

Sea-Cage Aquaculture, Sea Lice, and Declines of Wild Fish

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Abstract: *A sea cage, sometimes referred to as a net pen, is an enclosure designed to prevent farm fish from escaping and to protect them from large predators, while allowing a free flow of water through the cage to carry away waste. Farm fish thus share water with wild fish, which enables transmission of parasites, such as sea lice, from wild to farm and farm to wild fishes. Sea lice epidemics, together with recently documented population-level declines of wild salmon in areas of sea-cage farming, are a reminder that sea-cage aquaculture is fundamentally different from terrestrial animal culture. The difference is that sea cages protect farm fish from the usual pathogen-control mechanisms of nature, such as predators, but not from the pathogens themselves. A sea cage thus becomes an unintended pathogen factory. Basic physical theory explains why sea-cage aquaculture causes sea lice on sympatric wild fish to increase and why increased lice burdens cause wild fish to decline, with extirpation as a real possibility. Theory is important to this issue because slow declines of wild fish can be difficult to detect amid large fluctuations from other causes. The important theoretical concepts are equilibrium, host-density effect, reservoir-host effect, and critical stocking level of farmed fish (stocking level at which lice proliferate on farm fish even if wild fish are not present to infect them). I explored these concepts and their implications without mathematics through examples from salmon farming. I also considered whether the lice-control techniques used by sea-cage farmers (medication and shortened grow-out times) are capable of protecting wild fish. Elementary probability showed that $W \approx W^* - \epsilon F$ (where W is the abundance of wild fish, W^* is the prefarm abundance, F is the abundance of farm fish, and ϵ is the ratio of lice per farm fish to lice per wild fish). Declines of wild fish can be reduced by short growing cycles for farm fish, medicating farm fish, and keeping farm stocking levels low. Declines can be avoided only by ensuring that wild fish do not share water with farmed fish, either by locating sea cages very far from wild fish or through the use of closed-containment aquaculture systems. These principles are likely to govern any aquaculture system where cage-protected farm hosts and sympatric wild hosts have a common parasite with a direct life cycle.*

Keywords: aquaculture, fish parasite, host density effect, reservoir-host effect, sea lice, wild fish

Acuicultura en Jaulas Marinas, Piojos de Mar y Declinaciones de Peces Silvestres

Resumen: *Una jaula marina, a veces conocida como corral de red, es un encierro diseñado para prevenir que los peces de granja escapen y para protegerlos de depredadores mayores, al mismo tiempo que permite el libre flujo de agua para arrastrar desechos. Los peces de granja, por lo tanto, comparten agua con peces silvestres, lo cual permite la transmisión de parásitos, como los piojos marinos, de peces silvestres a peces de granja y viceversa. Las epidemias de piojos marinos, aunadas a declinaciones poblacionales de salmón silvestre documentadas recientemente en áreas de cultivo en jaulas marinas, son un recordatorio de que la acuicultura en jaulas marinas es fundamentalmente diferente a la crianza de animales terrestres. La diferencia estriba en que las jaulas marinas protegen a los peces de granja de los mecanismos usuales de control de patógenos de la naturaleza, como los depredadores, pero no de los patógenos mismos. Por lo tanto, una jaula marina se convierte en un centro de cultivo de patógenos. La teoría física básica explica porqué la acuicultura en jaula marina causa el incremento de piojos marinos en peces silvestres simpátricos y porqué*

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las mayores cargas de piojos causan la declinación de peces silvestres, con la posibilidad real de extirpaciones. La teoría es importante en este tema porque las declinaciones lentas de peces silvestres pueden ser difíciles de detectar entre grandes fluctuaciones debidas a otras causas. Los conceptos teóricos importantes son equilibrio, efecto de la densidad de huéspedes, efecto del reservorio de huéspedes y el nivel crítico de siembra de peces de granja (nivel de siembra en el que los piojos proliferan en peces de granja aunque no haya presencia de peces silvestres que los infecten). Exploré estos conceptos y sus implicaciones sin matemáticas sino por medio de ejemplos del cultivo de salmón. También consideré si las técnicas de control de piojos utilizadas en las jaulas marinas (medicación y acortamiento de tiempo de engorde) son capaces de proteger a peces silvestres. La probabilidad elemental mostró que $W \approx W^* - \varepsilon F$ (donde W^* es la abundancia pregranja de peces silvestres y ε es la proporción de piojos por pez de granja a piojos por pez silvestre). Las declinaciones de peces silvestres se pueden reducir con ciclos de crecimiento cortos para peces de granja, la medicación de peces de granja y el mantenimiento de niveles bajos de siembra de peces de granja, ya se mediante la ubicación de jaulas marinas muy lejos de los peces silvestres o mediante el uso de sistemas de acuicultura con contenedores cerrados. Es probable que estos principios rijan cualquier sistema de acuicultura en el que huéspedes protegidos en jaulas y huéspedes silvestres simpátricos tienen un parásito común con un ciclo de vida directo.

Palabras Clave: acuicultura, densidad de huéspedes, efecto huésped reservorio, parásito de peces, peces silvestres, piojos de mar

Introduction

Sea cages have an understandable appeal to business, to those concerned that wild stocks are overfished, and to governments hoping to make nutritious seafood more affordable. Nevertheless, there is a growing amount of data that show wild fish can decline—sometimes to near extinction—in areas where the density of sea cages is high (Krkošek et al. 2007a; Ford & Myers 2008). Often the decline is associated with infestations of sea lice (Copepoda). In Scotland (Butler & Watt 2003), Norway (Holst et al. 2003; Heuch et al. 2005), and western Ireland (Gargan et al. 2003), stocks of wild salmon and sea trout (*Salmo* spp.) have declined in areas with sea cages compared with areas without them. On Canada's Pacific coast, infection of wild juvenile salmon by sea lice from farm salmon (Morton et al. 2004; Krkošek et al. 2006) has caused similar, population-level declines (Krkošek et al. 2007a). The salmon louse *Lepeophtheirus salmonis* is an obligate parasite of salmonids; hence, sympatric fishes that are not salmonids cannot be the cause of *L. salmonis* epidemics on wild salmon. Sea lice epidemics are frequent in the sea-cage culture of many other fishes (Ho & Lin 2004), with presumably similar effects on sympatric wild fish.

Population-level changes in the abundance of wild animals are difficult to detect (Hilborn & Mangel 1997), and the difficulty is especially pronounced for migratory fishes such as salmon (Peterman et al. 2000). In some areas of sea-cage farming, declines of wild fish were not noticed until years after farming had begun, which has led to the hope that sea-cage farming is harmless to wild fish (e.g., Beamish et al. 2006). Moreover, 2 wild species may decline at different rates if one is more susceptible than the other or one is a predator of the other. In this situation, it is necessary to use basic principles to try to

understand the problem. Such principles cannot predict exactly what will happen in any given situation because there are too many unknowns, but they can elucidate what is likely to happen. I applied basic physics to the exchange of sea lice between wild and farm fish, avoiding nonessential mathematics wherever possible. My results showed strong theoretical support for population-level declines of sympatric wild fish in areas of sea-cage aquaculture if the wild fish and farm fish have a common parasite with a direct life cycle.

Predators Large and Small

Salmon in the wild are subject to predation by a multitude of micropredators (e.g., bacteria, viruses, parasites) and a few large predators (macropredators), such as sharks, seals, sea lions, and orcas (*Orcinus orca*). Both categories of predator serve to regulate salmon populations. When an animal is preyed on by micropredators, it is referred to as a host rather than prey. Experts speak of host-parasite systems and prey-predator systems, and this nomenclature reminds us that in the first case the predator is tiny and seldom kills its prey immediately, whereas in the second case the predator is comparable in size to the prey and usually kills the prey during capture. Many parasites regulate their prey (Tompkins et al. 2002; Moller 2005) (i.e., limit the prey population).

Large predators usually prefer vulnerable prey (Moore 2002), and parasitized fish are often solitary or found on the periphery of a shoal where they are more vulnerable (e.g., Ward et al. 2002). Capture of infected prey by large predators has a regulatory effect on micropredators (Packer et al. 2003) because they are consumed along with the prey, or if they escape, they may die without

finding another host. The exceptions to this rule are parasites with life cycles requiring multiple hosts. For them, being eaten is part of the plan. Parasites, such as sea lice, that require only one host are said to have a direct life cycle.

A sea cage excludes large predators but not micropredators. Water flows freely through the mesh of the cage, carrying micropredators in and out. Thus, in a sea cage the regulatory effect of large predators on micropredators is prevented. Moreover, because farm fish are fed, the self-regulating effect of micropredators—a cycle in which reduced host fitness due to infection reduces host foraging efficiency, increasing host mortality and thus micropredator mortality—is avoided. Accordingly, disease is one of the greatest problems in sea-cage aquaculture. Atlantic salmon (*Salmo salar*, the most-farmed species) have presented over 200 known infections, most of which are infrequent or rare in wild Atlantic salmon (Bakke & Harris 1998). Sea lice are an important example of such an infection. Sea-lice-associated costs are 20% of revenue in salmon farming (MacKinnon (1997), and those losses exceed US\$100 million annually (Johnson et al. 2004).

Sea Lice

Sea lice are parasitic copepods that graze on the surface of fish (e.g., Boxshall & Defaye 1993; Pike & Wadsworth 1999; Boxaspen 2006; Costello 2006). Lice consume the mucus layer of the skin, the skin itself, and tissues beneath the skin. External layers of mucus and skin are important, both as barriers to infection and as part of the osmotic system that controls the salinity of internal tissues. For example, when salmon begin their life cycle in fresh water, their skin works to prevent fresh water from entering tissues, and after they enter the ocean it works to prevent fresh water from leaving tissues. Lice-infected juvenile salmon sacrifice foraging opportunities to return to fresh water (Webster et al. 2007), where lice cannot survive. Punctures and lesions created by feeding sea lice compromise the osmotic system and lower the fitness of the host (Bjørn & Findstad 1997). Healing of wounds created by sea lice requires metabolic resources from the host, and wounds provide a pathway into the host for bacteria and viruses. When newly infected with sea lice, juvenile salmon leap and roll, increasing their visibility to predators (Grimnes & Jakobsen 1996; Webster et al. 2007).

Most species of sea lice have similar life cycles, and *L. salmonis* (colloquially, lep) is an important example (Johnson & Albright 1991; Stien et al. 2005). Leps have 10 life stages. Adult lice meet and mate on the host, and the female louse generates up to 11 clutches of 200–800 eggs in paired strings (Heuch et al. 2000). The eggs hatch

into planktonic nauplii, which do not feed, are incapable of swimming or attaching to a host, and drift near the surface. After drifting in the ocean for 3–5 days, the nauplii transform into copepodids, which also do not feed but can propel themselves toward a close-passing host and attach to it. If a copepodid does not find a host within about 5 days, it dies. About 50% of nauplii survive to become copepodids (Johnson & Albright 1991). If it captures a host, the copepodid transforms into a chalimus stage, attached to the host with a small filament around which it grazes. Eventually the chalimus becomes a preadult, which can move around on the host to feed, and after several more transformations it becomes an adult, which can mate. In modeling infection data on pink salmon (*Oncorhynchus gorbuscha*) fry, Krkosek et al. (2005) found that 10–20% of the copepodids that capture a host survive to the adult stage. (Lice on those unusually small hosts likely interfere with each other, so 25% survival may be more accurate.) Male lice leave females after mating, to seek other females, and females produce several clutches of eggs during their adult life. A complete life cycle takes about 2 months, depending on factors such as temperature and salinity. Under optimal conditions it can be as short as a month, and at low temperature it can stretch to over 6 months (Heuch et al. 2000). Leps can survive for a time on hosts other than salmonids, but have been observed to reproduce only on salmonids. Leps and other sea lice cannot survive in fresh water for more than a few weeks.

The key to sea lice physics is the large number of larvae that a female sea louse must release to ensure a new generation of lice. The exact number is not important, but for concreteness, let us say that a female lep succeeds if she produces 1600 or more live nauplii. Assuming equal numbers of males and females, only 2 of those 1600 nauplii will eventually succeed. Therefore, to maintain the louse population, each nauplius must have a 1/800 chance of eventually succeeding. From the survival percentages, it follows that $1/800 = p_n p_c^e p_s$, where $p_n \approx 1/2$ is the probability that a nauplius survives to the copepodid stage, $p_c^e \approx 1/100$ is the equilibrium probability that a copepodid will capture a host, and $p_s \approx 1/4$ is the probability that a copepodid that has captured a host survives to adulthood and successfully reproduces.

The important facts about sea lice are (1) sea lice steal metabolic resources from the host, damage the host's osmotic system, provide a pathway for secondary infections, and increase a host's risk of being eaten by large predators, (2) a successful female sea louse produces about 1600 nauplii, and (3) copepodids drift in currents and can migrate only by varying their depth. From point 1, it follows that sea lice increase, however slightly, the mortality rate of their host. From point 2, it follows that only 1% of copepodids must capture a host to maintain the lice population. From points 2 and 3, it follows that

capture of a host by a copepodid has a large element of luck.

The Host-Density Effect

Consider a large area of ocean that contains wild fish, no farm fish, and a drifting copepodid. Depending on the currents and the habits of the fish, the copepodid may at some time have a fish pass near enough for it to attach. The important thing is that its chances of capturing a fish go up if there are more fish and down if there are fewer fish. In other words, no matter what the environmental variables might be, or the spatial distribution of fish, its chance of attaching to a fish is roughly proportional to the total number of fish. This is called the host-density effect.

After a few days, the copepodid's energy stores are exhausted. If fish are so few that its chances of attaching before it dies are $<1\%$, the next generation will be smaller than the present generation. On the other hand, if the fish are so numerous that its chances of attaching are $>1\%$, the next generation will be larger than the present one. Thus, with a small number of fish, sea lice will gradually die out, whereas with a large number of fish, sea lice will increase. The host-density effect is well known from studies of direct-life-cycle parasites in mammals (Arneberg et al. 1998; Arneberg 2001; Arneberg et al. 2002), where parasite abundance, prevalence, and community richness all increase with host density. There is no reason to suppose that fish are exempt from this effect (Frazer 2007).

But sea lice have been in existence for a very long time without dying out or filling up the ocean. What prevents either of those things from happening? The answer lies in the regulatory effect of sea lice under natural conditions. Sea lice injure their hosts, and although the injury is usually slight, it does reduce the chance that a wild fish will survive, especially if the wild fish is a juvenile. If lice become very numerous, wild fish suffer higher mortality rates and their numbers decline. Conversely, if lice become scarce, wild fish enjoy lower mortality rates and their numbers increase. Population levels of lice and fish fluctuate, but neither one of them grows without bound. In biology as in physics, this situation leads to equilibrium. Natural systems are never quite at equilibrium because of the time lag between input and response variables. Nevertheless, it is still helpful to remember that there is an equilibrium and that the system is more likely to be moving toward that equilibrium rather than away from it. Experts use definitions of *equilibrium* general enough to include environmental and demographic stochasticity, but no harm is done if one thinks of equilibrium values of fish, lice, and larvae as the values to which they would tend if environmental variables had the same seasonal pattern every year.

Epidemics in Farm Fish—Critical Stocking Level

Suppose there are no wild fish present, only farm fish in cages, a copepodid carried by currents into a cage would likely attach to a host. If the copepodid is not carried into a cage, it dies. If there are not many fish in the cages and its chance of attaching to a host is just 0.5% instead of the 1% needed to maintain the population, then each generation will be half as large as the one before it. Because a generation requires about 2 months, after 6 months, the number of copepodids will decline to $1/8$ ($1/2 \times 1/2 \times 1/2$) of its original level. On the other hand, if there are so many farm fish in the cages that the chance of attachment is 2% , then after 6 months, the number of copepodids will increase to 8 times ($2 \times 2 \times 2$) their original level.

This exponential growth behavior shows that sea cages and lice by themselves are an unstable system. If the number of farm fish is over a certain level (i.e., the critical stocking level), lice increase exponentially, but if the number of farm fish is under that level, lice decline exponentially. This unknown critical level depends on currents, temperature, salinity, harvest rates, treatment rates (the frequency at which farmers medicate their fish for lice), and many other variables that are difficult to estimate. The only way of knowing the critical level has been reached is that an epidemic of sea lice occurs. If the stocking level of farm fish is subcritical and then temperature and salinity suddenly move into the optimal range for lice, the critical level can drop below the actual stocking level, causing an epidemic.

Wild Fish and Farm Fish Together

In a model system of wild fish and sea lice, there is an equilibrium to which the system tends to return when perturbed. In the real world, this equilibrium is a moving target because of exogenous variables such as climate, so populations of fish and lice are constantly trying to catch up with their changing equilibrium values. To understand the interaction of wild and farm fish, I assumed these exogenous variables are constant.

Imagine an area of ocean containing wild fish, sea lice, and seals that eat fish. Imagine the system is close to equilibrium, so each copepodid drifting in the water has a 1% chance of attaching to a fish. Occasionally, a lot of copepodids get lucky at the same time, and lice numbers increase. Seals then find those infected fish easier to catch, so the number of fish decline. Copepodids then have less luck finding a host, so lice numbers decline toward their original level where each copepodid has a 1% chance of finding a host. Although these fluctuations are interesting, the goal is only to track the equilibrium point.

Now put a sea cage into the area, with a few farm fish in it. Currents carry larvae into the cage, increasing their chance of finding a host (host-density effect), and lice numbers rise. The farm fish are protected from the seals by their cage, so their number stays the same, even though they have more lice on them. Nevertheless, as the wild fish become infected with lice, the seals find them easier to catch, so wild fish decline. How far do they decline? Recall that the equilibrium point is the point at which each copepodid has a 1% chance of capturing a host; therefore, the wild fish decline until that is again the case. If the circulation in the area is such that copepodids have equal exposure to farm fish and wild fish and if farm fish are not medicated to kill their lice, wild fish decline by an amount equal to the number of farm fish.

What effect does the grow-out time (time fish are in the cage) have on sea lice? If farmers harvest their fish and replace them with young fish at about the same rate the wild fish die and are replaced, then from a copepodid's point of view, a farm fish is much like a wild fish. If farmers leave their fish in the cage for only a fraction of the life cycle of wild fish, then the copepodids that capture those farm fish are less likely to reach adulthood. Thus, lice levels rise less and wild fish decline less. Sea-cage farmers of coho salmon (*O. kisutch*) in Japan use short grow-out times to help control lice (Nagasawa 2004).

Up to this point, I have considered a sea cage that held only a few farm fish. What if the cage is filled with farm fish or more sea cages are added? Remember, there is a critical stocking level of farm fish. If the stocking level is above the critical level, lice continue increasing, and wild fish continue declining—toward extinction. That last wild fish, covered with sea lice, is easily caught by seals. Of course, in real situations farmers do not allow lice levels on their fish to grow without bound, so wild fish may or may not go extinct; it depends on how many farm fish are present. If enough farm fish are present, then even low levels of lice on farm fish can extinguish local wild fish.

What about spatial effects? If wild fish and seals keep to one area of ocean so far from farm fish that only a small fraction of larvae reach it, then the decline of wild fish due to farm fish is much reduced. Nevertheless, if the farm system is supercritical, wild fish can still be extinguished because a small fraction of a very large number of copepodids is still a large number of copepodids. Finally, note that the seals in my thought experiment are just a proxy mechanism for the reduction in survival due to infection. For example, as the somatic fitness of wild fish is reduced by infection, their foraging efficiency is reduced, so their mortality increases. This is not the case for farm fish because they are fed. In this more general sense, the important thing about sea-cage farming is not so much that farm fish are protected from predators but that they are protected from the increased mortality due to infection.

The Reservoir-Host Effect

Consider 2 areas of ocean (with no farm fish) so distant from each other that there is little exchange of water between the areas. Call them area A (for adults) and area B (for birth). Suppose adult wild fish spend most of their time in area A, but every autumn a fraction of them migrate to area B for a brief period to mate and spawn. The eggs in area B incubate for about 6 months during which time there are no hosts present, so lice larvae decline. In the spring, wild juvenile fish emerge into waters free of larvae, where they forage and grow, and then they slowly migrate back toward area A to join the adults. Area B functions as a refuge for juveniles from the parasites of adults. Krkošek et al. (2007b) refer to this life history as migratory allopatry to distinguish it from migrations in which all cohorts move together. Pacific salmon typify migratory allopatry, with the offshore zone as area A and the coastal zone as area B. Every autumn a fraction of the adult salmon migrate from the offshore to the coast, where they spawn in their natal streams and then die.

The reservoir-host effect is triggered when salmon sea cages are located on coasts with stocks of wild salmon (Fig. 1). When adult wild salmon migrate from the offshore to their natal rivers, they pass the sea cages and infect the farm salmon. The farm salmon provide a reservoir host for lice over the winter when wild salmon are absent. In the spring, wild juveniles are infected by farm salmon as they pass the farms during their out-migration. Many of them die because the effects of sea lice on mortality are inversely proportional to body mass—lice on an adult fish increase the chance of death only slightly, whereas a few lice on a juvenile fish make its death nearly certain (Bjørn & Finstad 1997; Morton & Routledge 2005; Krkošek et al. 2006). Wild pink and chum (*O. keta*) salmon are especially vulnerable because they enter salt water soon after hatching, when they weigh about 0.2 g and do not have scales. M. Krkošek and coworkers (2005, 2006, 2007a, 2007b) analyze the reservoir-host effect in detail for pink salmon in the Broughton Archipelago of Pacific Canada.

Note that the reservoir-host effect depends on the host-density effect and on equilibrium and criticality. When adult wild salmon pass the farms during their autumn in-migration, host density near the farms rises steeply, and lice proliferate. During the winter lice on farm salmon decline if the farm stocking level is subcritical, but if the stocking level is close to critical, lice decline only a little. If the critical level drops below the actual stocking level (perhaps from an increase in salinity), lice on the farm salmon increase rapidly over the winter. In the spring when juvenile wild salmon exit their natal streams, host density rises again, and lice proliferate.

Virtually all observations of sea lice exchange between wild and farmed salmon are well explained by the reservoir-host effect. In the salmon farming areas of Norway, Scotland, and western Ireland, sea trout (*S. trutta*),

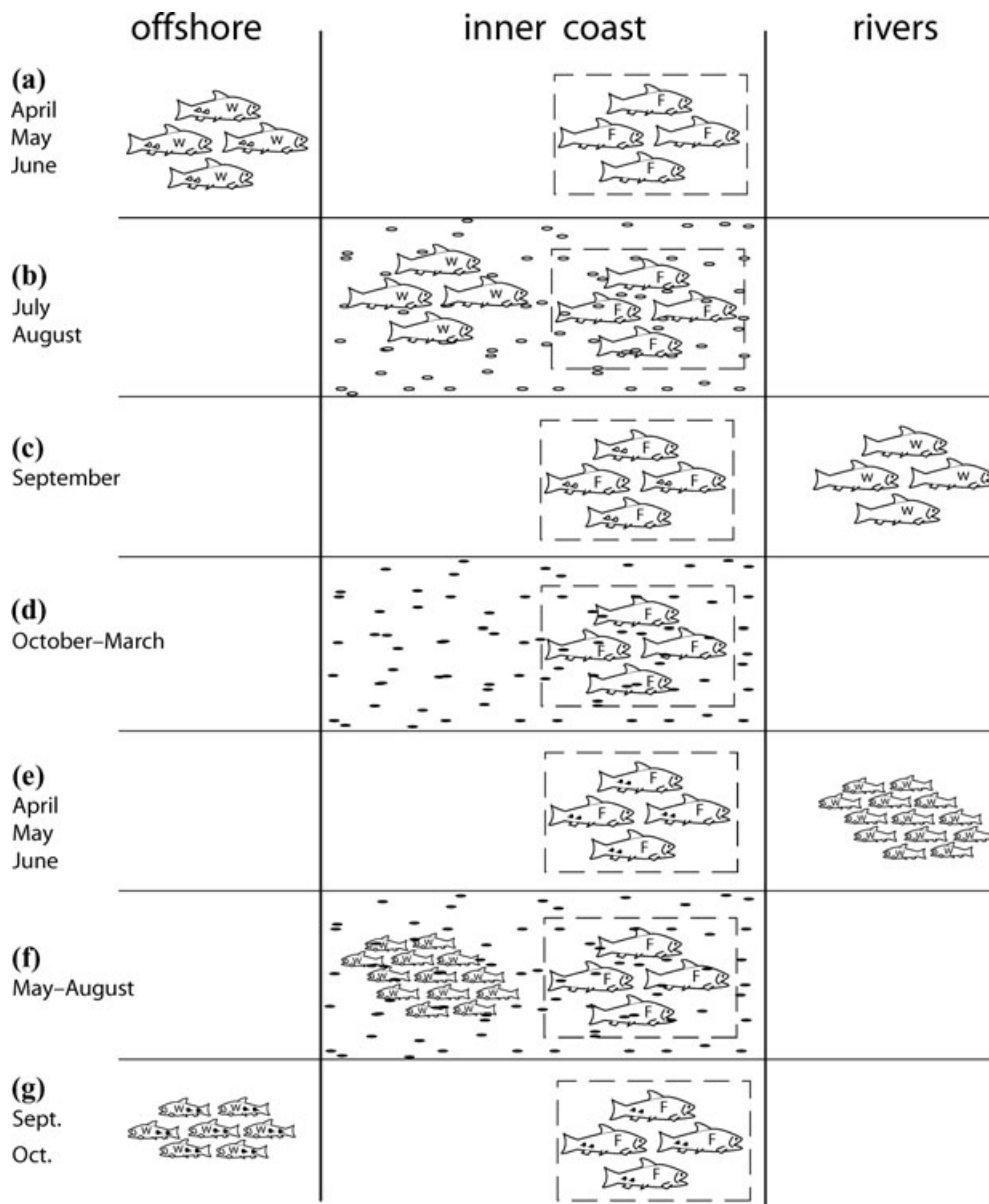


Figure 1. The reservoir-host effect for sea lice in sea-cage salmon farming (modified from Frazer 2007). Time intervals in the left column are for pink salmon in Pacific Canada. Wild-origin lice and larvae are drawn open, and farm-origin lice and larvae are solid. In (a) spring, adult wild salmon (W) with lice are in the offshore zone, and in (b) summer, larvae from in-migrating wild adults disperse in the coastal zone, where they infect farm hosts. By (c) fall, farm salmon (F) have lice, and wild adults have entered the rivers where their lice die. Pacific salmon die shortly after spawning. In (d) winter, larvae from lice on farm salmon reinfest farm salmon, while the eggs of wild salmon incubate in streams. As rivers freeze, increased salinity of coastal waters can cause the critical stocking level to drop below the actual stocking level (supercriticality). In (e) early spring, wild juveniles emerge from the gravels of their natal streams. Pink and chum salmon leave rivers immediately after emergence, weighing less than 0.5 g. In (f) spring and summer, larvae from farm salmon infect juvenile wild salmon as they pass the farms and forage in coastal areas. In (g) fall, juvenile wild salmon migrate offshore in numbers reduced by infection and predation. Out-migrating wild juveniles encounter in-migrating wild adults near the end of July.

whose marine phase is entirely in coastal waters, are declining more rapidly than Atlantic salmon (*S. salar*), which migrate rapidly past farms to the offshore. Wild Atlantic salmon are not declining in areas distant from

farms (reviews cited earlier). Juvenile pink salmon near salmon farms in Pacific Canada have over 8 times more lice than those sampled distant from farms (Morton et al. 2004), and lice-infection pressure on juvenile salmon near

a salmon farm is over 70 times greater than infection pressure distant from farms (Krkošek et al. 2005), which is likely an underestimate (Frazer 2008). Temporarily reducing farm stocking levels benefits local pink salmon (Morton et al. 2005), and a repeat of that experiment is needed to avoid extirpation of wild stocks (Krkošek et al. 2007a).

Medication

When lice infestations become severe, sea-cage operators medicate their fish. For example, salmon farmers in Canada mix chemicals such as emamectin benzoate with their salmon feed. Such chemicals are costly, and their toxicity is not limited to sea lice, so farmers medicate only when lice on their fish exceed an abundance threshold specified by regulations. What threshold is necessary to protect wild fish? To address this question, I considered the mathematics of the host-density effect. The type of calculation needed is usually done with differential equations (Anderson & May 1978), but a probability calculation suffices to find the equilibrium point.

The method is based on the fact that a larva's probability of success (i.e., completing its life cycle by capturing a host and reproducing) is the probability it succeeds on a wild host plus the probability it succeeds on a farm host (because these are mutually exclusive possibilities), and that, at equilibrium, the sum of these probabilities must equal a larva's prefarm probability of success. The equation for equilibrium is therefore

$$p_n p_{cw} p_{s|w} + p_n p_{cf} p_{s|f} = p_n p_c^e p_{s|w}. \quad (1)$$

The right-hand side is the prefarm equilibrium probability that a newly hatched larva eventually succeeds. On the left-hand side, the first addend is the probability that the larva succeeds on a wild host, and the second addend is the probability that it succeeds on a farm host. Elements on the right-hand side are defined earlier. Because the development from nauplius to copepodid occurs in the water, it does not depend on host type; hence, p_n is the same in each term and cancels out of the equation. On the left-hand side, $p_{s|f}$ is the probability of success of a copepodid that has captured a farm host. In general, this will be different from $p_{s|w}$ because farmers can shorten the grow-out time or medicate their fish to kill lice. The quantity $\varepsilon = p_{s|f}/p_{s|w}$ is the ratio of gravid female lice per farm fish to gravid female lice per wild fish, an observable factor. The equilibrium equation becomes

$$p_{cw} + \varepsilon p_{cf} = p_c^e, \quad (2)$$

where p_{cw} is the probability that a new copepodid eventually attaches to a wild host, and p_{cf} is the probability that it attaches to a farm host.

In Eq. 2 the point of view is that of a single copepodid, and the key quantity is its probability of attaching to a host. Attachment probability vanishes if no hosts are present, and it approaches unity if the number of hosts tends to infinity. Hence I took it to be $H/(H_{0.5} + H)$, in which H is the number of hosts in the region of ocean under study and $H_{0.5}$ is the host level at which the capture probability is 0.5. When both host types are present, the probability of capturing a wild host is the probability of capture, $(W + F)/(H_{0.5} + W + F)$, times the fraction of hosts that are wild, $W/(W + F)$, hence $p_{cw} = W/(H_{0.5} + W + F)$. Similar reasoning gives $p_{cf} = F/(H_{0.5} + W + F)$, and the equilibrium equation becomes

$$\frac{(W + \varepsilon F)}{(H_{0.5} + W + F)} = p_c^e. \quad (3)$$

I wanted to write the equilibrium equation in terms of prefarm equilibrium wild fish W^* and the critical stocking level F_x discussed earlier. The first step was to rearrange terms in Eq. 3 to obtain

$$W(1/p_c^e - 1)/H_{0.5} + F(\varepsilon/p_c^e - 1)/H_{0.5} = 1. \quad (4)$$

Recall that if $F = 0$, $W = W^*$, and if $F = F_x$, $W = 0$. Equation 4 therefore yields

$$W^* = H_{0.5}/(1/p_c^e - 1) \text{ and } F_x = H_{0.5}/(\varepsilon/p_c^e - 1). \quad (5)$$

Substituting these back into Eq. 4 then gives

$$W/W^* + F/F_x = 1. \quad (6)$$

The equation for conservation of probability (Eq. 1) has become an equation for conservation of hosts. Next, I used Eq. 5 to substitute for F_x in Eq. 6 to obtain

$$W = W^* - F(\varepsilon - p_c^e)/(1 - p_c^e). \quad (7)$$

This equation says that if farmers medicate and shorten grow-out times to keep lice abundance on their fish $< p_c^e$ times lice abundance on wild fish, then their fish will be a sink for lice rather than a source, and wild fish will increase slightly. We saw above that $p_c^e \approx 0.01$, so keeping farm abundances at such a low level would be difficult. In the usual case, $\varepsilon \gg p_c^e$, and Eq. 6 simplifies to

$$W \approx W^* - \varepsilon F. \quad (8)$$

In other words, wild fish decline in proportion to the ratio of lice abundance on farm fish to lice abundance on wild fish. In view of the large stocking levels on salmon farms in Norway, it is not surprising that a treatment threshold of 0.5 female lice per farm salmon has not been low enough to reverse the decline of local wild salmon (Holst et al. 2003).

Conclusions

The main point of this essay is that population-level declines of wild fish in areas of sea-cage farming are unsurprising and that extirpation is a real possibility. Basic physics dictates that declines of sympatric wild fish due to parasites such as sea lice should always be the null hypothesis, and that such declines are likely in any aquaculture where protected farm hosts and sympatric wild hosts have a common parasite with a direct life cycle. This is a consequence of the randomness of larval capture and the large numbers of larvae produced by female sea lice. To conserve sympatric wild fish at pre-farm levels, lice abundance on farm fish must be kept to less than a few percent ($100p_c\%$) of their abundance on wild fish. Difficult mathematics are not required to understand these results and their implications.

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