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# Transmission dynamics of parasitic sea lice from farm to wild salmon

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Marine salmon farming has been correlated with parasitic sea lice infestations and concurrent declines of wild salmonids. Here, we report a quantitative analysis of how a single salmon farm altered the natural transmission dynamics of sea lice to juvenile Pacific salmon. We studied infections of sea lice (*Lepeophtheirus salmonis* and *Caligus clemensi*) on juvenile pink salmon (*Oncorhynchus gorbuscha*) and chum salmon (*Oncorhynchus keta*) as they passed an isolated salmon farm during their seaward migration down two long and narrow corridors. Our calculations suggest the infection pressure imposed by the farm was four orders of magnitude greater than ambient levels, resulting in a maximum infection pressure near the farm that was 73 times greater than ambient levels and exceeded ambient levels for 30 km along the two wild salmon migration corridors. The farm-produced cohort of lice parasitizing the wild juvenile hosts reached reproductive maturity and produced a second generation of lice that re-infected the juvenile salmon. This raises the infection pressure from the farm by an additional order of magnitude, with a composite infection pressure that exceeds ambient levels for 75 km of the two migration routes. Amplified sea lice infestations due to salmon farms are a potential limiting factor to wild salmonid conservation.

**Keywords:** salmon conservation; aquaculture; sea lice; reservoir host; macroparasite; emergent disease

## 1. INTRODUCTION

Diseases can threaten wildlife when reservoir host populations are created, from which diseases can spill over into wildlife populations (McCallum & Dobson 1995; Daszak *et al.* 2000). Marine salmon farms located along wild salmon migration routes are spatially concentrated host populations that may serve as reservoirs and perturb the dynamics of any sympatric natural salmonid host–parasite system. Industrial-scale salmon farming has grown 55-fold over the last two decades (Porter 2003), and has spread globally, throughout and beyond the native ranges of wild salmonids. Sea lice (*Lepeophtheirus salmonis* Krøyer and *Caligus* spp.) are directly transmitted marine ectoparasites of salmonids: their life cycles have obligate parasitic and free-swimming stages, but no obligate intermediate host. Lice frequently infest farm salmon, and many studies have linked planktonic lice and lice parasitizing wild salmonids with the presence of farms (Tully & Whelan 1993; Costelloe *et al.* 1996, 1998*a*,*b*; Todd *et al.* 1997; Mackenzie *et al.* 1998; Tully *et al.* 1999; Bjørn *et al.* 2001; Bjørn & Finstad 2002; Marshall 2003; Morton & Williams 2004; Morton *et al.* 2004; McKibben & Hay 2004; Penston *et al.* 2004; Carr & Whoriskey 2004). Sympatric wild salmonid populations may then be affected: farms have been implicated in the infestation and collapse of pink salmon (*Oncorhynchus gorbuscha*) cohorts in Pacific Canada (PFRCC 2002; Morton & Williams 2004; Morton *et al.* 2004), wild sea trout (*Salmo trutta*) and Atlantic salmon (*Salmo salar*) populations in Europe (McVicar 1997, 2004). Assessing the role of salmon farms in these declines first requires a comprehensive understanding of how farms alter the natural transmission dynamics in this system, particularly involving the vertical transmission of lice from adult salmonid cohorts to susceptible juveniles. Here, we use mechanistic spatial models coupled with extensive field data to analyze how an isolated salmon farm perturbed the natural transmission dynamics of sea lice to juvenile pink and chum (*Oncorhynchus keta*) salmon along two narrow and restricted migration corridors in British Columbia, Canada.

Two native sea louse species coexist on salmonids in Pacific waters off North America: *L. salmonis* and *C. clemensi* (Parker & Margolis 1964). Both species have planktonic larval stages, and juvenile and adult parasitic stages. Planktonic nauplii hatch from gravid parasitic females and develop into infective copepodids. After settling on a host fish, copepodids develop through distinct chalimus and motile pre-adult and adult stages (Kabata 1972; Johnson & Albright 1991*a*,*b*). Attached stages feed on the mucus, scales and blood of the host fish leading to osmotic stress and emaciation of sufficiently infected hosts (Pike & Wadsworth 2000). The ecologies of these species differ in that *L. salmonis* are salmonid specialists, whereas *C. clemensi* are generalists occurring on members of several piscine families (Parker & Margolis 1964).

Pink and chum salmon are unique among salmonids in their precocious entry into marine waters: juveniles

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emerge from gravel and immediately leave their natal streams at approximately 28–35 and 30–40 mm fork length, respectively (Groot & Margolis 1991; personal observation). This makes them the smallest salmonids to contend with marine parasites. In this early marine phase, these species form mixed schools and occupy the same near-shore (often intertidal) habitats during their seaward migration (Groot & Margolis 1991). Marine entry begins in March (Groot & Margolis 1991) with juveniles initially free of lice. Direct vertical transmission of sea lice from returning conspecifics does not begin until late July, but transmission can occur indirectly immediately upon marine entry of juveniles via alternate wild hosts and farm salmon. The magnitudes of these transmission routes are unknown, but if the primary natural route is direct then fish farms may provide a significant transmission pathway for lice that operates much earlier in the salmon life cycle then would otherwise naturally occur.

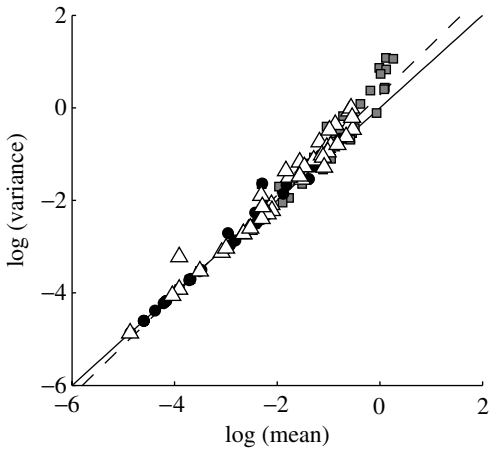


Figure 1. Log variance versus log mean for all 41 samples of copepodids (black circles) chalimi (grey squares) and motiles (clear triangles). There are 79–237 fish per sample. The solid line is the variance = mean line which accounts for 96% of the variation. The dashed line is the best-fit linear model, which has slope 1.08 and accounts for 97% of the variation. Compare with fig. 5 in Shaw & Dobson (1995).

## 2. FIELD METHODS

We sampled juvenile pink and chum salmon at 1–4 km intervals for 40 and 60 km along two narrow and restricted migration routes relative to an isolated salmon farm, and quantified the abundance of copepodid, chalimus and motile stages. The migration routes are labelled route I and II, respectively, and were located in marine fjordic habitats in British Columbia, Canada. An isolated salmon farm (farm A) was situated midway along both migration routes. A second salmon farm (farm B) was situated such that migratory salmon indirectly passed within 7 km of it toward the end of route I. We did not sample for approximately 20–60 km of the migration routes between the landward end of the study area and the various natal streams of the studied populations. Two replicate sets of samples (79–237 juvenile pink and chum salmon per sample) were obtained from each site in the spring of 2003 (17–27 April and 9–23 May). Four datasets result: two replicates of the two migration routes, each representing a spatially structured snapshot of louse population structure. Datasets are labelled: I-April, I-May, II-April, II-May. Route I and route II datasets share the same data landward of farm A. Further details of the field site are withheld to maintain industry anonymity.

At each site, juvenile pink and chum salmon (measuring 2.8–10 cm fork length) were captured by beach seine (30 m long, 4 mm mesh size). The beach seine was drawn in to approximately 1 $m^2 \times 30$ cm and a live subset was retained in 30 l buckets of seawater using a $15 \times 15$ $cm^2$ dipnet. Care was taken to maintain randomness by varying the location, depth and speed of the subsampling procedure. Individual fish were removed from a bucket with a $15 \times 15$ $cm^2$ dipnet and placed in a $15 \times 27$ $cm^2$ clear plastic envelope for analysis. Fins and lateral, dorsal and ventral surfaces were scanned with a $10\times$ magnification hand lens and the number of copepodid, chalimus and motile lice was recorded. Ovigerous females were classified as either *L. salmonis* or *C. clemensi*. Fish were then returned to buckets where more than 99% recovered and were subsequently released at the location of capture. This nonlethal assay is known to produce slight underestimates of copepodid and chalimus abundances and is only feasible for fish less than 10 cm fork length (Krkošek *et al.* in press). Temperature readings were taken at most sites and salinity readings were taken at a subset of sample sites around farm A with a Hydrolab Quanta electronic water quality meter.

## 3. MODEL

The large-scale movement of louse larvae in long and narrow fjordic habitats is limited to movements up and down the habitat length, and we model it with a one-dimensional domain. Juvenile salmon migrate down this domain, initially free of lice, and first encounter infective copepodids that originate from two primary host populations: farm salmon and sympatric wild hosts. As the infection progresses, juvenile salmon become a secondary source of louse larvae themselves. A distinction between the primary sources is their spatial distributions: a farm is a *point source* of lice whereas wild hosts are a *distributed source*. Each source corresponds to distinct spatial profiles in the dynamics of free-swimming and parasitic stages, which form the basis for distinguishing between farm-origin and natural-origin lice in spatially structured data.

Three models were developed (see Appendix A): $\Psi_0$ includes only natural primary sources of lice; $\Psi_1$ includes only farm sources of lice; and $\Psi_2$ includes both farm and natural primary sources of lice. Larvae from wild hosts are described by a uniform spatial distribution; a diversity of free-swimming hosts combined with the planktonic stages of lice suggests these larvae are well mixed. Advection–diffusion submodels describe the movements of nauplii and copepodids from the farm and the juvenile salmon. Infection dynamics on migratory juvenile salmon are linked to these larval distributions by a spatially dependent Poisson process (Papoulis 1963) that assigns a probability to each datum. In contrast to the common aggregated distributions of macroparasites on their host populations (Shaw & Dobson 1995; Shaw *et al.* 1998), the sea lice data conformed well to the characteristics of a Poisson process (see figure 2). The mean abundances of copepodid,

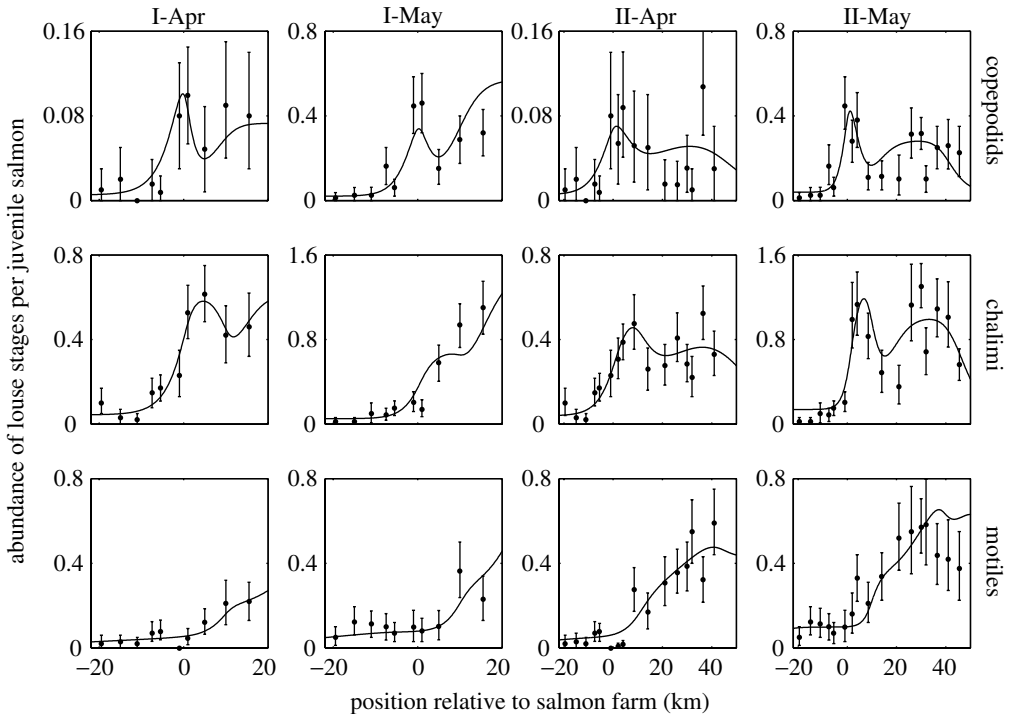


Figure 2. Abundance of parasitic louse stages on juvenile pink and chum salmon at points along their migration routes relative to a salmon farm located at $x=0$ (farm A). Salmon migrate in the rightward (seaward) direction. Columns correspond to datasets and rows correspond to louse stages. Error bars are bootstrapped 95% confidence intervals. Solid lines are the maximum likelihood best fits of model $\Psi_2$ to each dataset. Sample sizes are in the range 79–237 fish per sample.

chalimus and motile stages are

$$C(x) = \beta \frac{1}{v} \int_{x-\lambda_c}^{x} L(u)\, \mathrm{d}u,$$

$$H(x) = s_c \beta \frac{1}{v} \int_{x-\lambda_h}^{x-\lambda_c} L(u)\, \mathrm{d}u,$$

$$M(x) = s_c s_h \beta \frac{1}{v} \int_{x-\lambda_m}^{x-\lambda_h} L(u)\, \mathrm{d}u,$$

where $v$ is the mean seaward migration velocity of salmon, $\beta$ is the transmission coefficient and $s_c$ and $s_h$ are the proportions of lice surviving copepodid and chalimus stages. The $\lambda$s are the distances salmon travel in the cumulative mean durations of copepodid (c), chalimus (h) and motile (m) stages. The spatial distribution of infective larvae is $L(x)$: the number of planktonic copepodids within the unit of volume defined by the detection radius of lice centred at $x$. Only relative values become important, so the detection radius is not explicitly required. Maximum likelihood was used to fit models, likelihood ratios (Hilborn & Mangel 1997) to test if farms do not infect wild juvenile salmon and if secondary infection from the juvenile salmon is significant, and Akaike information criteria (AIC; Burnham & Anderson 2002) to select the best model among $\Psi_0$, $\Psi_1$ and $\Psi_2$. Both species of lice and juvenile salmon were included in the same analysis due to similarities in host behaviour and parasite life cycles.

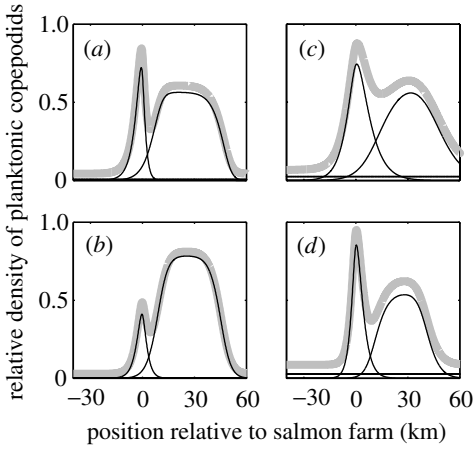


Figure 3. The spatial distributions of planktonic copepodids inferred by model $\Psi_2$ on a relative scale. Juvenile salmon migrate in the rightward (seaward) direction. The thick grey line is the total abundance of copepodids produced by all sources. The horizontal lines near zero are the ambient infection pressures. The thin dark curves oriented about $x=0$ are the distributions of copepodids produced directly by farm A and the second curves to the right are the distributions of copepodids produced by the farm-origin cohort of lice on the juvenile salmon. The latter distribution was found by solving the model with $\kappa=0$ to eliminate any contribution of lice from natural sources. Corresponding datasets are I-April (*a*), I-May (*b*), II-April (*c*), and II-May (*d*).

## 4. RESULTS

A total of 41 samples were collected across the four datasets yielding a total of 5514 juvenile salmon that were sampled for sea lice infections. From these fish, we counted a total of 552 copepodids, 2078 chalimi and 1015 motiles. Of these motiles, there were 12 ovigerous *L. salmonis* females and 53 ovigerous *C. clemensi* females. The distributions of lice on juvenile salmon ranged from slightly underdispersed to slightly overdispersed, and all samples closely followed the 1 : 1 variance to mean line,

Table 1. Summary of the spatial infection pressures caused by the salmon farm relative to ambient levels.
(Larval production per unit space was calculated based on a farm length of 0.2 km. Secondary larval production from the farm-origin cohort of lice on the juvenile salmon was calculated by solving the model with $\kappa = 0$ to eliminate any contribution from natural origin lice. Infection pressure is the local abundance of planktonic copepoidids.)

| statistic | KN-Apr | KN-May | TR-Apr | TR-May | average |
|---|---|---|---|---|---|
| primary larval production per unit space $\alpha \cdot (0.2\ \kappa)^{-1}$ | $3.19\times10^4$ | $5.48\times10^4$ | $2.52\times10^4$ | $1.37\times10^4$ | $3.14\times10^4$ |
| primary and secondary larval production per unit space $(\alpha + \int L_2(x)\ \mathrm{d}x)(0.2\kappa)^{-1}$ | $1.67\times10^5$ | $5.54\times10^5$ | $6.95\times10^4$ | $4.42\times10^4$ | $2.09\times10^5$ |
| maximum primary infection pressure $\max_x\{\alpha k(x)\}\kappa^{-1}$ | 87 | 144 | 30 | 32 | 73 |
| distance primary infection pressure exceeds ambient levels (km) | 25 | 27 | 45 | 23 | 30 |
| distance primary and secondary infection pressure exceeds ambient levels (km) | 73 | 76 | 91 | 62 | 75 |

which accounted for 96% of the variability in those data (figure 1). Sea temperatures were 8–10 °C and salinity readings were in the range 27.6–30.3‰.

The analysis revealed that lice from farm salmon infected wild juvenile salmon (likelihood ratio test, $R > 242.4$, d.f. $= 8$; $p < 0.001$ for all datasets) and that juvenile salmon were a secondary source of infection (likelihood ratio test, $R > 19.8$, d.f. $= 1$, $p < 0.001$ for all datasets). Model selection statistics (AIC) indicated $\Psi_2$ was the superior model for all datasets; the minimum $\Delta$AIC between all models and $\Psi_2$ was 22.4 and the probability $\Psi_2$ was the best model approached unity in each dataset (minimum Akaike weight $= 0.9998$). Statistical details are provided in the Electronic Appendix. These results strongly suggest that both wild and farm primary hosts were important sources of sea lice infecting juvenile salmon and in addition that juvenile salmon themselves were an important secondary source of sea lice.

The sea lice data were spatially structured: most lice were observed on juvenile salmon after they passed farm A (figure 2). Juvenile salmon carried low burdens of lice prior to their encounter with farm A. Near farm A, a large abundance of parasitic copepodids was observed followed by subsequent peaks in copepodids, chalimi and motiles further down the migration routes. The fit of model $\Psi_2$ agrees well with these data (figure 2) and explains these patterns. Prior to passing farm A, louse abundances were at natural ambient levels determined by a balance between immigration and emigration/death rates through each stage. Near farm A, a large cohort of lice colonized the juvenile salmon. These lice developed through subsequent chalimus and motile stages as their hosts migrated, producing the spatially displaced peaks in chalimi and motiles. Larvae were subsequently produced from the motile population on the juvenile hosts and produced the secondary infection waves of copepodids and chalimi apparent in the data.

Due to the mechanistic structure of the model, it was possible to analyse the transmission dynamics across each component in this system using parameter estimates from the maximum likelihood fits of model $\Psi_2$ to each dataset (figure 3). The analysis reveals that larvae originating from the farm and from the farm-produced cohort of lice on the juvenile salmon were responsible for the majority of the infection dynamics observed in the data. These differences can be quantified (table 1). Assuming the farm is 0.2 km in length, then the production of infective copepodids by the farm was on average $3.14\times10^4$ times greater than natural production in this spatial interval. This corresponds to an infection pressure near the farm that was on average 73 times greater than ambient levels and exceeded ambient levels for an average of 30 km. Inclusion of the dynamics of the farm origin cohort of lice on the juvenile salmon suggests that the production of larvae due to the farm was $2.08\times10^5$ times greater than ambient levels per unit space, with a composite spatial profile of infection pressure that exceeded ambient levels for 75 km (figure 3).

## 5. DISCUSSION

Our analysis identifies a signal amidst noise in empirical data. The signal is the ensemble of spatial distributions of lice predicted by the models. Any source of error that confounds the model predictions would detract from the results, while any unaccounted sources of variation that add noise to the data reduce the statistical power of the analysis. There were many unaccounted sources of noise in the sea lice data: variation in temperature and salinity; inter- and intra-specific variations in lice life-history parameters; inter- and intra-specific variations in juvenile salmon behaviour and host–parasite interactions; mixing of juvenile salmon with different immunological histories; deviations from a mean seaward migration velocity; deviations from a uniform juvenile salmon spatial distribution; temporal variations in the infestation levels on the farm; infection originating from farm B; density-dependent effects on louse survivorship and/or host mortality, potential patchiness in planktonic louse distributions; and generally, the 1D mathematical representation of a dynamic 3D biological system. The only confounding source of error would occur if a population of natural hosts was aggregated around the farm, producing the spatial distributions we attributed to the salmon farm. Were this to occur, our calculations indicate such an aggregated wild population would have to be either four orders of magnitude more dense than anywhere else in the study area or four orders of magnitude more infested than other wild hosts. Furthermore, the spatial distributions in the data require a point source that is stationary for at least two louse life cycles. For *L. salmonis*, this is at least 100 days at 10 °C (Johnson & Albright 1991*a*,*b*). Therefore, not only would such a population be unrealistically dense or infested, it would also be unrealistically stationary.

Given the paucity of confounding factors and abundant sources of noise, the strength of our results suggests the unaccounted sources of variation must be trivial relative to the effect of the salmon farm. Indeed, our calculations

suggest the farm raised infection levels by four orders of magnitude, and it is unlikely that other sources of error could vary by such a magnitude. However, a degree of noise is reflected in the variability of some parameter estimates (see Electronic Appendix) and in the deviation of some data from the best-fit models in figure 3. The statistical results are strong because those deviations are small relative to the overall dynamic pattern observed in the data and that pattern could only occur were a salmon farm the primary driver of sea lice dynamics on wild juvenile salmon. This is in agreement with Morton *et al.* (2004), who found virtually no lice on juvenile pink and chum salmon in several regions of British Columbia without salmon farms. However, a natural infestation of juvenile pink salmon with *C. clemensi* has been reported (Parker & Margolis 1964). Other European studies have found external oceanographic co-variates more important in determining louse dynamics (e.g. Marshall 2003), but such studies taken over seasonal time-scales import large temporal variations in temperature and salinity that affect the dynamics of all lice, but tell little about the interactions across wild and farm host populations. We have avoided such confounds by focusing on smaller temporal scales and explicitly examining the interactions among all host populations.

The models predict a close proximity of louse larvae to their source (figure 4*a*) and this agrees well with the abundance of copepodids around the salmon farm and around the peaks in the motile stages in the data. These results also agree with other investigations of spatial distributions of louse larvae that found consistent high levels near farms (Costelloe *et al.* 1996, 1998*a*,*b*). In contrast, one may expect most larvae to be transported long distances from their source owing to the one to two week lifespan of planktonic stages. This may be correct for a temporal *pulse* of larvae (figure 4*b*), but a farm is a *continuous* source, and it is the continuous production of larvae that produces the sustained maximum density of larvae near a farm. However, while a farm is a continuous source, population dynamics of lice on farms tend to oscillate due to population growth between intermittent treatment events (Revie *et al.* 2002) and this may affect the distribution of larvae around the source. Using biologically realistic parameter values, we found that the spatial profile of larvae produced by an oscillating source with amplitude half the mean tends to the predicted distribution of the constant mean source (figure 4*c* and *d*).

While no general conclusions can be made on the transmission dynamics of lice from farm to wild salmon based on this study alone (we only considered a single isolated salmon farm), the summation of these results with those of the current literature (listed in §1) strongly suggest these processes pervade most systems where wild and farm salmonids occur in sympatry. The data and analysis presented here extend our current understanding of lice interactions between wild and farm salmon by providing quantitative estimates of transmission rates across host populations while also considering subsequent transmission dynamics of lice within the wild population; initial transmission from farm to wild juvenile salmon may be minor compared with the subsequent population dynamics of the farm-produced *lineage* of lice. The spatial profiles of copepodid abundances inferred by the models

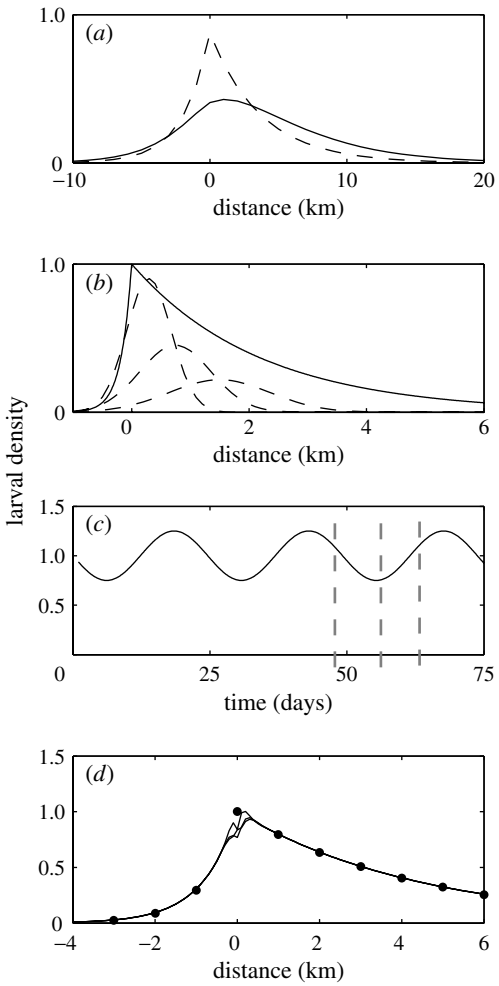


Figure 4. The spatial distributions of sea lice larvae around a point source. (*a*) The expected differences in the spatial distributions between nauplii (dashed line) and copepodids (solid line). (*b*) Differences in the spatial distributions of nauplii produced by a pulse release (dashed lines at successive time intervals) and by a continuous constant source (solid line) under strong advection. The dynamics of an oscillating source of amplitude half the mean (*c*). The frequency of oscillations is 14.8 per year, calculated from Revie *et al.* (2002). Three solutions for the spatial distributions of nauplii about the oscillating source are plotted in (*d*) for the time points indicated by the vertical dashed lines in (*c*). The expected distribution for the constant mean source is shown by the filled circles.

are strikingly consistent across all four datasets, lending support to this result.

Louse transmission from farm to wild salmon occurs during the most vulnerable salmon life-history phase (because of their small size and recent marine transition). For pink and chum salmon, this is a period prior to the return of adult conspecifics when near-shore native adult salmon abundances are lowest within annual variations (Groot & Margolis 1991). Presumably then, the natural rates of transmission to these juvenile fish are also at their lowest during this time. This agrees with the low rates of natural transmission shown here and by Morton *et al.* (2004) and suggests the primary natural route of vertical transmission to juveniles is direct: from returning conspecifics months later in the salmon life cycle after significant body growth in the juveniles has occurred.

The effect of a single salmon farm was to raise infection pressure by four to five orders of magnitude over natural rates, exceeding ambient levels for 75 km along two wild salmon migration routes. In some areas, juvenile salmon must migrate past several salmon farms, compounding these effects. This may have severe consequences, as these species may not have evolved mechanisms to contend with such high infestation pressures so early in their life-history. In fact, the near absence of natural vertical transmission of lice during this time would facilitate the evolution of the precocious marine phase evidenced by these species. Currently, the lethal infection levels for juvenile pink and chum salmon are unknown, but given their small size on marine entry (0.2–0.5 g; Groot & Margolis 1991, personal observation) and the known lethal limits for other species (1 motile louse per 0.75–1.6 g body weight for European salmonids; Grimnes & Jakobsen 1996; Bjørn & Finstad 1997), any amplification of louse transmission during this life-history phase may threaten the viability of the affected populations. The European experience is that louse management on farms has not resulted in positive effects on depressed wild salmonid populations (McVicar 2004). The rate of industry expansion in British Columbia is much faster than evolutionary time-scales, and therefore, an adaptive response by native salmonids is unlikely. Declines of wild salmonid populations correlated with sea lice infestations and salmon farms in Canada (PFRCC 2002; Morton & Williams 2004; Morton *et al.* 2004) and Europe (McVicar 1997, 2004) demand a cautionary approach to continued industrial expansion.

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## APPENDIX A

### (a) *Larval distribution models*

The study area is modelled in one spatial dimension. The distribution of planktonic copepodids from natural sources is approximated by a uniform spatial distribution: $L_0(x)=\kappa$. We now derive probability density functions (PDFs) for the spread of nauplii and planktonic copepodids around a point source of arbitrary strength at an arbitrary location, $x=y$. These PDFs then define the spatial distributions of larvae produced by a farm and by lice on the juvenile hosts. The spread of nauplii is modelled by the advection–diffusion equation

$$\frac{\partial n}{\partial t} = D\frac{\partial^2 n}{\partial x^2} - \gamma\frac{\partial n}{\partial x} - \mu_n n,$$

with the conditions $\lim_{x\to\pm\infty} n(x)=0$. The diffusion coefficient $D$ accounts for the combined effect of tides and random movements of individuals, $\gamma$ is the advection of larvae due to currents, and individuals die at a per capita rate $\mu_n$. The spatial steady-state solution yields the PDF for the distribution of larvae around the source:

$$k_n(x) = c_n\begin{cases} e^{a_1(x-y)}, & x\le y \\ e^{a_2(x-y)}, & x>y \end{cases} \quad a_2<0<a_1,$$

where $a_{1,2}=[\gamma\pm(\gamma^2+4\mu_n D)^{0.5}]\ (2D)^{-1}$ and $c_n$ ensures the PDF integrates to 1.

If most planktonic copepodids do not find a host, the spread of copepodids around a point source of arbitrary strength at $x=z$ is given by the same advection–diffusion equation with $\mu_n$ replaced by $\mu_p$, the per capita rate death rate of copepodids. This produces a similar PDF for the distribution of copepodids around $x=z$, given by

$$k_p(x) = c_p\begin{cases} e^{b_1(x-z)}, & x\le z \\ e^{b_2(x-z)}, & x>z \end{cases}, \quad b_2<0<b_1,$$

where $b_{1,2}=[\gamma\pm(\gamma^2+4\mu_p D)^{0.5}]\ (2D)^{-1}$ and $c_p$ ensures the PDF integrates to 1. In an effort to minimize unidentifiable parameters we fixed $\mu_p=2\mu_n$, based on longevity experiments (Johnson & Albright 1991*b*). The distribution of nauplii around $x=y$ forms a distributed source of copepodids, $k_n(x)$, and the PDF for the resulting distribution of copepodids around $x=y$ is given by the convolution

$$k(x) = \int_{-\infty}^{\infty} k_n(z)k_p(x-z)\,\mathrm{d}z.$$

The spread of $\alpha$ copepodids produced by a farm at $x=0$ is then $L_1(x)=\alpha k(x)$.

Assuming that the spatial distribution of juvenile salmon is uniform, then $M$ parasitic motile lice per juvenile salmon at location $y$ will produce $\varphi$ planktonic copepodids, and these copepodids will be distributed according to

$$L_2(x) = \varphi\int_{-\infty}^{\infty} M(y)k(x-y)\,\mathrm{d}y.$$

The composite spatial distribution of infective copepodids from all three sources is simply their summation: $L=L_0+L_1+L_2$.

### (b) *Infection model*

We approximate the migration of juvenile salmon by a mean rightward (seaward) velocity ($v$). Salmon encounter planktonic copepodids, which attach at a rate $\beta$ per unit time or $(1/v)\beta$ per unit space. Let $\tau_c$, $\tau_h$ and $\tau_m$ be the mean durations of copepodid, chalimus and motile stages, respectively. We define $\lambda_c=v\tau_c$, $\lambda_h=v(\tau_c+\tau_h)$ and $\lambda_m=v(\tau_c+\tau_h+\tau_m)$, which are the distances a juvenile salmon travels in the cumulative mean durations of copepodid, chalimus and motile stages, respectively. Let $N_c(x)$, $N_h(x)$ and $N_m(x)$ be spatially explicit discrete random variables for the number of copepodid, chalimus and motile lice on an individual juvenile salmon, respectively. If we assume infection events occur independently then $N_c$ is a variation on the Poisson process with a variable rate parameter (Papoulis 1963) and spatially explicit mean, $C(x)$, given in the main text. A count of $k$ chalimus lice on an individual salmon could occur from any $k$ of $n$ attached copepodids surviving to the chalimus stage with probability

$s_c$. It follows that

$$P\{N_h = k\} = \sum_{n=k}^{\infty} \left[ \binom{n}{k} (s_c)^k (1 - s_c)^{n-k} \left( \frac{[I_h(x)]^n}{n!} e^{-I_h(x)} \right) \right]$$

$$= \frac{1}{k!} [s_c I_h(x)]^k e^{-s_c I_h(x)},$$

where $I_h$ is the mean number of attached copepodids available for recruitment into the chalimus stage at location $x$:

$$I_h(x) = \beta \frac{1}{v} \int_{x-\lambda_h}^{x-\lambda_c} L(u)\, du.$$

Thus, $N_h$ is a Poisson random variable with mean, $H(x)$, given in the main text. In the same way, we define $s_h$ as the probability a chalimus louse survives to the motile stage and arrive at a Poisson distributed spatially explicit mean for motile stages, $M(x)$.

We assume a time-scale such that only two complete louse life cycles could occur on the juvenile hosts. The model was then solved numerically using a fast Fourier transform algorithm in MATLAB by first finding the solution for the distributions of parasitic stages arising from the primary larval distributions (from farm and non-juvenile salmon wild hosts) and then allowing reproduction and spatial redistribution of secondary larvae produced by the motile population on the juvenile hosts.

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