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REVIEW

Host density thresholds and disease control for fisheries and aquaculture

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ABSTRACT: The outbreak, persistence, and eradication of infectious diseases often depend on the density of hosts. In coastal seas, many fisheries are fully or over-exploited; meanwhile, farmed populations are increasing rapidly with aquaculture growth. Marine aquaculture facilities are typically open to the surrounding ecosystem and, therefore, wild and farmed populations are connected by their shared parasites. At the core of epidemiological theory are host density thresholds, above which diseases can persist or invade and below which diseases can be eradicated. Host density thresholds in aquaculture–fishery interactions likely function at regional scales that encompass multiple farms, which are connected by pathogen dispersal and the movement of wild hosts. Sudden outbreaks of parasitic copepods in wild-farmed salmon systems may be linked to aquaculture growth exceeding host density thresholds. Abiotic (e.g. temperature and salinity), management (e.g. husbandry and farm siting), and biotic factors (e.g. migrations of wild hosts) likely affect threshold values. A connected wild-farmed host population can exceed a host density threshold due to an influx of wild hosts via migration, increases in aquaculture production, or environmental change such as climate warming. Coastal management and policy should heed the disease implications of climate warming, aquaculture growth, and fisheries restoration that suggest increasing host densities and decreasing threshold values.

KEY WORDS: Epidemiology · Aquaculture · Fisheries · Conservation · Threshold · Disease

INTRODUCTION

The dynamics of infectious diseases are related to the density of host populations (Grenfell & Dobson 1995, Hudson et al. 2001). High densities of host populations can lead to increased contact rates among individuals or between hosts and a pathogen, resulting in increased transmission and persistence of disease (May & Anderson 1991, Hudson et al. 2001). Examples include host density as an explanation for patterns of parasite abundance in nature (Arneberg et al. 1998), the disease consequences of social organization in animal communities (Altizer et al. 2003), and the persistence and spread of measles among human populations (Keeling & Grenfell 1997). In contrast, at low host densities, the contact rate among susceptible host individuals or the rate of encounter between susceptible hosts and a pathogen can be lower, resulting in slower disease spread and disease eradication (May & Anderson 1991, Hudson et al. 2001). Such is the rationale for vaccination (e.g. Hampson et al. 2009) and culling (e.g. White et al. 1997) as strategies for disease control because they reduce the density of susceptible hosts.

There are cases where the dynamics of parasites are related to host densities. One example is the cyclical population dynamics of red grouse in Britain, which are at least partially controlled by the gastrointestinal nematode *Trichostrongylus tenuis* (Hudson et al. 1998). For grouse and *T. tenuis*, recruitment of adult worms is positively dependent on grouse density and grouse reproductive success is negatively related to parasite abundance (Hudson et al. 1992). Similar

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effects of parasite infection on host fecundity and of host density on parasite recruitment suggest the gastrointestinal nematode *Ostertagia gruehneri* may regulate a reindeer population in northern Norway (Albon et al. 2002). The population dynamics of measles in the UK is also an example of the effects of host population size, influx of susceptible hosts, and vaccination (Grenfell et al. 2001). Across many taxa, there is a positive relationship between the density of host populations and the average number of parasites per host (Arneberg et al. 1998).

HOST POPULATIONS AT SEA

In marine settings, fisheries reduce the abundance of fish host populations which should reduce parasite abundance (Dobson & May 1987), a process known as fishing out parasites. Indeed, fish communities in tropical reefs show reduced host density, reduced parasite abundance, and reduced parasite species richness in fished compared to protected areas (Lafferty et al. 2008). Trophic cascades, usually associated with depletion of top predators, include increases in the abundance of populations at lower trophic levels (e.g. Myers et al. 2007), with implications for disease outbreaks. In California, fisheries have depleted populations of predatory spiny lobsters, causing an increase in population density of their sea urchin prey, which, in turn, has caused recurrent *Vibrio* epidemics in urchin populations (Lafferty 2004). In the oceans, host density is also increased by the aggregated nature of marine populations, which may increase disease outbreak and spread relative to terrestrial systems (McCallum et al. 2003, 2004).

While fisheries have caused declines in the abundance of marine populations (Jackson et al. 2001, Myers & Worm 2003, Lotze et al. 2006), the growth of industrial aquaculture has caused an enormous increase in the abundance of domesticated marine populations (Goldburg & Naylor 2005, Duarte et al. 2007, FAO 2007). For example, populations of farmed salmon (usually Atlantic salmon, *Salmo salar*) are orders of magnitude larger than sympatric wild salmonid populations in salmon farming regions of Norway, Scotland, Ireland, New Brunswick, and British Columbia (Heuch & Mo 2001, Butler 2002, Krkošek 2009). In western North America, populations of the non-indigenous Pacific oyster *Crassostrea gigas* in aquaculture facilities dwarf the remnant and depleted populations of native Olympia oysters *Ostrea lurida*, the only native oyster in the region (McGraw 2009). For crustaceans, production of farmed prawns and shrimp has exploded and is poised to overtake global capture fisheries in the next few years (FAO 2007).

The transition from fisheries to aquaculture in the global production of seafood creates a major change in the abundance and distribution of marine host populations. This is particularly the case in coastal seas, where natural populations are often depleted (Jackson et al. 2001, Lotze et al. 2006) and aquaculture production is concentrated. There, aquaculture facilities typically occur in net pens, cages, rafts, or on ropes, all of which are open to the surrounding marine ecosystem. Wild and domestic marine populations are therefore connected by their shared parasites, which can be freely transmitted between the aquaculture environment and the surrounding ecosystem (Kent 2000, Krkošek et al. 2005). Such transmission between wildlife and domestic animal populations is termed spillover and spillback of parasites, and is a primary mechanism in the emergence of infectious diseases (Daszak et al. 2000). In this situation, wild populations are a natural reservoir for a large variety of pathogens which can spill over into aquaculture settings where they can be modified and/or amplified and then spill back into natural populations, with possible adverse affects on domesticated (Johnson et al. 2004, Murray & Peeler 2005) and natural populations (Krkošek et al. 2006, 2007a).

Disease control in aquaculture is a major challenge for the aquaculture industry (Johnson et al. 2004, Costello 2009b), and is a conservation concern for marine ecosystems and fisheries (Heuch et al. 2005, Krkošek et al. 2007a). However, not all coastal ecosystems that contain similar native populations and aquaculture facilities are affected by the same diseases or by the same dynamics of a disease. Epidemics may be isolated events, such as the 2001–2004 outbreak and subsequent eradication of infectious haematopoetic necrosis (IHN) virus in British Columbia salmon farms (Saksida 2006). Epidemics of native parasites may begin abruptly and then remain a chronic management challenge, such as the emergence of sea lice infestations in the Broughton Archipelago region of British Columbia (Morton & Williams 2003, Morton et al. 2004, Krkošek 2009) or the Passamaquoddy Bay region of the Bay of Fundy, New Brunswick (Hogans 1995). Spatially, there can be substantial variation in parasite abundance on wild and farmed fish populations (Tully et al. 1999, Heuch et al. 2003). While many factors such as veterinary practices or abiotic variation likely contribute to the observed variation in parasite outbreaks in aquaculture–fisheries interactions, the effects of host population density have not been carefully considered. The shifts in density and distribution of marine host populations due to fisheries and aquaculture have profound implications for disease dynamics, much as they do for humans and wildlife (May & Anderson 1991, Hudson et al. 2001).

HOST POPULATION THRESHOLDS

The density of host populations can affect transmission rates of a pathogen primarily because as host density increases so too does the rate at which susceptible hosts encounter an infectious individual or a parasite larva (May & Anderson 1991, Grenfell & Dobson 1995). This is because a pathogen, either in an infected individual or free-living in the environment, has a greater chance of encountering a new host individual if there are a greater number of hosts in the immediate environment. Such density-dependent transmission is common in host–parasite dynamics (May & Anderson 1991, Hudson et al. 2001), although there are exceptions (McCallum et al. 2001, Wonham et al. 2006, Smith et al. 2009). Density-independent transmission in terrestrial environments typically involves vector or sexually transmitted diseases; these are rarer in aquatic environments. However, it is important to keep in mind that the mechanism for density-dependent transmission is different than changes in host susceptibility due to decreased immuno-competence that may be associated with high host density due to, for example, increased stress or food shortages. Because the transmission rate of a pathogen can depend on host density, the density of hosts can have important consequences for the outbreak, persistence, or eradication of a disease (May & Anderson 1991, Grenfell & Dobson 1995).

The most common measure of disease persistence or eradication, and indeed, the most fundamental quantity in epidemiology, is known as R_0, the net reproductive value (May & Anderson 1991, Reno 1998, Hudson et al. 2001, Heesterbeek 2002). The value of R_0 is: (1) the number of secondary infections generated by a single infected individual in a susceptible host population for microparasites such as viruses, bacteria, and protists; or (2) the number of adult parasites generated in the lifetime of a single parasite for macroparasites such as arthropods or helminthes (Hudson et al. 2001). In both cases, if $R_0 > 1$, then the parasite population can invade the host population, leading to disease outbreak and persistence. In contrast, if $R_0 < 1$, then infections do not on average replace themselves and the parasite population will eventually die out, leading to disease eradication. To see how R_0 depends on the density of the host population, we first need a quantitative description of the population dynamics of the host–parasite system. Such models depend on whether the pathogen is a microparasite or a macroparasite, which differ fundamentally in the life cycle of the pathogen and how the distribution of the pathogen among hosts is characterized (May & Anderson 1991, Grenfell & Dobson 1995).

Host density thresholds for microparasites

Microprasites are primarily viruses, bacteria, and protists, which typically replicate within an individual host, spread by host-to-host contact, and do not have an obligate free-living stage in the life cycle (May & Anderson 1991), although aquatic microparasites may also be transported by water movement (Gustafson et al. 2007, Viljugrein et al. 2009). The dynamics of a microparasitic disease can be modeled by dividing the host population into classes according to their infection status, such as susceptible, infected, or recovered (SIR) (May & Anderson 1991, Grenfell & Dobson 1995). Variations of this SIR model framework have been used to characterize disease dynamics and evaluate health policy. Examples include measles (Grenfell et al. 2001) and smallpox (Ferguson et al. 2003) in human populations, *Renibacterium salmoninarum* in wild Chinook salmon *Oncorhynchus tshawytscha* populations (Fenichel et al. 2009), and, generally, aquatic animal populations, including aquaculture–fishery interactions (Murray 2009). To determine the host density threshold, separating disease outbreak and disease eradication conditions for microparasites, we need a quantitative description for the dynamics of the parasite in the host population — typically an SIR model.

Following the structure of a classic microparasite model (Anderson & May 1979), the basic form of an SIR model with density-dependent transmission is:

$$\begin{aligned} \frac{\mathrm{d}S}{\mathrm{d}t} &= bN + \nu R - dS - \beta SI \\ \frac{\mathrm{d}I}{\mathrm{d}t} &= \beta SI - (d + \alpha + \gamma)I \\ \frac{\mathrm{d}R}{\mathrm{d}t} &= \gamma I - (d + \nu)R \end{aligned} \quad (1)$$

where the host population size is N, which is divided into susceptible S, infected I, and recovered R classes ($N = S + I + R$). The host population has birth rate b and death rate d that are independent of infection status. Susceptible individuals become infected by their interaction with infected individuals, the rate of which is tuned by the transmission coefficient β. Once infected, hosts can die due to disease at rate α or recover at rate γ. Once recovered, hosts no longer die of the disease but they also lose immunity over time and therefore transition back into the susceptible class at rate ν.

To calculate the net reproductive value for a host–microparasite system governed by Eq. (1), we first can make some simplifying approximations according to the conditions under which R_0 is calculated. First of all, because R_0 is equal to the number of secondary infections generated by a single infected individual in a completely susceptible population, we let $S = N$, $I = 1$, and $R = 0$. Further, because the duration of a single infection is usually short compared to the time scale of

a host lifespan, we assume that natural birth and death rates of the host population have a negligible effect on host population size during the course of a single infection and therefore set $b = d = 0$; this simplification, for example, was used by Murray et al. (2001) to model short-lived epidemics of Australian pilchard herpesvirus. This means that the net reproductive value, R_0, can be calculated from the simplified model:

$$\begin{aligned} \frac{dS}{dt} &= -\beta SI \\ \frac{dI}{dt} &= \beta SI - (\alpha + \gamma)I \end{aligned} \tag{2}$$

known as a simple S-I model, even though the overall disease dynamics are determined by Eq. (1). With these approximations, and recalling that $S = N$ and $I = 1$, the net reproductive value for the disease is modelled by Eq. (2) as:

$$R_0 = \beta N \frac{1}{\alpha + \gamma} \tag{3}$$

Interpreting Eq. (3) is straightforward. The term $1/(\alpha + \gamma)$ is the average duration of the infectious period of an individual host, whereas the term βN is the rate at which hosts become infected. Thus, multiplication of these 2 terms equals the number of infected individuals generated by a single infected individual in the host population. Note that the net reproductive value depends on the size of the host population, which can be interpreted as host density if we consider a particular reference area such as an aquaculture net pen, an embayment with wild and farmed shellfish, or a fjord with wild and farmed salmon. The host density threshold separating disease outbreak and disease eradication conditions is found by setting $R_0 = 1$ and solving for N, giving:

$$N_c = \frac{\alpha + \gamma}{\beta} \tag{4}$$

According to this framework, a microparasite will invade a host population with density exceeding the critical population size N_c and will be eradicated from a host population with host density below N_c. While this SIR modeling framework may be simplistic, it is useful to capture the basic principles of microparasite disease transmission and to characterize the general dynamics, such as host density thresholds. Further, it forms a basis from which more detailed models can be constructed and applied to specific systems.

Host density thresholds for macroparasites

Macroparasites are primarily helminthes and arthropods, such as intestinal worms and ectoparasitic copepods (Hudson et al. 2001, Costello 2006). The key difference separating macroparasites from microparasites, aside from body size, is that the life cycle of a macroparasite has an obligate free-living larval stage (Anderson & May 1978). That is, in order to complete its life cycle, an individual parasite must leave the host in which it was born, survive a developmental progression in the outside environment and ultimately encounter another host individual, invade that individual, and reproduce. Furthermore, reproduction in macroparasites is often sexual, meaning that macroparasites have the added challenge of locating a mate inside or on the host individual in order to complete the life cycle, whereas reproduction in microparasites is primarily asexual. Well-known examples of macroparasite life cycles are intestinal nematodes whose eggs are distributed in the outside environment in the host feces and must be encountered and consumed by another host individual before development and reproduction can be completed. Another familiar example for aquatic animals is parasitic copepods, sea lice (*Lepeophtheirus* spp. and *Caligus* spp.), which reproduce sexually on the surface tissues of host fish, have free-swimming nauplii that hatch from egg strings on parasitic females, and which develop through non-feeding larval stages before they must attach to a new host fish or die (Kabata 1988, Costello 2006).

These differences require a different modeling framework for macroparasites that tracks the dynamics of free-living stages as well as the number of parasites per host in the host population (May & Anderson 1991, Grenfell & Dobson 1995). Following the structure of the classic host–macroparasite model formulation (May & Anderson 1979), we write:

$$\begin{aligned} \frac{dN}{dt} &= (b - d)N - \alpha P \\ \frac{dP}{dt} &= \beta LN - (d - \mu_P - \alpha)P - \frac{\alpha(k+1)P^2}{kN} \\ \frac{dL}{dt} &= \lambda P - \mu_L L - \beta LN \end{aligned} \tag{5}$$

where N is the size of the host population, P is the total parasite population size (not the average number of parasites per host), and L is the free-living larval population size. The host population has birth rate b and death rate d. Larvae, which have natural death rate μ_L, encounter and infect a host at a rate determined by the transmission coefficient β after which they are in the parasitic stage which dies at rate μ_P. Parasites also die when their host dies and impose a per-parasite host mortality rate α. The average number of free-living larvae produced per parasite is λ. The quantity k comes from the negative binomial distribution, which accounts for the aggregated nature of parasites on the host population (Shaw & Dobson 1995, Shaw et al. 1998).

If we model the average number of parasites per host $\bar{P}$ as opposed to the total parasite population size, the model (Eq. 5) becomes:

$$\frac{dN}{dt} = (b-d)N - \alpha\bar{P}N$$
$$\frac{d\bar{P}}{dt} = \beta LN - (b+\mu_P+\alpha)\bar{P} - \frac{\alpha}{k}\bar{P}^2 \quad (6)$$
$$\frac{dL}{dt} = \lambda N\bar{P} - \mu_L L - \beta LN$$

From here, a more intuitive model can be obtained if we assume parasites are Poisson distributed over the host population for which we let $k \to \infty$ and gives $\frac{d\bar{P}}{dt} = \beta LN - (b+\mu_P+\alpha)\bar{P}$ for the parasite population dynamics. However, parasites tend to be aggregated rather than randomly distributed, necessitating the common practice of using the negative binomial formulation similar to Eq. (5) (Grenfell & Dobson 1995).

Nevertheless, for all of these macroparasite models, the calculation of R_0 is the same because the average number of parasites produced during the lifetime of an individual parasite in an uninfected population is the same. Similar to microparasites, we assume the lifespan of parasites is short compared to hosts, and therefore host birth and death would have negligible effects on host population size and parasite population size over a time span of an individual parasite. The net reproductive value for a macroparasite governed by Eq. (6) is given by:

$$R_0 = \left(\frac{\lambda}{\mu_P+\alpha}\right)\left(\frac{\beta N}{\mu_L+\beta N}\right) \quad (7)$$

The first term in Eq. (7) is the total number of larvae produced by a parasite — the rate of larvae production λ multiplied by the lifespan of a reproductive stage parasite $1/(\mu_P + \alpha)$. The second term in the equation is the probability a larva will infect a host. Note that Eq. (7) is an increasing function of host population size. The critical host population size (N_c) separating parasite population persistence and eradication is given by setting $R_0 = 1$ and solving for N to give:

$$N_c = \frac{\beta}{\mu_L}\left(\frac{\mu_P+\alpha}{\lambda-\mu_P-\alpha}\right) \quad (8)$$

A macroparasite will invade a host population or persist if it is already present if host density exceeds N_c and will be eradicated from a host population with host density below N_c. Similar to the caveats for SIR models, this host–parasite modeling approach may be simplistic, but it is useful to characterize the dynamics of the system, such as host density thresholds, and forms a basis from which more detailed models can be developed for specific systems.

EMPIRICAL EVIDENCE FOR THRESHOLDS

While theory predicts that host density thresholds may be an important part of host–parasite dynamics, clear empirical examples are rare (Lloyd-Smith et al. 2005). Only in well-understood and data-rich systems such as measles in human populations is there compelling empirical evidence (Keeling & Grenfell 1997, Bjørnstad et al. 2002, Grenfell et al. 2002). There is a general positive relationship between host density and parasite abundance among species (Arneberg et al. 1998), but examples of abrupt outbreaks associated with host density thresholds for specific host–parasite systems is a separate matter. The rarity of clear empirical examples is not evidence that host density thresholds do not exist; rather, statistical difficulties arise with confounding factors or inadequate data (Lloyd-Smith et al. 2005). Indeed, the theory of host density thresholds forms the basis of controlling and eradicating disease in wildlife and domesticated animals (Grenfell & Dobson 1995, Hudson et al. 2001). For example, fertility control may be a form of culling that could eradicate bovine tuberculosis from badger *Meles meles* populations in the UK (White et al. 1997).

For aquaculture–fisheries interactions, there are at least 2 putative empirical examples of sudden outbreaks of salmon lice *Lepeophtheirus salmonis* from endemic to epidemic levels that might be linked to host density thresholds. The first example occurred in the Passamaquoddy Bay region in the Bay of Fundy, New Brunswick, in 1993–1994 (Hogans 1995). There, average sea lice abundances on farmed salmon remained at <20% prevalence with intensities of <5 lice per fish during 1988 to 1993. During the winter of 1993–1994, sea lice levels suddenly increased to reach prevalences approaching 100% and intensities of 25 lice per fish. The outbreak was associated with increased morbidity and mortality of farmed salmon, reduced market value of surviving fish, and a need for veterinary interventions to combat sea lice populations. Sea lice control remains an important management consideration in the Bay of Fundy, where attention is focused on the reliance of management on the parasiticide, emamectin benzoate, and the risk of sea lice evolving resistance to the chemical (Westcott et al. 2004).

The second putative example of sea lice outbreaks from endemic to epidemic conditions occurred in the Broughton Archipelago region of British Columbia, where reports of sea lice epidemics on wild juvenile salmon began in 2001 (Morton & Williams 2003). Data from non-farming areas indicate endemic levels of lice are ~5%, which jumped to epidemic levels of over 75% in 2001 and 2002 (Morton & Williams 2003, Morton et al. 2004), spurring management changes of farms, such as fallowing and chemical treatment, to

reduce lice (Morton et al. 2005, Krkošek 2009). Epidemics continued through 2005, and were associated with high mortality (Morton & Routledge 2005, Krkošek et al. 2006) and decline of local wild pink salmon populations (Krkošek et al. 2007a). There are recent changes in management, focused on the collective management of farms to either fallow or treat with emamectin benzoate during the juvenile salmon outmigration period. There are no data from wild fish preceding 2001 nor are there corresponding data available from aquaculture companies with which to evaluate the suddenness of the epidemics. However, because the outbreaks on wild juvenile salmon are highly conspicuous and the region is populated with fishermen, biologists, and First Nations peoples, it is unlikely that epidemics would have occurred prior to 2001 without notice.

For sea lice and salmon in Passamaquoddy Bay and the Broughton Archipelago, the epidemics appear to be marked by a sudden — rather than gradual — transition from endemic low levels of the parasite to epidemic levels where continuous surveillance and management of sea lice populations on farmed salmon is required. One characteristic that is common to both cases is that the outbreaks occurred amidst periods when aquaculture production was growing with slow incremental steps (Ford & Myers 2008). That is, the sudden increase in lice was not associated with a sudden increase in farmed salmon production but rather small incremental changes. These patterns are consistent with those predicted by theory, in which a small increase in host density that crosses the critical host density threshold, N_c, triggers a sudden outbreak of disease. It is further possible that changes in environmental conditions, such as temperature, may have decreased N_c, thereby accelerating the transition from endemic to epidemic dynamics. These patterns are suggestive of host density thresholds in sea lice and salmon population dynamics, but further data and detailed analysis are needed to detect and estimate N_c.

Other examples of host density thresholds in application are vaccination programs. Because vaccination reduces the density of susceptible hosts, the effective host population size for a pathogen is reduced, which can lead to eradication if the density of susceptible hosts remains below the host density threshold sufficiently long for the disease to fade out. Specifically, the fraction of the population that must be vaccinated, or for which the vaccine is effective, to eradicate a pathogen is $\nu = 1 - 1/R_0$ (Hudson et al. 2001). This concept is known as herd immunity (Anderson & May 1985), where a pathogen can be eliminated from a population because a sufficient proportion, but not all, of the host population has acquired immunity (through natural processes or vaccination) such that $N < N_c$ and $R_0 < 1$. Vaccination can also be effective in controlling density-independent transmission by reducing the proportion of infectious contacts with susceptible hosts. These considerations underlie the design and motivation for vaccination strategies for many diseases such as rabies in African domestic dogs (Hampson et al. 2009), the endangered Ethiopian wolf (Haydon et al. 2006), or the red fox in Europe (Suppo et al. 2000), or bovine tuberculosis from possum populations in New Zealand (Tompkins et al. 2009).

Vaccination has contributed to disease control in aquaculture (Sommerset et al. 2005). For example, despite a large increase in salmon farming production, furunculosis epidemics in Europe that persisted for a decade have been eradicated by vaccination (Johnsen & Jensen 1994, Lillehaug et al. 2003, Sommerset et al. 2005). Similarly, the development of a vaccine against *Vibrio* spp. in Norwegian salmon farms has curtailed outbreaks of vibriosis that required heavy antibiotic use and challenged the productivity of the industry (Lillehaug et al. 2003, Sommerset et al. 2005). However, vaccines can be imperfect: sub-clinical infections may persist — such as furnculosis in Atlantic salmon (Skugor et al. 2009) — or some fraction of the host population may become diseased. Imperfect vaccination has implications for virulence evolution (Gandon et al. 2001) and indicates host density thresholds may still affect disease outbreaks, with threshold values that are higher than those in the absence of vaccination (Hudson et al. 2001). In aquaculture, most vaccines that exist are for viral and bacterial diseases (Sommerset et al. 2005). Vaccines for parasitic disease, particularly sea lice, are an important research area but have not yet been successfully developed and implemented (Raynard et al. 2002, Boxaspen 2006, Alvarez-Pellitero 2008)

SPATIAL SCALE OF THRESHOLDS AT SEA

Domesticated marine populations exist at nested spatial scales, ranging from a net pen, raft, cage, or rope, to a farm or a collection of farms in an embayment or region. Furthermore, there may be mobile wild host populations in the surrounding environment that vary in abundance through time and space, and which can connect distant aquaculture facilities (Uglem et al. 2009). The spatial scale which defines the relevant host population upon which host density thresholds might act is likely determined by the disease connectivity within and among these host subpopulation units. This, in turn, depends on physical characteristics influencing the dispersion and survival of free-living infectious agents — wind, currents, salinity, and temperature, for example, for sea lice (Murray

& Gillibrand 2006, Amundrud & Murray 2009, Foreman et al. 2009). For these reasons, it may be difficult to generalize the spatial scale at which host density thresholds act. Rather, it is likely that this will vary on a case-by-case basis, based on physical features, biological vectors, and the species of hosts and pathogens.

However, it is likely that, in general, the spatial scale of host density thresholds is larger in the oceans than it is on land. In the marine environment, pathogens are likely more long-lived and more widely dispersed then they are on land (McCallum et al. 2004). Physical oceanographic processes can transport infectious agents over long distances and a marine environment may provide pathogens more stable physical conditions of temperature and moisture (McCallum et al. 2004). Furthermore, wild marine host populations tend to consist of host aggregates (such as a shoal of fish) which are highly mobile or migratory, contributing to the spatial spread of infection (McCallum et al. 2003). These characteristics of the marine environment likely underlie the observation that diseases spread at rates an order of magnitude faster in the oceans than they do on land (McCallum et al. 2003). These characteristics, in addition to connectivity of farms by wild fish movement (Uglem et al. 2009), suggest that the spatial scale for host density thresholds in the oceans is likely larger than an individual farm and likely occur at regional scales such as large embayments, fjords, or archipelagos.

For sea lice, dispersive free-living stages (nauplii and copepedites) can persist for several days up to a few weeks, depending on temperature (Boxaspen 2006). During that time, sea lice larvae can be widely dispersed, depending on oceanographic conditions (Murray & Gillibrand 2006, Amundrud & Murray 2009, Foreman et al. 2009). Important factors influencing dispersal include the physical forcing of wind and river discharge on currents, as well as vertical diel migrations of sea lice, which can expose the parasite to currents that reverse in direction as depth increases (Gillibrand & Willis 2007, Foreman et al. 2009). Models from fjordic habitats in Europe and Canada indicate sea lice can be dispersed on the order of 10 to 100 km from their source location before becoming infectious (Murray & Gillibrand 2006, Amundrud & Murray 2009, Foreman et al. 2009). Those predictions are supported by empirical studies of the planktonic stages as well as the young parasitic stages on wild hosts in the surrounding environment (Krkošek et al. 2005, Costello 2006). The high dispersion of sea lice larvae indicates that parasite populations on domesticated fish can have high connectivity among farms. They also suggest that only a small proportion of sea lice hatched at a particular farm may remain at that location to reinfect the same stock. This partially underlies the development of management where farms are collectively managed at regional scales, so-called coordinated area management plans, where stocking, harvesting, and chemical treatment are coordinated to minimize transmission among farms and between wild and farmed fish.

Although microparasites tend to be more short-lived, they can also be transported considerable distances by seawater movement, for example, spread between salmon farms of infectious salmon anemia (Gustafson et al. 2007) and pancreas disease (Viljugrein et al. 2009) outbreaks follow the directions of currents predicted using hydrodynamic modeling. Infection pressure, however, likely declines rapidly with distance from source population. In addition, microparasites may spread among farms over broad spatial scales due to the movement of wild fish populations (Uglem et al. 2009). For example, herpes epidemics of Australian pilchard stocks in 1995 and 1998–1999 spread at rates of 30 km d^{-1} or 10 000 km yr^{-1} (Jones et al. 1997, Gaughan et al. 2000, McCallum et al. 2003). Outbreaks of viral diseases in farmed fish can similarly occur at large spatial scales. For example, the IHN epidemic in salmon farms along the British Columbia coast in 2001–2003 occurred over a region of hundreds of kilometers (Saksida 2006). The outbreak of infectious salmon anemia in Chilean salmon farms has occurred at yet larger scales and has decimated industry production by 75% (Asche et al. 2009, Mardones et al. 2009). Both dispersal among farms and industry practices such as fish transport likely led to the spread of these outbreaks (Saksida 2006, Mardones et al. 2009).

PREDICTABILITY AND VARIATION OF THRESHOLDS

Host density thresholds in disease dynamics may be difficult to detect and predict (Lloyd-Smith et al. 2005). Detection is limited by the lack of baseline data before diseases emerge, because confounding factors make inference difficult or because disease dynamics are complicated by alternate host species. Because many parameters in parasite life cycles are influenced by environmental factors, environmental variation can cause R_0 and N_c to vary. This creates the intriguing possibility that a host population can cross a host density threshold when host density has not changed or is even in decline if R_0 increases substantially due to environmental effects on life cycle parameters. Demographic rates of parasite life cycles are often dependent on temperature, moisture, or salinity, creating the possibility that environmental variation can have a large influence on disease outbreaks (Pascual et al. 2000, Koelle et al. 2005). Disease dynamics in oysters,

for example, are particularly sensitive to temperature and salinity (Burreson & Calvo 1996, Soniat et al. 2009). Global climate change may thus have profound implications for disease dynamics due to influences on pathogen demographic rates (Harvell et al. 1999, Harvell et al. 2002). However, other diseases that are associated with cold water, such as winter ulcer disease and cold-water vibriosis in farmed salmon, may be reduced (Nordmo & Ramstad 1999, Colquhoun & Sorum 2001, Tunsjø et al. 2007).

Climate warming may have important implications for host–parasite dynamics in aquaculture–fisheries interactions, such as sea lice (Costello 2006), because developmental and fecundity rates are highly dependent on temperature (Stien et al. 2005, Boxaspen 2006). The net effect is a decrease in life cycle duration, an increase in R_0, and a decrease in N_c. Shorter-term, seasonal variation in abiotic factors may have a comparatively large influence on host density thresholds. Natural seasonal variation in temperature and salinity in coastal marine ecosystems can span a huge range of parameter values in demographic rates for sea lice (Johnson & Albright 1991, Stien et al. 2005, Bricknell et al. 2006), potentially generating high amplitude seasonal variations in R_0 and N_c. Overlaid on this seasonal environmental variation in abiotic factors can be seasonal variation in host abundance due to host migration (Krkošek et al. 2007b). For example, migrations of wild Pacific salmon between freshwater and offshore marine ecosystems cause the density of hosts for salmon lice in coastal ecosystems to vary seasonally by several orders magnitude between winter when few hosts are present, spring when large populations of juvenile salmon are present, summer when large populations of adult and juvenile salmon populations are present, and fall when adult populations are present and moving into freshwater to spawn (Groot & Margolis 1991, Quinn 2005).

It is easy to see how models that are more complex than those presented earlier in this article are needed to detail the disease interactions of fisheries and aquaculture. Such complexities are more likely the norm rather than the exception, due to the ubiquity of abiotic variation in coastal marine ecosystems and the migratory or highly dispersed life cycles of marine taxa. Nevertheless, this does not imply that host density does not affect disease dynamics. The influence of host density on disease dynamics lies at the core of epidemiological theory and practice for human and terrestrial animals, which themselves are not without complexity (May & Anderson 1991, Hudson et al. 2001). It can be helpful to turn to stochastic models of disease dynamics, which reveal that environmental stochasticity may cause host–density thresholds to appear more gradual rather than as abrupt transitions implied by the deterministic models (Lloyd-Smith et al. 2005). A central challenge to scientists is to distill from the complexities the essential processes that drive disease dynamics in aquaculture–fishery interactions into quantitative models where disease thresholds can be investigated. Such endeavours must address the challenges of uncertainty arising from environmental variation and uncertainty in parameter values. Thus, while quantitative prediction of thresholds may be difficult, it may still be possible to evaluate if and how such thresholds may arise.

THRESHOLDS, MANAGEMENT, AND POLICY

The concept that host density thresholds can separate eradication and outbreak conditions of an infectious disease has important implications for coastal planning. The critical host population density of a wild-farmed fish system may be maximized by minimizing pathogen transmission among farms and between wild and farmed hosts. This can be accomplished by situating farms distant from wild fish migration routes and in locations where ocean tides and currents connecting farms are minimized but flushing rates of farms are high. Furthermore, aquaculture tenures may be situated strategically so that natural variation in abiotic factors such as temperature and salinity minimize the survival rates of pathogens and/or the environmental stress that farmed fish populations experience, which thereby minimizes pathogen survival and therefore reduces R_0 and increases N_c. Such planning requires knowledge of coastal hydrodynamics, wild fish behaviour, and the environmental stress responses of farmed fish before the locations of farms are determined.

There are also management practices that may contribute to reducing R_0 and increasing N_c. These include breeding programs that select fish for resistance to diseases, which would act to reduce pathogen survival, reduce R_0, and increase N_c. Management of aquaculture vessel traffic and water circulation of live-tanks on vessels could be conducted with the view of minimizing cross-contamination among farms. Fish health programs such as vaccination or the application of chemical therapeutants can have similar effects. However, it is important to note that the evolution of resistance in pathogens to chemical therapeutants — such as for sea lice and emamectin benzoate — can undermine the sustainability of this management strategy. One means of minimizing this risk is by maintaining farmed fish density below N_c such that the application of chemotherapeutants is minimized and therefore selection for resistance is minimized. Indeed, careful planning and management may reduce N below N_c and relieve the reliance of management on chemotherapeutants.

CONCLUSIONS

Coastal seas have experienced major declines in the abundance of some species due to fisheries (Jackson et al. 2001, Lotze et al. 2006). Meanwhile, there are large increases in the abundance of other species due to trophic cascades associated with fisheries (Shears & Babcock 2003, Myers et al. 2007), as well as the rapid growth of marine aquaculture (Goldburg & Naylor 2005, Duarte et al. 2007). These shifts in the abundance and distribution of marine species have important implications for the dynamics of infectious disease, owing to the influence of host density on the outbreak, persistence, and eradication of disease (Lloyd-Smith et al. 2005). Reduction of host abundance due to fisheries may reduce the abundance and diversity of parasite species (Dobson & May 1987, Lafferty et al. 2008). Increases in host abundance due to fishery-induced trophic cascades may increase the rate of disease outbreaks (Lafferty 2004). Increases in the abundance of domestic species may contribute to the emergence of marine infectious disease in aquaculture (Murray & Peeler 2005, Mardones et al. 2009), and can change transmission dynamics of native parasites (Krkošek et al. 2007b, Krkošek 2009), with adverse effects on aquaculture (Johnson et al. 2004, Costello 2009b) and ecosystems (Krkošek et al. 2007a, Costello 2009a).

Diseases rates in the oceans are rising, though not for all taxa, and the causes are likely multi-factorial (Harvell et al. 2004, Lafferty et al. 2004, Ward & Lafferty 2004). Meanwhile, aquaculture is growing rapidly (Goldburg and Naylor 2005, FAO 2007) and fisheries are fully exploited, overexploited, or rebuilding (Pauly et al. 2002, Pauly et al. 2003, Worm et al. 2009). These trends indicate that disease interactions between wild and farmed fish are likely to intensify (Daszak et al. 2000), with disease outbreaks that can be triggered by changes in host density (May & Anderson 1991, Grenfell & Dobson 1995). However, such thresholds in host population density may be unpredictable (Lloyd-Smith et al. 2005). The development of vaccines can increase threshold values (Hudson et al. 2001), thereby relieving some challenges of host density and disease on aquaculture production. The spatial scale at which host density thresholds act is likely to be variable among pathogen species and among localities within pathogen species due to the complexities of ocean currents, abiotic factors, and wild hosts. However, the spatial scale of host population thresholds is not an individual farm but rather a collection of farms in a coastal unit such as an embayement, fjord, or archipelago. While host density thresholds may be difficult to predict, scientists and managers should appreciate that they likely exist and may not be detectable until after host densities exceed critical threshold levels, which may occur due to changes in host density or via changes in the threshold itself due to environmental change.

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