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ARTICLE

Estimation of spatiotemporal transmission dynamics and analysis of management scenarios for sea lice of farmed and wild salmon

Stephanie J. Peacock, Martin Krkošek, Andrew W. Bateman, and Mark A. Lewis

Abstract: Parasite transmission between farmed and wild salmon affects the sustainability of salmon aquaculture in Pacific Canada. Understanding and managing parasites in aquaculture is challenged by spatial and temporal variation in transmission dynamics. We developed a mechanistic model that connects sea louse (*Lepeoptheirus salmonis*) outbreak and control on farmed salmon (*Salmo salar*) to spatiotemporal dynamics of sea lice on migrating wild juvenile salmon (*Oncorhynchus keta* and *Oncorhynchus gorbuscha*). We fitted the model to time series of sea lice on farmed salmon and spatial surveys of juvenile wild salmon in the Broughton Archipelago. We used the parameterized model to evaluate alternative management scenarios based on the resulting sea louse infestations and predicted mortality of wild salmon. Early and coordinated management of sea lice on salmon farms was most effective for controlling outbreaks in wild salmon, while uncoordinated treatments led to a resurgence of sea lice on salmon farms during the juvenile salmon migration. This study highlights the importance of incorporating spatiotemporal variability when considering infectious disease dynamics shared by farmed and wild hosts, particularly when migratory wildlife are involved.

Résumé : La transmission de parasites entre saumons d'élevage et saumons sauvages a une incidence sur la pérennité de la salmoniculture sur la côte ouest du Canada. Les variations spatiales et temporelles de la dynamique de la transmission compliquent la compréhension et la gestion des parasites en aquaculture. Nous avons mis au point un modèle mécaniste qui relie les éclosions et le contrôle des poux du poisson (*Lepeoptheirus salmonis*) chez les saumons d'élevage (*Salmo salar*) à la dynamique spatiotemporelle des poux du poisson se trouvant sur des saumons sauvages (*Oncorhynchus keta* et *Oncorhynchus gorbuscha*) juvéniles en migration. Nous avons calé le modèle sur des séries chronologiques de données sur les poux du poisson sur des saumons d'élevage et sur des données d'évaluations spatiales de saumons sauvages juvéniles dans l'archipel Broughton. Nous avons utilisé le modèle paramétré pour évaluer différents scénarios de gestion à la lumière des infestations de poux du poisson en résultant et avons prédit la mortalité des saumons sauvages. La gestion précoce et coordonnée des poux du poisson dans les fermes salmonicoles constitue la mesure la plus efficace de contrôle des éclosions chez les saumons sauvages, alors que des traitements non coordonnés mènent à une résurgence de poux du poisson dans les salmonicultures durant la migration des saumons juvéniles. L'étude souligne l'importance d'incorporer la variabilité spatiotemporelle dans l'examen de la dynamique des maladies infectieuses communes à des hôtes sauvages et d'élevage, particulièrement dans les systèmes comprenant des espèces migratrices. [Traduit par la Rédaction]

Introduction

The effective management of disease depends on a solid understanding of the spatial and temporal processes affecting transmission dynamics (Keeling and Eames 2005; White et al. 2018). For wildlife, disease dynamics can be complicated by the movement of hosts over large distances and the associated variability in infection pressure that they experience (Altizer et al. 2011). Further, at any given location, temporal changes in infection pressure due to seasonality of both parasite and host life cycles (Altizer et al. 2006) or disease dynamics in reservoir hosts (Krkošek et al. 2006*a*) may result in fluctuating sources of infection (Hudson et al. 2002). Examples include social dynamics and dispersal of badgers (*Meles meles*) that underlie spatiotemporal variation of infection risk of bovine tuberculosis to domestic cattle (Delahay et al. 2000), oscillations in measles dynamics in urban centres causing periodic traveling waves of infection through rural communities (Grenfell et al. 2001), and parasite dispersal from and control on salmon farms causing spatiotemporal variation in infection risk along migration routes of wild salmon (Krkošek et al. 2010). Failure to consider the inherent spatiotemporal variability in infection dynamics can lead to erroneous conclusions about the risk of infectious disease to host populations and ineffective management recommendations.

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Sea louse transmission between farmed salmon and migrating wild salmon in coastal environments is one example of a system that exhibits strong spatial and temporal variability (Rees et al. 2015; Groner et al. 2016*b*). Sea lice (*Lepeoptheirus salmonis*) are copepod parasites that infect salmonids, feeding on epidermal tissues, muscle, and blood (Costello 2006). Sea lice hatch as free-living and nonfeeding nauplii that can disperse tens of kilometres in ocean currents before finding a suitable host, attaching, and developing through copepodite, chalimus, and motile adult stages (Stucchi et al. 2011; Amundrud and Murray 2009). High infestation intensities on adult hosts may cause host morbidity and mortality (Pike and Wadsworth 2000) and have major biological and economic impacts on the salmon-farming industry (Costello 2009; Abolofia et al. 2017). Although Pacific salmon (*Oncorhynchus* spp.) have been found to be more resistant to infestation (Jones et al. 2008; Johnson and Albright 1992), even low abundances on small, juvenile salmon may cause mortality (Morton et al. 2005) or sublethal effects on physiology (Nendick et al. 2011; Brauner et al. 2012) and behaviour (Krkošek et al. 2011*a*; Godwin et al. 2015).

Open-net salmon farms provide a reservoir host for sea lice that results in spatial variability in infestation pressure along the migration routes of wild juvenile salmon (Krkošek et al. 2006*a*). Transmission of sea lice from farmed salmon has been implicated in epizootics of wild salmon in Norway (Bjørn et al. 2001), Ireland (Gargan et al. 2003), Scotland (Butler and Watt 2003), and Canada (Krkošek et al. 2006*a*). In Pacific Canada, in particular, these epizootics pose a conservation risk to ecologically, culturally, and economically important wild salmon (Krkošek et al. 2011*b*). Outmigrating juvenile pink (*Oncorhynchus gorbuscha*) and chum (*Oncorhynchus keta*) salmon are most vulnerable due to their small size and underdeveloped scales when they enter the marine environment (Brauner et al. 2012).

The strength of infestation pressure from salmon farms changes depending on environmental conditions such as temperature and salinity that affect sea louse development and survival, but more importantly on the management of sea lice by the industry (Rogers et al. 2013). The cost of sea lice to the salmon-farming industry is on the order of hundreds of millions of US dollars annually (Abolofia et al. 2017), and there have been numerous management strategies to reduce louse abundances on farmed salmon. In Pacific Canada, in-feed treatments with emamectin benzoate (EMB; trade name SLICE) are the most common treatment for sea lice, and farms are required to treat (or harvest) if the number of sea lice per farmed salmon exceeds three motile lice (Fisheries and Oceans Canada 2018). For the most part, EMB has been effective at reducing sea louse infestations of farmed salmon (Saksida et al. 2010), although sporadic and localized reports of resistance to the drug among Pacific sea lice indicate this may change (Messmer et al. 2018). Nonetheless, chemical treatments have and continue to be a strong driver of sea louse population dynamics on salmon farms (e.g., Krkošek et al. 2010), which in turn influence infestations of sympatric wild salmon.

Although the environmental and management factors affecting sea louse dynamics on farmed and wild salmon have received considerable attention (e.g., Revie et al. 2005; Jansen et al. 2012; Rogers et al. 2013; Bateman et al. 2016), no studies have integrated the spatial dynamics of wild salmon migration and temporal dynamics of sea lice on farmed salmon. Interannual changes in average parasite abundance show a positive correlation between sea lice on farmed salmon and infestations of wild juvenile salmon (Marty et al. 2010; Peacock et al. 2013). However, within-year transmission dynamics that govern if and how an outbreak will emerge are mediated by ocean currents, the life cycle of sea lice, and migration patterns of wild salmon in relation to farms. To understand how chemical treatments on farms influence transmission dynamics of sea lice from farm to wild salmon, we must explicitly consider such complexity.

Some of these complex physical and biological processes have been modelled with respect to sea louse dispersal from salmon farms. At a fine resolution, Stucchi et al. (2011) conducted particle-tracking simulations that captured the three-dimensional dispersal of sea louse nauplii from a salmon farm, including the effects of wind, tides, and freshwater input as well as the vertical migration of sea louse nauplii and the effects of temperature and salinity on sea louse survival and development. These simulations were more recently expanded on to examine the roles of individual farms within the Broughton Archipelago (hereinafter BA), Canada, as "emitters" or "receivers" of sea louse infestations (Cantrell et al. 2018). At a coarser scale, Aldrin et al. (2013) modelled dispersal using simple seaway distance metrics, but included transmission of lice among numerous salmon farms and from nonspecified reservoirs for sea lice such as wild salmonids. Complex simulation models like the former can suggest major drivers of spatiotemporal variability in infestations and yield specific, detailed predictions, but one advantage of simpler models is that they can be fit to data to infer unknown parameters. This simpler approach has been taken to quantify the relative importance of salmon farms in driving sea louse infestations of wild salmon. Krkošek et al. (2005*a*, 2006*a*) modelled the broad-scale ocean currents as an advection–diffusion process, yielding a steady-state spatial distribution of infectious sea lice around salmon farms and the subsequent attachment and development of sea lice on migrating wild salmon. However, unlike the previously mentioned studies, Krkošek et al. (2005*a*, 2006*a*) ignored the sea louse population dynamics on salmon farms and considered farms to be a constant source of infectious-stage sea lice. Because of the large temporal fluctuations in sea louse numbers on farmed salmon (e.g., Krkošek et al. 2010; Jansen et al. 2012), ignoring the source dynamics could lead to erroneous conclusions about how farm management influences parasite population dynamics as well as survival of wild salmon.

In this study, we develop a mechanistic model that connects temporal dynamics of sea louse populations on farmed salmon to the spatiotemporal infestations of wild juvenile salmon and fit this model to data from sea louse monitoring of both farmed and wild salmon in BA. We use the fitted model to evaluate different farm management scenarios by the resulting juvenile salmon infestation dynamics, building on previous studies that have evaluated treatment timing based on effective control on salmon farms alone (e.g., Revie et al. 2005). The results may inform the management of salmon farms for the benefit of wild salmon, but are also an example of how spatiotemporal variability in infection pressure can be incorporated into models used to inform management of diseases.

Methods

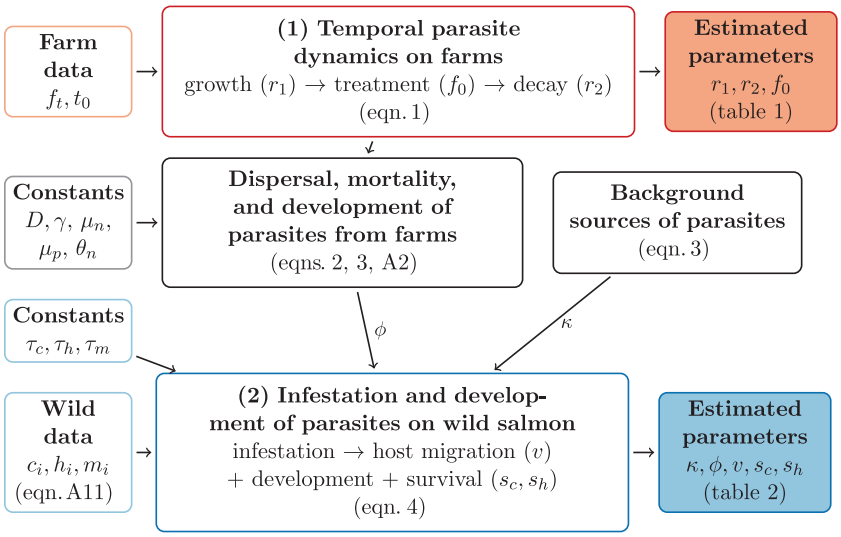
Model

We connected sea louse infestations of farmed salmon to observed louse abundances on juvenile wild salmon using a mechanistic model that includes sea louse population dynamics on farms in response to parasite control (Krkošek et al. 2010), dispersal of sea lice from farms, and infestation and development of lice on wild juvenile salmon (Krkošek et al. 2005*a*) (Fig. 1).

Sea louse populations on farmed salmon tend to grow exponentially in the absence of control and decline exponentially after treatment with parasiticide (Krkošek et al. 2010; Rogers et al. 2013). These temporal fluctuations in louse abundance on farmed salmon impact the infestation pressure on juvenile wild salmon migrating past salmon farms. We modelled the mean number of motile sea lice per farmed salmon as follows:

$$(1)\qquad f(t) = f_0 \begin{cases} e^{r_1 (t-t_0)} & t < t_0 \\ e^{r_2 (t-t_0)} & t \geq t_0 \end{cases}$$

Fig. 1. Schematic of the modelling framework for the sea louse transmission model incorporating temporal dynamics of lice on salmon farms. The model was fit to data in two stages: (1) the farm dynamics were fit to counts of lice on farmed salmon (red), and (2) the final predictions of lice per wild juvenile salmon were confronted with data of sea louse abundances on wild salmon throughout their migration route (blue). [Colour online.]



where $f(t)$ is the mean number of motile sea lice per farmed salmon at time t, r_1 is the population growth of lice before treatment, r_2 is the rate of decay after a treatment, f_0 is the mean number of motile sea lice per farmed salmon at the time of treatment, and t_0 is the treatment date. We assumed that host population size on salmon farms is approximately constant when farms are stocked, and thus the effect of host population size on sea louse population growth is included in the growth parameter for each farm, r_1 (Krkošek et al. 2010). Equation 1 does not include negative or positive density dependence of sea lice. Negative density dependence is unlikely on farmed hosts due to management interventions at low to moderate louse densities. Although several modelling studies have included positive density dependence (i.e., mate limitation at low densities; Groner et al. 2014; McEwan et al. 2019), we found that the exponential growth model fit the data well, perhaps because mate limitation is less likely on farmed salmon that are larger and in higher densities than juvenile wild salmon (Cox et al. 2017). We also found that sea lice were overdispersed on farmed hosts (see Results), which reduces the probability of mate limitation (Stormoen et al. 2013).

Several studies have found that in-feed treatments with EMB are effective for a period of ~3 months (e.g., Saksida et al. 2010; Rogers et al. 2013). Therefore, at time $t_0 + 90$ days, we assumed the treatment efficacy to have declined to the extent that sea louse population growth was again possible, and the growth rate returned to r_1.

Sea louse nauplii hatch from gravid motile sea lice and can disperse tens of kilometres from the open-net pens containing farmed salmon (Foreman et al. 2009). The temporal dynamics of sea louse populations at discrete farm locations can therefore lead to spatiotemporal patterns of infestation pressure on juvenile wild salmon migrating past farms. To capture this spatial dimension, we considered the dispersal of naupliar sea lice from salmon farms along the migration corridor of juvenile salmon through Knight Inlet – Tribune Channel (Fig. 2). This migration corridor is much longer (>100 km) than it is wide (≈1 km), and in this model we follow Krkošek et al. (2005*a*, 2006*a*) and consider the migration corridor to be a one-dimensional domain along which sea lice disperse and juvenile salmon migrate. The dispersal of nauplii is described by

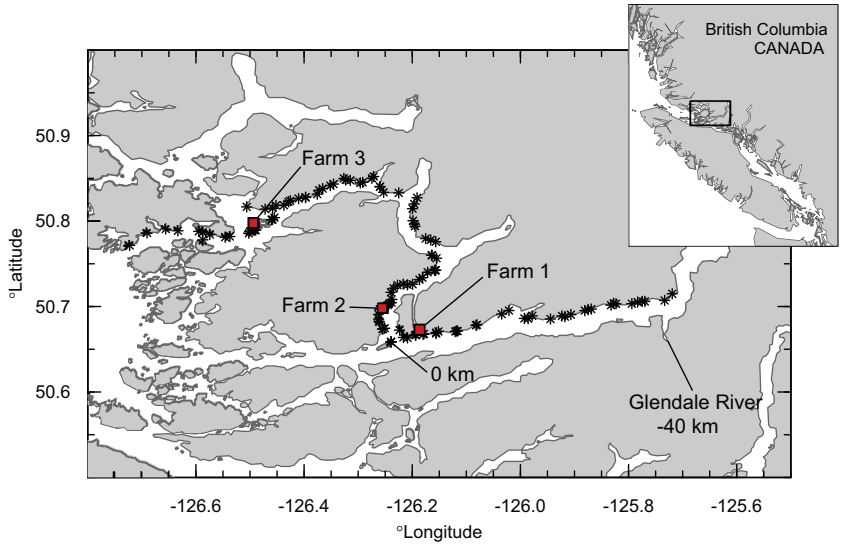
$$(2)\qquad \frac{\partial n_i}{\partial t} = \underbrace{f_i(t)\ \omega S_i(t)\ \delta(x - y_i)}_{\text{(1) production}} + \underbrace{D\frac{\partial^2 n_i}{\partial x^2} - \gamma\frac{\partial n_i}{\partial x}}_{\text{(2) diffusion–advection}} - \underbrace{(\mu_n + \theta_n)n_i}_{\text{(3) mortality–development}}$$

where $n_i(x, t)$ is the density of nauplii at location x and day t, originating from farm i. Nauplii are produced by motile sea lice at farm i, where $f_i(t)$ is the mean number of motile lice per salmon on farm i from eq. 1, ω is an unknown parameter for the fecundity of motile lice times the dilution of nauplii in three dimensions, and $S_i(t)$ is the number of farmed salmon on farm i at time t (Marty et al. 2010). In our case, the number of salmon in each farm was similar and relatively constant throughout the period considered, and so we assume that $\omega S_i(t)$ is constant (see the online Supplementary material, Fig. S1[1] for comparison of average versus total motile *L. salmonis* per farmed salmon). The production of sea lice from farmed salmon occurs at exactly location y_i and is described by a delta function, $\delta(x - y_i)$, which assumes that the length of the farm is small (i.e., on the scale of metres) relative to the spatial domain of the juvenile salmon migration route (tens of kilometres). The inclusion of a time-varying source of sea lice from salmon farms is a novel development from previous work (Krkošek et al. 2005*a*) and one that is necessary when lice on farmed salmon are changing dramatically in response to treatment.

The second part of eq. 2 captures random diffusion due to winds and tides, where D is the diffusion coefficient, and a general seaward advective flow due to high freshwater influx at the heads of inlets, where γ is the advection coefficient. These parameters have been estimated in previous studies as D = 22.67 km^2·day^{-1} (Krkošek et al. 2006*a*) and γ = 1.56 km·day^{-1} (Brooks 2005), and we fix them at these values to avoid identifiability problems with

[1]Supplementary data are available with the article through the journal Web site at http://nrcresearchpress.com/doi/suppl/10.1139/cjfas-2019-0036.

Fig. 2. Models were fit to data from the Broughton Archipelago, on the south-central coast of British Columbia, Canada. Sampling of farmed salmon took place on the three active salmon farms in the study region in 2006 (squares). Approximately 100 juvenile wild pink and chum salmon were sampled every ~1 km (stars) along their migration past these farms along the Knight Inlet – Tribune Channel migration corridor. The model was simulated from −60 to 80 km along the migration corridor, with 0 km being a reference point at the confluence of Knight Inlet and Tribune Channel. The map was produced using the R package PBSmapping (Schnute et al. 2015) with shoreline data from Wessel and Smith (2016).



other parameters in our model (e.g., the migration speed of salmon; see below), which are directly confounded.

The third part of eq. 2 describes natural mortality of nauplii at rate μ_n and development into copepodites at rate θ_n. Experimental data of nauplii mortality and development rates indicate $(\mu_n + \theta_n) = 4/5$ day^{-1} (Krkošek et al. 2006*b*). We calculated the solution to eq. 2, yielding the density of nauplii at any point along the migration x and time t, by numerically convolving the solution for advection–diffusion-decay (parts 2 and 3 of eq. 2), known as a Green's function, with the production term (part 1; Polyanin and Nazaikinskii 2016). Fixing the diffusion, advection, mortality, and development parameters allowed us to calculate the distribution of nauplii outside of the estimation of parameters for infestation and survival of wild salmon (see below) and thus increased computational efficiency and feasibility of model fitting. In the Discussion, we further justify these assumptions and consider errors they may introduce. Details of this solution are given in Appendix A.

Nauplii develop into copepodites, which can attach to susceptible juvenile salmon in the vicinity. These copepodites diffuse and advect via the same process described by eq. 2, except with mortality $\mu_p = 1/5$ day^{-1} (Krkošek et al. 2006*b*) and production $\theta_n n_i(x, t)$. We calculated the distribution of copepodites numerically by convolving the distribution of nauplii with the Green's function described above (see Appendix A for details). The total distribution of farm-source copepodites is the sum of the copepodid densities from all farms along the migration route, which we call $L_1(x, t)$. The infestation pressure for migrating wild salmon is a combination of background and farm sources of sea lice:

$$L(x,t) = \kappa + \phi L_1(x,t) \tag{3}$$

where κ is the background density of copepodites from distributed sources (e.g., returning wild adult salmon), and $\phi = \omega S_i(t)$ is the unknown fecundity–dilution parameter for sea lice from farmed salmon from eq. 2.

Copepodites attach to juvenile salmon and subsequently develop through chalimus and motile stages. The expected number of attached copepodid, chalimus and motile lice on a wild juvenile salmon at any point (x, t) is proportional to the density of infectious copepodites that fish previously encountered along its migration (Krkošek et al. 2005*a*). We assumed that wild salmon migrate at a constant speed v and calculate the expected number of sea lice on a wild juvenile salmon as the integral of the distribution of infectious copepodites, $L(x, t)$, along the migration path of juvenile salmon through space and time (i.e., the line integral):

$$C(x,t) = \beta \int_0^{\tau_c} L(x - vu, t - u)du \tag{4a}$$

$$H(x,t) = \beta s_c \int_{\tau_c}^{\tau_c+\tau_h} L(x - vu, t - u)du \tag{4b}$$

$$M(x,t) = \beta s_c s_h \int_{\tau_c+\tau_h}^{\tau_c+\tau_h+\tau_m} L(x - vu, t - u)du \tag{4c}$$

where $C(x, t)$, $H(x, t)$, and $M(x, t)$ are the expected number of attached copepodid, chalimus, and motile lice, respectively, on a juvenile salmon at kilometre x and day t; τ_c, τ_h, and τ_m are the respective developmental times of copepodites, chalimus, and motiles; and s_c and s_h are the respective survival of copepodites and chalimus to the next stage. The developmental times of copepodites, chalimus, and motiles (τ_c, τ_h, and τ_m) have been previously estimated, and therefore we assumed developmental to be constant at their 10 °C averages (Stien et al. 2005; Table A1). This assumption seemed reasonable given that the mean (±SE) temperature over the period of wild salmon sampling was 9.5 ± 0.12 °C.

We modelled the transmission coefficient, β, to be a Gamma random variable with mean β_0 and shape parameter r. Variability in transmission coefficients among individual juvenile salmon within a school may occur due to heterogeneity in host susceptibility and (or) small-scale patchiness in the distribution of copepodites due to swarming (Murray 2002) and leads to overdispersion of parasites among hosts. We assumed that infestation occurred as a Poisson process, which (with the Gamma-distributed transmission coefficient) gave rise to a Gamma–Poisson process that captures this overdispersion (Greenwood and Yule 1920) and is equivalent to a

negative binomial distribution of sea lice among hosts. Previous models have assumed that sea lice are evenly dispersed among hosts according to the Poisson distribution with constant rate parameter (Krkošek et al. 2005*a*, 2006*a*). Details of the infestation model are given in Appendix A.

Model fitting

The sea louse transmission model was fit to data collected in BA on the west coast of British Columbia, Canada (Fig. 2), in 2006. In this region, wild juvenile pink and chum salmon migrate through narrow fjords to the open ocean each spring, and in the year of the study, these migrating salmon passed by several active salmon farms. The model was fit to data of sea louse abundances on both farmed and wild salmon in two steps.

First, we obtained counts of motile-stage *L. salmonis* on salmon from three farms along the Knight Inlet – Tribune Channel migration corridor of the BA (Fig. 2) and were active in 2006. The number of motile lice on individual farmed salmon was available from November 2005 through June 2006 from a previous study of sea louse dynamics on Farm 1 (n = 1659 fish sampled) and Farm 2 (n = 1080 fish sampled) (Krkošek et al. 2010). For Farm 3, we had the total number of motile lice per sampling event (which included 20 fish), totalling 39 sampling events from July 2005 through June 2006 (Cohen Commission 2011). Sea louse counts were done at irregular intervals of anywhere from 7 to 44 days, with more frequent sampling during the juvenile salmon outmigration (Fig. 3).

We fit the model of exponential growth and decay of motile lice on farmed salmon eq. 1 to counts of sea lice on each farm separately. The different format of the data for Farms 1 and 2 versus Farm 3 required slightly different assumptions in the statistical analyses; for Farms 1 and 2 we assumed that the number of lice per fish was distributed according to the negative binomial with mean predicted by eq. 1 and overdispersion parameter k to be estimated, and for Farm 3 we assumed normally distributed error between the mean number of lice per fish from the 20-fish sample and the model prediction, with the residual variance to be estimated. Maximum likelihood estimates for growth rate parameters (r_1 and r_2) and the average number of lice per farmed salmon at the time of treatment (f_0) were then used to simulate a distribution of infectious copepodites originating from all three salmon farms throughout the migration corridor for the period of the juvenile salmon migration, using the advection–diffusion–decay processes described by eq. 2 (with details in Appendix A).

We fit the model of infestation and development of sea lice on wild juvenile salmon (eqs. 4–5) to spatiotemporal data of sea louse abundance on wild juvenile salmon. Wild juvenile pink and chum salmon were collected by beach seine along 60 km of the Knight Inlet – Tribune Channel corridor (Fig. 2). At each site, a maximum of 100 pink salmon and 100 chum salmon were live-assayed for sea lice (Krkošek et al. 2005*a*, 2005*b*). Fish collection and examination protocols were approved by the University of Alberta Animal Care Committee and carried out in accordance with the Guide to the Care and Use of Experimental Animals (www.ccac.ca). A total of 128 sites were sampled from 10 April – 22 May 2006, totalling 6593 pink, 6016 chum salmon samples, and 6428 associated *L. salmonis*. The surface water temperature at sample locations ranged from 7 to 16 °C (mean 9.5 °C), and salinities ranged from 9‰ to 33‰ (mean 28.3‰). Some of these data have been analysed previously as part of a larger project involving sea louse data from several sources (the Broughton Archipelago Monitoring Program; Rees et al. 2015; Patanasatienkul et al. 2015; Cox et al. 2017).

By assuming infestation occurred as a Gamma–Poisson process (see Appendix A), we were able to assign probabilities to each of our observations of sea lice on wild juvenile salmon and calculate the likelihood of these data given a parameter set. Maximum likelihood estimates for both the growth and decay of sea louse populations on farmed salmon and transmission of sea lice to wild salmon were obtained using a statistical tool called data cloning (Lele et al. 2010, 2007). Briefly, data cloning applies the Markov chain Monte Carlo (MCMC) algorithm in a Bayesian framework to obtain maximum likelihood parameter estimates (Lele et al. 2007). Owing to the complexity of the model and potential for parameters to be confounded and thus nonidentifiable or nonestimable given our data, we also investigated the estimability of model parameters using data cloning (Peacock et al. 2017). If parameters are estimable, the theory of data cloning posits that the

Fig. 3. Growth and decay of sea louse populations on three salmon farms before and after parasiticide treatments: (*a*) Farm 1 at x = –3.7 km, (*b*) Farm 2 at x = 4.0 km, and (*c*) Farm 3 at x = 53.0 km. Black lines are the model predictions for $f(t)$ from eq. 1, with grey zones indicating the bootstrapped 95% confidence intervals on model predictions. Open points are the average lice per farmed salmon ± 95% bootstrapped confidence intervals. Vertical dashed lines indicate treatment dates. Note that points in panel (*c*) are average lice per fish per pen, not counts of number of lice per fish. Corresponding parameter estimates are in Table 1.

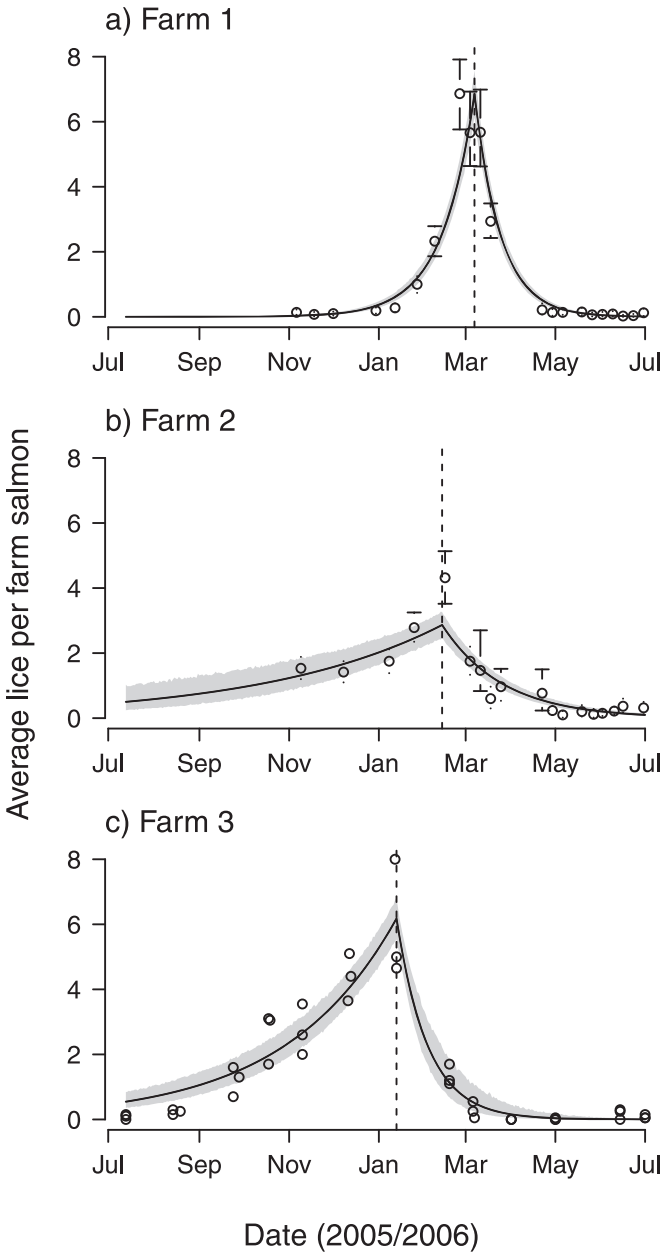
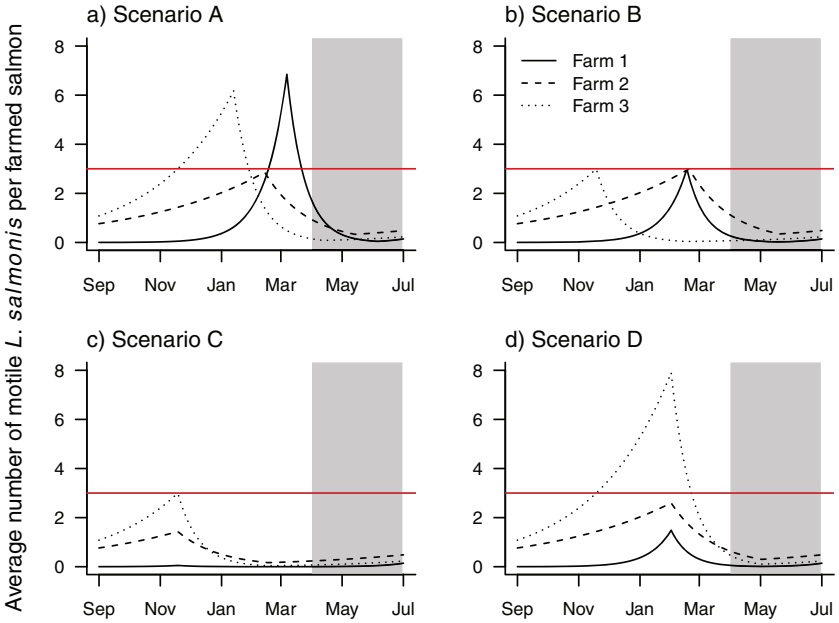


Fig. 4. The average number of motile *L. salmonis* per farmed salmon on three salmon farms under four different treatment scenarios: (*a*) independent treatments on the observed date, (*b*) independent but immediate treatment when threshold is reached, (*c*) coordinated treatment of all farms when the first farm reaches the threshold, and (*d*) coordinated treatments of all three farms prior to the juvenile salmon migration. The horizontal (red) line indicates the treatment threshold of three motile lice per farmed salmon (Fisheries and Oceans Canada 2018). [Colour online.]



variance in the posterior distribution should decline at a rate of $1/K$ when the likelihood is raised to the power K (or, equivalently, the data are "cloned" K times; Lele et al. 2010). We implemented data cloning in R (R Core Team 2018) using the software JAGS (Plummer 2003) and package dclone (Sólymos 2010). In fitting the transmission model to sea louse data from wild salmon, we used the simulated distribution of infectious sea lice from salmon farms over a 150-day period from 1 January 2006 to 31 May 2006 with a time-step of 0.05 days and over a 140 km long migration corridor (Fig. 1) with a grid space of 0.05 km. This grid was sufficient to cover the period and locations of wild salmon sampling for model fitting. Details of the model fitting methodology and results are provided as Supplementary material online[1] and R code is available (see section on Data Accessibility).

The free parameters that we estimated were the background louse density (κ), the fecundity–dilution parameter controlling the rate of production of nauplii at farm locations (ϕ), juvenile salmon migration speed (v), survival of copepodites and chalimus to the next stage (s_c and s_h, respectively), and the shape parameter (i.e., dispersion parameter of the negative binomial distribution, r; Fig. 1; Table A1). We were unable to estimate the mean transmission coefficient β_0 because we lack data on planktonic sea louse densities, and thus this parameter was confounded. Therefore, we could only estimate the parameter groupings of $\beta_0\kappa$ and $\beta_0\phi$ representing the background infestation pressure and farm infestation pressure. The survival of attached lice on pink and chum salmon was not assumed to be the same because, for example, the immune response may differ between host species (Jones et al. 2007; Sutherland et al. 2014). To consider the impact of this on the attachment process, we also fit a model allowing $\beta_0\kappa$ to differ for pink and chum hosts, but the estimates were not significantly different between host species. All other parameters were the same for both pink and chum salmon.

Simulations

We used the parameterized model to explore the effect of the timing of treatments relative to the wild salmon migration and relative to treatments on other farms on sea louse infestations of juvenile pink salmon in a simulation framework. Previous studies of farm networks have found that the timing of treatments among salmon farms influences the rate of sea louse population recovery and thus the frequency of treatments needed within a production cycle (e.g., Revie et al. 2005; Peacock et al. 2016). However, the influence of treatment timing relative to juvenile salmon migration has not been investigated (although see Bateman et al. 2016), and we aimed to understand whether coordinated treatments are beneficial for wild salmon and, if so, when treatments should occur. We investigated four different treatment scenarios (Fig. 4):

(A) independent treatments on farms at the observed date (Fig. 3, Fig. 4*a*),
(B) independent but immediate treatment of each farm when the louse abundance reached the treatment threshold of three motile lice per fish (Fisheries and Oceans Canada 2018; Fig. 4*b*),
(C) coordinated treatments of the three farms when the first farm reaches the treatment threshold on 18 November 2005 (even though the two other farms are below the threshold at that time; Fig. 4*c*), and
(D) coordinated treatments of all three farms on 1 February 2006 prior to the juvenile salmon outmigration (Rogers et al. 2013), even if it means delaying treatment of some farms after they have reached the threshold (Fig. 4*d*).

For each scenario, louse abundances on the three farms were simulated using the growth rates r_1 and r_2 estimated for each farm (Table 1). For each farm, simulations started at the predicted louse abundance on 1 September 2005 (Fig. 3) and ran to 1 July 2006, with one treatment per farm at the date specified by the scenario (Fig. S5[1]). We assumed that treatment efficacy lasted 90 days (Saksida et al. 2010), after which time the growth rate changed from r_2 back to r_1 (Rogers et al. 2013).

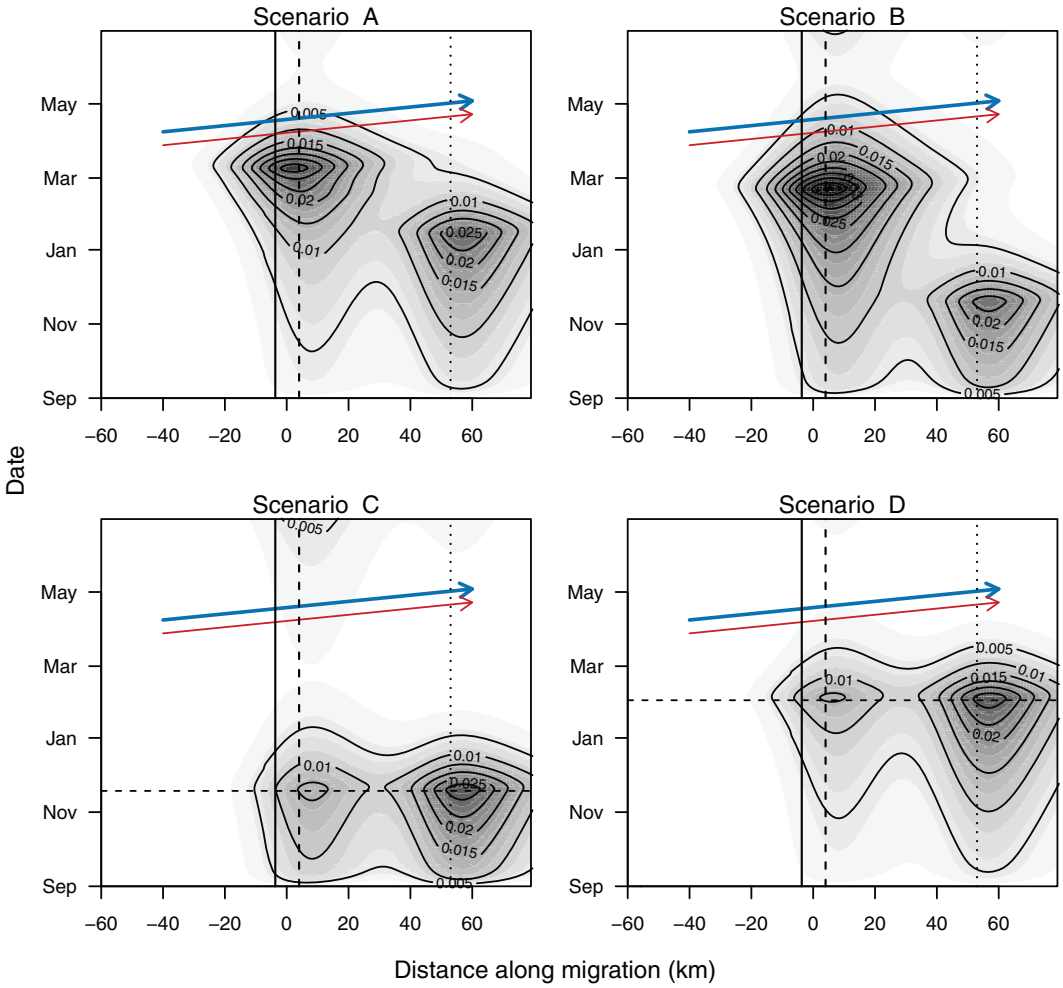
The migration path taken by a juvenile salmon through the simulated density of infectious larvae will influence the infestation pressure they encounter and the intensity of the resulting

Table 1. Parameter estimates (±95% confidence intervals) for eq. 1 fit to lice counts on farm salmon at three different salmon farms.

	Farm 1	Farm 2	Farm 3
r_1	0.045 (0.041, 0.048)	0.008 (0.005, 0.011)	0.013 (0.011, 0.016)
r_2	−0.056 (−0.06, −0.053)	−0.024 (−0.027, −0.022)	−0.048 (−0.062, −0.033)
f_0	6.848 (6.121, 7.574)	2.861 (2.444, 3.279)	6.177 (5.524, 6.83)
k or σ*	1.547 (1.231, 1.863)	1.284 (0.986, 1.582)	0.638 (0.494, 0.781)

*For Farm 1 and Farm 2, k is the dispersion parameter of the negative binomial distribution, whereas for Farm 3, σ is the residual standard deviation of Gaussian errors.

Fig. 5. The simulated densities of infectious copepodites (darker = higher density) along the 1D migration corridor during 2005–2006 under four treatment scenarios. The x axis is the Knight Inlet – Tribune Channel migration corridor (Fig. 2) from −60 to 80 km. The y axis is time from 1 September 2005 to 1 July 2006. The locations of Farm 1, Farm 2, and Farm 3 (Fig. 2) are indicated by vertical solid, dashed, and dotted lines, respectively. For Scenarios C and D, the treatments on farms were coordinated, with the single treatment date indicated by the horizontal dashed line. The thick (blue) and thin (red) arrows show wild juvenile salmon migration routes under normal (solid blue point) and early (open red point) migration timing, respectively. When calculating metrics, we used 1000 such migration paths starting at different points in time to capture the uncertainty emergence time of juvenile salmon migrating from Glendale River. [Colour online.]



infestation (Fig. 5). We estimated the migration path of salmon based on data of emergence timing from Glendale River, the major salmon-bearing river in BA located at approximately −40 km along our migration corridor (Fig. 2). We obtained dates of emergence of pink salmon fry from Glendale River from 2007 to 2012 from the Glendale Creek Juvenile Downstream Program (Pieter vanWill, DFO, personal communication). In 2010, pink salmon fry seemed to emerge about 2 weeks earlier than in other years (Fig. S1[1]), and early migration timing was anecdotally reported to have exacerbated sea louse exposure of juvenile salmon in the 2015 outbreak in BA (Bateman et al. 2016). To account for variability in migration timing, we considered an "early" migration scenario in which the population of juvenile salmon emerged as observed in 2010 and "normal" migration timing based on emergence data for 2007–2009 and 2011–2012 (Fig. 5). Within each of those scenarios, we incorporated stochasticity in migration timing by resampling with replacement from the emergence dates of fry (Fig. S1[1]) 1000 times, yielding 1000 different migration paths through the spatiotemporal distribution of infectious sea lice. All migration paths start at −40 km along the migration route (Glendale River; Fig. 2). We assumed the migration speed of the juvenile salmon was constant at the speed estimated in the model fitting.

Table 2. Maximum likelihood parameter estimates (95% confidence intervals) for the model fit to lice counts on wild juvenile salmon from the Knight Inlet – Tribune Channel migration corridor of the Broughton Archipelago (Fig. 2) in April and May 2006.

Parameter	Description	Estimate (95% CI)
$\kappa\beta_0$	Background infestation pressure	0.010 (0.009, 0.011)
$\phi\beta_0$	Farm infestation pressure	4.79 (4.41, 5.21)
v	Migration speed	4.09 (3.79, 4.40)
s_c^{pink}	Survival of copepodites on pinks	0.95 (0.70, 0.99)
s_h^{pink}	Survival of chalimus on pinks	0.46 (0.41, 0.52)
s_c^{chum}	Survival of copepodites on chums	0.78 (0.69, 0.85)
s_h^{chum}	Survival of chalimus on chums	0.29 (0.25, 0.33)
r	Overdispersion parameter for negative binomial	0.59 (0.54, 0.65)

We summarized the effect of treatment timing on wild juvenile salmon using three metrics: (*i*) total infestation pressure encountered along the migration, (*ii*) the maximum expected number of sea lice per juvenile salmon, and (*iii*) the expected mortality of juvenile pink salmon due to infestation. For each migration path we simulated, we calculated the overall infestation pressure as the line integral of the migration path over the spatiotemporal distribution of infectious copepodids. The maximum number of lice per juvenile salmon was the maximum sum of copepodid, chalimus, and motile lice at any point along the migration path. We calculated the expected host mortality using previous estimates of louse-induced mortality from Peacock et al. (2013). That study used a time series of the mean number of sea lice (copepodid, chalimus, and motile stage) at three locations in BA that have been monitored since 2001 (Peacock et al. 2016) together with salmon spawner and recruitment data over 60 years to estimate the per-sea-louse mortality rate, c, for pink salmon populations. To estimate population-level mortality under our four treatment scenarios, we simulated the mean louse abundance at those same three monitoring locations on the migration route. We calculated the mortality of wild salmon due to sea lice per generation of salmon as $1 - e^{-cL}$ (Krkošek et al. 2011*b*), where $c = 0.190$ is the estimated louse-induced mortality from Peacock et al. (2013) and L is our simulated mean louse abundance. We report the 2.5%, 50%, and 97.5% quantiles of all three metrics from the 1000 migration paths for both early and normal migration timing. R (R Core Team 2018) code reproducing simulations is available online (see Data Accessibility section).

Results

Farm dynamics

The three salmon farms under study showed clear patterns of exponential growth of louse populations until treatment dates, after which louse populations declined (Fig. 3). Growth rates and average lice per fish at time of treatment were in agreement with previous estimates (Krkošek et al. 2010; Table 1); slight differences were likely due to different assumptions about the statistical distribution of lice per fish. Krkošek et al. (2010) assumed lice were Poisson-distributed, whereas we found lice were overdispersed on their hosts, and the negative binomial was a better fit to the data.

Transmission to wild salmon

The production of sea louse copepodites at salmon farms was around 2400 times greater (95% CI: 2311–2525) than background sources, assuming a farm footprint of 0.2 km (i.e., $\phi/(0.2 \times \kappa)$; Krkošek et al. 2005*a*). Dispersal and mortality resulted in relatively low densities of infectious sea lice along the migration route (i.e., $L_F(x, t) \ll 1$; Fig. 5) and a maximum infestation pressure from farms that was 30 times greater than background sources ($\max_{x,t} \phi L_F(x, t)/\kappa$). This maximum occurred down-current from Farm 1 between 0.65 and 0.85 km along the migration on 9 March 2006, just 2 days after Farm 1 treated with EMB and prior to the treatment of Farm 2 (Fig. 3). Overall, the infestation pressure from farms was greater than that from background sources for 12 500 km × days, covering most of the migration corridor from January through May (Fig. S6[1]).

As well as identifying the sources of sea lice, parameter estimates give insight into both the life history of lice, survival rates, and differences in susceptibility of salmonid hosts to infestation (Table 2). We found that the background infestation pressure, $\kappa\beta_0$, was the same between pink and chum salmon, suggesting there was no difference in the susceptibility of those host species to initial infestation. Indeed, the data show similar numbers of copepodites on both host species; however, the survival of both copepodite-stage and chalimus-stage sea lice was significantly higher on pink salmon than on chum salmon (Table 2). Previous studies have assumed that juvenile salmon migrate at $v = 1\ \text{km}\cdot\text{day}^{-1}$ (Krkošek et al. 2005*a*, 2006*a*), but we found this parameter was much higher at $v = 4.09$ (3.79–4.40) $\text{km}\cdot\text{day}^{-1}$.

Sea lice were overdispersed on their juvenile salmon hosts, distributed according to the negative binomial with an overdispersion parameter of $r = 0.59$ (0.54–0.65), significantly less than 1 (Table 2). Previous studies using similar models assumed a Poisson distribution (Krkošek et al. 2005*a*, 2006*a*), but we found the negative binomial fit the data much better despite adding an extra parameter (likelihood ratio test, $\chi^2 = 1249$, df = 1, $p < 0.001$).

Model predictions captured the main peaks in infestations of juvenile salmon, especially for chalimus-stage lice (Fig. 6*b*). However, the model underpredicted sea louse abundance on juvenile salmon towards the end of the migration route, particularly for motile-stage lice (Fig. 6*c*). Data cloning showed that the parameters in both the farm model and the spatiotemporal infestation model for wild salmon were clearly estimable given the available data (Figs. S2 and S5[1]).

Simulations

The infestation pressure, maximum number of sea lice, and estimated mortality of juvenile salmon due to sea lice were all lower for treatment scenarios that coordinated among farms (i.e., scenarios C and D; Fig. 7). For Scenarios A, B, and D, early migration timing resulted in higher numbers of lice and higher mortality (Fig. 7), as salmon migrated closer in time to the peak infestation pressure at treatment (Fig. 5). Mortality did not increase with early migration timing for Scenario C where treatment was administered in November, far in advance of the juvenile salmon migration. Even though our simulations incorporated a recovery in sea louse population growth rates on farms 3 months after treatment, and louse populations in Scenario C had started to recover by the time the juvenile salmon migrated past, lice did not reach high enough numbers on farms to result in significant infestations of wild salmon (Fig. 5).

Fig. 6. The abundance of (*a*) copepodid, (*b*) chalimus, and (*c*) motile sea lice per wild juvenile pink salmon along their migration corridor (x, km) from 10 April 2006 (day t = 100) to 24 May 2006 (day t = 144). The grey surface is the model prediction using fitted parameter estimates (Table 2). Points are the observed mean number of copepodid, chalimus, or motile sea lice on juvenile salmon (±95% bootstrapped confidence intervals). Dark blue points indicate observations that are higher than model predictions, and light red points indicate observations that are lower than model predictions. Arrows indicate 95% confidence intervals that extend beyond the plotting region. See Fig. S7[1] in the online Supplement for fits to chum salmon data. [Colour online.]

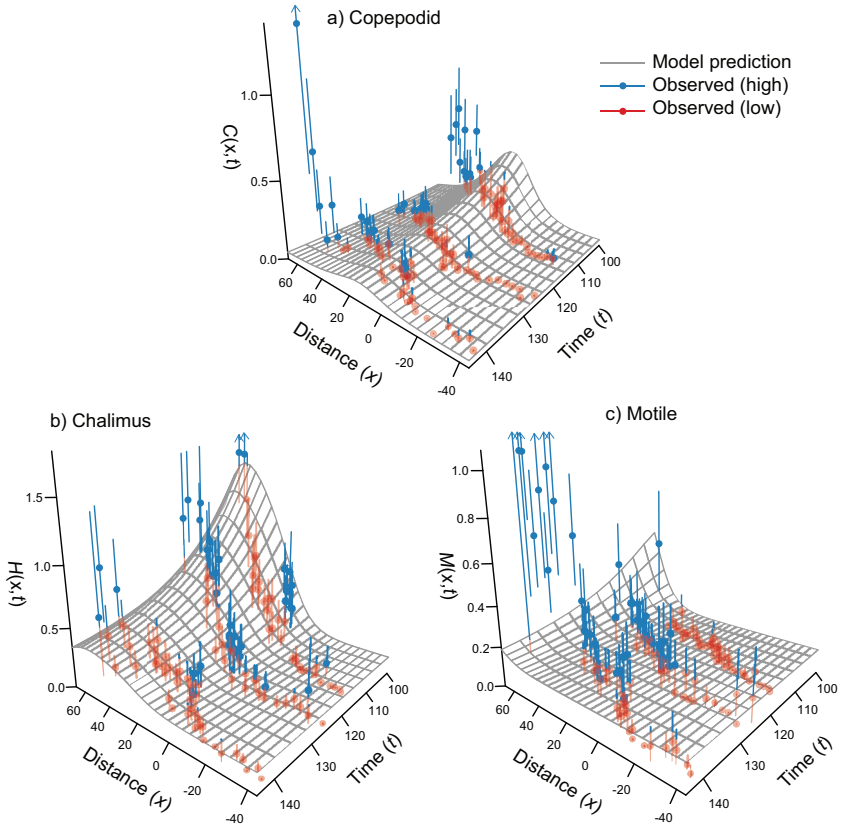
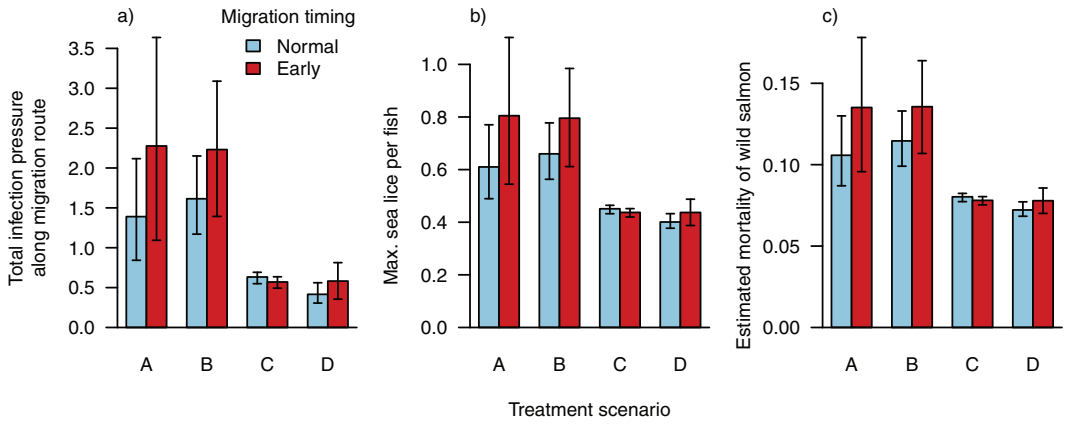


Fig. 7. Three metrics of wild salmon health calculated over four treatment scenarios (A–D) under normal (light blue) and early (dark red) migration timing of juvenile salmon. Metrics are (*a*) total infestation pressure encountered by a juvenile salmon along their migration, calculated as the line integral over the distributions in Fig. 5, (*b*) maximum number of lice (copepodite, chalimus, and motile stages) attached to a juvenile salmon during their migration, and (*c*) the mortality of wild salmon due to sea lice, calculated as $1 - e^{-cL}$, where c is the estimated per-sea-louse mortality rate for the generation of salmon (Peacock et al. 2013), and L is the mean sea louse abundance on out-migrating juvenile salmon, calculated here from simulations. See main text for details.



Discussion

The temporal dynamics of sea lice on salmon farms have been well studied in relation to management and environmental variables (e.g., Revie et al. 2003, 2005; Rogers et al. 2013), but there have been few attempts to connect those dynamics with empirical data on wild juvenile salmon migrating through those areas using mechanistic models. In this study, we empirically modelled the temporal dynamics of sea louse populations on farmed salmon and the consequences for the infestation and development of sea lice on juvenile wild pink and chum salmon in BA, which has been

a major salmon farming region in Pacific Canada. Since 2006, salmon farms in BA have been more proactive in treating farmed salmon for lice to ensure low prevalence during the juvenile salmon out-migration (Peacock et al. 2013), but recent outbreaks highlight the challenges to successful proactive treatments given variability in sea louse population growth rates and in the migration timing of juvenile salmon (Bateman et al. 2016). Our fitted model suggested that in 2006, the infestation pressure from salmon farms was 24 times greater than that from background sources of sea lice, such as returning wild adult salmon. Although this result is not new — many studies have identified salmon farms as the main source of sea lice on wild juvenile salmon in regions of salmon farming (e.g., Krkošek et al. 2005*a*, 2006*b*; Marty et al. 2010) — there are several aspects of this study that advance our understanding of the system in an important way.

Previous models describing observations of sea lice on juvenile salmon assumed that sea louse production at salmon farms was constant (Krkošek et al. 2005*a*, 2006*a*). Sea louse populations on three salmon farms that were active in 2005–2006 in BA showed clear patterns of exponential growth and decay, with louse abundances peaking just prior to treatments of farmed salmon. The temporal dynamics of louse populations on these farms in 2006 suggest that treatments were effective at reducing sea louse abundances on farmed salmon; however, the timing of the treatment could have been more precautionary to prevent peaks in louse numbers as juvenile salmon migrate past farms (Rogers et al. 2013). For example, the peak in louse production occurred on 9 March 2006, close to when juvenile salmon began their migration. The two farms that were treated in January were minor sources of lice at that time. Including the temporal dynamics of sea louse infestation pressure from farms allowed us to more accurately assess the importance of salmon farms as sources of infestation for wild salmon, but also to investigate other potential treatment strategies using a simulation approach.

Our estimates of louse-induced mortality from the true treatment schedule were on par with previous estimates of 8.3%–22.3% (mean 15.9%) from an independent data set (cf. figure 6 of Peacock et al. 2013). Using our parameterized model, we were able to explore how that mortality might have changed if farms had treated differently. We found that coordinated treatment among salmon farms and early precautionary treatments would have reduced sea louse abundance and minimized sea-louse-induced mortality of wild salmon. Our results likely underestimated the importance of coordinated treatments, as we did not account for transmission and infestation among salmon farms that could hinder area-wide control if management is not coordinated (Bateman et al. 2016). Early treatment was especially important when the migration timing of juvenile salmon was earlier than usual. Emergence of juvenile salmon has been shown to advance with warmer temperatures (Holtby et al. 1989), and thus earlier migration timing may become the norm under climate change. In Pacific Canada, current license conditions require that salmon farms take management action when louse populations exceed an average of three motile lice per farmed salmon (Fisheries and Oceans Canada 2018), but there is no formal area-wide management plan that requires coordinated treatment among farms. Early treatment would seem to be optimal after the influx of lice with returning wild salmon in the fall and only if farms coordinate to avoid reinfestation prior to the juvenile salmon migration. This coordination is particularly important to minimize infestation of juvenile salmon at the start of their migration, so that the migrating juvenile salmon themselves do not spread sea lice among farms (Krkošek et al. 2006*a*). These benefits of early intervention would have to be weighed against drawbacks of potentially more frequent treatments throughout a production cycle, such as cost and increased opportunity for sea lice to develop resistance to treatments.

Although we have addressed several shortcomings of previous modelling efforts, there remain assumptions and caveats to our model. The estimability of parameters in previous models of sea louse transmission (Krkošek et al. 2005*a*, 2006*a*) was not clear from published point estimates. The original models attempted to estimate 14 free parameters, and we found many of these parameters were not estimable given the available data. Using data cloning (Lele et al. 2010), we identified which parameters were estimable and simplified the model to seven free parameters. This involved making some assumptions and fixing some parameters (e.g., the developmental times of louse stages) and assuming other parameters did not differ between pinks and chum salmon (e.g., the background infestation pressure). Although these added assumptions may weaken the generality of the model, the simplified model was a better fit to the data. In this particular case, the main conclusion that salmon farms are a main source of sea lice on juvenile salmon was unchanged when the model was improved to ensure estimability. This may not always be the case, and we highlight that considering parameter estimability is important when fitting mechanistic models to data (Peacock et al. 2017), especially if the results have implications for the conservation and management of endangered or keystone species.

Our model greatly simplifies the processes driving the dispersal of sea lice from salmon farms. More complex hydrodynamic models have considered ocean currents and sea louse dispersal in three dimensions (Foreman et al. 2009; Stucchi et al. 2011; Cantrell et al. 2018), which undoubtedly yields more precise predictions of infestation pressure but is dependent on the wind, tide, and freshwater forcing of the specific time period being modelled. Our approach, although simpler, yields more generalizable insights into infestation patterns and has been shown to capture the major spatial patterns in infestation (Krkošek et al. 2005*a*, 2006*a*). Further, we were able to fit the model to data and infer parameters. One exciting area for future research would be to bridge these two approaches and confront the more complex simulation models with spatiotemporal data of infestations on wild salmon, although there are substaantial computational challenges involved.

Our model appears to do a poor job of predicting lice on juvenile salmon towards the end of their migration route, failing to capture rises in all lice stages, but particularly in motiles, in the 40–60 km range (Fig. 6). The more complex hydrodynamic models of BA have suggested that salmon farms north of Farm 3 (Fig. 2), which were not modelled in this study, may be major contributors of infestation pressure in the area of Farm 3 (Cantrell et al. 2018). Sea lice emitted from these more northern farms may be driving the higher-than-predicted infestations of wild salmon later in the migration.

Several other simplifying assumptions in our model may explain the discrepancy towards the end of the migration. For example, it is possible that sea lice develop to sexual maturity on juvenile salmon, and this may act as a third source of infestation pressure that we did not account for. The generation time of *L. salmonis* is 4–8 weeks (depending on temperature; Costello 2006) and juvenile salmon may take up to 3 months to complete their migrations (Krkošek et al. 2009). Reproduction of sea lice on wild salmon has been accounted for before (Krkošek et al. 2006*a*), but given the complexity of this model already, we could not include it. Once again, a simplification had to be made to ensure that the model did not outstretch the data.

Our assumption of a constant migration speed along the course of the juvenile salmon migration may also have contributed to the poor fit of the model towards the end of the migration. If salmon slowed their migration around certain farms, the infestation pressure may be effectively higher because of increased exposure time. Previous estimates for the migration speed of juvenile salmon were ~1 km·day^{-1} (Krkošek et al. 2006*a*; Morton et al. 2010), but our results suggest juvenile salmon migrate four times as fast. There are several reasons to believe that migration speed is

not constant. In the simplest case, migration speed may increase as the salmon grow from ~20 mm at ocean entry to over 100 mm towards the end of our study period. Acoustic tagging studies have shown considerable variability in the migration speed of juvenile salmon. For example, Welch et al. (2011) report a standard deviation in migration speed of juvenile coho (*Oncorhynchus kisutch*) of ±4.93 body lengths per second, or roughly 10–40 km·day^{-1} for fish between 30 and 100 mm. More complicated dynamics may arise if migration speed depends on sea louse infestation intensity; juvenile salmon may slow their migration as they acquire more lice (Nendick et al. 2011; Brauner et al. 2012). In such a scenario, juvenile salmon may get caught in infestation hotspots (Altizer et al. 2011; Peacock et al. 2018), and infestation pressure in those areas may increase out of proportion to the densities of sea louse larvae. There have been no direct studies of the routes and timing of migration for juvenile salmon in BA to assess support for these hypotheses, but such biological complexity may mean that our assumption of constant migration speed misses key aspects of the host–parasite dynamics.

We assumed that all parameters, not just migration speed, were constant over the period of juvenile salmon outmigration. It is well established that many of these parameters can vary — in particular development times and survival rates of sea lice are known to depend on temperature and salinity, respectively (e.g., Fig. S8[1]). Development is slower at colder temperatures (Stien et al. 2005; Costello 2006), and temperature generally increased throughout our study period (Fig. S8*a*[1]). However, in the spatial dynamics of the model, the development times of attached lice are multiplied by the migration speed of juvenile salmon (eqs. 4*a*, 4*b*, 4*c*), and migration speed may also have been increasing throughout the study as fish grow. Thus, faster sea louse development associated with higher temperatures in combination with faster migration speeds of hosts may lead to the same overall spatial dynamics of infestation. However, the development of pre-infective-stage larvae is predicted to have ranged from 2.6 to 5.3 days given the range in temperature observed during our sampling (Fig. S8*a*[1]), and this may have introduced errors into our calculation of the farm footprint and infestation pressure on wild salmon. Although it must be acknowledged, the uncertainty introduced by fluctuating temperatures within the range that we observed is not substantially greater than the uncertainty in the parameter estimates themselves (Stien et al. 2005).

Salinity is also known to affect sea louse population dynamics with the survival of attached lice declining below 15 psu (Johnson and Albright 1991; Connors et al. 2008) and pre-infective stages being even more sensitive (Bricknell et al. 2006; Groner et al. 2016*a*). During this study, only three out of 128 sampling events for juvenile salmon had salinities below 15 psu, and two of these occurred at adjacent sites on the same day and may be considered an anomaly (Fig. S8*b*[1]). Although salinities were frequently below 29 psu throughout our study, the concentration below which attachment success has been shown to decline (Bricknell et al. 2006), there were no obvious spatial or temporal patterns in salinity in our data (Fig. S8*b*[1]). Thus, the impact of salinity on attachment success would be averaged and included in the transmission coefficient (β_0) within the farm and background infestation pressures. Including relationships between temperature and developmental times or survival and salinity, such as those described by Groner et al. (2016*a*), may increase the realism of our model but are not likely to have changed the main results.

Some of our parameter estimates were somewhat surprising in light of laboratory studies and previous models of sea louse transmission in BA. We found that sea lice apparently survive better on pink salmon than on chum salmon, in contrast with laboratory studies suggesting that pink salmon mount a more effective immune response (Jones et al. 2007; Sutherland et al. 2014). In the field, some studies have found higher apparent survival on chum salmon (Morton et al. 2010), whereas others are inconsistent in which host species sea lice survive better on (Krkošek et al. 2006*a*). This uncertainty highlights the differences between laboratory and field studies and the need to consider ecological effects of sea louse infestation (e.g., Krkošek et al. 2011*a*) as well as physiological (Brauner et al. 2012). For example, predation by coho salmon on both pink and chum salmon may alter the host–parasite dynamics in the natural environment (Peacock et al. 2014).

The importance of salmon farms in driving sea louse infestations of wild juvenile salmon has by now been well established (e.g., Bjørn et al. 2001; Krkošek et al. 2006*a*; Marty et al. 2010). The more critical problem from a conservation standpoint is determining best practices for salmon farms to minimize impacts to wild salmon populations. By parameterizing a spatiotemporal model that connects management actions on farms to infestations of migrating wild salmon, we were able to explore different management scenarios in an empirically grounded framework. Our simulations suggest that precautionary treatments in advance of the juvenile salmon migration that reduce sea louse populations on salmon farms before they grow to critical levels and siting salmon farms as far away from the migration routes as possible will minimize the impact of farm-origin sea lice on wild pink and chum salmon. However, given recent reports of sea louse resistance to chemical treatments in the Pacific (Messmer et al. 2018), any management strategy will also have to consider the evolutionary consequences for resistance to ensure long-term viability.

More generally, this study demonstrates the importance of considering spatial and temporal patterns in infection dynamics when attempting to understand and manage emerging infectious diseases in wildlife populations. Mechanistic models are a powerful tool for understanding and predicting complex ecological and epidemiological processes (White et al. 2018), but there are few examples where such models are fit to data to estimate parameters of the system. The development of new analytical and statistical approaches, such as the data cloning (Lele et al. 2007, 2010) we applied, have opened the door to fitting mechanistic models to complex ecological data sets, with the potential to deepen our understanding of even well-studied systems such as sea louse transmission between farmed and wild salmon.

Data accessibility

All code and data for this study can be accessed at https://github.com/sjpeacock/Spatiotemporal-infection-model/.

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Appendix A

Advection, diffusion, and decay of farm-source sea lice

Sea louse nauplii from salmon farms advect, diffuse, die, and develop according to eq. 2. The transient solution to the advection, diffusion, decay equation can be written in the form of a Green's function:

$$G(x,t) = \frac{1}{\sqrt{4\pi D t}}\exp\left[-(\mu_n + \theta)t - \frac{(x-\gamma t)^2}{4Dt}\right] \tag{A1}$$

To account for the dynamic production of lice at farms, we calculated the convolution of eq. A1 with the point source forcing function eq. 1 multiplied by the Dirac delta function, $\delta(y)$, for a farm located at y. This assumes that lice are produced at exactly the farm location, with production proportional to the number of motile adult lice at that farm, and then disperse according to the advection–diffusion–decay process. The resulting distribution kernel of nauplii in space and time is

$$\begin{aligned} k_n(x,t) &= \int_0^t \int_{-\infty}^{\infty} G(x-\xi, t-\tau) f(\tau)\delta(\xi)\, d\xi d\tau \\ &= \int_0^t G(x, t-\tau) f(\tau)\, d\tau \\ &= \int_0^t \frac{1}{\sqrt{4\pi D(t-\tau)}}\exp\left[-(\mu_n+\theta)(t-\tau) - \frac{[x-\gamma(t-\tau)]^2}{4D(t-\tau)}\right] f(\tau)\, d\tau \end{aligned} \tag{A2}$$

These distributed nauplii then develop into infectious copepodites, which can attach to susceptible juvenile salmonids in the vicinity. To obtain the distribution of infectious copepodites, we consider the nauplii as a distributed source that develop and subsequently diffuse and advect, leading to a second convolution for the distribution of infectious copepodites:

$$k_c(x,t) = \int_0^t \int_{-\infty}^{\infty} G(x-\xi, t-\tau) k_n(\xi,\tau)\, d\xi d\tau \tag{A3}$$

where the parameters in $G(x, t)$ are the same as before except μ_c, the mortality rate of copepodites, replaces $(\mu_n + \theta)$. In practice, we calculated the solutions given by eqs. A2 and A3 numerically, applying a fast Fourier transform (FFT) algorithm to ease the convolution step. We assume that the probability of successfully finding a host is low, and so we ignore removal of planktonic copepodites through attachment. This solution, $k_c(x, t)$, is proportional to the infestation pressure on wild juvenile salmon from copepodites originating from salmon farms. We did not know the transmission coefficient (i.e., the proportion of infectious copepodites that attach to a host), and so we considered only relative densities of copepodites and normalized $k_c(x, t)$ so the area under the highest peak of $k_c(x, t)$ in time equalled one. We also assumed the distributions originating from different farms were independent, and therefore the total farm-source copepodite density, $L(x, t)$, was the normalized sum of the copepodites originating from each of the three farms:

$$L_1(x,t) = \frac{\sum_{i=1}^{3} k_{c,i}(x,t)}{\max_{i,j}\left[\int_{-\infty}^{\infty} k_{c,i}(u,j)du\right]} \tag{A4}$$

Table A1. List of parameters and variables referred to throughout the text.

Type*	Symbol	Description	Equation†	Value and (or) unit
Variable	$f(t)$	The expected number of sea lice per farmed salmon at time t	1	motiles·fish^{-1}
Parameter	f_0	The average number of motile sea lice per farmed salmon at the time of treatment	1	motiles·fish^{-1}
Parameter	r_1, r_2	Rates of exponential growth and decay of lice on farmed salmon	1	day^{-1}
Parameter	k, σ	Dispersion parameter (k, for Farm 1 and Farm 2) or residual standard deviation (σ, for Farm 3) for modelling lice dynamics on farms	Table 1	
Variable	$n(x, t)$	The density of nauplii at kilometre x and day t	2	
Constant	D	Diffusion coefficient	2	22.67 km^2·day^{-1}
Constant	γ	Advection coefficient	2	1.56 km·day^{-1}
Constant	μ_n	Mortality rate of nauplii	2	1/5 day^{-1}
Constant	μ_c	Mortality rate of copepodites	A3	4/5 day^{-1}
Variable	$L_1(x, t)$	Distribution of infectious copepodites from farm sources	A4 and 3	
Parameter	κ	Scale of background infestation pressure	3	copepodites·fish^{-1}
Parameter	ϕ	Scale of farm infestation pressure	3	
Variable	$L(x, t)$	Distribution of infectious copepodites from all sources	3	
Variable	$C(x, t)$	Expected number of copepodites per wild juvenile salmon	4	
Constant	β	Transmission coefficient	4	1
Constant	τ_c	Developmental time of the copepodid stage	4	3.6 days
Parameter	V	Migration speed of juvenile salmon	4	km·day^{-1}
Variable	$H(x, t)$	Expected number of chalimus per wild juvenile salmon	4	
Parameter	s_c	Survival of copepodites to chalimus stage	4	
Constant	τ_h	Developmental time of the chalimus stage	4	16.0 days
Variable	$M(x, t)$	Expected number of motiles per wild juvenile salmon	4	
Parameter	s_h	Survival of chalimus to motile stage	4	
Constant	τ_m	Developmental time of the motile stage	4	28.6 days
Parameter	r	Dispersion parameter in negative binomial distribution	A5	

*Type distinguishes variables from parameters and specifies which parameters are constant (i.e., not free).
†Equation is the equation in which the parameter or variable first appears.

where $k_{c,i}(x, t)$ is the copepodite density from farm i.

Infestation model

We model infection as a Gamma–Poisson process, where the transmission coefficient β is a Gamma random variable with mean β_0 and dispersion parameter r. The expected number of copepodites per juvenile salmon $C(x, t)$ is therefore also a Gamma random variable with mean $\beta_0 L_0$ and dispersion parameter r:

$$\text{(A5)}\qquad g(C;\, \beta_0 L_0, k) = C^{k-1}\left[\frac{k}{\beta_0 L_0}\right]^k \frac{\exp\left[-\left(\frac{k}{\beta_0 L_0}\right)C\right]}{\Gamma(k)}$$

where $L_0 = \int_0^{\tau_c} L(x - vu, t - u)du$ (eq. 5*a*). The number of copepodid sea lice on an individual fish, N_c, will be a random variable with probability density:

$$\text{(A6)}\qquad \Pr\{N_c = c\} = \int_0^\infty g(C;\, \beta_0 L_0, k)\frac{C^c}{c!}e^{-C}dC$$

$$\text{(A7)}\qquad = \frac{(c + k - 1)!}{c!(k - 1)!}\left(\frac{k}{\beta_0 L_0 + k}\right)^k\left(1 - \frac{k}{\beta_0 L_0 + k}\right)^c$$

which is the negative binomial distribution with mean $C(x, t)$ and dispersion parameter r. A count of h chalimus sea lice on an individual fish can result from any of i attached copepodites surviving to the chalimus stage with probability s_c. We define N_i as the discrete random variable for the number of attached copepodites available for recruitment into the chalimus stage on an individual fish at point (x, t). Therefore, the probability of having a fish with h chalimus sea lice is

$$\text{(A8)}\qquad \Pr\{N_h = h\} = \sum_{i=h}^\infty \left[\binom{i}{h}s_c^h(1 - s_c)^{i-h}\Pr\{N_i = i\}\right]$$

The distribution for N_i is

$$\text{(A9)}\qquad \Pr\{N_i = i\} = \int_0^\infty g(I;\, \beta_0 L_1, k)\frac{I^i}{i!}e^{-I}dI$$

$$\text{(A10)}\qquad = \frac{(i + k - 1)!}{i!(k - 1)!}\left(\frac{k}{\beta_0 L_1 + k}\right)^k\left(1 - \frac{k}{\beta_0 L_1 + k}\right)^i$$

yielding the probability of observing h chalimus sea lice as

$$\Pr\{N_h = h\} = \sum_{i=h}^\infty \left[\frac{i!}{h!(i - h)!}s_c^h(1 - s_c)^{i-h}\int_0^\infty g(I;\, \beta_0 L_1, k)\frac{I^i}{i!}e^{-I}dI\right]$$
$$= \binom{h + k - 1}{h}\left(\frac{k}{k + s_c\beta_0 L_1}\right)^k\left(1 - \frac{k}{k + s_c\beta_0 L_1}\right)^h$$

which is the negative binomial distribution with mean value $s_c\beta_0 L_1$ and dispersion parameter r, where $L_1 = \int_{\tau_c}^{\tau_c+\tau_h} L(x - vu, t - u)du$. Similar logic can be followed to arrive at the distribution for the number of motile lice per fish, giving formulas for the probabilities of observing numbers of copepodite, chalimus, and motile sea lice on individual fish. We assume that sea louse infestations on an individual fish are independent and that observations of sea lice on different fish at the same sample site are independent. Therefore, the likelihood of the data given a certain parameter set $\boldsymbol{\theta}$ is

$$\text{(A11)}\qquad \mathcal{L}(\text{data}|\boldsymbol{\theta}) = \prod_{i=1}^N \Pr\{N_c(x_i, t_i) = c_i\}\cdot\Pr\{N_h(x_i, t_i) = h_i\} \times \Pr\{N_m(x_i, t_i) = m_i\}$$

where N is the total number of fish sampled; c_i, h_i, and m_i are the observed number of copepodites, chalimus, and motile sea lice, respectively, on fish i; and x_i and t_i are the place and time that fish i was sampled.