when males of two biotypes were present with a female of a given biotype in the same arena, the B male and female responded by increasing their frequency of courtships, leading to more copulation events, whereas the ZHJ1 male and female did not do so. Moreover, although courtships between the two biotypes occurred, copulation never resulted; this confirmed that both B and ZHJ1 share incompletely isolated mate recognition systems. Further, B males not only courted females of either biotype more frequently than did ZHJ1 males, they also interfered more frequently with the courtships initiated by ZHJ1 males than did ZHJ1 males with courtships initiated by B males (tables S2 and S3). The mating behavior and interactions between B and AN differed in ways similar to what we found for B and ZHJ1, although details varied between the two combinations (*18*) (tables S4 and S5).

These results help to explain the underlying basis of the B biotype's capacity to invade and displace indigenous populations. The strong competitive ability of B results partly from its capacity to adjust sex ratio in favor of its population increase, and partly from its capacity to interfere with the mating of indigenous individuals. When the proportion of males is increased, B adults respond by increasing the frequency of copulation and consequently increasing the proportion of female progeny. Critical to this is that B responds independently of whether the males are all B or a mix. This interaction is extraordinary because the indigenous males actually help to promote copulation among the invaders and consequently increase the invaders' competitive capacity. In contrast, the indigenous females do not respond to increased numbers of adult males. Moreover, copulation by indigenous individuals is partly blocked by B males that readily attempt to court with females of either biotype—a behavior not reciprocated by the indigenous males. These asymmetric mating interactions have obvious population-level implications because the increase in the proportion of B females and the concomitant decrease in the proportion of indigenous females results in an immediate higher population growth rate for B and a lower growth rate for the indigenous population. As the abundance of B increases relative to the indigenous individuals, the increased allocation of eggs to female progeny and the active interference of mating of indigenous males by B males combine to drive the indigenous population to local extinction.

Mating interactions between closely related but reproductively isolated genetic groups are likely a common phenomenon (*3*, *22*–*24*) and are expected given the widespread existence of hybridization and introgression (*25*). Although examples of asymmetric competition are well known (*26*–*28*), asymmetric mating interactions are less well described (*28*). The rarity of examples may be, as illustrated by this study, the consequence of such interactions leading to the rapid displacement of the disadvantaged organisms. Biological invasions offer opportunities to gauge and characterize the potential magnitude and form of asymmetric mating interactions before species are lost through competitive exclusion or before the importance of competition is reduced over evolutionary time through niche partitioning and character displacement.

Allopatric species often demonstrate greater similarity in mating signals than do sympatric species, even when they have been diverging for a similar length of time (*3*). As a consequence of biological invasions, previously allopatric species are brought together and their partially similar mate recognition systems may promote asymmetric mating interactions between them. As we have shown, these interactions may play a critical role in determining the capacity of the invader to establish itself and the consequences for indigenous species.

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Declining Wild Salmon Populations in Relation to Parasites from Farm Salmon

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Rather than benefiting wild fish, industrial aquaculture may contribute to declines in ocean fisheries and ecosystems. Farm salmon are commonly infected with salmon lice (*Lepeophtheirus salmonis*), which are native ectoparasitic copepods. We show that recurrent louse infestations of wild juvenile pink salmon (*Oncorhynchus gorbuscha*), all associated with salmon farms, have depressed wild pink salmon populations and placed them on a trajectory toward rapid local extinction. The louse-induced mortality of pink salmon is commonly over 80% and exceeds previous fishing mortality. If outbreaks continue, then local extinction is certain, and a 99% collapse in pink salmon population abundance is expected in four salmon generations. These results suggest that salmon farms can cause parasite outbreaks that erode the capacity of a coastal ecosystem to support wild salmon populations.

The decline in ocean fisheries (*1*, *2*) and rise in global demand for fish have driven the rapid growth of aquaculture (*3*, *4*). Although aquaculture may augment fish supply (*3*), there are ecological risks, including competition and interbreeding of escaped farm fish with wild fish (*5*, *6*), depletion of wild fish caught to feed farm fish (*3*, *4*), and the spread of infec-

tion from farm fish to wild fish (*7*, *8*). Disease threats of aquaculture to wild fish populations have long been contentious because of the uncertainty in impacts on those populations (*9*–*12*). We assess the impact of recurrent aquaculture-induced salmon lice (*L. salmonis*) infestations on wild pink salmon (*O. gorbuscha*) populations.

The salmon louse is a native marine ectoparasitic copepod of salmonids that feeds on surface tissues and causes stress, osmotic failure, viral or bacterial infection, and ultimately death (*13*). Lice are directly transmitted via planktonic nauplii and copepodids that can persist for several days. In areas without salmon farms, the prevalence of *L. salmonis* on juvenile pink salmon 2 to 3 months after marine emergence is low (<5%) (*14*–*16*), because returning adult salmon are mostly offshore when juvenile salmon enter the sea (*16*, *17*). Louse infestations of wild juvenile salmon have occurred throughout the Broughton Archipelago in Pacific Canada (Fig. 1) from 2001 to 2005 (*7*, *8*, *14*, *18*, *19*). There, salmon farms situated in inlets and channels near rivers can increase copepodid densities above background levels for more than 80 km of wild salmon migration routes or, equivalently, for the first 2.5 months of the wild salmon's marine life (*8*). In response to a pink salmon population collapse in 2002, a primary migration corridor was fallowed in 2003 (i.e., farm salmon were removed from aquaculture facilities in Tribune Channel through Fife Sound, but farms peripheral to this route remained active) (Fig. 1). For that salmon cohort, *L. salmonis* abundance declined (*19*), and pink salmon marine survival increased (*20*).

To test for effects of lice on salmon population dynamics, we compiled Fisheries and Oceans Canada escapement data (the number of salmon per river), from 1970 to the present, for all pink salmon populations from rivers in the central coast of British Columbia, Canada (Fig. 1). There were 64 rivers whose salmon populations were not exposed to salmon farms and 7 rivers whose salmon populations must migrate past at least one salmon farm. Because pink salmon have a 2-year life cycle, there are distinct odd- and even-year lineages (*21*), which amount to 128 unexposed populations and 14 exposed populations. Rivers with substantial enhancement (e.g., spawning channels) were excluded because any increased salmon abundances in these rivers confound our estimates of natural changes in abundance. Unexposed populations had been and continue to be commercially fished. Exposed populations were commercially fished before the infestations, but the fishery remains closed since the onset of the infestations, when the data show a marked decline in productivity (Fig. 2 and fig. S1).

The analysis was based on the Ricker model (*22*), which is commonly used to model time-series data from density-dependent populations (*23*–*26*), including pink salmon (*24*, *26*), and provides robust estimates of population growth rates (*24*). The model is $n_i(t) = n_i(t-2)\exp[r - bn_i(t-2)]$, where $n_i(t)$ is the abundance of population i in year t, r is the population growth rate, and b determines density-dependent mortality. Upon log transformation to $\log[n_i(t)/n_i(t-2)] = r - bn_i(t-2)$, the Ricker equation becomes a linear model with intercept r and slope b that can be estimated by linear regression and hierarchical mixed-effects modeling (*23*, *24*, *27*, *28*). A preliminary model selection analysis did not support including random effects on r or b (fig. S2 and tables S1 and S2) (*27*). We therefore pooled data from multiple populations (*27*) and used linear regression to estimate parameters and parametric bootstrapping to construct 95% confidence intervals (CIs) on the parameter estimates (*23*). This allowed us to statistically compare parameters from pooled populations subjected to infestations, which is not possible with hierarchical mixed-effects models because there are only two data points per population during infestation years.

We compared parameter estimates among three groups: unexposed populations, exposed

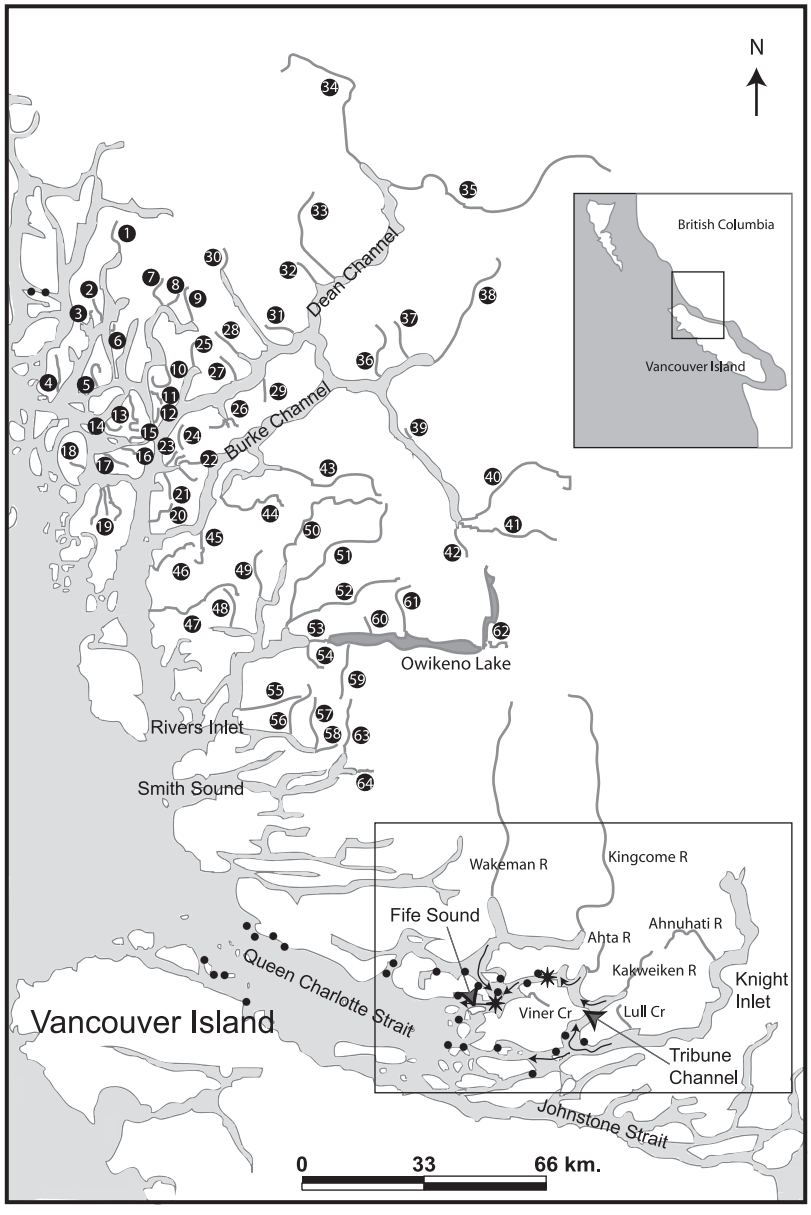

Fig. 1. Study area in the Broughton Archipelago (boxed area in inset), depicting pink salmon populations from unexposed rivers (numbered circles) and exposed rivers (directly labeled within the lower rectangular frame). Inferred migration routes in the Broughton Archipelago are shown by the small arrows. Salmon farms are shown by black dots and sample sites by stars. Salmon farms south of Knight Inlet are not shown. Identities of the numbered (unexposed) rivers are provided in data set S1 (*28*).

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preinfestation populations, and exposed populations during infestations (excluding the fallow year). The groups did not differ in b, and so we reanalyzed the data with b fixed among the three groups. Unexposed populations did not differ from exposed preinfestation populations in growth rate (unexposed populations: $r = 0.62$, 95% CI: 0.55 to 0.69; exposed preinfestation populations: $r = 0.68$, 95% CI: 0.46 to 0.90). The growth rate of exposed populations during the infestations was significantly lower and significantly negative ($r = -1.17$, 95% CI: –1.71 to –0.59; Fig. 3), meaning that if infestations are sustained, then local extinction is certain (*29*). Population viability analysis (*28*, *29*) revealed the mean time to 99% population collapse is 3.9 generations, with the 95% CI from 3.7 to 4.2. During two generations of infestations, some exposed populations have declined to <1%, whereas others have exceeded their historical abundance. We initially excluded the fallow data, because they contain only 1 year of observations and correspond to a nonrandom management action. By fixing $b = 0.64$, as estimated above, and estimating r from the remaining seven data points, we found the growth rate of fallow populations was significantly increased ($r = 2.50$, 95% CI: 1.28 to 3.62). The maximum reproductive rate for pink salmon is $r^* = 1.2$ (*24*). Fishing mortality probably reduced r for unexposed and exposed preinfestation populations. The depressed growth rate of exposed salmon populations during the infestations indicates that previous fishing mortality (now ceased) has been greatly exceeded by louse-induced mortality.

To estimate the mortality of pink salmon caused by lice, we extended the Ricker model to directly accommodate louse data collected from exposed populations during the infestations (*14*, *18*, *19*, *28*). We constrained the model by fixing $b = 0.64$ and by requiring $r = r^* = 1.2$, because there was no fishing mortality. Louse-induced mortality is represented by multiplying by $\exp[-aP_i(t-1)]$, where P is the mean abundance of motile (adult and preadult) lice per juvenile salmon from population i that spawned in year t. We log-transformed the model to $\log[n_i(t)/n_i(t-2) = r - bn_i(t-2) - aP_i(t)]$ and used linear regression to estimate a. The term $\exp[-aP_i(t-1)]$ significantly improved the fit of the model ($t = -5.019$, df = 33, $P = 1.74 \times 10^{-5}$; fig. S3), and results remained strong when the data were restricted by averaging populations and excluding some population groups ($P < 0.005$ for all groups; table S3). The parameter a corresponds to the rate of parasite-induced host mortality multiplied by the time that juvenile salmon are exposed to the parasites, $a = \alpha T$. The exposure time, T, is about 2 months (based on the migration speed of juvenile pink salmon through the archipelago), and the value of α has been estimated at 0.022 (motile lice × day)$^{-1}$ (based on survival experiments of naturally infected juvenile pink salmon) (*8*). Dividing the estimated $a = 0.89$ (95% credible intervals are from 0.46 to 1.34) by 60 days reveals an excellent correspondence between these two independent estimates of pathogenicity ($a/60 = 0.015$, with 95% credible estimates from 0.0077 to 0.022). Using a hierarchical Bayesian simulation (*28*) that represents uncertainty in the model fit as well as in the distribution of r^* (*12*), we found the estimated mortality of pink salmon, $1 - \exp[-aP_i(t-1)]$, caused by lice ranged from 16% to over 97% and was commonly over 80% (Table 1). The lowest mortality comes from fallow populations when louse abundance was nevertheless elevated, possibly resulting from transmission from active farms outside the fallowed corridor (*7*, *19*, *20*).

These results provide strong empirical evidence that salmon farm–induced *L. salmonis* infestations of juvenile pink salmon have depressed wild pink salmon populations and may lead to their local extinction. However, this parasite threat may not exist at low farm salmon abundances; the delay between the onset of salmon aquaculture in 1987 and louse infestations in 2001 (Fig. 2) may be explained by farm fish abundance crossing a host density threshold above which outbreak conditions occur (*30*). It is unlikely that another factor is responsible: The increased growth rate in response to fallowing rules out other factors that could have affected exposed, but not unexposed, populations. The results rely on extensive spatial replication to compensate for short time series in infestation

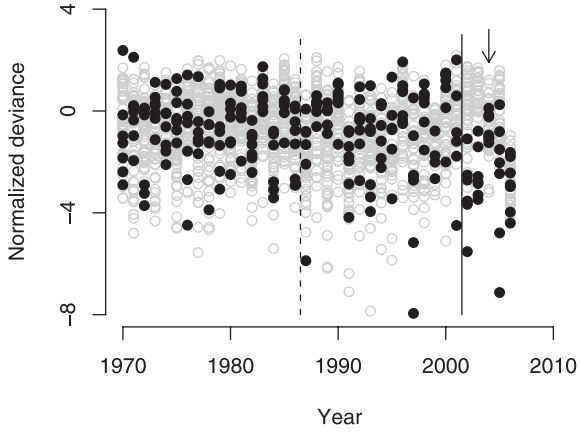

Fig. 2. Time series of normalized population deviances {$\log[N_i(t)/m_i]$, where $N_i(t)$ is the population estimate for population i in year t and m_i is the time-series mean abundance for population i} for 128 control populations of pink salmon (open gray circles) and 14 pink salmon populations exposed to salmon farms (black circles). The vertical dashed line marks the beginning of salmon aquaculture in the Broughton Archipelago. The vertical solid line marks the onset of louse infestations (and the commercial fishery closure) affecting the exposed populations. The arrow indicates data for exposed pink salmon cohorts that, as juveniles, experienced a fallowed migration corridor.

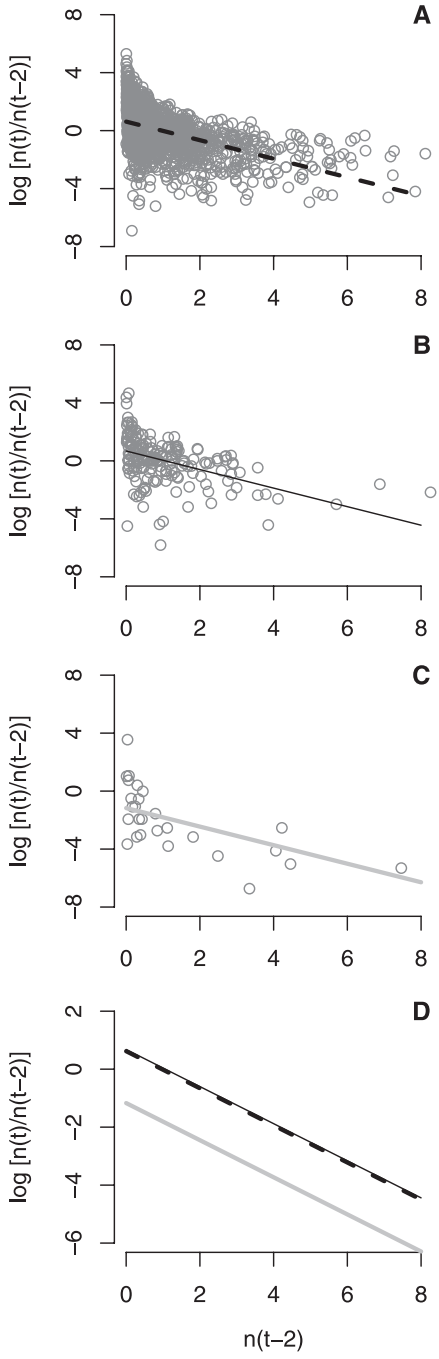

Fig. 3. Fits of the log-transformed Ricker model to escapement data for unexposed populations (**A**), exposed populations before infestations (**B**), and exposed populations during the infestations (**C**), and a comparison of the log-transformed Ricker model for the three groups in panels (A) to (C) (**D**). The intercept (growth rate) is lower for the exposed population during the infestations than for exposed populations before the infestations and the unexposed populations.

Table 1. Mean abundances, *P*, of motile *L. salmonis* on juvenile pink salmon and estimated parasite-induced host mortality, *M* (with upper and lower bounds of the 95% credible interval in parentheses), for exposed populations during infestations.

River	2002		2003		2004*		2005		2006	
	P	*M*	*P*	*M*	*P*	*M*	*P*	*M*	*P*	*M*
Ahta	3.4	95.21 (79.07, 98.95)	1.0	59.09 (36.87, 73.82)	0.3	23.52 (12.89, 33.10)	2.6	90.21 (69.76, 96.93)	0.4	30.06 (16.81, 41.49)
Kakweiken	3.4	95.21 (79.07, 98.95)	1.0	59.09 (36.87, 73.82)	0.3	23.52 (12.89, 33.10)	2.6	90.21 (69.76, 96.93)	0.4	30.06 (16.81, 41.49)
Viner	4.0	97.20 (84.12, 99.53)	2.2	86.00 (63.65, 94.76)	0.2	16.37 (8.79, 23.51)	2.3	87.20 (65.29, 95.41)	1.4	71.39 (47.48, 84.68)
Wakeman	4.0	97.20 (84.12, 99.53)	2.2	86.00 (63.65, 94.76)	0.2	16.37 (8.79, 23.51)	2.3	87.20 (65.29, 95.41)	1.4	71.39 (47.48, 84.68)
Kingcome	4.0	97.20 (84.12, 99.53)	2.2	86.00 (63.65, 94.76)	0.2	16.37 (8.79, 23.51)	2.3	87.20 (65.29, 95.41)	1.4	71.39 (47.48, 84.68)
Ahnuhati	2.6	90.21 (69.76, 96.93)	0.7	46.51 (27.53, 60.86)	0.2	16.37 (8.79, 23.51)	1.9	81.70 (58.27, 92.16)	0.3	23.52 (12.89, 33.10)
Lull	2.6	90.21 (69.76, 96.93)	0.7	46.51 (27.53, 60.86)	0.2	16.37 (8.79, 23.51)	1.9	81.70 (58.27, 92.16)	0.3	23.52 (12.89, 33.10)

*These data correspond to the salmon cohort responding to the fallow treatment in 2003.

years. The time to reach sufficient temporal replication to support hierarchical mixed-effects modeling, say 10 generations (which equals 20 years), greatly exceeds the predicted time to extinction. That is, there is a major risk associated with waiting for large data sets to accumulate before implementing conservation policy. Industrial aquaculture is rapidly expanding to new species, regions, and habitats (*31*), which can create parasite outbreaks that contribute to the decline of ocean fisheries and ecosystems.

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Supporting Online Material

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2 August 2007; accepted 2 November 2007
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Habitat Split and the Global Decline of Amphibians

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The worldwide decline in amphibians has been attributed to several causes, especially habitat loss and disease. We identified a further factor, namely "habitat split"—defined as human-induced disconnection between habitats used by different life history stages of a species—which forces forest-associated amphibians with aquatic larvae to make risky breeding migrations between suitable aquatic and terrestrial habitats. In the Brazilian Atlantic Forest, we found that habitat split negatively affects the richness of species with aquatic larvae but not the richness of species with terrestrial development (the latter can complete their life cycle inside forest remnants). This mechanism helps to explain why species with aquatic larvae have the highest incidence of population decline. These findings reinforce the need for the conservation and restoration of riparian vegetation.

Amphibian populations are declining worldwide (*1*, *2*). Among the factors determining the amphibian declines are habitat loss and fragmentation, which affect amphibians just as they affect any other organisms: through population isolation, inbreeding, and edge effects (*3*–*5*). Another important factor is the fungus *Batrachochytrium dendrobatidis*, a highly virulent pathogen that attacks many amphibian species and has been responsible for the decline of many populations even in undisturbed environments (*6*, *7*). Amphibians can also be threatened by climate shifts (*7*), ultraviolet-B radiation (*8*), introduction of exotic species (*9*), and agrochemical contaminants (*10*). We investigated the role of a further factor, which we define as "habitat split."

Amphibian species with aquatic larvae typically undergo a major ontogenetic niche shift, whereby tadpoles and adults occupy two distinct habitats (*11*). In pristine environments, the aquatic habitat of the tadpoles and the terrestrial habitat of the postmetamorphics grade into each other. However, in landscapes occupied by humans, land use has often resulted in a spatial separation between remnants of terrestrial habitat and breeding sites (*12*). Adults of species with aquatic larvae, in order to breed, are obliged to abandon forest remnants to reach water bodies, and at the end of the reproductive season, both