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Population biology of infectious diseases shared by wild and farmed fish

Martin Krkošek

Department of Ecology and Evolutionary Biology, University of Toronto, 25 Harbord St, Toronto, ON, M5S 3G5

Salmon Coast Field Station, Simoom Sound, BC, V0P 1S0

Abstract

Global fisheries landings ceased increasing decades ago, causing an increasing shortfall in wild seafood supply and an expansion of aquaculture. The abundance of domesticated fishes now dwarfs related wild fishes in some coastal seas, changing the dynamics of their infectious diseases. Transport and trade of seafood, feed, eggs, and broodstock bring pathogens into new regions and into contact with naïve hosts. Density-dependent transmission creates threshold effects where disease can abruptly switch from endemic to epizootic dynamics. Hydrodynamics allow pathogens to disperse broadly, interconnecting farms into meta-populations of domesticated host fish in regions that also support related species of wild fish. Spill-over and spill-back dynamics of pathogen transmission between wild and farmed fish can create novel transmission pathways or bioamplify pathogen abundance, potentially depressing or endangering wild fish. Mortality from natural predator-prey interactions may be synergistic or compensatory with these increased infections. Domestic environments may favour the evolution of undesirable pathogen traits, such as virulence and drug resistance, leading to the emergence of strains that cause high mortality and/or evade treatment. Overall, these changes to the dynamics of infectious disease in coastal seas impose new constraints on the sustainability of both wild and farmed fish.

Introduction

It has been about 30 years since global fisheries landings stopped increasing (Figure 1)(FAO 2016). While fisheries and conservation science have contested the sustainability of marine resource use (Pauly et al. 1998, Myers and Worm 2003, Walters 2003, Worm et al. 2009, Branch et al. 2010), it has become clear that fisheries have not and cannot keep pace with the growing global demand for seafood (FAO 2016). The resulting deficit in the supply of wild seafood has caused a switch from hunting to farming (i.e., aquaculture) in coastal seas (Figure 1) (Goldburg and Naylor 2005, Duarte et al. 2007). Overall, aquaculture now supplies as much seafood for human consumption as global fisheries (FAO 2016), and trends in fish production indicate continued saturation of aggregate fisheries production and rapid growth in domesticated marine and diadromous fish populations (Figure 1).

With this blue revolution, some coastal seas are becoming managed for aquaculture production (Goldburg and Naylor 2005). Large populations of domesticated fish now inhabit some ocean regions, where marine ecosystems provide both services and disservices to the aquaculture industry. Services include the provisioning of fresh seawater and the assimilation of waste, whereas disservices include a diversity of parasite infections (ranging from viruses to metazoans) that originate from sympatric wild fishes (Nowak 2007, Walker and Winton 2010b). Infectious diseases cost shellfish and finfish aquaculture industries billions annually and pose disease risks for wild fish (Krkošek et al. 2006, Lafferty et al. 2015). The population biology of fish and parasites should alert us to changes in the epidemiology of infectious diseases in a future of domesticated seas (Grenfell and Dobson 1995, Mennerat et al. 2010).

In this paper I discuss the ecological and evolutionary theory of marine infectious diseases in the context of global trends in aquaculture and fisheries. To be clear, I refer to parasites as the full assemblage of microparasites (e.g. viruses and bacteria) and macroparasites

(e.g. helminthes and sea lice). I begin by situating fisheries and aquaculture within the framework of emerging infectious diseases (EID), and then discuss the main ecological and evolutionary dynamics that characterize the population biology of these host-parasite systems. My focus is on population level theory of parasites and wild fish, and how the introduction of domesticated fish may affect dynamics. I conclude with a discussion of how the ecological and evolutionary dynamics described may affect future epidemiology of marine infectious diseases given a global context of saturated fisheries and expanding aquaculture.

Emerging Infectious Diseases of Fish

The emergence of infectious diseases is often traced to the intensification of parasite transmission among humans, domesticated animals, and wildlife (Daszak et al. 2000). Diseases such as plague, avian flu, HIV, and Ebola, are all examples of emergence and spread in this way. The growth of domesticated fish populations raised in coastal seas has created the spill-over (parasite transmission from wild to farmed animals) and spill-back (parasite transmission from farmed to wild) dynamics that are key ingredients for disease emergence (Daszak et al. 2000). Further, there are unique characteristics of marine epidemiology that may facilitate disease emergence and spread in coupled wild and farmed fish populations.

The dynamics of infectious diseases in the oceans differ from those on land in several ways (McCallum et al. 2004). First, ocean environments have fewer barriers to the movement of hosts and pathogens. Second, large migrations of fish are common, and ocean currents can carry both hosts and pathogens for long distances. Third, common behaviours such as aggregation of fish hosts in shoals and schools can further facilitate disease spread. As a result of these differences infectious diseases have rates of spatial spread in the oceans that are one to two orders of magnitude faster than on land (McCallum et al. 2003). For example, herpes epidemics

of Australian pilchards in the 1990s (Jones et al. 1997, Gaughan 2001) spread at 10,000 km per year (McCallum et al. 2003).

Analyses have shown that cases of marine infectious disease are increasing for some host taxa but decreasing for other taxa (Lafferty et al. 2004, Ward and Lafferty 2004). Overall, diseases in wild marine fish have been decreasing (Ward and Lafferty 2004), potentially because of a process known as fishing out parasites (Dobson and May 1987, Wood et al. 2010). The reduction in wild fish population sizes due to fishery harvests has the potential consequence of reducing the transmission success of parasites that have density-dependent transmission. Comparisons have suggested that fish populations in marine protected areas have higher abundances and diversity of parasites (Lafferty et al. 2008).

At the same time, however, infectious diseases of farmed fish are a significant impediment for aquaculture production (Walker and Winton 2010a, Lafferty et al. 2015). A recent review of EID (there defined as emergence of previously unknown diseases, or spread of existing disease into new host species or geographic locations) in wild vertebrates indicated that freshwater and marine fishes have the highest rates of disease emergence amongst vertebrates, which is at least partially because they are the most speciose group (Tompkins et al. 2015). The most common mechanism of EID in other taxa was human-mediated transmission whereas for wild fish, it was mostly direct transmission between wild and domesticated fish. Approximately 75% of new and ongoing cases of EIDs in wild fish since 2000 have co-occurred in aquaculture. These patterns contain a sample bias for temperate species of commercial importance, like salmon.

Overall, these trends of disease cases in wild and farmed fish suggest that coastal seas utilized for fishing and aquaculture have seen a decline in wild fish stocks and their parasites due to fisheries effects on fish stocks, but an increase in domesticated fish populations and their

parasites due to aquaculture expansion. In seas where wild and farmed fish share parasites, this creates parasite-mediated apparent competition that is modified by economic and policy feedbacks. Theory of such interactions is currently lacking but is needed to understand the dynamics of such coupled human and natural systems. In the next sections I discuss key ecological and evolutionary processes that are likely to modify the epidemiology of infectious diseases in coastal seas due to the transmission of parasites between wild and domesticated fish.

Parasite Dispersal

Hydrodynamics of coastal seas can allow parasite transmission to occur over long distances. Physical hydrodynamic variables such as salinity, temperature, UV radiation, wind, river discharge, and tidal currents all can affect the distance that parasites can spread in a coastal marine environment (Garver et al. 2013, Foreman et al. 2015). For example, the dispersal of sea lice from salmon farms can extend for 30 km or more into the surrounding ecosystems (Krkošek et al. 2005). Hydrodynamic models of parasite dispersal that incorporate the dependency of parasite demographic rates on environmental variables have been useful for studying the spatio-temporal dynamics of parasite dispersal among fish farms (Amundrud and Murray 2009, Stucchi et al. 2011). Thus, there is a need for a regional perspective on marine epidemiology, in which the spatial scale includes metapopulation structure where subpopulations of parasites in fish farms are interconnected (Aldrin et al. 2013). As a corollary, the population dynamics of parasites shared between wild and farmed fish will likely be organized at regional scales that involve aggregations of aquaculture activity and wild fish habitat/migrations.

Host Migration

Migrations of marine fish species are common, and migrations of host populations have a number of implications for infectious disease dynamics including migratory culling, escape, stalling, and allopatry (Altizer et al. 2011). Migratory culling refers to situations where infected individuals are lost from the migratory population due to mortality or an inability to keep pace (Bradley and Altizer 2005). Migratory culling can lead to declines in both host population size and parasite abundance during migration. Migratory escape refers to the situation where hosts migrate to new environments where infection risk is lower because local parasite abundance has not yet accumulated (Hall et al. 2014). Alternatively, a negative effect of infection on migration speed may create a dynamic that may result in a slowing or stoppage of the migration, depending on how the collective behavior of hosts in a migrating population responds to parasitism. Migratory allopatry describes a situation where the natural host lifecycle involves a migration that separates age-classes, effectively stopping transmission from old to young individuals for long periods during ontogeny (Krkošek et al. 2007).

Aquaculture may interact with these migratory processes in a variety of ways. In particular, it can situate large populations of domesticated hosts on the migration routes of wild fish (Figure 2), creating opportunities for transmission to occur in phases of the ontogeny of wild fish for which there is little evolutionary history or pre-adaptation to elevated levels of parasite exposure (Krkošek et al. 2005). Fish with life histories that use coastal habitats for juvenile feeding and growth or with life histories entirely situated in coastal habitats are particularly vulnerable to parasites whose levels may be elevated by the growth of domesticated fish populations. Migratory stalling may also cause migrating fish populations to extend their stay in habitats where parasite abundance is high, potentially creating a positive feedback between parasite levels, slowing migration speed, and mortality. In addition, parasite exposure may alter the timing and duration of migrations. For example, there is evidence of migratory escape in sea

142 trout, where they prematurely terminate their marine migration and return to freshwater habitats
143 to escape high sea-louse parasite abundances (Birkeland and Jakobsen 1997). Also, sea lice
144 infection experienced by juvenile Atlantic salmon is associated with delayed maturity and
145 therefore delayed spawning migrations of adult salmon (Vollset et al. 2014).
146
147 **Parasite translocation**
148 A common mechanism for EID in coastal marine fish is the transport of seafood products, feed,
149 eggs, or broodstock that can contain parasites. A consequence is that parasites become
150 introduced into domesticated and wild fish populations in new regions, contributing to spatial
151 spread of infection. Further, the recipient host fish populations may have little evolutionary
152 history with the parasites (i.e. naïve host populations vulnerable to epizootics).
153 For example, it is thought that frozen whole pilchards that are imported to Australia as
154 feed for tuna ranches were a source of two epidemics of herpes virus that swept through
155 Australian and New Zealand pilchard stocks in the late 1990s (Jones et al. 1997, Gaughan 2001).
156 Those epidemics spread at 10,000 km per year (McCallum et al. 2003), causing mass mortality
157 of ~30,000 tonnes in Western Australia in 1998-99, (Gaughan et al. 2000) and a 75% decline in
158 pilchard spawning biomass (Ward et al. 2001b). Such parasite induced mass mortality can have
159 cascading ecological effects; the pilchard die-off led to the expansion of competitor species
160 (Ward et al. 2001a), reproductive failure in penguins (Dann et al. 2000), and diet shifts in
161 gannets (Bunce and Norman 2000).
162 In farmed fish, epidemics of infectious salmon anemia virus (ISAv) in Chilean salmon
163 farms have been traced to the introduction of an endemic Norwegian strain (Kibenge et al. 2009),
164 causing a 75% decline in farmed salmon production from the world's second biggest producer
165 (Asche et al. 2009). Both ISAv and Piscine Reovirus (PRV – the agent associated with heart and

skeletal muscle inflammation disease) (Palacios et al. 2010) are considered exotic to the North Pacific Ocean, but PCR tests of farmed and wild salmon have indicated that strains of these ancestrally Norwegian viruses may now be present in both wild and farmed salmon in British Columbia (Kibenge et al. 2013, Marty et al. 2015, Kibenge et al. 2016). These examples underscore the need for continuing and increasing biosecurity practices that focus on the trade and movement of seafood products - including aquaculture products, feed, eggs, and broodstock - at regional and global scales.

Host-density thresholds

The population dynamics of infectious diseases commonly involve threshold effects due to density dependent transmission (Grenfell and Dobson 1995, Lloyd-Smith et al. 2005). Density dependent transmission occurs for diseases where the transmission rate is an increasing function of the density of hosts. Exceptions occur where parasites retain high levels of transmission success even if hosts are not abundant because of host aggregation (e.g. sexually transmitted diseases) as well as vectors that actively search for hosts (e.g. mosquitos). In these cases, the transmission rate is frequency dependent (i.e. it depends on the prevalence of infection but not on the density of hosts). For fish, it is common for macroparasites to have an indirect lifecycle that involves invertebrate intermediate hosts, but active vector-borne transmission appears to be relatively rare. Due to external fertilization, sexually transmitted diseases are likely to also be rare for fish. These patterns mean that parasite life-histories that have density dependent transmission are likely to make up the majority of fish diseases, although such patterns have not been empirically quantified.

Density dependent transmission creates a threshold effect in the population dynamics of disease. At low host densities an infection will fade out after being introduced, whereas at high

host densities an initial infection will, on average, spread to at least one or more other hosts. There is therefore a critical host density threshold that separates two sets of dynamics, one where diseases fade out and another where diseases spread. Because the critical host density is a function of environmentally-dependent demographic parameters, the threshold itself is also likely to be a function of such factors as temperature and salinity (e.g. sea lice), or UV radiation (e.g. degradation of viruses). For fisheries and aquaculture, sea lice and salmon are an example of a macroparasite-host system that has density dependent transmission and thresholds that divide endemic and epidemic conditions (Frazer et al. 2012, Jansen et al. 2012).

A simple theoretical example using a susceptible-infected epidemiological model suggests how microparasite (e.g. viruses and bacteria) dynamics may switch from a low endemic state to epizootic dynamics as aquaculture production increases (Figure 3). Infection levels initially occur at a low endemic state that is maintained by pathogen immigration when farmed fish abundance is below the threshold ($N < N_c$). Dynamics then transition into epizootic when farmed fish abundance exceeds the host density threshold ($N > N_c$)**.** Empirically, the dynamics of microparasitic disease in farmed fish will be more variable than this simple model, due to the effects of environmental and demographic stochasticity, variation in management and biological variables, and spatial factors discussed earlier such as parasite dispersal, fish migration, and metapopulation dynamics. Nonetheless, the model illuminates how the epidemiology of disease can change abruptly in response to gradual growth in domesticated fish populations.

Allee effects

Allee effects – reduced fitness at low population abundance - may create other thresholds in the population dynamics of host-parasite systems. Whereas the critical host density thresholds discussed above involve dynamics where parasite demography is dependent on host density,

Allee effects refer to situations where parasite demography depends on parasite abundance and where host demography depends on host abundance.

For parasites, demographic Allee effects may occur through at least two mechanisms. Mate limitation likely occurs for dieocious parasite species (males and females are separate individuals) because the probability a male and a female occur in the same host decreases at low parasite abundances (May 1977). Separately, Allee effects may occur for parasites that have positive-sloped dose-response curves (probability of pathogen invasion in an individual host is an increasing function of the abundance of pathogens to which it is exposed) (Regoes et al. 2002). These mechanisms can create a breakpoint in the parasite population dynamics that divides initial conditions under which a large founding parasite population will grow versus those in which a small initial number of parasites will fail to establish new infections in the host population. Thresholds arising from Allee effects in parasite demography may slow the spread of infection between wild and farmed fish populations and provide a target for managing parasite abundance (Krkošek et al. 2012).

For wild fish exposed to parasites from a reservoir host population, an Allee effect can occur because the abundance of parasitic infectious stages is decoupled from host abundance and so large numbers of parasites can remain in the environment and become concentrated on the few remaining hosts (Krkosek et al. 2013). Here, the dynamics are conceptually analogous to the Allee effect that occurs in the population dynamics of a prey population subject to predation by a type II predator (McLellan et al. 2010). Thus for wild fish exposed to parasite transmission from a reservoir domesticated host population, the emergent Allee effect may contribute to population decline and/or endangerment.

Foodweb dynamics

In many infectious disease systems, there are multiple host populations that interact directly and indirectly within a foodweb. An example of a negative indirect interaction involving hosts is apparent competition in which a parasite that is shared by two host populations creates a negative correlation between the demographies of the two host populations, creating a false impression that the two hosts are competitors (Hatcher et al. 2012). Two examples of parasites with multi-host dynamics that also involve domesticated fish are infectious hematopoietic necrosis virus (IHNv) and the sea louse Caligus clemensi, both of which infect salmonids, herring and other species. Even for salmonid-specific parasites, such as salmon lice, there are multiple salmon species that may interact via competition, predation, and shared parasites.

The indirect effects created by foodweb interactions among hosts can also involve predators that exacerbate or counteract parasitism in multi-host communities. If predators have a type-II functional response (i.e., attack rate is limited by the predator's capacity to process prey), in which the attack rate increases with the parasite load of prey, then predators may exacerbate mortality rates from parasites consuming more hosts when host abundance is low, but may also reduce the overall parasite load without changing overall mortality when hosts are abundant by focusing predation on the fraction of infected hosts (Krkošek et al. 2011) (Figure 4). In communities with both parasite and predator-mediated apparent competition, the combination of predator selectivity and parasite effects on predation risk of prey may result in predation becoming concentrated on the preferred prey, exacerbating mortality in the preferred host species but also releasing the less-preferred host from predation (Peacock et al. 2014, Peacock et al. 2015).

Climate Change

Parasite demographic rates and fish population sizes and ranges are all affected by variation in ocean conditions. Theory and empirical work indicate that parasites and hosts have optimal thermal ranges, and so depending on the juxtaposition of host and parasite responses to changes in climate, the outcomes may involve range shifts and local decreases or increases in the intensity of an infectious disease (Lafferty 2009, Molnar et al. 2013). For example, some cold-water diseases of fish may become less problematic or may no longer constrain habitat used by wild and farmed fish as temperature increases (Nematollahi et al. 2003). For other disease systems, parasite demographic rates may accelerate with small increases in temperature, generating faster population growth and elevated risk of epizootics (Groner et al. 2014, Bateman et al. 2016). Climate variation may also have important implications for parasite dispersal and persistence in the marine environment (O'Connor et al. 2007), since the demography of infective stages depends on temperature, salinity, UV radiation and other physical factors (Groner et al. 2014, Foreman et al. 2015). Both long-term and seasonal climate variation may therefore contribute substantially to disease outbreaks or the release of farmed or wild fish populations from disease burden.

Virulence

Evolutionary theory indicates that increases in host density should select for increased levels of virulence (reduction in host fitness due to the parasite) (Day 2001, Day and Gandon 2007). This is the result of a trade-off between parasite transmission and parasite-induced host mortality. In an environment of low host abundance, natural selection will favor a parasite phenotype that has low virulence so as to not kill its current host before the parasite's offspring are transmitted to a new host. In an environment where host density is high, transmission success of individual parasite offspring becomes saturated and so parasite phenotypes that have high fecundity are

favoured, but that usually occurs at the expense of host demography, typically survival. Further ecological factors such as predation or food limitation that differ between wild and domesticated environments may cause the transmission-virulence trade-off curves to shift in favour of increased virulence in domesticated environments (Figure 5). For fisheries and aquaculture, there is evidence that trait variation and conditions in domesticated environments can select for increased virulence of parasites on fish farms, including bacterial (Pulkkinen et al. 2010) and macroparasitic diseases (Mennerat et al. 2012).

Kennedy et al. (2016) identified eight factors that may lead to selection for increased virulence of pathogens in aquaculture systems. These included four related to intensive aquaculture operations: high-density rearing of farmed fish, which aids transmission; shortened rearing cycles of farmed fish, which selects for faster transmission rates of parasites; low genetic diversity of broodstock, which allows invasion of virulent specialist strains of parasites; and endemic infections in farmed stocks, which creates opportunities for adaptation of parasites. An additional four factors relate specifically to disease control practices: incomplete vaccination, which creates dynamics similar to the endemic infection described above; breeding for resistance, which because it doesn't completely prevent infection or transmission can similarly select for increased virulence; chemotherapy, which can select for virulence if the mechanism that selects for drug resistance is also linked to virulence; and management practices that favour horizontal transmission (among conspecifics) over vertical transmission (parent to offspring).

Drug resistance

Parasites of farmed fish have evolved resistance to antibiotics and pesticides used in fish health management, creating a need for new medicines and treatment applications that forestall the evolution of resistance. For bacterial diseases, antibiotic resistance in pathogens of farmed fish

also carries risks to human health, since the bacteria under selection on fish farms can exchange genes with bacteria that affect humans (Cabello 2006). For example, molecular studies indicate the genetic determinants of resistance to drugs like tetracycline, trimethoprim, sulfonamide and streptomycin can and in some cases have been exchanged between the salmon pathogen *Aeromonas salmonicida* and a variety of human pathogens including *Vibrio cholerae, Escherichia coli, Aeromonas hydrophila,* and *Aeromonas caviae* (Cabello 2006). While wild fish may ultimately be the origin of most infections of farmed fish, continued transmission between wild and farmed fish may be important for the sustainability of health management practices in aquaculture (Kreitzman et al. 2016). That is because wild fish provide a refuge from selection for antibiotic resistance in parasites that also infect domesticated fish. Thus, maintaining genetic connectivity between the domesticated fish and the wild refuge may be important for slowing or stalling the evolution of resistance in parasites of domesticated fish.

Management and innovation

The challenges of infectious disease in fisheries and aquaculture have already spurred innovations to control disease and promote fish health (Pohlenz and Gatlin 2014, Jones et al. 2015, Naslund and Johnsson 2016). The obvious example is the production of new pesticides, medicines, and vaccines (Lorenzen and LaPatra 2005, Shoemaker et al. 2009). Some viral diseases that previously caused epidemics in farmed fish are now effectively controlled with new vaccines (Sommerset et al. 2005). Evolutionary responses of parasites to drug-based disease control has led to treatment failure in some areas, further spurring the need for new drugs and find alternative means of controlling disease and slowing parasite evolution. The idea that wild fish may provide an evolutionary ecosystem service of slowing the emergence of drug resistance

is not a formal part of management, yet the service may well explain why drug resistance in sea lice has evolved in the North Atlantic but not the North Pacific (Kreitzman et al. 2016).

A move to coordinated area management recognizes that dispersal connects disease dynamics among farms and between wild and farmed fish, and so parasite management on farms is becoming increasingly coordinated at regional scales (Jones et al. 2015, Ellis et al. 2016). Coordination of the timing of parasite treatment on farms to minimize parasite levels during periods of wild fish migrations may effectively protect wild fish without increasing the overall frequency of drug use (Peacock et al. 2013, Rogers et al. 2013).

Furthermore, knowledge from foodweb ecology has helped identify and implement parasite control systems that include the co-stocking of cleaner fish, which consume parasites on farmed fish, as well as multi-trophic aquaculture systems where filter feeders utilize the waste from finfish farms, and kelp utilize the nutrient enrichment from both finfish and filter feeders (Mordue and Pike 2002, Treasurer 2002). Domesticated fish populations have also become more resistant to disease outbreaks via selective breeding as well as improved husbandry and site selection that reduce sources of stress that can make fish more prone to disease (Pohlenz and Gatlin 2014, Ellis et al. 2016, Naslund and Johnsson 2016).

Conclusions

The theory of parasite population biology indicates that numerous ecological and evolutionary dynamics of parasites depend on host abundance and transmission between multiple host populations, particularly wild and domesticated animals. There are therefore clear predictions from theory as to the epidemiological consequences of aquaculture growth, which have important implications for both wild and farmed fishes. Some predictions are already realized, such as the evolution of drug resistance (Westcott et al. 2010), increased virulence (Sundberg et

al. 2016), and density-dependent shifts from endemic to epizootic dynamics (Frazer et al. 2012). Theory of disease risk and animal migration is clearly relevant for wild and farmed fish, particularly aspects that elevate disease, such as migratory allopatry and stalling (Altizer et al. 2011), but the generality of these processes is not yet clear. Other aspects such as Allee effects or evolutionary ecosystem services of slowing drug resistance remain mostly theoretical and although they may mediate disease dynamics on farms, it is not clear to what extent they will be taken up or be of practical use in management.

A key consideration not yet discussed is uncertainty. While theory provides clear predictions as to the epidemiological consequences of aquaculture growth, it is much more difficult to predict the details of where, when, and for whom EID will occur. For example, while theory predicts continued emergences of virulent parasite strains it is not possible to presage the exact details of timing, location, and extent because any true model of such dynamics would be inherently stochastic. The issue of parameter uncertainty and parameter dependency on abiotic variables also makes it difficult to make precise predictions, for example, when a host-density threshold for a disease outbreak may be exceeded (Frazer et al. 2012). There is also an issue of structural uncertainty in spatial scale, spatial structure, biological variables, or their interactions so that it is not possible to know with certainty the exact model from which to make theoretical predictions for particular contexts. Models will also vary among species, and a crux for future research is to understand that variation to explain why only some parasites emerge as disease problems for wild and farmed fish despite a diverse range of parasites that can be exchanged.

Nevertheless, while the theory of parasite population biology will necessarily fall short of making precise predictions on epidemiological changes, it does provide an informative framework for interpreting the causes of observed outbreaks, changes in pathogen traits, management successes and failures, and future expectations (Peacock et al. 2013, Bateman et al.

2016, Groner et al. 2016). This connection between theory and practice does not only point to some utility for management but also opportunities to develop, test and refine theory. Looking forward, both theory and empirical work suggest that a future of continued aquaculture growth includes an intensification of ecological and evolutionary processes that facilitate disease emergence. EID events in coastal seas with intensive aquaculture will likely continue with little or no warning, and they will likely occur with increasing frequency, challenging innovation in disease management to keep pace. Understanding and managing marine diseases will therefore be critical to navigating towards a future for coastal seas in which prosperous aquaculture activity coexists with productive coastal ecosystems and fisheries.

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645 Figure 1.

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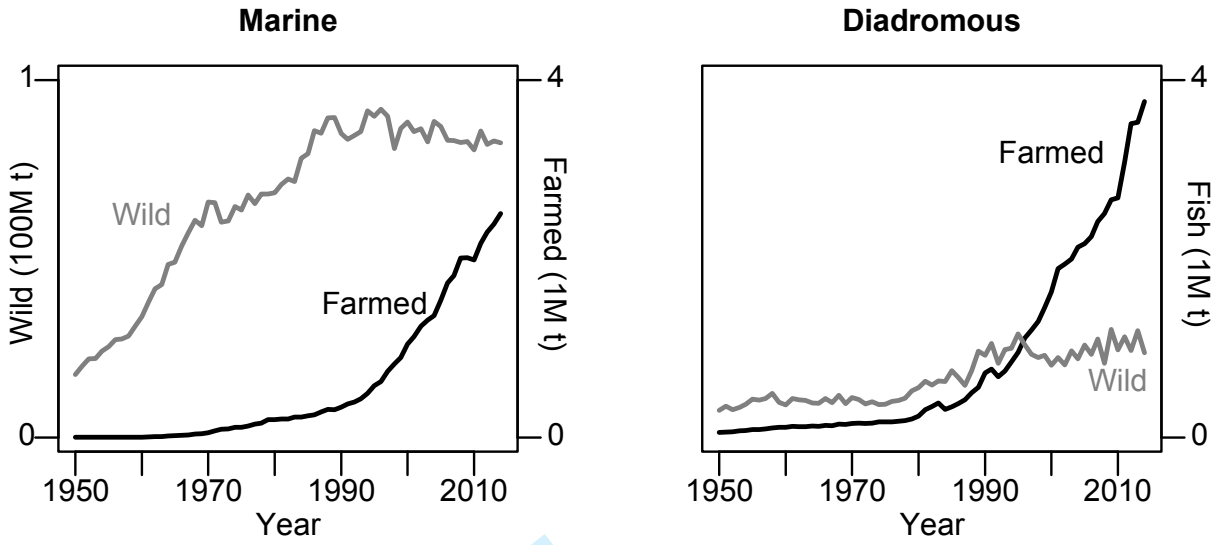

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649 **Figure 1.** Global production from fisheries and aquaculture of marine (left panel) and
650 diadromous (right panel) fish in marine and brackish waters. The diadromous panel excludes
651 marine and freshwater fishes and the marine panel excludes diadromous and freshwater fishes.
652 Note that the y-axis scale for marine fish differs between wild and farmed fish but is the same for
653 diadromous fish of wild and farmed origin. Data source: (FAO 2016).
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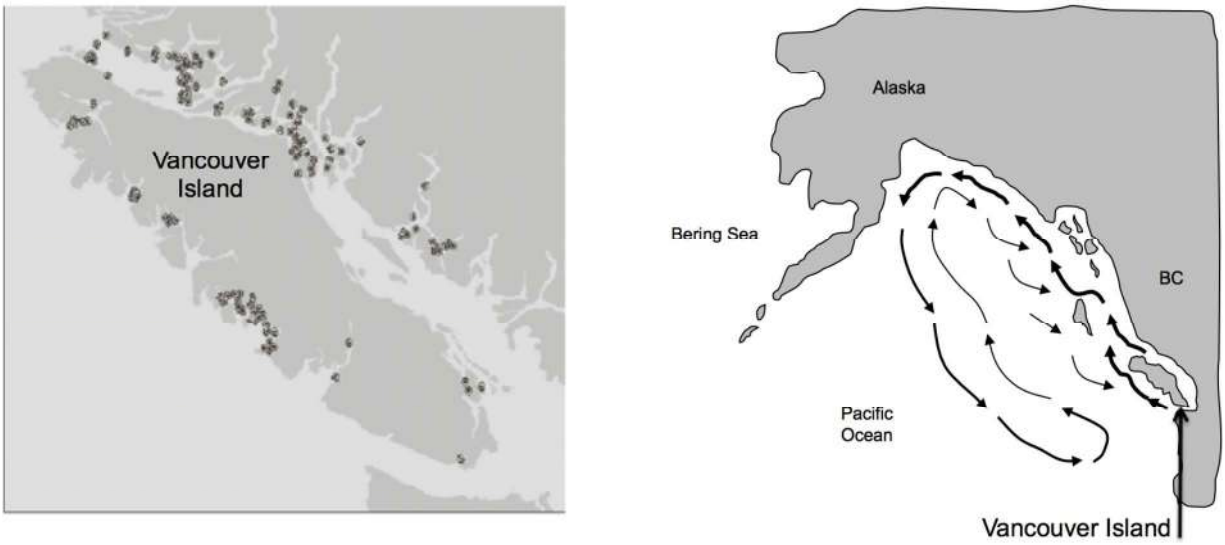
657 Figure 2



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660 **Figure 2.** Distribution of salmon farm tenures (left) around Vancouver Island, British Columbia
661 and the migration of wild pink salmon from the Fraser River (right). Parasite dispersal connects
662 the population dynamics of disease amongst farms within a region as well as between farmed
663 salmon and the wild salmon populations that migrate through the aquaculture regions as
664 juveniles and as adults.
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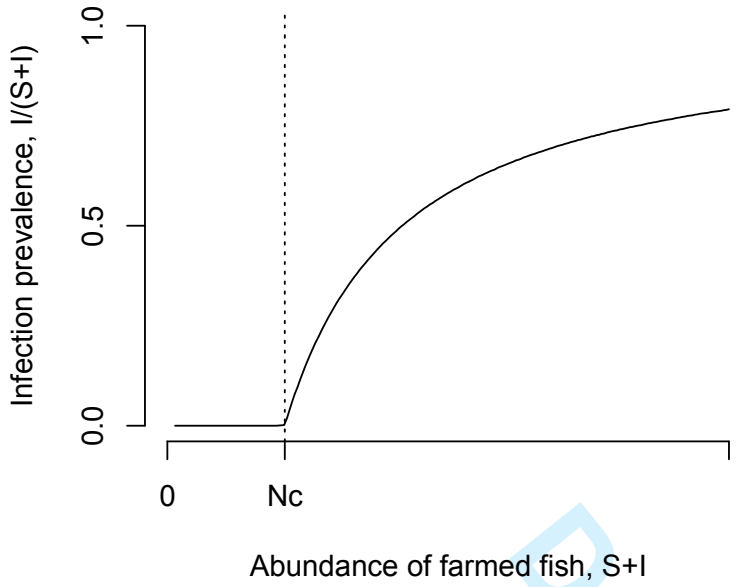
668 **Figure 3.**



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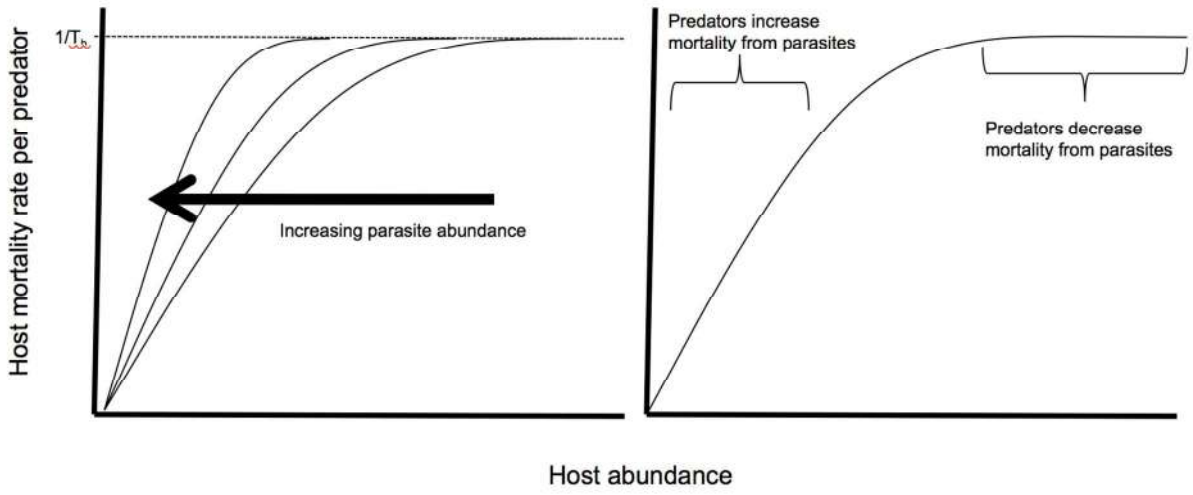
670 **Figure 3.** Equilibrium levels of infection prevalence as a function of farmed fish abundance
671 predicted by a susceptible-infected model. At abundances below the host-density threshold (*Nc*)
672 infection is at a low (non-zero) endemic state, maintained by immigration of parasites from the
673 surrounding environment. At abundances above the host density threshold, the dynamics switch
674 to epidemic conditions where infection can be sustained and spread within the farmed fish
675 population. Above *Nc*, equilibrium levels of infection sharply increase as a function of farmed
676 fish abundance and eventually saturate at 100%. The model: Farmed fish are categorized into
677 susceptible (*S*) and infected (*I*) classes. New fish are stocked into the production system at rate
678 ψ, infected individuals transmit the parasite to new hosts at rate β, and infection enters the
679 domesticated environment from an external (wild) reservoir host population at rate γ. The natural
680 mortality plus harvest rate of fish is μ and the rate of parasite induced host mortality is α. The *S-I*
681 dynamics can be characterized by the equations $\frac{dS}{dt} = \psi - \beta SI - \gamma S - \mu S$ and $\frac{dI}{dt} = \beta SI + \gamma S -$
682 $(\mu + \alpha)I$. The net reproductive value, R_0, which is the average number of secondary infections
683 in a completely susceptible population caused, on average, by an initial infection is $R_0 =$
684 $\beta N/(\mu + \alpha)$ where *N* is the size of the host population when all hosts are susceptible prior to
685 disease introduction. The critical host density threshold that divides endemic versus epizootic
686 dynamics is found by solving for *N* when $R_0 = 1$, which gives $N_c = (\mu + \alpha)/\beta$. The equilibrium
687 expressions for susceptible and infected hosts are given implicitly as $S^* = \psi/(\beta I^* + \gamma + \mu)$ and
688 $I^* = \gamma S^*/(\mu + \alpha - \beta S^*)$. Parameter values used for this plot are $\psi = 0.01$ to 1 (controls the
689 total fish abundance along the x-axis), $\beta = 0.001$, $\mu = 0.01$, $\alpha = 0.005$, and $\gamma = 0.0000001$.
690

691 Figure 4.



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693 **Figure 4.** Functional response of a predator when its attack rate is an increasing function of
694 parasite abundance (Krkošek et al. 2011). The functional response is given by $(\gamma + p\theta)N/[1 +$
695 $(\gamma + p\theta)T_hN]$ where γ is the base attack rate of a predator on prey in the absence of parasites, p
696 is the average abundance of parasites, θ is the per-parasite rate of increased attack rate of the
697 predator on the prey, T_h is the handling time, and N is the abundance of the prey population. In
698 the model, increasing levels of parasite abundance cause the functional response curve to rise
699 more quickly but do not change the saturation level of prey consumption. The result is that at low
700 prey abundance, predation indirectly intensifies mortality arising from parasites whereas at high
701 prey abundance predation rates are unaffected by parasite levels but instead become focused on
702 the infected portion of the prey population.
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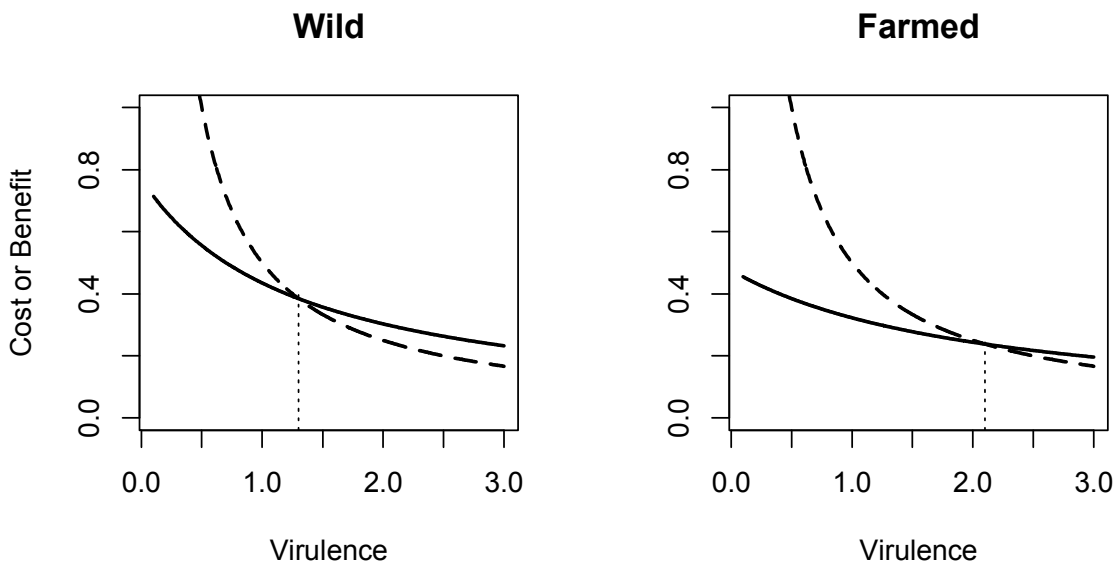
705 Figure 5



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707 **Figure 5.** Simple model of how virulence may change for a parasite in wild versus farmed fish
708 conditions. The model assumes that both transmission β and host mortality ψ are functions of
709 virulence α, where virulence is proportional to the rate of host-exploitation of the parasite
710 resulting in increased parasite propagules being produced (increasing β) but at the expense of
711 host mortality (increasing ψ). A measure of the fitness of the parasite is the net reproductive
712 value, R_0 (i.e., the number of secondary infections generated by a single infection in a
713 susceptible host population), and so $R_0 = [\beta(\alpha)/\psi(\alpha)]\, S$, where S is the abundance or density
714 of susceptible hosts. Optimizing parasite fitness over virulence (i.e. $dR_0/d\alpha = 0$) gives the
715 evolutionary stable strategy for the virulence trait when $(1/\beta)(d\beta/d\alpha) = (1/\psi)(d\psi/d\alpha)$
716 where the benefit to increased transmission of increasing virulence ($(1/\beta)(d\beta/d\alpha)$, dashed
717 lines) equals the cost to increased host mortality of increasing virulence ($(1/\psi)(d\psi/d\alpha)$, solid
718 lines). The benefit curve is hypothesized to be steeper and the cost curve shallower in
719 domesticated environments relative to wild environments due to the lack of migration, predation,
720 and food limitation in domesticated environments. Such shifts in the trade-off curves cause the
721 evolutionary equilibrium of the virulence trait to shift to the right (vertical dotted lines).
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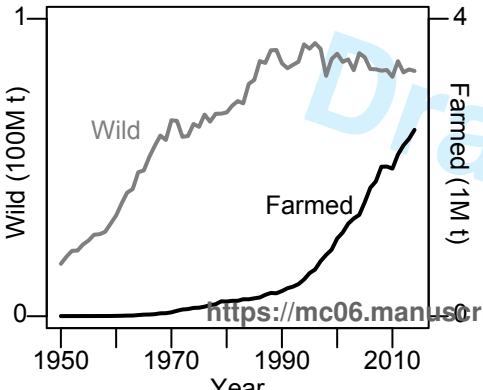
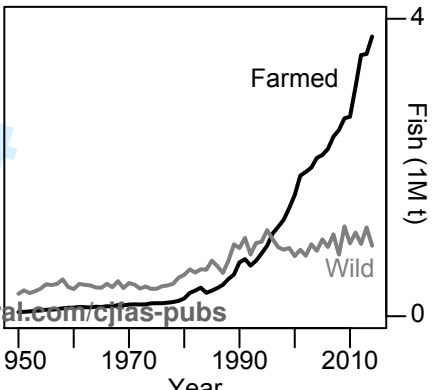
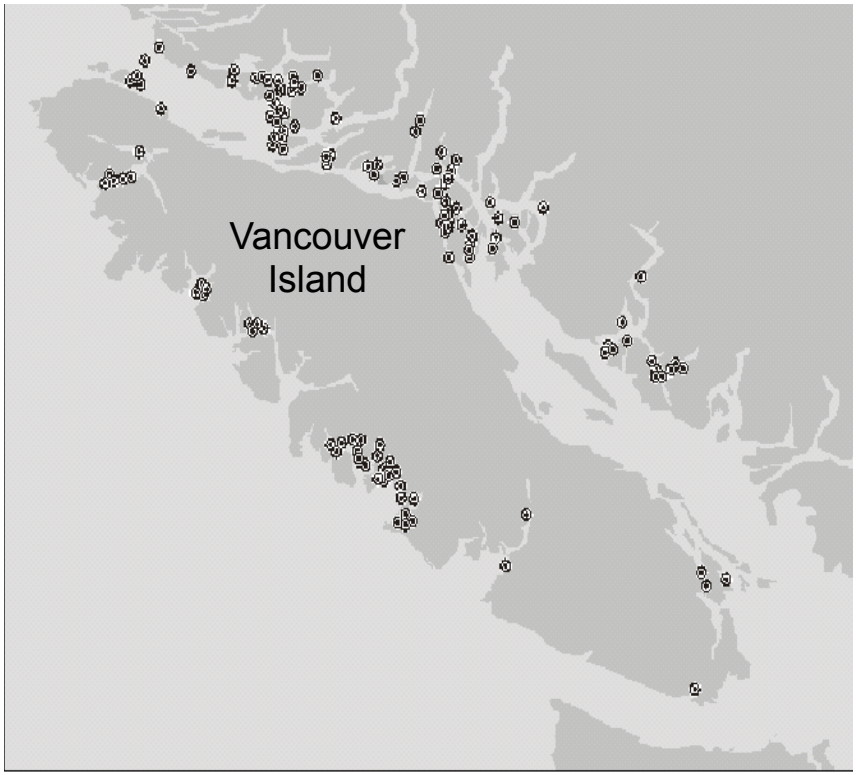
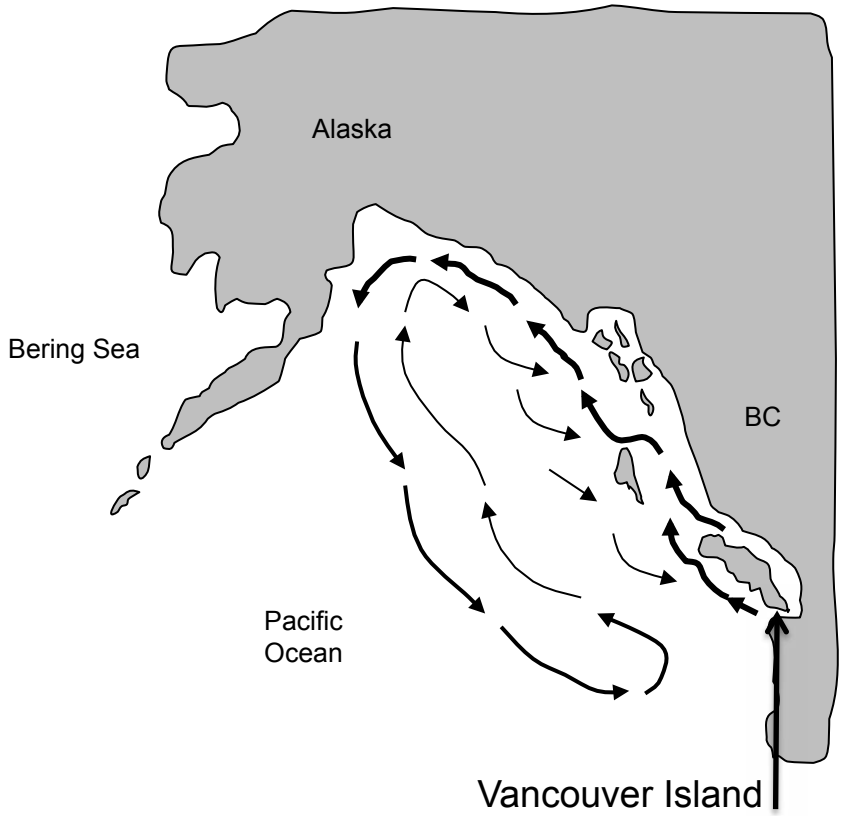





Vancouver
Island


Alaska
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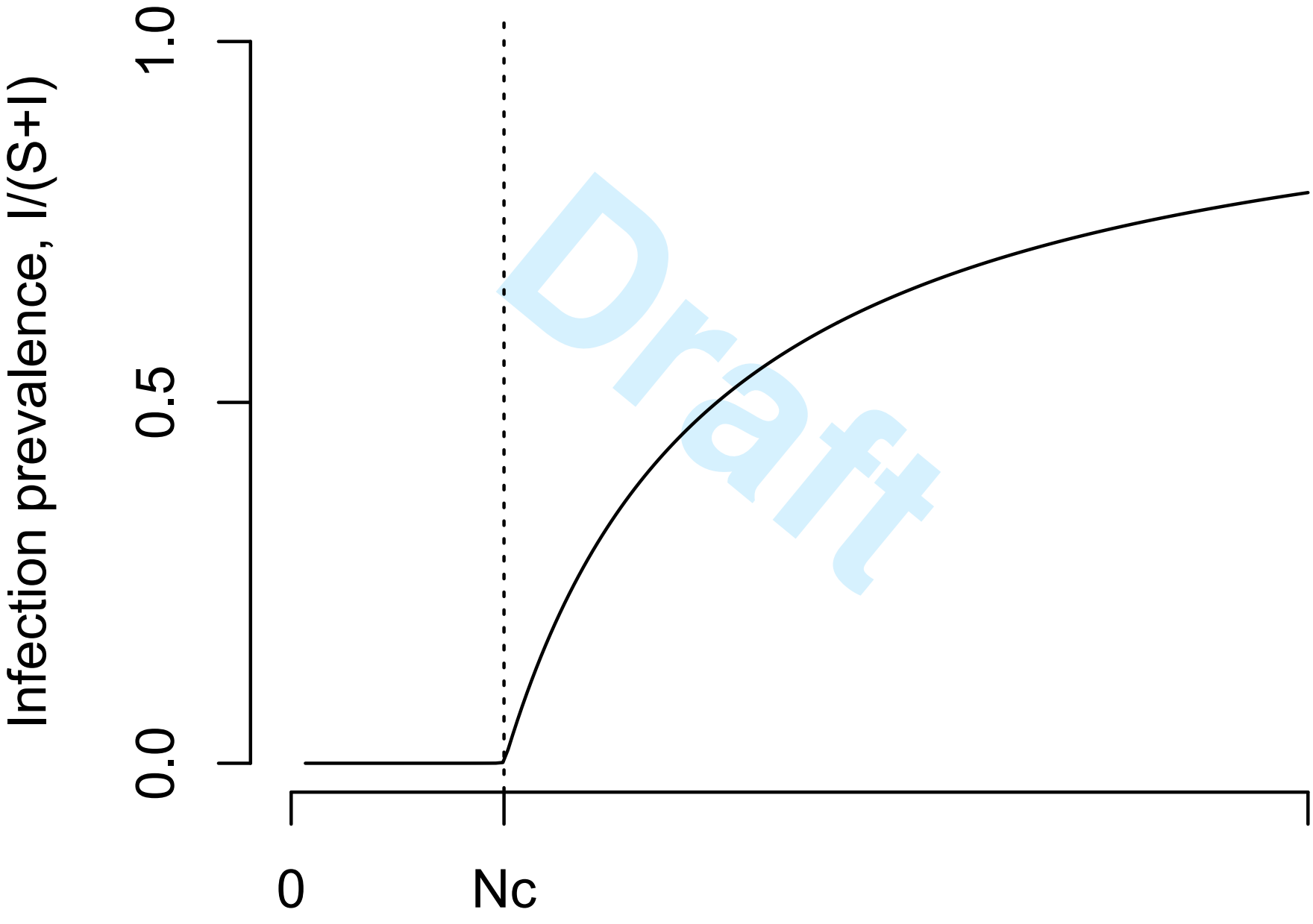
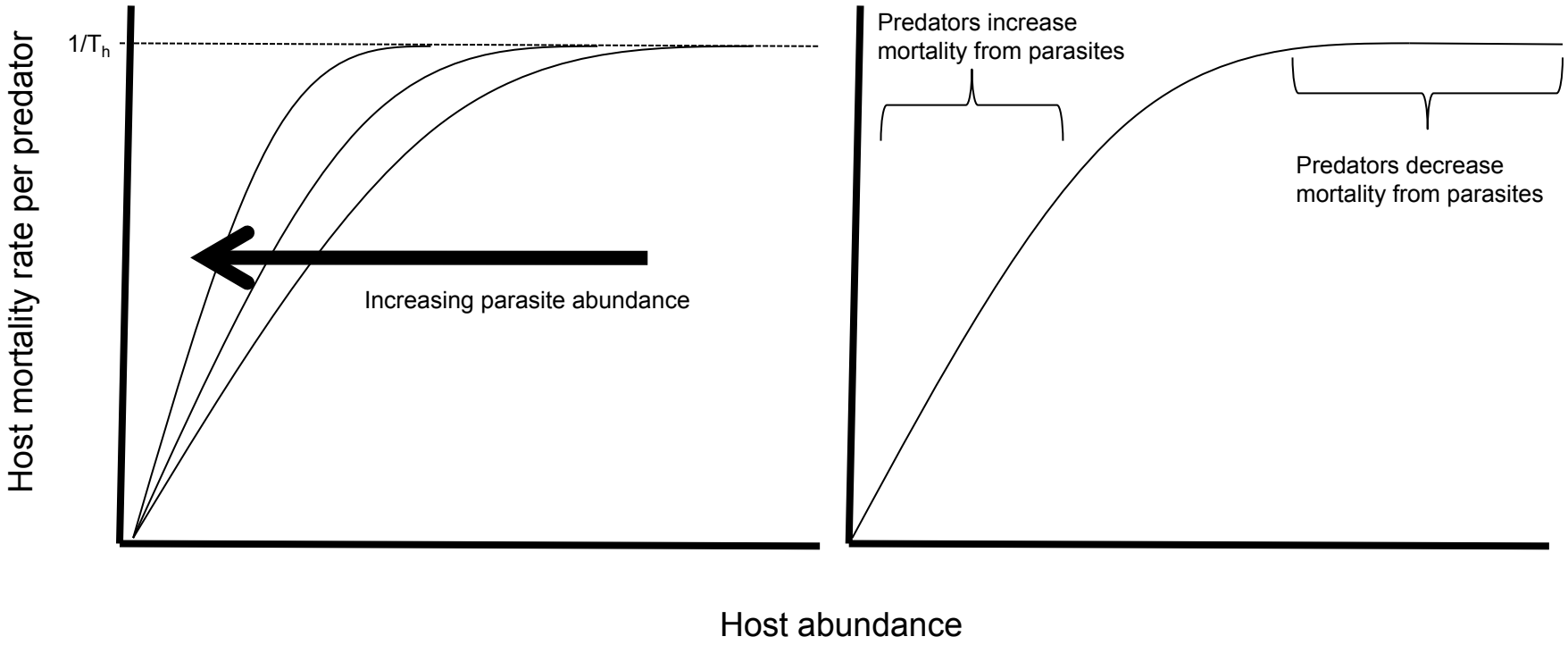
Vancouver Island


Infection prevalence, I/(S+I)
1.0
0.5
0.0
0
Nc
Abundance of farmed fish, S+I


Host mortality rate per predator
1/T_h
Increasing parasite abundance
Predators increase mortality from parasites
Predators decrease mortality from parasites
Host abundance