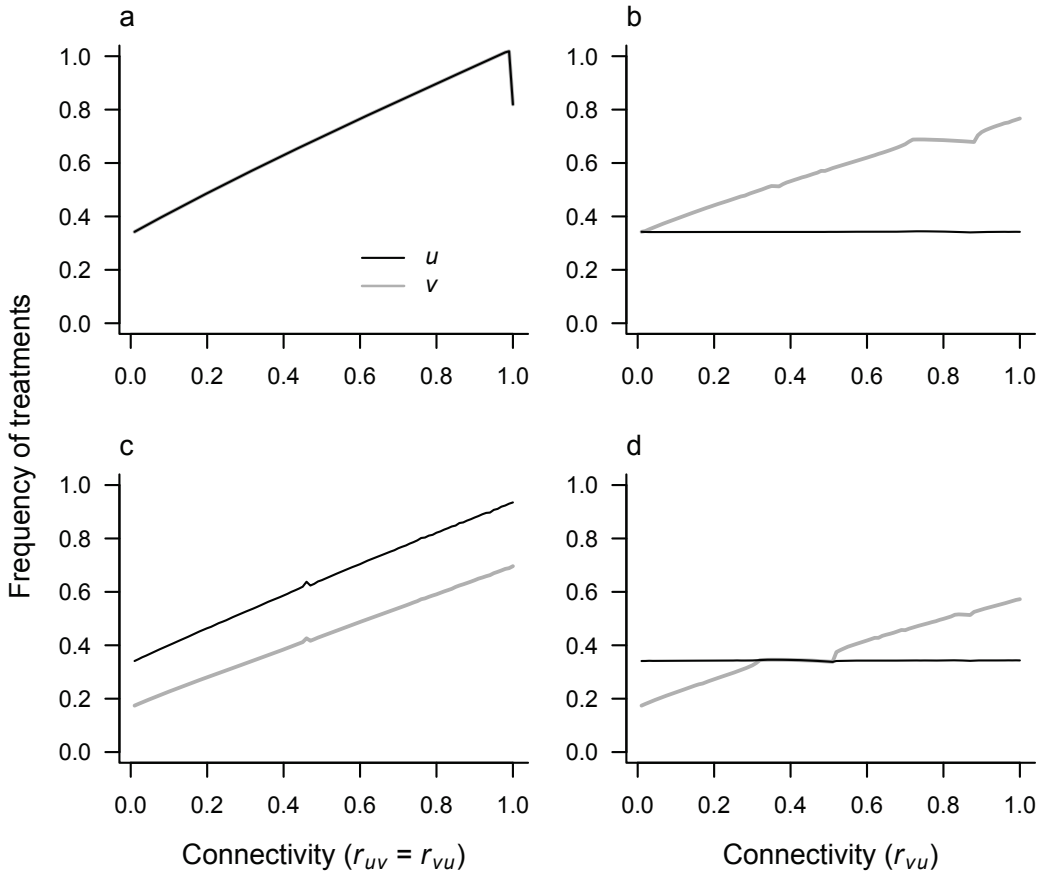

Figure 2.4: The frequency of treatments on patch one (black line) and patch two (grey line) under four different scenarios of increasing connectivity (table 2.1). The frequency of treatments was calculated as the inverse of the mean time between successive treatments over 500 iterations (see Section 2.3).

2.3.4 Testing for chaos

The time series of population density appeared chaotic when the period of the population cycles in the two patches was different (figure 2.1c). The bifurcation diagrams showed large regions of parameter space that had seemingly chaotic dynamics (figure 2.3b-d). However, a lack of pattern in the time series was not a good indicator of chaos (Hastings et al., 1993). The Lyapunov exponent was not greater than zero in any of the scenarios (figure 2.5), indicating the dynamics were not chaotic but were quasiperiodic. Quasiperiodic cycles are differentiated from exactly periodic cycles by cobwebbing the return map; over several treatments on patch two, $\phi(u)$ returned to the original branch of the return map very near to the starting point but not exactly at the starting point, such that the dynamics were shifted slightly (e.g., figure 2.3b at $r_{vu} = 0.65$ and figure 2.6d). For exactly periodic cycles, after iterating the return map a finite number of times, I returned to the exact value at which I started (e.g., figure 2.3b at $r_{vu} = 0.80$ and figure 2.6b).

For some values of r_{ij}, the Lyapunov exponent was negative infinity for all three starting values I tried. This indicated that the fiducial and perturbed trajectories converged to the precision of the numerical calculations such that $\epsilon_n = 0$ after iterating the return map (figure 2.5, red arrows).

2.3.5 Stochasticity

Small amounts of stochasticity added to the return map tended to shift quasiperiodic dynamics towards chaos. However, when the dynamics were exactly periodic, the stochasticity was damped such that the fiducial trajectory and the perturbed trajectory remained close over 200 iterations of the return map (figure 2.6a). A small change in r_{vu} from 0.72 to 0.71 in scenario C caused

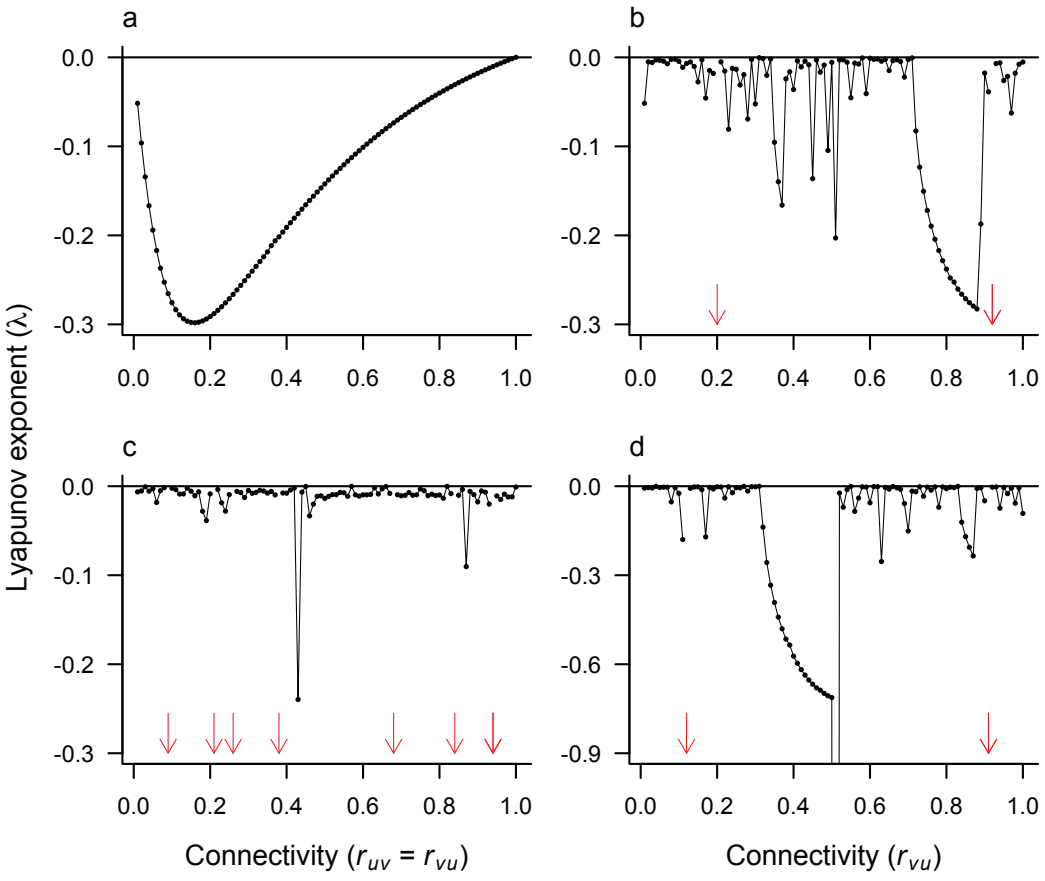

Figure 2.5: The Lyapunov exponents λ (black points) for scenarios A-D (a-d) over the control parameter (table 2.1). Red arrows indicate values of r_{ij} for which the Lyapunov exponent was $-\infty$ for all three starting values.

a transition from periodic to quasiperiodic dynamics (figure 2.6b,d). In the quasiperiodic case, the two trajectories drifted apart as the stochasticity accumulated (figure 2.6c). For scenario D, when r_{vu} was increased from 0.31 to 0.32, the deterministic dynamics went from quasiperiodic to phase locking (figure 2.3d). In this case, as in scenario C, stochasticity caused the trajectories to diverge for $r_{vu} = 0.31$ corresponding to the quasiperiodic dynamics, but stochasticity was damped when the deterministic dynamics exhibited phase locking. This shows that small amounts of stochasticity can accumulate when dynamics are not stable or exactly periodic, and result in sensitivity to initial conditions that is characteristic of chaotic dynamics.

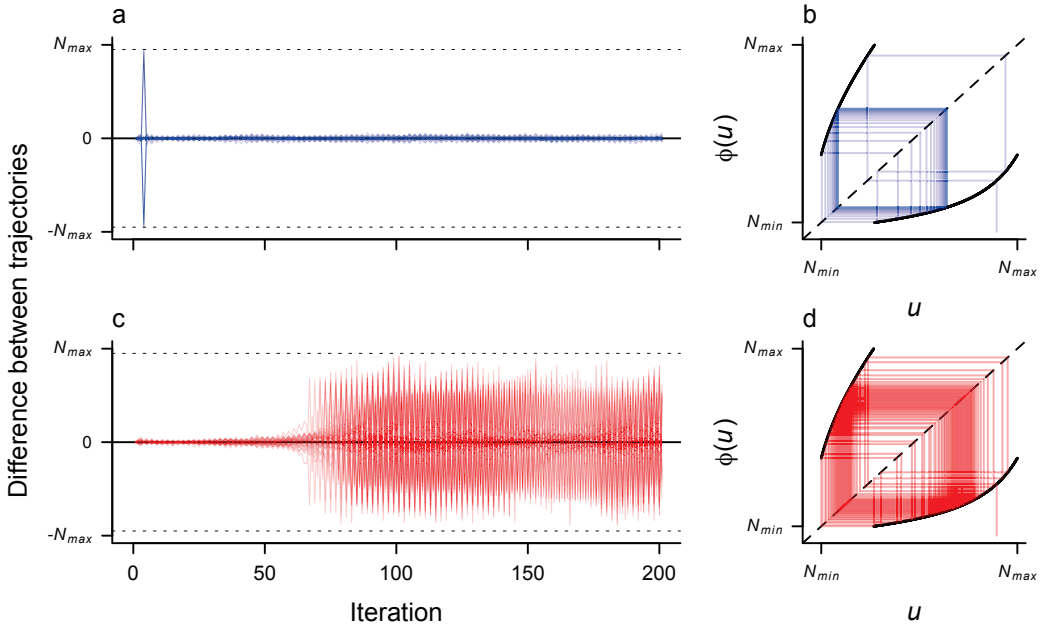

Figure 2.6: The effect of stochasticity differed with small changes in parameters. The difference between two trajectories initially separated by ϵ_0 remained small for parameters under which the deterministic model showed exactly periodic dynamics (a), but increased for parameters under which the deteministic model showed quasiperiodic dynamics (c). The corresponding deterministic return maps of the fiducial trajectory for scenario B with $r_{vu} = 0.72$ (b) and $r_{vu} = 0.71$ (d) (see figure 2.3b).

2.4 Discussion

The current magnitude and extent of human-natural couplings is unprecedented and there is an urgent need to better understand the consequences of accelerating human impacts on natural ecosystems and the services that they provide (Millennium Ecosystem Assessment, 2005). Unfortunately, social and ecological systems are often studied in isolation and we fail to consider the reciprocal interactions between human and natural systems that can lead to surprisingly complex dynamics (Liu et al., 2007a). My model of parasite populations was based on simple exponential growth of two connected parasite populations. The addition of human intervention representing the application of chemotherapeutants when parasite abundance reached a threshold resulted in surprisingly complex dynamics, from synchrony to chaos. Unexpected behaviour such as this can result in policies that are well-meaning but have unintended and perverse consequences for the health of ecosystems.

2.4.1 Implications for sea lice management

As global fisheries decline, aquaculture has seen rapid growth to meet the global demand for seafood (FAO, 2014). A healthy environment in which to grow fish is an ecosystem service that open-net pen aquaculture relies upon. Many approaches have been taken to minimize epizootics of sea lice and other pathogens for the health of coastal ecosystems, including biomass restrictions meant to limit host density, strategic siting of farms, the use of cleaner-fish that prey on sea lice, and the application of chemotherapeutants (Brooks, 2009; Costello, 2004; Rae, 2002). My simple model for coupled parasite populations subject to control revealed one instance where a conservation policy intended to improve the health of coastal ecosystems may actually hinder the

control of parasites on salmon farms. In Pacific Canada, salmon farms must treat with chemotherapeutants when sea louse populations exceed three lice per fish; a guideline that was meant to protect juvenile wild salmon from sea louse infestations during the vulnerable period of their juvenile migration (British Columbia Ministry of Agriculture and Lands, 2005; Brooks, 2009). However, as my model showed, strict threshold control of parasites according to this policy may lead to asynchronous or even chaotic dynamics on adjacent farms that are difficult to predict and control. As a result, the threshold treatment policy in British Columbia may actually harm wild salmon via two mechanisms. First, unpredictable dynamics make it difficult to plan for the wild juvenile salmon migration, which could mean high sea louse abundances on salmon farms along the migration route, transmission to juvenile salmon (Krkošek et al., 2006a; Marty et al., 2010) and adverse impacts on wild salmon populations (Krkošek et al., 2011b; Peacock et al., 2013). Second, uncoordinated treatments allow sea lice that are resistant to treatment to disperse and find mates on nearby, untreated farms, thus facilitating the evolution of sea louse resistance to current chemotherapeutants (Aaen et al., 2015). Resistance to chemotherapeutants is a major threat to both the aquaculture industry and sympatric wild salmon (Igboeli et al., 2014).

Coordinated efforts to synchronize the pathogen dynamics among farms may reduce reliance on chemotherapeutants. My results suggest that many small, weakly connected farms may be the worst-case scenario in terms of minimizing sea louse populations. A more favourable option might be fewer farms with more hosts in each farm. In that case, although the internal growth rate of parasites on a farm will likely be higher due to a higher number of hosts, low connectivity will make treatments more effective. This is the same conclusion reached by Salama and Murray (Salama and Murray, 2011), who used a susceptible-exposed-infected-recovered (SEIR) model, coupled with a

hydrodynamic model, to investigate how farm size and separation distance influenced transmission of infectious waterborne pathogens. For populations that are weakly coupled, but have similar internal growth rates (e.g., have a similar number of hosts and are exposed to similar environmental conditions), synchrony can be induced by either treating populations at the same time or coordinating stocking and harvest among adjacent farms. Such strategies may reduce the potential for the rescue effect in louse populations on adjacent farms and therefore lower the frequency of treatments, but require coordinated effort among multiple stakeholders (e.g., different levels of government and industry). Pest management plans that require this kind of cooperation have been recommended (e.g., Brooks, 2009; Costello, 2004; Peacock et al., 2013), but are still not implemented in many areas including Pacific Canada.

2.4.2 Model limitations

My simple model did not consider exogenous forces on the population dynamics of sea louse parasites. Such forces are likely due to the effect of temperature and salinity on settlement success (Bricknell et al., 2006), developmental rates (Groner et al., 2014; Stien et al., 2005) and survival (Johnson and Albright, 1991a). Environmental conditions have been proposed to result in synchrony of local population dynamics over wide geographic scales (i.e., Moran effects; Moran, 1953). Indeed, such an effect has been shown in a variety of systems (e.g., Cheal et al., 2007; Grenfell et al., 1998; Koenig and Knops, 2013). Sea louse populations on farmed salmon show annual cycles (Marty et al., 2010) that may be driven, in part, by changes in salinity and/or temperature (Johnson and Albright, 1991b). The relative contributions of dispersal versus environment in driving synchrony of local populations is an ongoing question in ecology (Koenig, 1999; Lande et al., 1999), and sea lice in networks of salmon

farms may provide an ideal model system due to the extensive monitoring of louse populations and environmental conditions on salmon farms. These data have been used in statistical analyses aimed at management applications (e.g., Jansen et al., 2012; Revie et al., 2003; Rogers et al., 2013), but could also be useful in answering questions of general interest in ecology.

2.4.3 Dynamics of coupled populations

There has been considerable theoretical interest in how dispersal affects the dynamics of coupled populations (e.g., Dey et al., 2014, 2015; Franco and Ruis-Herrera, 2015; Goldwyn and Hastings, 2011; Hastings et al., 1993; Kendall and Fox, 1998). My analysis expands on previous theoretical work in several ways. First, I considered control of populations when a threshold abundance was reached. Previous work has considered density dependence as part of the intrinsic dynamics of local populations (e.g., the Ricker model, Dey et al., 2015; Hastings et al., 1993) or periodic interventions such as feeding and harvest (e.g., Chau, 2000). Treatment thresholds, which are a nonlinear reciprocal interaction between parasite populations and human intervention, had not previously been explored to my knowledge. Second, I analyzed a continuous-time population model that may be more representative for some species, but were able to simplify my analysis by considering a discrete time return map for the population density in patch one at the time of treatment on patch two. This dynamical systems approach has gained attention recently in the context of peak to peak dynamics (Rinaldi et al., 2001) and statistical methods for analyzing time series data (Sugihara et al., 2012), but also has broader applications for simplifying analyses of continuous-time models for interacting populations (Schaffer, 1985). Finally, I varied both the internal growth rates and connectivities in my populations to explore scenarios where growth rates

of the two populations differed and connectivity was not necessarily reciprocal. Many studies of coupled populations only consider equal connectivity (although see Dey et al., 2014; Franco and Ruis-Herrera, 2015).

Increasing dispersal between two populations subject to control was expected to increase the frequency of treatments, but the simple model I developed displayed much more complex dynamics. My results were consistent with other population models that show high dispersal leads to synchrony of populations while lower levels of dispersal lead to out-of-phase dynamics (Ben Zion et al., 2010; Dey et al., 2014, 2015). If the two populations had different periods due to unequal growth rates, the dynamics underwent periodic or quasiperiodic cycles. When dynamics were exactly periodic, added stochasticity was damped such that the difference between nearby trajectories remained small. Hastings (Hastings, 1993) analyzed a coupled discrete logistic model and also found that the addition of stochasticity resulted in chaos for parameter values corresponding to a four-point cycle in the deterministic model, but stable population densities for parameter values corresponding to a two-point cycle in the deterministic model. This result highlights the fine line between predictable deterministic dynamics and chaos (Hassell et al., 1991; Hastings, 1993).

2.4.4 Conclusion

The complexity of coupled human and natural systems has been gaining attention as we recognize and attempt to understand our impact on natural ecosystems. For aquaculture, the interaction between farm management and natural pathogen dynamics, including dispersal among farms, may lead to unpredictable dynamics that undermine our ability to maintain a healthy environment for both farmed and wild salmon. The successful management of disease in coastal ecosystems likely requires cooperation among different com-

panies to synchronize and stabilize pathogen dynamics. This example emphasizes that human-natural couplings cross the boundaries that define reaches of policy and governance, and cooperation among stakeholders at different levels is required to achieve the common goal of healthy and sustainable ecosystems that can support adaptive human populations.

Appendix

2.A Solution to ODE

The solutions to equation 2.1 are:

$$
\begin{aligned}
u(t) &= f_u(t, u_0, v_0) \\
&= c_1 \exp\left[\frac{r_{uu} + r_{vv} + \alpha}{2} t\right] + c_2 \exp\left[\frac{r_{uu} + r_{vv} - \alpha}{2} t\right] \qquad (2.9)
\end{aligned}
$$

$$
\begin{aligned}
v(t) &= f_v(t, u_0, v_0) \\
&= c_1 \left(\frac{r_{vv} - r_{uu} + \alpha}{2r_{uv}}\right) \exp\left[\frac{r_{uu} + r_{vv} + \alpha}{2}\, t\right] \\
&\quad + c_2 \left(\frac{r_{vv} - r_{uu} - \alpha}{2r_{uv}}\right) \exp\left[\frac{r_{uu} + r_{vv} - \alpha}{2}\, t\right], \qquad (2.10)
\end{aligned}
$$

where

$$c_1 = \frac{2r_{uv}v_0 - u_0(r_{vv} - r_{uu} - \alpha)}{2\alpha} \tag{2.11}$$

$$c_2 = \frac{u_0(\alpha + r_{vv} - r_{uu}) - 2r_{uv}v_0}{2\alpha} \tag{2.12}$$

$$\alpha = \sqrt{(r_{uu} - r_{vv})^2 + 4r_{uv}r_{vu}}. \tag{2.13}$$

We can rearrange equations 2.9 - 2.10 to get the time of the next treatment given the growth rates and initial conditions. I denote the time of the next treatment of patch one and patch two as T_u and T_v, respectively. The equations for T_u and T_v are:

$$\begin{aligned} 2\alpha N_{\max} = \exp\left(\frac{r_{uu} + r_{vv}}{2}T_u\right) \Bigg[& \left(\exp\left(\frac{\alpha}{2}T_u\right) - \exp\left(\frac{-\alpha}{2}T_u\right)\right) \\ & \times (2r_{uv}v_0 + u_0(r_{uu} - r_{vv})) \\ & + u_0\ \alpha \left(\exp\left(\frac{\alpha}{2}T_u\right) + \exp\left(\frac{-\alpha}{2}T_u\right)\right)\Bigg] \end{aligned} \tag{2.14}$$

$$\begin{aligned} 4\alpha r_{uv}N_{\max} = \exp\left(\frac{r_{uu} + r_{vv}}{2}T_v\right) \Bigg[& (2r_{uv}v_0\ (r_{vv} - r_{uu}) + 4u_0\ r_{vu}r_{uv}) \\ & \times \left(\exp\left(\frac{\alpha}{2}T_v\right) - \exp\left(\frac{-\alpha}{2}T_v\right)\right) \\ & + 2r_{uv}v_0\alpha \left(\exp\left(\frac{\alpha}{2}T_v\right) + \exp\left(\frac{-\alpha}{2}T_v\right)\right)\Bigg]. \end{aligned} \tag{2.15}$$

In equations 2.14-2.15, T_u and T_v cannot be solved for explicitly, so I used a numerical root finding algorithm (`uniroot` in R (R Development Core Team, 2014)) to determine T_u and T_v.

2.B Algorithm describing return map

Because equations 2.14-2.15 can not be solved for T_u and T_v, model analysis by the return map involved simulating successive treatments until patch two was treated next. The recursive algorithm I applied to calculate the population density in patch one when patch two was treated next is:

```
function u_next(u, v, R)
    evaluate T_u(u, v, R) = Tu          Calculate the time to the
                                        next treatment of u
    evaluate T_v(u, v, R) = Tv          and the time to the next
                                        treat of v.
    if (T_u>T_v) then                   If the time to treatment
                                        of v is less,
        return f_u(T_v, u, v)               return u when v is
                                        treated.
    else                                Otherwise, patch 1 is
                                        treated.
        v_new = f_v(T_u, u v)               Calculate v when u
                                        is treated and
        return u_next(Nmin, v_new, R)       repeat function with
                                        new initial values.
    end if
```

Chapter 3

Cessation of pink salmon decline with control of parasites

3.1 Introduction

The resilience of social-ecological systems depends on their adaptive capacity to respond to human and environmental change (Liu et al., 2007a; Walker et al., 2004). For coastal systems, this may include the adaptive change of aquaculture management in response to disease outbreaks in farm and wild fishes. Farm fish raised in sea cages are vulnerable to native pathogens from wild populations (Saksida, 2006). Wild fish populations are vulnerable to bio-amplification of native pathogens in farming regions (Krkošek et al., 2006a), as well as the introduction of novel pathogens (Gaughan, 2001). Precautionary

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regulatory approaches include protected areas (Bjørn et al., 2011), parasite limits on farm fish (Heuch et al., 2005), and integrated coastal planning (Gudjonsson and Scarnecchia, 2009). However, empirical evaluations of adaptive farm management and the resultant conservation gains have been rare.

Host migration may drive the dynamics of infectious disease in coastal ecosystems that support wild and farm salmon populations Krkošek et al. (2007b, 2009). For example, the large abundance of wild salmon in coastal seas of the north Pacific is seasonally ephemeral, limited to the spring out-migration of juveniles transiting to offshore waters and the summer and autumn return of adult salmon to freshwaters to spawn (Quinn, 2005). Effective disease control may require breaking transmission cycles between wild and farm salmon by timing parasite control strategies relative to migration schedules of wild salmon populations.

Here, I examine the links between adaptive changes in management of sea lice on salmon farms, observed infections on wild juvenile salmon, and wild salmon population dynamics (figure 3.1). In particular, I elucidate connections that have not yet been made between adaptive changes in management and parasites on wild juvenile salmon, and the sea lice observed on wild juvenile salmon and wild salmon population productivity. Drawing on data from a nine-year study of parasitic sea lice (*L. salmonis*) and pink salmon (*O. gorbuscha*) from Pacific Canada, this analysis indicates positive conservation outcomes due to adaptive changes in management of parasites in salmon aquaculture facilities. These results provide an example of how management of sea lice on farm salmon can be improved, with relevance to management of sea lice on farm salmon in Canada, Europe, and other areas of the world where the expansion of aquaculture has been accompanied by environmental concerns of parasite transmission to wild salmonids (e.g., Bjørn et al., 2001; Butler and Watt, 2003; Gargan et al., 2003).

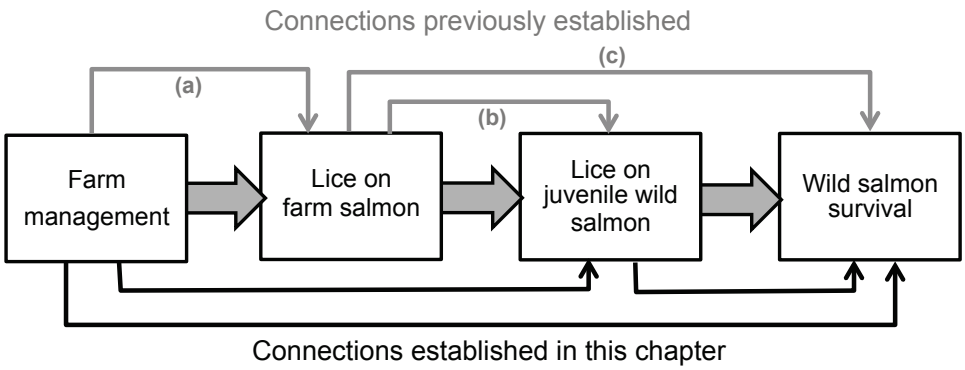

Figure 3.1: This study addresses unanswered questions about the efficacy of sea lice management on salmon farms in reducing wild juvenile salmon epizootics and increasing wild salmon survival (black lines, bottom). Connections previously established (grey lines, top) include (a) the effect of treatments on sea louse abundance on farm salmon (Krkošek et al. 2010, Revie et al. 2003), (b) the transmission of lice from farm to wild salmon (Bjørn et al., 2001; Gargan et al., 2003; Krkošek et al., 2006a; Marty et al., 2010; Morton et al., 2004) and (c) the effect of sea louse abundance on farm salmon on wild salmonid survival (Gargan et al., 2003; Krkošek et al., 2011b).

3.2 Methods

3.2.1 Farm data

The farm data consist of monthly estimates of farm Atlantic salmon (*Salmo salar*) abundance and average number of adult female sea lice (*L. salmonis*) per farm salmon per farm in the Broughton Archipelago (figure 3.2) from 2001 to 2009, reported in Marty et al. (2010). I focused my analysis on farms located on the Knight Inlet - Tribune Channel - Fife Sound (KTF) corridor of the Broughton in order to compare the farm data with field monitoring programs of juvenile salmon that migrate through that corridor (figure 3.2). The total louse population per farm and per region was estimated by multiplying the average number of lice per farm salmon by the number of salmon per farm, and then summing over farms (Marty et al., 2010; Orr, 2007). The data also include records of in-feed parasiticide treatments with emamectin benzoate.

I categorized advance, or "winter" treatments as those that occurred in January through March or October through December, prior to juvenile salmon migrations, which typically occur in March through June (Jones et al., 2007; Krkošek et al., 2006a; Krkošek, 2010b; Morton et al., 2004).

3.2.2 Weekly louse monitoring of wild fish

I assembled data from a long-term monitoring program that sampled juvenile pink salmon for sea lice at weekly intervals during March through June between 2001 and 2009 at three sites in the Broughton Archipelago (figure 3.2). Data collection involved searching nearshore waters ($\sim$ 2-5 m from shore) visually for schools of juvenile salmon in the surface 0.3-2 m, depending on visibility. Once spotted, salmon were collected by dip net (45 cm diameter with 5 mm knotless mesh on a 2.45 pole from a 7.5 m boat) between 2001 and 2003 (Krkošek et al., 2005a; Morton et al., 2004; Morton and Williams, 2003). For 2004 through 2009, juvenile salmon were collected from the same sites using a beach seine net, whose dimensions varied among years ranging from the smallest dimensions of 15.2 m long by 1.8 m deep with 6 mm knotless mesh (2004-2007) to a larger net that was 40 m long by 2.5 m deep with 6 mm knotless mesh (2008-2009).

Upon collection, juvenile salmon were transferred into seawater-filled buckets either directly from the dip net (2001 through 2003) or from the bunt of the purse seine net using smaller dip nets (10 to 15 cm square on a 30 cm handle with 2 mm knotless mesh) during 2004 through 2009. From 2001 through 2004, juvenile salmon were placed individually from the buckets into individual sample bags, then placed on ice and then frozen for subsequent laboratory analysis. Frozen samples were analyzed under a dissecting microscope at 30 $\times$ magnification and species and stages of lice were identified according to

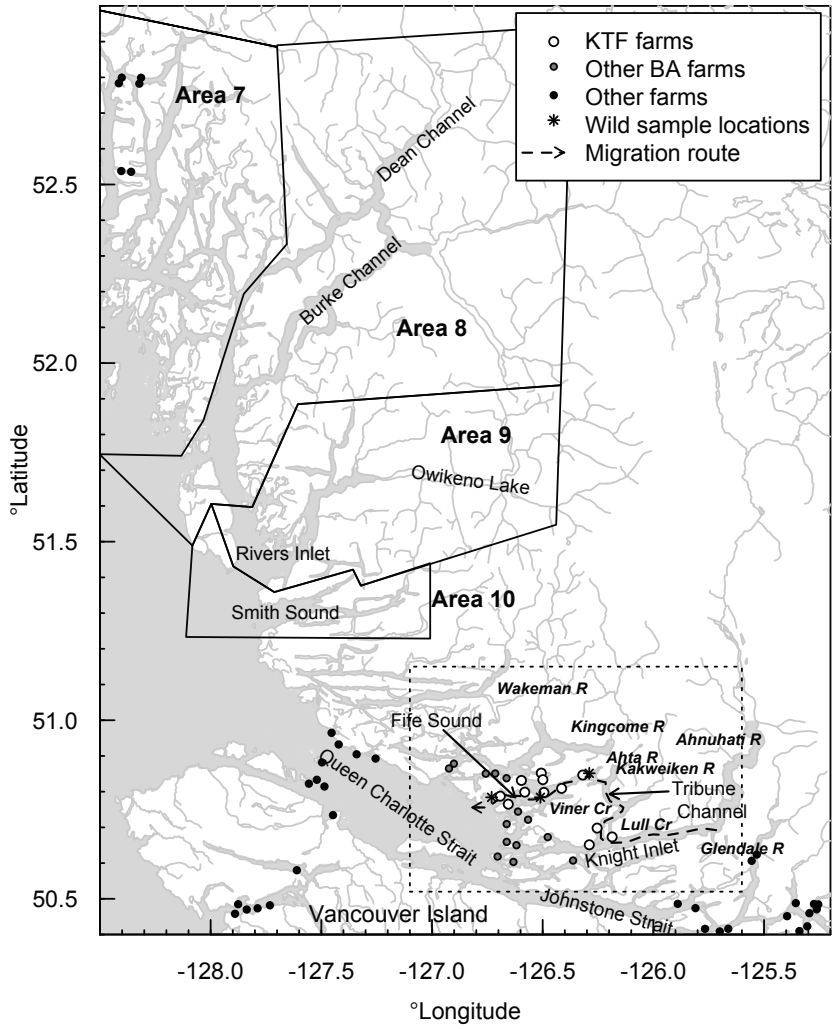

Figure 3.2: Salmon populations on the coast of British Columbia were categorized as unexposed (fishery management areas 7-10) or exposed to salmon farms (area 12, the Broughton Archipelago, labelled rivers in the lower right). The focal study area is the Broughton Archipelago, outlined by the dotted box, and the Knight Inlet - Tribune Channel - Fife Sound (KTF) wild salmon migration corridor (black line). Salmon farm locations are shown by small black circles outside the Broughton Archipelago, while inside the Broughton Archipelago, small grey circles indicate those farms distant from the KTF migration corridor and white circles indicate those farms on the KTF migration corridor. Stars indicate the three weekly sample sites for wild juvenile salmon from 2001-2009.

Johnson and Albright (1991b). From 2005 through 2009, juvenile salmon were analyzed non-lethally on site, using a 16 × magnification hand lens to visually assay individual fish in zip-locked plastic bags (Krkošek et al., 2005b). Pink and chum (*O. keta*) salmon were collected, however only data of lice on pink salmon are presented here. I report the lice per wild juvenile pink salmon as the sum of all attached stages of *L. salmonis*.

3.2.3 Intensive louse surveys of wild fish

To provide a more detailed analysis of changes in sea lice transmission dynamics in relation to management changes, I also assembled data from an intensive sampling program that studied sea lice infections on juvenile salmon as they migrated through the KTF corridor. These data consisted of 100 juvenile pink (*O. gorbuscha*) salmon, collected at 1-3 km intervals along the length of the corridor (figure 3.2). Salmon were caught by beach seine and non-lethally assayed for lice as described above and in Krkošek et al. (2005b). At each sample location in weekly and intensive surveys of wild fish, temperature and salinity were recorded using a thermometer and a salinity refractometer. I contrast average numbers of sea lice on pink salmon from 2009 with data from 2004 that were previously published by Krkošek et al. (2006a).

3.2.4 Salmon spawner-recruit data

I obtained estimates of pink salmon spawner abundance for populations near active salmon farms in the Broughton Archipelago (fishery management area 12) and reference populations not exposed to salmon farms (areas 7-10; figure 3.2) from the Pacific salmon escapement database (Fisheries and Oceans Canada, 2011b). These data contained spawner abundance estimates (with

missing values) for 277 rivers, each with independent even and odd year populations, spanning 60 years from 1950 to 2010. Fisheries and Oceans Canada (DFO) personnel generated the spawner data via analyses of data from stream walks and overhead flights, also conducted by DFO personnel.

To calculate recruitment, I added the estimated abundance of pink salmon caught in fisheries (i.e., catch) to spawner abundance enumerated in rivers (i.e., escapement). I obtained raw catch data or estimates of exploitation rates of pink salmon from DFO stock assessment biologists for each year in each DFO management area that contained rivers in my study area (fishery management areas 7,8,9,10,12; 12 includes the Broughton Archipelago). For areas 7-10, catch was assumed to consist of local populations within the management areas. For these areas, exploitation rates were calculated as $h_{i,t} = C_{a,t}[C_{a,t}+E_{a,t}]^{-1}$, where $C_{a,t}$ is the catch for area a in year t and $E_{a,t}$ is the estimated total escapement for the area, expanded from counts of spawner abundance using the $\mathrm{P_{max}}$ technique (for a description of this technique, see Appendix A of Peacock et al. 2013). For area 12, exploitation rates were provided directly by DFO, and also accounted for fisheries targeting primarily non-local populations who are fished in area 12 on their migration to rivers further south (e.g., the Fraser River). I assumed that returns to each river in a management area experienced the same exploitation rate in a given year. Recruitment, $R_{i,t}$, to river i in year t was calculated as $R_{i,t} = N_{i,t}[1 - h_{i,t}]^{-1}$, where $N_{i,t}$ is the spawner abundance of pink salmon from river i in year t and $h_{i,t}$ is the exploitation rate for river i in year t.

I structured the escapement data into odd and even year populations for each river, which is standard practice for pink salmon due to their two-year lifecycle and intrinsic differences between odd and even year lineages (Dorner et al., 2008; Pyper et al., 2001). I then screened the data, keeping only populations for which there were at least twenty spawner-recruit data pairs. I further kept

only the eight rivers for the Broughton Archipelago region that were used in previous analysis of the area (figure 3.2, Krkošek et al. 2007a; Krkošek and Hilborn 2011). This reduced the dataset to 179 populations of odd or even year lineages of pink salmon, yielding 2385 spawner-recruit pairs distributed over 99 rivers from 1962-2010.

3.2.5 Analysis

Estimates of the mean abundance of *L. salmonis* per wild juvenile pink salmon per year were calculated from the weekly monitoring data via a generalized linear mixed-effects model (GLMM), with fixed effects for year and random effects for sample site and week. The data were highly over-dispersed, and found to be best represented by a zero-inflated negative binomial distribution. This model was fitted using the package `glmmADMB` (Skaug et al., 2012), using the software R (R Development Core Team, 2014).

I investigated several relationships linking the effect of changes in management (e.g., timing of parasiticide treatments) to wild salmon population growth rates (figure 3.1). First, the trends over time in the total number of parasiticide treatments and proportion of those treatments occurring in winter (January-March or October-December, prior to the juvenile salmon outmigration) in the KTF corridor and the Broughton Archipelago were quantified using regression analysis. The yearly estimates of average lice per wild juvenile salmon from the aforementioned GLMM were then related to the total lice on farm salmon, and to the management changes, quantified as the total number of treatments, number of winter treatments, and proportion of total treatments occurring during winter on farms in the Broughton Archipelago. I then analyzed the survival of pink salmon populations in the Broughton Archipelago in relation to the average number of lice per wild juvenile salmon, on the premise that

the latter was related to both farm lice and adaptive changes in management and provides a covariate that more closely captures the actual effect on wild salmon survival. Previous studies have related wild salmon survival to lice on farm salmon (Krkošek et al., 2011b; Marty et al., 2010), which is a less direct approach to determine the population-level effect of sea lice.

For this population-level analysis, I applied a hierarchical Ricker model to the pink salmon spawner-recruit data described above. The data were spatially structured, first by populations exposed to salmon farms (Broughton Archipelago) and a reference regions where populations were unexposed to salmon farms (figure 3.2). The unexposed region was further partitioned into four fishery management areas used for reporting of catch by the DFO. Exposed populations all existed within management area 12.

The model allowed for several levels of synchrony in salmon survival by treating year and management area within year as random and nested random effects, respectively. Density dependent mortality was treated as a fixed factor per population (i.e., different for even and odd year populations within the same river). The growth rate was treated as a fixed factor, and the average lice per wild juvenile salmon (as estimated from the GLMM) was included as a covariate. The full model was:

$$R_{i,t} = N_{i,t-2} \exp\left[(r + \theta_t + \theta_{a,t}) - b_i N_{i,t-2} - cW_{a,t-1} + \epsilon_{i,t}\right], \qquad (3.1)$$

where $R_{i,t}$ is recruitment of population i in year t and $N_{i,t-2}$ is the abundance of spawners of population i in year $t-2$. Here, t is lagged two years to account for the two-year lifecycle of pink salmon. The growth rate, r, was the same for the entire region, but the density dependence parameter, b_i, was different for each population as it relates to the habitat characteristics unique to each river and density-dependent competitive interactions within populations.

To test for an effect of sea louse infestations on survival, I included the average lice per wild juvenile salmon the previous year, $W_{a,t-1}$, as a covariate. I assumed the lice per wild juvenile salmon to be zero for reference regions, as louse abundance on juvenile pink salmon in nearshore waters is extremely low in the absence of salmon farms (Gottesfeld et al., 2009). Any sea lice on juvenile salmon in reference regions are likely originating from returning adult salmon (Gottesfeld et al., 2009), and transmission occurs further offshore and later in the season when juvenile pink salmon are less vulnerable to infection. For return years 2002-2010 in the Broughton Archipelago, $W_{a,t-1}$ was taken as the mean louse abundance per wild juvenile salmon, estimated from the GLMM for 2001-2009. Data describing louse abundances on farm and wild salmon from the onset of salmon farming in the Broughton Archipelago to the first reported infestation in 2001 (Morton and Williams, 2003) were not available, but it is reasonable to assume that sea louse abundances were not epizootic during this period as outbreaks were not reported on salmon farms (Marty et al., 2010) or noticed on wild juvenile salmon (Morton and Williams, 2003). However, to address this uncertainty, I treated $W_{a,t-1}$ as missing data for return years 1991 to 2001. Prior to 1991, I assume $W_{a,t-1} = 0$ for the Broughton Archipelago, as salmon farming production was low (Pearsall, 2008). The strength of the relationship between pink salmon survival and lice on wild juvenile salmon was controlled by the parameter c. The estimated percent mortality of pink salmon due to sea lice on wild juvenile salmon is therefore equal to $1 - \exp(-cW_{a,t-1})$ (Krkošek et al., 2011b).

Environmental stochasticity was represented by spatially coherent variation among all populations (θ_t, a random normal variable for year with mean zero and variance to be estimated), spatially coherent variation for populations within a management area ($\theta_{a,t}$, a random normal variable for areas within years that has a mean of zero and variance to be estimated), and random

annual variation that is independent among populations ($\epsilon_{i,t}$, also a random normal variable for each river in each year that has mean of zero and variance to be estimated). The random effect of area within year is also needed to accommodate the non-independence of survival observations among rivers within a management area in a given year, due to the assumption that rivers within an area experience the same harvest rate. For this analysis, I ignore measurement error associated with the observation of spawners.

As is common in the application of the Ricker model to data, equation 3.1 was fit in its linear form:

$$\ln\left[R_{i,t}/N_{i,t-2}\right] = (r + \theta_t + \theta_{a,t}) - b_i N_{i,t-2} - cW_{a,t-1} + \epsilon_{i,t}, \qquad (3.2)$$

using the lme4 package in R (Bates et al., 2011). Confidence intervals on model parameters were calculated via parametric bootstrapping as described in Krkošek et al. (2007a) and Krkošek and Hilborn (2011).

3.3 Results

The total number of lice on farm salmon has been steadily declining over the last decade, with no corresponding declines in farm salmon production (figure 3.3a, 3.3b). The dynamics of lice on farm salmon in the KTF corridor of the Broughton Archipelago (figure 3.2) are characterized by large fluctuations in abundance that have a clear annual cycle (figure 3.3a). Louse abundances on farm fish increase during winter and sometimes spring months until parasiticide treatments appear to reduce sea lice to lower levels during spring and summer months. During autumn months, the cycle of louse population growth, treatment, and decline appears to begin anew. Farms were largely compliant with the regulatory guidelines of treatment (or harvest) when the

abundance of motile stage lice exceeded three lice per farm fish during the wild juvenile salmon out-migration season (March-June, table 3.3). During the remaining months of the year, the abundance of lice that triggered treatment declined from 2004 onwards (table 3.3). The total number of treatments in the KTF corridor and the Broughton Archipelago have not increased significantly over time (linear regression; KTF: $df = 7$, $F_{1,7} = 0.374$, $p = 0.611$; BA: $df = 7$, $F_{1,7} = 0.806$, $p = 0.399$). However, the proportion of these treatments occurring in winter, preceding the juvenile salmon out-migration, has increased over time in both the KTF corridor (logistic regression; $df = 7$, $\exp(\hat{\beta}) = 1.198$, $z = 1.807$, $p = 0.071$) and across the Broughton Archipelago ($df = 7$, $\exp(\hat{\beta}) = 1.123$, $z = 1.790$, $p = 0.073$; table 3.2).

The dataset on weekly monitoring of lice on juvenile salmon comprised 19 113 lice on 7907 pink salmon sampled over nine years (table 3.3). There were substantial inter-annual variations in louse abundance on wild juvenile salmon as well as farm salmon (table 3.3 and figure 3.3b, 3.3c). The period 2001-2005 was characterized by relatively high sea louse abundance on wild juvenile pink salmon, with the exception of 2003 when the fallowing management intervention was implemented by provincial regulators and farming companies (Morton et al., 2005). The period of high louse abundance on wild juvenile salmon corresponded to years when louse abundance on farm salmon was also high during the outmigration season (figure 3.3b, 3.3c). The fallow year (2003), showed a declining trend in louse abundance on farm fish in the early part of the migration season, which was not associated with parasiticide treatment, but rather a management intervention implemented by provincial regulators that fallowed most of the farms along the KTF corridor.

At a coarse scale, inter-annual patterns in the average abundance of lice on wild juvenile pink salmon in the KTF corridor are related to the total annual number of gravid lice on farm fish in the corridor during the outmigration

Table 3.1: Average abundance of motile stage *L. salmonis* per farm fish for the month prior to treatment with parasiticide in the Knight Inlet - Tribune Channel - Fife Sound (KTF) migration corridor and the entire Broughton Archipelago (see figure 3.2) in 2001-2009.

Area	Year	Sea lice* per farmed salmon			
		Mar-Jun		Jan-Feb; Jul-Dec	
		Mean	SE	Mean	SE
KTF corridor	2001†	3.61	0.62	16.74	3.49
	2002†	5.55	0.12		
	2003‡			3.09	1.59
	2004	2.85	0.86	6.47	
	2005	2.55	0.90	3.10	0.94
	2006	4.52	0.52	2.41	0.59
	2007			2.38	
	2008	1.92	0.34	1.63	0.44
	2009			1.04	0.52
BA	2001†	3.39	0.40	16.74	3.02
	2002†	7.68	1.01		
	2003‡	12.28	2.20	4.04	1.04
	2004	6.44	1.18	12.25	2.62
	2005	2.66	0.44	5.36	1.18
	2006	2.63	0.58	4.78	0.97
	2007			4.98	1.96
	2008	1.92	0.25	2.13	0.42
	2009	2.23		1.86	0.51

*Motile stage *L. salmonis*.

†Years 2001 and 2002 did not yet have systematic monitoring programs implemented and there are numerous instances of missing data (Marty et al., 2010).

‡The year 2003 corresponds to the fallowing intervention.

Table 3.2: Number of treatments with SLICE® on farms along the Knight Inlet - Tribune Channel - Fife Sound (KTF) migration corridor and the entire Broughton Archipelago (BA; see figure 3.2) for 2001-2009. Winter treatments are those occurring in January-March or October-December, in advance of a juvenile salmon outmigration.

Area	Year	Number of treatments		Proportion
		Total	Winter	winter
KTF corridor	2001*	6	2	0.33
	2002†	3	0	0.00
	2003†	2	2	1.00
	2004	7	1	0.14
	2005	6	3	0.50
	2006	9	8	0.89
	2007	1	1	1.00
	2008	6	5	0.83
	2009	7	7	1.00
BA	2001*	8	2	0.25
	2002†	7	3	0.43
	2003†	7	7	1.00
	2004	19	8	0.42
	2005	17	11	0.65
	2006	16	12	0.75
	2007	4	4	1.00
	2008	11	10	0.91
	2009	17	16	0.94

*Years 2001 and 2002 did not yet have systematic monitoring programs implemented and there are numerous instances of missing data (Marty et al., 2010).

†The year 2003 corresponds to the fallowing intervention.

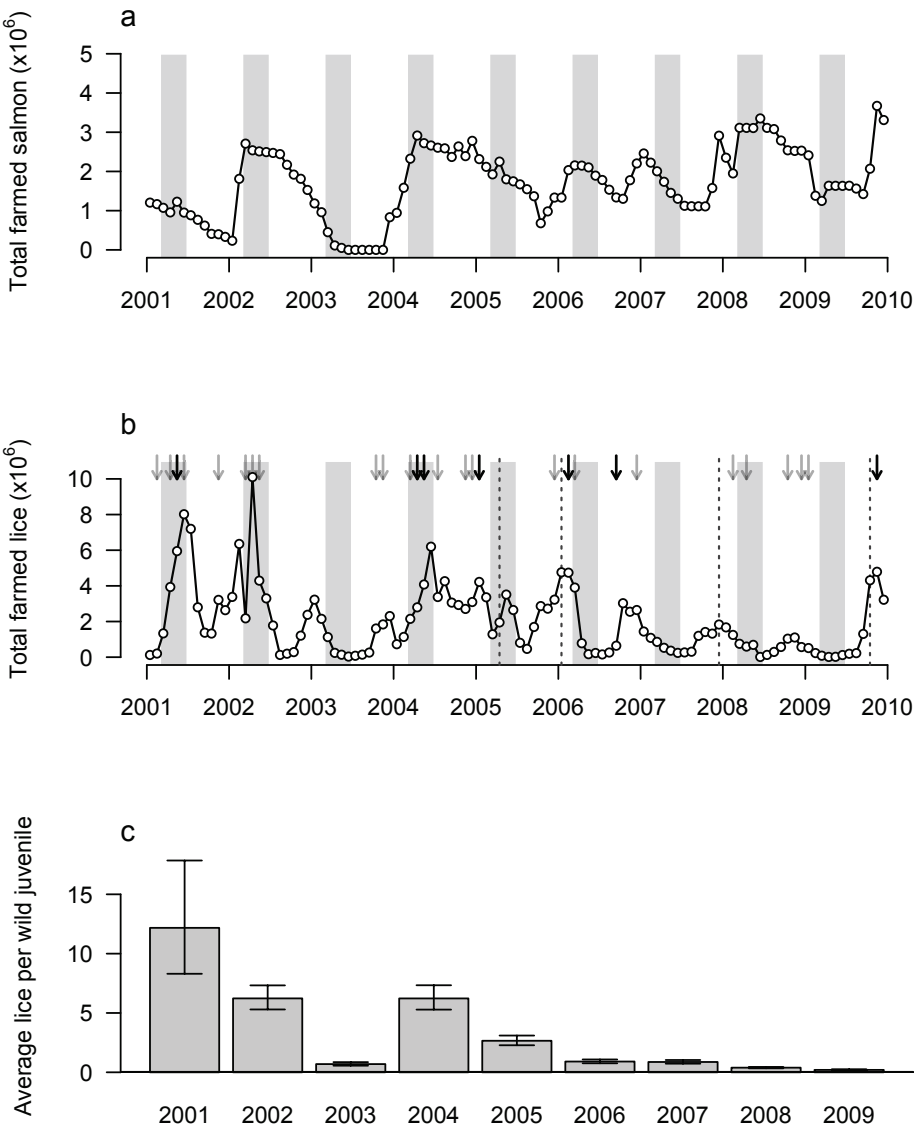

Figure 3.3: Total number of farm salmon in their second year of production (a), total number of gravid *L. salmonis* on farm salmon (b), and average abundance (±95% CI) of *L. salmonis* (all stages) per juvenile salmon in Tribune Channel and Fife Sound (c). Light grey vertical bars in (a) and (b) represent the time period of the juvenile salmon outmigration (March-June) and the vertical dotted lines in (b) indicate the occurrence of at least three parasiticide treatments of a salmon farm in that month, while grey and black arrows represent one or two treatments in that month respectively.

Table 3.3: Summary of annual characteristics of lice on wild and farm fish in the KTF corridor of the Broughton Archipelago between 2001 and 2009.

Year	Farmed salmon		Juvenile pink salmon	
	Lice ($\times 10^6$)*	Winter treatments†	Lice ($\pm$ 95% CI)‡	n
2001	19.2	No	12.17 (8.30, 17.85)	268
2002	19.9	No	6.23 (5.30, 7.33)	490
2003	1.5	Yes	0.69 (0.56, 0.86)	367
2004	15.2	No	6.23 (5.28, 7.34)	546
2005	9.4	Yes	2.66 (2.28, 3.10)	1892
2006	5.1	Yes	0.91 (0.76, 1.08)	726
2007	2.0	Yes	0.87 (0.73, 1.04)	1000
2008	2.0	Yes	0.39 (0.34, 0.45)	2075
2009	0.2	Yes	0.20 (0.16, 0.26)	543

*Farm salmon lice were the total number of female lice (millions) on farm salmon in the KTF corridor during the out-migration season (March 1 - June 30) of each year.
†Winter treatments indicates whether the proportion of parasiticide treatments occurring in winter (January-March or October-December of the given year) was ≥ 0.50 (note the fallowing management intervention in 2003).
‡The average louse abundance (all stages) on wild juvenile pink salmon (95% confidence intervals) as estimated by the generalized linear model, and number of juvenile pink salmon sampled each year (n).

season (figure 3.3a). In 2006 and later years, treatment of farm fish with parasiticide occurred more frequently prior to the juvenile salmon outmigration season (table 3.2), and louse abundance on wild juvenile salmon shows a corresponding decline (table 3.3, figure 3.4b). In particular, 2006 appears to be a turning point in management actions on the migration corridor and sea louse abundance on wild juvenile salmon in the migration corridor (Figs. 3.3, 3.4). The proportion of total treatments occurring in winter had a greater effect on the average lice per wild juvenile salmon ($R^2 = 0.777$, $p = 0.002$, AIC = 8.0), than did the total number of treatments ($R^2 = 0.000$, $p = 0.981$, AIC = 21.5; table 4).

Intensive louse surveys of wild juvenile salmon in 2004 and 2009 involved 6384 and 9482 pink salmon, respectively. These samples were separate and in addition to the weekly monitoring of lice on wild salmon described above. The

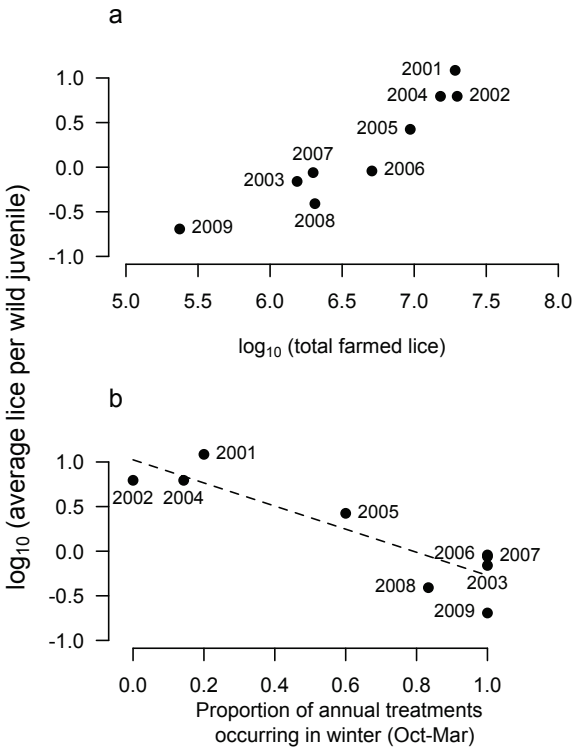

Figure 3.4: Relationship between the estimated average number of *L. salmonis* per juvenile pink salmon per year and the total abundance of gravid *L. salmonis* on farm salmon in the Knight Inlet - Tribune Channel - Fife Sound migration corridor per year (a), and the proportion of all treatments of farm salmon with parasiticide that occur in advance of the juvenile salmon outmigration each year (i.e., winter treatments) (b). The dashed line in (b) is the linear regression of log_{10}(avg. *L. salmonis* per juvenile pink salmon) over the proportion of winter treatments (table 4).

2004 data consisted of three replicate surveys, and the 2009 data consisted of five replicate surveys of the KTF corridor (Krkošek et al., 2006a). The spatial survey data indicated major declines in the magnitude of transmission from farm salmon to wild juvenile salmon in 2009 relative to 2004 (figure 3.5). Focusing on two surveys in May, the peak of the mean number of motile lice per wild juvenile salmon was nearly 20 times lower in 2009 than 2004 (1.55 motiles/fish versus 0.08 motiles/fish, figure 3.5i,l). Further, the peaks in infection of wild juvenile salmon after they migrate past salmon farms that characterize the 2004 data (Krkošek et al., 2006a, 2005a) were not apparent in 2009. These changes are consistent with changes between 2004 and 2009 in the abundance of farm fish and gravid lice per farm fish in farms on the migration route (figure 3.3b). The decline in abundance and spatial pattern of sea lice infection in 2009 was common to all replicate surveys. Differences in salinity between years, while known to affect louse survival (Pike and Wadsworth, 2000), were unlikely to have confounded the results. A paired t-test on salinities in 2004 and 2009, paired by month and distance along the migration route, suggested no significant difference between years ($t = 0.047$, $df = 14$, $p = 0.963$). Ranges in temperature and salinity were similar between 2004 and 2009 (see table 5 in Peacock et al. 2013).

There were significant declines in the survival of pink salmon populations with sea louse infestations in juveniles (figure 3.6). Including the covariate of average lice per wild juvenile salmon improved the fit of the model (Likelihood ratio test, $\chi^2 = 12.128$, $df = 1$, $p < 0.001$). The growth rate for pink salmon over all areas was $r = 1.088$ (95% CI = 0.873, 1.302) and the parameter for the effect of lice on survival was significantly different from zero ($c = 0.190$, 95% CI = 0.087, 0.299), indicating a reduction in survival with increasing abundance of sea lice on wild juvenile salmon. The estimated percent mortality of pink salmon in the Broughton due to sea louse infestations ranged from 90.1% for

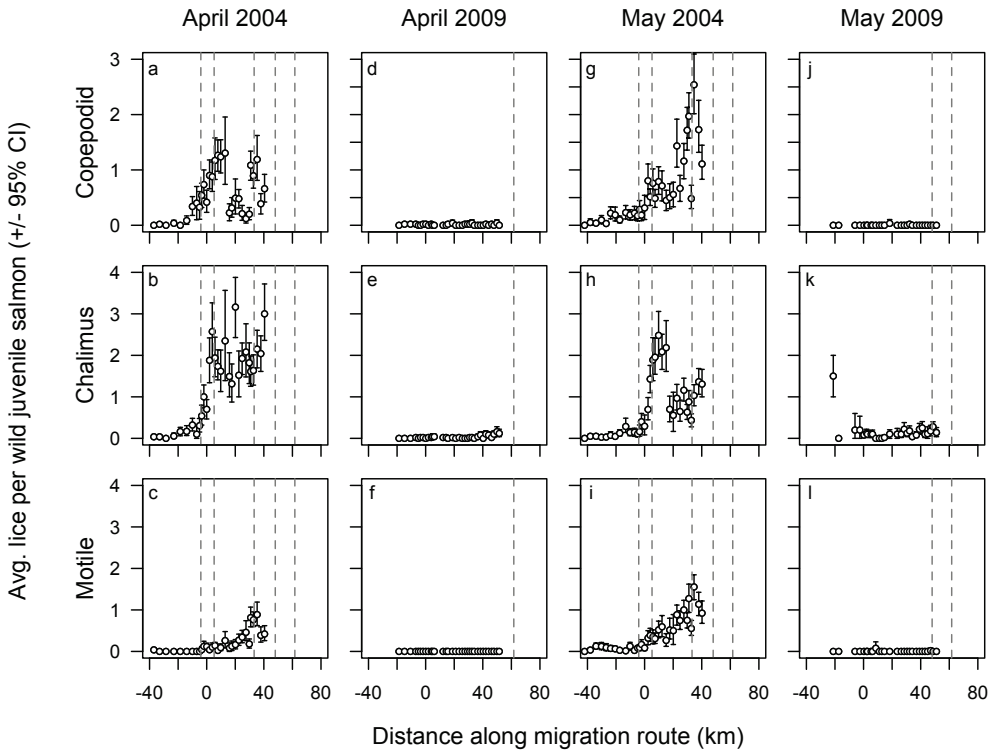

Figure 3.5: Spatial distributions of the average number of L. salmonis developmental stages on wild juvenile pink salmon in April and May of 2004 and 2009 along the Knight Inlet - Tribune Channel - Fife Sound migration corridor (direction of fish migration is from left to right within each panel). The top, middle, and bottom rows of panels show the average abundance of parasitic copepodites, chalimus and motile stages per fish, respectively (with 95% bootstrapped CI). Vertical dashed lines indicate the locations of the active salmon farms in each year.

returns in 2002, to 3.8% for returns in 2010, and showed a declining trend between 2002 and 2010 (figure 3.6b).

3.4 Discussion

The spread of infection from domesticated animals can threaten wildlife (Krkošek et al., 2007a; Pedersen et al., 2007) and create situations of high management urgency and uncertainty (Haydon et al., 2002; Krkošek, 2010b). For salmon

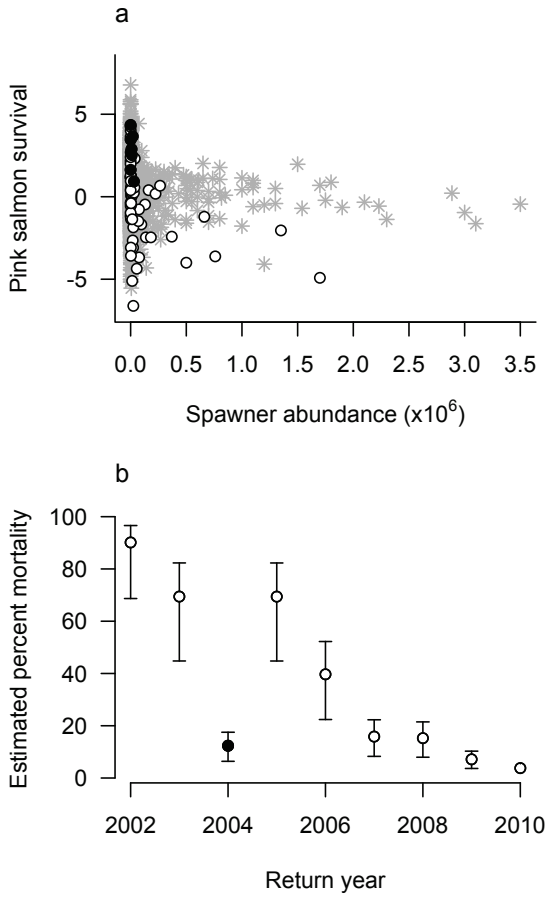

Figure 3.6: (a) Pink salmon survival ($\log(R_{i,t}/N_{i,t})$) for reference populations and the Broughton Archipelago prior to the onset of sea louse infestations (grey stars), and during sea louse infestations in the Broughton Archipelago (2002 to present, circles). (b) The percent mortality of Broughton populations due to sea louse infestations of juvenile salmon ($1-\exp(-cW_{a,t-1})$), as estimated by a modified Ricker model fit to the data in (a). Prior to 2001 (return year 2002), there are no data on sea louse abundances on juvenile salmon and mortality due to infestations was assumed to be negligible. Error bars indicate the range of mortality arising from a 95% bootstrapped CI on the parameter c. Closed circles in both (a) and (b) correspond to salmon that migrated through the Broughton Archipelago during the fallowing intervention of 2003.

and sea lice in the Broughton Archipelago, I found that infections of wild juvenile salmon increased with sea louse abundance on farm salmon. Intensive spatial surveys of sea lice on wild juvenile salmon showed low infection levels on juvenile salmon prior to migration past salmon farms, suggesting that sea lice transferred from farm salmon to wild salmon. Management actions, such as fallowing farms along the migration routes of juvenile salmon and winter treatments with parasiticide, lowered the abundance of sea lice on farm salmon, and therefore reduced infection of wild salmon. Finally, there was a strong negative relationship between pink salmon survival and sea lice infection of juveniles, implicating that efforts by the salmon farming industry to reduce sea lice levels during the wild salmon out-migration have positive implications for wild salmon survival and productivity.

Lepeophtheirus salmonis epizootics of wild juvenile salmon in the Broughton Archipelago were first observed in 2001 (Morton and Williams, 2003). Earlier, lice were noted on salmon farms in the area but outbreaks were not sufficiently widespread to require regular monitoring and treatment (Marty et al., 2010). The sudden nature of sea lice epidemics suggests a critical host density threshold in the region, above which sea lice population growth will occur exponentially if left untreated, was exceeded (Frazer et al. 2012), and is consistent with louse outbreaks elsewhere (Jansen et al., 2012) and epidemiological theory (Krkošek, 2010a). The absence of lice data on wild juvenile salmon prior to 2001 necessitates making assumptions on lice abundance during the 1990s, when farms were present but outbreaks were not reported. In my analysis, I assume these abundances on wild juvenile salmon were at roughly natural levels due to host-density thresholds having not been exceeded. The effects of lice on salmon survival during the 1990s are therefore absorbed into the estimation of the population growth rate, which would include louse-induced host mortality at natural louse levels. After outbreaks began, it took two years

for louse monitoring in the Broughton Archipelago to become systematic (in 2003; Jones et al. (2006); Marty et al. (2010)), and the results indicate it took another three years before treatment became adjusted to the outmigration of wild juvenile salmon.

In the midst of the outbreaks in the early 2000s, a fallowing management intervention closed most farms on the migration route (Morton et al., 2005). This was implemented by provincial regulators, partially in response to the population collapse in the preceding year (PFRCC, 2002). The fallowing management intervention reduced infection rates on wild juvenile salmon (Morton et al. 2005) and improved survival of the affected pink salmon cohorts (Beamish et al., 2006). Another study comparing parasite loads and marine survival between fallow and active migration routes in the same year reached similar conclusions (Morton et al., 2011). While fallowing is an effective management tool for controlling outbreaks, it is less economical for farms to be fallowed on an annual basis because salmon production cycles usually exceed one year.

Reliance on parasiticide use in sea-cage salmon aquaculture therefore appears to be inevitable if current aquaculture production cycles continue and overall production exceeds regional host-density thresholds below which outbreaks do not occur (Frazer et al., 2012). However, in a regime of parasite population growth in the Broughton Archipelago, I found that meeting conservation objectives for wild salmon did not involve a significant increase in the number of parasiticide treatments over previous management, but rather, a change in the timing of treatment in advance of wild salmon migration schedules. These winter treatments were not due to compliance of management with policy, as they occurred during months when regulatory policy did not necessitate management intervention, only increased monitoring frequency when sea louse abundances reach three motiles per farm salmon.

The changes in parasite management I have documented occurred during a period of intensive scientific study (Krkošek, 2010b; PFRCC, 2002). During this period, many multi-stakeholder processes connected research scientists with fisheries managers, aquaculture veterinarians, policy representatives, conservation organizations, First Nations, eco-tourism operators, commercial fishing interests, and other groups. Examples include the Broughton Archipelago Monitoring Program (`www.bamp.ca`), the British Columbia Pacific Salmon Forum (PSF, 2009), and Simon Fraser University's Speaking for the Salmon Series (Gallaugher and Wood, 2004; Routledge et al., 2007). These processes may have been vital in exchanging and interpreting scientific information that aided a response from management during a period of high scientific progress and uncertainty.

Nevertheless, progress on science, management, and policy of salmon aquaculture and sea lice is constantly challenged by the correlative nature of analyses such as this. The inter-annual changes in louse abundance on wild and farm salmon as well as salmon population growth rates are consistent with a process of disease outbreaks and subsequent control. However, these linkages are not the product of formal scientific principles of replication and randomization, possible in a controlled setting, but rather correlations within components of a dynamic social-ecological system. It is therefore possible that the results presented here are the product of other unknown processes that were spatially and temporally correlated with sea lice and salmon management and population dynamics, although no such alternative process has yet been identified. Despite such uncertainty, I found effective advance louse management on farms has appeared to yield positive conservation benefits.

Another limitation of this analysis is the use of the Knight-Inlet - Tribune Channel - Fife Sound migration corridor as an indicator of how sea louse abundance has changed among years in relation to farm management and

influenced productivity of exposed pink salmon populations. Based on 10 years of fieldwork in the Broughton Archipelago, I understand that the migration route I studied is the primary corridor through which the main biomass of juvenile salmon transits from rivers in the Broughton Archipelago to Queen Charlotte Strait. This also accords with traditional knowledge of local residents and aboriginals in the area, but is nevertheless an untested assumption and alternate migration routes are possible (e.g., directly to the mouth of Knight Inlet; figure 3.2). The locations of the various rivers in relation to the salmon farms will introduce some variability among populations in their exposure levels, and I have not directly accounted for such effects except for the area within year random effect in the spawner-recruit model.

Estimates of lice on wild juvenile pink salmon from weekly monitoring of three sites in the Broughton Archipelago are challenged by changing methodologies in sample collection and analysis throughout the study period. Juvenile salmon were collected by dip net (2001-2003) and beach seine (2004-2009), and each method has potential biases. Dip netting may select for weaker fish that are slower to evade the net, but reduces the time fish spend in the net. Beach seining reduces selection bias (although fitter fish may be able to escape the net in sub-optimal conditions), but increases the time spent in the net when lice and scales may be rubbed off (Morton et al., 2004). I assume the potential errors associated with each of these methods are small relative to the yearly differences in infection pressure, as there is no abrupt change in infection intensity with the change in collection methods. Similarly, switching from lethal examination in a laboratory under a microscope (2001-2004) to visual assays of live salmon using a hand lens in the field (2005-2009) may have confounded results as visual assays of live salmon have been shown to slightly underestimate the abundance of copepidite and chalimus stages (Krkošek et al., 2005b). However, these errors are again likely small relative to annual changes in in-

fection intensity. Indeed, when I analyze the counts of motile lice only, which are enumerated equally well on live and euthanized salmon (Krkošek et al., 2005b), the annual trends are the same and these results are unchanged.

Clearly, the magnitude and uncertainty of the linkages between lice on farms, lice on wild juvenile salmon, and salmon population growth are sensitive to assumptions of various plausible migration routes (Krkošek et al., 2011b; Marty et al., 2010). Here, I have applied similar methodologies that previously documented epizootics and population decline of wild pink salmon populations in the Broughton Archipelago (Krkošek et al., 2007a, 2006a, 2005a; Krkošek and Hilborn, 2011) to new data from the area in more recent years, and uncovered a significant negative relationship between lice on juvenile salmon and salmon survival. Together with the relationship between lice on juvenile salmon and management of lice on salmon farms, these results suggest that recent adaptive changes in parasite management have had positive effects for conservation of pink salmon in the Broughton Archipelago.

The changes in parasite management on salmon farms in the Broughton are not an example of formal adaptive management (Walters, 1997). These were not experimental changes that were planned according to a quantitative framework designed to systematically evaluate management effectiveness, but nor were they strictly trial and error. Rather, these results are likely the product of a contentious and productive scientific debate with continuing disagreement, multi-stakeholder involvement, and management responses. These processes may have nevertheless led to adaptive change in a social-ecological system, with at least temporary conservation gains. It is not clear if adaptive changes in management and policy in social-ecological systems are more commonly attributable to formal adaptive management or the more contentious multi-stakeholder process that has occurred for sea lice and salmon in the Broughton Archipelago. I suspect the latter, and there has been increasing interest in

alternative views of adaptive management that regard conservation as a social process, where alternative objectives and perceptions must be considered (e.g., Cundill et al., 2012).

Sea louse outbreaks and concerns of transmission to wild salmonids are not new issues unique to the Broughton Archipelago. Concerns of declines of wild salmon and trout in Europe (e.g., Bjørn et al., 2001; Butler and Watt, 2003; Gargan et al., 2003) and elsewhere (Ford and Myers, 2008) have spurred coordinated area management and strategic delousing treatments of farm salmon in these areas. Winter treatment of farm salmon prior to wild salmon migrations and before warming temperatures spur sea lice population growth has been recommended in Europe and eastern Canada for almost a decade (Costello, 2004). Some of these changes to sea lice management have been met with success, decreasing the infection pressure and numbers of sea lice on wild salmonids (e.g., Bjørn et al., 2011; Heuch et al., 2009), although connections from management changes to the productivity of wild fish populations have rarely been made.

The long-term sustainability of social-ecological systems that depend on wild and farm salmon remains to be resolved. In the Broughton Archipelago, current louse management could be undermined by parasite evolution of resistance to chemical treatments, as has occurred or is occurring elsewhere (Lees et al., 2008; Westcott et al., 2010). In addition, there is little known of potential impacts of parasiticide use on other ecosystem components or processes (Burridge et al., 2010). Beyond sea lice, other infectious diseases, such as Infectious Salmon Anemia (Olivier, 2002), or ecological effects of farming non-native species (Volpe et al., 2001) may be of concern. Coordinated fallowing of farms after harvest may help break the cycle of infection for sea lice and other pathogens (Costello, 2004). As global aquaculture growth continues (FAO, 2012), adaptive changes in disease management may be fundamental to

resilience of social-ecological systems dependent on both wild and farm fish.

Chapter 4

Can reduced predation offset negative effects of sea louse parasites on chum salmon?

4.1 Introduction

By definition, parasites harm their hosts (Anderson and May, 1978). The fitness of parasitized individuals can decrease through direct parasite-induced mortality, reduced fecundity, for example via parasitic castration (Baudoin, 1975), or reduced reproductive success, for example via sexual selection (Hamilton and Zuk, 1982). The impact of parasites on host individuals is invariably negative when considered in isolation, but may be complex and unexpected

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in nature. Parasitism is interdependent with other ecological interactions that the host experiences, such as predation and competition (Hatcher et al., 2012). For example, parasites may increase host susceptibility to predation (Johnson et al., 2006; Krkošek et al., 2011a) and, in turn, parasite populations may be regulated when infested hosts are preyed upon (Packer et al., 2003). Feedbacks between parasitism and predation can be further complicated by non-linear predator-prey dynamics and clumping of parasites among hosts.

The direct impact of parasites on host population dynamics may be weak relative to other drivers such as predation because parasite infestations are often sub-lethal (Hatcher et al., 2012). Therefore, understanding the indirect effects of parasitism on processes such as predation may actually be more important than understanding the direct effects of parasites on isolated host individuals. If parasites act to reduce predation on hosts then the net effect of infestation may be negligible or even positive for the host if gains from reduced predation offset or exceed direct costs of parasitism. Here, I explore this idea in the context of parasitic sea louse infestations of juvenile pink and chum salmon (*Oncorhynchus* spp.) in Pacific Canada.

I first report on an analysis of chum salmon spawner-recruit data in which I did not find an effect of sea lice on chum salmon productivity despite high statistical power. This unexpected result led me to investigate the effects of parasitism on interactions within a juvenile salmon food web that may mitigate the impact of sea lice for chum salmon. Predation by coho salmon (*O. kisutch*) is an important source of mortality for both juvenile pink and juvenile chum salmon (Groot and Margolis, 1991) that may be mediated by sea lice (Krkošek et al., 2011a). Field-based experiments suggest that coho salmon prefer to consume pink salmon over chum salmon (Hargreaves and LeBrasseur, 1985). In the second part of this chapter, I use a mathematical model to explore the conditions under which a parasite-induced shift in predation to pink salmon may

lead to higher chum salmon survival in a regime of sea louse infestations. The results indicate that the ecological context of host-parasite interactions may alter, or even reverse, the expected impact of parasites on host populations.

4.2 Chum salmon productivity

4.2.1 Methods

In fisheries, productivity can be calculated as the mean number of offspring that survive to adulthood and are either caught in fisheries or return to freshwater to spawn (i.e., recruits per spawner) (Hilborn and Walters, 1992). I modelled chum salmon productivity using a Ricker spawner-recruit model (Hilborn and Walters, 1992; Ricker, 1954). The full model included hierarchical terms to account for spatial and temporal covariation among populations (described below) and a covariate describing the effect of sea lice on chum salmon productivity (Krkošek et al., 2011b),

$$\underbrace{\ln[R_{i,t}/S_{i,t}]}_{\text{population productivity}} = \underbrace{(r+\theta_t+\theta_i+\theta_{CU}+\theta_{t/a})}_{\text{growth rate with random effects}} - \underbrace{b_i S_{i,t}}_{\text{density dependence}} - \underbrace{cL_{a,t+1}}_{\text{effect of sea lice}} + \underbrace{\varepsilon_{i,t}}_{\text{residual variation}} \tag{4.1}$$

where $R_{i,t}$ are the recruits to population i produced by spawners in brood year t, $S_{i,t}$ is the spawner abundance, r is the overall growth rate, $b_i S_{i,t}$ is the population-specific within brood year density-dependent mortality, and $cL_{a,t+1}$ is the estimated mortality of chum salmon due to sea lice. Residual variation, $\varepsilon_{i,t}$, is normally distributed with mean zero and variance to be estimated. I

ignored measurement error associated with the enumeration of spawners, as in previous studies of spawner-recruit data in relation to sea lice (e.g., Krkošek et al., 2011b; Peacock et al., 2013), because accounting for both process and measurement error greatly complicates the analysis. Further, it has been shown that explicitly including measurement error in a state-space framework does not improve parameter estimates in the range of growth rates that I encountered (Su and Peterman, 2012).

For species that return at different ages, such as chum salmon, recruits in a given return year need to be assigned to brood years based on the distribution of ages at return. I compiled spawner-recruit time series by brood year for river populations on the south-central coast of British Columbia, Canada from spawner, catch, and age-at-return data provided by Fisheries and Oceans Canada (Fisheries and Oceans Canada, 2011a). River populations were excluded from the analysis if there were spawner abundance estimates for less than one-third of the years analyzed. Chum salmon return to spawn as 3-, 4- or 5-year-old adults. The distribution of ages for multiple return years was therefore needed to calculate total recruitment corresponding to a single brood year. In years and/or areas where age data were not available, I imputed age data in by assuming all returns in a given year were 4-year old fish (Pyper et al., 2002). I tested the sensitivity of my results to this assumption by also imputing missing ages-at-return assuming that missing return years has the same age structure as other years for that area, and areas without any age data were the same as adjacent areas, and by running the entire analysis assuming a constant 3-, 4- and 5-year age-at-return across all areas and years. In total, I analyzed trends in productivity from 1980-2005 for 63 chum salmon river populations; 53 unexposed and 10 exposed to salmon farms.

Adult Pacific salmon tend to return to their natal rivers to spawn (Quinn, 2005), allowing an analysis of factors affecting chum salmon productivity at

the spatial scale of river populations. However, chum salmon display lower fidelity to natal rivers than some other salmon species (e.g., sockeye salmon). I accounted for synchrony in productivity among river populations at larger spatial scales by modelling variability in growth rates among years, regions, statistical management areas and ecologically and/or genetically distinct biological units termed Conservation Units under Canada's Wild Salmon Policy (Holtby and Ciruna, 2007; Irvine, 2009). Variability in growth rates among years common to all populations in the region was included as θ_t (Mueter et al., 2002; Pyper et al., 2002). Variability in growth rates among populations was included as θ_i (Dorner et al., 2008). Variability in growth rates among Conservation Units was included as θ_{CU}. Finally, Variability in growth rates among Pacific fishery management areas was included as $\theta_{t/a}$ and accounts for the non-independence of recruitment estimates within management areas due to common harvest rates and the non-independence of sea louse abundance which is measured at the scale of management areas. These random effects were assumed to be normally distributed random variables with means of zero and variances to be estimated.

The covariate $L_{a,t+1}$ is an estimate of parasite exposure for populations in area a and year $t+1$ when juvenile chum salmon from brood year t enter the ocean and migrate past salmon farms. I investigated two different forms of this covariate. First, I used the sum of adult female sea lice in April on all farmed salmon in the vicinity of the juvenile salmon migration route (Krkošek et al., 2011b). For years 2000-2002, some of these salmon farms did not report sea louse abundances, and so I estimated these abundances under four different scenarios (F1-F4 (Krkošek et al., 2011b)). The second form of the covariate was the average number of attached sea lice (copepodid, chalimus, and motile stages) per juvenile wild pink and chum salmon (Peacock et al., 2013). Due to the absence of data for sea louse abundances on farmed and wild salmon in

the 1990s (Marty et al., 2010; Peacock et al., 2013), brood years 1990-1998 for the farm sea louse covariates and 1990-1999 for the wild sea louse covariate were excluded from the analysis. I tested the significance of the sea louse covariate using a likelihood ratio test with the null model $c = 0$ indicating no correlation between sea louse abundance and chum salmon productivity. I performed a retrospective power analysis to determine my power to detect an effect of sea lice if an effect indeed existed.

4.2.2 Results

There was no evidence of reduced productivity of chum salmon populations exposed to sea louse infestations on farmed salmon (figure 4.1). The model fit was not improved by including a sea louse covariate (table 4.2.2). Populations exposed to salmon farms showed no obvious declines in productivity associated with either the expansion of salmon farming circa 1990 or sea louse infestations (1999-2005, figure 4.1). These results were consistent across all age-at-return scenarios that I considered. I found significant covariation among populations within the study region in each year, within populations, Conservation Units, and within management area each year, as indicated by an improvement of the model when all random effects were included.

The lack of a significant correlation alone is not reason to discount a possible impact of sea louse infestations on chum salmon populations. However, I had high power to detect changes in growth rate that would have resulted in population declines. Simulations incorporating model estimates of variability indicated I had 80.1 - 99.8% power to detect a rate of decline of $c = 0.20$, depending on the form of the covariate used. I had $> 70\%$ power to detect effects in the range of those found for pink salmon (Krkošek et al., 2011b; Peacock et al., 2013), except for the F1 covariate, for which I had just 46.2%

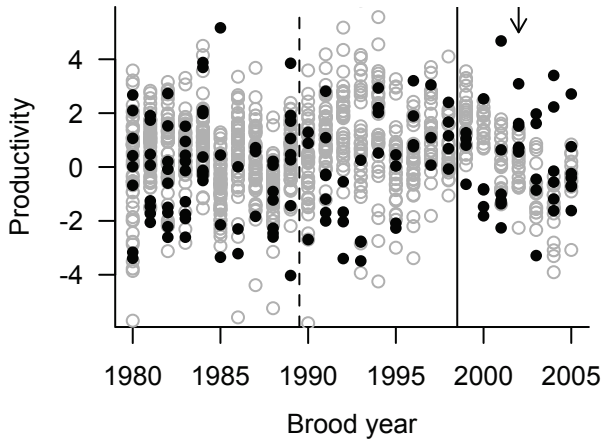

Figure 4.1: Productivity (log recruits per spawner) of chum salmon river populations in south-central British Columbia that were unexposed (grey open circles) or exposed to sea lice from farmed salmon in the Broughton Archipelago (black closed circles). Salmon farming was expanding in the Broughton Archipelago circa 1990 (dashed line), while the onset of recorded sea louse infestations wasn't until 2000 (affecting chum salmon from brood year 1999, solid line). A fallow management intervention in 2003 affected those salmon migrating from brood year 2002 (arrow).

Table 4.1: The parameter for sea louse-induced mortality of chum salmon, c, was not significantly different from zero for all forms of the sea louse covariate, $L_{a,t+1}$ (equation 4.1). Likelihood ratio tests with the null model showed no improvement with the inclusions of the sea louse covariate. Results for different age-at-return scenarios are provided in Table S4.

Louse covariate	c^*	logLik†	$\chi^2_{0.05,1}$	p
F1	0.064 (-0.025, 0.155)	-1615.1	3.601	0.058
F2	0.077 (-0.077, 0.222)	-1616.0	1.798	0.180
F3	0.068 (-0.054, 0.185)	-1615.8	2.260	0.133
F4	0.069 (-0.055, 0.189)	-1615.8	2.262	0.133
W	0.109 (-0.048, 0.251)	-1609.0	3.084	0.079

*Maximum likelihood parameter estimate (95% bootstrapped CI).
†log Likelihoods are not directly comparable among covariate models, as the wild and sea louse datasets had different amounts of missing data.

power to detect the effect size found for pink salmon. Although sea lice increase mortality rates of individual chum salmon in captivity (Krkošek et al., 2006a; Morton and Routledge, 2005), the results suggest this does not translate to a measurable impact on chum salmon at the population level.

4.3 Parasite-mediated changes to predation

In the following section, I develop a host-macroparasite model describing the population dynamics of a generalist parasite and two hosts in the presence of a common predator. The objective is to determine the biologically relevant conditions under which reduced predation may lead to a negligible net impact of parasites on the survival of one of the host populations. The model has general applicability, but I employ parameters from the literature for sea lice and Pacific salmon hosts to determine whether parasite-mediated changes to predation may offset direct parasite-induced mortality for chum salmon (figure 4.2).

4.3.1 The functional response

The functional response describes the consumption rate of a predator as a function of prey abundance or density. Holling's type II functional response predicts increasing predation rates with increasing prey abundance to a satiation point, above which predators are limited by the time it takes to handle and digest prey (Holling, 1959). For a predator of more than one prey species, consumption rates of a particular prey species are lower because the predator spends time handling alternative prey (Chesson, 1983; Smout et al., 2010). The type II functional response for a generalist predator of m different prey

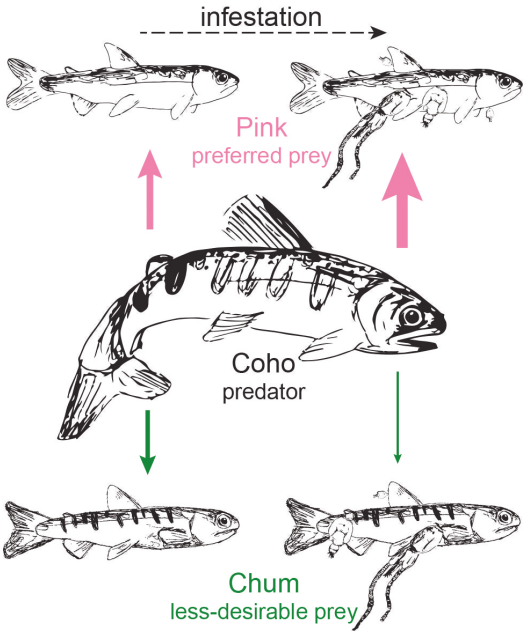

Figure 4.2: Predation pressure, indicated by the thickness of the solid arrows, is higher for pink salmon as they are a preferred prey of coho salmon (Hargreaves and LeBrasseur, 1985). As prey become parasitized, they are easier to identify and/or catch allowing predators to more easily express their prey preference. As a result, infestation may decrease predation pressure on less desirable prey species. In this case, chum salmon may experience lower predation pressure in a regime of sea louse infestations.

species is:

$$f_i = \frac{a_i N_i}{1 + T \sum_{j=0}^{m} a_j N_j}, \tag{4.2}$$

where f_i is the instantaneous rate of consumption of prey species i by a single predator, a_i is the maximum rate at which predators attack and capture prey species i (henceforth referred to as the attack rate), N_i is the number of individuals of species i that are available to predators, and T is the handling time it takes a predator to consume and digest a single prey item, which can differ among prey species but we assume is the same for all prey species.

The influence of parasites on predator-prey interactions varies, depending largely on whether parasites make prey more or less vulnerable to predation (Hall et al., 2005). Infested individuals may incur additional costs that require them to display riskier foraging behaviour or they may be physiological impaired and more likely to succumb to predation (e.g., Hall et al., 2005; Hudson et al., 1992). I adapted the multi-prey type II functional response to include a linear increase in the attack rate with increasing number of parasites, such that the attack rate on host species i with x parasites is $a_{i,x} = \gamma_i + \sigma_i x$ where γ_i is the attack rate on species i in the absence of parasites and σ_i is the per-parasite increase in attack rate (Krkošek et al., 2011a). I assumed a linear increase in attack rate with parasites because for juvenile salmon, a single sea louse may have detrimental affects on performance. However, there may be thresholds in the number of parasites below which parasites have little effect on host behaviour, particularly for larger fish (Wells et al., 2006), and so I also considered non-linear increases in attack rates (results not shown in detail, but see electronic supplement for Peacock et al. 2014).

Including the parasite-mediated attack rate in the multi-prey functional response (equation 4.2), I obtain the following equation for the predation rate

on individuals of species i with x parasites in the presence of an alternative species j that is also affected by parasites:

$$f_{i,x} = \frac{q_i(x)N_i(\gamma_i + \sigma_i x)}{1 + T\left[\sum_{y=0}^{\infty}\left\{q_i(y)N_i(\gamma_i + \sigma_i y)\right\} + \sum_{z=0}^{\infty}\left\{q_j(z)N_j(\gamma_j + \sigma_j z)\right\}\right]} \tag{4.3}$$

where $q_i(x)$ is the proportion of prey species i with x parasites. The summations in the denominator arise because the saturation point of the functional response is determined by the attack rates of both species with all possible numbers of parasites. The proportions of the total prey of species i and j with y and z parasites are $q_i(y)$ and $q_j(z)$. These are the probability mass functions for the distributions of parasites per host and may differ between host species i and j. Parasites are often overdispersed among host individuals (Shaw et al., 1998), and sea lice are no exception (Murray, 2002). We assumed that parasites were distributed among hosts according to the negative binomial distribution, for which $\bar{P}_i = \sum_{y=0}^{\infty}\{q_i(y)\ y\}$ is the expected number of parasites on prey species i and $\bar{P}_j = \sum_{z=0}^{\infty}\{q_j(z)\ z\}$ is the expected number of parasites on prey species j. (See Appendix 1 of Anderson and May (1978) for the expected values of other discrete distributions commonly applied to parasites.) We can then simplify equation 4.3 to:

$$\begin{aligned} f_{i,x} &= \frac{q_i(x)N_i\left(\gamma_i + \sigma_i x\right)}{1 + T\left[N_i\left(\gamma_i + \sigma_i\sum_{y=0}^{\infty}\left\{q_i(y)y\right\}\right) + N_j\left(\gamma_j + \sigma_j\sum_{z=0}^{\infty}\left\{q_j(z)z\right\}\right)\right]} \\ &= \frac{q_i(x)N_i\left(\gamma_i + \sigma_i x\right)}{1 + T\left[N_i\left(\gamma_i + \sigma_i\bar{P}_i\right) + N_j\left(\gamma_j + \sigma_j\bar{P}_j\right)\right]}. \end{aligned} \tag{4.4}$$

4.3.2 Host-parasite population dynamics

To evaluate the combined effects of parasite-mediated changes to predation and direct parasite-induced mortality, I consider the above functional response in a mathematical model describing the change in abundance of two host populations and each of their associated parasite populations. I treat predator abundance as a constant (Hall et al., 2005), independent of host/prey abundance and parasites. That is, I consider the functional response of predators to the abundance of prey but do not include a numerical response in the predator. This was in part to simplify the model, but also because numerical responses of coho salmon likely occur on much longer timescales than the within-season dynamics of juvenile salmon hosts and parasites that I consider. The model builds upon the original host-macroparasite model by Anderson and May (1978) and more recent work by Krkošek et al. (2011a) who considered just a single host species and associated parasite population.

The first pair of equations describes the decline in abundance of two host populations due to predation and direct parasite-induced mortality, where the host species interact through a common predator. The general equation for the change in population i in the presence of alternate prey j is:

$$\frac{dN_i}{dt} = \underbrace{-C\sum_{x=0}^{\infty}\left\{q_i(x)\,\frac{N_i\left(\gamma_i+\sigma_i x\right)}{1+T\left[N_i\left(\gamma_i+\sigma_i\bar{P}_i\right)+N_j\left(\gamma_j+\sigma_j\bar{P}_j\right)\right]}\right\}}_{\text{host mortality from predation}}$$

$$\underbrace{-\alpha\sum_{x=0}^{\infty}\left\{q_i(x)\,x\,N_i\right\}}_{\text{host mortality from parasites}}$$

$$= -C\frac{N_i(\gamma_i+\sigma_i\bar{P}_i)}{1+T\left[(N_i(\gamma_i+\sigma_i\bar{P}_i)+N_j(\gamma_j+\sigma_j\bar{P}_j)\right]} - \alpha\,\bar{P}_i\,N_i. \qquad (4.5)$$

Host populations decline due to predation at a rate predicted by the multi-prey functional response (equation 4.4) and due to direct parasite-induced mortality at rate $\alpha\bar{P}_i$, where $\bar{P}_i = P_i/N_i$ is the average number of parasites per host. There is no source term for N_i because I considered the survival of a cohort of hosts, not including host reproduction. This approach is applicable to migrating juvenile salmon, and also avoids having to account for the potentially large difference in generation times of hosts and parasites. The equation for the rate of change in the number of prey species j is the same as equation 4.5, with i's and j's reversed.

The second pair of equations describes the change in the total number of parasites on each host population, P_i and P_j. Once again, I present the general

form for the total number of parasites on host/prey population i:

$$\frac{dP_i}{dt} = \underbrace{\beta L N_i}_{\text{attachment}} - \underbrace{\mu P_i}_{\text{natural mortality}}$$

$$- \sum_{x=0}^{\infty} \left\{ \underbrace{x}_{\text{killed with hosts}} \left[\underbrace{C \frac{q_i(x)\, N_i(\gamma_i + \sigma_i x)}{1 + T\left[N_i(\gamma_i + \sigma_i \bar{P}_i) + N_j(\gamma_j + \sigma_j \bar{P}_j)\right]}}_{\text{host mortality from predation}} \right. \right.$$

$$\left. \left. + \underbrace{\alpha\, q_i(x)\, x\, N_i}_{\text{host mortality from parasites}} \right] \right\}. \tag{4.6}$$

Parasites attach at rate βL, where β is the transmission coefficient and L is the density of free-living infectious-stage parasites. Parasites have a natural mortality rate, μ. I assumed that attachment and mortality were the same on both hosts populations, although this assumption could be relaxed for other systems. Finally, parasites were assumed to die when their host dies. Although it has been shown that parasites can be trophically transmitted from prey to predator (Connors et al., 2010a), I do not consider the infection level of the predators in my model and so parasites that might jump onto successful predators are considered removed from the system.

The inclusion of parasite mortality due to host mortality results in a quadratic term in equation 4.6. This quadratic term in the rate of parasite mortality arises because host mortality is linearly dependent on x, with all x parasites being killed when their host is killed (see Anderson and May, 1978, page 225). In the following calculations, I abbreviate the denominator of the type II functional response as $\phi = 1 + T\left[N_i(\gamma_i + \sigma_i \bar{P}_i) + N_j(\gamma_j + \sigma_j)\bar{P}_j)\right]$. Equation 4.6 can then be simplified to:

$$\frac{dP_i}{dt} = \beta L N_i - \mu P_i - \frac{C N_i \gamma_i}{\phi} \sum_{x=0}^{\infty} \left\{ q_i(x) x \right\} + \frac{C N_i \sigma_i}{\phi} \sum_{x=0}^{\infty} \left\{ q_i(x) x^2 \right\}$$

$$+ \alpha N_i \sum_{x=0}^{\infty} \left\{ q_i(x) x^2 \right\}$$

$$= \beta L N_i - \mu P_i - \frac{C N_i \gamma_i}{\phi} \sum_{x=0}^{\infty} \left\{ q_i(x) x \right\} - \left(\frac{C N_i \sigma_i}{\phi} + \alpha_i N_i \right) \sum_{x=0}^{\infty} \left\{ q_i(x) x^2 \right\}$$

$$= \beta L N_i - \mu P_i - \frac{C N_i \gamma_i \bar{P}_i}{\phi} - N_i \left(\frac{C \sigma_i}{\phi} + \alpha_i \right) \left(\frac{\bar{P}_i^2}{k} + \bar{P}_i \right), \tag{4.7}$$

where $\sum_{x=0}^{\infty} \{ q_i(x) x^2 \} = E[x^2] = \bar{P}_i^2/k + \bar{P}_i$, and k is the overdispersion parameter for the negative binomial distribution (Anderson and May, 1978).

Equation 4.7 gives the change in the total number of parasites on all hosts of species i, but it may be more ecologically relevant to consider the mean number of parasites per host. The mean number of parasites per host species i is $\bar{P}_i = P_i/N_i$. The rate of change in the mean number of parasites per host species i can be computed using the chain rule:

$$\frac{d\bar{P}_i}{dt} = \frac{d}{dt}\frac{P_i}{N_i}$$

$$= \frac{1}{N_i}\frac{dP_i}{dt} - \frac{P_i}{N_i^2}\frac{dN_i}{dt}$$

$$= \beta L - \mu\bar{P}_i - \frac{C\gamma_i\bar{P}_i}{\phi} - \left(\frac{C\sigma_i}{\phi} + \alpha_i\right)\left(\frac{\bar{P}_i^2}{k} + \bar{P}_i\right)$$

$$- \bar{P}_i\left[-\frac{C\gamma_i}{\phi} - \frac{C\sigma_i\bar{P}_i}{\phi} - \alpha_i\bar{P}_i\right]$$

$$= \beta L - \mu\bar{P}_i - \left(\frac{C\sigma_i}{\phi} + \alpha_i\right)\left(\bar{P}_i^2\left(\frac{1-k}{k}\right) + \bar{P}_i\right). \qquad (4.8)$$

The equation for the mean number of parasites per host species j is the same as equation 4.8, with subscripts i and j reversed. I assumed the same overdispersion parameter for both host species.

4.3.3 Parameterization

The host-parasite model described by equations 4.5 and 4.8 could be applied to any pair of host species that share a common parasite and a common predator. I developed the model to investigate whether parasite-mediated changes to predation could offset direct effects of sea lice on chum salmon. When possible, parameter values for salmon and sea lice were drawn from the literature (Table 4.1). For lesser-known parameters, I investigated the sensitivity of model output to a biologically-reasonable range of parameter values.

The functional response required estimates for handling time, capture rates

and per-parasite increases in capture rates. Handling time includes the time taken to consume and digest prey and is therefore related to the size of the prey item. Juvenile pink and chum salmon are similar in size during their migration, so I assumed the same handling time of $T = 1$ day for both species. This estimate was based on our own observations and previous group predation experiments indicating individual coho salmon consume approximately one juvenile salmon per day (Krkošek et al., 2011a).

The difference between host species in capture rates and per-parasite increases in capture rates dictates how predation rates will change with infestation. The base capture rate for pink salmon was set at $\gamma_p = 3.40 \times 10^{-6}$ day^{-1} based on estimates of juvenile pink salmon mortality in the absence of sea lice as calculated by Krkošek et al. (2011a). I assumed that the base capture rate was greater for pink salmon than chum salmon based on experiments in ocean enclosures suggesting that coho salmon prefer to consume pink salmon, even when chum salmon are more abundant and smaller (Hargreaves and LeBrasseur, 1985). The base capture rate for chum salmon was set at $\gamma_c = 2.72 \times 10^{-6}$ day^{-1}, 80% that of pink salmon. This estimate of preference is likely conservative based on the strong preference for pinks found by Hargreaves and LeBrasseur (1985).

Multiple experiments suggest that juvenile pink and chum salmon become easier for predators to catch as the number of attached motile sea lice increases (Krkošek et al., 2011a). As prey become easier to catch, coho salmon may more strongly express their preference for pink salmon (Hargreaves and LeBrasseur, 1985). Therefore, I assume that the per-parasite increase in capture rate is greater for pink salmon ($\sigma_p = 5 \times 10^{-4}$; Krkošek et al., 2011a) than for chum salmon ($\sigma_c = 5 \times 10^{-5}$). I investigated the sensitivity of juvenile salmon survival to different ratios of σ_c/σ_p. In the following chapter of this thesis, I empirically determine the relationship between σ_c and σ_p.

Attachment rates of sea lice to juvenile salmon hosts depend on the density of sea lice originating from ambient and farmed sources (Krkošek et al., 2006a), and will likely vary from year to year with environmental conditions and farm activity. As a base-value, I consider $\beta L = 0.05$ parasites$\cdot$(host$\cdot$day)$^{-1}$ (Krkošek et al., 2011a) and consider the sensitivity of model output to a range of infestation pressures, from low to high. I define low infestation pressure as the rate of attachment and survival to the motile stage of sea lice from ambient sources only ($0 < \beta L \leq 0.001$) as found in studies of juvenile salmon and sea lice in the Broughton Archipelago (Krkošek et al., 2006a). Moderate infestation pressure includes sea lice from farmed salmon where farms have relatively low levels of infestation (≤ 3 sea lice per farmed salmon) or migration routes are distant from point sources of sea lice at salmon farms ($0.001 < \beta L \leq 0.2$). Finally, high infestation pressure refers to periods of infestations of more than three sea lice per farmed salmon for farms directly along migration routes of wild juvenile salmon ($\beta L > 0.2$; Krkošek et al., 2006a).

The rates of direct parasite-induced host mortality ($\alpha = 0.02$) and natural parasite mortality ($\mu = 0.24$) were taken from field-based experiments on the survival of juvenile pink and chum salmon infested with sea lice (Krkošek et al., 2006a, 2009). These rates were assumed to be the same for pink and chum salmon hosts because differences between host species were not significant in experiments (Krkošek et al., 2009).

To analyze equations 4.5 and 4.8, I had to specify the probability density function for the number of sea lice per host, $q_i(x)$ and $q_j(x)$ where x is the number of parasites and subscripts i and j indicate that these distributions may differ between host species. Sea lice are generally over-dispersed on their hosts, and so I assumed that x was a negative binomial random variable with dispersion parameter $k = 1.199$. The value of k was the same for pink and chum salmon, as determined from the dataset used to calculate the wild sea

louse covariate in the chum salmon population analysis.

The survival of juvenile pink and chum salmon was calculated by numerically solving equations 4.5 and 4.8 using R (R Development Core Team, 2014) and the package *deSolve* (Soetaert et al., 2010). I assumed an initial population of $N_i(0) = N_j(0) = 10^6$ salmon that leave a river with $\bar{P}_i(0) = \bar{P}_j(0) = 0$ sea lice per host. The dynamics were simulated over the first 90 days of the juvenile salmon migration. This brief window was chosen because pink and chum salmon are the predominant prey of coho salmon starting when coho salmon follow the pink and chum salmon migration out of rivers into the nearshore marine environment and ending 6-8 weeks later when the prey outgrow their predator (Groot and Margolis, 1991). During this time, pink and chum salmon are also most susceptible to the effects of sea louse infestation (Brauner et al., 2012).

4.3.4 Results

The multi-prey type II functional response predicted higher predation rates on the preferred prey - pink salmon (figure 4.3a). Pink salmon made up proportionally more of the predators' diet as parasite abundance increased because the per-parasite increase in attack rates of pink salmon was greater than for chum salmon. Overall predation rates on chum salmon therefore declined with increasing number of parasites when prey were abundant (figure 4.3b). However, at low prey abundance, predation rates were not in the saturation region of the type II functional response and parasites increased predation rates on both pink and chum salmon.

These changes in predation rates with the number of parasites were reflected in the population dynamics of pink and chum salmon and associated sea lice. The survival of chum salmon was greater than the survival of pink salmon with

Table 4.1: Parameters, symbols, and units used in the functional response and host-parasite population dynamics.

Description	Symbol	Base value & units*	Source
Abundance of coho salmon predators	C	5000 predator	[1]
Handling time	T	1 day	[1]
Attack rate	γ_p	3.40×10^{-6} (predator $\cdot$ day)$^{-1}$	[1]
	γ_c	2.72×10^{-6} (predator $\cdot$ day)$^{-1}$	[1,2]
Per-parasite increase in attack rate	σ_p	5×10^{-4} hosts $\cdot$ (predator $\cdot$ parasite $\cdot$ day)$^{-1}$	[1]
	σ_c	5×10^{-5} hosts $\cdot$ (predator $\cdot$ parasite $\cdot$ day)$^{-1}$	†
Rate of parasite-induced host mortality	α	0.02 hosts $\cdot$ (parasite $\cdot$ day)$^{-1}$	[3,4]
Infection pressure	βL	0.05 parasites $\cdot$ (host $\cdot$ day)$^{-1}$	[3]†
Natural mortality rate of sea lice	μ	0.24 day^{-1}	[4]
Dispersion parameter	k	1.199	[5]

*Basic physical dimensions of units are time for days and number of individuals for all other variables.
†Model output investigated under a range of values.
References: [1] Krkošek et al. (2011a), [2] Hargreaves and LeBrasseur (1985), [3] Krkošek et al. (2006a), [4] Krkošek et al. (2009), [5] Peacock et al. (2013).

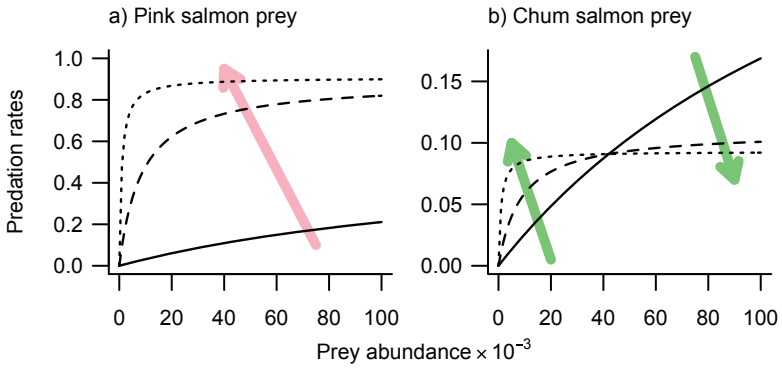

Figure 4.3: Mean predation rates, calculated as $\sum_{x=0}^{\infty} f_{i,x}$, on (a) pink salmon and (b) chum salmon over the number of prey available. I assumed that the number of parasites is the same on both prey species (i.e., $\bar{P}_i = \bar{P}_j$): solid line $\bar{P}_i = 0$, dashed line $\bar{P}_i = 0.2$, dotted line $\bar{P}_i = 2.0$. Light arrows indicate the direction of change in predation rates with increasing parasitism. For chum salmon (b), predation rates decrease with increasing number of parasites at higher prey abundance because predators can more easily capture their preferred prey - pink salmon. The abundance of alternate prey species was assumed to be the same as the focal prey species ($N_j = N_i$), and all other parameters were kept at their base values (table 4.1).

base parameter values (figure 4.4a). The average number of sea lice was also greater on chum salmon than on pink salmon (figure 4.4b) because predators preferentially culled infected pink salmon. Predation rates on pink salmon increased steeply as the number of parasites per host increased at the start of the migration, whereas predation rates on chum salmon decreased initially (figure 4.4c). As parasitized individuals were removed from the population over the course of the migration and juvenile salmon abundance declined, predators were less able to focus on preferred prey and predation rates on pink salmon declined. Near the end of the migration, chum salmon were more abundant, had a higher parasite load and experienced similar predation rates as pink salmon.

The prediction of higher sea louse abundance on chum salmon (figure 4.4b) was supported by data from a long-term monitoring program of sea lice on juvenile salmon in the Broughton Archipelago. I found higher average numbers of copepodid and chalimus sea lice on juvenile chum salmon than on pink salmon caught in the same sample (table S5 of Peacock et al., 2014). Numbers of motile sea lice did not differ significantly between host species, but motiles are known to move amongst hosts in search of mates (Connors et al., 2011) or when their host is attacked by a predator (Connors et al., 2008b).

In the model, the per-parasite increase in attack rates was relatively large compared to the base attack rates. The results were therefore extremely sensitive to the relative per-parasite increase in attack rates for pink and chum salmon. If I assume the per-parasite increases in attack rates are the same for both pink and chum salmon, survival is similar for the two species even though pink salmon are far more likely to be captured in the absence of parasites (figure 4.5a). This is because the difference in overall attack rates between pink and chum salmon gets smaller as the number of parasites increases. However, if the per-parasite increase in attack rate is higher for pink salmon then for

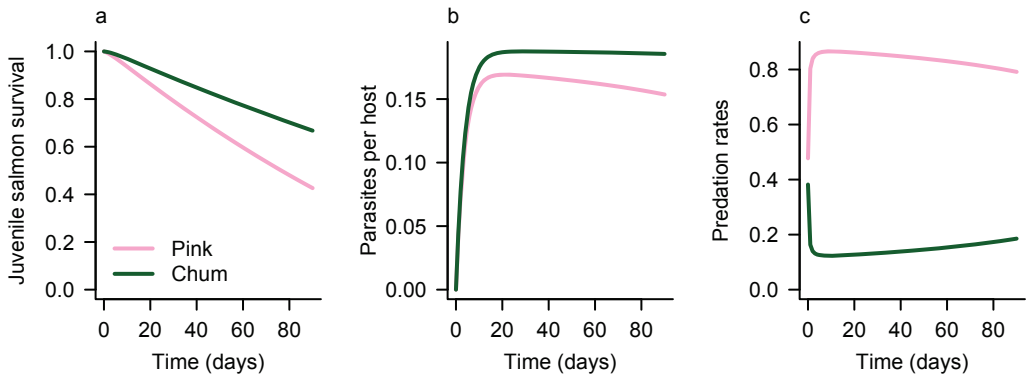

Figure 4.4: (a) Predicted survival of pink salmon (light pink line) and chum salmon (dark green line) over the period of juvenile salmon outmigration ($t = 0 - 90$ days). In the model, survival of pink and chum salmon declined with predation and direct parasite-induced mortality (equation 4.5). (b) The predicted average number of sea lice per pink salmon (light pink line) and per chum salmon (dark green line), which changed with sea louse attachment, natural mortality and host mortality (equation 4.6). (c) Predicted predation rates on pink salmon (light pink line) and on chum salmon (dark green line) over the period of the migration according to equation 4.3. Parameter values are given in table 4.1.

chum salmon, the difference in overall attack rates between pink and chum salmon grows as the number of parasites increases. Therefore, the assumption that predators such as coho salmon will focus predation on pink salmon as they become parasitized is essential for chum salmon survival to increase with infestations.

The population dynamics of juvenile salmon and sea lice were also sensitive to different levels of sea louse infestation pressure. Experimental work in the absence of predators indicates that survival of juvenile salmon declines with as few as one sea louse per fish (Krkošek et al., 2006a; Morton and Routledge, 2005). I might expect that as infestation pressure increases, modelled by an increase in the attachment rate, βL, direct parasite-induced mortality may become important and survival of chum salmon would decline. I found that at low infestation pressure, survival of chum salmon was greater than the survival of pink salmon. At moderate infestation pressure and base parameter values (table 4.1), survival of pink salmon declined steeply as predation on pink salmon increased but the survival of chum salmon actually increased due to reduced predation (figure 4.5b). This increase in chum survival was due to lower predation pressure at the beginning of the migration, when prey were still abundant and a reduction in predation rate had a large impact on the number of chum salmon consumed. As infestation pressure increased above the base value of $\beta L = 0.05$, survival of chum salmon declined to match that of pink salmon for two reasons: steeper declines in survival of pink salmon meant a low abundance of preferred prey, resulting in higher predation on chum salmon earlier in the migration and direct parasite-induced mortality of chum salmon became increasingly important at high infestation pressure. This highlights the sensitivity of these results to infestation pressure, with the survival of chum salmon increasing only at moderate infestation intensity.

All of the results presented thus far refer to the model in which attack rates

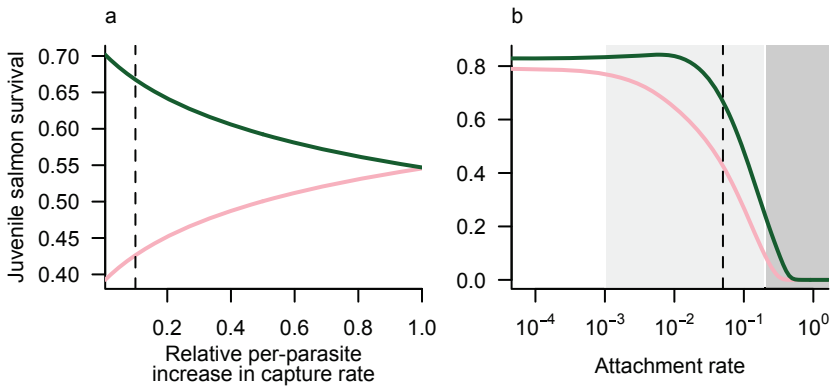

Figure 4.5: Predicted survival of pink salmon (light pink lines) and of chum salmon (dark green lines) as a function of (a) the ratio of per-parasite increase in attack rate for chum salmon over that for pink salmon, and (b) infestation pressure (i.e., attachment rate βL). Low, moderate and high infestation pressure are indicated by light, medium and dark grey shading in (b). Base parameter values are indicated by vertical dashed lines (Table 4.1).

and host mortality increase linearly with the number of parasites. Some studies suggest that there may be thresholds for impact depending on the number of parasites (Wells et al., 2006) and the size of the hosts (Brauner et al., 2012). When the linear assumption was relaxed to include sigmoidal responses in attack rates and host mortality rates to the number of parasites, the results were largely unchanged; chum salmon survival and associated sea-louse abundance were consistently higher than those for pink salmon.

4.4 Discussion

Parasites are generally considered a villainous guild, causing host morbidity and mortality. However, I hypothesized that in certain situations, the net effect of parasitism on hosts may be nullified or possibly positive when considering indirect effects of parasites on predator-prey interactions within a multi-host community. For communities of juvenile salmon, the physiological impact of

sea louse parasitism has been well-studied (Brauner et al., 2012), and both pink and chum salmon in captivity show decreased survival with as few as one attached sea louse (Krkošek et al., 2009; Morton and Routledge, 2005). Multi-year studies of pink salmon population abundance data indicate that the net impact of sea louse infestations on pink salmon is likely negative (Krkošek et al., 2011b), suggesting that direct parasite-induced mortality translates to reduced productivity of affected populations for pink salmon. However, this analysis of chum salmon population abundance data suggests the existence of an ecological mechanism that confers resilience to chum salmon populations despite the direct effects of infestations on host individuals. Indirect ecological effects of sea lice on salmon predator-prey interactions may be a key determinant of host survival. Sea louse parasites are known to increase the susceptibility of juvenile pink and chum salmon to predation (Krkošek et al., 2011a) and coho predators prefer to consume pink salmon over chum salmon (Hargreaves and LeBrasseur, 1985). If infestations intensify predation on pink salmon, this may partially release chum salmon from predation, offsetting direct mortality costs of parasites on chum salmon.

Using a host-macroparasite model, I evaluated the conditions under which parasite-mediated changes to predation may offset direct impacts of parasites on host populations. I considered predation by a generalist predator (coho salmon) on two prey populations (pink and chum salmon) that share a common parasite (sea lice). The model built upon previous experimental evidence that coho salmon predators exhibit a strong preference for pink salmon over chum salmon, even when pink salmon are larger (i.e., harder to catch) and less abundant than chum salmon (Hargreaves and LeBrasseur, 1985). The model allowed parasites to cause an increase in predation rates that was larger for the preferred prey, which can reduce predation on the other less-desirable prey in the saturation region of the type II functional response. The less-desirable prey

had higher survival in regimes of moderate-intensity infestations. However, if the intensity of infestations was high enough, the negative direct impact of parasites overwhelmed any gains from reduced predation on the less-desirable prey (figure 4.5c). Therefore, the potential indirect benefits that parasites may confer to hosts are likely constrained to a limited range of infestation levels.

Interestingly, the model predicted that the prey population with higher survival also had the higher parasite abundance. I understand this counterintuitive feature as follows: predation was focused on the preferred prey species and on parasitized individuals. This group of prey - preferred and parasitized - had the lowest survival. Less-desirable prey species with parasites had higher survival and therefore the mean number of parasites per host was higher for less-desirable prey (figure 4.3). In this case, the survival of hosts influenced their parasite load, rather than the parasite load influencing host survival, a reverse direction of causality than is usually assumed. This highlights the interdependence of parasite and host survival, and that host survival is not necessarily negatively related to the number of parasites.

The effects of sea lice on juvenile chum salmon survival are sensitive to the level of infestation pressure. An increase in chum salmon survival with sea lice only occurred over a moderate range of infestation levels. However, studies of infestation pressure from salmon farms in the Broughton Archipelago suggest that this may be the range of infestations that have occurred over the past decade (Krkošek et al., 2006a, 2005a). At these moderate levels of infestation, pink salmon may experience significant mortality because parasites increase predation and thus mortality of pink salmon. Meanwhile, chum salmon populations may incur lower overall mortality because the redirection of predation mortality onto pink salmon caused by sea lice compensates or exceeds the direct impact of sea lice on chum salmon mortality. At high infestation levels, however, the model predicts a decline in chum salmon survival due to over-

whelming direct parasite-induced mortality. The sensitivity of both pink and chum salmon survival to high infestation levels in the model highlights the potential for decline in both pink and chum salmon populations should epizootics occur at sufficiently high levels. From a conservation perspective, it is therefore important to reduce abundances of sea lice in coastal regions shared by wild salmon and aquaculture.

The form of the functional response may influence the model outcome. I chose a type II functional response that has been used previously for salmon and sea lice (Krkošek et al., 2011a) and has been recommended more generally for piscivorous fishes that actively pursue prey (Moustahfid et al., 2010). Coho salmon are active predators that prey on schooling pink and chum salmon. Nonetheless, different functional responses may alter the outcome of predator-prey interactions. For example, a type III functional response has decreasing attack rates when prey abundance is low because prey may be better able to seek refuge or the predator may shift focus to more abundant prey species (Moustahfid et al., 2010). This may result in much higher predation on chum salmon if they outnumber the alternate prey, even if the alternate prey are preferred.

Non-linear changes in host attack rates and survival with sea lice are also worth consideration. Studies of the physiological impact of sea louse infestation on salmonid smolts indicate thresholds in louse abundance below which the impact is negligible (Wells et al., 2006). However, for studies of juvenile pink and chum salmon, the presence of thresholds depends on the size of the host (Brauner et al., 2012). For salmon less than 0.5 grams in weight, a single sea louse can reduce swimming performance (Nendick et al., 2011), trigger measurable physiological changes (Sackville et al., 2011) and cause mortality (Morton and Routledge, 2005). However, as juvenile salmon grow and develop scales, they can survive low levels of infestation with little effect. This study

focuses on juvenile salmon in the first 2-3 months of their migration when they are the primary prey for coho salmon predators. During this period they are mostly below the 0.5 gram threshold (Brauner et al., 2012). In the absence of more detailed studies of non-linear effects of sea lice on such small hosts, I continued with the assumption of linear increases in host mortality and attack rates with the number of attached sea lice.

Coho salmon are major predators of juvenile pink and chum salmon (Groot and Margolis, 1991; Parker, 1971), but also prey upon other species of fish (e.g., Pacific herring, sand lance) and zooplankton (Brodeur, 1991). I ignore these other prey species in my analysis and focus on predation rates on pink and chum salmon. Including additional prey species would not affect the results unless the alternative prey were both more numerous and preferred by coho salmon. Coho salmon often follow pink and chum salmon out of the rivers and pink and chum salmon dominate the coastal ecosystem over the subsequent weeks (Groot and Margolis, 1991). Therefore, pink and chum salmon are likely the primary prey for coho salmon until they outgrow their predators 6-8 weeks later (Groot and Margolis, 1991).

There may be explanations for my inability to detect an effect of sea lice on the productivity of chum salmon other than parasite-mediated predation. First, chum salmon often return to larger geographic areas than specific rivers, potentially blurring the differences in survival between river populations exposed and not exposed to sea lice from farmed salmon. Chum salmon that emerge from a river outside of the region of salmon farming will not pass by salmon farms as susceptible juveniles, but may return to a river within the region of salmon farming. High survival of such fish may confound a decline in survival of chum salmon migrating past salmon farms. Conservation Units, defined by Fisheries and Oceans Canada, include river populations with similar genetic and life-history traits, suggesting gene-flow among river populations within

a Conservation Unit (Holtby and Ciruna, 2007). The Conservation Unit for chum salmon in the Broughton Archipelago includes river populations exposed and unexposed to salmon farming. However, the Conservation Unit for pink salmon in the Broughton Archipelago also includes both river populations exposed and unexposed to salmon farms (Holtby and Ciruna, 2007), and yet the correlation between sea louse infestations on salmon farms and pink salmon survival in the Broughton Archipelago was significant (Krkošek et al., 2011b). However, if the effect size for chum salmon were smaller than for pink salmon, this movement of spawners may bolster survival of exposed river populations just enough to conceal any real impact of sea lice on chum salmon survival.

Second, inaccuracies in the chum salmon data may introduce uncertainty, making it harder to detect a statistically significant effect of sea lice. Fisheries and Oceans Canada aims to enumerate as many salmon species as possible while minimizing the cost of stock assessment programs. Chum salmon may be counted at sub-optimal times because they are usually the last species to return within the season. Observation error is likely large due to the nature of enumeration methods (e.g., helicopter flights, stream walks). Return estimates do not include catch of chum salmon in First Nation fisheries or unreported catch of chum salmon in fisheries targeting other species (English et al., 2012). Variable age-at-return in chum salmon introduces the potential for additional error that is not present in analyses of pink salmon population, which have a consistent two-year lifecycle. While the data on age-at-return for populations exposed to salmon farms were limited, the results were robust to different imputation methods for missing age-at-return. As a base case, I imputed missing age-at-return data with a constant 4-year age-at-return as this assumption minimizes spurious autocorrelation and cross-correlations between time series for different river populations (Pyper et al., 2002). Although these sensitivity analyses and a power analysis indicate the results are robust, one cannot

forget that errors accumulate across the different types of data used in the spawner-recruit analysis.

The role that parasites are traditionally cast in is changing as we uncover the influence of parasites on competitive or predator-prey interactions in the host community (Hatcher et al., 2012). Thinking beyond the direct impact of parasites on hosts is particularly important in the context of species conservation, where multi-host dynamics are often a necessary ingredient for disease to threaten biodiversity (i.e., reservoir hosts; De Castro and Bolker, 2005). While results of theoretical models such as the one I present here are sensitive to certain parameters and assumptions, they provide valuable insight into host-parasite dynamics under different ecological conditions. My results indicate that parasite effects on predator-prey interactions in multi-host dynamics may sometimes protect, or even enhance, the persistence of some host species, but this occurs at the expense of other species.

Chapter 5

Parasitism and food web dynamics of juvenile Pacific salmon

5.1 Introduction

Predators kill their prey, whereas the effects of parasites on hosts are often sublethal. Therefore, the way in which parasites affect predator-prey interactions in food webs can have implications for host population dynamics and communities (Hatcher et al., 2014, 2012). In some cases, parasite-induced changes in host behaviour that make prey more susceptible to predation may have evolved to facilitate transmission to a definitive host (e.g., Carney, 1969; Lafferty and Morris, 1996). However, there is growing recognition that parasites can influ-

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ence food web dynamics of host populations more broadly (Hatcher et al., 2006, 2014). Parasite-induced changes to host behaviour may increase predation by non-host species (e.g., Marriott et al., 1989), adding to direct effects of parasites on hosts. Alternatively, if parasites reduce predation, parasite-mediated relief from predation may offset direct effects of parasites on hosts. Whether parasites increase or decrease predation mortality of hosts therefore becomes a key question in determining the net impact of parasites on host populations.

Most theoretical models of predation and parasitism assume that predators will prefer parasitized prey because they may be easier to detect and catch (e.g., Ives and Murray, 1997; Kisdi et al., 2013; Lafferty, 1992; Toor and Best, 2015). Empirical evidence of this has been reported for red grouse infested with nematodes (parasitized individuals appear to emit more scent and are more easily detected Hudson et al., 1992) and selective predation of snowshoe hares infested with nematodes (Murray et al., 1997). Selective predation on parasitized prey can have implications for population dynamics, destabilizing predator-prey cycles (Ives and Murray, 1997) and potentially improving the health of host populations by reducing the overall prevalence of parasites (Hudson et al., 1992; Packer et al., 2003). There are numerous studies using mathematical models to explore how predation and parasitism might interact (e.g., Hatcher et al., 2014; Lafferty, 1992; Packer et al., 2003; Peacock et al., 2014), but connections between theory and data are rare, particularly for multi-host systems.

In this chapter, I experimentally test for evidence of parasite-mediated changes to the food web dynamics of juvenile salmon. In the previous chapter, I found that chum salmon populations did not decline with increasing sea louse infestations on out-migrating juveniles, unlike pink and coho salmon populations (Krkošek et al., 2011b). A mathematical model tailored to the system suggested that if a predator preference for pink salmon (Hargreaves and Le-

Brasseur, 1985) were amplified by infestations, then sea lice may reduce predation on chum salmon, offsetting the direct effects of parasitism and dampening the population-level impact. However, empirical evidence of sea lice changing predation dynamics in mixed-species schools is needed to substantiate this hypothesis.

In this chapter, I build upon the theoretical modelling in chapter 4 and experimentally test for evidence of parasite-mediated changes to the food web dynamics of juvenile salmon. The results further our understanding of how sea lice affect juvenile Pacific salmon and are an example of the unexpected outcomes of parasitism in multi-host systems.

5.2 A general model for parasite-mediated predation

I begin with a simple model for parasite-mediated predation and build upon previous simulation work in chapter 4 by analytically determining the condition under which predation on chum salmon would be expected to decline with parasites. I then frame this condition in the context of predator preference and form the hypotheses to be tested in experiments.

5.2.1 The functional response

A type II functional response (Holling, 1959) describes an increase in predation rate with increasing prey abundance until a saturation point where predators are limited by the time it takes to handle and digest prey. The functional response of a generalist predator depends on both the abundance of alternate prey species and the preference the predator displays for each species. The

instananeous rate of prey consumption by a single generalist predator on prey species N_i in the presence of alternate prey N_j is described by

$$f_i = \frac{a_i N_i}{1 + T_h(a_i N_i + a_j N_j)}, \tag{5.1}$$

where a_i and a_j are the rates of successful attack resulting in capture, henceforth referred to as attack rates, and T_h is the handling time for predators to consume and digest prey (Holling, 1959; Lawton et al., 1974). This type of functional response is common in piscivorous fishes such as coho salmon (Moustahfid et al., 2010), and has been applied previously to juvenile salmon (Krkošek et al., 2011a; Peacock et al., 2014).

Attack rates may differ among prey species, in which case the predator is said to have a preference for the species with the higher attack rate (Chesson, 1983). Due to previous evidence of selective predation on pink salmon (Hargreaves and LeBrasseur, 1985), I chose the attack rate on pink salmon to be greater than the attack rate on chum salmon. Handling time may also differ between prey species, but here I assumed that it was the same for both prey species because juvenile pink and chum salmon are morphologically and behaviourally similar. Throughout the chapter, I use subscripts i and j to denote different prey species, and subscripts c and p to refer specifically to chum and pink salmon.

I included an effect of parasites on predation susceptibility of prey by incorporating a linear increase in the attack rate with the mean number of parasites per prey, x:

$$a_i = \gamma_i(1 + \omega_i x), \tag{5.2}$$

where γ_i is the base attack rate in the absence of parasites and ω_i is the per-parasite proportional increase in the attack rate (Krkošek et al., 2011a; Peacock et al., 2014). (This notation differs slightly from the previous chapter,

where the per-parasite increase was defined as σ_i such that $\omega_i \gamma_i = \sigma_i$.) The impact of parasites on host susceptibility to predation is likely non-linear, but a linear approximation is acceptable for low to moderate parasite abundances. I assume that the number of parasites is the same on both prey species because no significant difference in infection levels between juvenile pink and chum salmon has been reported in the wild (Patanasatienkul et al., 2013).

To determine the conditions under which predation on chum salmon might decline with parasites, I consider how the per-capita predation rate, $g_c = f_c/N_c$, changes with respect to the number of parasites. Inserting equation 5.2 into equation 5.1, and solving $dg_c/dx < 0$ leads to the following condition:

$$\frac{\omega_c}{\omega_p} < \frac{T_h \gamma_p N_p}{1 + T_h \gamma_p N_p}. \tag{5.3}$$

In words, equation 5.3 indicates that in order to observe a decline in predation on chum salmon with increasing number of parasites, the per-parasite increase in predation on chum salmon must be less than the per-parasite increase in predation on pink salmon (i.e., $\omega_c < \omega_p$). Under this condition, the attack rate on pink salmon would increase more quickly with the number of parasites than the attack rate on chum salmon. More specifically, equation 5.3 says that the ratio of ω_c/ω_p must be less than the proportion of time that predators would spend consuming pink salmon if there were no chum salmon present and no parasites. The more pink salmon there are in the environment (i.e., $N_p \to \infty$), the longer it takes predators to handle prey (i.e., $T_h \to \infty$), or the higher the base attack rate on pink salmon (i.e., $\gamma_p \to \infty$), the more occupied predators will be with their preferred prey, therefore requiring less of a difference in per-parasite increases in attack rates to see a decline in predation on chum salmon.

5.2.2 Prey preference

Rivers in coastal British Columbia see hundreds to millions of pink and chum salmon returning to spawn each fall (Fisheries and Oceans Canada, 2011b), and the following spring their offspring emerge from the gravel and migrate by the millions through coastal waters for period of two to three months (Heard, 1991). It is therefore reasonable to assume that during this time, N_p is large, and if γ_p and T_h are not too small (Krkošek et al., 2011a) then I can approximate equation 5.3 by $\omega_c < \omega_p$ (I revisit this approximation later in light of my experimental results). This approximate condition can be stated in terms of the change in predators' preference for pink salmon with parasites. The preference for prey type j is defined as the probability that prey type j will be consumed next given equal availability of all prey types, and can be calculated as $\alpha_j = a_j / \sum_i a_i$ (Chesson, 1983). Values of $\alpha_j > 0.5$ indicate a preference for species j. Incorporating the effect of parasites on the attack rate (equation 5.2), the preference for pink salmon when prey are infested with x lice is:

$$\alpha = \frac{\gamma_p(1+\omega_p x)}{\gamma_c(1+\omega_c x)+\gamma_p(1+\omega_p x)}. \tag{5.4}$$

The rate of change in preference with respect to the number of parasites is

$$\frac{d\alpha}{dx} = \frac{\gamma_c\gamma_p(\omega_p-\omega_c)}{[\gamma_c(1+\omega_c x)+\gamma_p(1+\omega_p x)]^2}. \tag{5.5}$$

Therefore, the condition that $\omega_c < \omega_p$ is equivalent to $d\alpha/dx > 0$, i.e., that the preference for pink salmon increases with the number of parasites. In the following section, I describe a series of field-based experiments designed to test the hypothesis that predator preference for pink salmon increases with the number of parasites, and that predation mortality of chum salmon declines with infestations.

5.3 Empirical evidence from a juvenile salmon food web

5.3.1 Experimental methods

I conducted a series of field-based predation experiments in the Broughton Archipelago, Canada (126.5° W, 50.8° N) in the springs of 2013 and 2014. The goals of these experiments were (1) to test for species-selective predation by coho salmon on pink salmon, (2) to test for selective predation on parasitized prey and (3) to determine if preference for pink salmon increases with parasitized prey.

I collected coho predators and pink and chum prey by beach seine and transported them to a floating research facility where they were housed in flow-through ocean enclosures until being used in experiments. Two days prior to an experiment, I haphazardly selected the required number of coho predators and transferred them to a separate ocean enclosure where they were deprived of food until experiments.

Each experiment consisted of paired trials, one with pink and chum prey that were infested with sea lice and one with uninfested prey. Prior to an experiment, I sorted prey into lousy and clean infestation categories by examining each fish in a clear plastic bag with seawater using a 16 × hand lens (Krkošek et al., 2005b). I classified prey as lousy if they were infested with at least one *L. salmonis* sea louse of a chalimus II or motile stage, and clean if they had no sea lice of any stage or species and no signs of louse-induced morbidity. I size-matched pink and chum within and between infestation categories to minimize the impact of prey size as a confounding factor (Hargreaves and LeBrasseur, 1986). There may be other factors that increase the susceptibility of certain

individuals to infection and would thus be confounded with sea louse infestation. This is an unavoidable consequence of using naturally-infested prey. However, research suggests that aggregation of sea lice among hosts is likely due to small-scale patchiness in the spatial distribution of infectious parasites and there is limited evidence for selection among host individuals by sea lice (Murray, 2002).

I transferred equal numbers of sorted pink and chum prey to one side of a divided experimental net pen. There were two experimental net pens, one with clean prey and one with lousy prey, and I randomly assigned which of the two experimental net pens housed the lousy trial. The food-deprived coho predators were then transferred to the empty side of experimental net pens, and predators and prey acclimatized to the divided experimental net pens for a minimum of four hours and up to a maximum of 20 hours before trials began. The variation in acclimatization period was unavoidable due to the variable time required to collect and sort prey, and the constraint of starting and ended trials during daylight hours only.

Trials began by dropping the divider of the net pen to allow coho predators access to the mixed school of pink and chum prey. For experiments in 2014, a one-hour observation period followed during which I recorded the number of prey successfully captured by coho predators. Trials ran for between 4 and 24 hours; the length of the trial was pre-determined, but varied among experiments depending on the number of predators and prey I had (described below). At the end of trials, I divided the net pens and separated the coho predators from the remaining pink and chum prey. I counted coho predators and returned them to the holding pen. I measured the remaining pink and chum prey and visually inspected them for sea lice and other markings (e.g., scars from lice or predation strikes) using a 16 $\times$ hand lens (Krkošek et al., 2005b) and then released them near their location of capture.

The number of predators, number of prey, and the length of the trials varied depending on how many clean and lousy pink and chum prey I was able to obtain. Lousy prey were often limiting, as the natural prevalence of sea lice was low throughout the study. When trials were run with less than 100 prey, the lengths of the trials and/or number of predators were predetermined with the goal of having approximately one-third of the available prey consumed based on consumption rates in previous trials. The variable number of predators, prey and length of the trails did not affect the preference for pink salmon.

I ran a total of 27 experiments, all but one of which consisted of paired trials with lousy and clean prey. Within these experiments, I performed eight control trials without coho salmon predators. The objectives of control trials were (1) to test if mortality in the absence of predation due to handling or sea lice was substantial or different between prey species, (2) to test if size-matching between pink and chum salmon prey was effective, and (3) to assess observation error in counting prey in and out of net pens. Any difference between prey species in mortality from sources other than predation may have confounded a predator preference, as I did observe coho consuming moribund prey.

5.3.2 Data analysis

Assuming the only substantial mortality of pink and chum prey in experiments was due to predation (I verified this assumption in the control experiments), the rate of change in prey species i throughout an experiment can be described by the functional response introduced in equation 5.1,

$$\frac{dN_i(t)}{dt} = -C\frac{a_i N_i(t)}{1 + T_h(a_i N_i(t) + a_j N_j(t))}, \tag{5.6}$$

where $N_i(t)$ and $N_j(t)$ are the number of prey species i and j available at time t and C is the number of coho predators in the experiment. Although I did not have a direct measure of the attack rates, I can solve for a_i as a function of known variables by integrating the coupled equations for the change in prey, $dN_i(t)/dt$ and $dN_j(t)/dt$ (Lawton et al., 1974), obtaining

$$N_i(t) = N_i(0) \exp\left[-a_i \left(tC - T_h \left[(N_i(0) - N_i(t)) + (N_j(0) - N_j(t))\right]\right)\right], \quad (5.7)$$

where $N_i(0)$ is the number of prey species i at the beginning of the trial and $N_i(t)$ is the number of prey species i remaining at the end of the trial. A similar equation, with i's and j's exchanged, results for the remaining prey $N_j(t)$. Solving equation 5.7 for a_p and a_c, the attack rates on pink and chum, and taking the ratio of $a_p/(a_c + a_p)$, I arrive at an equation for the preference for pink salmon (Chesson, 1983):

$$\alpha = \log\left(\frac{N_p(t)}{N_p(0)}\right) \Big/ \log\left(\frac{N_p(t)N_c(t)}{N_p(0)N_c(0)}\right). \quad (5.8)$$

I calculated α for each experimental trial. Values of α are constrained between zero and one, where $\alpha > 0.5$ indicates selective predation on pink salmon and $\alpha < 0.5$ indicates selective predation on chum salmon.

To determine if preference for pink salmon prey (α) increased when prey were parasitized, I fit a linear mixed-effects model with a fixed-effect for treatment (lousy or clean) and random effects that accounted for for experiment number and coho group. The random effects accounted for possible variation among experiments conducted on different days due to weather, the age and size of prey, etc. and for possible variation among the coho groups collected from different areas at different times. I applied a logit transformation to α, which satisfied the assumptions of the linear model.

The predation scars on surviving prey and changes in louse abundances during experiments also carried information about predator preference. I estimated the proportion of remaining prey that had predator scars using a binomial generalized linear mixed-effects model (GLMM; logit link), with fixed effects for prey species and treatment (lousy or clean trial) plus an interaction term that allowed for a disproportionate effect of sea lice on one species. To account for non-independence of observations, I included nested random effects for trial within experiment number within coho group. I also included an observation-level random effect to account for overdispersion in the proportion of remaining prey with predator scars (Warton and Hui, 2011).

I estimated the number of sea lice on prey using a Poisson GLMM (log link) with fixed effects for prey species and a factor indicating whether the data refer to before or after experiments, plus an interaction term, and a random effect for experiment number nested within coho group. Although sea lice are often overdispersed on hosts (Murray, 2002), my group of hosts had been sorted and had low but non-zero infestation intensity that was better represented by the Poisson distirbution.

Finally, I estimated the predation mortality of both pink and chum as a function of the mean number of lice per pink or chum at the beginning of each trial. The proportion of available prey that were consumed was estimated using a binomial GLMM (logit link function) with fixed effects for prey species and the mean number of lice per fish, plus an interaction term that allowed for a disproportionate effect of sea lice on predation mortality of one species. I also included nested random effects for trial within experiment number within coho group, accounting for the non-independence of pink and chum predation mortality estimates from the same trial/experiment/coho group. Again, I included an observation-level random effect to deal with overdispersion in the proportion of prey consumed by predators (Warton and Hui, 2011).

For each set of models described above I fit all nested models and compared them using AIC_c (table 5.1). In all cases no single model had overwhelming support, so I based my inference on the weighted average prediction of top models comprising 90% of the cumulative Akaike weight (Burnham and Anderson, 2002), thereby accounting for both parameter uncertainty and model uncertainty. I report model-averaged predictions, as opposed to model-averaged parameters, to avoid errors in model-averaged parameters that can result from collinearity among predictors and different methods of averaging parameters (i.e., natural average vs. zero method Grueber et al., 2011). All models were fit in R (R Development Core Team, 2014), using the library `lme4` for mixed-effects models (Bates et al., 2014) and `AICcmodavg` for model averaging (Mazerolle, 2014).

5.3.3 Results

I conducted 45 predation trials and eight control trails, involving a total of 524 different coho predators and 3674 pink and chum prey. The eight control trials without coho predators had no substantial mortality of pink and chum salmon. A total of 911 pink salmon were consumed during predation experiments, while only 564 chum salmon were consumed. The eight control trials without coho predators had no substantial mortality of pink and chum salmon.

Coho predators showed a preference for consuming pink salmon ($\alpha > 0.5$) in 40 out of the 45 predation trials (figure 5.1a). In trials with clean prey, the model-averaged preference was $\alpha_0 = 0.665$ (95% CI: 0.611, 0.715), indicating an overall preference for pink salmon prey over chum salmon (figure 5.1b). There was considerable uncertainty as to whether this preference changed when prey were infested with sea lice. I averaged predictions for preference over models that did and did not include an effect of sea lice as there was no obvious

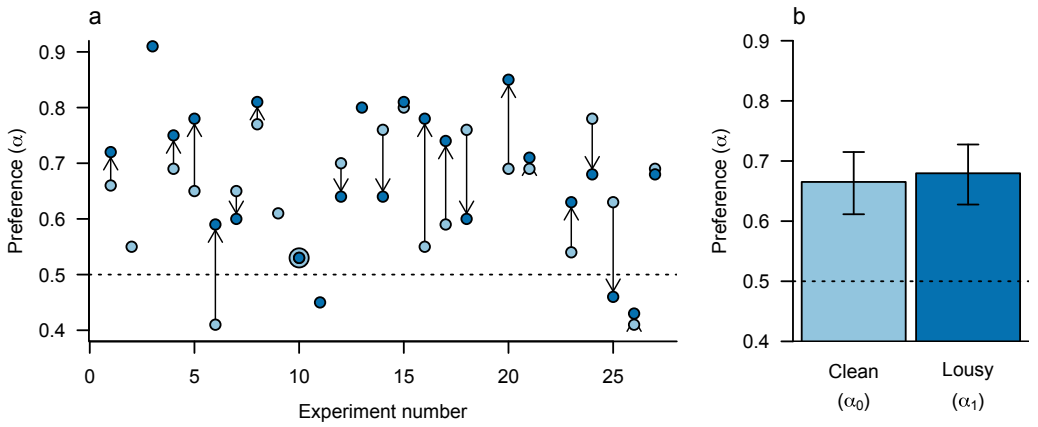


Figure 5.1: (a) A predator preference for pink salmon was evident across experiments, with $\alpha > 0.5$ (dotted line) in 40 out of 45 of the trials. Preference was higher in lousy trials (dark points) than clean trials (light points) in 12 experiments. Arrows indicate the direction of change in preference with sea lice. (b) The model-averaged estimate ($\pm$ 95% CI) of preference was higher in lousy trials than in clean trials, although there was considerable uncertainty in estimates.

support from the data for a single top model (see table 5.1 for model selection statistics and table 5.2 for parameter estimates from the top models). The model averaged estimate of preference for pink salmon increased only slightly in lousy trials to $\alpha_1 = 0.680$ (0.628, 0.727), and confidence intervals for preference in clean trials overlapped the estimate for preference in lousy trials (figure 5.1b).

Fresh predation scars were clearly identified on surviving prey as semi-circular tooth marks, often accompanied by haemorrhaging. The proportion of remaining prey that had predator scars was higher for pink salmon and higher when prey were infested with sea lice (figure 5.2a) with a weak interaction between prey species and lice suggesting that the difference in predation scars between pink and chum salmon was higher when prey were infested (table 5.1 and 5.2). This suggests that the observed species-selective predation on pink salmon (figure 5.1) is not the result of chum salmon escaping predators, but that pink salmon were more likely to be targeted by predators.

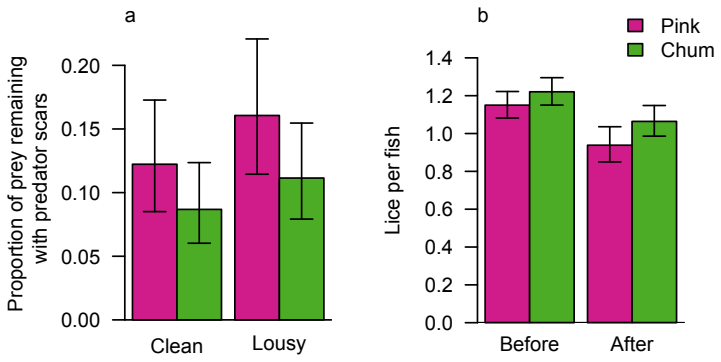


Figure 5.2: (a) The proportion of remaining prey (± 95% CI) that had predator scars was higher for pink salmon than for chum salmon and higher in trials where prey were lousy. (b) The estimated number of lice per fish (± 95% CI) was slightly higher for chum salmon both before and after experiments, but decreased during experiments for both pink and chum.

The number of chalimus- and motile-stage sea lice on prey was lower after experiments (figure 5.2b). Furthermore, at the end of experiments, the number of sea lice on pink salmon was lower than the number of sea lice on chum salmon (figure 5.2b and table 5.2). In control experiments, the number of lice did not differ between prey species or before and after experiments. Therefore the pattern of reduced infestations after predation experiments, with a stronger effect for pink salmon, suggests selective predation of infested prey.

As the analysis of preference would suggest, the predation mortality of chum salmon was lower than the predation mortality of pink salmon (figure5.3a). There was little evidence that sea lice affected the predation mortality of pink and chum salmon differently, although the suite of top models did include an interaction by which the predation mortality of pink salmon increased with the mean sea lice per fish at the start of the trial, but the predation mortality of chum salmon decreased with sea lice (table 5.2). The model-averaged odds ratio, describing the increase in the odds of predation corresponding to an increase in one sea louse per fish, was 1.01 (95% CI: 0.93, 1.09) for pink salmon

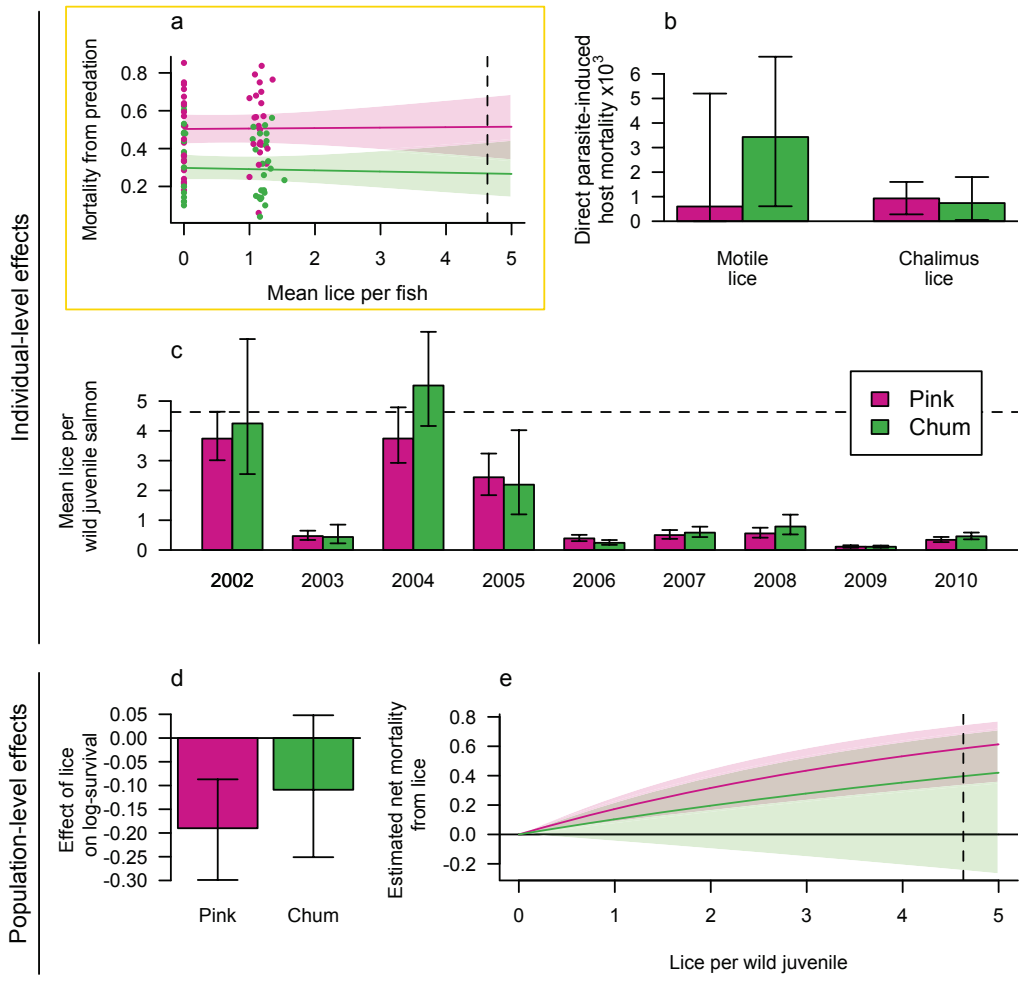


Figure 5.3: (a) Predation mortality over louse abundance* for pink and chum salmon. The x-axis is extended to show louse abundances corresponding to peak epizootics in 2004 (dashed line in panels a, c and e*). (b) Rates of direct parasite-induced mortality per chalimus- and motile-stage louse from a survival analysis of captive juvenile wild salmon (Krkošek et al., 2009). (c) The mean number of sea lice per juvenile wild salmon, estimated from monitoring data (Peacock et al., 2013). (d) The population-level effect of sea lice estimated from spawner-recruit data using a Ricker model: $\log(R/S) = r - bS - cL$, where R are recruits, S are spawners, and L is the mean number of sea lice* per juvenile wild salmon (Peacock et al., 2014, 2013). (e) The predicted mortality due to lice $(1 - \exp(-cL))$ over louse abundance. In all panels, shaded regions and error bars are 95% confidence intervals. *Note: Louse abundance includes chalimus II and motile stages in a, all louse stages in c, d, and e.

Table 5.1: Model selection statistics for analysis of experiments. Only models comprising 90% of the cumulative Akaike weight (cum. w_i) are shown.

Response (dist^{n}, link)	**Predictors***	**K†**	**AIC$_c$‡**	**$\Delta_i^{\S}$**	**w$_i$¶**	**cum. w$_i$**
Preference	Null	4	80.93	0.00	0.588	0.588
(Normal, logit)	Lice	5	81.65	0.71	0.412	1.000
Proportion with	Species + Lice	7	380.84	0.00	0.544	0.544
predator scars	Species $*$ Lice	8	382.85	2.01	0.200	0.744
(Binomial, logit)	Species	6	383.03	2.19	0.182	0.926
Lice per fish	After $*$ Species	6	6742.23	0.00	0.517	0.517
(Poisson, log)	After + Species	5	6742.71	0.48	0.407	0.924
Predation	Species	6	561.74	0.00	0.628	0.628
mortality	Species + Lice	7	563.99	2.25	0.204	0.832
(Binomial, logit)	Species $*$ Lice	8	564.38	2.64	0.168	1.000

$^{\star}*$ = interactive and additive effects included.
$^{\dagger}K$ is the number of parameters.
$^{\ddagger}AIC_c = AIC + (2K(K+1)) \,/ (n-K-1)$.
$^{\S}\Delta_i = AIC_c(i) - \min(AIC_c)$.
$^{\P}w_i = \exp(-\Delta_i/2) / \sum_j \exp(-\Delta_j/2)$.

and 0.97 (0.88, 1.06) for chum salmon.

There was considerable uncertainty in the estimated predation mortality, particularly at high louse abundances because the range of infestation pressure I observed was limited – most prey in the lousy infestation category had just one sea louse of chalimus II or motile stage (figure 5.3a). At the peak of sea louse infestations in the study area, louse abundance was much higher (figure 5.3c). Nonetheless, the difference in predation mortality of pink and chum salmon (figure 5.3a) is consistent with the estimates of population-level mortality (Peacock et al., 2014, 2013, figure 5.3d-e), while individual-level direct louse-induced mortality (Krkošek et al., 2009, figure 5.3b) and the abundance of lice (Peacock et al., 2013, figure 5.3c) have been similar between the species.

Table 5.2: Parameter estimates on the scale of the linear predictor from the top models for each of the response variables I considered. See figures 5.1-5.3 for model predictions on the scale of the response.

Response	$\mathbf{w_i}^{\dagger}$	**Fixed effect**[‡]	**Est.**	**SE**	**95% CI**[§]		
logit pref. for pink	0.59	(Intercept)	0.720	0.106	0.513	0.927	*
	0.41	(Intercept)	0.638	0.120	0.403	0.874	*
		Treatment=Lice	0.159	0.116	-0.069	0.387	
logit proportion of remaining prey with predation scars	0.59	(Intercept)	-2.402	0.187	-2.770	-2.035	*
		Species=Pink	0.406	0.143	0.125	0.687	*
		Treatment=Lice	0.369	0.165	0.045	0.694	*
	0.22	(Intercept)	-2.349	0.203	-2.747	-1.952	*
		Species=Pink	0.301	0.217	-0.124	0.726	
		Treatment=Lice	0.279	0.216	-0.145	0.703	
		Pink:Lice interaction	0.185	0.287	-0.377	0.748	
	0.20	(Intercept)	-2.212	0.169	-2.542	-1.882	*
		Species=Pink	0.400	0.143	0.120	0.680	*
log number of sea lice per fish	0.56	(Intercept)	0.191	0.030	0.132	0.250	*
		After	-0.116	0.048	-0.210	-0.021	*
		Species = Pink	-0.043	0.043	-0.127	0.041	
		After:Pink interaction	-0.118	0.075	-0.266	0.029	
	0.44	(Intercept)	0.210	0.027	0.156	0.263	*
		After	-0.165	0.037	-0.237	-0.092	*
		Species = Pink	-0.081	0.035	-0.150	-0.012	*
logit proportion of available prey consumed	0.63	(Intercept)	-0.876	0.163	-1.196	-0.556	*
		Species = Pink	0.896	0.103	0.695	1.098	*
	0.20	(Intercept)	-0.858	0.173	-1.196	-0.520	*
		Species = Pink	0.895	0.103	0.694	1.097	*
		Mean lice per fish	-0.030	0.091	-0.209	0.149	
	0.17	(Intercept)	-0.783	0.180	-1.136	-0.431	*
		Species = Pink	0.749	0.143	0.469	1.029	*
		Mean lice per fish	-0.150	0.123	-0.391	0.091	
		Pink:Lice interaction	0.243	0.169	-0.089	0.575	

[†]Akaike weights normalized to include only the top models comprising 90% total Akaike weight from table 5.1.

[‡] The (Intercept) refers to the parameter estimate for the base factor level while other parameters indicate the change for the specified factor level (e.g., "Species = Pink") or the slope with respect to the continuous variable (e.g., "Mean lice per fish"). Base factor levels are "Treatment = No lice", "Species = Chum", and "Before" predation experiments.

[§]Stars denote parameters for which the 95% CI does not overlap zero.

5.3.4 Relating experimental results to functional response model

An increase in preference for pink salmon with parasites was an approximate condition for predation on chum salmon to decline with parasites, but this approximation held only for large N_p. Given the relatively small increase in preference with parasites that I observed, I consider the full condition given by equation 5.3 to determine what abundance of pink salmon is required for equation 5.3 to be satisfied.

First, I consider the equation for preference in terms of the base attack rates, γ_p and γ_c, and per-parasite proportional increases in attack rates, ω_p and ω_c. For the trials with clean prey, I substitute $x = 0$ into equation 5.4 to yield the equation for the preference for pink salmon in the absence of sea lice:

$$\alpha_0 = \frac{\gamma_p}{\gamma_p + \gamma_c}. \tag{5.9}$$

The corresponding equation for preference in lousy trials, denoted α_1, with pink and chum infested with x parasites is given by equation 5.4. Solving equation 5.9 for γ_c and substituting into equation 5.4, I arrive at the following expression for ω_c:

$$\omega_c = \underbrace{\frac{\left[\left(\frac{\alpha_0}{1-\alpha_0}\right)\left(\frac{1-\alpha_1}{\alpha_1}\right) - 1\right]}{1.19}}_{\text{intercept}} + \underbrace{\left(\frac{\alpha_0}{1-\alpha_0}\right)\left(\frac{1-\alpha_1}{\alpha_1}\right)}_{\text{slope}} \omega_p. \tag{5.10}$$

My estimates of $\alpha_0 = 0.665$ and $\alpha_1 = 0.680$ yield an intercept in equation 5.10 that is negative and a slope that is less than one. In that case, ω_c is less than ω_p for all positive values of ω_p, and the condition in equation 5.3 is met as N_p approaches infinity. Given my estimates of α_0 and α_1, what is the minimum

N_p for the condition in equation 5.3 to be met? If the intercept of equation 5.10 is negative, as my estimates suggest, then it follows that:

$$\frac{\omega_c}{\omega_c} < \left(\frac{\alpha_0}{1-\alpha_0}\right)\left(\frac{1-\alpha_1}{\alpha_1}\right), \tag{5.11}$$

and I can write the full condition for predation on chum salmon to decline with parasites in terms of the estimates of preference:

$$\left(\frac{\alpha_0}{1-\alpha_0}\right)\left(\frac{1-\alpha_1}{\alpha_1}\right) = \frac{T_h N_p^* \gamma_p}{1 + T_h N_p^* \gamma_p}. \tag{5.12}$$

Rearranging equation 5.12, I arrive at an equation for the minimum number of pink salmon, N_p^*, required for predation on chum salmon to decline with increasing parasites:

$$N_p^* = \frac{\alpha_0(1-\alpha_1)}{T_h \gamma_p(\alpha_1 - \alpha_0)}. \tag{5.13}$$

The calculation of N_p^* from equation 5.13 requires estimates for the handling time, T_h, and base attack rate on pink salmon, γ_p (figure 5.4). To get an estimate of handling time, I consider the maximum number of prey the average coho could consume in a day. In my experiments, the mean number of prey consumed per predator per day ranged from 0.25 to 10, but seemed biased high in shorter experiments. Considering the 24-hour experiments only, predators each ate an average of 1.88 prey. This was consistent with observations by Hargreaves and LeBrasseur (1985) who found that coho ate an average of 2-3 pink or chum salmon per day at the beginning of their experiments (as experiments progressed and coho grey this increased to $\sim$ 6 prey per day) and observations from Krkošek et al. (2011a) who estimated that coho ate approximately one prey per day in their group predation experiments. Based

on this information, I assumed that the average coho satiates at two prey per day and set $T_h = 0.5$ days.

To get an approximate estimate of the successful attack rate on pink salmon in the absence of parasites, γ_p, I used my recorded observations from the first hour of experiments, where I noted each successful attack on the school of prey by a coho predator ($n = 12$ trials). Based on these observations, the average attack rate per coho predator was 16.0 day^{-1} (range 4.8 to 24.0 day^{-1}). I was not able to distinguish between pink and chum prey in these observations, so I denote this overall attack rate as $\bar{\gamma} = \gamma_p + \gamma_c$. I then used the estimate of preference in the absence of parasites from the number of each prey species consumed to calculate the attack rate on chum salmon in terms of preference and the attack rate on pink salmon: $\gamma_c = \gamma_p(1 - \alpha_0)/\alpha_0$. It follows that $\gamma_p = \bar{\gamma}\alpha_0$, or $\gamma_p = 10.6$ day^{-1}. Using these estimates of $T_h = 0.5$ days and $\gamma_p = 10.6$ day^{-1}, the critical number of pink salmon in the school from equation 5.13 is $N_p^* \approx 3$ (figure 5.4).

The parameter estimates for handling time and attack rate derived from my experiments reflect the scale of a single school of pink and chum prey with a single group of coho predators over a maximum period of 24 hours. Previous studies have considered the population-level impacts of parasite-mediated predation over the entire juvenile salmon migration of 3 months (Krkošek et al., 2011a; Peacock et al., 2014). At this population scale, Krkošek et al. (2011a) estimated $T_h = 1$ day and a much smaller attack rate of $\gamma_p = 3.4 \times 10^{-6}$ day^{-1}. These population-level parameters result in a very different estimate of $N_p^* \approx 4$ million pink salmon. Although this estimate may seem large, at the population-scale it is not unreasonable that there would be millions of juvenile pink salmon transiting through coastal waters during the spring migration. Estimates of abundance from river surveys indicate that returns of adult pink salmon to rivers in the Broughton Archipelago may be as high as ~ 2 million

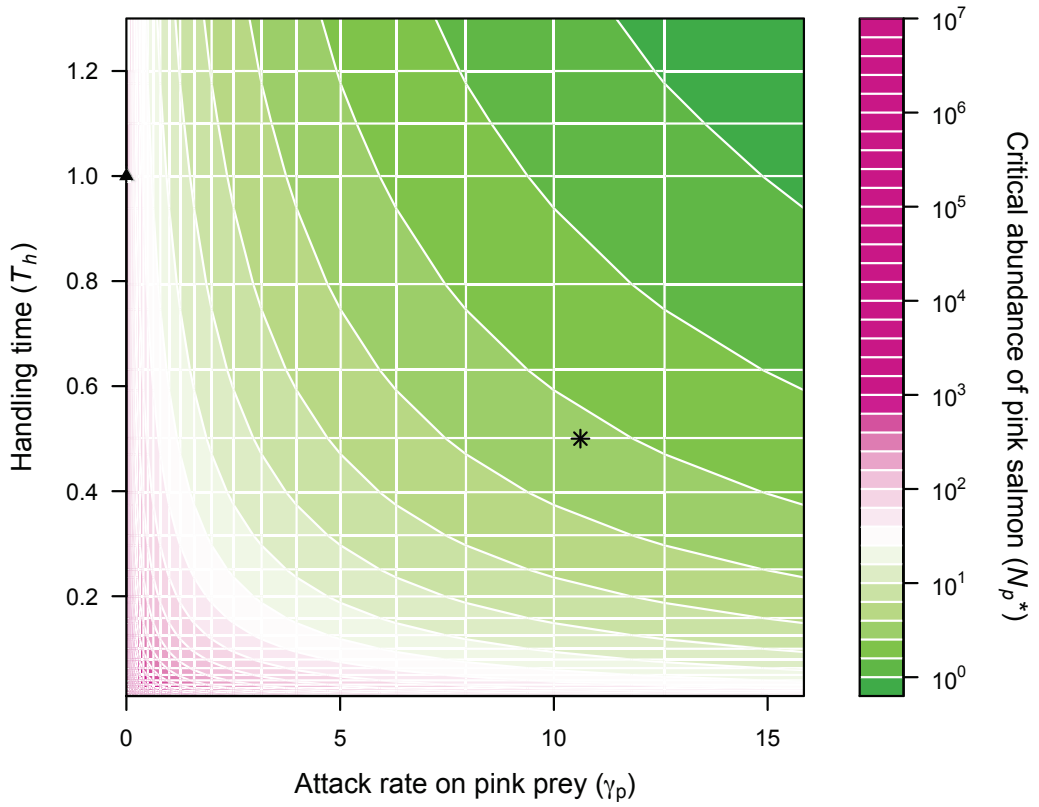


Figure 5.4: The condition for predation on chum salmon to decline with parasites depended on the attack rate on pink salmon in the absence of parasites (γ_p; x-axis), the handling time of prey (T_h; y-axis) and the number of pink salmon available to predators (N_p^*; filled contours). Approximate estimates of γ_p and T_h from my experiments suggested that the condition was met for $N_p^* \geq 3$ pink salmon (star), while previous population-level estimates of those parameters suggest this critical value of pink salmon is much higher at $N_p^* \geq 4$ million pink salmon (black triangle).

spawners (see figure 6a of Peacock et al., 2013), and the survival rate of salmon from juveniles to adults is relatively low (Parker, 1968) suggesting that there would be many millions of juveniles migrating in the spring.

5.4 Discussion

Parasite infestations can have diverse outcomes for host populations depending on how parasites affect host interactions in the broader community (Hatcher et al., 2012). Theoretical studies have suggested that predation can play an important role in mediating host-parasite interactions (Ives and Murray, 1997), particularly if predators display selective predation on parasitized prey (Hall et al., 2005). However, empirical work on the interactions between generalist parasites and predators in multi-host systems has been rare.

In this chapter, I investigated how parasites influenced selective predation in a juvenile salmon food web. I found that predators preferentially consumed pink salmon and preferentially consumed both pink and chum infested with sea lice, but there was uncertainty regarding how the predator preference for pink salmon changed with parasite infestation. The small increase in preference for pink salmon when prey were infested with sea lice was not statistically significant, but may be biologically significant. My calculations suggest that, given this small increase in preference, predation on chum salmon may decline with sea lice if enough pink salmon are present to occupy choosy predators.

The minimum number of pink salmon required to observe a decline in predation on chum salmon with parasites was highly dependent on the scale at which I considered parameter estimates. Observations from my experiments suggest that this critical abundance of pink salmon is as low as three, while parameters from other population-level studies (Krkošek et al., 2011a; Peacock et al.,

2014) put the minimum number of pink salmon in the millions. This difference reflects the importance of scale when interpreting the results of experiments such as ours. A type II functional response describing the consumption rate of individual predators over increasing abundance of prey may not be directly applicable at the population scale. For social species like juvenile salmon that migrate or hunt in groups, the number of groups, not individuals, may be the appropriate unit when considering population dynamics (Fryxell et al., 2007). The number of groups may not increase linearly with the number of individuals and, for juvenile salmon, the relative numbers of pink and chum salmon may vary considerably among schools. Therefore, it may not be trivial to understand population-level responses from individual-level experiments. In interpreting my results, I have implicitly assumed that the responses at the level of single schools of predators and prey would be observed at the population-scale, but more careful consideration of how these effects scale up should be incorporated into in future work.

My results clearly indicated that coho predators preferentially consumed pink salmon over chum salmon, consistent with a previous study reporting species-selective predation by coho salmon (Hargreaves and LeBrasseur, 1985). In the absence of sea louse infestations, the predation mortality of pink salmon was significantly higher than that of chum salmon (figure 5.3a). The preference for pink salmon did not change significantly in trials with lousy prey, but there was a trend towards increased preference with sea lice. When prey were infested, predation mortality of pink salmon tended to increase, as expected from previous work indicating that sea lice make juvenile salmon more vulnerable to predation (Krkošek et al., 2011a), but predation mortality of chum salmon tended to decline (figure 5.3a). Although the increase in predator preference for pink salmon with parasites was small and uncertain, it does point to a mechanism that may explain the different population-level responses of pink

and chum salmon. The effect of sea lice on predation of pink and chum salmon is consistent with observed population-level effects (figure 5.3a,e).

The experimental data did not offer clear support for a single hypothesis regarding prey preference or estimates of predation mortality, and so I used model averaging to account for the uncertainty in both parameter values and model selection (Burnham and Anderson, 2002). In this way, I avoided overestimating effect sizes, as would have occurred if I had used the full models to draw inference. For example, the impact of sea lice on predator preference (figure 5.1) was larger if the effect was estimated only from model that included treatment (i.e., lousy or clean trial) as a covariate. However, there was only a 41.2% chance that was the correct model over the null model with no effect of treatment (table 5.1). Evidence of a change in preference with sea lice was weaker when accounting for this model uncertainty by averaging the predicted preference between the null model and the model including lice as a factor. I acknowledge that the use of AIC for mixed-effects models is an active area of research and that there are concerns regarding model-averaged parameter estimates for models with multiple predictor variables (e.g., different parameter estimates among models for the same predictor due to collinearity among predictors Grueber et al., 2011). To avoid some of these pitfalls, I maintained the same random-effect structures among all models I compared and mainly reported model-averaged predictions rather than model-averaged parameters.

Several limitations in experiments may underlie the uncertainty in the estimates of preference. The experiments included a limited number of predators and prey, which may have increased variability in consumption of each prey species among trials due to the unavoidable stochastic nature of the order in which predators will encounter the different prey species. Future work over a wider range of prey abundances with higher replication would allow parameterization of the functional response parameters directly, and allow a more direct

test of how these parameters differ for different prey species. I used naturally-infested pink and chum salmon, and so the effect-size in my experiments was limited by the level of infestation in the wild. At the time of experiments, sea lice were not abundant on juvenile wild salmon. As a result, prey in lousy trials often had just one chalimus II stage louse, which may have had little effect on prey susceptibility to predation (Brauner et al., 2012; Krkošek et al., 2011a). Effect sizes may have been much larger for infestation levels such as those measured in the early 2000s (figure 5.3c), but that was extrapolating beyond the range of the data (figure 5.3a).

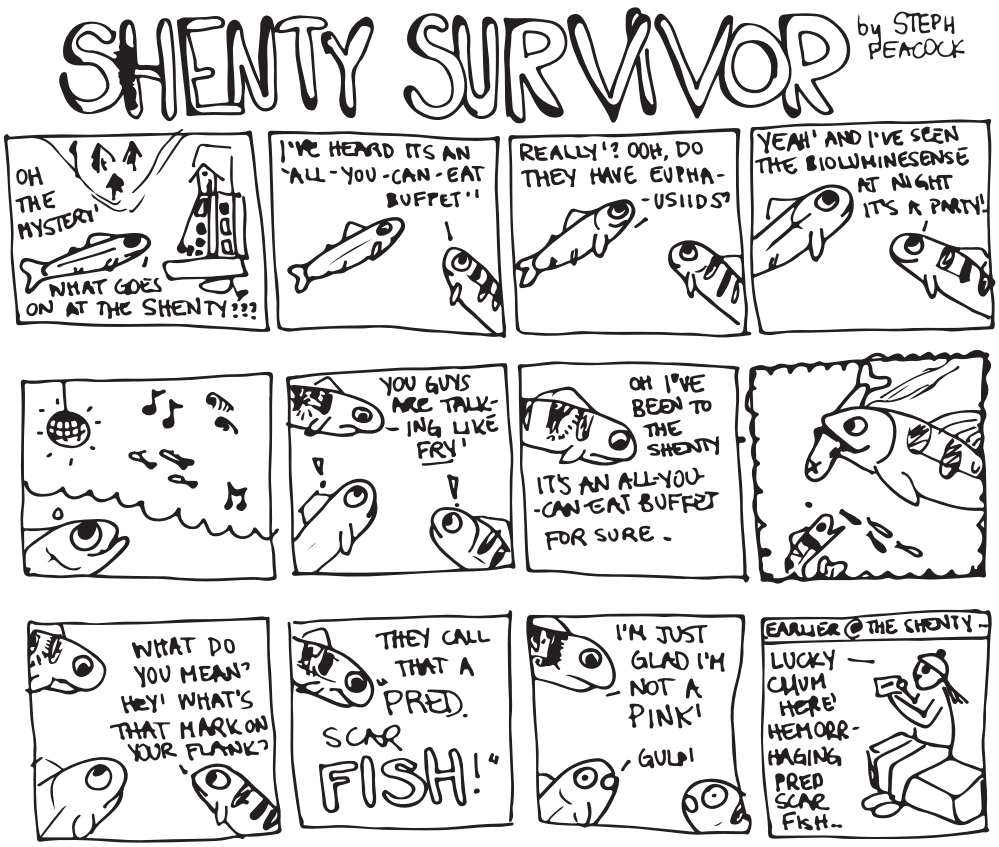
The ways in which generalist parasites affect food web dynamics is gaining attention (e.g., Hatcher et al., 2006, 2012), but the impact that parasites can have on interactions in host communities has long been recognized. In particular, parasite-mediated apparent competition, by which generalist parasites cause declines for host species that are more vulnerable to infection or have lower growth rates (Hudson and Greenman, 1998), has been cited as a major factor shaping the structure of ecological communities (Bonsall and Hassell, 1997). For juvenile salmon, parasite-mediated apparent competition could explain the observed differences in population-level survival of pink and chum salmon if high chum salmon abundance caused a rise in parasite numbers overall that had a disproportionate negative impact on sympatric pink salmon. However, experimental work has shown that, if anything, chum salmon incur higher direct parasite-induced mortality (Krkošek et al., 2009, ; figure 5.3). Further, pink salmon are the more abundant species in Broughton Archipelago, where population-level impacts have been estimated. The main source of sea lice on juvenile pink and chum salmon in my study area is farmed salmon in open-net pens along the migration route (Krkošek et al., 2006a), with secondary infection among juvenile salmon being lower, particularly at the beginning of the migration when juvenile salmon are most vulnerable to sea lice (Krkošek

et al., 2005a). It therefore seems unlikely that apparent competition is a reason why pink salmon seem to be more affected by sea lice at the population level, though this hypothesis may warrant further investigation.

5.4.1 Conclusions

In this chapter, I have shown that sea louse parasites can alter the food web dynamics of their juvenile salmon hosts and potentially shift predation towards preferred prey, leading to unexpected outcomes of parasite infestations for salmon populations. Initial research on sea lice and juvenile salmon focused on direct effects of parasites on host physiology (Brauner et al., 2012) and mortality in isolation (Krkošek et al., 2009; Morton and Routledge, 2005). In the wild, host survival is also influenced by community interactions and the ecological effects that parasites might have on processes such as competition and predation are of key importance (Hatcher et al., 2006).

In general, where predators display species-selective predation, even a small increase in prey preference with parasites can result in parasite-mediated release from predation for less-desirable prey under the right conditions. This is contrary to conventional thinking, which posits that parasites make hosts more vulnerable to predation by altering host behaviour or other traits (Hudson et al., 1992). Indeed, it was initially reported that sea lice increase predation susceptibility of both juvenile pink and chum salmon (Krkošek et al., 2011a). However, this straightforward interpretation can be complicated in multi-host systems with generalist predators, where parasites may alter food web dynamics. In such cases, the potential for interactions among host species through predation needs to be considered. This study contributes to an increasing realization of the diverse mechanisms by which parasites influence the dynamics of host populations and communities.


SHENTY SURVIVOR
by STEPH PEACOCK
OH THE MYSTERY! WHAT GOES ON AT THE SHENTY???
I'VE HEARD ITS AN "ALL-YOU-CAN-EAT BUFFET"!
REALLY!? OOH, DO THEY HAVE EUPHA-USIIDS?
YEAH! AND I'VE SEEN THE BIOLUMINESENSE AT NIGHT IT'S A PARTY!
YOU GUYS ARE TALK-ING LIKE FRY!
OH I'VE BEEN TO THE SHENTY IT'S AN ALL-YOU-CAN-EAT BUFFET FOR SURE.
WHAT DO YOU MEAN? HEY! WHAT'S THAT MARK ON YOUR FLANK?
THEY CALL THAT A "PRED. SCAR FISH!"
I'M JUST GLAD I'M NOT A PINK! GULP!
EARLIER @ THE SHENTY -
LUCKY CHUM HERE! HEMORR-HAGING PRED SCAR FISH..

Chapter 6

General discussion

Human-mediated changes to ecosystems are affecting infectious disease dynamics in wildlife across the globe (Pedersen et al., 2007). In particular, the spill-over and spill-back of pathogens between domesticated animals and wildlife can change natural disease dynamics in a way that hinders pathogen control and challenges biological conservation (Lafferty and Gerber, 2002; Pedersen et al., 2007). Such feedbacks have historically been studied in the terrestrial environment, but the rapid expansion of aquaculture in recent decades has seen a rise in new and re-emerging pathogens in the marine environment with high transmissibility (Kent, 2000). In this thesis, I considered the dynamics of sea louse parasites infesting farmed and wild salmon. The insights gained have application to the management of salmon aquaculture and conservation of wild salmon but are also of broad interest in ecology.

6.1 People and parasites

In the first part of this thesis, I considered how management intervention affects parasite population dynamics and transmission to wild salmon. In chap-

ter 2, I analyzed a general model for coupled populations subject to control that included the reciprocal interactions between human management of the system and natural parasite dynamics. Model simulations revealed that synchronizing dynamics of louse populations between farms lead to longer periods between treatments and thereby reduced reliance on parasiticides. Synchrony was achieved by high dispersal of parasites or coordinating stocking and harvesting between farms with similar internal growth rates or entrainment of a slower growing population by a faster growing one (i.e. source-sink dynamics). If growth rates differed between populations or dispersal was low and uneven, the parasite dynamics were difficult to predict in practice, and tended to be chaotic when small amounts of stochasticity were added. The unexpected complexity of the model emerged from the combination of parasite dispersal and a strict control threshold. Although treatment thresholds were implemented to reduce epizootics of sea lice, the approach may actually hinder parasite control by producing asynchronous parasite dynamics on adjacent farms. More predictable and controllable parasite dynamics could be attained if management on farms aimed to synchronize parasite populations through coordination of treatment and stocking dates.

The parasite dynamics and treatment timing on salmon farms in the Broughton Archipelago provided some empirical support for the purely theoretical conclusions of chapter 2. In chapter 3, I considered the linkages between the timing and frequency of sea lice treatments on salmon farms and sea lice abundances on farmed and wild fish in the Broughton Archipelago. As the theoretical model suggested, when treatments of farms within a region occurred at similar times in the winter, prior to the wild juvenile salmon outmigration, the abundance of lice on both farmed salmon and wild juvenile salmon was lower than in years when treatments appeared more reactionary. Interestingly, the synchronization of treatments in winter was not a formally coordinated man-

Figure 6.1: A juvenile pink salmon infested with *L. salmonis*, collected June 5, 2015 in the Broughton Archipelago. The prevalence and intensity of sea lice observed on juvenile salmon in 2015 was the highest since 2005, when management of sea lice on salmon farms seemed to adopt winter SLICE® treatments (S. Peacock, pers. obs.).

agement action mandated by any policy or guideline. The model results from chapter 2 suggest that the synchronization of treatments among farms could have been a fortuitous consequence of high louse dispersal.

One aspect that was missing from both chapter 2 and chapter 3 was the potential for environmental conditions, such as temperature and salinity, to affect sea louse dynamics and synchronize adjacent farms (i.e., the Moran Effect, Goldwyn and Hastings, 2011; Moran, 1953; Ranta et al., 1995). Modelling the effect of temperature and salinity on growth rates and survival of sea lice would be a worthwhile extension of the theoretical model (Groner et al., 2014; Stien et al., 2005) and necessary if one were to try and fit the model to data. The effect of temperature and salinity has been included in a statistical analysis of individual farms of the Broughton Archipelago (Rogers et al., 2013), but not in an analysis of the relative importance of dispersal versus internal popula-

tion growth (but see Aldrin et al., 2013, for a European example of such an analysis). If historic environmental data associated with sea lice monitoring on farms in Pacific Canada were available, fitting a mechanistic population model to those data could provide an empirical test of the importance of dispersal versus environmental conditions in synchronizing populations, which is a question of broad theoretical interest (Koenig, 1999; Lande et al., 1999; Ranta et al., 1995).

A common theme that emerged from both the theoretical and applied investigations of sea louse management was the importance of timing. Timing of treatments relative to adjacent farms could theoretically make treatments much more effective, as dispersal from an adjacent farm would be minimized post-treatment. Synchronized treatments occurring in January/February have the added benefit that louse numbers on farms tended to remain low throughout the juvenile salmon outmigration in March-June. Well-timed treatments of farmed salmon can therefore benefit both the salmon farming industry and wild salmon. Others have also recognized the importance of coordination and timing (e.g., Brooks, 2009; Costello, 2004; Kristoffersen et al., 2013), but unfortunately area-wide coordination of treatments is not mandated in many areas, including Pacific Canada. Despite the evidence pointing towards some positive adaptive changes in management, there was a resurgence of sea lice on juvenile salmon in the Broughton Archipelago in the spring of 2015 (figure 6.1). Although the cause of these recent elevated numbers of sea lice has yet to be determined, it serves as a reminder that the problem persists and farms must remain precautionary in their approach to sea lice.

6.2 Predators and parasites

Research on sea lice and salmon has been focused on management of sea lice in aquaculture and effects of sea lice on farmed salmon, perhaps because that is where human interest and influence lie. Studying the effects of sea lice on migrating wild juvenile salmon is much more complex as they are embedded in an ecosystem and subject to interacting pressure from predation and competition (Godwin et al., 2015; Krkošek et al., 2011a). In the second part of my thesis, I focused on how predation may mediate the effect of parasites in multi-host systems, seeking an explanation for why chum salmon populations have not declined with sea louse infestations in the Broughton Archipelago, though pink and coho salmon populations have (Krkošek et al., 2011b). Pink and chum salmon have very similar early life histories (Groot and Margolis, 1991), similar infection levels as juveniles (Patanasatienkul et al., 2013) and experience similar rates of direct parasite-induced mortality (Krkošek et al., 2006a). In light of these similarities between juvenile pink and chum salmon, the different population-level responses was unexpected. This apparent paradox could be dismissed due to the uncertainty in the chum population estimates and the potential for straying spawners to confound effects of sea lice at the population-level, as discussed in chapter 4, but I was interested in an ecological explanation for why chum salmon may be less affected by lice. Thus, I considered how species-selective predation and parasitism might interact to influence predation mortality of pink and chum salmon differently.

As in the first part of my thesis, there were some unexpected results. Previous work on single prey species suggested that predation increased with parasites becuase juvenile salmon infested with sea lice displayed deviant schooling behaviour, increased risk taking, and were preferred in individuals and group predation experiments (Krkošek et al., 2011a). In chapter 4, simulations from

a theoretical model suggested that if a predator preference for pink salmon increased with parasitism, predation mortality of chum salmon may decline with parasites. The decline in predation mortality of chum salmon may be large enough to offset direct negative effects of sea louse parasites, thereby explaining the lack of a population-level effect of sea lice on chum salmon. The details of how predators prey upon mixed-species schools may actually mean opposite effects of parasitism on the survival of pink and chum populations! A series of field-based experiments in chapter 5 were somewhat inconclusive, but could not rule out the possibility that the preference for pink salmon increased with parasites. The results emphasized that selective predation, on certain species and on parasitized prey, is an important consideration when assessing the impacts of parasites on host populations.

My study of the interacting effects of coho predation and sea lice on juvenile pink and chum salmon showed the complexity of parasitism in multi-host systems, but there is even more to the story than what I have investigated here. Trophic transmission of sea lice from prey to predators may influence parasite dynamics in the juvenile salmon food web. Previous studies have found that trophic transmission of lice from pink salmon prey to cutthroat trout predators was successful in 70% of trials (Connors et al., 2008b), and up to 2/3 of the motile sea lice on coho smolts come from pink and chum salmon prey (Connors et al., 2010a). In the host-parasite model that I introduced in chapter 4, I assumed that parasites were killed if their host was consumed, but this is clearly not always the case. Trophic transmission of sea lice intensifies parasite exposure for coho predators (Connors et al., 2010a), likely contributing to lower survival of coho associated with sea louse infestations (Connors et al., 2010b), but may also affect parasite dynamics of pink and chum salmon prey. The accumulation of sea lice on predators may affect their predation ability and/or survival, with implications for predation pressure on

juvenile pink and chum salmon. Trophic transmission of sea lice is sex biased, with adult males being much more likely to transfer to predators than other motile stages (Connors et al., 2008a). These male sea lice may transfer back to pink and chum salmon in search of mates (Connors et al., 2011), changing the parasite dynamics from those investigated in this thesis. There is room to explore these additional complexities through models, experimentation, and examination of existing data from long-term monitoring of sea lice on juvenile salmon.

My thesis work on juvenile salmon and sea lice adds to a growing body of research on the interacting effects of parasites and predators in ecological communities (Hatcher et al., 2006, 2012). There have been numerous theoretical studies of parasites and predation (e.g., Bairagi and Adak, 2015; Ives and Murray, 1997; Lafferty, 1992; Packer et al., 2003), but empirical examples are only just beginning to surface. Predation on larval anurans infected with trematode parasites is become one well-studied system for how community interactions can mediate host-parasite interactions (e.g., Marino and Werner, 2013; Orlofske et al., 2012; Raffel et al., 2010). Results from that work have emphasized the importance of trait-mediated effects of parasites on hosts; even the presence of parasites in the environment can result in increased avoidance activity of hosts which in turn increases host susceptibility to predation (Marino and Werner, 2013). More examples are needed to test model predictions for predator-prey-parasite systems. In particular, field studies on parasitism in rare species are needed to understand how ecological impacts of parasitism may affect host survival in nature.

6.3 Conclusions

The work I have presented builds upon a solid foundation laid down over the past decade through intensive research into the interactions between sea lice and their hosts. Previous work from the Broughton Archipelago has uncovered the importance of sea louse transmission from farmed to wild salmon (Krkošek et al., 2005a, 2006b), the impact of sea lice on wild salmon physiology (Brauner et al., 2012), ecology (Krkošek et al., 2011a), and population productivity (Krkošek et al., 2011b), and described the ecology of sea lice (Connors et al., 2008b, 2011). As our understanding of this system grows, so does the list of potential research questions. Further investigation often lead to surprises – complex parasite dynamics emerged from the combination of dispersal and threshold control; an unintended consequence of a well-meaning policy. Chum salmon populations did not follow the trend of decline associated with sea lice that was found for pink and coho salmon in the Broughton Archipelago (Krkošek et al., 2011b). Species-selective predation on pink salmon may lead to opposite effects of sea lice on predation mortality of pink and chum salmon.

There are few universal laws in ecology, perhaps because the complexity of ecological systems eludes unifying principles (Lawton, 1999). Pieces of ecosystems may be well-understood when separated in laboratory or mesocosm experiments, but the full suite of ecological interactions in nature is beyond characterization. Furthermore, humans are becoming an increasingly important part of ecosystems, and can change ecosystem dynamics in unanticipated ways (Liu et al., 2007a). My thesis research showed that unexpected dynamics can emerge from a combination of factors - people, predators and parasites - even when each part is well understood in isolation. If there is any unifying law in ecology perhaps it is that we should never assume that we know how an ecological system will react. This is important for ecologists and policy

makers to consider as we enter an era of unprecedented human growth and anthropogenic impacts on natural ecosystems (Ceballos et al., 2015; Millennium Ecosystem Assessment, 2005).

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