Sea lice in the North Pacific: from sub-lethal effects on wild salmon to parasite management and policy

by

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B.Sc. McGill University, 2012

Thesis Submitted in Partial Fulfillment of the
Requirements for the Degree of
Doctor of Philosophy

in the
Department of Biological Sciences
Faculty of Science

SIMON FRASER UNIVERSITY
Fall 2018

Approval

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Abstract

Since wild-capture fisheries production plateaued in the early 1990s, the world's dependence on aquaculture has grown steadily. This 'blue revolution' may have helped the conservation of some wild aquatic species by decreasing fishing pressure, but for others it has depleted their populations through habitat degradation, harvest for feed, and the spread of infectious disease. This thesis examines how parasites from aquaculture facilities can indirectly influence wild host survival and assesses how improvements to policy could limit these effects. I explore these topics in British Columbia, Canada, where wild Pacific salmon (*Oncorhynchus* spp.) are commonly infested with parasitic sea lice (*Lepeophtheirus salmonis* and *Caligus clemensi*) from open-net salmon farms. In Chapter 2, I use a field experiment to demonstrate that heavy sea louse infestation is associated with decreased competitive foraging ability for juvenile sockeye salmon (*O. nerka*). In Chapter 3, I show that this louse-associated reduction in competitive ability leads to decreased foraging success for juvenile sockeye in the wild. In Chapter 4, I analyse the otoliths (i.e., ear stones) of juvenile sockeye to reveal that highly infested fish grow more slowly than uninfested individuals. Each of these responses – competitive ability, foraging success, and growth – has major implications for salmon survival. In Chapter 5, I then investigate the ways in which parasite control policy could be improved on salmon farms to limit transfer of sea lice to wild salmon. I demonstrate that there is considerable underestimation bias in self-reported sea lice counts from industry, which determine when delousing treatments are used to control sea lice outbreaks on farms. I also show that current parasite control policy is not resilient to changing environmental conditions and I assess the potential effectiveness of alternative policies. Ultimately, the sustainability and success of the blue revolution will depend on our understanding of the full impacts of disease on wildlife and our ability to limit them.

Keywords: aquaculture, Pacific salmon, sea lice, sub-lethal effects, host-parasite, environmental policy

Dedication

This thesis exists because of my late grandfather, Lloyd (Bud) Fell.

When I was a kid, there was nothing I looked forward to more than fishing with grandpa. I'd stay over at my grandparents' house, and the next morning my grandpa and I would wake up way too early, have a piece of toast, and strike out on the water in search of salmon. To him, it was a routine he'd done countless times, having spent much of his life catching fish to stock the family freezer before another deployment in the Navy. To me, it was always an epic adventure with a man I hero-worshipped.

I'll never forget leaning over the gunwale to see my first orcas (with his hand on my life jacket) or my first time behind the wheel of a boat (on his lap). I won't forget the early morning rituals, the high fives when we landed a fish, or the conversations when we didn't. But most importantly, I won't forget the deep love for this coast that he instilled in me at such a young age.

It wasn't until after he was gone that I realised my life's passions and career path could all be sourced to the experiences I shared with my grandpa while growing up. I can't count the number of times I thought of him while doing fieldwork for this thesis, looking over the side of the boat at whales or sitting behind the wheel for the boat ride home. I'm sure he knew how much he influenced my life, but I regret not realizing it earlier and telling him. Whether he meant to or not, he taught me lessons that will stay with me for my entire life, especially the importance of hard work and love for those closest to you. I miss him more than I can explain and I wish he was still here to see this chapter of my life come to a close.

This one's for you, grandpa. Tight lines.

Acknowledgements

First, a massive thank you to my supervisors for their unending support during my graduate career. John Reynolds, Larry Dill, and Marty Krkosek have been with me through the highs and lows of my PhD, and somehow they stuck with me. John: I'll never know how Larry convinced you to take on a kid you'd never met as a graduate student, but for that kindness (or foolishness?) I will always be tremendously thankful. One day I hope to provide the same insight, enthusiasm, and genuine care to my students that you have shown to me. Larry: I will forever be proud to have been your last student (if only because I took so damn long at it). Thank you for your incredible generosity with your time, for your infectious passion about ecology and conservation, and for all the fantastic conversations (both over beers and not). Marty: it's hard to believe that we first met almost 10 years ago, when you came to rescue me and my crewmates after we broke down in your boat in Tribune Channel. Thank you for sharing the beauty of the Broughton Archipelago with me, for providing so many years of mentorship in the field, and for showing me just how rewarding the life of a coastal researcher can be.

I am immensely thankful to Craig Orr for his support and mentorship throughout my PhD. Craig - you are an inspirational advocate for salmon conservation, and if I ever get too deep in code or papers and start wondering why I do what I do, I only have to look to you. I also thank Jonathan Moore and Julia Baum for their time and expertise while serving as the internal and external examiners for my defence.

Despite the first person active voice, this thesis was not a solo venture. In addition to the people listed above, I also thank Brendan Connors for his feedback on the early chapters and Andrew Bateman for the massive amount of time he spent collaborating with me on Chapter 5. I also thank Brian Hunt for the expertise he shared with me during this thesis.

I am very grateful to Sandra Vishloff, Marlene Nguyen, and Susan Riviere for constantly steering me in the right direction while at SFU. I'm sure it can sometimes feel that your hard work behind the scenes goes unnoticed, but we (the students) know how integral you are to our graduate careers.

I have been very fortunate with financial support during my time at SFU. I am thankful to NSERC for giving me a Postgraduate Scholarship and an Industrial Postgraduate

Scholarship (IPS). The funding for that NSERC IPS was shared with Watershed Watch Salmon Society, and I cannot thank that organization enough for taking a risk on an unproven graduate student. I have also received several financial awards from Simon Fraser University and its Department of Biological Sciences, for which I'm very appreciative. I also thank TIDES Canada, MITACS, and the W. Garfield Weston Foundation for their considerable support over the years. Finally, I am immensely grateful to the Hakai Institute for financially supporting me personally as well as the Salmon Early Marine Survival Program.

I am incredibly thankful to my family for all their support throughout my life and during this thesis. Thank you to my mom for raising me and my sister through difficult circumstances. Mom: you never stopped and were rarely thanked, but I know how much you shaped me into the person I am today. Thank you to my dad and my stepmom, Lin, for their continuous love and support. Dad, without you I never would have made it through undergrad; I can't tell you who many times a phone call to you 'levelled the ship', even if you didn't know it. Thank you to my sister, Nicole, for tolerating two and a half decades of my version of brotherly love and for always putting a smile on my face. Thank you to my grandparents, Barbara and Bud Fell, for helping provide me a childhood full of love and happy memories. And thank you to the Fliggs for punctuating my years during this thesis with games and laughter.

The people of Salmon Coast Field Station have played a huge role in my life and thesis. I can't imagine what my field seasons would have been like without late night chats in the purple room, beach fires, Shenty sleepovers, walks to Billy's, fishing expeditions, flat calm mornings on the boat, fiery red sunsets on Grandma's porch, and so many other shared experiences. Many of the friendships that began at Salmon Coast will last my entire life. In particular I thank Lauren Portner, Dylan Smyth, Luke Rogers, Emma Atkinson, and Andrew Bateman for many years of comradery and support, and Stephanie Peacock for showing me the ropes way back in 2009. I also thank Mirko Diaz, Carson White, Leah Walker, Shannon Mendt, Jenni Schine, Clare Atkinson, Chris Guinchard, Heather Forbes, Peter Harrington, Coady Webb, Zephyr Polk, and the dozens of other Salmon Coasters whose company I have had the pleasure of enjoying over the years.

Earth to Ocean is a very unique lab group, and I'm grateful to have had the opportunity to interact with so many of its brilliant young scientists and all of its PIs. Many of the Côté and Reynolds lab members have been pillars of my life both inside and outside of work. There's something special about a group of people that can always put a smile on your face even while at the office; for this especially, I thank Debora Obrist, Brett Howard, Jane Pendray, Luis Malpica-Cruz, and Kirsten Wilcox.

I am so grateful for all the memories that I have made with my Vancouver friends, some of whom have put up with me for six years now. Many of these people I met at Salmon Coast or SFU. Most have lived with me at some point. All are extremely dear to my heart. Thank you to Evan Colyer for being a truly great friend and for living with me for two of the best years of my life. Thank you to Brett Howard for all the chats over beers and the evening games that have been staples to me for the last half decade. Thank you to Cole Howard for making several climbing trips genuinely unforgettable experiences (and for the occasional Shakespearean argument with your sibling). Thank you to Jeremy Enns for the ice cream and disc golf dates. Thank you to Lauren Portner for being an absolute rock in my life for years and for our many dog walks. Thank you to Dylan Smyth for the being an excellent pseudoroommate, road trip companion, and scavenger hunt creator. Thank you to Debora Obrist for sweeping into my life with laughs and chocolate. Thank you to Hannah Watkins for helping me get through the final stretch. And a huge thank you to Kevin Lu for being a wonderful (and tolerant) roommate, an inspirational climbing partner, and an all-around good guy.

Finally, I thank my partner Fiona Francis for four amazing years together. I honestly could not have made it through the final year of this PhD without Fiona's perpetual encouragement and friendship. She stuck with me through the worst of times and was at my side through the best of them. Because of her, I'll always look back at these years fondly and I am already looking forward to the next ones with her and our crazy pup, Cashew. With all due respect to this thesis, the best thing that came out of this degree was meeting Fiona. Thank you for all your love and support, Fi.

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Chapter 1.

General introduction

The rise of aquaculture

With wild-capture fisheries production having plateaued nearly three decades ago, aquaculture has been increasingly relied upon to provide protein to the world's growing human population. Aquaculture is now the fastest-growing food sector in the world (Subasinghe et al. 2009), accumulating over CAD $200 billion in sales each year and providing 18 million jobs globally (FAO 2016a). As of 2014, aquaculture contributes more to global fish consumption than capture fisheries, and this is predicted to increase drastically over the next decade (FAO 2016a). Never has the world's per capita fish supply been so high, and this is directly attributable to the rapid growth of the aquaculture industry (often called the 'blue revolution').

The blue revolution is regularly promoted as the solution for declining wild fish and shellfish populations (Natale et al. 2013), and in some instances this is probably true. For example, the farming of carp in China and India has led to increased food security without threatening wild fish stocks (Ahmed and Lorica 2002). In other cases, aquaculture may have negative effects on wild fish and shellfish populations through habitat degradation, bycatch of fish in seedstock collections, harvest of wild fish for feed, or other mechanisms. In southeast Asia, large swathes of mangrove forests have been destroyed to make way for shrimp ponds, eliminating critical nursery habitat for many wild fish and shellfish (Paul and Vogl 2011). The farming of milkfish (*Chanos chanos*) in the Philippines requires collections of wild juveniles that can kill 10 billion juvenile finfish annually due to bycatch mortality (Naylor et al. 2000). Aquaculture also directly uses wild fish resources for protein in feed (24% of the feed, on average; Tacon and Metian 2015) and most of these fish are food-grade for humans (Cashion et al. 2017). Although the relative dependence of aquaculture feed on wild-caught fish is predicted to decline over the next decade, the overall harvest of wild fish for feed is still expected to increase as the industry grows (Msangi et al. 2013). There are similar concerns over several other effects of aquaculture on wildlife, including effluent discharge and introduction of exotic

species (Klinger and Naylor 2012), but perhaps the most notable is the transfer of infectious diseases to wild fish.

Infectious disease in aquatic wildlife

Infectious diseases have long been the bane of the aquaculture industry. Like terrestrial agriculture (Harwood 1990), aquaculture facilities provide ideal breeding grounds for disease by farming high densities of aquatic organisms in environments not subject to natural selection. As a result, diseases cost the marine aquaculture industry billions of dollars each year, mainly through mortality, decreased marketability, diminished growth, or the necessity of vaccinations or treatments (Lafferty et al. 2015). And for every disease brought under control, a new one is bound to arise; as Lafferty et al. (2015) put it: “aquaculture’s history is one of victories over diseases followed by new challenges.”

The disease dynamics between wild and farmed aquatic species are often difficult to disentangle. Sometimes, as in the case of the fungal crayfish plague in Europe (Alderman 1996) or the polychaete outbreaks in California abalones (Culver and Kuris 2004), diseases are exotic and epizootics in wildlife populations are obviously attributable to aquaculture activities. When disease outbreaks in wildlife are from native pathogens or parasites, however, it can be challenging to determine whether farmed species were involved. Aquaculture facilities can act as reservoirs to which native pathogens and parasites can transfer, before amplifying in numbers and transferring back to wild hosts (Daszak et al. 2000). Differentiating a natural epizootic and one caused by farmed species can be very challenging (Lafferty et al. 2015).

Even if the role of aquaculture in disease outbreaks of aquatic wildlife can be confirmed, identifying the impacts of disease can be just as, if not more, problematic. Theoretically, the addition of disease reservoirs should increase extinction risk for a focal host species (De Castro and Bolker 2005), but the actual effects of disease on wildlife depend on the extent of exposure, the characteristics of the infectious agent, and the susceptibility and resistance of the host (Lafferty et al. 2015). We can estimate direct mortality from disease using laboratory studies (e.g., Hameed et al. 2003; Skall et al. 2004; Schikorski et al. 2011), but disease can also interact with ecological processes to determine, for example, the effectiveness of individuals at competing for resources or avoiding predation (Hatcher et al. 2006). These sub-lethal (or ‘indirect’ or ‘ecological’) effects are

harder to quantify because they require ecological context and therefore cannot be assessed in a laboratory environment. It stands to reason that species whose mortality rates are determined primarily through the outcomes of ecological processes like competition and predation (e.g., fish; Hixon and Jones 2005) may be heavily influenced by the sub-lethal effects of disease. Despite their potential importance to individuals and populations, the sub-lethal effects of disease are rarely examined in wild fish.

The salmon farming industry and its notorious parasites

The difficulty and importance of understanding how aquaculture influences disease dynamics in wildlife populations is perhaps best exemplified by the salmon farming industry. Salmon farms generally raise hundreds of thousands of domesticated Atlantic salmon (*Salmo salar*) in open-net pens that occupy the coastal waters of Norway, Chile, the United Kingdom, Ireland, Canada, and several other countries (Groner et al. 2016). Marine Atlantic salmon farming is a CAD $19 billion global industry, making it the most valuable variety of aquaculture despite being only the ninth largest in terms of biomass (FAO 2016b).

Like other forms of aquaculture, salmon farming is highly susceptible to disease outbreaks. Epizootics of infectious salmon anemia have caused mass mortality events in salmon farms around the world (Cottet et al. 2011). The Norwegian industry is currently battling with heart and skeletal muscle inflammation, a relatively common disease caused by piscine orthoreovirus (Di Cicco et al. 2017; Wessel et al. 2017), which produces mortality rates up to 20% and morbidity rates as high as 100% (Kongtorp et al. 2004; Palacios et al. 2010). The Chilean salmon farming industry has been devastated by the bacterial disease piscirickettsiosis for decades (Rozas and Enríquez 2014), while Canadian farms have experienced sporadic but major losses due to the viral disease infectious hematopoietic necrosis (St-Hilaire et al. 2002; Saksida 2006). The list goes on, with most diseases causing major economic losses to farms (Asche et al. 2009; Rozas and Enríquez 2014; Lafferty et al. 2015). By far the most significant infectious agent on salmon farms, however, is a group of ectoparasites called sea lice (primarily *Lepeophtheirus salmonis* and *Caligus* spp.), which cost the global salmon farming industry over CAD $600 million each year (Costello 2009a).

In every salmon farming country (e.g., Buschmann et al. 2009; Peacock et al. 2013; Hersoug 2015), policies have been implemented to control the spread of disease from farmed to wild salmon and trout (*Oncorhynchus* spp. and *Salmo* spp.). Most of these policies were put in place specifically to control sea lice outbreaks. Sea lice feed on the mucus and surface tissues of fish and are native to each of the world's salmon farming regions (Costello 2006). Wild adult salmon are often infested with sea lice, but the more vulnerable juvenile salmon are separated spatially and temporally from adults (called 'migratory allopatry'), so they are normally rarely infested (Costello 2009a). Salmon farms break this separation by providing year-round reservoirs for sea lice (Krkošek et al. 2007a), which can result in high louse infestation rates in wild juvenile salmon (Bjørn et al. 2001; Marty et al. 2010; Price et al. 2011).

Sea lice negatively affect wild salmon individuals and populations. At the individual level, sea lice can cause moderate levels of direct mortality to juvenile salmon in laboratory environments (Finstad et al. 2000; Jones and Hargreaves 2009; Jakob et al. 2013), especially at small host sizes (Jones et al. 2008). Juvenile pink salmon (*O. gorbuscha*) infested with sea lice are more likely to be eaten by predators (Krkošek et al. 2011a; Peacock et al. 2015), but other than that the sub-lethal effects of sea lice are poorly understood. Since competition and predation are the primary causes of mortality for salmon (Groot and Margolis 1991; Hixon and Jones 2005), any effect of sea lice on these critical ecological processes could have a large impact on overall mortality. At the population level, juvenile exposure to sea lice reduces adult returns of wild Atlantic salmon by 18% (Vollset et al. 2016) or 39% (Krkošek et al. 2013a), depending on the analysis. No equivalent experimental studies have been performed for Pacific salmon (*Oncorhynchus* spp.), but correlational work has shown that sea lice abundance on farms is associated with reduced productivity for populations of pink and coho salmon (*O. kisutch*; Connors et al. 2010; Krkošek et al. 2011b), but not for chum salmon *(O. keta*; Peacock et al. 2014).

Coexistence of wild and farmed salmon in Pacific Canada

British Columbia (BC), Canada is the only region in the world that is a large producer of both wild and farmed salmon (Groner et al. 2016). After growing steadily for decades, the Atlantic salmon farming industry now yields 81% of all salmon production in BC (FAO 2016c). In contrast, BC's wild salmon have generally been in decline over the

same time frame (Price et al. 2017). Consequently, the potential effects of salmon farms on BC's wild salmon have been hotly debated (e.g., Marty et al. 2010; Krkošek et al. 2011b), with frequent disagreements between academics and scientists from government and industry about the extent of the problem.

Sea lice have been the main focus of scientific research on the impacts of BC salmon farming (e.g., Krkošek et al. 2007b; Connors et al. 2010; Bateman et al. 2016), nearly all of which has focussed on *L. salmonis*, a salmonid specialist (Pike and Wadsworth 1999). The other species of sea louse in BC, *C. clemensi*, is a generalist that infects other nearshore marine fishes in addition to salmonids (Parker and Margolis 1964); until this thesis there was no research on the potential effects of *C. clemensi* on wild salmon. Despite the scientific disagreements regarding sea lice in BC, significant progress has been made in minimizing their effects, at least for *L. salmonis*. High sea louse infestation rates were first observed on wild juvenile salmon in 2001, and shortly thereafter new regulations were implemented to control *L. salmonis* numbers on farms (Peacock et al. 2013). Like every other country that farms salmon, these regulations require farms to perform a chemical delousing treatment or harvest their fish if sea lice counts exceed a threshold; in BC, that threshold is three pre-adult or adult *L. salmonis* per fish (Saksida et al. 2010). BC farms are also required to treat more quickly after a high louse count between March and June (just before and during the wild juvenile salmon migration) than in other months (Fisheries and Oceans Canada 2016). Salmon farms perform their own sea lice counts to inform these management decisions, and the department of Fisheries and Oceans Canada (DFO) attempts to ensure accuracy in these data by performing intermittent audits of the counts.

Sea louse control policy may have improved the outlook for some of BC's wild salmon (Peacock et al. 2013), but recent evidence suggests that there may be opportunities to improve. In the Broughton Archipelago of BC, sea louse epizootics on out-migrating juvenile salmon were greatly reduced after the new regulations were introduced (Peacock et al. 2016). These regulations seemed to slow or cease the decline of local pink salmon populations (Peacock et al. 2013). In 2015, however, parasite control lapsed in Broughton Archipelago salmon farms, resulting in the highest louse levels observed on wild juveniles in a decade (Bateman et al. 2016). This event made it clear that while sea louse control policy in BC may be effective in most years, we know little

about how what factors influence that effectiveness and whether policy improvements ought to be made for the sake of imperilled wild salmon populations.

Fraser River sockeye salmon (*O. nerka*) are one of the most important sets of salmon populations in the world (Northcote and Larkin 1989). They are an economic staple of Canada's west coast and for millennia have been a vital food source for local Indigenous peoples (Jacob et al. 2010). In the 1990s and 2000s, however, Fraser River sockeye experienced a two-decade decline in productivity (Peterman and Dorner 2011), followed by record-low adult returns in 2009 and 2016 (Pacific Salmon Commission 2016). This productivity decline spurred a $37 million federal inquiry led by BC Supreme Court Justice Bruce Cohen (Cohen 2012c; 2012b; 2012a). Salmon farming was a major focus of this inquiry because Fraser River sockeye migrate through a hotspot of BC salmon farms and the abundance of sea lice on juvenile Fraser River sockeye had already been linked to these facilities (Price et al. 2011). As a result, the Cohen Commission's recommendations explicitly outlined the need for research into the potential effects of sea lice on Fraser River sockeye (Cohen 2012a).

Overview of thesis chapters

This thesis examines the potential sub-lethal effects of sea lice, specifically *Caligus clemensi*, on wild salmon (Chapters 2-4) and the effectiveness of policy designed to control sea lice on salmon farms (Chapter 5). I use British Columbia (BC), Canada as a model system and, for Chapters 2-4, I use juvenile Fraser River sockeye as my study organism.

In Chapter 2, I consider the potential effects of sea lice on the competitive abilities of juvenile sockeye salmon. This involves a field experiment using wild-caught Fraser River sockeye to determine whether heavily infested juvenile salmon are worse competitors for food than fish with fewer or no sea lice. In Chapter 3, I assess whether that louse-associated reduction in competitive foraging ability translates to reduced foraging success in the wild. I accomplish this using a field survey of wild Fraser River sockeye during their juvenile migration to determine the amount of food in the stomachs of fish with different infection intensities of sea lice. In Chapter 4, I evaluate whether heavily infested juvenile sockeye experience decreased growth – an important determinant of survival for salmon. For this chapter, I analyze the microstructure of sockeye otoliths to

establish the recent growth history of fish prior to capture and relate it to their sea louse infestation rate.

After examining potential sub-lethal effects of sea lice on wild juvenile salmon in Chapters 2-4 and finding extremely high louse infestation rates in each study, the natural follow-up question was: can policy be improved to better control sea lice on farms during the juvenile salmon migration? In Chapter 5, I use parasite control policy in BC as a case study to examine several ways in which environmental policies can be undermined. Specifically, I assess the extent to which wild fish populations transfer sea lice to farmed salmon, the potential bias of self-reported sea lice counts from industry, and the resilience of parasite control policy to environmental change. To answer these questions, I fit a hierarchical Bayesian model to a long-term dataset of sea lice counts on salmon farms. In doing so, I perform the first analysis of the spatial and temporal dynamics of *L. salmonis* and *C. clemensi* on BC farms and provide possible solutions for improving existing parasite control policy. In my final chapter, I synthesize the results of the previous chapters and discuss how they can inform future research and management.

Chapter 2.

Sea lice, sockeye salmon, and foraging competition: lousy fish are lousy competitors[1]

Abstract

Pathogens threaten wildlife globally, but these impacts are not restricted to direct mortality from disease. For fish, which experience periods of extremely high mortality during their early life history, infections may primarily influence population dynamics and conservation through sub-lethal effects on ecological processes such as competition and predation. I conducted a competitive foraging experiment using out-migrating juvenile Fraser River sockeye salmon (*Oncorhynchus nerka*) to determine whether fish with high abundances of parasitic sea lice (*Caligus clemensi* and *Lepeophtheirus salmonis*) have reduced competitive abilities when foraging. Highly infected sockeye were 20% less successful at consuming food, on average, than lightly infected fish. Competitive ability also increased with fish body size. My results provide the first evidence that parasite exposure may have negative sub-lethal effects on fitness of juvenile sockeye salmon, and suggest that sub-lethal effects of pathogens may be of key importance for the conservation of marine fish.

Introduction

Pathogens are a major threat to wildlife around the world. From outbreaks of protozoan parasites in wild bumble bees (Meeus et al. 2011) to viral epidemics in wild carnivores and African apes (Hofmeyr et al. 2000; Leroy et al. 2004; Origgi et al. 2012), new cases of pathogen-induced mortality and population declines continue to be identified, with few management attempts showing clear benefits of intervention (Woodroffe 1999). The

[1] A version of this chapter appears as Godwin, S. C., Dill, L. M., Reynolds, J. D., & Krkošek, M. 2015. Sea lice, sockeye salmon, and foraging competition: lousy fish are lousy competitors. Canadian Journal of Fisheries and Aquatic Sciences 72(7): 1113-1120.

emergence of diseases in many wildlife populations is attributable to anthropogenic influences that promote pathogen transmission and virulence such as climate change, habitat loss, introduced species, pollution, and domesticated animals (Daszak et al. 2000; Harvell et al. 2002; Brearley et al. 2013). For extinction risk, pathogens are primarily a threat when a reservoir host population maintains high pathogen prevalence in a sympatric threatened host species as it declines towards extinction (De Castro and Bolker 2005; Krkošek et al. 2013b).

Impacts of pathogens on their hosts go beyond direct mortality from disease. Host-parasite systems provide particularly noteworthy examples of behavioural (Carney 1969), physiological (Kristan and Hammond 2000), and morphological (Johnson et al. 1999) changes to hosts that can indirectly reduce their survival. Pathogens can modulate crucial ecological processes affecting the host such as competition or predation (Hatcher et al. 2012a), and this may be particularly important for fishes whose life histories typically include extreme mortality from these interactions (Groot and Margolis 1991; Hixon and Jones 2005). These high mortality rates of fish in the absence of pathogens make it difficult to understand the implications of pathogen infection on fish populations. Consequently, studies restricted to direct effects on survival or physiology (e.g., Jakob et al. 2013) are only tangentially relevant for fish populations because they ignore how pathogens interact with other processes that can lead to high mortality, such as competition and predation.

Salmon (*Oncorhynchus* spp. and *Salmo salar*) exemplify the complexity of fish disease ecology. Epidemics in wild fish created by parasite spill-back from farmed fish that act as reservoir hosts for ectoparasitic sea lice (*Lepeophtheirus salmonis*) can cause mortality that exceeds previous fisheries catch and may threaten persistence for some species (Krkošek et al. 2007b). In other systems, impacts of parasitism do not scale up to the population level, potentially due to modulation of predation pressure (Peacock et al. 2014). Theoretically, parasitism can act synergistically or antagonistically with predation depending on where the predator-prey system lies along a type II functional response whereby predator intake rate increases with prey density toward an asymptote (Krkošek et al. 2011a). Sockeye salmon (*O. nerka*) are of great ecological, cultural, and economic importance to the west coast of North America (Cooke et al. 2004; Eliason et al. 2011) and are therefore a strong candidate species for conservation research. Recently, the decline of Canada's iconic Fraser River sockeye stocks triggered a $37 million dollar

federal judicial inquiry into the causes of the decline (Cohen 2012c; 2012b; 2012a), which identified disease interactions of sockeye with salmon aquaculture operations as a major management uncertainty and research priority.

Populations of domesticated salmon represent a reservoir host population of *Caligus clemensi* sea lice, which can infect wild juvenile sockeye (Price et al. 2011). Infection risk for juvenile sockeye is exacerbated by the generalist nature of *C. clemensi*; Pacific herring (*Clupea pallasii*) can also be infected by *C. clemensi* at high abundances and are likely a second sea louse reservoir to sockeye (Morton et al. 2008; Beamish et al. 2009). However, it is not known how or whether parasite exposure fits into the long-term decline of sockeye productivity. Direct mortality of juvenile sockeye from *C. clemensi* infection has not been estimated, but it is likely very low; in a laboratory setting, *L. salmonis* infection does not cause direct mortality except at extreme abundances not seen in wild fish (Jakob et al. 2013). Because Pacific salmon experience high mortality during their early marine life (Parker 1968; Bax 1983) and early marine growth is crucial for survival (Beamish et al. 2004; Moss et al. 2005; Farley et al. 2007), any impact of *C. clemensi* on sockeye population dynamics is probably indirect, by modulating competition or predation. Here, I performed a foraging experiment to test whether the competitive ability of juvenile Fraser River sockeye differs with sea louse abundance. Competitive ability, in this case, was defined as intake during a food pulse of limited quantity and duration between hungry fish. I found that infection is associated with reduced competitive ability, whereas large body size is associated with greater competitive ability. Collectively, these results indicate that elevated parasitism, potentially due to infection from farmed salmon, may have sub-lethal effects on survival and conservation of an iconic fish.

Materials and methods

Sea louse life cycle

Sockeye salmon on the south coast of British Columbia (BC) are infected by two species of sea louse: *L. salmonis*, a specialist on salmonids, and *C. clemensi*, a generalist on many fish species. The life cycle of the sea louse has an early free-living phase (nauplius stages), a phase in which the louse is attached to its host (copepodid and chalimus stages), and a mobile phase during which the louse can move around on the surface of its host (pre-adult and adult stages). The number of chalimus stages differs

among louse genera. Here, I use the term ‘large chalimus’ to describe the final two chalimus stages for *C. clemensi*, which has four chalimus stages, and the final chalimus stage for *L. salmonis*, which has two (Kabata 1972; Hamre et al. 2013). I use ‘motile’ to encompass all pre-adult and adult stages of louse development. Adult lice reproduce sexually, with attached females extruding pairs of egg strings whose eggs hatch into nauplii. The generation time for *L. salmonis* is 40 to 52 days at 10°C and shorter at warmer temperatures (Johnson and Albright 1991), and the generation time for *C. clemensi* is probably similar based on other *Caligus* species (Hogans and Trudeau 1989; Piasecki and MacKinnon 1995).

Study area and sampling design

I collected juvenile sockeye salmon in northern Johnstone Strait, BC every two to nine days from May 29 to June 13, 2013 (Figure 2.1) during the three and a half weeks that sockeye were observed in this region. My collection area is a migration bottleneck for juvenile sockeye and individual collections were made after visual confirmation of sockeye presence, usually through surfacing behaviour. Fish were captured by purse seine (bunt: 27 x 9 m with 13 mm mesh, tow: 46 x 9 m with 76 mm mesh) designed for hand-retrieval from a small (6 m) motorized vessel. Capture typically occurred between 5 and 40 m from shore.

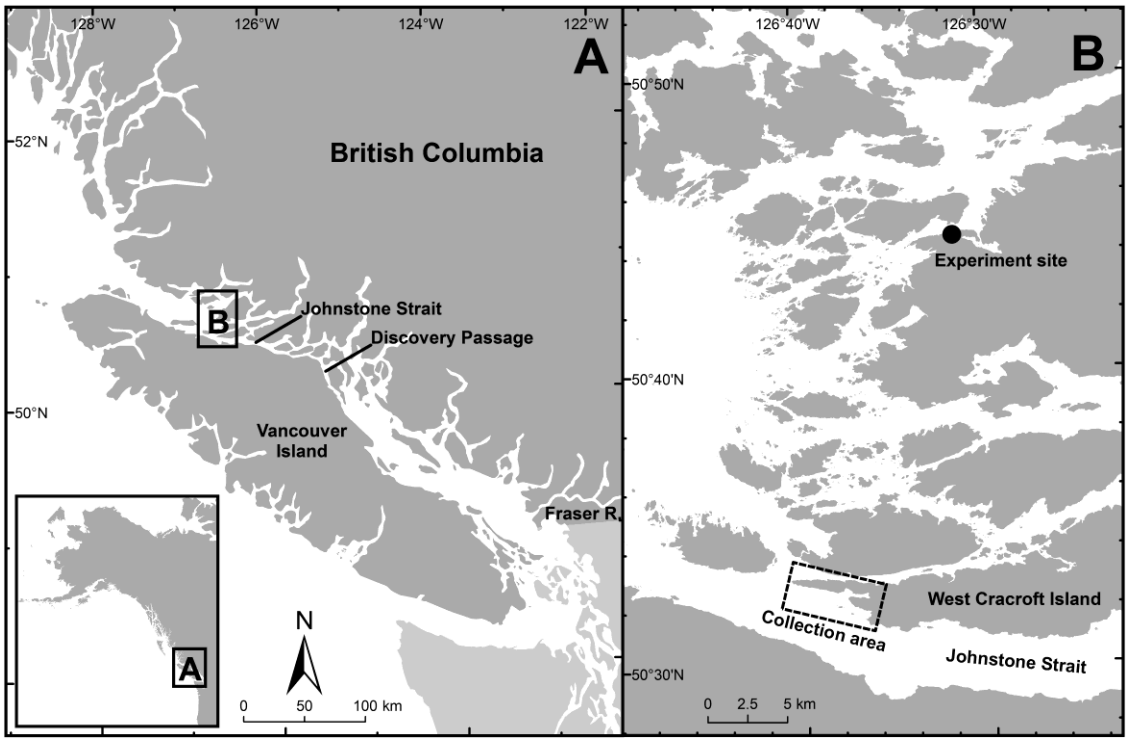

Figure 2.1 Locations of the collection area and the competitive foraging experiment.

Out-migrating juvenile sockeye salmon were captured in the region defined by the dashed rectangle in May and June, 2013.

Captured fish were initially held alongside the boat in a pocket of the bunt end of the net that was carefully set with sufficient depth and width to allow the fish to swim with minimal apparent stress and to prevent contact with the netting. The maximum time that fish were held in the net was approximately 0.5 h, during which time sockeye were removed from the net. The vast majority of bycatch was other juvenile Pacific salmon, but Pacific sand lance (*Ammodytes hexapterus*), threespine stickleback (*Gasterosteus aculeatus*), rockfish (*Sebastes* spp.), and Pacific herring were also captured in low numbers (fewer than 10 non-salmon bycatch individuals in each set). Fish were transferred from the net by allowing them to swim into a seawater-filled 3.79 L milk jug with the base cut off, and then transferred into one of three seawater-filled insulated fish totes (0.58 m deep and 0.97 x 0.55 m across) by submerging the milk jug and allowing the fish to swim out. All subsequent transfers of fish were performed using this technique. During initial collections, fish were observed in 13.2 L transparent plastic aquaria to check carefully for sea louse detachment resulting from transfer. No sea lice were seen detaching from their host during these or subsequent collections. Collection ceased when I had reached a density of approximately 150 fish per tote. I did not always

capture enough fish in my first set to reach the desired density in the three totes; in these cases I set the net again and continued adding fish to the totes. When the first fish had been in a tote for 2 h, I ended the collection and no more fish were added. No species other than sockeye salmon were kept or assessed for sea louse infection.

Fish transportation and holding

The juvenile sockeye were transported for approximately 1 h by boat to the experimental facility in Cramer Passage, in the Broughton Archipelago, BC. I used ice packs and battery-powered aquarium bubblers to ensure adequate temperature and aeration during transportation. Approximately 1 600 fish were captured and transported over the course of the sampling period. All fish survived transportation except during one collection due to poor weather during the transportation process. Fish from this collection were released and not used in the experiment. Apart from the fish in this collection, transported sockeye did not exhibit behaviours indicative of stress such as gasping, clamped fins, or unusual movement other than being easily startled into the corners of the tote.

The experimental facility consisted of several floating wooden docks that supported four ocean net pens in a location chosen for shelter from wave action. Upon arrival at the facility, sockeye were transferred immediately to one of three net pens. One net pen was 2.1 m deep and 2.8 x 2.8 m across, another was 2.3 m deep and 4.4 x 3.2 m across, and the third was 2.8 m deep and 6.1 x 6.1 m across; each had 4 mm mesh walls and floors. Each collection of approximately 450 fish was stored in a separate net pen.

The fish were fed frozen adult brine shrimp (Brine Shrimp Direct, Ogden, Utah, USA) in the holding pens. The shrimp had minimal size variation and were added into the middle of the net pen after being thawed in an equal volume of seawater. During this acclimatization period, feeding occurred every 2 h during daylight hours such that an average of 3 g of brine shrimp was fed to each fish over the course of the day.

Competitive foraging experiment

After three to ten days in the holding pen, sockeye were selected for the foraging experiment. Each trial used fish from only one collection, i.e., all fish caught in a single

day. Individual fish were transferred from the holding pen to 13.2 L transparent plastic aquaria and visually assessed for infection of large chalimus and motile stage lice. A more detailed assessment of louse infection could not be performed without handling the fish, which would have increased the likelihood of louse detachment. Due to the high prevalence of infection (> 99%), it was not possible to obtain enough uninfected fish to form infected and uninfected groups. Instead, I selected 10 fish in each of the upper and lower extremes of the distribution in louse load from among approximately 300 fish, on average. My selection criteria for the infection categories changed between trials depending on the abundance of lice on the fish, but for all trials the minimum difference in louse load between the lightly infected and heavily infected groups was three large chalimus stage lice and one motile louse (Table 2.1). Fish with scars, open wounds, hemorrhaging fins, or other external signs of poor health were not used in the experiment. Five trials of 20 fish (10 in each infection category) were initiated, though one fish escaped during a trial.

Table 2.1 Infection abundances of sea lice before and after the competitive foraging experiment with juvenile sockeye salmon.

	Pre-experiment criteria				Post-experiment means[a]			
	Lightly infected		Heavily infected		Lightly infected		Heavily infected	
Trial	Large chal.[b]	Motile[c]	Large chal.	Motile	Large chal.	Motile	Large chal.	Motile
1	≤ 2	0	≥ 5	≥ 2	1.1 ± 0.2	1.4 ± 0.4	4.4 ± 0.5	2.3 ± 0.5
2	≤ 2	0	≥ 5	≥ 2	0.6 ± 0.3	0.8 ± 0.3	3.3 ± 0.2	3.2 ± 0.4
3	0	0	≥ 4	≥ 2	0.4 ± 0.2	0.8 ± 0.2	2.3 ± 0.2	2.7 ± 0.5
4	≤ 2	0	≥ 5	≥ 1	0.8 ± 0.1	0.4 ± 0.2	4.3 ± 0.7	1.6 ± 0.3
5	≤ 2	0	≥ 5	≥ 2	0.3 ± 0.2	0.7 ± 0.2	3.1 ± 0.4	2.1 ± 0.4
Overall					**0.3 ± 0.1**	**0.8 ± 0.1**	**3.5 ± 0.2**	**2.4 ± 0.2**

[a] Louse means are given ± SE

[b] 'Large chal.' includes chalimus III and IV stage *C. clemensi* and chalimus II stage *L. salmonis*

[c] 'Motile' encompasses the pre-adult and adult stages of both louse species

After fish were assigned to an infection category, they were transferred to the experimental net pen (2.3 m deep and 4.4 x 3.2 m across). Once there, fish were fed an excess quantity of brine shrimp every hour during daylight hours to ensure that all had the opportunity to feed. I began depriving the fish of food approximately 24 h after they were placed into the experimental net pen. The foraging experiment was initiated 30 min after sunrise two days later, which corresponded to 36 h after the beginning of food deprivation.

To initiate the experiment, 20 g of frozen brine shrimp was fed to the fish in the same manner as in the holding pens. After 2.5 min of foraging, the fish were startled by a deliberate sudden movement by the experimenter so that they would not feed on any remaining brine shrimp. In a test run, most of the brine shrimp were consumed at the end of 2.5 min and the most successful foragers stopped feeding after this point. Following the experiment, the holding pen was pulled up to form a shallow pool and the fish were captured using milk jugs. They were then transferred to individual sterile sample bags (Whirl-Pak® Write-On Bags; Nasco, Fort Atkinson, Wisconsin, USA) and euthanized with a lethal dose of MS-222 (240 mg L-1). I assessed each fish for sea louse infection by hand lens (Krkošek et al. 2005a), including smaller louse life stages, and placed them on ice. Temperature and salinity were measured inside the net pens at the surface and 1 m depth immediately following the experiment (Table A.1).

Dissection

I recorded the fork length, body depth and wet body weight of each fish in the laboratory and noted any tissue damage or evidence of poor health. Stomachs were excised between the lower oesophagus and the pyloric sphincter, and their wet contents weighed. For each fish, half of the heart and two gill arches with filaments were removed and stored in an RNA preservative (RNAlater®; Life Technologies, Burlington, Ontario, Canada) at 4°C overnight and -20°C thereafter.

To further assess the health status of my experimental fish, I screened the tissue samples from all the fish in the first trial and half the fish in trials 2-5 for salmonid alphavirus (SAV), infectious salmon anemia virus (ISAV), and piscine orthoreovirus (PRV) (see Appendix B for methods). The samples tested from trials 2-5 were from the five least successful foragers in the heavily infected group and the five most successful foragers in the lightly infected group, as determined by their stomach content weights after the experiment. These fish were chosen to provide the largest difference in competitive abilities while maintaining even ratios of lightly infected and heavily infected fish.

Statistical analyses

To test for differences in wet stomach content weights and post-experiment louse loads between infection categories, I used Welch's *t* test for two samples of unequal variance. I used linear regression to determine whether wet stomach content weights increased with fish body size. This body size term was the result of a principal component analysis (PCA) used to convert my three measures of body size (fork length, body depth, and wet body weight discounted by stomach content weight) into one linearly uncorrelated body size variable. The data from the first principal component explained 93% of the original variation in body size data.

To evaluate which parameters best predicted the wet stomach content weights, and therefore the competitive abilities of my experimental fish, I performed model selection using Akaike Information Criterion corrected for small sample sizes (AICc) (Hurvich and Tsai 1989). I fit eight mixed-effects models to the data using trial number as a random effect on the intercept in each. My models were constructed *a priori* according to my hypotheses and included the biologically relevant combinations and pairwise interactions of three fixed effects: the fish's infection category (i.e., highly or lightly infected), the total number of lice on the fish after the experiment, and the body size variable from the PCA. I included the post-experiment louse total term in my candidate model set to assess whether louse dispersal among fish during the experiment and development from chalimus to motile stages influenced my ability to predict competitive ability from the initial conditions of the fish. All analyses were performed in R 3.0.1 (R Foundation for Statistical Computing, 2013) using the nlme and MuMIn packages.

Results

Fish that had been assigned to the lower infection category before the experiment had less than one-third as many sea lice after the experiment as those in the higher infection category (Figure 2.2A). Sockeye in the lower infection category had no motile sea lice before entering the experimental pen, but they averaged 0.8 motile lice per fish after the experiment (Table 2.1), which equated to 58% of the fish having motile lice. Despite the increase in motile lice and decrease in large chalimus lice over the course of the experiment, I was able to identify the original infection categories of the fish after the experiment because of the criteria that distinguished them initially (see Figure A.1). Each

trial initially had a minimum difference of three large chalimus between the two infection categories; after each trial, the two groups of fish still had different numbers of large chalimus, even though the overall chalimus abundances decreased. Although 96% of motile lice infecting the experimental fish were *C. clemensi*, a second species of louse, *L. salmonis*, accounted for 4% of motile lice. The mean (± SD) length of my experimental fish was 11.2 cm ± 1.1 cm, their mean depth was 2.0 cm ± 0.2 cm, and their mean body weight discounted by stomach content weight was 12.2 g ± 4.0 g. Mean fish body size did not differ between infection categories (two sample *t* test, $t = 0.34$, df = 97, $p = 0.74$).

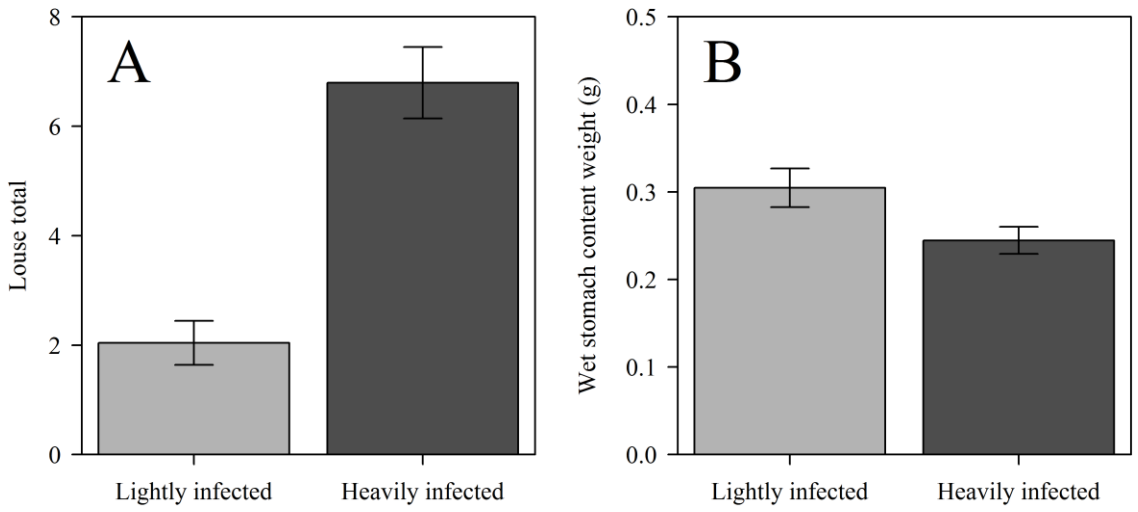

Figure 2.2 Louse total and wet stomach content weight recorded post-experiment for both infection categories of juvenile sockeye.
Error bars show the 95% confidence intervals for the mean value.

Adding brine shrimp to the experimental net pen caused a feeding frenzy, suggesting that the experiment conditions were highly competitive. All fish were observed feeding in each trial, and brine shrimp were the only organisms found in the digestive tracts of the fish after the experiment.

Of the 60 experimental sockeye screened for viruses, none were positive for ISAV or SAV. Two fish tested positive for PRV, one from each infection category.

Juvenile sockeye competitive ability was related to both sea louse infection and body size. The top model of eight models fit to the stomach content weight data included terms for both of these predictors. This model accounted for 97.7% of model support using Akaike weights and was 8.21 AICc units lower than the second top model (Table 2.2), indicating that it had substantially more empirical support than any other model in

my model set (Burnham and Anderson 2002). The standard deviation of the random intercept was 0.0245. The top three models accounted for 99.8% of model support and all included a term for the infection category of the fish.

Table 2.2 Model selection statistics for the mixed-effects models fit to the stomach content weight data from the competitive foraging experiment.

Rank	Model[a]	ΔAICc[b]	w_i[c]
1	*pre-infection + size*	0	0.977
2	*pre-infection*	8.21	0.016
3	*pre-infection * size*	10.33	0.006
4	*size*	13.93	0.001
5	*post-infection + size*	15.09	0.001
6	*intercept only*	20.66	0
7	*post-infection*	23.84	0
8	*post-infection * size*	28.68	0

[a] All models include a random effect on the intercept for trial number. Terms included were the infection category of the fish before the experiment (*pre-infection*), the number of lice on the fish after the experiment (*post-infection*), and the body size variable from the principal component analysis (*size*). Parameter estimates (± SE) for the top model were 0.304 ± 0.014 for the intercept, -0.058 ± 0.011 for the highly infected group, and 0.020 ± 0.004 for the body size variable derived from the principal component analysis (n = 99). Models with interactions (e.g. "*pre-infection * size*") include lower order main effects.
[b] Difference from the top model AICc
[c] Akaike model weight

As predicted, highly infected sockeye had lower competitive abilities in the experiment relative to lightly infected fish. Highly infected fish were 20% less successful at consuming food than lightly infected fish, on average (Figure 2.2B): the mean (± SE) stomach content weight of the fish was 0.305 g ± 0.011 g for the lightly infected sockeye (n = 50) and 0.245 ± 0.008 for the highly infected sockeye (n = 49). Also as predicted, wet stomach content weights increased with body size for each infection category (Figure 3), and this relationship was consistent among all trials (see Figure S2).

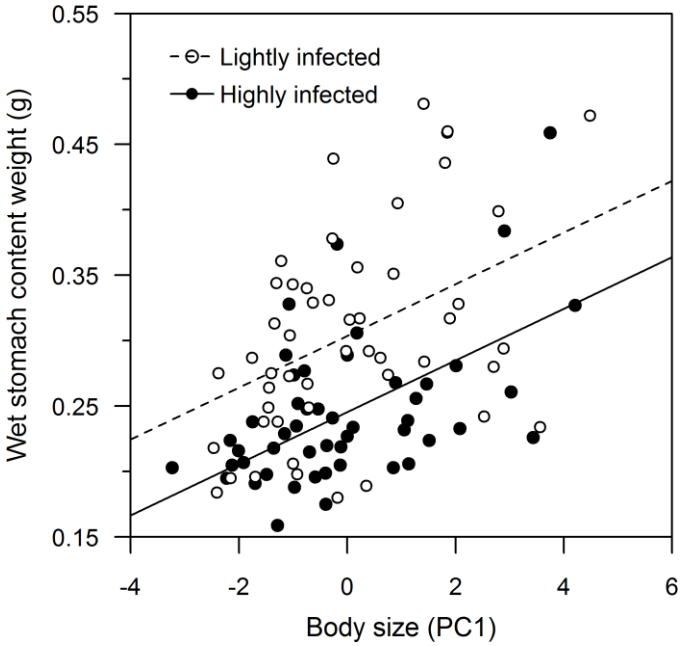

Figure 2.3 Wet stomach content weights as a function of body size for fish of both infection categories.

Each point represents an individual fish. The unitless body size variable was derived from a principal component analysis using three correlated measures of body size (PC1 = the first principal component). Regression lines use the coefficients from the main effects of the top mixed-effects model, which includes terms for infection category and body size (Table 2.2).

Discussion

Theory predicts that complex host-pathogen dynamics may arise in multi-host systems, particularly when pathogens modulate other ecological processes such as competition and predation (Hatcher et al. 2012a). My results suggest that parasitic sea lice may modulate intra-specific competition among wild juvenile sockeye salmon, where higher sea louse abundances produce lower competitive abilities in hosts. My results also indicate higher competitive abilities in larger fish, but that there was little evidence that larger fish were less impaired by infections than smaller fish.

The relevance of my results to wild-feeding fish depends on how well the experimental environment represented the natural conditions encountered by out-migrating juvenile sockeye. My experiment simulated a limited patch of a single prey type. The physical characteristics of northern Johnstone Strait cause biological production in this body of water to be extremely low (McKinnell et al. 2014). Juvenile sockeye prey availability is thought to be so low in this region that McKinnell et al. (2014) put forth a ‘trophic gauntlet hypothesis’, in which sockeye suffer an energy deficit while travelling the 150 km through

Johnstone Strait and the individuals that survive this journey are only those with sufficient energy reserves. Although the patchiness and diversity of sockeye prey in Johnstone Strait are not yet known, the prey limitation in this region is such that feeding opportunities for wild juvenile sockeye may be limited and individuals must therefore compete.

There are several potential mechanisms by which sea lice might reduce the competitive ability of juvenile sockeye. These include, but are not limited to: visual impairment, swimming impairment, stamina reduction, and antagonistic behaviour from larger or more dominant fish. Further research is needed to determine which mechanisms apply; only antagonistic behaviour from larger fish can be discounted because there was no difference in size between the infection categories of my experiment. My objective was to determine whether highly infected fish have reduced competitive abilities because of the implications of such an outcome. In equal-opportunity prey-limited environments, lower competitive abilities should reduce foraging success, which determines the energy that can be allocated to growth. For fish, growth can be used as a surrogate component of fitness (Schluter 1995) and can determine the outcome of competition and predation (Sogard 1997). Indeed, as shown in my experiment, size also influences competitive foraging success, suggesting a further delayed effect of louse infection on competitive ability.

Despite having no motile sea lice upon entering the experimental net pen, 58% of the fish in the lower infection category had motile lice afterwards (Table 2.1). This indicates either the development of chalimus stage lice into motile lice, the transfer of motile lice from individuals in the higher infection category, or the attachment of motile lice from the environment during the 60 h that the fish were held in the experimental pen. I believe louse development was the primary driver of this result for four reasons: (1) no sea lice were observed to detach from their hosts during the handling process, (2) the mesh size of the net pens was small enough to hinder motile louse influx, (3) abundances of large chalimus stage lice were lower after the experiment than they were before (Table 2.1), and (4) although no study has clarified the timing of the life cycle for *C. clemensi*, the dominant species in my experiment (96%), work on two other *Caligus* species suggests that a realistic estimate of development time between the final *C. clemensi* chalimus stage and the adult stage could be between 21 and 71 degree days, which would result in a considerable portion of large chalimus stage lice being able to molt into motiles

between my infection assessments (on average, 29 degree days at 1 m depth) (Piasecki and MacKinnon 1995; González and Carvajal 2003).

Since the treatment groups were not created experimentally, it is possible that fish with different parasite loads differed in some aspect of condition that may have predisposed them to louse infection. One such possibility is pre-existing viral disease. For a separate investigation, I screened a subset of my fish for three viruses of potential concern: SAV, ISAV, and PRV. None of the 60 experimental fish tested positive for ISAV or SAV, and only two were positive for PRV, one from low and one from high louse infection categories. In addition to viral screening following the experiment, I assessed the external conditions of the fish before the experiment and their internal conditions immediately after. I chose fish for the experiment that did not have significant scarring, open wounds, or hemorrhaging fins. During dissection, I confirmed that the fish did not have swollen kidneys, pale gills, discoloured livers, internal bleeding, or any other obvious internal sign of poor health. Though not conclusive evidence, the absence of the three viruses tested in all but two fish and the apparently healthy internal and external conditions of the fish help argue against the hypothesis that the driver of the effect seen in my competitive foraging experiment was an underlying health difference rather than sea louse infection.

Juvenile sockeye salmon were primarily infected with *C. clemensi*, although *L. salmonis* accounted for 4% of motile lice on experimental fish. This result is consistent with the relative abundances of louse species reported by Price et al. (2011) on juvenile Fraser River sockeye earlier along their migration route. However, the highest infection prevalences reported by Price et al. were considerably lower (84% in 2007 and 62% in 2008 for *C. clemensi*) than the prevalence I observed immediately after capture (> 99%), which prevented me from challenging groups of uninfected fish in the experiment. This discrepancy could be a result of year-to-year variation in environmental conditions, increased transmission from salmon farms in the Discovery Passage, BC (Figure 2.1A) (Price et al. 2011), or transfer from another louse source further along the migration route, such as Pacific herring. Regardless, the high infection prevalence observed is of concern given the potential fitness consequence of sea louse exposure demonstrated by my experiment.

A potential alternative explanation for my results could be that highly infected fish have reduced appetites. Farmed Atlantic salmon (*Salmo salar*) adults with high numbers of *L. salmonis* can have reduced appetite, but there has been no published evidence of such an effect in wild or juvenile fish. In fact, previous work indicates that infected wild juvenile pink salmon (*O. gorbuscha*) are willing to accept higher predation risk to access food (Krkošek et al. 2011a). Additionally, all of my experimental fish joined the feeding frenzy upon food introduction. One other potential alternative explanation for my results is that highly infected fish have increased metabolic rates and therefore faster digestion. Should this effect occur, it is unlikely to have confounded my data because the fish were immediately euthanized after the experiment ended and therefore differences in stomach content weight are unlikely to be the result of differences in digestive rates. Differential competitive ability between infection categories of fish therefore remains the most plausible mechanism behind my results.

Although I was not able to confirm the origin of my fish genetically, there is a very high probability that they were from the Fraser River, given the timing and the large pulse of fish moving through the region, rather than from the few small local systems (Groot and Cooke 1987; Price et al. 2013). I ensured that I collected juvenile sockeye during the peak of the Fraser River sockeye out-migration by monitoring the progress of the run through observers in the Discovery Islands.

Fraser River sockeye are of substantial economic and ecological importance. After a two-decade decline in Fraser River sockeye productivity, record-low adult returns in 2009 prompted intense public and scientific scrutiny of their management. The resulting federal judicial inquiry by the Cohen Commission provided 75 recommendations to the Canadian government on the management and conservation of Fraser River sockeye. The final report postulated that the causes of the decline may originate in the nearshore marine waters where the fish grow (Cohen 2012c) and one of the recommendations explicitly stated the need for research on determining “what pathogens are encountered by Fraser River sockeye salmon along their entire migratory route, and the cumulative effects of these pathogens” (Cohen 2012c). By showing that juvenile Fraser River sockeye with high abundances of sea lice have reduced competitive abilities, my work provides the first evidence for a fitness consequence of parasite exposure in Fraser River sockeye.

Sea louse infestations of domesticated salmon in BC are currently managed using in-feed treatment with a parasiticide (emamectin benzoate (‘‘SLICE’’); Intervet/Schering-Plough Animal Health, Boxmeer, The Netherlands) (Saksida et al. 2010). In the Broughton Archipelago, treatments preceding juvenile pink (*O. gorbuscha*) and chum salmon (*O. keta*) out-migrations have resulted in dramatic reductions in louse levels of wild fish and positive conservation outcomes for local wild populations (Peacock et al. 2013). Louse loads of out-migrating sockeye are still very high in Johnstone Strait, as I observed, and elevated sea louse abundances have been linked to salmon farms earlier on the sockeye migration route in the Discovery Passage (Price et al. 2011). Under the conditions of their Licence for Finfish Aquaculture, farms are only required to initiate treatment when the abundance of lice exceeds three motile *L. salmonis* per fish. No policy exists for reducing *C. clemensi* levels, despite this being the primary louse species infecting juvenile sockeye migrating through this region. A treatment regime aimed to minimize *C. clemensi* infestations on farmed fish in the Discovery Passage prior to and during the migration of juvenile sockeye in this region could reduce one potentially significant stressor on one of the most important salmon populations in the world.

My results are important within the context of marine conservation for species like sockeye salmon that are susceptible to infection by pathogens with complex multi-host dynamics. Both fish farms and Pacific herring act as reservoir hosts from which *C. clemensi* can be maintained in the environment even when there are few or no sockeye present. Other species of salmon for which sea lice have been implicated as causes of concern are primarily infected with the salmonid specialist *L. salmonis* (e.g., Krkošek et al. 2007b; Gargan et al. 2012), so the potential for highly abundant reservoirs of other fish species is considerably reduced. Fraser River sockeye, however, are mainly parasitized by the generalist *C. clemensi*, which infect other species such as Pacific herring. This additional reservoir host exacerbates any threat to sockeye from sea louse infection, such as reduced ability to forage successfully, because the potential exists for a stronger Allee effect and greater extinction risk if the sockeye population becomes sufficiently low (Krkošek et al. 2013b). This complexity is compounded by sub-lethal effects of parasites on fish populations. For salmon, early marine survival largely depends on growth (Beamish et al. 2004; Farley et al. 2007), so the results of resource competition among individuals likely govern their survival. The implications of a pathogen-induced reduction in competitive ability could therefore determine the survival

of infected individuals. Understanding how pathogens like sea lice interact with fundamental ecological processes that determine fish survival is essential for effective marine conservation of populations vulnerable to pathogen infection.

Chapter 3.

Heavy sea louse infection is associated with decreased stomach fullness in wild juvenile sockeye salmon[2]

Abstract

Foraging success can be mediated by parasites, but this is poorly understood for marine fish whose aggregations and patchy prey fields create conditions for intense intraspecific competition. I evaluated whether sea louse infection is associated with decreased stomach fullness of wild juvenile sockeye salmon (*Oncorhynchus nerka*) in Johnstone Strait, BC during their marine migration from the Fraser River. *Caligus clemensi* comprised 98.6% of the pre-adult and adult lice and 86.5% of the copepodites (freshly attached juvenile lice); the rest were *Lepeophtheirus salmonis*. I found that infection status was an important predictor of relative stomach fullness for juvenile sockeye (wet stomach content weight divided by body weight), as indicated by mixed-effects model selection, and that highly infected fish had 17% ± 8% lower relative stomach fullness than did lightly infected fish. This louse-associated reduction in relative stomach fullness occurs as the juvenile sockeye migrate through a food-limited environment, and presumably elevated competition. Given that early marine growth for juvenile salmon is often a predictor of survival, my results highlight the importance of understanding sub-lethal effects of parasites on salmonids and possibly other fish species.

[2] A version of this chapter appears as Godwin, S. C., Krkošek, M., Reynolds, J. D., Rogers, L. A., & Dill, L. M. In press. Heavy sea louse infection is associated with decreased foraging success in wild juvenile sockeye salmon. Canadian Journal of Fisheries and Aquatic Sciences.

Introduction

Foraging success is tightly linked to growth and survival of individuals, both of which are predictors of fitness and population dynamics (Crombie 1947; Sutherland 1996). Fish frequently experience patchy foraging opportunities and aggregate in large groups that intensify competition (see review by Ward et al. (2006)), and consequently survival often depends on these competitive outcomes (e.g., Welker et al. 1994; Resetarits 1995). This is especially the case for juvenile fishes, whose early growth depends on food supply and often determines survival and recruitment (Houde and Hoyt 1987; Anderson 1988; Bergenius et al. 2002). Evidence suggests that parasites affect competitive foraging outcomes of hosts, thereby influencing host population dynamics, community structure, and biodiversity (see Hatcher et al. 2006). However, this evidence is primarily from terrestrial species (e.g., Grosholz 1992; Schall 1992; Maksimowich and Mathis 2000). Despite what is likely a more competitive environment with food patchiness and consumer aggregation for fishes, there is little work in the fisheries literature on how parasites mediate competition in wild fish and whether this translates to decreased survival through reduced foraging success and growth (but see Finley and Forrester 2003).

Pacific salmon (*Oncorhynchus* spp.), like many fishes, experience high juvenile mortality from predation and starvation (Parker 1968; Groot and Margolis 1991). Consequently, the impacts of parasitism may be primarily expressed through the mechanisms by which parasitism affects inter- and intra- specific interactions. Juvenile Pacific salmon migrate in large groups to swamp and evade predators (Eggers 1978; Furey et al. 2016), so competition for food is probable in regions with low prey availability (McKinnell et al. 2014). Foraging success during the marine migration of juvenile salmon likely affects growth, which is often a predictor of survival (Moss et al. 2005; Farley et al. 2007; Duffy and Beauchamp 2011), so competition in environments of low foraging opportunity is one plausible mechanism through which parasites may affect survival of juvenile salmon (Godwin et al. 2015). Although parasitism can raise energetic requirements and thereby increase foraging rate (e.g., Giles 1987; Shi et al. 2002), parasitism may also interfere with the behavioural process of obtaining food in a food-limited environment and thereby reduce foraging success (e.g., Barber and Ruxton 1998).

During the ocean phase of their juvenile migration, Pacific salmon are susceptible to infection by sea lice (*Lepeophtheirus salmonis* and *Caligus clemensi*), which are native ectoparasites that feed on the surface tissue of their host (Wootten et al. 1982). Juvenile Pacific salmon normally have low infection levels of sea lice, especially the salmonid specialist *L. salmonis*, because these salmon are temporally and spatially separated from adult Pacific salmon (Krkošek et al. 2007a). However, in recent decades domesticated Atlantic salmon (*Salmo salar*) farmed in open-net pens in coastal British Columbia (BC) have provided year-round reservoirs for sea lice that allow substantial transmission of *L. salmonis* to juvenile Pacific salmon (Costello 2009a; Groner et al. 2016). For the generalist *C. clemensi*, there also exist other natural host infection reservoirs, such as Pacific herring (*Clupea pallasii*) (Morton et al. 2008; Beamish et al. 2009), which share nearshore coastal waters with juvenile salmon as spawners and larvae (Beamish et al. 2012).

In recent years, the generalist *C. clemensi* has infected over 98% of out-migrating juvenile sockeye salmon (*O. nerka*) from the Fraser River (Godwin et al. 2015; Godwin et al. 2017), an iconic set of Pacific salmon populations that forms Canada's largest sockeye run. Juvenile sockeye salmon that experience high infection intensity by *C. clemensi* exhibit reduced foraging success in a competitive and food-limited experimental setting (Godwin et al. 2015). Whether this translates to reduced foraging success in the wild is still unknown, but is the focus of this work. Here, I tested whether the relative stomach fullness (wet stomach content weight divided by body weight) of wild juvenile sockeye is lower when sea louse infection intensity is higher, which has implications for our understanding of how parasites mediate competition in fishes and potentially for the management of *C. clemensi* in BC.

Methods

Fish collection

I collected juvenile sockeye salmon in the wild as they migrated through western Johnstone Strait, BC, between May 26 and June 7, 2014 (Figure 3.1). At this point in their migration, the sockeye post-smolts have well-developed scales and average 114 mm in fork length. I used a hand-operated purse seine net (bunt: 27 x 9 m with 13 mm mesh, tow: 46 x 9 m with 76 mm mesh) that I set from a small (6 m) open boat. After

surrounding the fish, I brought the net next to the boat to form a pocket of sufficient width and depth to allow the fish to swim freely and minimize their contact with the mesh.

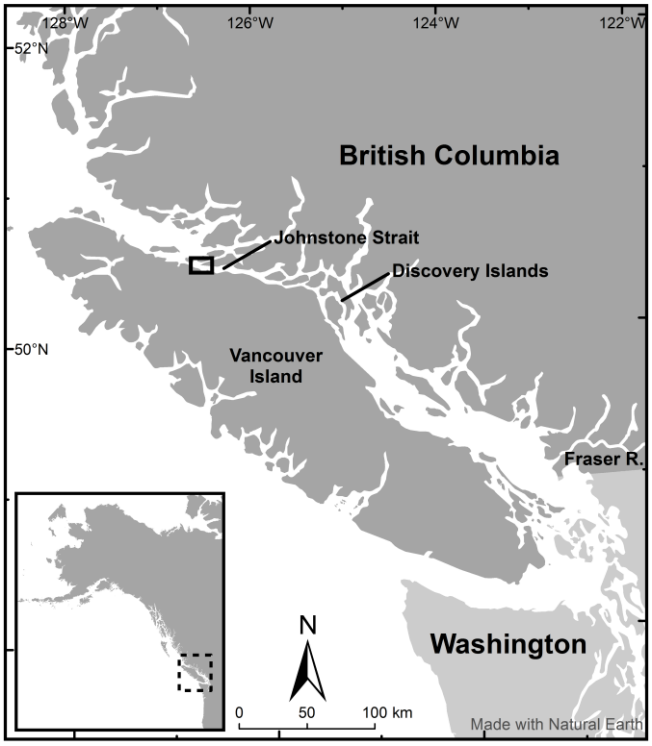

Figure 3.1 Map of study region.
The solid black box indicates the area in which salmon collections occurred.

I transferred captured fish from the net into an insulated fish tote (0.58 m deep and 0.97 x 0.55 m across) filled with fresh seawater. I moved fish from the net into the tote by dipping them and their surrounding seawater into a 3.79 L container (an inverted milk jug with the top capped and bottom cut off). This transfer method minimized or prevented sea louse detachment as fish were never exposed to air and there was minimal contact between the fish and sampling equipment (Godwin et al. 2015). All subsequent transfers were also performed using the same method. I used ice packs to regulate water temperature in the tote and aquarium bubblers to maintain adequate aeration. I transferred 50–100 sockeye into the tote during each capture event. See Table B.1 for collection locations, catch sizes, and oceanographic data.

Infection status assessment

I transferred sockeye individually from the tote into 13.2 L clear plastic aquaria and assessed them for sea louse infection by eye. If a fish appeared to be in one of my two infection categories (see below), I recorded that category and transferred the fish to an individual sterile 532 mL sample bag (Whirl-Pak® Write-On Bags; Nasco, Fort Atkinson, Wisconsin, USA) and euthanized it with an overdose of MS-222 (240 mg L^{-1}). After euthanizing a fish, I performed a full assessment of its infection status using a hand lens (Krkošek et al. 2005a) to confirm its infection category. Fish that were euthanized but found not to meet my infection category criteria were not used; such fish accounted for approximately 10% of euthanized fish and were usually identified initially to be in the lightly infected category by eye but then found to have too many small juvenile lice upon inspection by hand lens. I alternated between processing highly infected and lightly infected fish so as to not confound digestion time with infection status.

Larger, more developed sea lice have greater effects on their hosts (Wootten et al. 1982; Nendick et al. 2011; Jakob et al. 2013). I created infection categories that reflected this differential level of pathogenicity, so that small juvenile lice were not weighted equally to large adult lice (similar to Peacock et al. 2015). Sea lice initially attach to their host as copepodites, then develop through two (*L. salmonis*; Hamre et al. 2013) or four (C. clemensi; Kabata 1972) attached chalimus stages of increasing size before molting into their motile pre-adult and adult stages. Here, I consider individuals in their second *L. salmonis* chalimus stage or their third or fourth *C. clemensi* chalimus stages as 'large chalimus' sea lice; I also consider pre-adult and adult individuals as 'motile' sea lice. To weight the infection statuses of fish according to the development of lice infecting them, I defined a louse infection scale in which one large chalimus louse was equal to one infection unit, one motile louse was equal to two infection units, and copepodite and small chalimus lice were equal to zero. Because of the high infection prevalence observed (> 98%), I was unable to create a category for uninfected fish. Instead, I created a 'lightly infected' category in which all the fish had zero infection units and no more than three copepodite or small chalimus lice (Table 3.1). In all collections, the 'lightly infected' and 'highly infected' categories differed by a minimum of three infection units. See Table B.2 for the detailed sea louse infection data.

Table 3.1 Infection categories and sample sizes for each fish collection.

		Lightly infected		Highly infected	
Collection	Fish pairs	Infection scale	Max. lice	Infection scale	Min. lice
1	5	0	3	4	6
2	6	0	1	3	2
3	7	0	1	4	5
4	7	0	2	4	4
5	6	0	3	4	4
6	7	0	3	4	7
7	6	0	3	4	6
8	5	0	3	5	7
9	5	0	3	5	4
10	6	0	3	5	9
11	5	0	3	5	10
Average	**5.9**	**0.0**	**2.5**	**4.3**	**5.8**

The infection scale was weighted such that one large chalimus louse was equal to one infection unit, one motile louse was equal to two infection units, and copepodite and small chalimus lice were equal to zero units. For each collection, a single fish pair was comprised of one fish from the highly infected category and one from the lightly infected category.

Approximately one hour after capture, I released the remaining fish at the collection site. In each collection, I retained five to seven pairs of fish, each consisting of a lightly and highly infected sockeye. In total, I retained 130 juvenile sockeye salmon from across 11 collections to analyze stomach fullness in relation to infection status.

Zooplankton sampling and analysis

Immediately following the infection status assessments and subsequent release of fish, I collected zooplankton samples with a horizontal plankton tow at the fish capture site. Plankton tows were performed with a 0.5 m diameter plankton net with 250 µm mesh. The top of the net was kept 5-10 cm below the ocean surface, on average, and the tow lasted for 30 s. I attached a calibrated flow meter (General Oceanics, Miami, Florida, USA) at the mouth of the net to measure the volume of water sampled. I used horizontal tows instead of vertical ones because plankton samples near the surface are more similar to sockeye diets than those from deeper in the water column (Landingham et al. 1998) and my field observations indicate that juvenile sockeye frequently occupy the surface waters at the collection sites.

Each plankton sample was placed in a glass jar containing 250 mL of 10% formalin-seawater solution. Within three days of the collection, the samples were poured into a 63 µm sieve and rinsed with distilled water. The samples were then halved repeatedly using a Folsom plankton splitter (Aquatic Research Instruments, Hope, Idaho, USA) until approximately 200-250 individuals remained, after which they were transferred to a Bogorov counting tray (Wildlife Supply Company, Yulee, Florida, USA). From these samples, I identified and enumerated individuals from the high-level taxa previously found in juvenile Fraser River sockeye salmon stomachs (Price et al. 2013); these were termed 'sockeye prey'. These high-level taxa from Price et al. (2013) were: Copepoda, Brachyura, Oikopleura, Euphausiacea, Cladocera, Pteropoda, Decapoda, Amphipoda, Insecta, Cumacea, fish, and eggs. I calculated sockeye prey density by multiplying the number of sockeye prey in the Bogorov tray by the reciprocal of the splitting fraction, then dividing by the volume of water that passed through the plankton net.

Determination of relative stomach fullness

I used wet stomach content weight as my measure of stomach fullness and divided this by the fish's body weight to calculate relative stomach fullness. Following the field collections, I transported the euthanized fish on ice to a laboratory facility 45 min away by boat. Upon arrival at the laboratory, fish were analyzed in the original order of collection by dissecting them immediately to weigh the wet stomach contents. Stomachs were excised between the lower oesophagus and the pyloric sphincter, and their contents extruded with forceps and weighed, as in Godwin et al. (2015).

Statistical analysis

To determine which biological variables best explained juvenile sockeye salmon relative stomach fullness, I fit a suite of 13 mixed-effects models to my data (see Table B.3 for the full model set). My models included the biologically relevant combinations of five fixed effects: infection status, body size, prey density, the two-way interaction between infection status and body size, and the two-way interaction between infection status and prey density. I included infection status as a fixed effect to test my main hypothesis that relative stomach fullness decreases with high intensities of sea louse infection. I included body size and its interaction with infection status to account for any additional benefit of a larger body size to relative stomach fullness, and the potential decreasing

effect of infection with body size (Godwin et al. 2015). Finally, I included prey density and its interaction with infection status to account for the probable association between relative stomach fullness and prey density, and the possible decreasing effect of infection with increasing density of prey. Prey density and relative stomach fullness were centered and scaled by one standard deviation to allow the model-fitting optimizer to function correctly, since their variances were different by 10 orders of magnitude. Each model included a random effect of collection number on the intercept, which was determined *a priori* to account for the hierarchical structure of the sampling design, and a variance structure allowing for different variances in each collection to account for heteroscedasticity in the residuals. I performed model selection using Akaike Information Criterion corrected for small sample sizes (Hurvich and Tsai 1989) as my measure of model parsimony. I calculated relative variable importance (RVI) values based on the AICc weights.

I derived my measure of body size from a principal component analysis using three highly correlated body metrics: fork length, body depth, and weight. The first principal component explained 98% of the original variation in these metrics and so I used that as the variable representing body size in the statistical analyses.

I tested for differences in body size between infection categories using a two-sample *t*-test, and used linear regression to assess whether motile or overall louse abundance increased with body size for the highly infected fish. I completed all my analysis in R 3.2.1 (R Foundation for Statistical Computing, 2015) using the nlme and MuMIn packages.

Results

The juvenile sockeye salmon in my highly infected category were primarily infected by *C. clemensi* rather than *L. salmonis*, with 98.6% of the motile sea lice and 86.5% of the copepodid lice infecting these fish belonging to the former species. Neither motile abundance (R^2 = 0.009, d.f. = 63, p = 0.455) nor overall louse abundance (R^2 = 0.025, d.f. = 63, p = 0.207) increased with body size for fish in the highly infected category. Body size also did not differ between infection categories (t = 0.282, d.f. = 128, p = 0.779). The mean (± SE) zooplankton density across all collections was 941 individuals L^{-1} ± 141 individuals L^{-1}, and the mean sockeye prey density was 772 individuals L^{-1} ±

134 individuals L^{-1}, indicating that potential sockeye prey constituted 82% of the zooplankton community in the surface waters during my collections.

Lightly infected fish had higher relative stomach fullness than highly infected fish (Figure 3.2), and this trend held in each of the 11 collections (Figure 3.3). Infection status was the most important predictor of relative stomach fullness; of the 13 mixed-effects models, the top nine all included an infection status term, while none of the bottom four did (Table B.3). The importance of infection status to relative stomach fullness was corroborated by this predictor having the highest RVI (0.97), compared with body size (0.66), prey density (0.60), the interaction between infection status and prey density (0.16), and the interaction between infection status and body size (0.14). Regardless of their rank, all models with the relevant terms revealed that relative stomach fullness was higher for lightly infected fish, that it increased with body size, and that it increased with prey density. The top-ranked model included only an infection status term, but two other models were within 2 AICc units of the top model and therefore also had substantial support (Burnham and Anderson 2002; Table 3.2). One of these highly-supported models included a body size term, while the other included a predictor for prey density (Table 3.2).

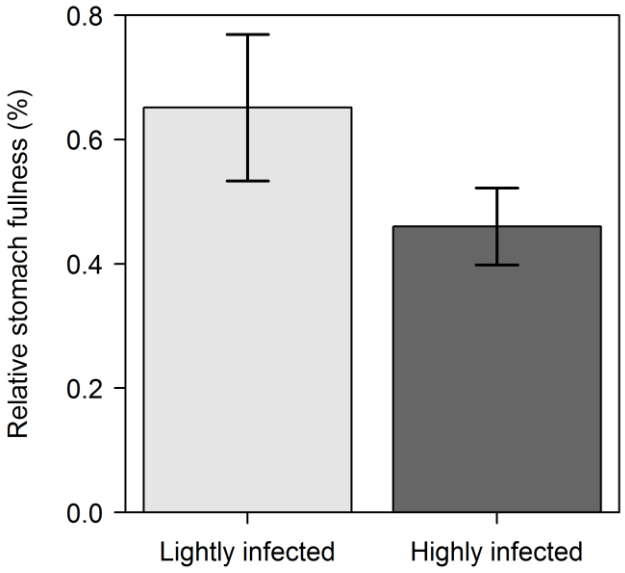

Figure 3.2 Relative stomach fullness of juvenile sockeye salmon for the two categories of sea louse infection.

Error bars indicate the 95% confidence intervals around the relative stomach fullness for each infection category.

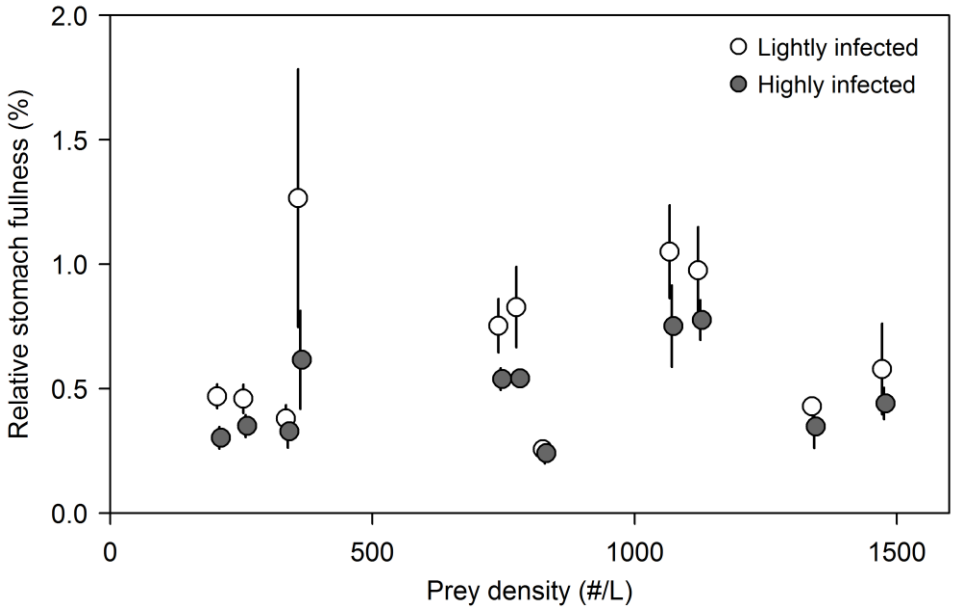

Figure 3.3 Mean relative stomach fullness (± SE) for fish in both infection categories of each collection.

Each vertical pair of points (one grey, one white) comprises a single collection. The relationship between relative stomach fullness and prey density had equivocal support in my model selection results (see Table S3).

Table 3.2 Model selection results for the six models of relative foraging success that accounted for at least 5% of model support.

Rank	Model	ΔAICc[a]	w_i[b]	R^2 [c]
1	*infection*	0	0.358	0.549
2	*infection + size*	1.24	0.192	0.530
3	*infection + prey*	1.66	0.156	0.542
4	*infection + size + prey*	2.66	0.095	0.510
5	*infection * size*	3.85	0.052	0.530
6	*infection * prey*	3.91	0.051	0.536

Relative stomach fullness was calculated as wet stomach content weight divided by body weight. The models included combinations of infection category (*infection*), body size (*size*; see description of principal component analysis), and prey density (*prey*) fixed effects. Each model included a random effect on the intercept for collection number. Interaction terms are distinguished with an asterisk symbol. See Table B.3 for the full model set and selection results.

[a] Difference from the top model AICc

[b] Akaike model weight

[c] R^2 for mixed-effects models calculated using the method developed by Nakagawa and Schielzeth (2013)

The highest-ranked model without an infection status predictor was 7.8 AICc units higher than the top model and accounted for only 0.7% of model support, as judged by AICc weights (Table B.2). The top-ranked model was 51 times more likely than the highest-ranked model without an infection status predictor, and its coefficients indicated that

highly infected fish had 17% ± 8% lower relative stomach fullness than lightly infected fish, on average.

Discussion

Theory and empirical evidence suggest that pathogens and parasites can influence host survival and population dynamics by modulating competitive foraging interactions (Hatcher et al. 2006). However, there is little evidence of parasite-mediated intraspecific competition in wild fishes, for whom intraspecific competition may be particularly intense due to fish aggregation and food patchiness. My results indicate that, for wild juvenile sockeye salmon, high levels of sea louse infection are associated with reduced relative stomach fullness. The juvenile sockeye used in this study were captured during their early marine migration from their natal freshwater systems, which based on the timing of capture and genetic analyses from previous studies (Groot and Cooke 1987; Price et al. 2011; Godwin et al. 2017) were mostly in the Fraser River watershed of BC.

While my stomach fullness data describe the quantity of prey consumed by sockeye, it should be noted that stomach fullness is not a true measure of foraging success or efficiency. Stomach fullness does not account for the energy densities or digestibility of prey, which vary among the zooplankton prey items that dominate the diet of juvenile sockeye (Lee 1974; Foy and Norcross 1999) as well as spatially and temporally with the availability of those items (Landingham et al. 1998; Tanasichuk and Routledge 2011; Mackas et al. 2013). Parasitized individuals can shift their diet to prey items of lower energy density or digestibility when they struggle to compete with unparasitized conspecifics for higher-quality prey (Milinski 1984). Since juvenile sockeye with heavy sea louse infection have lower competitive foraging abilities (Godwin et al. 2015), it is possible that they too shift toward capturing more prey items of lower quality. If that were the case, then by using stomach fullness data I produced conservative estimates of the differences in foraging success between infection categories.

Animal migration is generally demanding metabolically, so managing energy gain and depletion is vital for most migrating animals to avoid starvation or the sub-lethal effects of depleted energy reserves (Sapir et al. 2011). Unlike some migratory species, juvenile sockeye salmon forage during their migration, but this foraging is temporally variable because feeding opportunities are patchy (Parsons et al. 1970; McKinnell et al. 2014). In

regions with relatively high productivity, such as the northern Strait of Georgia, BC (Parsons et al. 1970; Masson and Peña 2009), no evidence of food limitation has been observed (Price et al. 2013). By contrast, the region in which I captured fish for this study, Johnstone Strait, has a sparse prey field due to strong tidal mixing (B. Hunt et al., Hakai Institute, unpublished data), which may cause high mortality for juvenile sockeye that enter the Strait with insufficient energy reserves (McKinnell et al. 2014). Sea lice levels on juvenile sockeye are also considerably higher in Johnstone Strait than in the more productive southern regions (Price et al. 2011; Godwin et al. 2015). Hence, the potential effects of sea lice on sockeye salmon growth and survival in Johnstone Strait are likely to involve the elevated abundances of the parasite itself, its effects on intraspecific competition, and the intensification of competition due to food limitation and the energy expenditure of migration.

My results provide equivocal evidence for an association between prey density and the relative stomach fullness of juvenile sockeye. If food competition is indeed higher in Johnstone Strait for sockeye than in nearby regions and that contributed to my finding that relative stomach fullness is associated with heavy sea louse infection, then I might have expected to find a stronger relationship between prey density and relative stomach fullness. Only one my three models with considerable support (as judged by AICc values; Burnham and Anderson 2002) contained a prey density predictor, which across my model set was my third most important predictor variable (RVI = 0.60). It is possible that prey density did not severely limit relative stomach fullness of sockeye in Johnstone Strait in 2014 even if food availability is commonly much lower there than in the Strait of Georgia (McKinnell et al. 2014; B. Hunt et al., Hakai Institute, unpublished data.). However, if the prey field was not limiting sockeye foraging rates then I might not expect to observe the differences in relative stomach fullness between infection categories that I did, since highly infected (and therefore less competitive (Godwin et al. 2015)) individuals would still have the opportunity to feed to satiation. It is also possible that the prey density estimates from my zooplankton collections did not represent the prey field encountered by the fish when they were feeding, due to patchiness in time or space (Parsons et al. 1970) or the strong tidal currents known to occur in Johnstone Strait (Sutherland et al. 2007). Most of the sockeye in this study must have eaten in the preceding eight hours (see Appendix B), which limits the potential effects of patchiness and currents on my results, but these remain possibilities that cannot be excluded.

Perhaps the most likely explanation for my equivocal evidence of a relationship between prey density and relative stomach fullness was that the sample size of 11 collection sites was too small to detect an effect. While my study design was suitable for looking at consistent differences in relative stomach fullness within groups, the sample size was limited for investigating variation among collections.

The relationship that I found between sea louse infection and relative stomach fullness is correlative, but the weight of evidence is building that sea lice have sub-lethal effects on important determinants of sockeye salmon survival. Sea lice are associated with Pacific salmon population declines (Connors et al. 2010; Krkošek et al. 2011b), but these declines cannot be explained by direct mortality alone, which may only be significant at small host sizes (Jones et al. 2008). However, sea lice also appear to have sub-lethal effects that influence mortality through their hosts' ecological interactions, for example by reducing swimming endurance (Mages and Dill 2010), increasing risk-taking behaviours (Krkošek et al. 2011a), and elevating predation (Peacock et al. 2015). For juvenile sockeye salmon specifically, individuals that are heavily infected by sea lice, primarily *C. clemensi*, have lower competitive foraging ability (Godwin et al. 2015), reduced body growth (Godwin et al. 2017), and actively attempt to dislodge these parasites by leaping (Atkinson et al. In press). Nonetheless, laboratory studies using experimental infections are needed to help differentiate two alternative interpretations of my results: 1) that sea lice abundance is a consequence rather than a cause of variation in relative stomach fullness; or 2) that sea louse abundance and relative stomach fullness are both correlated with (signals of) fundamental underlying fitness variation among individuals. I consider these alternative explanations to be unlikely because they both require sustained differences in relative stomach fullness that would have led to a difference in body size between the two infection categories, which was not observed. It is also striking that the observed louse-associated differences in relative stomach fullness occurred in each of the 11 collections (Figure 3.3), so any correlation between an underlying condition and sea louse infection would have to be very strong indeed.

In addition to my main result that heavy sea louse infection is associated with reduced sockeye stomach fullness, I also found moderate evidence that relative stomach fullness increased with body size. This result runs counter to the negative exponential relationship between relative stomach fullness and body size reported by Brett (1971) for juvenile sockeye, suggesting that larger juvenile sockeye may have a foraging

advantage due to their body size. The obvious potential mechanism for this is that smaller sockeye are prevented from foraging on larger (and possibly more abundant) prey due to gape limitation, which often, but not always, determines foraging success in young fish (Hargreaves and LeBrasseur 1986; Bremigan and Stein 1994; Devries et al. 1998; Scharf et al. 2000). Together with the negative relationship between infection status and relative stomach fullness, this potential effect of body size would be consistent with my previous findings that high infection intensities and smaller body sizes are associated with lower competitive foraging ability (Godwin et al. 2015).

Over 98% of the motile sea lice infecting the juvenile sockeye salmon were *C. clemensi*, but although these infections have been linked to open net-pen salmon farms (Price et al. 2011), there are currently no management actions directed at regulating this louse species on farms in BC. *Lepeophtheirus salmonis*, the main species of sea louse that causes fish mortality and financial loss to salmon aquaculture in the northern hemisphere (Mustafa et al. 2001; Johnson et al. 2004; Costello 2009b), is controlled on BC farms through application of in-feed parasiticide (emamectin benzoate; Saksida et al. 2010) when their abundance exceeds three motile lice per fish (Fisheries and Oceans Canada 2016a). While emamectin benzoate also reduces *C. clemensi* numbers, *C. clemensi* abundance is not directly managed. Since *Caligus* spp. are considered less pathogenic than *L. salmonis* (Johnson et al. 2004; Igboeli et al. 2014) and most of the research into the effects of sea lice on wild salmon has focussed on *L. salmonis* (e.g., Johnson et al. 1996; Krkošek et al. 2005b; Connors et al. 2010), there has been little reason to target *C. clemensi* with treatments on farms until now. However, given the mounting evidence for sub-lethal effects of *C. clemensi* on wild salmon, and the ability for *C. clemensi* to reach extreme abundances on farms without targeted treatment (e.g., 100% prevalence and 47.2 lice per fish (Di Cicco et al. 2017)), it seems prudent to start considering *C. clemensi* in the fish health management plans of farmed salmon in BC.

There is increasing concern over the potential impacts of *C. clemensi* on wild Fraser River sockeye salmon (e.g., Moore et al. 2017), especially in the context of the record-low Fraser sockeye returns in 2009 and 2016 (Pacific Salmon Commission 2016). Unlike *L. salmonis*, which can only infect salmonids, the generalist *C. clemensi* may have multiple sea lice reservoirs along the juvenile sockeye migration route, including Pacific herring, which may amplify extinction risk for salmon by allowing parasite abundances to remain high when an imperiled host population declines (De Castro and Bolker 2005)

and perhaps threaten herring stocks as well. My results shed further light on the sometimes subtle interactions between parasites and wild Pacific salmon (Miller et al. 2014; Peacock et al. 2014), and underscore the need to study not only the direct mortality from parasites but their sub-lethal effects as well. The impacts of parasite-mediated intraspecific competition on host survival and recruitment may be particularly influential for fishes, which often experience intense food competition and growth-dependent mortality, but there has been little to no work in this area. Competition is a fundamental driver of populations, and identifying how and when parasites mediate the competitive interactions of their hosts may be essential to understanding the host-parasite dynamics of many systems.

Chapter 4.

Reduced growth in wild juvenile sockeye salmon infected with sea lice[3]

Abstract

I examine daily growth rings in the otoliths of wild juvenile sockeye salmon (*O. nerka*) to determine whether infection by ectoparasitic sea lice (*Caligus clemensi* and *Lepeophtheirus salmonis*) was associated with reduced host body growth, an important determinant of survival. Over 98% of the sea lice proved to be *C. clemensi*, and my results suggest that highly infected fish may grow more slowly than uninfected individuals and that larger fish may grow faster than smaller fish. Finally, I found evidence of an interaction between body size and infection status, indicating the potential for parasite-mediated growth divergence.

Introduction

Pathogens are known to influence wildlife populations, especially when they interact with other stressors (Smith et al. 2009; Pacioni et al. 2015). Impacts on biota resulting from the spread of pathogens are commonly associated with anthropogenic activities, such as through the introduction of non-native species (Dunn 2009), climate change (Harvell et al. 2009; Maynard et al. 2015), and spill-over from domestic animal stocks (Daszak et al. 2000). Indeed, nearly one quarter of the planet's most threatening invasive species cause disease in invaded systems (Hatcher et al. 2012b), and epizootics from the wildlife trade alone are responsible for large-scale economic and conservation impacts (Karesh et al. 2005). Marine pathogens specifically can cause large population declines (e.g., Friedman et al. 2000; Hewson et al. 2014) and billions of dollars in losses for

[3] A version of this chapter appears as Godwin, S. C., Dill, L. M., Krkošek, M., Price, M. H. H., & Reynolds, J. D. 2017. Reduced growth in wild juvenile sockeye salmon infected with sea lice. Journal of Fish Biology 91(1): 41-57.

fisheries and aquaculture globally (Lafferty et al. 2015) in part because the absence of dispersal barriers facilitates rapid disease spread (McCallum et al. 2003).

Outbreaks of disease are often prominent and can lead directly to mass mortality events for host species (e.g., Lessios 1988; Skerratt et al. 2007), yet sub-lethal effects on host survival through traits such as growth, behaviour, and competitive ability are much less understood despite their potential importance to host survival. Growth is a particularly important component of fitness for juvenile fish, because it both expands their prey size range and it reduces predation risk (Parker 1971; Hargreaves and LeBrasseur 1986; see review by Sogard 1997). It has long been hypothesized that there is a 'critical period' in the early life history of marine fish that determines overall survival (Hjort 1914). Evidence for the critical period hypothesis has been mixed (Elliott 1989), and the concept is usually applied to larval mortality soon after yolk sack absorption (May 1974), but Pacific salmon (*Oncorhynchus* spp.) may well be a group of fish whose differential survival within and among year-classes is determined during their early marine life. The importance of early marine growth to overall survival has been demonstrated in coho (*O. kisutch*; Beamish et al. 2004), Chinook (*O. tshawytscha*; Duffy and Beauchamp 2011), pink (*O. gorbuscha*; Moss et al. 2005), and sockeye salmon (*O. nerka*; Farley et al. 2007).

For over a decade, conservation concerns have been raised about elevated levels of parasites - in particular sea lice - on Pacific salmon (Naylor et al. 2003; Krkošek et al. 2007b). Two main species of sea lice infect Pacific salmon: *Lepeophtheirus salmonis* (Krøyer 1837), a salmonid specialist (Pike and Wadsworth 1999), and *Caligus clemensi* (Parker & Margolis 1964), a generalist that also parasitizes other nearshore marine fish, including Pacific herring (*Clupea pallasii*; Parker and Margolis 1964). These ectoparasites feed on the epidermis and, in extreme cases, the musculature of host fish, and *L. salmonis* has been shown to alter host physiology (Wagner et al. 2003; Sutherland et al. 2011), influence behaviour (Krkošek et al. 2011a), and increase mortality (Morton and Routledge 2005; Krkošek et al. 2007b; Connors et al. 2010).

Most research on Pacific salmon and sea lice has focussed on *L. salmonis* and its effects on juvenile pink and chum salmon (*O. keta*; Jones et al. 2007; Brauner et al. 2012). Juvenile sockeye salmon migrating northward along the south coast of British Columbia, however, are primarily infected by *C. clemensi* (Price et al. 2011; Godwin et

al. 2015) and are post-smolts (having spent at least one year in freshwater before migrating to the sea) rather than young-of the-year. While direct mortality from sea lice is likely very low for sockeye salmon (Jakob et al. 2013), the sub-lethal effects of louse parasitism on these fish are unknown apart from an association with reduced competitive ability in highly infected fish (Godwin et al. 2015). Sockeye salmon are of particular interest because of their iconic status, their importance to fisheries (FAO 2015), and their complex multi-host dynamics with sea lice – *C. clemensi* infestations of wild juvenile sockeye are linked to domestic Atlantic salmon (*Salmo salar*; Price et al. 2011), and Pacific herring commonly infected as well (Morton et al. 2008; Beamish et al. 2009).

Investigations into the effects of pathogens on the growth of fish are scarce, with little research having been performed on salmonids (Speare et al. 1998) or outside of the laboratory (Sandell et al. 2015). For salmon and other teleost fish, body growth can be inferred from their otoliths - calcified structures in the inner ear whose incremental depositions are proportional to changes in fish length (Campana and Neilson 1985; Campana 1990). Here, otolith microstructure analysis was used to determine whether out-migrating wild juvenile sockeye salmon exhibit differential growth based on sea louse infection status. Specifically, it was hypothesized that i) highly infected juvenile sockeye grow more slowly than uninfected individuals, ii) larger fish grow faster than smaller fish, and iii) infection status and body size interact such that smaller fish experience a greater infection-associated reduction in growth compared with larger fish.

Material and Methods

Sampling design and fish selection

The wild juvenile sockeye salmon used in this study were collected for a separate ecological investigation (see Price *et al.,* 2011, 2013). These fish were collected from marine waters surrounding the Discovery Islands, between Vancouver Island and the mainland of British Columbia (BC), Canada (Figure 4.1). Collections occurred from May 28 to July 7, 2009 and June 4 to June 21, 2010 using a modified purse seine (70 m x 10 m with 6 mm mesh). After pursing the net, the catch was concentrated in the bunt of the seine, and fish were dip-netted out and euthanized with a swift blow to the head. The fish were immediately frozen individually and labelled for subsequent laboratory

analyses. Individual fish were thawed, weighed, measured for fork length, and assayed for sea lice using a dissecting microscope. Species of motile (i.e., pre-adult and adult) stages of sea lice were identified directly by morphology. Younger copepodid and chalimus stage lice were removed from the fish, mounted on slides, and examined under a compound microscope for species determination based on detailed morphology (Kabata 1972; Johnson and Albright 1991). In total, 2,401 juvenile sockeye salmon were captured over 79 collections (1,420 in 2009, and 981 in 2010).

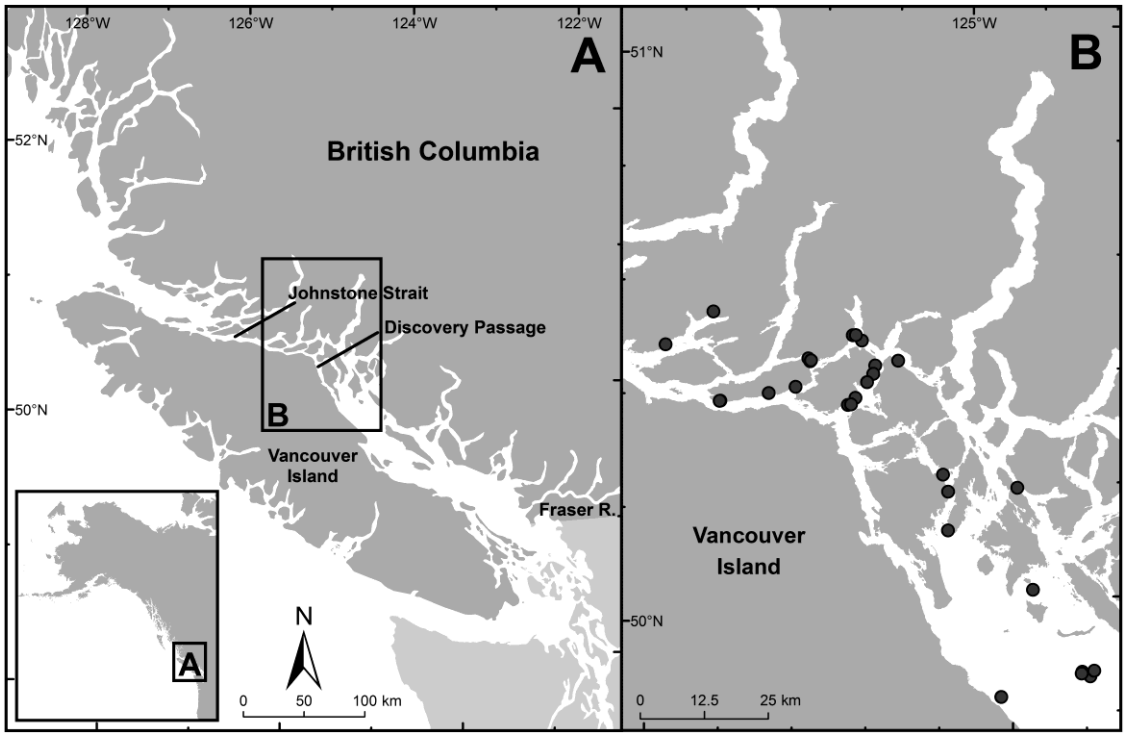

Figure 4.1 Map of the study area.
The extent of panel B is depicted by the black rectangle in panel A. Collection locations for out-migrating juvenile sockeye are shown in panel B as black points.

To select fish for otolith analyses, I excluded all collections that had fewer than two uninfected fish, or fewer than 10 total fish. From each of the remaining collections, I selected the two most highly infected sockeye, giving priority to number of motile lice, followed by the total number of lice. All of these infected fish had motile infection intensities at or above the 90th percentile intensity from the 2,401 sockeye captured, so hereafter they are termed ‘highly infected’. For each collection, I chose two uninfected fish by selecting those most similar in fork length to the two highly infected fish already selected. The mean difference in fork length between each pair of infected and uninfected fish (± SE) was 5.1 mm ± 0.8 mm, which was approximately 4.7% of overall

mean fish length (Table 4.1). The resulting dataset included 116 fish across 29 collections.

Table 4.1 Mean (± SE) fork length (mm) and sea louse infection status of size-matched juvenile sockeye salmon examined for otolith analysis.

Year	Infection status	n	Fork length	Motile lice	Total lice
2009	highly infected	26	106.5 ± 2.9	2.38 ± 0.29	8.04 ± 0.96
2009	uninfected	26	106.9 ± 2.1	–	–
2010	highly infected	32	109.9 ± 1.9	1.56 ± 0.16	4.66 ± 0.41
2010	uninfected	32	108.0 ± 2.0	–	–

Sea louse infection intensities are presented as combined intensities for *C. clemensi* and *L. salmonis*, but are dominated by *C. clemensi* (98.3%).

Otolith preparation and interpretation

I removed both sagittal otoliths from the selected fish and placed them in 2 cm^3 Eppendorf tubes pre-filled with 95% ethanol. For each fish, I chose one of the otoliths randomly and carefully cleaned it of soft tissue using distilled water and fine-tipped forceps under a dissecting microscope. Once clean, I mounted the otoliths sulcus side up on a glass microscope slide using a clear thermoplastic adhesive (Crystalbond 509-3; Aremco Products Inc., Valley Cottage, New York, USA). I then polished the mounted otoliths with wetted 30 µm and 3 µm lapping film (Digikey Corporation, Thief River Falls, Minnesota, USA) until the 10 outermost daily rings were visible on the pararostrum when viewed under a Leica DM1000 (Leica Microsystems Inc., Concord, Ontario, Canada) compound microscope at 400 x magnification (Figure 4.2). The polishing process was finished using wet 0.3 µm lapping film to remove any scratches caused by the coarser grit film. During this process, I photographed otoliths frequently at 400 x magnification using a Leica ICC50 HD camera to ensure that an image was obtained before over-polishing. If, after polishing, damage prevented at least 10 successive peripheral rings to be discerned, I discarded the otolith and the second otolith was used in its place; this occurred six times. Comparable methods for mounting and polishing juvenile sockeye otoliths were developed and validated independently by Stocks *et al.* (2014) and Freshwater *et al.* (2015a), and have since been used successfully in multiple ecological studies (Freshwater et al. 2015b; 2016).

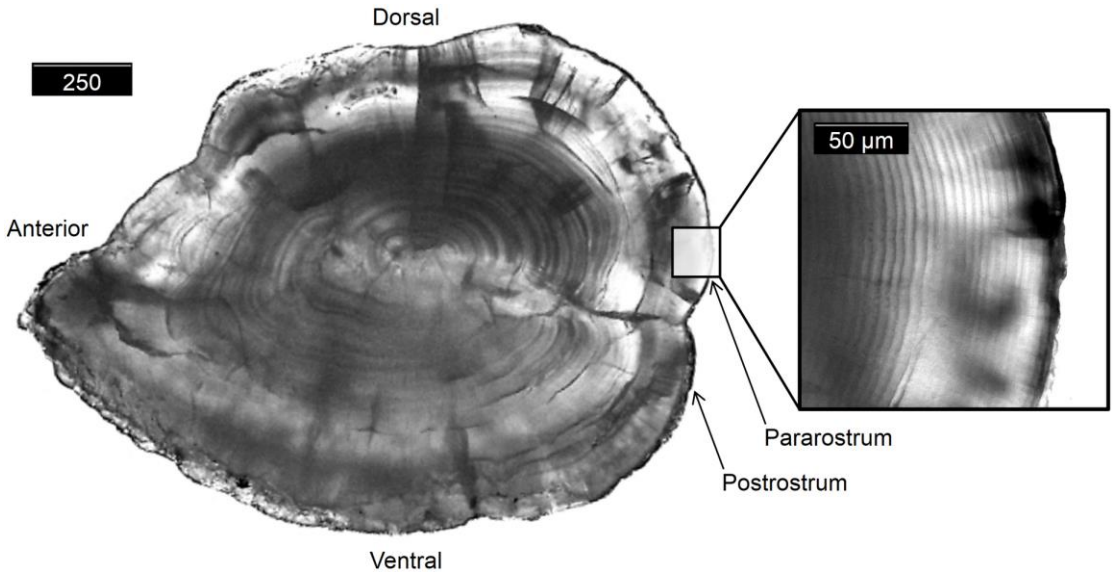

Figure 4.2 Photograph of a whole otolith after polishing (40 x magnification) and its daily growth increments (400 x magnification).

I quantified otolith growth for the 10 full days preceding capture by measuring the combined width of the 10 outermost daily increments. First, I photographed each prepared otolith with a scale bar in each image as a reference. I then used ImageJ 1.48v (National Institutes of Health, 2014) to measure the distance from the outermost ring on the pararostrum to the 10th ring inwards. I took measurements from the outermost ring rather than from the edge of the otolith to ensure that only full days of growth were considered. Finally, I measured the radius (the distance from the otolith's core to the farthest point on the pararostrum) at 400 x magnification for 30 otoliths using the same photographic and image processing software.

Statistical analysis

To confirm that otolith growth was an acceptable proxy for body growth in this study's juvenile sockeye salmon, I calculated the Pearson correlation coefficient between fork length and otolith radius for otoliths that had not been over-polished during preparation (n=30). I then estimated body growth for all 116 fish by multiplying their 10-day otolith growth by the slope of a linear regression between fork length (mm) and otolith radius (µm). I used two-sample *t-tests* to determine whether fish fork length differed between infection categories, both within and across years. I assessed whether louse abundance increased with body size using simple linear regression. To determine whether motile

abundance and total louse abundance differed between years on highly infected fish, I used two-sample Wilcoxon-Mann-Whitney non-parametric tests.

I tested the three main hypotheses by competing mixed-effects models with different combinations of infection status, fork length, and their two-way interaction as model variables. Because sea louse abundance was higher in 2009 than in 2010 for the highly infected fish (Table 4.1), I included a year variable to determine whether growth was reduced in 2009. For the same reason, I included the three-way interaction between infection status, fork length, and year to assess whether the size-mediated effect of infection status differed with year. I fit all 11 combinations of these fixed effects because all were considered biologically plausible. Each mixed-effects model had a random effect structure describing how the intercept varied according to collection number, which was determined *a priori*, as well as a power variance function structure needed to account for heteroscedasticity in the residual variation. I performed model selection using Akaike Information Criterion corrected for small sample sizes (AICc; Hurvich and Tsai 1989). I predicted that otolith growth would be higher in uninfected fish and larger fish, that the effect of infection on otolith growth would decrease with fish length, and that models including fish length, infection status, and their two-way interaction – and not year or the three-way interaction – would have the most support.

I estimated differences in body growth between highly infected and uninfected fish by generating model-averaged predictions for otolith growth and multiplying these by the rate of fork length growth per µm of otolith growth (i.e., the slope of Figure 4.3). These body growth estimates were used to calculate growth reductions for highly infected fish relative to uninfected fish of the same length by dividing the growth divergence between infection categories – the difference between the growth of uninfected and highly infected fish – by the growth of uninfected fish. All analyses were completed in R 3.1.1 (R Foundation for Statistical Computing, 2014) using the nlme, MuMIn, and AICcmodavg packages.

Genetic analyses

As part of the broader study from which these fish were obtained (Price et al. 2013), a subset of samples was genetically analysed to determine the population to which fish belonged. Forty-three of the 116 fish examined for otoliths had corresponding tissue

samples extracted for genetic analysis. Tissue samples were analysed at the Fisheries and Oceans Canada (DFO) molecular genetics laboratory in Nanaimo, BC (see Price *et al.*, 2011) for details on genetic analyses). Briefly, the 43 tissue samples had DNA extracted (Withler et al. 2000) and amplified using polymerase chain reaction; samples were assigned to source populations using a Bayesian mixed stock analysis (Pella and Masuda 2001) and a baseline of 85 sockeye populations when at least seven of 14 microsatellite loci amplified (Beacham et al. 2004).

Results

The majority (98.3%) of the sea lice in this study were *C. clemensi* rather than *L. salmonis*, which is consistent with previous reports (Price et al. 2011; Godwin et al. 2015). As only six of the 369 lice were *L. salmonis*, sea louse infection intensities were analysed and are presented as the combined *C. clemensi* and *L. salmonis* infection intensities.

Fork length was strongly correlated with otolith radius (r = 0.89; Figure 4.3), indicating that otolith growth is a reasonable proxy for body growth during this period of the sockeye salmon life cycle. The fork length of the size-matched fish did not differ between the two infection categories in 2009 (t = 0.098, d.f. = 50, p = 0.923) or 2010 (t = -0.712, d.f. = 62, p = 0.479), nor across the two years (t = -0.424, d.f. = 114, p = 0.672; Table 4.1). Louse abundance did not increase with body size (R^2 = 0.006, d.f. = 114, p = 0.400). The abundance of motile lice on the highly infected fish was higher in 2009 than in 2010 (U = 546, p = 0.034; Table 4.1), as was total louse abundance (U = 594, p = 0.005; Table 4.1).

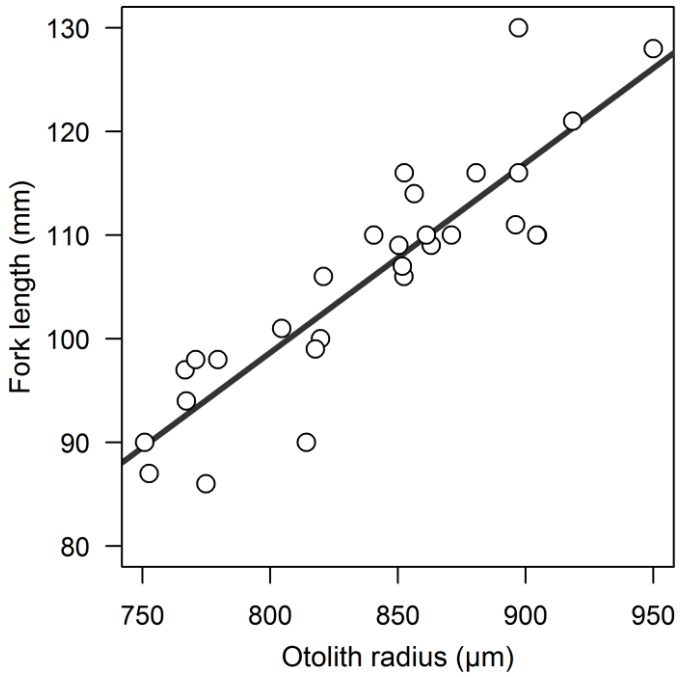

Figure 4.3 Sockeye smolt fork length in relation to otolith radius.
The equation of the line is: $y = -47.74 + 0.18x$.

Otolith growth was best explained by fork length and infection status (Table 4.2). As predicted, I found evidence that larger fish grew faster than smaller fish, and uninfected fish grew faster than highly infected fish (Figure 4.4). No single model had overwhelming support; instead, the top three models were all within 1.27 AICc units of each other, and the third-ranked model still accounted for 16% of overall model support. The model in which otolith growth was affected both by fish size and infection status was 1.89 times more likely than the model in which otolith growth was only affected by fish size.

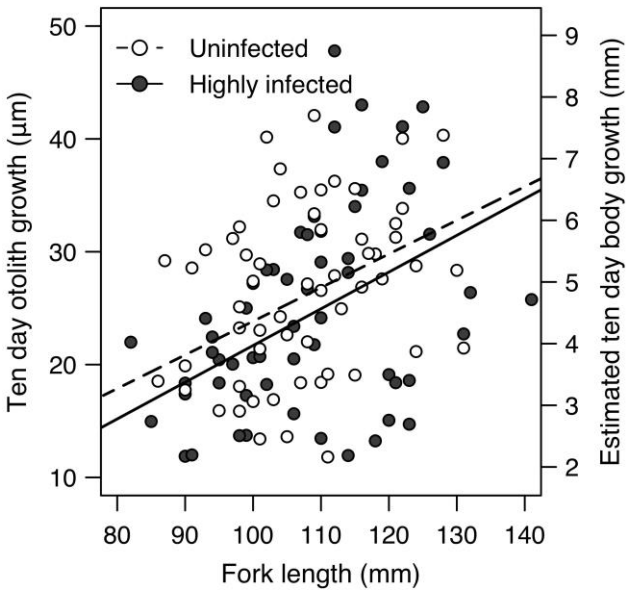

Figure 4.4 Ten-day otolith growth increases with fork length for uninfected and highly infected juvenile sockeye.

Estimated 10-day body growth is represented on the secondary y axis. Lines depict the model-averaged predictions.

Table 4.2 Mixed-effects model selection results for otolith growth in juvenile sockeye.

Rank	Model[a]	ΔAICc[b]	w_i[c]
1	*infection + length*	0	0.310
2	*infection * length*	0.48	0.244
3	*length*	1.27	0.164
4	*infection + length + year*	1.97	0.115
5	*infection * length + year*	2.17	0.105
6	*length + year*	3.39	0.057

Only the six models with greater than 1% model support are shown.

[a] Models had a random intercept for collection number. The fixed effects used were infection category (infection), fork length (length), their two-way interaction, year, and the three-way interaction.

[b] Difference from the top model AICc

[c] Akaike model weight

Daily growth divergence between infection categories was evident in the model-averaged predictions. These predictions indicated that 90 mm uninfected fish – those in the 10th percentile of fork length – grow at the same rate as 97.4 mm infected fish. However, the effect of infection diminished slightly as body size increased, supporting the hypothesis for a size-mediated effect of infection. For example, the model-averaged predictions suggested that 125 mm uninfected fish – those in the 90th percentile of fork length – grow at the same rate as 128.7 mm infected fish; thus, highly infected 90 mm

sockeye experience an 11.6% reduction in daily body growth relative to uninfected fish. In comparison, highly infected 125 mm fish only experience a 4.7% reduction, and median-sized (105 mm) fish experience a 7.9% reduction (Figure 4.5).

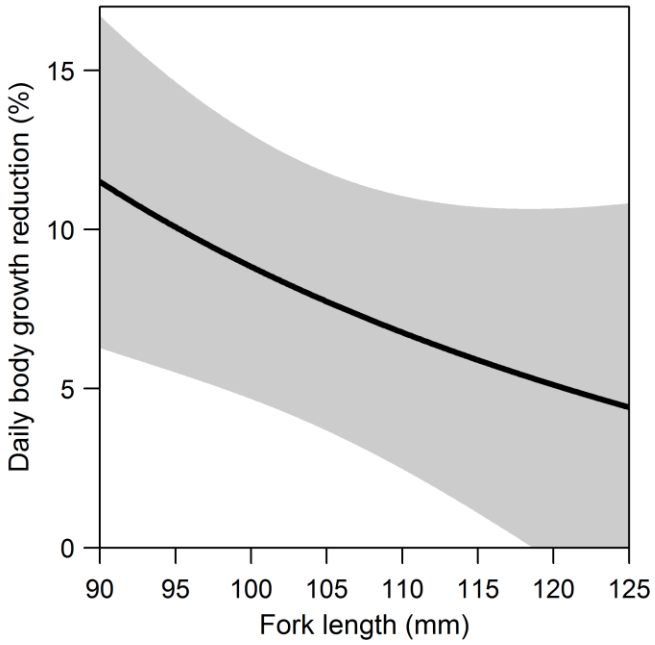

Figure 4.5 Size-dependent reductions in daily body growth for highly infected juvenile sockeye relative to uninfected fish.
Growth estimates for each infection category were calculated by generating model-averaged predictions for otolith growth and multiplying these by the rate of fork length growth per µm of otolith growth (i.e., the slope of Figure 4.3). Grey area denotes the 95% confidence region, which incorporates the unconditional standard errors of averaged predictions and the standard error of *R*, the rate of fork length growth per µm of otolith growth.

Genetic analyses indicated that the majority (91%) of the 43 fish examined were from 16 spawning systems within the Fraser River: Chilko (42%), Weaver (9%), Pitt (7%), Dolly Varden Creek (7%), Adams (2%), Birkenhead (2%), Bowron (2%), Cultus (2%), Nadina (2%), Nahatlatch (2%), North Thompson (2%), Raft (2%), Stellako (2%), Tachie (2%), and Taseko (2%). The remaining 9% were from the Phillips River system in the Discovery Islands region.

Discussion

Pacific salmon may experience a critical period in their development where early marine growth determines overall survival, and my results suggest that pathogens may play a role in modulating that growth. My analysis of otolith rings from juvenile sockeye salmon

indicates that larger fish, and those uninfected by sea lice, may grow faster than their smaller and highly infected counterparts.

I evaluated the growth history of juvenile sockeye over the 10 days preceding capture. I chose ten days as the duration for three reasons: 1) the 10 most recent daily rings could consistently be exposed on each of the otoliths, 2) little to moderate turnover of sea lice would be expected from development alone in 10 days (Hogans and Trudeau 1989; Johnson and Albright 1991; Piasecki and MacKinnon 1995), and 3) *C. clemensi* can disperse among hosts in its pre-adult and adult stages, so a longer time period would result in a higher chance that a host's infection status at the time of capture will not represent its infection status throughout the entire time period.

Juvenile sockeye generate daily rings in their otoliths (Wilson and Larkin 1980), but otolith formation stops when fish are reared below 5 ºC (Marshall and Parker 1982), at least in freshwater. This cessation of daily ring formation under the influence of low temperature is coupled with a greatly reduced rate of body growth. Since the fish captured were feeding actively (Price et al. 2013) and travelling through waters well above 5 ºC (Price et al. 2011), I do not expect daily ring formation to have ceased prior to capture.

I cannot know for certain that the relationship identified between sea louse infection and growth rate is a causative one. Lice could differentially infect and survive on hosts that have lower initial body condition and could therefore be an indicator, rather than a cause, of condition differences among fish. While future laboratory research could come closer to identifying a causative relationship between sea louse infection and fish growth (e.g., Tveiten et al. 2010), the issue of condition differences among fish would still confound such studies, and the effect in wild fish could be much different depending on how sea lice influence interacting factors that are absent in the laboratory. For example, juvenile sockeye have lower competitive abilities with sea louse infection (Godwin et al. 2015), which should influence foraging outcomes and, ultimately, growth. Field studies are therefore necessary to place such host-pathogen relationships within an ecological context.

Sea lice can cause salmon to reduce their feeding (Costello 2006; Bravo et al. 2008), which could cause reduced growth in their hosts. For Atlantic salmon infected with *L.*

salmonis, this reduced feeding is only a temporary phenomenon when lice are at the pre-adult stage – the first motile stage – and host feeding recovers by the time lice moult into adults (Dawson et al. 1999). I am not aware of any similar studies involving *C. clemensi*. Nevertheless, if the infected fish experienced a temporary reduction in feeding due to a heavy infection of pre-adult lice, it is possible that the observed reduction in growth was also temporary. This is unlikely considering that 89% of the motile lice on this study's fish were adults, not pre-adults, but even if pre-adults were causing these fish to feed less, it would not be a temporary phenomenon. As 70% of the lice on the infected fish were non-motile (i.e., would later moult into pre-adults), these fish would likely not resume full feeding for several weeks under this scenario.

My results suggest that there may be a growth difference between infection categories of juvenile sockeye salmon despite a presumed lack of food limitation for these fish. Price *et al.* (2013) found empty stomachs in only 3.1% of juvenile sockeye migrating through the Discovery Islands in 2009 and 0.0% of them in 2010. More recently, McKinnell *et al.* (2014) proposed a 'trophic gauntlet hypothesis' in which juvenile sockeye migrating through Johnstone Strait, the body of water that they enter after migrating northward through the Discovery Islands, suffer an energy deficit due to a lack of prey. Although there is some overlap between the sampling area and the region described by McKinnell *et al.* (2014), growth differences would be expected to be accentuated further north where prey may be less available if differential foraging success between uninfected and highly infected fish contributes to the differences observed.

It is not surprising that larger juvenile sockeye salmon grow faster than smaller individuals; bigger fish are more likely to catch bigger prey (Hargreaves and LeBrasseur 1986) and hunt more efficiently due to higher visual acuity (Flamarique and Hawryshyn 1996) and faster burst swimming speed (Hale 1999). If this size-dependent growth is not a product of the fish being on different parts of a growth curve with an inflection point – in which case their body sizes could eventually converge – there may be growth divergence in this sockeye life stage. Growth curves for younger sockeye captured and reared in freshwater do not indicate an inflection point having already been reached (Brett et al. 1969; Bilton and Robins 1973; Brett and Shelbourn 1975), but to my knowledge no high resolution size-at-age data exist for out-migrating juvenile sockeye in marine waters. It may be that this relationship between age and increasing variance in

body size is the incipient phase of what ultimately becomes differential survival among individuals.

The effects of infection status and body size were not independent, as infection interacted with heterogeneity in body size to amplify divergent growth in smaller fish and dampen it in larger individuals. Model-averaged predictions indicated that highly infected fish in the 10th percentile of fork length experience an 11.6% reduction in daily body growth relative to uninfected fish, but median-sized fish experience a 7.9% reduction and those in the 90th percentile only experience a 4.7% reduction. Since infection intensity did not increase with host body size, this diminished infection-associated growth reduction in larger sockeye may be due to these fish hosting fewer parasites per unit mass. These results highlight one potential scenario in which small amounts of divergent growth due to parasitism may be subsequently reinforced and amplified by size-dependent divergent growth. Sea lice are associated with reduced competitive abilities in juvenile sockeye (Godwin et al. 2015), and this connection between sea lice and growth may demonstrate a second, delayed, effect of heavy sea louse infection on survival.

The belief that 'bigger is better' generally holds true with fish (Sogard 1997), and Pacific salmon are no exception (Beamish et al. 2004; Moss et al. 2005; Farley et al. 2007). The growth reductions that I observed in highly infected fish (see also Appendix C) are comparable to those shown to influence survival in early marine coho salmon. Calculations based on data in Beamish *et al.* (2004; their Figure 1) indicate that early marine growth was 9.5% lower for the average coho salmon soon after marine entry than for those that survived through their first marine winter. Juvenile sockeye and coho salmon have different early life histories, but both species usually enter the ocean after spending one year in freshwater. While the two species are not directly comparable, the growth reductions reported here in sockeye salmon are in the same range as those demonstrated to have survival consequences for their congener, which lends credence to the notion that these growth reductions could affect overall marine survival of sockeye. Furthermore, when projecting my results over 120 days, the resulting 6.2% difference in fork length for fish initially of median size (see Appendix C) appears similar to the size difference reported by Farley *et al.* (2007) to be associated with a decrease in marine stage survival rate of a few percent for juvenile sockeye.

In light of the growing evidence that *C. clemensi* infection is correlated with components of fitness for wild juvenile salmon, and the fact that this species constitutes 98.3% of the lice observed on juvenile sockeye, it is worth considering why there is no management plan for this louse species on farmed salmon in BC. In contrast, *L. salmonis* are managed on BC salmon farms using an in-feed parasiticide (emamectin benzoate (‘‘SLICE’’); Intervet/Schering-Plough Animal Health, Boxmeer, The Netherlands; Saksida et al. 2010). As mandated by their Licence for Finfish Aquaculture, marine farms perform a SLICE treatment when there is an average of at least three motile *L. salmonis* on a subset of their domestic fish, but there is no such threshold for *C. clemensi*. *Caligus clemensi* infestations of wild juvenile sockeye are associated with open net-pen salmon farms along their migration route (Price et al. 2011), and infection prevalence in wild juvenile sockeye continues to be extremely high (Godwin et al. 2015). There seems no obvious reason for this discrepancy in treatment between the two louse species. Additional parasiticide treatments on farms for *C. clemensi* outbreaks could increase risk of louse resistance (Denholm et al. 2002; Bravo et al. 2008; Igboeli et al. 2012), but at minimum, a combined threshold should be established in which *C. clemensi* and *L. salmonis* are considered together when determining when treatment should occur.

Fraser River sockeye salmon constitute a suite of populations under intense scientific and public scrutiny. After a two-decade decline in productivity, record low adult returns in 2009, and a $37 million federal judicial inquiry into the causes of the decline, Fraser River sockeye briefly showed a modest rebound in productivity (Fisheries and Oceans Canada 2015). Concern remains, however, as returns in 2016 were the lowest in over 100 years (PSC, 2016). Furthermore, while no single cause of the long-term decline in Fraser River sockeye productivity was identified during the federal inquiry (Cohen 2012c), the decline was correlated with competition with pink salmon and exposure to salmon farms during early marine migration (Connors et al. 2012). This has led to a call for research on the factors governing juvenile sockeye survival, particularly in regard to pathogens (Cohen 2012c). As 91% of the 43 fish genetically tested in this study were from the Fraser River, and 37% of these were from conservation units with amber (of concern) or red (threatened) statuses (Grant and Pestal, 2012), the results presented here provide insight into a potential pathogen-induced survival consequence for this salmon stock-complex.

Any potential effect of sea lice on juvenile sockeye is of particular concern given that *C. clemensi* is a generalist with multiple possible reservoir host populations. By infecting Pacific herring and domestic Atlantic salmon, *C. clemensi* can be maintained in the environment regardless of juvenile sockeye abundance, thus sustaining infection pressure even at low sockeye densities and creating the apparent competition structure that is most commonly associated with disease imperilment of wildlife (De Castro and Bolker 2005). The results presented here highlight the need to consider the sub-lethal effects of marine pathogens on host populations in addition to the traditional focus on direct mortality, as this may be vital for understanding wild fish survival and, ultimately, for conserving at-risk populations.

Chapter 5.

Parasite monitoring and management on North Pacific salmon farms

Abstract

Science can help mitigate threats to the environment by informing environmental policy, but often the effectiveness of these policies is poorly understood. Several marine policies are now in place to reduce the environmental effects of the marine aquaculture industry, whose rapid global expansion has triggered concerns about disease transmission between farmed and wild aquatic organisms. Open-net salmon farming has been at the forefront of these concerns, particularly due to the spread of parasitic sea lice (primarily *Lepeophtheirus salmonis* and *Caligus* spp.) and the resulting impacts on wild salmon (*Oncorhynchus* spp. and *Salmo salar*). I analyzed three critical issues for policy regulating parasite control on farmed salmon by modeling the population dynamics of sea lice using industry self-reported data. First, I found the first evidence that parasites with different levels of specialization can spread from multiple wild host species to farmed fish, and that the amount of transfer can be quite high. Next, I showed that bias can occur when industry self-reports data that are relied upon by policy. Finally, I demonstrate that current policy for parasite control does not have the capacity to remain effective in face of environmental change. My results illustrate several methods by which the effectiveness of environmental policies can be undermined and suggests potential solutions for improving future effectiveness.

Introduction

Informing environmental policy is a key pathway through which science helps moderate threats to the environment (Moore et al. 2018). Scientific evidence can identify environmental problems and trigger implementation of new policy (e.g., Schindler 1974; Farman et al. 1985; Carson 2002), or it can inform existing policies when their scientific basis is questionable (e.g., Hernandez and Kempton 2003; Artelle et al. 2013). Recently, research has focused on policy aimed at adaptation to future environmental change (Mawdsley et al. 2009; Ford et al. 2010; Grafton 2010), but the adaptive capacity of

existing policies to environmental change is rarely analyzed. This ability of environmental policies to remain effective under varying environmental conditions (hereafter termed 'resilience') will likely become more important in a changing global climate (Nelson et al. 2007; Berkes 2007), perhaps especially so in coupled human and natural systems already sensitive to human impacts (Walker et al. 2004).

Many policies rely on self-reported data from industry to detect and mitigate environmental problems. For example, national policies commonly rely on industry to self-monitor pollution discharge or to self-report violations of pollution standards (Livernois and McKenna 1999; Shimshack and Ward 2005; Barla 2007). Industry self-reporting enables monitoring programs that are otherwise infeasible due to costs or logistics (Gunningham and Rees 1997) and they provide opportunities for companies to demonstrate cooperation with regulatory authorities (Helland 1998). Still, an obvious question remains: can industry self-reported data be trusted? Audits, inspections, and threats of legal actions and financial penalties help maintain the accuracy of self-reported data from industry (Gray and Shimshack 2011), but the incentives for inaccurate self-reporting can be high (Gunningham and Rees 1997). Despite these incentives, the accuracy of self-reported data from industry is rarely investigated (but see De Marchi and Hamilton 2006 and Li et al. 2017, for example).

In recent decades, numerous marine policies have focussed on reducing the environmental impacts of aquaculture, an industry whose rapid expansion (termed the blue revolution) has resulted in a shift towards farming of unprecedented speed (Duarte et al. 2007). Although this shift may lead to welcome relief for certain over-exploited marine ecosystems, it can bring additional stressors in the form of emerging infectious diseases (Daszak et al. 2000), as did terrestrial agriculture before it (Harwood 1990). Aquaculture facilities can act as disease reservoirs that provide persistent infection pressure to wildlife even at low host densities, which may lead to elevated extinction risk for wildlife (De Castro and Bolker 2005; Krkošek et al. 2013b). Implementing effective policies that manage disease and allow aquaculture and marine wildlife to coexist, while sustaining a productive seafood supply, is imperative.

Of the many types of aquaculture, salmon farming has raised some of the greatest concerns in terms of its impacts on wildlife. Salmon farms typically raise hundreds of thousands of Atlantic salmon (*Salmo salar*) in open-net pens (Orr 2007) and operate in

the same nearshore marine waters through which wild salmon (*Oncorhynchus* spp. or *S. salar*) migrate (Ford and Myers 2008). Consequently, pathogens and parasites are easily transmitted between farmed and wild salmon (Krkošek 2017). For example, parasitic sea lice (primarily *Lepeophtheirus salmonis* and *Caligus* spp.) are thought to spread from wild salmon to farmed salmon when the wild adults return to coastal waters in the fall, after which they amplify on the farms over the winter and transfer back onto the migrating wild juveniles in the spring (Krkošek et al. 2007a; Krkošek et al. 2010; Marty et al. 2010). Some sea louse species have broad host ranges, which may result in additional parasite reservoirs and increase risk for wild salmon (De Castro and Bolker 2005), but it is not known whether non-salmonid hosts (e.g., Pacific herring, *Clupea pallasii*) actually play a role in louse transmission to farms. In the absence of salmon farms, juvenile salmon typically have low infestation rates of sea lice (Costello 2009a) and when they become infested they can suffer high levels of direct (Morton and Routledge 2005; Jones et al. 2008) or indirect mortality (Peacock et al. 2015; Godwin et al. 2017; Godwin et al. In press). This juvenile mortality due to sea lice reduces wild adult salmon returns in Europe (Krkošek et al. 2013a; Vollset et al. 2016) and potentially in North America (Krkošek et al. 2011b).

British Columbia (BC), Canada is the only region in the world that is a large global producer of both farmed and wild salmon (Groner et al. 2016). In BC, treatments to reduce sea lice almost exclusively involve use of an in-feed parasiticide called emamectin benzoate (trade name SLICE®; Saksida et al. 2010). Salmon farms themselves collect and self-report the sea louse count data used to determine infestation rates on the farms, and the Canadian federal fisheries department (colloquially referred to as DFO) occasionally conducts scheduled audits of the industry counts. Chapter 1 provides more background information on the local context.

I present a comprehensive analysis of the spatial and temporal variation in parasites on salmon farms in Pacific Canada, and I use this information to explore three key issues for aquaculture science and management that have relevance to other environmental policies. First, I clarify the disease ecology of this multi-host system by estimating, for the first time, transmission from wild to farmed hosts for parasite species with different levels of specialization. Second, I evaluate whether industry self-reporting results in underestimation bias in monitoring data relied upon by policy. Third, I test the resilience of current parasite control policy to changing environmental conditions. I address these

questions by analyzing a time series of industry self-reported parasite data and government audit data from 91 farms (Figure 5.1) over six years, using a hierarchical model and Bayesian fitting methods.

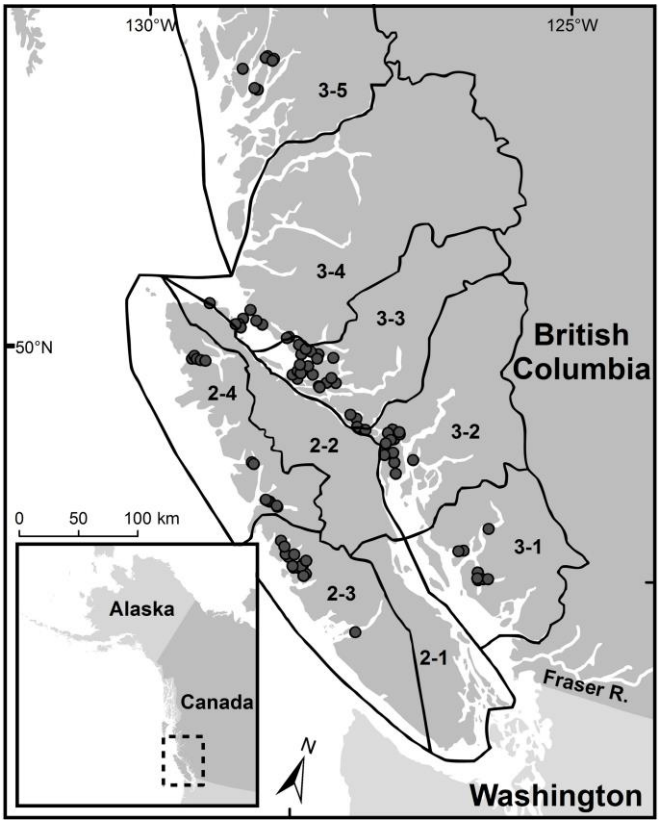

Figure 5.1 Locations of the 91 BC salmon farms in the industry sea lice dataset that were active (i.e., stocked with fish) for at least one month between 2011 and 2016.

Solid black lines demarcate the boundaries of DFO fish health surveillance zones, each of which is identified by number (e.g., 3-1, 3-2, etc.).

Methods

Data

I used the publicly available sea lice data collected by aquaculture industry staff on active salmon farms in BC from 2011 through 2016 (Fisheries and Oceans Canada 2017a). These data are monthly averages of industry louse counts on individual farms; data from individual sampling events or individual fish are not publicly available. During each sea lice sampling event, farm staff capture stocked fish by seine net in three net pens and collect at least 20 fish by dip net from each seine (Fisheries and Oceans

Canada 2016a). One of the net pens is a reference pen that is assessed in every sampling event, and the other two are selected randomly (Fisheries and Oceans Canada 2016a). The collected fish are then placed in an anaesthetic bath of tricaine methanesulfonate (TMS, or MS-222) and assessed for lice by eye. The sea louse counts concentrate on the more pathogenic and mobile pre-adult and adult life stages (hereafter termed 'motile') rather than the attached stages earlier in development. For each farm in the dataset, between 0 and 6 sea lice sampling events were conducted each month (mean = 1.64 events month^{-1}), and 99.1% of these months had 1 to 3 sampling events.

For every mean monthly motile louse count, the industry dataset includes the month and year of the count, the number of sampling events contributing to the count, the age class of the stocked fish, a farm facility identifier, the farm's fish health surveillance zone (Figure 5.1), whether chemical delousing treatment occurred in that month, and the previous month's mean louse count. I excluded mean monthly counts that were missing any of this information (n = 440). My final dataset comprised 2,626 mean monthly louse counts over six years, from seven health zones and 91 farms (Figure 5.1).

In an attempt to ensure the quality of industry sea lice data, DFO performs intermittent, pre-arranged audits of industry sea lice monitoring. The farm facilities to be audited are chosen randomly each month, and the audits are then scheduled to coincide with one of those farms' scheduled sampling events that month (Fisheries and Oceans Canada 2017b). During the audits, fish are selected in the same manner as for non-audited sampling events and divided equally between farm staff and DFO personnel for independent counting (Fisheries and Oceans Canada 2017b). Audit timing data are publicly available (Fisheries and Oceans Canada 2017c), and I therefore knew whether or not an audit was associated with each of the mean monthly louse counts. Audits occurred in 7.7% of the month-farm combinations in the dataset.

To assess relative effects of wild fish populations on farm sea louse counts, I estimated relative annual abundance indices of wild Pacific salmon (*Oncorhynchus* spp.) and Pacific herring (*Clupea pallasii*) adults for each DFO fisheries management area (different than the health zones; Government of Canada 2007) that contained at least one farm in my dataset. For the salmon abundance indices, I used DFO's estimates of returning adults for every salmon population in the New Salmon Escapement Database System (NuSEDS; Fisheries and Oceans Canada 2017d) that had data in all six years

(2011-2016). ‘Population’ is defined in NuSEDS as each unique combination of spawning stream, species, and run timing. I rescaled these annual abundance estimates (by one standard deviation) within each management area to obtain an annual index of adult salmon relative abundance for each area. The number of populations that contributed to each management area’s salmon abundance index ranged from five to 85 (mean = 21), and the species composition of these contributing populations varied among areas (Table D.1). I considered this acceptable for two reasons: 1) I rescaled the annual NuSEDS estimates within management areas, not among them, and 2) my abundance indices draw from the most data-rich populations, which are likely to be indicator streams for their management areas (Price et al. 2017). For Pacific herring (*Clupea pallasii)*, I used the DFO’s annual herring spawn index for each management area (Cleary and Thompson 2017) and scaled these in the same manner as for the salmon estimates. Since returning wild salmon and spawning herring are only present in certain months of the year, my model only applies the abundance indices in the relevant months (see below).

Model

I analyzed the publicly available industry sea lice dataset to determine relationships between several potential predictors and farm louse counts in BC. Specifically, I assessed the potential effects on farm louse counts of local wild salmon and herring abundance, DFO audits, and delousing treatment. I accounted for the effects of month, year, and fish health surveillance zone – which incorporate variation in environmental conditions – as well as farmed salmon age class and louse density dependence.

I fit a hierarchical model to the farm louse count data using a Markov chain Monte Carlo (MCMC) approach (see Table 5.1 for an overview of the model notation). Despite using Bayesian tools to facilitate fitting, I employed uninformative priors to focus on patterns indicated by the data. My model was divided into two nearly identical parts – one for *L. salmonis* and one for *C. clemensi* – and it was fit to all the data simultaneously. For simplicity, I present only the *C. clemensi*-focused components of my model and note where the *L. salmonis*-focused components deviated from these. I fit my single model and interpret its results rather than performing model selection on smaller models because the complexity of the model is justified by the size of the dataset and because

all the parameters have strong biological justifications (see Neal (2012) and Gelman et al. (2013) for discussion of this method of Bayesian model inference).

Table 5.1 Overview of model notation.

Symbol	Description	Data or prior details
	Response and continuous predictor variables	
N	motile louse count per ten fish	discrete count
S	local Pacific salmon abundance index	continuous
H	local Pacific herring abundance index	continuous
	Indices for predictor variables	
t	index for timestep (month)	72 months (January 2011 - December 2016)
f	index for farm	91 farms (all active BC farms in 2011 - 2016)
treat	index for treatment status	3 treatment statuses (treated in t, in t-1, or not)
zone	index for fish health surveillance zone	7 health zones (all health zones for the 91 farms)
year	index for year	6 years (2011 - 2016)
month	index for month-of-year	12 months-of-year (January - December)
class	index for age class of farmed fish	2 age classes (< 1 year in sea, > 1 year in sea)
area	index for management area	9 areas (all areas for the 91 farms)
	Model predictions and parameters	
μ	predicted motile louse count per ten fish	
λ	per-capita louse population growth rate	
γ	louse colonization rate	
τ	effect of delousing treatment	fixed, $U(0,100)$
α	effect of DFO audit	fixed, $U(0,100)$
ρ	negative binomial shape parameter	$U(0,100)$
	Sub-model predictions and parameters	
η	linear function for λ ($\lambda = e^{\eta}$)	
η_i	coefficient in η, associated with predictor i	fixed, all $U(0,100)$
φ	varying-coefficients term for farms in η	random, $U(0,100)$
β	linear function for γ ($\gamma = e^{\beta}$)	
β_i	coefficient in β, associated with predictor i	fixed, all $U(0,100)$
ψ	varying-coefficients term for farms in β	random, $U(0,100)$
	Subscripts	
L	subscript for *L. salmonis*	
C	subscript for *C. clemensi*	
o	subscript for observed louse count	
s	subscript for local wild Pacific salmon	
h	subscript for local wild Pacific herring	

In the industry dataset, the mean monthly counts are rounded to the nearest tenth, effectively rendering them discrete data. If I were to treat the counts as continuous data, the gamma distribution would be the natural choice to describe their variation, but the data also include a high proportion of zero counts. To overcome this, I model monthly louse counts per ten fish (i.e., I multiplied the mean monthly counts by ten) and assume a negative binomial error distribution:

$$N_{C,t,f} \sim negative\ binomial\ (\mu_{C,t,f}, \rho_C), \tag{5.1}$$

where $N_{C,t,f}$ is the observed mean motile louse count per ten fish in month *t* on farm *f*, $\mu_{C,t,f}$ is the predicted mean motile louse count per ten fish for that month and farm, and ρ_C is the negative binomial shape parameter, fit as an additional free parameter. This prevents complications associated with zeros, because unlike the gamma distribution, the negative binomial distribution allows for zero counts. I note that the standard error (and therefore the distribution) of the mean monthly louse counts will, in fact, be affected by the number of fish assessed on a farm in any given month, but this information is not available, and I use the negative binomial distribution as an empirical approximation of the true underlying distribution.

At their most basic, my models take the form:

$$\mu_{C,t+1,f} = N_{C,t,f}\lambda_{C,t,f} + \gamma_{C,t,f}, \tag{5.2}$$

where the mean motile count in month *t* ($\mu_{C,t+1,f}$) is predicted by the sum of intrinsic on-farm dynamics and external colonisation pressure. The on-farm dynamics are the product of the previous month's count on that farm ($N_{C,t,f}$) and a per-capita population growth rate ($\lambda_{C,t,f}$) affected by on-farm conditions, such as louse density and treatment status. The colonization rate ($\gamma_{C,t,f}$) is a function of extrinsic factors, such as wild-host abundance. Both the per-capita population growth rate and the colonization rate are modelled as transformed linear functions, where $\lambda_{C,t,f} = e^{\eta_{c,t,f}}$ and $\gamma_{C,t,f} = e^{\beta_{c,t,f}}$; thus $\eta_{c,t,f}$ and $\beta_{c,t,f}$ are analogous to linear predictors in generalized linear mixed models (GLMMs) with a logarithmic link function. Note that my model assumes key population processes can be captured by considering only motile lice, ignoring details of early developmental stages. But while my model does not explicitly consider larval lice, those that colonize as larvae and develop into motiles are still captured in the

colonization-rate component of my model. I also implicitly model the influence of environmental conditions on development time between larval attachment and the motile stage (~27 days for *L. salmonis* at 10°C (Johnson and Albright 1991) and unknown length for *C. clemensi*) in the growth-rate component. I do this by including spatial and temporal predictors (i.e., health zone, month, and year) that are strongly correlated with temperature and salinity (Pickard and McLeod 1953; Fisheries and Oceans Canada 2017e) – important drivers of development timing for *L. salmonis* (Johnson and Albright 1991). While it would be more direct to use actual temperature and salinity measurements from the farms, I was unable to obtain these data.

I allow delousing treatment to influence monthly louse counts, including the motiles that developed over the month, as:

$$\mu_{C,t+1,f} = e^{\tau_{C,treat,t,f}}(N_{C,t,f}\lambda_{C,t,f} + \gamma_{C,t,f}), \quad (5.3)$$

where $e^{\tau_{C,treat,t,f}}$ is a proportional mortality term for farm *f* in month *t* that results from delousing treatment. The exponent takes one of three levels: zero if treatment last occurred more than one month before *t*, or one of two levels to describe louse decline when treatment occurred in month *t* or *t*-1, corresponding to the two-month effectiveness previously described for emamectin benzoate (Lees et al. 2008; Saksida et al. 2010).

To account for the potential effects of DFO audits on industry louse counts, I extend the model such that

$$\mu_{C,t+1,f} = e^{\tau_{C,treat,t,f}} e^{\alpha_{C,t+1,f}} \left(\frac{N_{C,t,f,n}}{e^{\alpha_{C,t,f}}}\lambda_{C,t,f} + \gamma_{C,t,f}\right), \quad (5.4)$$

where $e^{\alpha_{C,t+1,f}}$ allows for a proportional change in a farm's counts in month *t* (or *t*+1, as appropriate), if indeed they are influenced by whether the DFO audits a farm. The exponent, $\alpha_{C,t,f}$, takes the value of 0, if no audit occurs, or a fitted estimate, if an audit occurs. When an audit does occur in month *t*, that month's observed louse count ($N_{C,t,f,o}$) is rescaled by $e^{\alpha_{C,t,f}}$ to account for any louse count observation error associated with audits.

The linear predictor for per-capita population growth rate in a given month and farm ($\ln(\lambda_{C,t,f})$ from Equation [2]) takes the form:

$$\eta_{c,t,f} = \eta_{C,0} + \eta_{CC,class}N_{C,t,f} + \eta_{CL,class}N_{L,t,f} + \eta_{class,t,f} + \eta_{C,zone,f} + \eta_{C,year,t} + \eta_{C,month,t} + \varphi_{C,f}, \quad (5.5)$$

The first term is an intercept ($\eta_{C,0}$) that defines growth rate at base factor levels (*class* = < 1 year in sea, *zone* = 2-3, *year* = 2011, *month* = January) and louse counts of zero for *C. clemensi* and *L. salmonis* abundance in the previous month. The next two terms represent the interspecific and intraspecific density dependence on a farm's per capita louse count in month *t*; they each incorporate a farm's louse counts in month *t-1* ($N_{C,t-1,f}$ and $N_{L,t-1,f}$) and a coefficient that describes density dependence due to either *C. clemensi* ($\eta_{C,C,class}$) or *L. salmonis* ($\eta_{C,L,class}$). These coefficients depend on the age class of the farm's stocked fish in month *t* (farm and month subscripts not shown), which can take one of two levels: fish that have spent less than one year in seawater, and fish that have spent greater than or equal to one year in seawater. Age class also directly affects the per-capita growth rate of a farm in month *t* ($\eta_{class,t,f}$) because fish surface area may influence louse survival (Tucker et al. 2002). There are three additional coefficients for categorical covariates: the age class of the fish for a given month and farm ($\eta_{class,t,f}$), the health zone of the farm ($\eta_{C,zone,f}$), and the year ($\eta_{C,year,t}$) and month ($\eta_{C,month,t}$) of the louse count. Among other things, these coefficients represent spatial and temporal variability in temperature and salinity. The last term in [5] is a varying coefficient (hereafter termed a 'random effect' to continue the parallel between my sub-models and GLMMs) describing how the intercept varies among farm facilities.

I modelled the linear predictor for a farm's colonization rate in month *t* such that

$$\beta_{C,t,f} = \beta_{C,0} + \beta_{class,t,f} + \beta_{C,zone,f} + \beta_{C.year,t} + \beta_{C,h,month}H_{year,area} + \beta_{C,s,month}S_{year,area} + \psi_{C,f}, \quad (5.6)$$

where $\beta_{C,0}$ is an intercept term describing the colonization rate at zero wild salmon and herring abundance, zero counts for *C. clemensi* and *L. salmonis* in the previous month, and base factor levels (*class* = <1 year in sea, *zone* = 2-3 and *year* = 2011). The age class of the farm's fish ($\beta_{class,t,f}$), the farm's fish health surveillance zone ($\beta_{C,zone,f}$), and the year of the count ($\beta_{C,year}$) affect colonization rate in the same manner that they affected per-capita growth rate in [5]. Next, there are two 'wild fish' terms that describe the effects of wild Pacific herring ($\beta_{C,h,month}H_{year,area}$) and wild Pacific salmon

($\beta_{C,s,month}S_{year,area}$) in a given year and management area. Adult wild salmon and wild herring spawners do not aggregate in their management area's marine waters year-round, so I only consider the effects of salmon and herring in select months of the year. Since I do not know the monthly abundance of herring and salmon in each management area, I set the coefficients for these terms to be zero in all months except those in which herring and salmon are most likely to be present and influencing farm louse counts. Pacific herring in BC typically spawn in February, March, and April (Benson et al. 2015), so I fit herring coefficients for each of these months plus May, to account for April colonization by larval lice that would develop and be enumerated in the May motile louse counts. July, August, and September experience the highest numbers of adult Pacific salmon in the marine environment (Groot and Margolis 1991), so I fit salmon coefficients for these months plus October. The *L. salmonis* model does not include a herring term because *L. salmonis* is a salmonid specialist (Pike and Wadsworth 1999). Finally, I include a random effect on the intercept of farm facility to account for the hierarchical nature of the data ($\psi_{C,f}$) while limiting the number of farm facility parameters in my model.

The full equation for the predicted mean motile louse count is as follows:

$$\mu_{C,t+1,f} = \overbrace{e^{\tau_{C,treat,t,f}}}^{\text{Treatment (5.3)}}\ \overbrace{e^{\alpha_{C,t+1,f}}}^{\text{Audit (5.4)}}\left(\overbrace{\frac{N_{C,t,f,n}}{e^{\alpha_{C,t,f}}}}^{\text{Rescale count (5.4)}}\ \overbrace{e^{\eta_{C,0}+\eta_{CC,class}N_{C,t,f}+\eta_{CL,class}N_{L,t,f}+\eta_{class,t,f}+\eta_{C,zone,f}+\eta_{C,year,t}+\eta_{C,month,t}+\varphi_{C,f}}}^{\text{Population growth rate (5.5)}} + \underbrace{e^{\beta_{C,0}+\beta_{C,C,class}N_{C,t-1,f}+\beta_{C,L,class}N_{L,t-1,f}+\beta_{class,t,f}+\beta_{C,zone,f}+\beta_{C.year,t}+\beta_{C,h,month}H_{year,area}+\beta_{C,s,month}S_{year,area}+\psi_{C,f}}}_{\text{Colonization rate (5.6)}}\right), \quad (5.7)$$

where the over- and under-braces reference the previously described equations and where the predicted mean motile louse count is the mean of the negative binomial probability density function in [1].

I used uninformative priors (uniform between -100 and 100) for all of my parameters except for a single $\alpha_{zone,c}$ coefficient. I constrained this parameter's uniform prior between -10 and 10 because the *C. clemensi* data for this particular fish health surveillance zone (zone 3-1) included two drastic month-to-month declines in mean louse counts that caused fitting complications. This constraint had no effect on the modal parameter estimate for $\alpha_{zone,c}$. I fit separate variance parameters for the two farm-facility

random effects ($\varphi_{C,f}$ and $\psi_{C,f}$), in addition to a parameter describing the correlation between the two. The random effects were each drawn from a multivariate normal distribution with a mean of zero and a covariance matrix determined by an inverse-Wishart distribution, which was in turn informed by the three random-effect parameters (Gelman and Hill 2007). Despite their complexity, I needed to fit these random effects to account for the hierarchical nature of the data while also avoiding fitting 180 separate fixed-effect parameters.

My analysis was performed using R 3.2.1 (R Core Team 2015) and JAGS 4.3.0 (Plummer 2017) with the R package R2jags 0.5-7 (Yu-Sung Su and Yajima 2015).

Simulations

To test the resilience of current parasite control policy to environmental change and assess the performance of other potential policy options, I used my model results to simulate louse counts under three management scenarios. The first scenario was the current parasite management strategy employed by BC farms, in which delays between simulated threshold-breaking counts and subsequent treatments were drawn from Poisson distributions using the observed mean delay for that month. The second scenario was one in which delousing treatment always occurred in the same month as the threshold-breaking count. The third scenario was the same as the first, but with an additional obligatory treatment in February. I simulated these three management scenarios for both an average year (i.e., all years except 2015) and for 2015, when sea surface temperatures were exceptionally high in the northeast Pacific (Peterson et al. 2015; Bateman et al. 2016).

I focussed on *L. salmonis* for this exercise because *C. clemensi* counts are not currently a consideration for parasite management on BC farms and because the audit effect was so strong for *C. clemensi* that simulating *C. clemensi* counts under existing conditions might not be meaningful. To reflect reality, I did not allow farms to treat in consecutive months.

I simulated counts over a calendar year, starting with the mean observed count in December of the previous year. For each iteration of the simulation, I drew the parameter estimates from one of my MCMC samples. I used the mean of the observed

values for the simulation data, except for treatment status, which was adjusted according to louse counts on the simulated farms and the simulated management scenario. I ran the simulation 10,000 times.

Results and Discussion

I present and discuss my results according to the three topics of concern outlined above. After describing the temporal trends from the raw data, I show that parasites with different levels of specialization can spread from wild salmonid and non-salmonid populations to salmon farms, and that the amount of transfer can be quite high. Next, I demonstrate that bias can occur in industry self-reported data relied upon by policy. Finally, I establish that current policy for parasite control on salmon farms has low resilience to rising ocean temperatures. Appendix D presents model diagnostics (Figure D.1-6 and Table D.2) and the results that pertain primarily to sea louse population ecology (i.e., the modeled effects of density dependence, farmed fish age class, and the spatial and temporal variables; Figure D.7-8).

Temporal trends

The data show temporal variation that is consistent with Canada's underlying (but not explicit) regulatory goal of minimizing sea lice on farms during the wild juvenile salmon migration (Peacock et al. 2013; Fisheries and Oceans Canada 2016a). Two species of sea lice are counted on salmon farms in BC: *L. salmonis* and *Caligus clemensi.* On average, *L. salmonis* counts were lowest during the wild juvenile salmon migration period (April to June), which was just after the highest frequency of delousing treatments (Figure 5.2). In contrast, mean *C. clemensi* counts were lowest in the fall when *L. salmonis* counts were at their peak (Figure 5.2). The *C. clemensi* counts are likely severe underestimates (Saksida et al. 2007), because these sea lice can readily leave their hosts (Jones and Nemec 2004; Saksida et al. 2015), especially when the hosts are handled or exposed to netting or air (Butterworth et al. 2006; Atkinson et al. In press). The intra-annual patterns were mostly consistent among years, with the notable exception of 2015, when sea surface temperatures in the northeastern Pacific Ocean were abnormally high (Peterson et al. 2015). In that year, counts were on average 3.1 times higher for *L. salmonis* during the salmon migration than in the other years, and 1.5

times higher for *C. clemensi* (Figure D.9). Temperature is a strong determinant of louse development rate (Johnson and Albright 1991), and the data suggest that when sea surface temperatures are higher, the risk for sea louse outbreaks increases dramatically

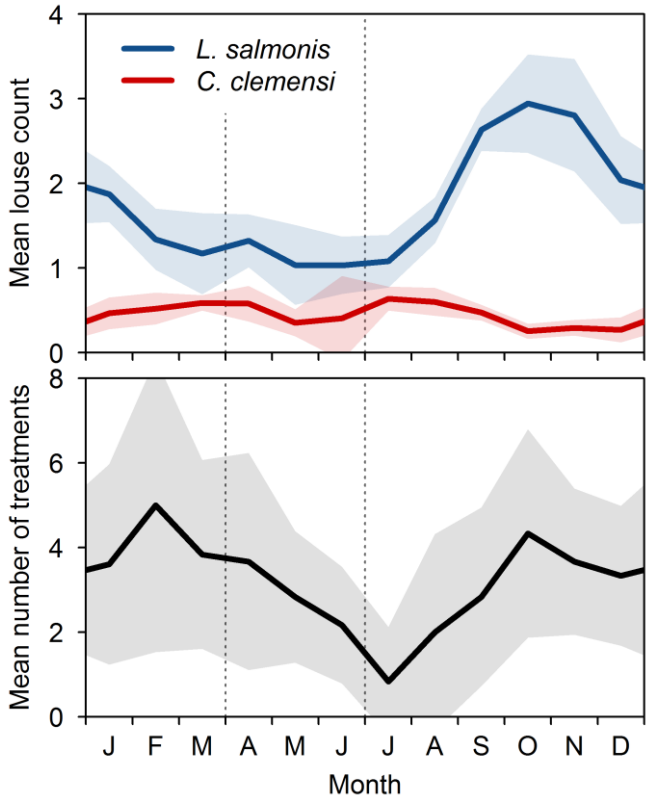

Figure 5.2 Monthly variation in the observed mean motile louse counts (top panel) and the mean number of delousing treatments (bottom panel) between 2011 and 2016 in BC.

Shaded regions depict the 95% confidence intervals for the mean. Dashed vertical lines delineate the wild juvenile salmon migration period (April to June). I show the monthly means connected by lines for visual effectiveness, but I note that these are not continuous data.

Wild fish as sources of sea lice to farms

My results indicate that wild adult salmon and herring function as natural reservoir host populations from which sea lice spread to salmon farms. Local wild salmon abundance had a positive effect on *L. salmonis* and *C. clemensi* colonization rates and overall counts in August (Figure 5.3), suggesting that wild salmon help spread not only salmonid specialists to farms, but also generalist parasites as well. These results corroborate and expand on a previous correlation that linked wild salmon abundance with *L. salmonis* infestation rates on farms in one region (Marty et al. 2010) by assessing the timing and strength of the effect.

I also show, for the first time, that a non-salmonid species can act as a source of parasites to salmon farms, as local wild herring abundance had a positive relationship with *C. clemensi* colonization in April (Beamish et al. 2009). It has long been speculated that non-salmonids are involved in the dynamics of parasite populations on farms (Jones et al. 2006; Beamish et al. 2009), but never confirmed. It is possible that some regions in BC have resident herring populations that provide year-round *C. clemensi* infestation pressure, but these populations are less common than the migratory ones (Beacham et al. 2008) and I lack the data to estimate their abundance. Also due to data limitations, I was unable to account for fish migrating through management areas in which they do not eventually spawn.

The impacts of disease in multi-host systems depend on the level of specialization of the pathogen or parasite. Specialist parasites with density-dependent transmission are unable to drive focal host species to extinction because their population densities track their hosts', but generalist parasites can avoid these host-density thresholds by persisting in the environment via other host reservoirs even when focal host population densities are low (De Castro and Bolker 2005). Salmon farms shift the relationship between *L. salmonis* and wild salmon to the generalist situation by providing a potential year-round reservoir of hosts for *L. salmonis* that does not exist naturally (Krkošek et al. 2007a; Costello 2009a), and my results reveal that this reservoir is supplied externally through the transmission of parasites from wild to farmed salmon in August. I also provide a rare empirical example of a generalist parasite (i.e., *C. clemensi*) spreading from more than one host species (i.e., wild salmon and herring) to a domesticated host reservoir. If a parasite has a negative population-level effect on the host, additional reservoirs should theoretically decrease the probability of parasite eradication and increase extinction risk for a focal host species (De Castro and Bolker 2005).

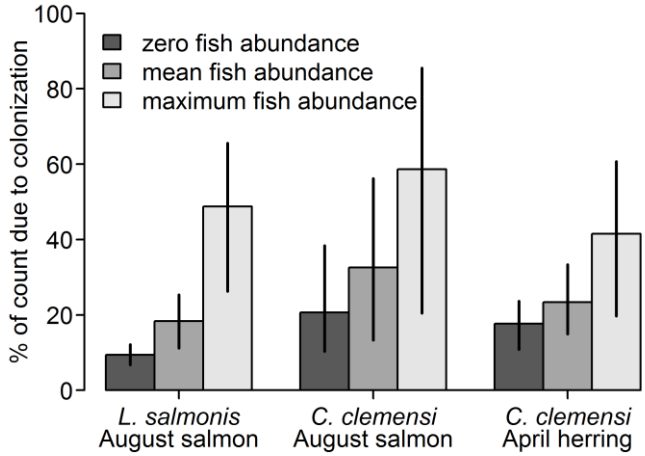

Figure 5.3 Influence of colonization on sea louse counts in the months that my model showed positive effects of fish (i.e., salmon or herring) abundance.

For each positive fish effect (e.g., the effect of wild salmon on *L. salmonis* counts in August), three levels of local wild fish abundance are presented. Predictions were made using the parameter values from the MCMC samples under the conditions that treatment last occurred more than two months ago and that the farm was not audited during the month in question. All other predictors were set to the mean of observed values. The remaining percentage of the sea louse counts is from population growth.

Bias in sea louse counts on salmon farms

Industry sea louse counts are biased downwards. In months when DFO performed its pre-arranged audits of industry sea louse counts (see methods for details), the industry's mean monthly *C. clemensi* counts were approximately 1.94 (95% credible interval: 1.58, 2.42) times higher than in months when DFO did not audit (Figure 5.4), after accounting for all the other variables in my model. Using the estimates for this *C. clemensi* audit effect and for the conservative background rate of *C. clemensi* underestimation (30%; Saksida et al. 2007), I expect that the true *C. clemensi* abundances on farms are at least 2.5 times higher than the reported counts. For *L. salmonis*, counts in audit months were 1.17 (1.04, 1.28) times higher than in non-audit months. The actual bias in *individual* counts (i.e., audited vs. non-audited counts) is probably much greater because the count data are reported as monthly means. Bias in audit counts was thus diluted in the pool of each month's counts (mean = 1.64 counts month^{-1}).

The natural solution to the bias in the industry's self-reported data would be for these monitoring data to be collected by an independent third-party instead. While third-party monitoring is often discussed as an optimal solution to ensure accurate industry data (e.g., Gunningham and Rees 1997), regulatory transitions from self-monitoring to third-

party monitoring are rare and to my knowledge the amount that they improve data accuracy remains unassessed. Other options for increasing data accuracy include improving data collection training for industry staff (Dasgupta et al. 2000) and performing audits or inspections without advance notice *after* data collection takes place, so that data are always collected with the risk of subsequent review (Laplante and Rilstone 1996). In the case of parasite management on salmon farms, the protocol for counting sea lice could also be improved by eliminating the use of dip nets, which would ensure that sea lice do not abandon their hosts before counts are performed. The current protocol requires high diligence to minimize or retrieve dislodged sea lice; this is especially true for *C. clemensi* (Butterworth et al. 2006; Atkinson et al. In press), which could explain why *C. clemensi* are more underestimated than *L. salmonis*.

Self-reported data are often thought to reflect reality because incentives exist for accurate self-reporting. For example: 1) self-reporting is done under substantial surveillance (Short and Toffel 2010), 2) audits or inspections are performed without advance notice (Helland 1998; Shimshack and Ward 2005), 3) misreporting is met with administrative, legal, or financial penalties for the employer and/or employees (Shimshack and Ward 2005; Gray and Shimshack 2011), 4) accurate data are easy to obtain (Gunningham and Rees 1997; Gray and Shimshack 2011), or 5) industry is not penalized when self-reporting demonstrates violations to regulations (Livernois and McKenna 1999). None of these conditions exist for sea louse counts by salmon farms; in particular, self-reported violations (i.e., *L. salmonis* counts above three lice per fish) result in the farm having to perform a costly delousing treatment or harvest its fish earlier than it would otherwise. My results suggest that when incentives for accurate self-reporting are not strong, bias can occur in self-reported industry data used by environmental policy.

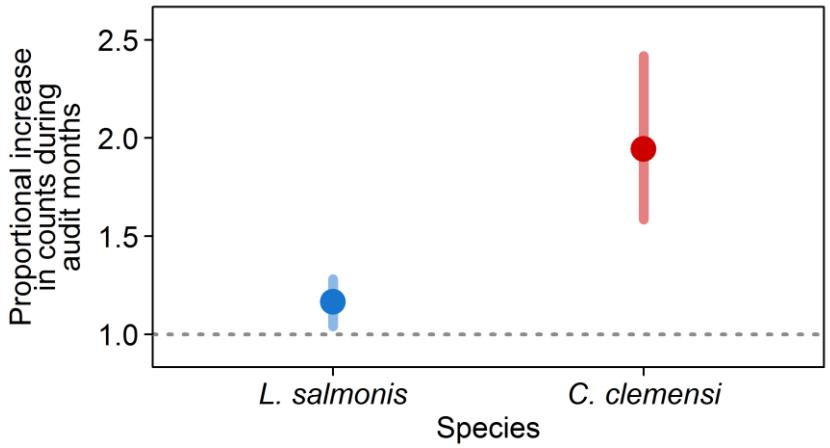

Figure 5.4 Proportional increase in farm louse counts (with 95% credible intervals) in months when DFO audited farms.
The horizontal dashed line indicates the value at which there would be no difference in counts between months with and without a DFO audit.

Delousing treatment policy: effectiveness, compliance, and resilience

Nearly every salmon farming region in the world is contending with resistance of sea lice to chemical delousing treatments (Westcott et al. 2008; Aaen et al. 2015). Recent theoretical evidence suggests that the North Pacific may be an exception to this, and that the large wild salmon populations in this region may delay or prevent resistance by acting as genetic refuges for treatment-susceptible sea lice that can re-infest farms (Kreitzman et al. 2017). Indeed, emamectin benzoate delousing treatment is still highly effective in the north Pacific (Figure D.10). *Lepeophtheirus salmonis* counts decreased by 62.4% in the first month after treatment (95% credible intervals: 56.5%, 67.0%) and by 56.8% (50.0%, 62.6%) compared to untreated counts in the second month. *Caligus clemensi* counts decreased by 69.8% (60.0%, 77.6%) and 63.4% (52.0%, 73.6%) in the first two months after treatment. These are the first estimates of treatment efficacy for *C. clemensi*. I also found no evidence of resistance evolution over the time range of my dataset; I attempted to include a logistic-shaped interaction between treatment and month (from January 2011 to December 2016), but the model would not converge with this term because the data showed no decline in effectiveness of treatment over time.

Salmon farms regularly require delousing treatment, and compliance with parasite control regulations is not perfect. In BC, when a farm's *L. salmonis* counts exceed an average of three motile lice per fish, it is required to perform a management action in the form of a delousing treatment or harvest (Fisheries and Oceans Canada 2016a),

although the latter option is rarely chosen. Mean monthly *L. salmonis* counts exceeded the three-louse threshold in 14.3% of all months, and in 8.5% of months during the wild juvenile salmon migration in spring. When *L. salmonis* counts exceeded the three-louse threshold, they were followed by prompt action (i.e., treating or harvesting within one month) 79.5% of the time between March and June – the months in which regulations require prompt action (Fisheries and Oceans Canada 2016a) – and 67.3% of the time between July and February. On average, farms waited 0.75 ± 0.22 months before action after a threshold-breaking *L. salmonis* count between March and June, while the mean delay was 1.32 ± 0.14 months between July and February. Some regions (e.g., zone 2-3: SW Vancouver Island) were faster at responding with management action than others (e.g., zone 3-3: Broughton Archipelago; Table D.3), suggesting that there is regional variation in how salmon farms interpret the regulations and their on-the-ground implementation.

My simulations indicated that current parasite control policy is effective at reducing farm lice during the wild juvenile migration in average years, but that it has limited capacity to remain effective when environmental conditions change. In 2015, the North Pacific experienced the most anomalous sea surface temperatures on record due to a warming event now known as the 'the blob' (Peterson et al. 2015; Bateman et al. 2016). Under these high-temperature conditions and the current parasite management strategy in BC, the mean simulated farm louse count during migration was 2.41 times greater than the mean simulated count during an average year's migration (hereafter termed 'baseline'; Figure 5.5). In comparison, the mean observed louse count during the 2015 migration was 3.12 times higher than the mean observed count in average years (Figure 5.5). To reduce farm louse counts during migration in a high temperature year to baseline levels under this status quo management strategy, my simulations indicated that the treatment threshold would have had to be cut in half, from 3 lice to 1.5.

I simulated two alternative parasite management strategies to assess their potential effectiveness at controlling parasite outbreaks in high-temperature years. The first strategy eliminated delays between diagnoses of sea louse outbreaks (i.e., *L. salmonis* counts greater than three lice per fish) and subsequent delousing treatments. This strategy reduced sea louse counts during migration in high-temperature years to 1.45 times greater than baseline (Figure 5.5) and only increased the number of annual treatments by 9.2% in average years. The second alternative parasite management

strategy was identical to the current strategy, but with an additional obligatory delousing treatment in February, just before the migration period. This strategy reduced migration sea louse counts in the high-temperature year to the same level as baseline (Figure 5.5), but it would be quite costly for the salmon farming industry; farms would have to treat 74.2% more often than they do now in average years and 10.9% more often in high-temperature years.

The resilience of coupled human and natural systems such as this one depends on their adaptability to environmental change (Folke 2006; Liu et al. 2007). Since environmental policy often dictates the extent of human impacts on these systems, effective long-term policy options must account not only for current conditions but for future ones as well (Nelson et al. 2007). Consequently, the resilience of *policy* to environmental change is beginning to be recognized as a key consideration for new policies (Berkes 2007; Adger et al. 2011), but the resilience of existing policies is largely unknown. As I show with the case of parasite control on salmon farms, it may be that current policies will need revision for continued effectiveness in the face of a changing global environment.

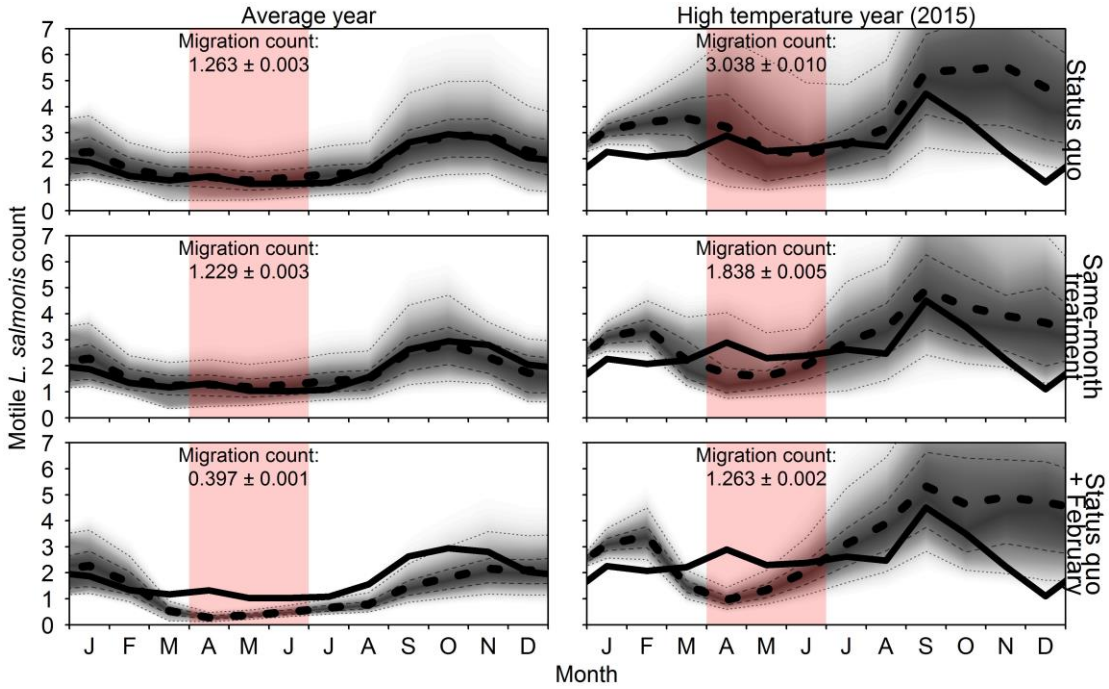

Figure 5.5 Simulated motile *L. salmonis* counts on farms in an average year (left panels) and in a high-temperature year (2015; right panels), for three different parasite management strategies.

The top panels show the current parasite management strategy employed by BC salmon farms, the middle panels depict a strategy in which delousing treatments always occur in the same month as a threshold-breaking count, and the bottom panels show the current strategy with an

additional obligatory treatment in February. The thick dashed solid lines indicate the monthly means of the simulated louse counts and the solid lines show the observed mean louse counts. The quantiles of the simulated counts are shaded in 0.5% increments and the thin dashed lines give the 50% and 90% quantiles. The wild juvenile salmon migration period is conveyed by the red shaded regions, and the mean *L. salmonis* counts (± SE) during the migration period are given. The top-left panel reflects current louse management practices in an average year. Note that the monthly data are discrete and only connected by lines for visualization purposes.

Conclusions

My results illustrate that the effectiveness of environmental policies can depend on many factors. For aquaculture policies, understanding the complexities of disease dynamics in multi-host systems may be crucial for minimizing disease outbreaks. Environmental policies regulating industry can be undermined by biased industry self-reported data when incentives for accurate self-reporting are not strong. Finally, my analysis shows that with a changing global environment, the effectiveness of policies at mitigating ecological threats may deteriorate if they are not resilient to environmental change. By demonstrating how the effectiveness of environmental policies can be weakened, I provide potential solutions for improving future effectiveness of these policies.

Chapter 6.

General discussion

If, how, and when aquaculture impacts wildlife through disease is a critical question that may determine the conservation of many aquatic species. My thesis examines the indirect pathways through which wild fish can be affected by parasites linked to aquaculture operations and the current effectiveness of management at controlling these parasites on farms. Specifically, I examine the potential effects of sea lice on factors critical to wild salmon survival and I assess several possible ways in which policy regulating sea louse control on salmon farms may be undermined.

The impacts of pathogens and parasites on host individuals and populations are not limited to direct mortality from disease. These infectious agents can also interact with fundamental ecological processes, such as competition and predation, to determine host survival (Hatcher et al. 2006). This may be especially true for fish, which often experience very high mortality rates due to competition and predation (Groot and Margolis 1991; Hixon and Jones 2005). However, empirical examples of sub-lethal effects of parasites on wild fish are few and far between. In Chapter 2, I found that heavy sea louse infestation was associated with compromised competitive foraging abilities in juvenile sockeye salmon. Using a field experiment, I observed that highly infested fish experienced a 20% decline in their ability to compete for food, on average. In Chapter 3, I found that this decrease in competitive foraging ability may lead to lower foraging success in the wild. By performing a field survey of out-migrating juvenile sockeye salmon, I discovered that highly infested fish had 17% less food in their stomachs, on average. In Chapter 4, I built on the results of Chapters 2 and 3 and established that sea louse infestation is associated with decreased growth for juvenile sockeye. I analyzed the microstructure of juvenile sockeye otoliths and found that when highly infested, median-sized fish experienced an average growth reduction of 7.9%. Cumulatively, these results indicate that sea lice may have sub-lethal effects on their hosts, which has major implications for wild salmon survival.

Together with previous research linking sea louse infestations of juvenile sockeye to salmon farms along their migration route (Price et al. 2011), my results in Chapters 2-4 suggest that parasite management on farms may be crucial for minimizing the effects of sea lice on wild salmon. In Chapter 5, I assessed parasite control policy for BC salmon farming to determine whether there is room to improve. In particular, I found that the self-reported sea lice counts performed by the salmon farming industry are biased; in months where DFO audited salmon farms, counts increased by a factor of 1.94 for *C. clemensi* and 1.17 for *L. salmonis*, on average. These counts are used to determine the timing of delousing treatments to control sea louse outbreaks, so this underestimation bias increases farm louse exposure for wild juvenile salmon. I also found that current parasite control policy is not resilient to environmental change, as counts in high-temperature years cannot be controlled during the juvenile salmon migration like they are in average years. Given these results, I assessed and suggested possible solutions to improve parasite control policy on salmon farms.

A second species of sea louse enters the ring

To me, the most surprising part of my thesis was the *C. clemensi* narrative that emerged. Until the work from Chapters 2-4 was published (Godwin et al. 2015; Godwin et al. 2017; Godwin et al. In press), few had paid this species of sea louse any attention. Several studies had documented high infestation rates of *C. clemensi* on juvenile salmon (e.g., Morton et al. 2008; Beamish et al. 2009) and *C. clemensi* had even been reported as the dominant louse species on out-migrating Fraser River sockeye and sometimes on farms as well (Price et al. 2011). Yet nobody had ever investigated the potential effects of *C. clemensi* on wild salmon, probably because this species is thought to be less pathogenic than *L. salmonis* (Johnson et al. 2004; Igboeli et al. 2014) and because the sub-lethal effects of sea lice are rarely examined. But even if *C. clemensi* had been considered a problem by some, I don't think anybody (including myself) would have expected that 99% of out-migrating sockeye would be infested with this sea louse in 2013 and 2014.

Since I published the first results from this thesis, there has been growing concern about BC's less-studied sea louse. In a report that made several aquaculture-related recommendations to improve the outlook for Pacific salmon, a panel of expert scientists specifically noted that *C. clemensi* may influence the foraging success and growth of

juvenile sockeye salmon (Moore et al. 2017). The advisory council to BC's Minister of Agriculture mentioned my work showing that "*Caligus* sea lice can impair feeding and growth of wild salmon" and remarked that *C. clemensi* is not targeted by delousing treatments on BC farms (Minister of Agriculture's Advisory Council on Finfish Aquaculture 2018). SeaChoice, Canada's sustainable seafood watchdog program, recently questioned the lack of *C. clemensi* control on farms and explicitly cited my publications in its rationale for rejecting BC farmed salmon as a responsible seafood choice for consumers (SeaChoice 2017). In short, *C. clemensi* and its potential sub-lethal effects are now on the map and questions are being raised about how salmon farms manage this species.

The unexpected *C. clemensi* results continued in my fifth chapter. I found that farm infestation rates of *C. clemensi* are probably at least 2.5 times higher than indicated by industry counts, due to significant underestimation bias in the counts and background underestimation from the counting protocol itself. Consequently, even if regulations were changed so that *C. clemensi* were targeted by parasite management on farms, the way that counts are performed would have to be improved drastically for the counts to be trusted. If those improvements were made, however, my results indicate that *C. clemensi* management could well be effective, since this louse species is just as affected by delousing treatments as *L. salmonis*.

It will be interesting to see the reaction of the salmon farming industry and government regulators when the results from Chapter 5 are published. The industry will likely experience pressure to eliminate the underestimation bias in their sea lice counts, at least from the public and NGOs. Given the federal government's dual mandates to promote the aquaculture industry while protecting wild salmon (Cohen 2012a), it will be illuminating to see if regulations get adjusted or if DFO changes the way they are enforced. It is also possible that momentum will build for targeted *C. clemensi* management, but that will not be a popular idea within the industry. *Lepeophtheirus salmonis* affect farm profits by increasing mortality and decreasing growth (Costello 2009b), so treatments are generally supported by industry even though they are costly. On the other hand, there is no evidence that *C. clemensi* harm farmed fish, so there will be minimal incentive for farms to increase the frequency of treatments to control this louse species. There will also be concerns that more treatments will lead to the development of resistance in sea lice to the delousing chemicals. This is a major issue in

other salmon farming regions (Aaen et al. 2015), and although Pacific Canada may be less susceptible to resistance development (Kreitzman et al. 2017) it is still a legitimate concern. If farms were to start treating for *C. clemensi* outbreaks, they would also have to contend with more external louse inputs than they do for *L. salmonis*, since my results suggest that both wild salmon *and* herring can transfer *C. clemensi* to farms. Ultimately, any modifications to the way that salmon farms manage parasites will probably be made through changes to policy regulating the industry.

The upcoming policy window for Canadian aquaculture

Over the ten years that I have been involved in salmon research, the issue of salmon farming has cycled in and out of public, media, and government attention. Since August 2017, however, concern about salmon aquaculture in Pacific Canada has risen to levels not seen in the last decade. The escalation began in the Broughton Archipelago, BC, where members of four BC First Nations began occupying two salmon farms (CTV News 2017). This protest stemmed from the long-standing conviction held by most coastal BC First Nations that salmon farms should not be allowed to operate in Indigenous territories without being granted explicit permission from the local Nations. Although the occupations were eventually ended by court order (The Tyee 2017), by that time they had already renewed the salmon farming controversy in Pacific Canada.

Since the occupations, several events have occurred that indicate we may be approaching a tipping point for salmon farming in BC. Cumulatively these events suggest there is a window opening for science to influence policy governing Canadian salmon farms. Potentially the biggest game changer was the April 2018 audit of Canada's salmon farming industry from the Canadian Auditor General's office (Office of the Auditor General of Canada 2018). The audit, led by environment commissioner Julie Gelfand, was critical of Fisheries and Oceans Canada for its management of Canadian aquaculture. Gelfand said the investigation revealed "the most number of gaps in any audit [she's] ever done" and that the "the department is at risk of being seen to promote aquaculture over the protection of wild salmon" (National Post 2018). The audit recommended that DFO "should more effectively enforce aquaculture regulations and pursue additional enforcement measures", to which DFO responded that it would assess the costs of full aquaculture enforcement by November 2019 (Office of the Auditor General of Canada 2018). This costing process could be informed by my fifth chapter,

which indicates that inadequate enforcement is undermining parasite management in BC and, more importantly, suggests possible solutions for improvement.

Still at the federal level, the Office to the Chief Science Advisor in Canada recently established an independent expert panel to assess the provision of aquaculture science advice in Canada (Government of Canada 2018). The expert panel will make formal recommendations for improvement to the Minister of Science and the Minister of Fisheries, Oceans and the Canadian Coastguard. These recommendations will explicitly cover how scientific evidence is considered "in risk-based decision-making and policy development processes that form the basis for the management of aquaculture". Additionally, the Government of Canada is currently developing a National Aquaculture Act (J. Hutchings, pers. comm.), although details for this are scarce. This thesis fills several knowledge gaps in our understanding of the management and potential impacts of Canadian aquaculture, so it may well be used to improve the evidentiary bases of these two processes.

As of 2010, the federal government regulates the Canadian aquaculture industry and provides licenses for companies to operate, while the BC provincial government controls tenures for siting operations (Fisheries and Oceans Canada 2018). Since the BC New Democratic Party (NDP) came into power in 2017, there has been much uncertainty over the renewal of these tenures, many of which were set to expire in 2018. The NDP campaigned on promises to "ensure that the salmon farming industry does not endanger wild salmon" (BC NDP 2017). In June 2018 the new provincial government made their first major decision about salmon farming, pushing the decisions on all tenure renewals to 2022 but also changing the rules regarding those renewals (Government of British Columbia 2018). As of 2022, tenures will only be renewed if DFO asserts that operations will not affect wild salmon stocks *and* if the companies have negotiated agreements with the local First Nations to operate in their territories (Government of British Columbia 2018). Both of these new rules provide opportunities for scientific evidence to inform the outcomes of tenure decisions. DFO will theoretically have to support its case with arguments grounded in science and First Nations will likely rely on research to inform their decisions on whether they want farms operating in their territories. With the upcoming opportunities to inform decision-making at both the provincial and federal levels, I am hopeful that the results from my thesis could help guide the forthcoming changes to aquaculture management and policy over the next few years.

Conclusion

This thesis fills key knowledge gaps on the stressors faced by what is often considered the most important set of salmon populations in BC (Northcote and Larkin 1989). Prompted by the long-term decline in Fraser River sockeye productivity, the $37 million Cohen Commission identified the need for research into the effects of pathogens and parasites faced by Fraser River sockeye during their juvenile out-migration. My second chapter provided the first evidence that parasite exposure may cause fitness consequences for these fish, and my third and fourth chapters further supported this assertion.

This thesis highlights the necessity for field studies that incorporate ecological context. Laboratory studies can identify cause-and-effect relationships between parasites and their hosts, but the actual effects of parasites in the wild are probably much different than in a laboratory setting. For example, if sea lice reduce juvenile sockeye growth by decreasing their competitive foraging ability, a standard laboratory study without food competition would not find an effect of sea lice on growth. The effects of parasites on their hosts therefore depend on ecological processes absent in the laboratory, and field studies are the only way to incorporate this complexity. With this ecological context, however, comes uncertainty; despite considerable effort to reduce potential confounding factors, all of the studies in this thesis are ultimately correlational. The obvious next step to confirm these effects would be a complex challenge study in a laboratory setting that incorporates all the variables found to be important in this thesis (e.g., food competition and body size variation). It is unclear whether such a study would be feasible, both in terms of its complexity and its facility requirement.

When considered together (Figure 6.1), the interconnected relationships I found in Chapters 2-4 suggest that sea louse infection may not just be accompanied by a single, temporary effect for the duration of infestation. In each chapter, I found that the response variable of interest (competitive foraging ability, foraging success, or growth) was not only associated with sea louse infestation, but also with body size. Infestation may therefore have long-lasting effects by initiating and/or intensifying divergent growth among individuals through differences in competitive ability and foraging success. These differences may then create and reinforce intraspecific heterogeneity in body sizes and ultimately differential survival through ecological processes such as predation (Sogard

1997). The potential for lasting effects of sea lice on factors critical to salmon survival highlights the need to consider the anthropogenic influence on the parasite burdens of these fish, and to reduce it through management actions when possible.

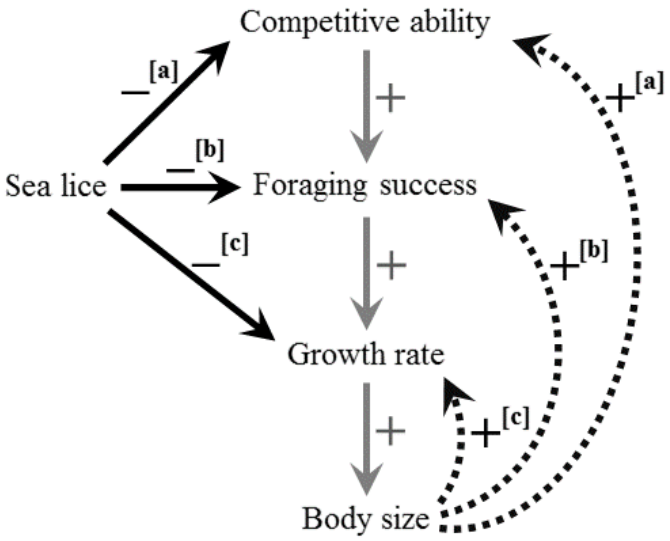

Figure 6.1 Relationships among juvenile sockeye salmon traits and sea louse infection.

Solid double-ended arrows indicate established correlative sea louse relationships ([a] Chapter 2, [b] Chapter 3, [c] Chapter 4). Dashed black arrows indicate established correlative body size relationships, and grey arrows indicate implicit mechanistic relationships.

This thesis reveals that that there is room for improvement when it comes to parasite management on salmon farms. I identify key areas where improvements could be made, namely the accuracy of sea lice counts on farms and the resilience of parasite control policy to increasing sea surface temperatures. Rather than simply pointing a finger at the problem, I discuss possible alternatives to current policy and their potential for increased effectiveness. These findings are directly applicable to broader environmental policies that rely upon self-reported data from industry or whose effectiveness may be vulnerable to changing environmental conditions.

Understanding and limiting the effects of diseases from aquaculture on aquatic wildlife will likely determine the sustainability and success of the blue revolution (Olesen et al. 2011). This thesis provides key insights into the nuanced ways in which disease may impact marine wildlife and helps chart a path forward for improving aquaculture policy in Canada and beyond.

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Appendix A.

Supporting information for Chapter 2

Description of methods for viral analysis

Virus screening was performed at the Atlantic Veterinary College. Salmonid alphavirus (SAV) was tested using TaqMan primers and probe sequences for the Q_nsP1 real-time PCR assay, a broad-spectrum assay for all salmonid alphavirus subtypes (Hodneland and Endresen, 2006). Real-time RT-PCR was also performed to detect infectious salmon anemia virus (ISAV) and piscine orthoreovirus (PRV) using TaqMan probes for ISAV segment 8 (Snow et al., 2006) and PRV segment L1 (Haugland et al., 2011). Samples were considered positive at Ct values less than or equal to 37.5 for SAV, 35.25 for ISAV, and 39.9 for PRV. Verification of ISAV results was accomplished with conventional RT-PCR using segment 8 (Devold et al., 2000) and segment 6 HPR primers (Kibenge et al., 2009).

Supplementary figures and tables

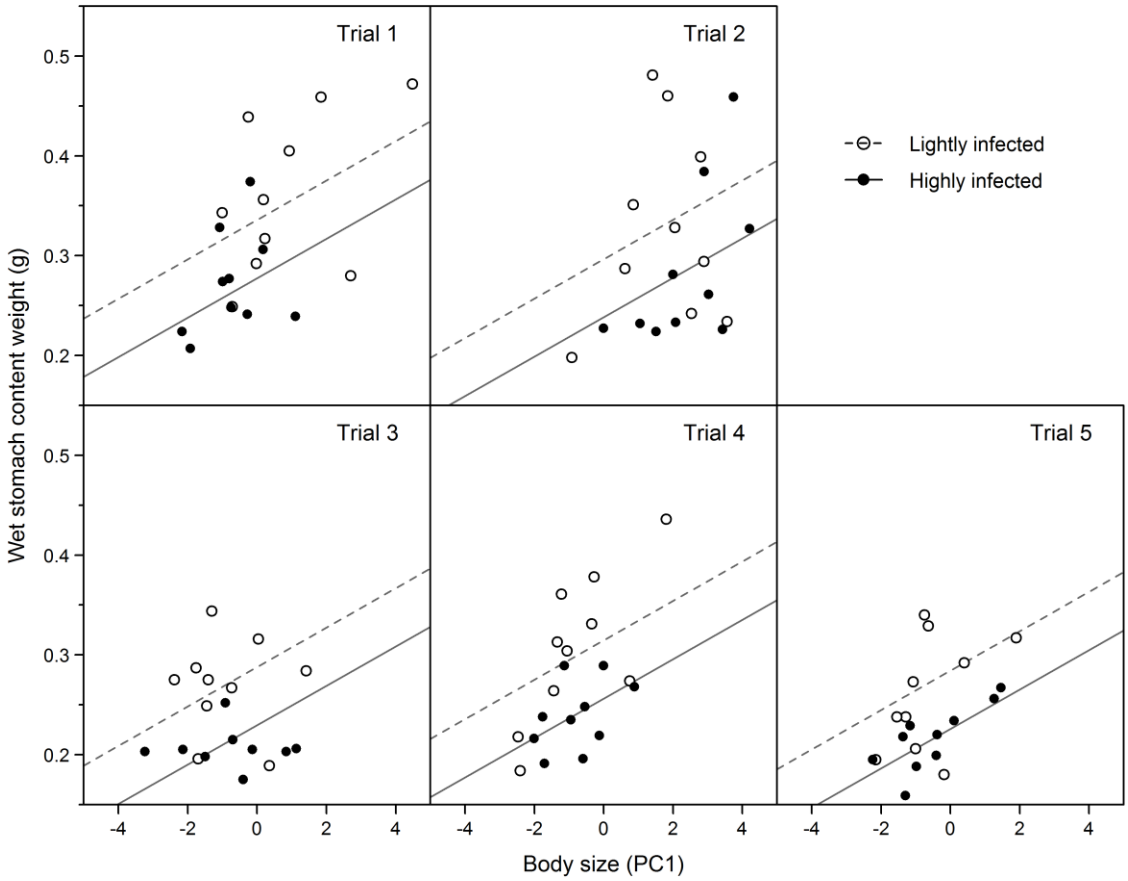

Figure A.1 Differences among trials in experimental foraging success.
Each point represents an individual fish. Regression lines use the parameter coefficients of the top model, which includes terms for infection category and body size (Table 2.2).

Table A.1 Temperature and salinity data from inside the net pens immediately following the experiment.

	Temperature (°C)		Salinity (ppt)	
Trial	Surface	1 m depth	Surface	1 m depth
1	11.5	10.8	29.4	26.7
2	12.6	12.5	23.8	24.2
3	14.1	13.1	21.6	24.8
4	12.6	12.6	23.4	23.6
5	11.6	11.5	25.8	26.3

Supplementary references

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Appendix B.

Supporting information for Chapter 3

Juvenile sockeye salmon digestion experiment

Methods

To assess the extent to which my prey density estimates were representative of the prey field met by the juvenile sockeye salmon (*O. nerka*) when they had been feeding, I needed to determine how quickly juvenile sockeye digested their prey. To accomplish this, I performed a small feeding experiment at a floating field facility comprised of several floating docks and net pens. I collected fish at the same Johnstone Strait location used in the main study and transported them by boat for 1 h to the experimental facility (see Figure 3.1 for map). During transport, the sockeye were again held in insulated fish totes with bubblers and ice packs. I did not collect temperature or salinity data for this experiment, but previous studies using the same experimental facility and juvenile salmon collection sites have indicated that their water temperatures and salinities are very similar (Atkinson et al. In review; Godwin et al. 2015).

Upon arrival at the facility, I transferred the fish to a large (2.8 m deep and 6.1 x 6.1 m across) net pen and weaned them onto frozen brine shrimp (Brine Shrimp Direct, Ogden, Utah, USA) over the next five days. Brine shrimp were thawed in freshwater and were fed to the fish by adding them to the center of the net pen. Medium-sized fish (between 107 and 120 mm) were removed for another study, leaving 31 smaller (104.1 mm ± 0.4 mm) and 37 larger (124.9 ± 0.7 mm) fish. The fork length range of these experimental fish was 97 mm to 132 mm, which was fully within the fork length range of the fish from the main study (88 mm to 133 mm).

One hour after sunrise on their sixth day at the experiment facility, the 68 fish were fed to satiation and ten were immediately sacrificed with a lethal dose of MS-222. At half, one, two, three, five hours after initial feeding, ten fish were again randomly removed and euthanized, leaving remaining fish. At eight hours, these eight fish were removed and euthanized. I weighed the wet stomach contents of the fish in the same manner as for

those sacrificed in the field, and calculated relative stomach fullness by dividing the weight of each fish by the weight of its wet stomach contents.

I fit a Michaelis-Menten curve to the relative stomach fullness data and used these parameter estimates to calculate the amount of digestion that occurred between time points in the experiment.

Results

The digestion rate of the experimental fish began high and gradually slowed over time (Figure B.1). Six fish did not consume any brine shrimp and were therefore removed from the dataset. After one hour, 37% of the initial stomach contents remained, and after the final hour of the experiment (hour eight) only 18% remained. The predicted relative stomach fullness of the experimental fish at hour eight (0.6%) was similar to the mean relative stomach fullness of the main study's fish (0.6%), suggesting that most of the main study's fish must have eaten in the previous eight hours since not all of them would have fed to satiation like the experimental fish.

Supplementary figures and tables

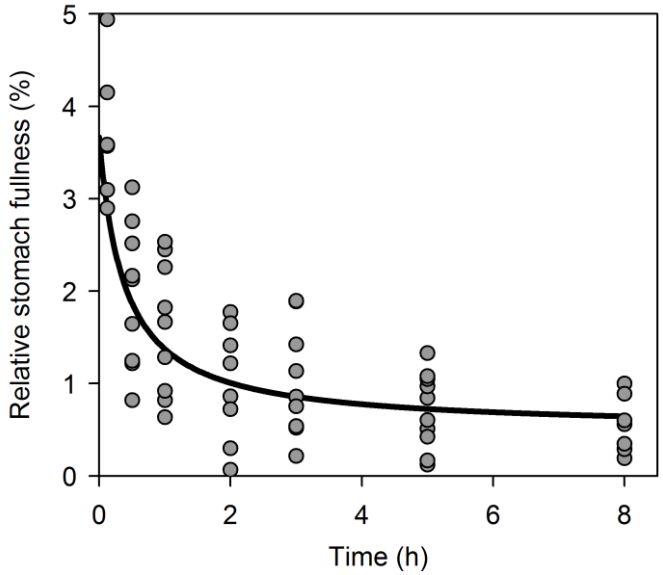

Figure B.1 Relative stomach fullness of juvenile sockeye salmon over the course of the digestion rate experiment.
The black line shows the Michaelis-Menten curve fit to the data. Six fish were removed from this dataset because they did not consume any food.

Table B.1 Detailed data for the juvenile salmon (*Oncorhynchus* spp.) collections.

Collection date	Latitude	Longitude	Temp. 0m (°C)	Temp. 1m (°C)	Salinity 0m (ppt)	Catch size
2014-05-26	50 32.474	-126 37.489	9	9	29	70
2014-05-27	50 32.741	-126 39.895	9	9	34	250
2014-05-29	50 32.591	-126 37.858	10	9	32	400
2014-05-30	50 32.787	-126 40.182	9	9	30	1000
2014-05-31	50 32.724	-126 39.689	9	9	30	400
2014-06-01	50 32.811	-126 40.069	9	9	30	350
2014-06-02	50 32.635	-126 39.299	9	9	30	800
2014-06-06	50 32.770	-126 39.923	9	9	29	150
2014-06-06	50 32.992	-126 40.773	10	9.5	28	1000
2014-06-07	50 32.958	-126 40.829	9.5	9.5	28	1500
2014-06-07	50 32.791	-126 39.986	9.5	9.5	29	1500

Catch size indicates the estimated number of all juvenile salmonids caught in the net, which included sockeye (*O. nerka*) as well as chum (*O. keta*), pink (*O. gorbuscha*), coho (*O. kisutch*), and chinook (*O. tshawytscha*). "Temp." indicates water temperature at the specified depth.

Table B.2 Detailed sea-louse infection data for juvenile sockeye (*O. nerka*) used in the stomach fullness analysis.

Collection	Infection category	Caligus copepodite	Lep copepodite	Small chalimus	Large chalimus	Motile Caligus	Motile Lep
1	highly	1	0	5	4	0	0
1	highly	0	0	4	2	1	0
1	highly	1	0	1	3	1	0
1	highly	1	0	3	2	2	0
1	highly	1	0	1	4	0	0
1	lightly	0	0	0	0	0	0
1	lightly	1	1	0	0	0	0
1	lightly	2	0	1	0	0	0
1	lightly	1	0	0	0	0	0
1	lightly	1	0	2	0	0	0
2	highly	0	0	2	2	1	0
2	highly	1	0	2	1	1	0
2	highly	0	0	0	0	1	1
2	highly	4	0	5	4	0	0
2	highly	1	0	3	1	1	0
2	highly	3	0	9	5	0	0
2	lightly	0	0	1	0	0	0
2	lightly	0	0	0	0	0	0
2	lightly	1	0	0	0	0	0
2	lightly	0	0	0	0	0	0

Collection	Infection category	Caligus copepodite	Lep copepodite	Small chalimus	Large chalimus	Motile Caligus	Motile Lep
2	lightly	1	0	0	0	0	0
2	lightly	0	0	1	0	0	0
3	highly	0	0	2	2	1	0
3	highly	0	1	3	2	1	0
3	highly	0	1	7	2	1	0
3	highly	2	0	7	2	1	0
3	highly	2	0	4	5	0	0
3	highly	1	0	7	7	0	0
3	highly	3	0	7	4	1	0
3	lightly	0	0	0	0	0	0
3	lightly	0	0	1	0	0	0
3	lightly	1	0	0	0	0	0
3	lightly	0	0	1	0	0	0
3	lightly	1	0	0	0	0	0
3	lightly	0	0	1	0	0	0
3	lightly	0	0	1	0	0	0
4	highly	0	0	2	2	2	0
4	highly	1	1	6	4	0	0
4	highly	0	0	4	2	2	0
4	highly	0	0	9	2	2	0
4	highly	0	0	1	1	2	0
4	highly	0	0	5	3	1	0
4	highly	0	0	2	5	2	0
4	lightly	2	0	0	0	0	0
4	lightly	0	0	1	0	0	0
4	lightly	1	0	0	0	0	0
4	lightly	1	0	1	0	0	0
4	lightly	0	0	0	0	0	0
4	lightly	0	0	1	1	0	0
4	lightly	0	0	1	0	0	0
5	highly	1	1	5	6	1	0
5	highly	2	0	4	3	1	0
5	highly	0	0	3	2	1	0
5	highly	0	0	1	2	1	0
5	highly	1	1	3	4	0	0
5	highly	2	0	4	4	0	0
5	lightly	1	0	2	0	0	0
5	lightly	0	0	2	0	0	0
5	lightly	0	0	0	0	0	0
5	lightly	0	0	0	0	0	0
5	lightly	1	0	1	0	0	0

Collection	Infection category	Caligus copepodite	Lep copepodite	Small chalimus	Large chalimus	Motile Caligus	Motile Lep
5	lightly	0	1	0	0	0	0
6	highly	2	0	12	1	2	0
6	highly	3	1	3	4	1	0
6	highly	0	0	5	5	0	0
6	highly	0	0	3	2	2	0
6	highly	1	0	6	1	3	0
6	highly	1	1	6	3	1	0
6	highly	0	0	4	4	0	0
6	lightly	0	1	2	0	0	0
6	lightly	0	0	0	0	0	0
6	lightly	0	0	1	0	0	0
6	lightly	0	0	3	0	0	0
6	lightly	0	0	3	0	0	0
6	lightly	2	0	1	0	0	0
6	lightly	0	0	2	0	0	0
7	highly	1	0	2	2	1	0
7	highly	2	1	6	4	0	0
7	highly	1	0	5	6	0	0
7	highly	1	0	1	6	1	0
7	highly	0	0	4	7	0	0
7	highly	0	0	2	2	2	0
7	lightly	0	0	1	0	0	0
7	lightly	0	0	1	0	0	0
7	lightly	0	0	0	0	0	0
7	lightly	1	0	1	0	0	0
7	lightly	1	0	2	0	0	0
7	lightly	1	0	0	0	0	0
8	highly	0	0	2	6	0	0
8	highly	0	0	2	5	0	0
8	highly	1	0	4	10	1	0
8	highly	0	0	5	6	4	0
8	highly	0	0	2	6	1	0
8	lightly	0	0	1	0	0	0
8	lightly	3	0	0	0	0	0
8	lightly	2	0	0	0	0	0
8	lightly	0	0	0	0	0	0
8	lightly	1	0	1	0	0	0
9	highly	1	0	3	2	2	0
9	highly	0	0	0	1	3	0
9	highly	1	0	2	0	3	0
9	highly	0	0	5	3	1	0

Collection	Infection category	Caligus copepodite	Lep copepodite	Small chalimus	Large chalimus	Motile Caligus	Motile Lep
9	highly	0	0	3	6	0	0
9	lightly	1	0	1	0	0	0
9	lightly	1	0	2	0	0	0
9	lightly	0	0	1	0	0	0
9	lightly	0	0	1	0	0	0
9	lightly	0	0	3	0	0	0
10	highly	3	0	5	3	1	0
10	highly	1	0	1	5	2	0
10	highly	1	0	3	6	1	0
10	highly	2	0	6	8	1	0
10	highly	2	0	4	9	1	0
10	highly	2	0	4	5	2	0
10	lightly	0	0	3	0	0	0
10	lightly	1	0	0	0	0	0
10	lightly	3	0	0	0	0	0
10	lightly	1	0	2	0	0	0
10	lightly	1	0	1	0	0	0
10	lightly	1	0	0	0	0	0
11	highly	0	2	11	5	2	0
11	highly	1	0	5	4	1	0
11	highly	2	0	3	1	4	0
11	highly	1	0	13	3	1	0
11	highly	1	0	3	8	1	0
11	lightly	0	0	3	0	0	0
11	lightly	0	0	0	0	0	0
11	lightly	1	1	0	0	0	0
11	lightly	1	1	1	0	0	0
11	lightly	0	0	1	0	0	0

Fish were categorized as highly infected or lightly infected based on the number and developmental stage of sea lice parasitizing them (Table 4.1). Two species of sea lice were observed: *Caligus clemensi* (Caligus) and *Lepeophtheirus salmonis* (Lep). Large chalimus lice were in their second *L. salmonis* chalimus stage or their third or fourth *C. clemensi* chalimus stage. Motile sea lice were pre-adults or adults.

Table B.3 Model selection results for the full model set.

Rank	Model	ΔAICc[a]	w_i[b]	R^2[b]
1	*infection*	0	0.358	0.549
2	*infection + size*	1.24	0.192	0.530
3	*infection + prey*	1.66	0.156	0.542
4	*infection + size + prey*	2.66	0.095	0.510
5	*infection * size*	3.85	0.052	0.530
6	*infection * prey*	3.91	0.051	0.536
7	*infection * prey + size*	4.42	0.039	0.500
8	*infection * size + prey*	5.31	0.025	0.510
9	*infection * size + infection * prey*	6.83	0.012	0.494
10	*size*	7.80	0.007	0.469
11	*intercept only*	8.18	0.006	0.469
12	*size + prey*	9.05	0.004	0.445
13	*prey*	9.80	0.003	0.461

The models included additive and multiplicative combinations of infection category (*infection*), body size (*size*; see description of principal component analysis), and prey density (*prey*) fixed effects. Each model included a random effect on the intercept for collection number.

[a] Difference from the top model AICc

[b] Akaike model weight

[c] R^2 for mixed-effects models calculated using the method developed by Nakagawa and Schielzeth (2013)

Supplementary references

Atkinson, E., Bateman, A. W., Dill, L. M., Krkošek, M., Reynolds, J. D., and Godwin, S. C. In press. Oust the louse: leaping behaviour removes sea lice from wild juvenile sockeye salmon. Journal of Fish Biology.

Godwin, S. C., Dill, L. M., Reynolds, J. D., and Krkošek, M. 2015. Sea lice, sockeye salmon, and foraging competition: lousy fish are lousy competitors. Canadian Journal of Fisheries and Aquatic Sciences 72(7): 1113-1120.

Nakagawa, S. and Schielzeth, H. 2013. A general and simple method for obtaining R^2 from generalized linear mixed-effects models. Methods in Ecology and Evolution 4(2): 133-142.

Appendix C.

Supporting information for Chapter 4

Recursive growth model

To investigate how sea louse-mediated growth effects may influence body size over a longer period of time, I constructed a recursive growth model. Using the model-averaged predictions for 10-day otolith growth, I calculated daily body growth, in terms of fork length, using the equation:

$$L_{t+1} = L_t + \frac{G_{t(L_t)}R}{10} \quad \text{(C.1)}$$

where L_t is fork length at the beginning of day t, $G_{t(Lt)}$ is the model-averaged prediction for 10-day otolith growth during day t at length L_t , and R is the rate of fork length growth per µm of otolith growth (i.e., the slope of Figure 4.3). For illustrative purposes I used initial fork lengths that corresponded to the 10th, 50th, and 90th percentile lengths from my dataset of 2,401 juvenile sockeye salmon, and I fit the model over 60 (Figure C.1) and 120 (Figure C.2) days for both infection statuses.

Supplementary figures

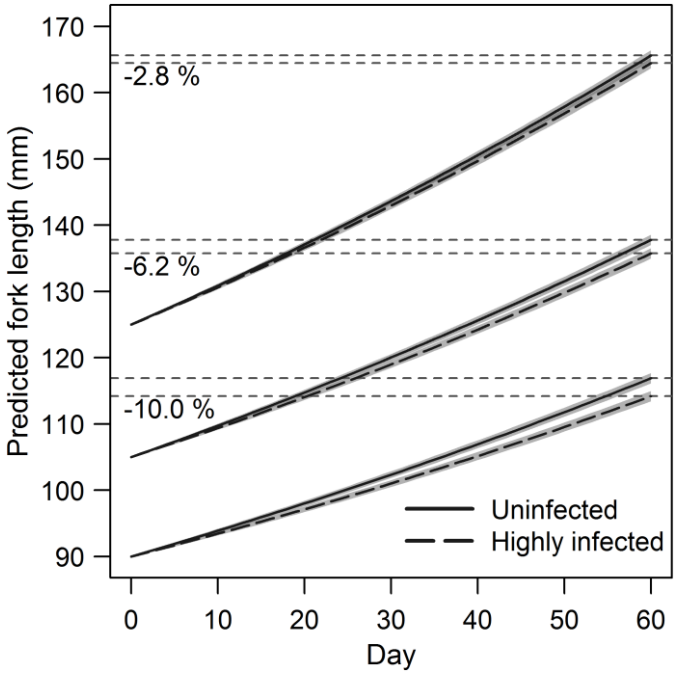

Figure C.1 Recursive growth models fit over 60 days using model-averaged predictions from the model set and initial lengths corresponding to the 10th, 50th, and 90th percentile lengths of captured sockeye salmon.

Percentages represent the growth reductions for highly infected juvenile sockeye relative to uninfected individuals after 60 days. 95% confidence intervals were calculated using the unconditional standard errors of averaged predictions and the standard error of *R*, the rate of fork length growth per µm of otolith growth.

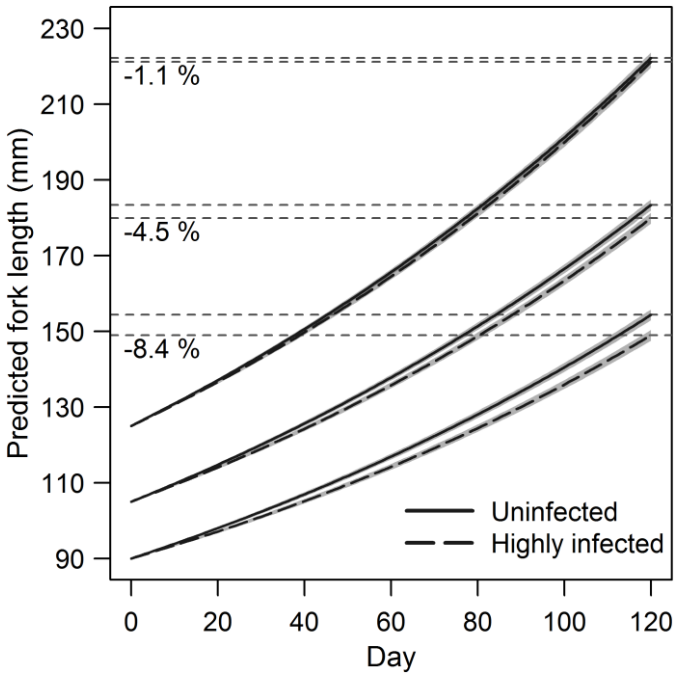

Figure C.2 Recursive growth models fit over 60 days using model-averaged predictions from the model set and initial lengths corresponding to the 10th, 50th, and 90th percentile lengths of captured sockeye salmon.

Percentages represent the growth reductions for highly infected juvenile sockeye relative to uninfected individuals after 60 days. 95% confidence intervals were calculated using the unconditional standard errors of averaged predictions and the standard error of *R*, the rate of fork length growth per µm of otolith growth.

Appendix D.

Supporting information for Chapter 5

Supplementary results

Model fit

My model predictions matched observed louse counts well for both *L. salmonis* and *C. clemensi*, except for a tendency to under-predict higher counts due to a scarcity of data points (Figure D.1-2). I present convergence diagnostics and posterior plots (Figure D.3-5), as well as the full set of parameter estimates (Table D.2 and Figure D.6). When parameter estimates are given in the text, I report them as modal estimates along with the lower and upper 95% credible intervals (LCI and UCI).

Spatial trends

The annual peaks and troughs in louse counts were relatively consistent among regions, with two exceptions. The Sunshine Coast (zone 3-1) had particularly low *L. salmonis* and *C. clemensi* counts throughout the time series. In contrast, the Central Coast (zone 3-5) experienced notably high *L. salmonis* counts compared with the other zones, likely due to a natural inclination towards high population growth rates in the region (Figure D.8) and delays between diagnoses of louse outbreaks and subsequent delousing treatments (see methods section in Chapter 5).

Effects of density dependence and age class

Neither interspecific nor intraspecific density dependence were important predictors of *C. clemensi* population growth rates on either of the two age classes of farmed salmon. For *L. salmonis*, intraspecific density dependence had a negative effect on louse population growth rate when farmed salmon had spent less than one year in seawater, and a positive effect on growth rate when farmed salmon had spent more than one year in seawater (Figure D.6). Interspecific density dependence did not influence *L. salmonis* population growth rate.

Farmed salmon age class had a positive effect on *L. salmonis* colonization rates and a negative effect on *C. clemensi* colonization rates (Figure D.6). The population growth rates of *L. salmonis* and *C. clemensi* were not influenced by farmed salmon age class.

Effects of spatial and temporal variables

Population growth rates for *C. clemensi* were consistently lower than those of *L. salmonis* across months, years, and health zones (Figure D.8). The mean predicted population growth rates for *L. salmonis* varied between 0.92 and 1.36 for most of the year, with an obvious one-month spike to 1.93 (95% credible intervals: 1.17, 2.92) in September. *Caligus clemensi* started the year with their highest predicted growth rate (1.02 (0.47,1.93) in January) and had a crash in October to 0.20 (0.07, 0.41). Predicted population growth rates were highest in 2015 for *L. salmonis*; at 1.57 (0.97, 2.38). *Caligus clemensi* population growth rates were fairly consistent among years (Figure D.8). *Lepeophtheirus salmonis* experienced faster population growth on the Central Coast (zone 3-5); compared to other health zones, with the mean predicted population growth rate in that region 1.93 (1.11, 3.02) being 1.70 times higher than the mean rate of the other six zones. The exceptionally low *C. clemensi* population growth rate in health zone 3-1 was driven by two strong month-to-month declines in mean louse counts and a small sample size in that zone (Figure D.8).

The high uncertainty of the predicted colonization rates prevented me from inferring differences among the effects of year and heath zone on these rates, both within and between species (Figure D.9).

Supplementary figures and tables

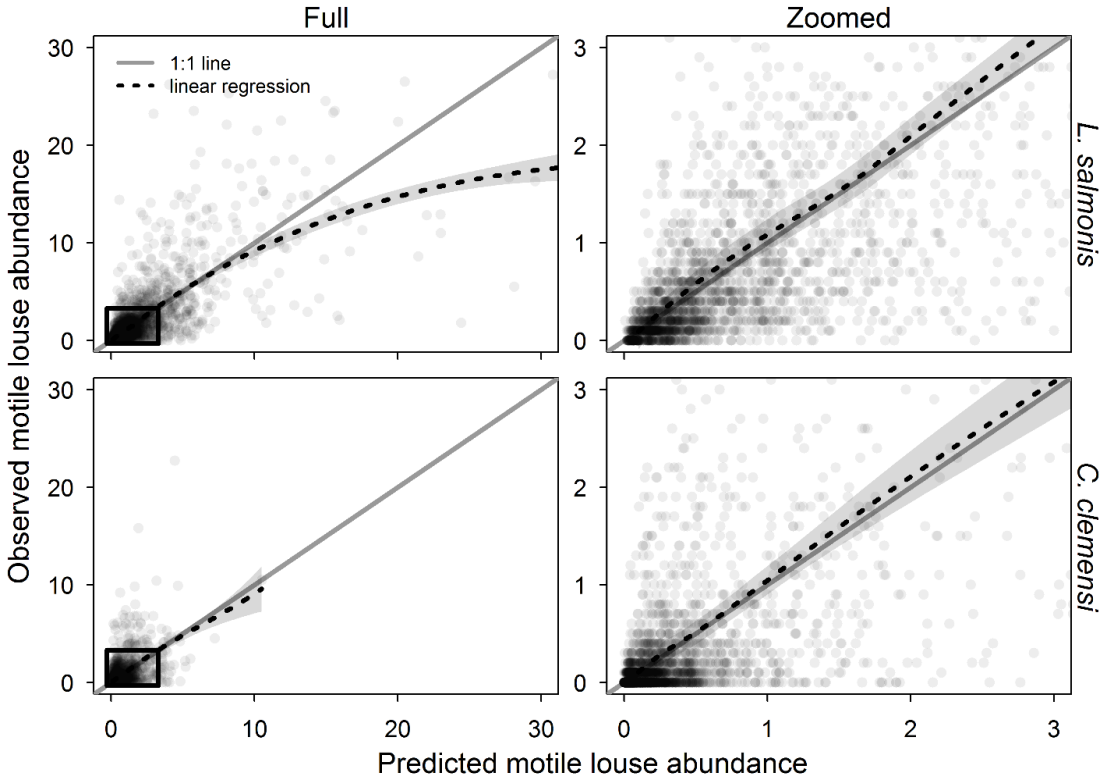

Figure D.1 Observed and predicted motile louse abundances for *L. salmonis* and *C. clemensi*.

The dashed black line is a LOESS regression fit between the observed and predicted values, while the solid grey line is the 1:1 line. The right panels are subsets of the left panels, and their extents are indicated in the left panels by the black rectangles. One *C. clemensi* data point is not visualized because it is beyond the bounds of the axes (observed motile louse count = 47.3). The model predictions match the observed louse counts well, except for a tendency to under-predict *L. salmonis* at high values due to a scarcity of data points (only 3.5% of observed counts had louse counts greater than 10 lice per fish).

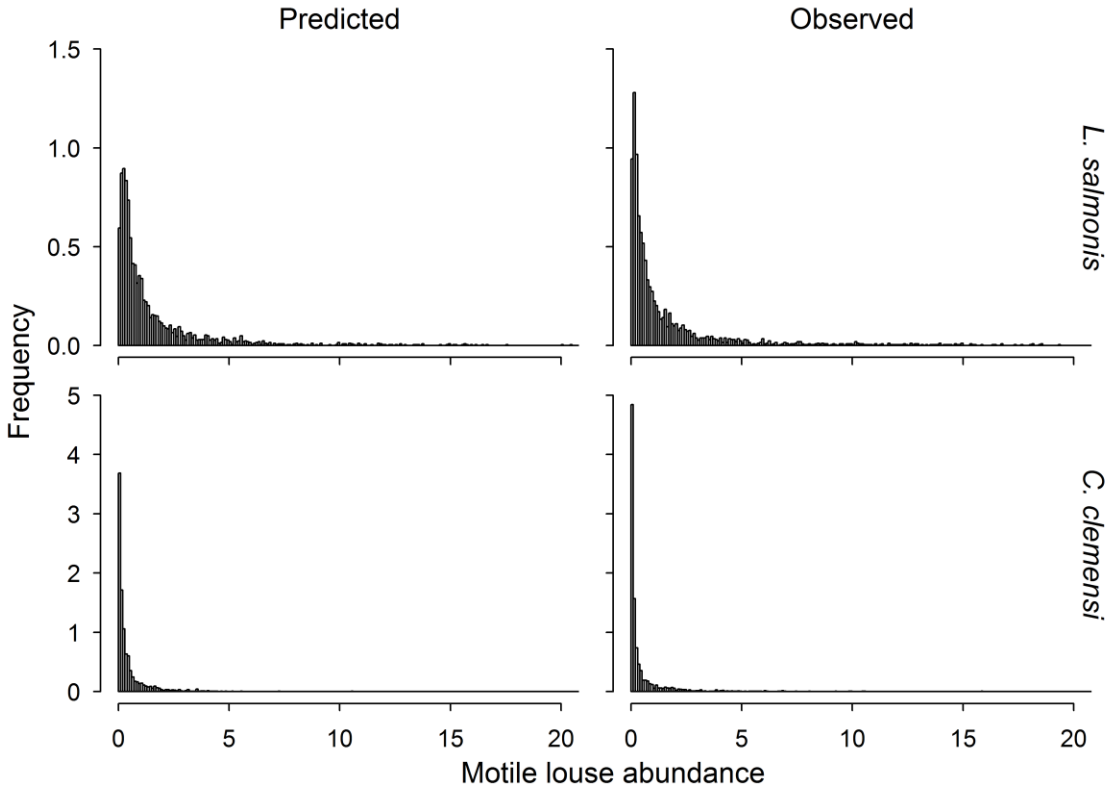

Figure D.2 Frequency histograms for predicted and observed motile louse counts.

Two *C. clemensi* and fourteen *L. salmonis* counts are not visualized here because they are beyond the bounds of the x axis. Note the different y axis limits between *L. salmonis* and *C. clemensi*.

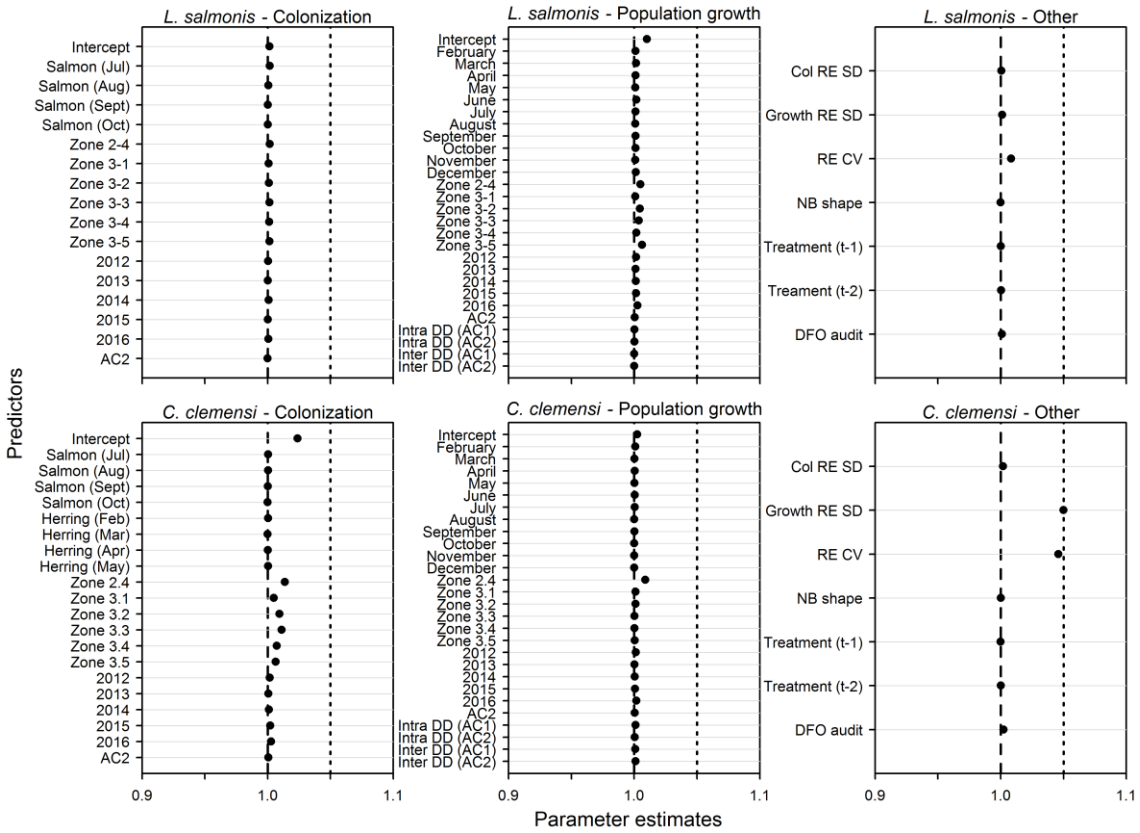

Figure D.3 Gelman-Rubin convergence diagnostics for my model parameters[1].
The two rows of panels differentiate the parameters for *L. salmonis* (top) and *C. clemensi* (bottom), and the three columns of panels distinguish the parameters from the colonization linear function of the model (left), the population growth function (center), and elsewhere (right). The finely-dashed vertical line at 1.05 describe the value under which parameters are considered to have converged, and the coarsely-dashed vertical line at 1.00 indicates the minimum possible value.

[1] 'AC1' = farmed salmon age class 1 (< 1 year in seawater), 'AC2' = farmed salmon age class 2 (>=1 year in seawater), 'Intra DD' = intraspecific density dependence, 'Inter DD' = interspecific density dependence, 'RE' = random effect, 'Col' = colonization, 'Growth' = population growth, 'SD' = standard deviation, 'CV' = covariation, 'NB shape' = negative binomial shape parameter.

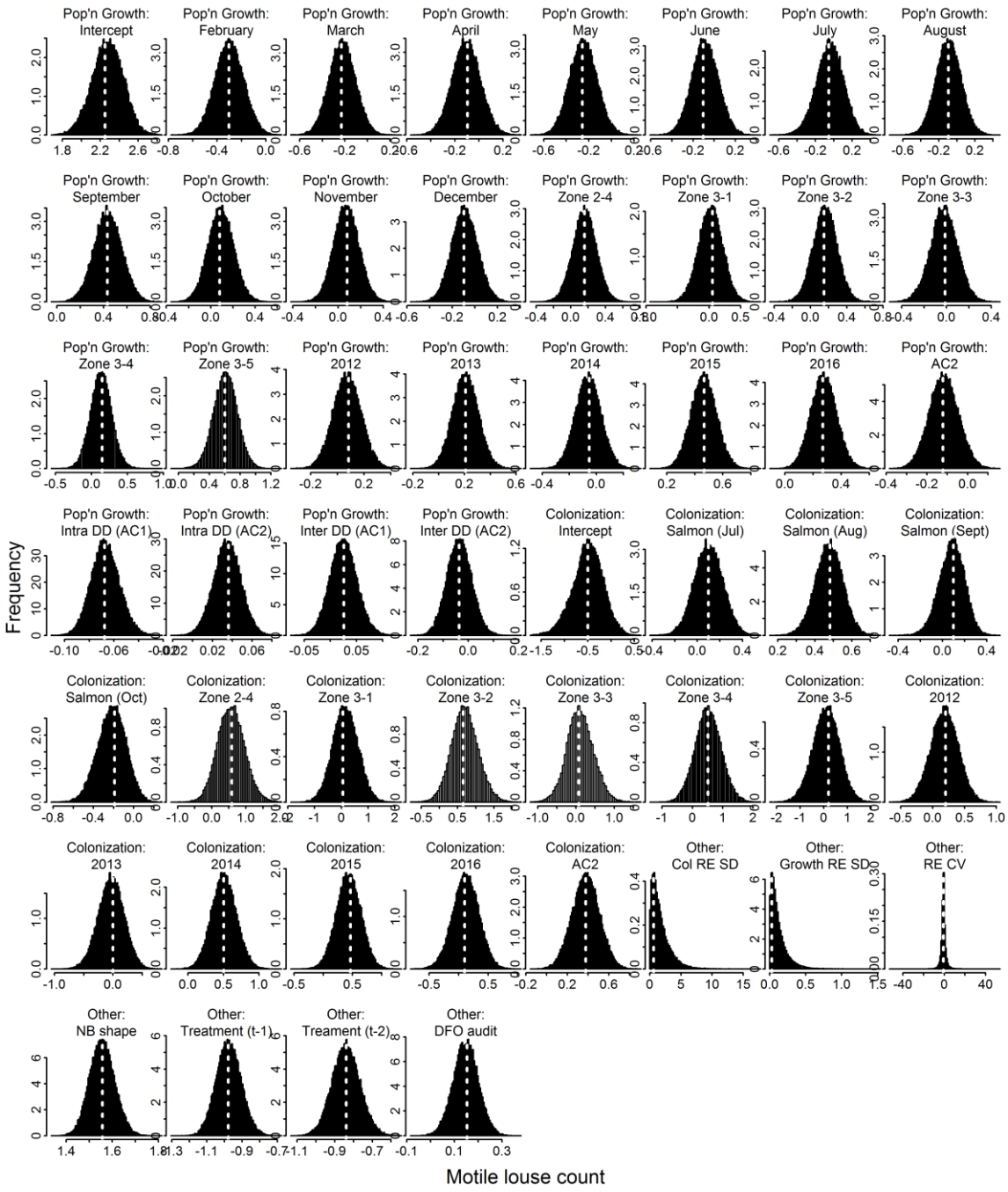

Figure D.4 Posterior distributions of the *L. salmonis* parameters[1].
The white dashed vertical lines denote the modal estimates. To best view the distributions, I do not constrain the axes to be consistent among panels.
[1] 'Pop'n growth rate' = population growth rate, 'AC1' = farmed salmon age class 1 (< 1 year in seawater), 'AC2' = farmed salmon age class 2 (>=1 year in seawater), 'Intra DD' = intraspecific density dependence, 'Inter DD' = interspecific density dependence, 'RE' = random effect, 'Col' = colonization, 'Growth' = population growth, 'SD' = standard deviation, 'CV' = covariation, 'NB shape' = negative binomial shape parameter.

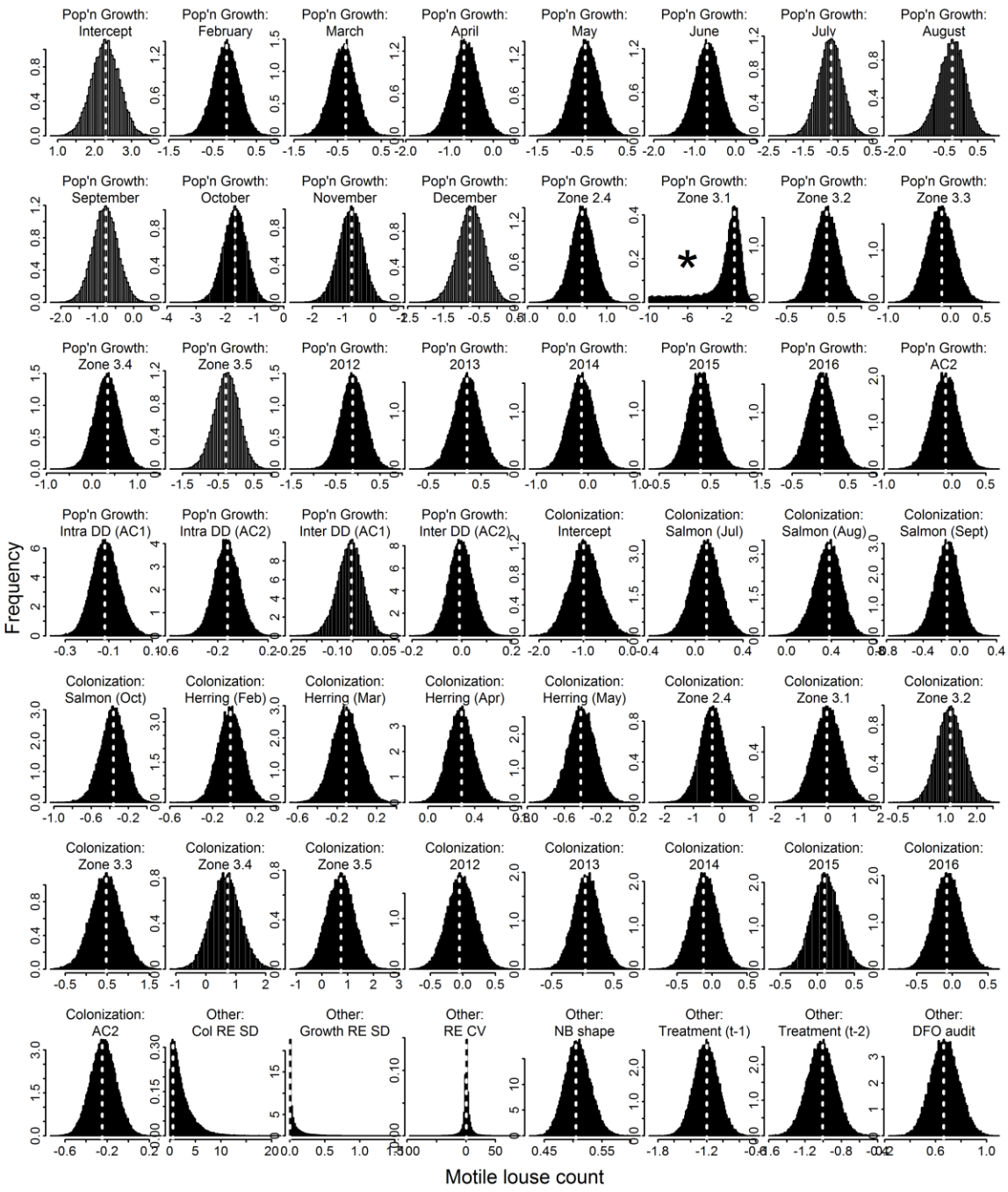

Figure D.5 Posterior distributions of the C. clemensi parameters[1].
The white vertical lines denote the modal estimates. To best view the distributions, I do not constrain the axes to be consistent among panels. The asterisk identifies the parameter that experienced fitting complications (see methods).
[1] 'Pop'n growth rate' = population growth rate, 'AC1' = farmed salmon age class 1 (< 1 year in seawater), 'AC2' = farmed salmon age class 2 (>=1 year in seawater), 'Intra DD' = intraspecific density dependence, 'Inter DD' = interspecific density dependence, 'RE' = random effect, 'Col' = colonization, 'Growth' = population growth, 'SD' = standard deviation, 'CV' = covariation, 'NB shape' = negative binomial shape parameter.

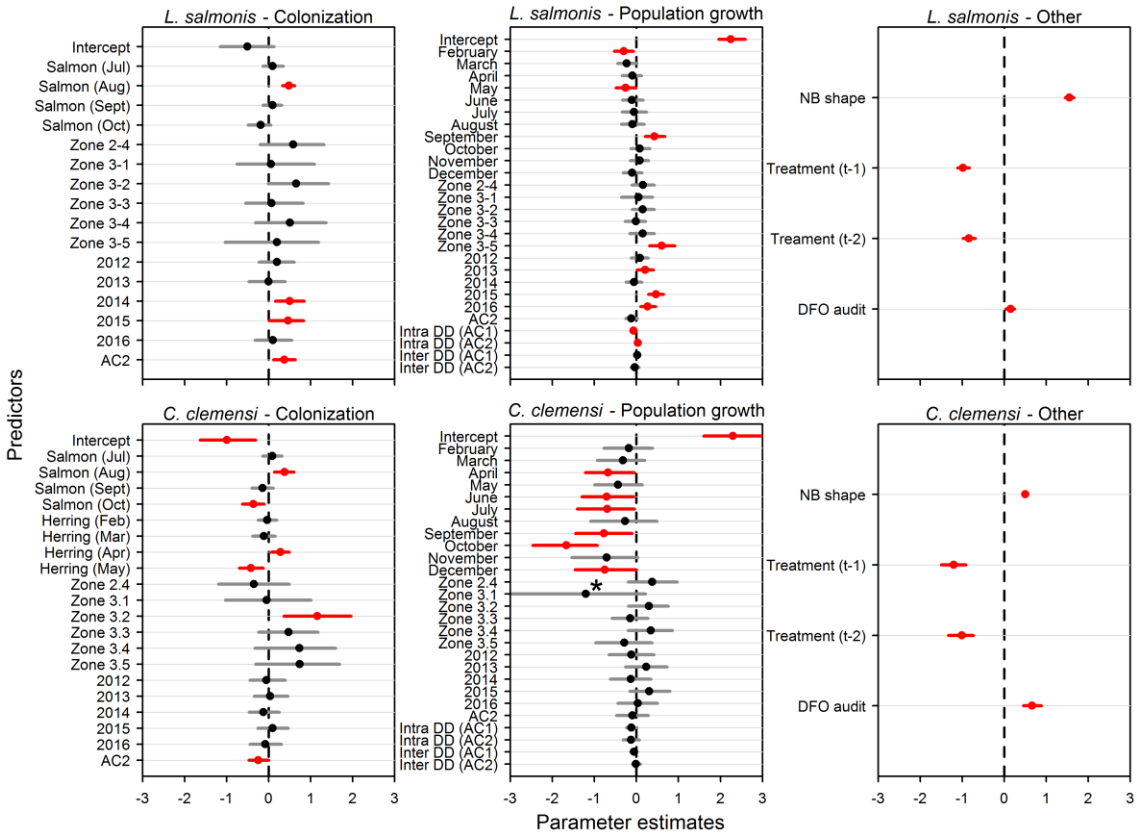

Figure D.6 Modal parameter estimates and their 95% credible intervals[1].
As in Figure D.3, the two rows of panels differentiate the parameters for *L. salmonis* (top) and *C. clemensi* (bottom), and the three columns of panels distinguish the parameters from the colonization linear function of the model (left), the population growth function (center), and elsewhere (right). Red points identify the parameters whose credible intervals do not overlap zero, which is indicated by the dashed vertical line. The asterisk identifies the parameter that had difficulty fitting (see methods).
[1] 'AC1' = farmed salmon age class 1 (< 1 year in seawater), 'AC2' = farmed salmon age class 2 (>=1 year in seawater), 'Intra DD' = intraspecific density dependence, 'Inter DD' = interspecific density dependence, 'NB shape' = negative binomial shape parameter.

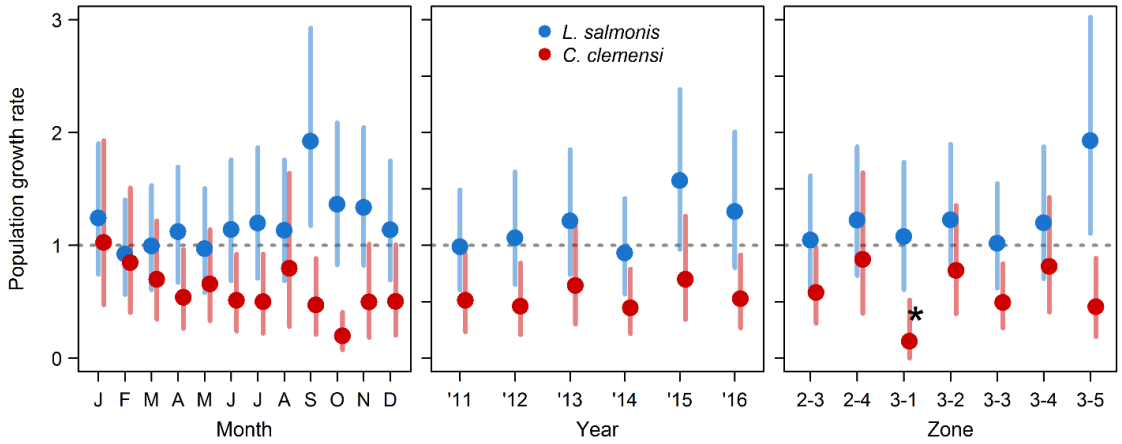

Figure D.7 Predicted effects of month (left panel), year (middle panel), and health zone (right panel) on population growth rate (λ) for motile lice on BC farms.

Predictions were made under the conditions that treatment last occurred more than two months ago and louse counts in the previous month were zero. Other predictors were set to the mean of observed values, except for the previous month's louse counts, audit, and wild local salmon and herring abundance, which were set to zero. The error bars give the 95% confidence intervals for the mean predictions and the horizontal dashed line indicates the replacement level, where the per-capita population growth rate is zero. The asterisk identifies the parameter that had difficulty fitting (see methods).

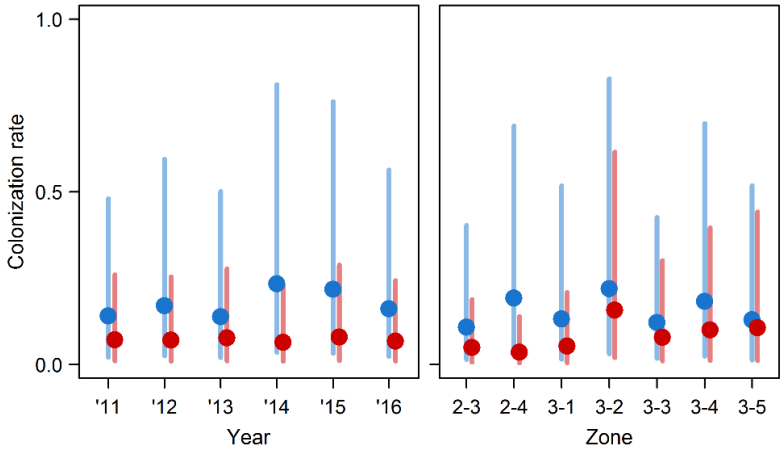

Figure D.8 Predicted effects of year (left panel), and health zone (right panel) on colonization rate (γ) for motile lice on BC farms.

Predictions were made under the conditions that treatment last occurred more than two months ago and louse counts in the previous month were zero. Other predictors were set to the mean of observed values, except for the previous month's louse counts, audit, and wild local salmon and herring abundance, which were set to zero. The error bars give the 95% confidence intervals for the mean predictions.

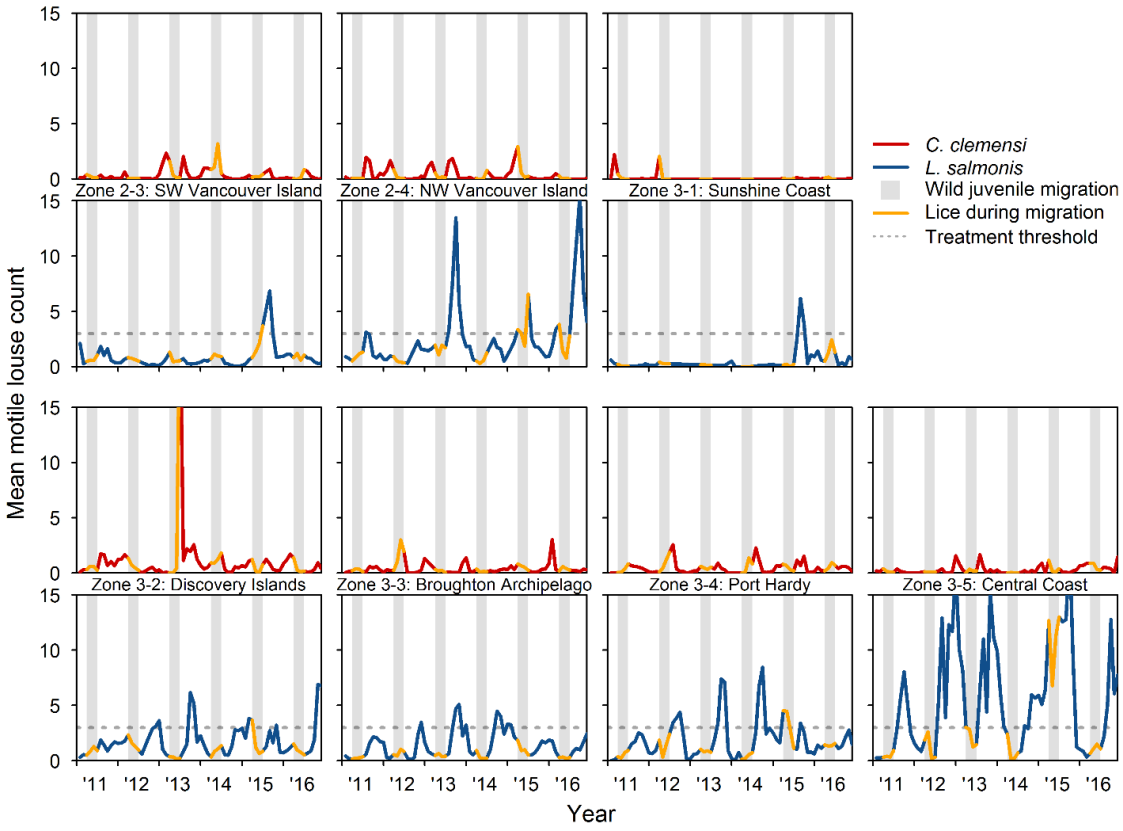

Figure D.9 Mean monthly motile louse counts on farms for each of the seven DFO fish health surveillance zones represented in the industry sea lice dataset between 2011 and 2016 in BC.

Each health zone (Figure 5.1) has a pair of panels, with *C. clemensi* counts shown in the top panel and *L. salmonis* counts in the bottom. Louse counts during the wild juvenile salmon migration, which occurs from April to June (i.e., the orange lines within the grey-shaded regions) are emphasized because louse management practices in BC focus on reducing louse abundances on farms during this time. The three-louse treatment threshold is only shown in the *L. salmonis* panels because *C. clemensi* counts do not trigger delousing treatment.

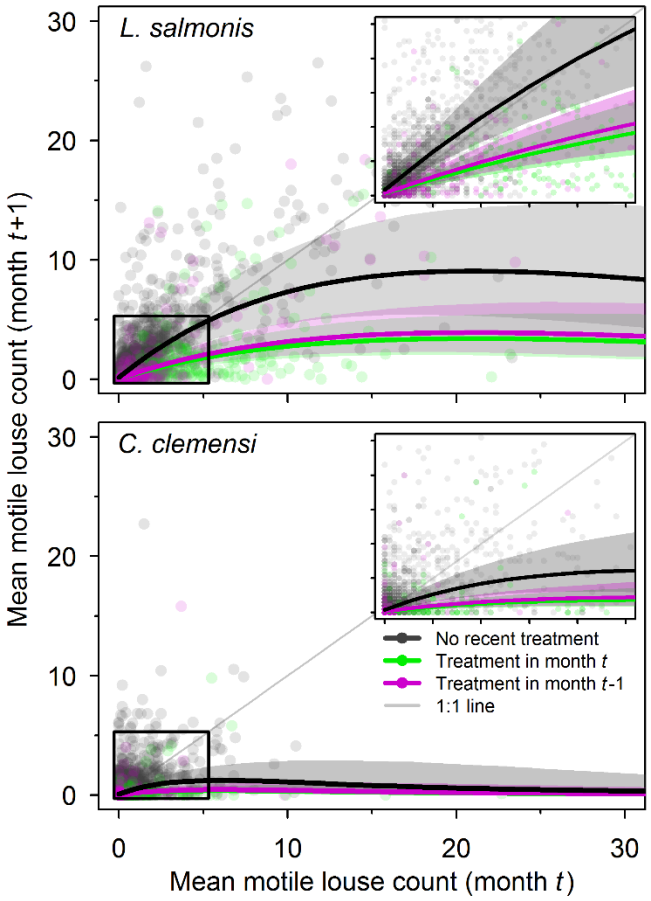

Figure D.10 Predicted effect of delousing treatment on population growth rate for *L. salmonis* (top panel) and *C. clemensi* (bottom panel).

Lines represent mean predictions for the three levels of the treatment predictor; all other predictors were set to the observed means, except for the previous month's louse counts, audit, and local wild salmon and herring abundances, which were set to zero. Shaded regions indicate the 95% confidence intervals for the mean predictions. Each point is a mean monthly count of motile lice from the publicly available industry dataset (n=2626). The asymptotic shape of the curves is due to density dependence. The black boxes in the main plot windows indicates the extent of the insets. One exceptionally high mean *C. clemensi* count was outside the bounds of the figure (count = 47.3), so two points are not visualized here (one with the high count in month *t* and one with the high count in month *t*+1).

Table D.1 Species composition of contributing populations to each management area's salmon abundance indices.

Area	Species	*n*	Proportion
12	chum	3	0.03
	coho	5	0.01
	pink	5	0.96
13	Chinook	4	0.01
	chum	11	0.06
	coho	8	0.02
	pink	7	0.91
	sockeye	1	0.00
15	Chinook	1	0.04
	chum	2	0.66
	coho	1	0.07
	pink	1	0.23
16	chum	5	0.99
	sockeye	1	0.01
23	Chinook	3	0.07
	chum	3	0.79
	coho	3	0.11
	sockeye	1	0.03
24	Chinook	5	0.09
	chum	5	0.53
	coho	5	0.19
	sockeye	4	0.19
25	Chinook	5	0.44
	chum	6	0.42
	coho	6	0.08
	sockeye	4	0.06
26	Chinook	4	0.02
	chum	6	0.72
	coho	6	0.25
	sockeye	3	0.01
27	Chinook	2	0.19
	chum	2	0.67
	coho	3	0.14
6	Chinook	1	0.00
	chum	27	0.08
	coho	10	0.01
	pink	40	0.90
	sockeye	7	0.01
7	chum	7	0.47
	pink	8	0.52
	sockeye	1	0.01

'Area' refers to the DFO management area, 'n' is the number of contributing populations for each species in that area, and 'proportion' is n divided by the number of contributing populations for the entire area.

Table D.2 Model parameter estimates, grouped by louse species and location in the model (i.e., the population growth linear function, the colonization linear function, or elsewhere)[1].

Species	Type	Name	Mode	LCI	UCI
C. clemensi	Pop'n Growth	Intercept	2.302	1.606	3.036
C. clemensi	Pop'n Growth	February	-0.179	-0.776	0.383
C. clemensi	Pop'n Growth	March	-0.317	-0.930	0.200
C. clemensi	Pop'n Growth	April	-0.672	-1.216	-0.046
C. clemensi	Pop'n Growth	May	-0.435	-0.997	0.138
C. clemensi	Pop'n Growth	June	-0.706	-1.300	-0.068
C. clemensi	Pop'n Growth	July	-0.695	-1.406	-0.050
C. clemensi	Pop'n Growth	August	-0.267	-1.096	0.485
C. clemensi	Pop'n Growth	September	-0.769	-1.448	-0.107
C. clemensi	Pop'n Growth	October	-1.667	-2.466	-0.931
C. clemensi	Pop'n Growth	November	-0.707	-1.535	0.027
C. clemensi	Pop'n Growth	December	-0.754	-1.463	-0.005
C. clemensi	Pop'n Growth	Zone 2.4	0.381	-0.194	0.971
C. clemensi	Pop'n Growth	Zone 3.1	-1.202	-8.274	0.214
C. clemensi	Pop'n Growth	Zone 3.2	0.302	-0.191	0.761
C. clemensi	Pop'n Growth	Zone 3.3	-0.142	-0.582	0.275
C. clemensi	Pop'n Growth	Zone 3.4	0.345	-0.198	0.857
C. clemensi	Pop'n Growth	Zone 3.5	-0.287	-0.969	0.374
C. clemensi	Pop'n Growth	2012	-0.122	-0.657	0.415
C. clemensi	Pop'n Growth	2013	0.234	-0.263	0.727
C. clemensi	Pop'n Growth	2014	-0.133	-0.620	0.345
C. clemensi	Pop'n Growth	2015	0.305	-0.156	0.795
C. clemensi	Pop'n Growth	2016	0.033	-0.442	0.503
C. clemensi	Pop'n Growth	AC2	-0.096	-0.474	0.280
C. clemensi	Pop'n Growth	Intra DD (AC1)	-0.118	-0.234	0.009
C. clemensi	Pop'n Growth	Intra DD (AC2)	-0.122	-0.321	0.068
C. clemensi	Pop'n Growth	Inter DD (AC1)	-0.054	-0.139	0.019
C. clemensi	Pop'n Growth	Inter DD (AC2)	-0.009	-0.095	0.093
C. clemensi	Colonization	Intercept	-0.991	-1.630	-0.307
C. clemensi	Colonization	Salmon (Jul)	0.093	-0.140	0.314
C. clemensi	Colonization	Salmon (Aug)	0.382	0.138	0.598
C. clemensi	Colonization	Salmon (Sept)	-0.141	-0.402	0.113
C. clemensi	Colonization	Salmon (Oct)	-0.362	-0.624	-0.111
C. clemensi	Colonization	Herring (Feb)	-0.032	-0.252	0.193
C. clemensi	Colonization	Herring (Mar)	-0.109	-0.381	0.158
C. clemensi	Colonization	Herring (Apr)	0.286	0.066	0.492
C. clemensi	Colonization	Herring (May)	-0.422	-0.693	-0.127
C. clemensi	Colonization	Zone 2.4	-0.349	-1.195	0.494
C. clemensi	Colonization	Zone 3.1	-0.042	-1.035	1.007
C. clemensi	Colonization	Zone 3.2	1.159	0.362	1.962

Species	Type	Name	Mode	LCI	UCI
C. clemensi	Colonization	Zone 3.3	0.476	-0.239	1.174
C. clemensi	Colonization	Zone 3.4	0.735	-0.331	1.593
C. clemensi	Colonization	Zone 3.5	0.740	-0.308	1.690
C. clemensi	Colonization	2012	-0.052	-0.444	0.391
C. clemensi	Colonization	2013	0.041	-0.339	0.461
C. clemensi	Colonization	2014	-0.121	-0.465	0.250
C. clemensi	Colonization	2015	0.096	-0.259	0.463
C. clemensi	Colonization	2016	-0.079	-0.441	0.306
C. clemensi	Colonization	AC2	-0.247	-0.472	-0.001
C. clemensi	Other	NB shape	0.505	0.465	0.550
C. clemensi	Other	Treatment (t-1)	-1.199	-1.495	-0.918
C. clemensi	Other	Treatment (t-2)	-1.007	-1.331	-0.734
C. clemensi	Other	DFO audit	0.665	0.460	0.883
L. salmonis	Pop'n Growth	Intercept	2.250	1.959	2.593
L. salmonis	Pop'n Growth	February	-0.302	-0.526	-0.074
L. salmonis	Pop'n Growth	March	-0.230	-0.447	0.007
L. salmonis	Pop'n Growth	April	-0.095	-0.337	0.121
L. salmonis	Pop'n Growth	May	-0.257	-0.485	-0.015
L. salmonis	Pop'n Growth	June	-0.106	-0.333	0.159
L. salmonis	Pop'n Growth	July	-0.053	-0.336	0.241
L. salmonis	Pop'n Growth	August	-0.094	-0.359	0.184
L. salmonis	Pop'n Growth	September	0.432	0.209	0.676
L. salmonis	Pop'n Growth	October	0.080	-0.134	0.318
L. salmonis	Pop'n Growth	November	0.080	-0.146	0.288
L. salmonis	Pop'n Growth	December	-0.097	-0.312	0.135
L. salmonis	Pop'n Growth	Zone 2-4	0.160	-0.108	0.425
L. salmonis	Pop'n Growth	Zone 3-1	0.055	-0.358	0.386
L. salmonis	Pop'n Growth	Zone 3-2	0.149	-0.098	0.424
L. salmonis	Pop'n Growth	Zone 3-3	-0.012	-0.266	0.214
L. salmonis	Pop'n Growth	Zone 3-4	0.150	-0.156	0.422
L. salmonis	Pop'n Growth	Zone 3-5	0.604	0.314	0.914
L. salmonis	Pop'n Growth	2012	0.085	-0.128	0.283
L. salmonis	Pop'n Growth	2013	0.209	0.017	0.405
L. salmonis	Pop'n Growth	2014	-0.053	-0.246	0.126
L. salmonis	Pop'n Growth	2015	0.466	0.285	0.640
L. salmonis	Pop'n Growth	2016	0.270	0.100	0.458
L. salmonis	Pop'n Growth	AC2	-0.121	-0.254	0.025
L. salmonis	Pop'n Growth	Intra DD (AC1)	-0.068	-0.089	-0.044
L. salmonis	Pop'n Growth	Intra DD (AC2)	0.036	0.008	0.062
L. salmonis	Pop'n Growth	Inter DD (AC1)	0.026	-0.026	0.074
L. salmonis	Pop'n Growth	Inter DD (AC2)	-0.036	-0.130	0.060
L. salmonis	Colonization	Intercept	-0.502	-1.151	0.128
L. salmonis	Colonization	Salmon (Jul)	0.098	-0.137	0.351

Species	Type	Name	Mode	LCI	UCI
L. salmonis	Colonization	Salmon (Aug)	0.482	0.329	0.619
L. salmonis	Colonization	Salmon (Sept)	0.097	-0.133	0.308
L. salmonis	Colonization	Salmon (Oct)	-0.190	-0.488	0.049
L. salmonis	Colonization	Zone 2-4	0.586	-0.209	1.318
L. salmonis	Colonization	Zone 3-1	0.059	-0.755	1.090
L. salmonis	Colonization	Zone 3-2	0.656	-0.014	1.425
L. salmonis	Colonization	Zone 3-3	0.074	-0.550	0.820
L. salmonis	Colonization	Zone 3-4	0.512	-0.316	1.368
L. salmonis	Colonization	Zone 3-5	0.203	-1.038	1.186
L. salmonis	Colonization	2012	0.202	-0.235	0.607
L. salmonis	Colonization	2013	0.001	-0.469	0.396
L. salmonis	Colonization	2014	0.504	0.160	0.843
L. salmonis	Colonization	2015	0.466	0.014	0.832
L. salmonis	Colonization	2016	0.102	-0.323	0.547
L. salmonis	Colonization	AC2	0.376	0.119	0.631
L. salmonis	Other	NB shape	1.558	1.450	1.661
L. salmonis	Other	Treatment (t-1)	-0.979	-1.109	-0.833
L. salmonis	Other	Treament (t-2)	-0.839	-0.983	-0.693
L. salmonis	Other	DFO audit	0.153	0.040	0.248

1 'LCI' = lower 95% credible interval, 'UCI' = upper 95% credible interval, 'Pop'n growth rate' = population growth rate, 'AC1' = farmed salmon age class 1 (< 1 year in seawater), 'AC2' = farmed salmon age class 2 (>=1 year in seawater), 'Intra DD' = intraspecific density dependence, 'Inter DD' = interspecific density dependence, 'NB shape' = negative binomial shape parameter.

Table D.3 Summary statistics for management responses to L. salmonis counts that exceeded the three-louse threshold for delousing treatment between 2011 and 2016 in BC.

Health zone	Number of counts	Proportion of counts over threshold	Mean # of months before action	Frequency of prompt action
2-3: SW Vancouver Island	509	0.06	0.52 ± 0.24	0.88
2-4: NW Vancouver Island	376	0.16	1.12 ± 0.33	0.69
3-1: Sunshine Coast	122	0.07	0.40 ± 0.37	0.67
3-2: Discovery Islands	430	0.14	1.03 ± 0.29	0.78
3-3: Broughton Archipelago	690	0.12	1.74 ± 0.36	0.62
3-4: Port Hardy	273	0.19	1.52 ± 0.36	0.63
3-5: Central Coast	226	0.37	1.52 ± 0.28	0.62

Management responses to a threshold-breaking counts can take one of two forms: delousing treatment or harvest of stocked fish. An 'action' refers to one of these two management responses, and a 'prompt action' is one that occurred by the end of the month following the threshold-breaking count. Means are given with standard errors.

Supplementary references

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