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RESEARCH ARTICLE

Spatial patterns of sea lice infection among wild and captive salmon in western Canada

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Abstract
Context Parasite transmission between captive and wild fish is mediated by spatial, abiotic, biotic, and management factors. More effective population management and conservation strategies can result from multivariable assessments of factors associated with spatial dynamics of parasite spillover.
Objective Our study characterised spatial patterns of sea lice (*Lepeophtheirus salmonis, Caligus clemensi*) infection on out-migrating chum (*Oncorhynchus keta*) and pink (*O. gorbuscha*) salmon in an area with Atlantic salmon (*Salmo salar*) farming.
Methods A multivariable statistical model for sea louse parasitism of out-migrating chum and pink salmon was developed from 166,316 wild salmon sampled in the Broughton Archipelago, British Columbia, Canada from 2003 to 2012. We assessed for factors hypothesized to influence sea lice infection levels, at the non-motile life stage, including spatial scales of infection sources.
Results Fish length, sampling year and method were strong explanatory factors. Infection was greatest in higher salinity water. Farmed and wild juvenile salmon infection levels were correlated, on average, within 30 km. Except for 2004, sea lice infection on farms were typically well below the regulatory level (3 motiles per fish). Average intensity of non-motile infections observed on the wild fish were 6.36 (SD = 9.98) in 2004 compared to 1.66 (SD = 1.25) for the other years.
Conclusions Accuracy of future model estimates will benefit by including hydrodynamic data accounting for anisotropic spread of sea lice from sources. Multivariable statistical modelling over long time series data strengthens understanding of factors impacting wild juvenile salmon infection levels and informs spatial patterns of aquatic epidemiology.

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Introduction

Pathogen–host dynamics in populations depend on the frequency of interaction between hosts and pathogens (Anderson and May 1991). In general, more interactions lead to more infected individuals—though the frequency of these interactions often depends on the spatial scale and extent of the biotic (host and pathogen), abiotic and disease management factors (Real and Biek 2007; Gilligan and van den Bosch 2008). For example, spatial biotic effects are evident in chronic wasting disease (CWD), an infectious prion disease of ungulates, where higher rates of infection among more closely related individuals suggest that social grouping behaviour is driving local scale disease persistence (Cullingham et al. 2011). Spatial abiotic effects have been observed from parasitic protozoan infections of *Perkinsus marinus* in oysters, when a warming trend during the mid-1980's along the east coast of North America resulted in infection of more northerly populations as the cold water barrier to pathogen growth was lost (Harvell et al. 2002). Spatial management factors can occur in salmonid aquaculture when spatially targeted fallowing of salt water net pen sites is used to reduce pathogen loads in the area by decreasing host density and by breaking the infection cycle between year classes of fish (Werkman et al. 2011).

Pathogens of wild and captive aquatic populations provide a study system for understanding the spatial scale of these factors. Disease transmission among captive and wild populations can occur where stationary aquaculture net pens are sympatric with wild populations (Tully et al. 1999; Johansen et al. 2011), as is reported for the spread of sea lice from farmed Atlantic salmon (*Salmo salar*) to wild Pacific salmon (*Oncorhynchus* family) populations (Marty et al. 2010). Sea lice are recognised as important pests on the skin and gills of marine fishes including wild and farmed salmonids (Costello 2006). These copepods progress through a series of life stages, broadly summarised as planktonic (drifting mostly passively as nauplii and then infectious copepodids), non-motile (physically attached to hosts, via a filament, as copepodids that then molt into chalimus) and motile (limited movement on host as pre-adult then adults).

In British Columbia, Canada, more attention has been given to sea lice, *Lepeophtheirus salmonis* and *Caligus clemensi*, since 2002 because these parasites were reported at high prevalence on wild out-migrating chum (*Oncorhynchus keta*) and pink (*O. gorbuscha*) salmon smolts. This pattern was observed specifically in the Broughton Archipelago coastal region where there has been active salmon aquaculture since 1987 (Morton and Williams 2003; Beamish et al. 2006). Upon hatching from and leaving natal streams in late winter and early spring, chum and pink salmon spend the first few months of their life in estuarine and related nearshore habitats prior to migrating to the open ocean (Heard 1991; Salo 1991). These nearshore staging habitats seem to provide the necessary time for exposure to the infective stages of sea lice in the study area (Krkosek et al. 2009). Similar to ectoparasites of terrestrial animals, substantial infestation may be required before sea lice have a direct negative impact on the health of their hosts, but this depends on host body size (Tucker et al. 2002; Jones and Hargreaves 2009).

As a precautionary measure to reduce levels of infective lice stages in areas where large numbers of juvenile chum and pink salmon reside in the spring and summer months, the Province of British Columbia implemented the Broughton Archipelago Sea Lice Action Plan in March 2003, and later expanded it to encompass the entire coast as the sea lice management strategy (Saksida et al. 2011). Since October 2003, these programs require mandatory monitoring of the abundance of *L. salmonis* and *C. clemensi* (Saksida et al. 2011). Furthermore, farms are required to treat their fish if the mean abundance of *L. salmonis* is $\geq$3 motile lice per fish during 1 March to 30 June (http://www.pac.dfo-mpo.gc.ca/aquaculture/reporting-rapports/lice-pou-eng.html).

The sea lice monitoring program has provided opportunities to better understand the spatial scale of transmission between sea lice on farmed and wild fish populations. Krkošek et al. (2005a) estimated infection pressure from a single farm in the Broughton to elevate ambient levels on juvenile salmon up to 30 km. The authors modelled transmission dynamics using a spatial model of parasite dispersal, development, and

fish migration that was fit to data on sea lice abundances on wild salmon migrating through the Broughton Archipelago. Though an elegant approach, there are other significant factors known to influence sea lice levels on farmed and wild salmon, such as accounting for the distance of wild salmon to more than one fish farm (Saksida et al. 2011; Middlemas et al. 2012), size of farmed populations (Jansen et al. 2012), environmental factors such as salinity (Bricknell et al. 2006; Bravo et al. 2009), and differential immunity responses among the wild salmon species (Fast et al. 2002).

This study presents the most comprehensive spatial and temporal analysis for quantifying sources of sea lice infection on wild chum and pink juvenile salmon using 10 years of data and accounting for confounding biotic, abiotic and management factors. The objectives of this study were to (1) identify locations where wild juvenile chum and pink salmon were associated with high abundances of non-motile sea lice, (2) determine the spatial scale over which there was an association between sea lice levels of farmed and wild juvenile salmon, and (3) compare how sea lice abundance on the wild juvenile salmon varied given different levels of farm infection for conditions favourable to sea lice infection on the wild salmon. More effective population management and conservation strategies can result from a multivariable assessment of the key factors associated with the spatial dynamics of parasite spillover in aquatic communities.

Methods

Study area and data

The Broughton Archipelago is located in the coastal waters of British Columbia (50.7°N, 126.5°W). During the study period (2003–2012) this area contained 20 active Atlantic salmon farms. In 2003–2012, out-migrating chum and pink salmon were sampled and assessed for sea lice primarily from April to June, and to a lesser extent in March and July. Over this period, sampling occurred one or more times at 148 sites (Fig. 1). From 2003 to 2009 sampling was carried out using a non-lethal sampling and field assessment method by the Krkošek team (Krkosek et al. 2005b) and a lethal sampling and laboratory assessment method used by the Department of Fisheries and Oceans Canada (DFO) (Jones et al. 2006a). The live sampling protocol was unable to identify lice species, sex and developmental stage for sea lice in early life stages, as was possible using the lethal protocol (Jones et al. 2006a; Jones and Hargreaves 2007). Differences in the sampling protocols, methods and also sampling locations, may introduce biases for analysis and interpretation. To help address this, a collaborative research program, the Broughton Archipelago Monitoring Program (BAMP; http://www.bamp.ca), for which study data are available, was developed in 2010 with involvement of the salmon farming companies operating in the Broughton Archipelago, the DFO, university researchers, and the Coastal Alliance for Aquaculture Reform (CAAR). As part of the BAMP initiative a standard sampling protocol was developed that merged the spatial distribution of sampling effort between the two previous programs and used lethal lab-based analysis of lice on fish. From 2003 to 2012, data collection took place weekly, bi-weekly, or monthly. Two fishing gear types, beach and purse seines, were used to collect samples. Purse seine was only used by DFO from 2003 to 2009. Beach nets are shorter ($\sim$46 m), targeting captures at shallower depths ($\sim$3.7 m), while purse seine nets are longer (180–275 m) and capture fish at deeper depths ($\sim$16 m; Jones et al. 2006a). Typically, at each site, a maximum of 100 specimens of each species (Krkošek team) or 30 of each species (DFO and BAMP) were randomly selected from the sample. Fish were measured for fork length (mm; head to fork). When using the lethal sampling protocol, data were collected on lice count, developmental stage (non-motile: copepodid, chalimus; motile: pre-adult, or adult), species (*L. salmonis*, *C. clemensi*, or unknown), and gender (for the adult stages). For non-lethal sampling, lice were recorded to broad developmental stage, either non-motile or motile, and only motile lice were identified to species. To retain maximum data for our analyses, we classified sea lice life stage as non-motile and motile, and did not discriminate between lice species. All teams recorded the geographic coordinates of the sampling location and often measured the surface water (<1 m) temperature (°C) and salinity (practical salinity units [psu]). Data from the Atlantic salmon farms were provided by the British Columbia Salmon Farmers Association and included farm location (longitude, latitude), mean monthly fish inventory, motile sea lice counts, and surface water (<1 m) temperature (°C) and salinity (psu) measurements.

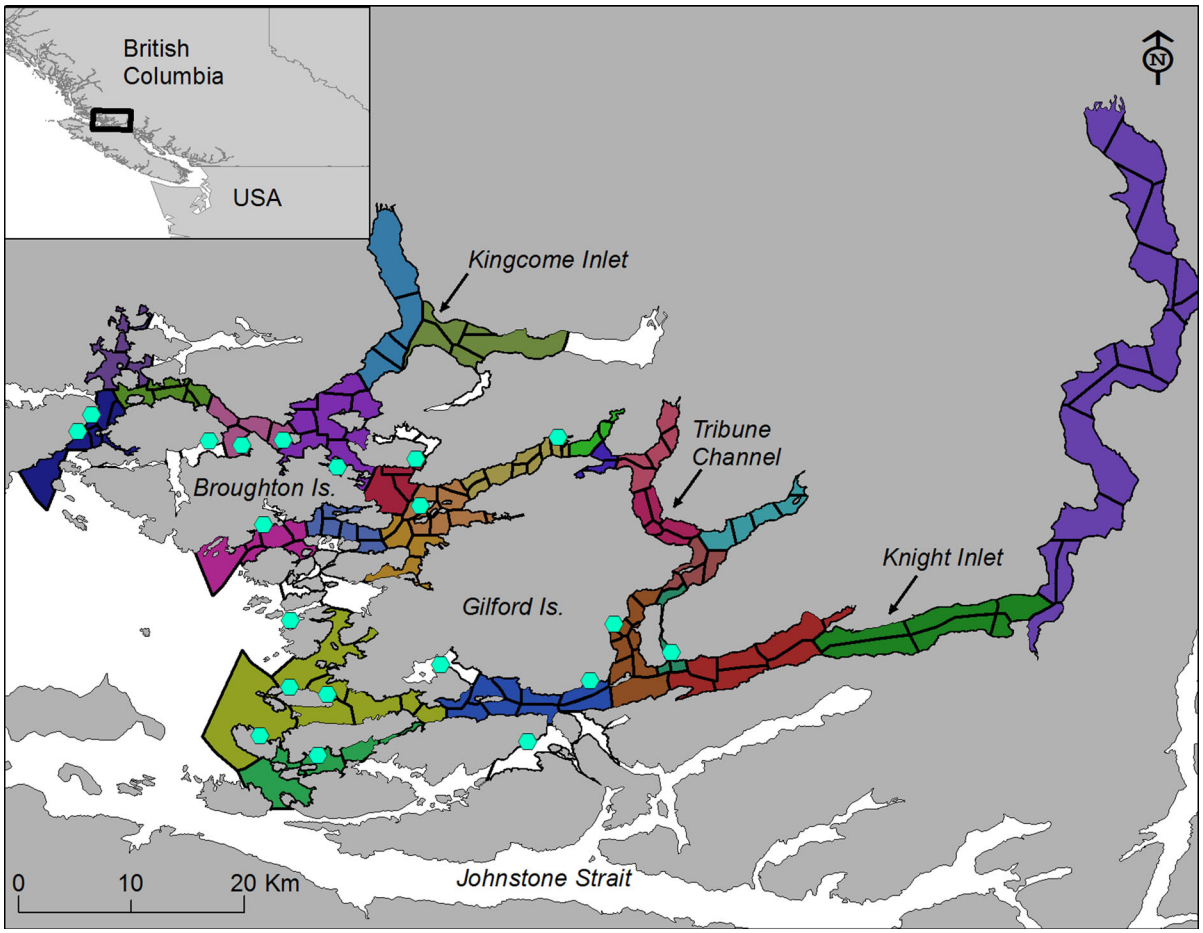


Fig. 1 Study area in the Broughton Archipelago, illustrating the sampling zones used to structure data for the multivariable model (*thick black outline*) and the sub-regions (*colour*) used for imputing missing data. Also shown are the locations of active Atlantic salmon farms during the study (turquoise hexagons)

Variables used in statistical analysis

Outcome and explanatory variables were aggregated to month t and at the sampling zone level i. We used Voronoi tessellation to divide the study area into non-overlapping zones for the 148 sampling sites (Fig. 1). This algorithm subdivides space such that the area within a zone is closer to its centre than the centre from any other zone (Okabe et al. 2000). Voronoi tessellation was based on the DFO sampling because the sites were more clustered among multiple sampling instances than from the Krkošek team sampling.

A challenge for quantifying infection pressure from sea lice sources is that movement patterns of wild salmon cause uncertainty pertaining to where and when infection may have occurred relative to the sampling event. Non-motile sea lice represent more recent infection events than motile sea lice. Therefore, we assumed that the location where wild salmon were captured with non-motiles was closer to the true location of infection than where infection had occurred with older motile staged lice, given that the fish would have had more time to move from the location of infection. Similarly, we assumed sources of motile lice releasing infectious copepodids into the water column were closer in space and time to the infection transmission events. We assessed variables representing sources of sea lice infection as motiles from (a) wild juvenile chum and pink salmon, and (b) farms at time $t-1$, affecting the non-motile sea lice abundance on the wild juvenile salmon at time t. The one-month time lag accounted for time required for motile sea lice

in time $t-1$ to produce copepodids infecting fish at time t within an area over which we assumed the host and parasite populations may interact. Wild juvenile chum and pink salmon as sources of sea lice infection were quantified as the mean abundance of motiles on the fish at time $t-1$ as defined from the: (i) sampling zone i ($A_{i,t-1}$), and proportionally weighted by sample size of fish assessed per zone for (ii) zones immediately adjacent with zone i ($B_{i,t-1}$), and (iii) both $A_{i,t-1}$ and $B_{i,t-1}$ (i.e. $AB_{i,t-1}$). Salmon farms as sources of sea lice infection were modelled as a kernel density weighted variable ($F_{i,t}$) exerting an estimated infection pressure up to seaway distance x. The infection pressure on sampling zone i at time t from neighbouring farms j at time t was determined by summing the potential output of motile sea lice from the farms weighted by their seaway distance to the centre of sampling zone i. Seaway distances, defined as the shortest distance through water around land, were calculated using statistical software R (R Development Core Team 2012) and the *gdistance* package (van Etten 2012). The potential output of sea lice from farms was computed by multiplying the abundance of motile sea lice, *L. salmonis* or *C. clemensi*, 1 month prior on each of the neighbouring farms ($G_{j,t-1}$) by the number of fish ($N_{j,t-1}$). The weights ($w(d_{i,j})$) were obtained from the seaway distance ($d_{i,j}$) and a Gaussian kernel density with a band width equal to approximately one-fourth of the total width, as used by Jansen et al. 2012. The resulting formula for farm infection pressure was:

$$F_{i,t} = \sum_{j \in A_i} w(d_{i,j}) \mathrm{G_{j,t-1}} N_{j,t-1} \quad (1)$$

where A_i was the area included within the range, x, of the kernel. To assess the distance, x, where the effect of neighbouring farms no longer contributed significantly to juvenile sea lice on wild salmon, we evaluated the output of lice from the farms at maximum distances of 10, 20, 30, 40, 50 and 60 km. Though results from Foreman et al. (2009) and Stucchi et al. (2011) suggest average mean flows (and hence an overall dispersion of particles) seaward from the heads of inlets, farm infection pressure, $F_{i,t}$, was assumed to be independent of direction in this study. However, to partially account for farms that reside in relatively sheltered regions and thus are subject to less dispersion than those in channels, all $F_{i,t}$ were standardised by dividing by the area of water within the seaway distance x. We acknowledge these $F_{i,t}$ assumptions are relatively simple. Nevertheless, they produce an initial set of results that can be compared with those of future analyses that incorporate more realistic physical oceanographic features (e.g. from Foreman et al. 2009).

In accounting for confounding factors, we assessed salinity (psu) of sampling zone i and month t. To fill in missing observations, we imputed values as the mean of the adjacent zones, or if also missing, as the mean of sampling zones at the sub-regional level (Fig. 1). Despite the same imputation approach, there were insufficient observations of temperature to model this variable. Thus, we included fixed variables for month and year to account for temperature and other unknown factors potentially influencing seasonal and annual levels of the outcome variable. We used mean length of fish captured in sampling zone i at time t to represent sea lice exposure time of the juvenile salmon in the near-shore environment, assuming that larger fish had spent more time staging (and growing) since leaving their natal streams than smaller fish. Also younger pink salmon are more susceptible while their scales and immune systems are still developing (Jones et al. 2008). We included a variable for wild salmon species (i.e. proportion of chum to pink salmon sampled in zone i at time t), and an interaction with fish length because salmonid species vary in their susceptibility to sea lice (Fast et al. 2002) and chum grow faster than pink salmon (Moss et al. 2009). The interaction was used to explore species differences in infection levels of sea lice over the sampling season. To account for sampling biases, we assessed variables for the proportion of fish captured by beach seine netting and assessed by non-lethal methods, both for sampling zone i at time t.

Statistical model

We used a multivariable two-part random effects model (Tooze et al. 2002; Liu et al. 2008) to determine factors affecting the mean abundance of non-motile sea lice on wild juvenile chum and pink salmon in sampling zone i for month t ($Y_{i,t}$). This model permitted us to account for a substantial proportion of zeros (i.e., $Y_{i,t} = 0$) in the dataset (25 %). We modelled the odds of the mean non-motile infection being greater than zero (part I) using a logistic regression, and the mean non-motile sea lice intensity

(part II), that is, number of lice given that counts were greater than zero using a gamma regression model. Both parts of the model included a sampling zone random effect to account for repeated measures within the zone. Potential autocorrelation between monthly measures of non-motile sea lice was assessed by including a time-lagged outcome (Y_{t-1}) among the predictor variables. In summary, the model equations were:

$$\log_{\text{it}}(\pi_{i,t}) = X^{I}_{i,t}\beta^{I} + \mu_{1i} \quad (2)$$

$$\ln(u_{it}) = X^{II}_{i,t}\beta^{II} + \mu_{2i} \quad (3)$$

where $\pi_{i,t} = \text{P}(Y_{i,t} > 0)$, is the probability of a non-zero abundance of non-motile sea lice, and $\mu_{it} = \text{E}(Y_{i,t} \mid Y_{i,t} > 0)$ is the mean non-zero intensity of non-motile sea lice, modeled by a gamma distribution with a constant shape parameter, θ. The pairs of random effects for sampling zones were modelled as $[u_1, u_2] \sim \text{N}([0,0], [\sigma_1^2, \rho\ \sigma_1\ \sigma_2, \sigma_2^2])$. The model was implemented in SAS PROC NLMIXED (SAS® 9.1.3 for Windows, SAS Institute Inc., Cary, NC) and estimated by adaptive Gaussian quadrature, as previously described (Tooze et al. 2002; Liu et al. 2008).

Model building

We determined the most appropriate combinations of the predictor variables, as well as their forms (i.e. binary or continuous on either original or log scale). Predictors were included or excluded from the analysis simultaneously in both parts of the model. To facilitate the model estimation algorithms, the time lagged sources of sea lice from wild salmon were included as binary predictors in Part I and as log scale continuous predictors in Part II of the model. Prior to log transformation, we added 1.0×10^{-3} to the predictor to enable transformation of zero values while still maintaining a quantitatively negligible value. Salinity was assessed both on original and restricted scales with lower cutoffs at 10, 15, 20 or 25 psu and an upper cutoff of 30 psu, based on known biological effects of salinity on sea lice (Bricknell et al. 2006; Bravo et al. 2009). We were unable to model the year effect for 2003 because there were no farm lice counts during the months of out-migrating wild chum and pink salmon for this year. All continuous explanatory variables were centred by their mean and then scaled to one unit of standard deviation to permit the direct comparison of the magnitude of the effect for each explanatory variable. We used Akaike's information criterion (AIC) model selection techniques (Burnham and Anderson 2002) to rank the candidate models. Models with a small difference in AIC, $\Delta\text{AIC} \leq 2$, from the top ranked model provided effectively equal support (Burnham and Anderson 2002).

As a diagnostic tool, the observed number of sampling zones with no non-motile sea lice (i.e. abundance = 0) was compared to the expected number of zero values across the dataset, as predicted by part I of the model. For part II of the model, the Anscombe residuals (McCullagh and Nelder 1989) were inspected visually by plotting them against all predictor variables and the fitted values. We visually assessed for unaccounted spatial autocorrelation by mapping the random effects and the Anscombe residuals.

We used cross-validation to assess the predictive ability of the top ranked model. The dataset was randomly divided to have 75 % of the data for model building and the remaining 25 % for comparing with model predictions. The goodness of fit between the predicted result from part I of the model and observed data was calculated using the AUC (area under the receiver operating characteristic curve value), which indicated the ability of the model to correctly classify the observed outcomes: 0.9–1.0 excellent, 0.8 to <0.9 good, 0.7 to <0.8 fair, 0.6 to <0.7 poor, <0.6 fail (Hosmer and Lemeshow 2004). The agreement between predicted and observed non-zero data for part II of the model was assessed using Spearman's correlation coefficient. The validation process was repeated five times with unique random divisions of the data.

Model predictions

We used the model to predict non-motile sea lice infection on wild juvenile chum and pink salmon. Predictions on wild fish in the sampling zones were generated for scenarios for (a) reported farm lice levels in June 2004 capped at an abundance of 3 motile sea lice, which is the maximum permitted under the current regulations, and (b) farms with no sea lice. The other predictors in our model were set to represent June 2004, the year in our study for which conditions were determined to be optimal for sea lice infection. We used Part I of the model to map the probability of sampling wild juvenile chum and pink salmon infected

with non-motile sea lice, and Part II of the model to map the intensity of infection.

Results

Data from 166,316 wild juvenile chum and pink salmon were available for the study. Mean intensity of non-motile sea lice on the wild salmon over the duration of the study period was 3.07 (SD = 5.91). In 2004, when conditions were favourable to sea lice survival the mean intensity was 6.36 (SD = 9.89), compared to the other years at 1.66 (SD = 1.25). Aggregation of data by sampling zones and month resulted in 1 061 analytical units, from a total of 148 zones ranging in size from 0.5 to 42.0 km^2 (mean 5.2 km^2; Fig. 1). A descriptive analysis assessing the relationship between the predictor variables and the outcome, non-motile abundance on the wild salmon, denoted several strong trends (Table 1). Mean seasonal abundance of non-motile sea lice increased from April to July. Mean yearly average abundance of non-motile sea lice on the wild salmon was less than 0.7 lice per fish, with the exception of 2004, the highest year of reported infection at 7.8. The average motile abundance on farmed salmon was also greatest in 2004 (Supplementary Material Table S1). The abundance of non-motile sea lice on the wild salmon positively varied with the abundance of motiles from wild salmon from the previous time unit in the same sampling zone and from the surrounding sampling zones, as well as salinity, proportion of chum salmon, and month (Table 1). The abundance of non-motile sea lice appeared to have a non-linear relationship with fish length in that mid-sized fish had higher levels than smaller or larger fish (Table 1). From 2003 to 2012, chum had a higher intensity of non-motile sea lice infection than pink salmon (Supplementary Material Fig. S1), and the proportion of non-motile to motile sea lice infection decreased more slowly in chum over the sampling season (Supplementary Material Fig. S2). Clearly these data contain a degree of variability; however, these patterns were largely reflected in the multivariable model.

For the multivariable model, we found the abundance of non-motile sea lice on wild juvenile chum and pink salmon was sensitive to the spatial scales of the predictor variables representing sources of sea lice (Table 2). Models ranked higher when a variable for farm infection pressure was included, with the top-ranked AIC model indicating this effect extended up to 30 km. Non-motile sea lice abundance on the wild juvenile salmon was positively associated with farm infection pressure (Table 3). We found that motile infection pressure from the wild juvenile salmon in the previous month was best explained by combining fish from sampling zone i and the immediate surrounding zones (i.e. $AB_{i,t-1}$). This factor had a positive association in Part I of the model and a negative association in Part II of the model. For both parts of the model, farms as sources of sea lice explained more variation in the response variable than did infection from the wild juvenile salmon.

In controlling for the confounding factors, the intensity of non-motile sea lice on wild salmon was less in all years compared to 2004 in part I and II of the model. Also, in both parts of the model, samples from June and July had higher abundances of sea lice than was the case for April. Our top model retained one variable for sampling biases, the proportion of fish assessed using non-lethal methods, such that the probability of wild salmon being infected with non-motile sea lice increased with an increasing proportion of fish assessed using non-lethal methods (part I); however, there was no apparent association with this variable and intensity of non-motile sea lice reported on infected wild juvenile salmon (part II). Catches with a higher proportion of chum had greater infection intensities (part II). Mid-sized fish were more likely to be infected (part I), and at a higher intensity than infected fish at smaller or larger sizes (part II). The interaction between fish length and species indicated that juvenile chum and pink had different non-motile abundance levels relative to their length. The continuous form of salinity best explained the outcome variable, and was found to be positively associated with an increased probability of sea lice infection and intensity.

Cross-validation results support that our model adequately represented the pathogen–host system. For part I of the model, the mean AUC for predicted to observed data was 0.88 (SD = 0.01). For part II of the model, the mean Spearman's rank correlation of predicted to observed data was 0.73 (SD = 0.03).

Our model predicted that the wild salmon have the greatest probability of infection in the main channels and out towards the open ocean (i.e. further from the upper inlets; Figs. 2, 3) regardless of whether there

Table 1 Description of candidate predictor variables and their relationship with the outcome, *Y*, non-motile sea lice abundance (*L. salmonis* and *C. clemensi*) on wild juvenile salmon (chum and pink) in sampling zone *i* at month *t*, unless otherwise stated, for 2003–2012

Variable	Variable mean	Variable 90 % range	Variable level	*Y* mean	*Y* 90 % range
Sampling zone motile abundance$_{t-1,i}$*	0.204	0–0.38	0–0.003	0.236	0–0.630
			>0.003–0.07	0.354	0–1.09
			>0.07	4.01	0–6.89
Neighbouring motile abundance$_{t-1,i}$*	0.191	0–0.33	0–0.01	0.230	0–0.621
			>0.01–0.09	0.280	0–0.961
			>0.09	4.04	0–6.86
Sampling zone and Neighbouring zone motile abundance$_{t-1,i}$*	0.182	0–0.32	0–0.01	0.210	0–0.532
			>0.01–0.09	0.293	0–0.920
			>0.09	3.98	0–6.78
Salinity (psu)	21.6	0.02–29.8	0.02–10.0	0.154	0–0.12
			>10.0–20.0	1.95	0–0.428
			>20.0	0.974	0–1.50
Fish length (mm)	61.6	29.5–86.4	29.5–40.0	0.176	0–0.455
			>40.0–60.0	0.571	0–1.14
			>60.0–80.0	0.851	0–0.55
			>80.0–100.0	6.78	0–8.66
			>100.0–120.0	1.50	0–5.35
			>120.0–140.0	0.455	0.05–0.934
			>140.0–143.0	0.0567	0.014–0.0940
Proportion of non-lethally sampled fish	0.245	0–1.0	0–0.25	1.36	0–1.12
			>0.25–0.50	0.097	0.028–0.243
			>0.50–0.75	0.408	0.018–0.582
			0.75–1	0.773	0.030–1.42
Proportion of beach sampled fish	0.770	0–1.0	0–0.25	1.33	0–2.34
			>0.25–0.50	1.01	0–2.06
			>0.50–0.75	0.612	0–0.638
			0.75–1	1.18	0–1.06
Proportion of chum	0.471	0–1.0	0–0.25	0.200	0–0.420
			>0.25–0.50	0.317	0–0.774
			>0.50–0.75	0.772	0–1.63
			0.75–1	4.06	0–6.42
Sampling month			April	0.238	0–0.632
			May	0.607	0–1.19
			June	1.91	0–1.67
			July	2.18	0–4.07
Sampling year			2003	0.325	0–0.932
			2004	7.80	0–10.6
			2005	0.510	0–0.982
			2006	0.232	0–0.480
			2007	0.655	0–1.31
			2008	0.0795	0–0.210
			2009	0.151	0–0.267
			2010	0.215	0–0.492
			2011	0.0869	0–0.269
			2012	0.174	0–0.492

* Variable levels defined at 50 and 75 % quartiles

Table 2 Top models estimating the abundance of non-motile sea lice (*L. salmonis* and *C. clemensi*) on wild juvenile salmon (chum and pink) in sampling zone i at month t

Variables	AIC	ΔAIC	K	−2ll
$Y_{i,t} + M_{i,t} + C_{i,t} + S_{i,t} + L_{i,t} + L_{i,t}^2 + E_{i,t} + AB_{i,t-1} + F_{i,t-1,30\ \mathrm{km}} + C_{i,t} \times L_{i,t} + C_{i,t} \times L_{i,t}^2$	944.6	0.0	42	849.2
$Y_{i,t} + M_{i,t} + C_{i,t} + S_{i,t} + L_{i,t} + L_{i,t}^2 + E_{i,t} + AB_{i,t-1} + F_{i,t-1,40\ \mathrm{km}} + C_{i,t} \times L_{i,t} + C_{i,t} \times L_{i,t}^2$	949.2	4.6	42	853.8
$Y_{i,t} + M_{i,t} + C_{i,t} + S_{i,t} + L_{i,t} + L_{i,t}^2 + E_{i,t} + AB_{i,t-1} + F_{i,t-1,20\ \mathrm{km}} + C_{i,t} \times L_{i,t} + C_{i,t} \times L_{i,t}^2$	951.0	6.4	42	855.6
$Y_{i,t} + M_{i,t} + C_{i,t} + S_{i,t} + L_{i,t} + L_{i,t}^2 + E_{i,t} + AB_{i,t-1} + F_{i,t-1,10\ \mathrm{km}} + C_{i,t} \times L_{i,t} + C_{i,t} \times L_{i,t}^2$	958.1	13.5	42	862.7
$Y_{i,t} + M_{i,t} + C_{i,t} + S_{i,t} + L_{i,t} + L_{i,t}^2 + E_{i,t} + AB_{i,t-1} + F_{i,t-1,50\ \mathrm{km}} + C_{i,t} \times L_{i,t} + C_{i,t} \times L_{i,t}^2$	960.7	16.1	42	865.3
$Y_{i,t} + M_{i,t} + C_{i,t} + S_{i,t} + L_{i,t} + L_{i,t}^2 + E_{i,t} + AB_{i,t-1} + F_{i,t-1,60\ \mathrm{km}} + C_{i,t} \times L_{i,t} + C_{i,t} \times L_{i,t}^2$	966.4	21.8	42	871.1
$Y_{i,t} + M_{i,t} + C_{i,t} + S_{i,t} + L_{i,t} + L_{i,t}^2 + E_{i,t} + AB_{i,t-1} + C_{i,t} \times L_{i,t} + C_{i,t} \times L_{i,t}^2$	1005.3	60.7	41	914.3

Models are ranked by Akaike's information criterion. ΔAIC is the difference in AIC from the top ranked model, K is the number of fixed effects parameters (including intercepts) in the nested model, and −2ll describes model fit as the −2 Log Likelihood

Y year (range: 2004–2012), *M* month (range: April–July), *C* proportion of wild juvenile salmon that are chum, *S* proportion of wild juvenile salmon assessed using non-lethal methods, *L* mean fish length, *E* mean salinity, *AB* mean sampling zone and neighbouring zones of motile abundance at month *t−1, F* kernel weighted measure of motile infection pressure from farms within 30 km at month *t−1*

Variables represent the averages at sampling zone *i* for month *t*, unless specified

were lice on farmed fish or not. We found the ambient mean intensity of non-motile sea lice infections on wild salmon (i.e. when there was no farm infection pressure) to be 1.85 (SD = 1.48; minimum = 0.36; maximum = 8.22). When the mean abundance on farm fish was set at the regulatory limit of 3 motile lice per fish, the mean non-motile sea lice intensity on wild salmon increased to 2.72 (SD = 2.21; minimum = 0.50; maximum = 13.0) (Figs. 2, 3).

Discussion

Wild juvenile chum and pink salmon enter the marine environment free of sea lice, but over time these fish can be exposed to and become infected with sea lice. Under conditions favourable for sea lice survival our model predicted that sea lice infection pressure from Atlantic salmon farms on wild juvenile salmon extended throughout most of the study region, with the exception of the upper inlets, and was highest near the farms. Though, with or without farms as a source of sea lice, infections on wild juvenile salmon were predicted to be highest in the main channels. Sea lice infection may be more likely in these areas for two primary reasons. Firstly, sea lice survival and infection viability are sensitive to salinity (Jones et al. 2006a, 2006b; Jones and Hargreaves 2007). The Broughton Archipelago inlets have lower salinity levels, especially during late spring and summer when inflowing river discharge is at its highest from snow and glacial melt water (Freeland and Farmer 1980). Sea lice can actively swim to depths with preferred salinity levels (Heuch 1995). Therefore, we hypothesize that the influx of freshwater into the Broughton Archipelago may enforce a natural separation between pathogens and hosts, reducing the probability of infection, as sea lice seek deeper depths to avoid the freshet (which is less dense than seawater) and the out-migrating chum and pink remain near the surface (upper 2 m) as per their natural behaviour (Heard 1991; Salo 1991). This effect is likely to be strongest closest to the river outlets in the upper inlets. Secondly, *L. salmonis* and especially *C. clemensi* are natural parasites to Pacific salmon, and are known to infect a broad range of non-salmonid host species including herring (*Clupea pallasi*) and sticklebacks (*Gasterosteus aculeatus*) that overwinter in the coastal area. Thus, these fish

Table 3 Standardised parameter coefficient estimates (β) and standard errors (SE) of the explanatory variables in the AIC top-ranked model for abundance of non-motile sea lice (*L. salmonis* and *C. clemensi*) on wild juvenile salmon (chum and pink) in sampling zone *i* at month *t*

	β	SE
(a) Part I		
Intercept	3.45	0.62
2005	−0.91	0.74
2006	−1.95	0.63
2007	−1.09	0.68
2008	−1.65	0.60
2009	−1.14	0.63
2010	−0.53	0.61
2011	−1.63	0.64
2012	−0.49	0.62
May	0.20	0.35
June	−2.42	0.55
July	−2.70	0.73
Proportion non-lethal sampling$_{t,i}$	1.67	0.20
Proportion chum$_{t,i}$	0.02	0.11
Length$_{t,i}$	4.32	0.77
Length$^2_{t,i}$	−4.12	0.82
Length$_{t,i}$ × Proportion chum$_{t,i}$	−0.43	0.40
Length$^2_{t,i}$ × Proportion chum$_{t,i}$	0.76	0.44
Salinity$_{t,i}$	0.89	0.12
Wild fish infection pressure, $AB_{i,t-1}$	0.42	0.26
Farm motile abundance$_{t,i,30\ km}$	0.89	0.27
Sampling zone deviation	0.52	0.05
(b) Part II		
Intercept	0.08	0.18
2005	−1.07	0.25
2006	−2.01	0.17
2007	−1.25	0.17
2008	−2.58	0.18
2009	−2.71	0.20
2010	−1.85	0.19
2011	−2.20	0.21
2012	−1.92	0.20
May	0.45	0.12
June	0.31	0.21
July	0.89	0.31
Proportion non-lethal sampling$_{t,i}$	0.002	0.04
Proportion chum$_{t,i}$	0.29	0.04
Length$_{t,i}$	1.73	0.31
Length$^2_{t,i}$	−1.93	0.33
Length$_{t,i}$ × Proportion chum$_{t,i}$	0.65	0.19
Length$^2_{t,i}$ × Proportion chum$_{t,i}$	−0.44	0.20
Salinity$_{t,i}$	0.44	0.05
Wild fish infection pressure, $AB_{i,t-1}$	−0.17	0.06
Farm motile abundance$_{t,i,30\ km}$	0.35	0.05
Sampling zone deviation	0.71	0.17
Sampling zone correlation (ρ)	0.59	0.62
Log of shape parameter (θ)	0.38	0.04

(a) Part I models the probability of non-motile abundance being greater than zero, and (b) Part II models the intensity of infection given that sampled wild juvenile salmon have this stage of sea lice. Coefficient estimates and SE for levels of year and month variables are in reference to year 2004 and month April, respectively. $AB_{i,t-1}$ denotes wild fish infection pressure resulting from juvenile chum and pink salmon assuming a binary (Part I) or log transformed distribution (Part II) with the response variable

† Variable is binary in Part I and log transformed in Part II

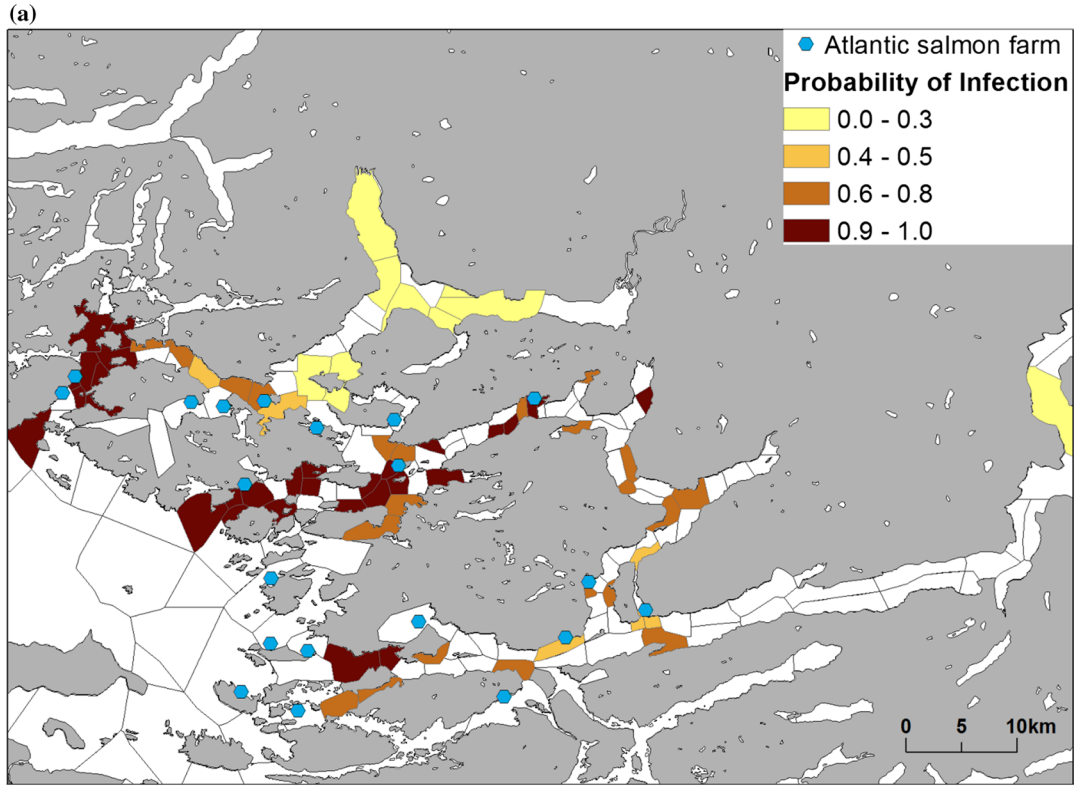
Fig. 2 Model predictions of non-motile sea lice infection levels (*L. salmonis* and *C. clemensi*) on wild juvenile salmon (*chum* and *pink*) in the study area for favourable sea lice conditions, given no simulated farm infection pressure (i.e. ambient level), for the **a** probability of infection (model Part I), and **b** intensity of infection (model Part II). *White* sampling zones had insufficient data to generate model estimates ▶

may also serve as sources of infection (Jones et al. 2006a; Beamish et al. 2009; Gottesfeld et al. 2009).

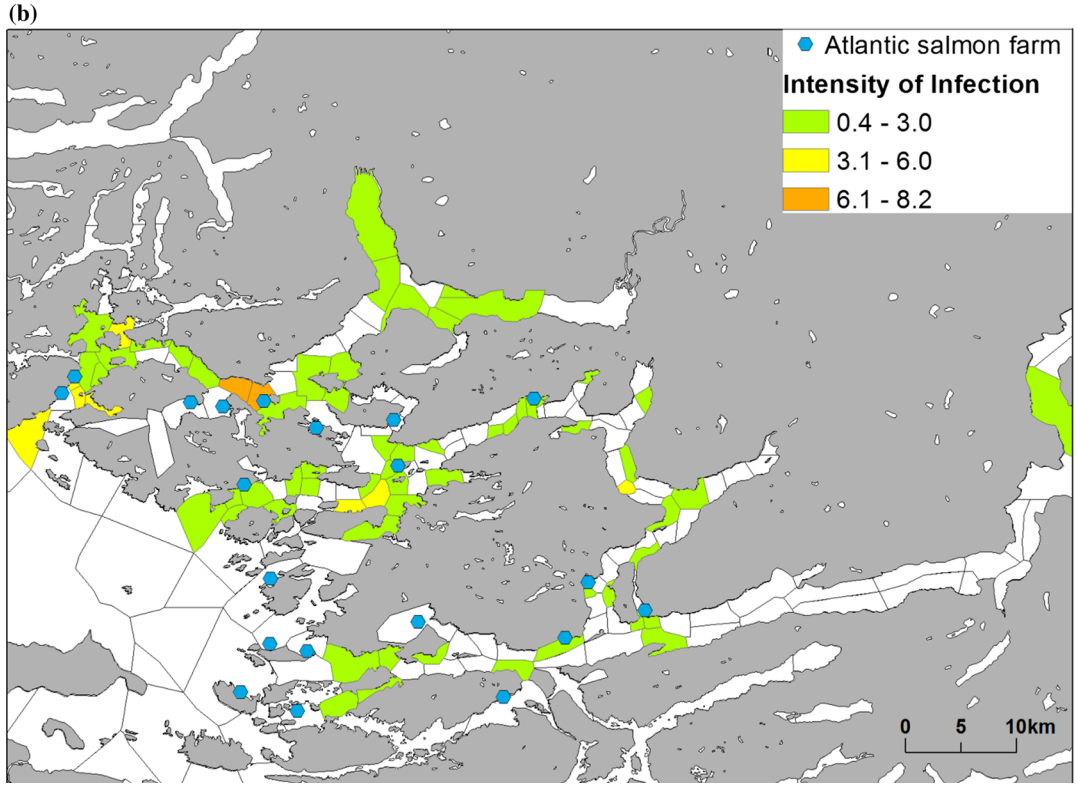
We did not find strong evidence of self-infection within the wild juvenile chum and pink salmon populations. Our assumption that sea lice levels on these fish in the previous month contribute to infection on fish sampled in the current month is tenuous because the fish and planktonic sea lice are not stationary. It is possible that the fish migrate beyond the immediate and neighbouring sampling zones over the one month period. Also, larvae produced by the adult lice within a zone may have dispersed to other zones over the week or two required to develop into infectious copepodids given typical study area water temperatures (approximately 7–12 °C from March to July; Stucchi et al. 2011).

Salmon farms are stationary through time and the number of fish within the farms is known. Therefore, we expect our estimate of infection pressure from farms to be more accurate than from the wild juvenile salmon. Non-motile sea lice abundance on the wild

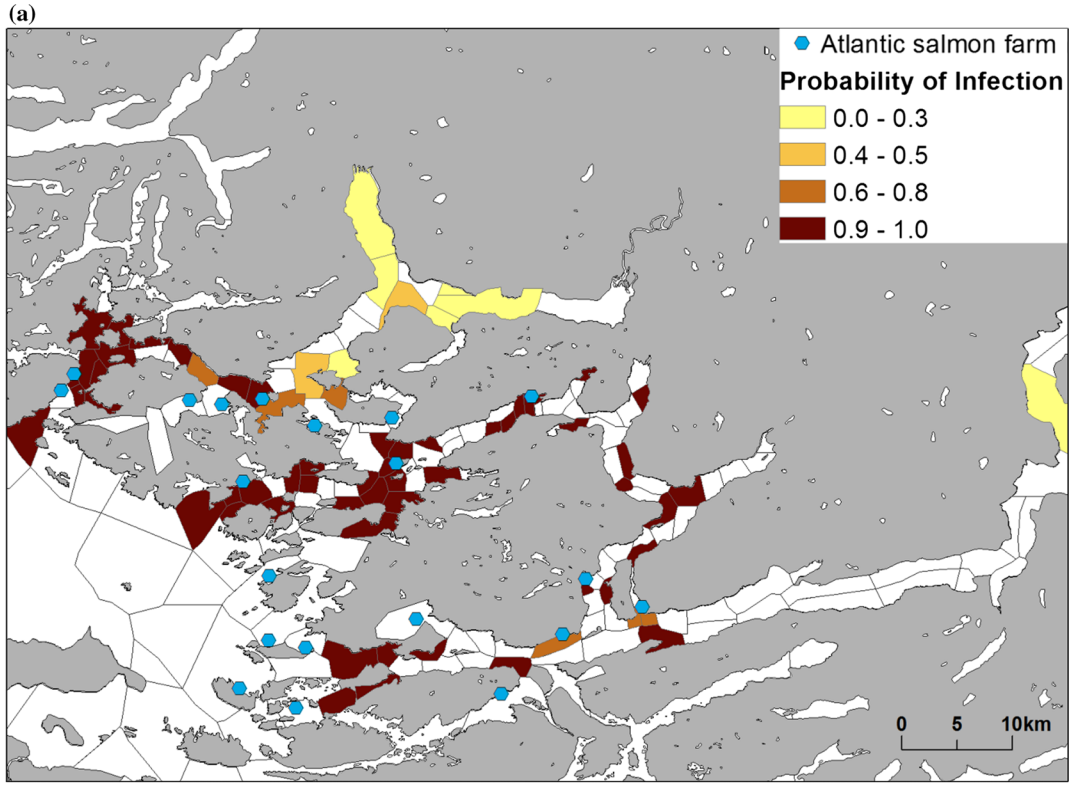
(a)


Atlantic salmon farm
Probability of Infection
0.0 - 0.3
0.4 - 0.5
0.6 - 0.8
0.9 - 1.0
0 5 10km

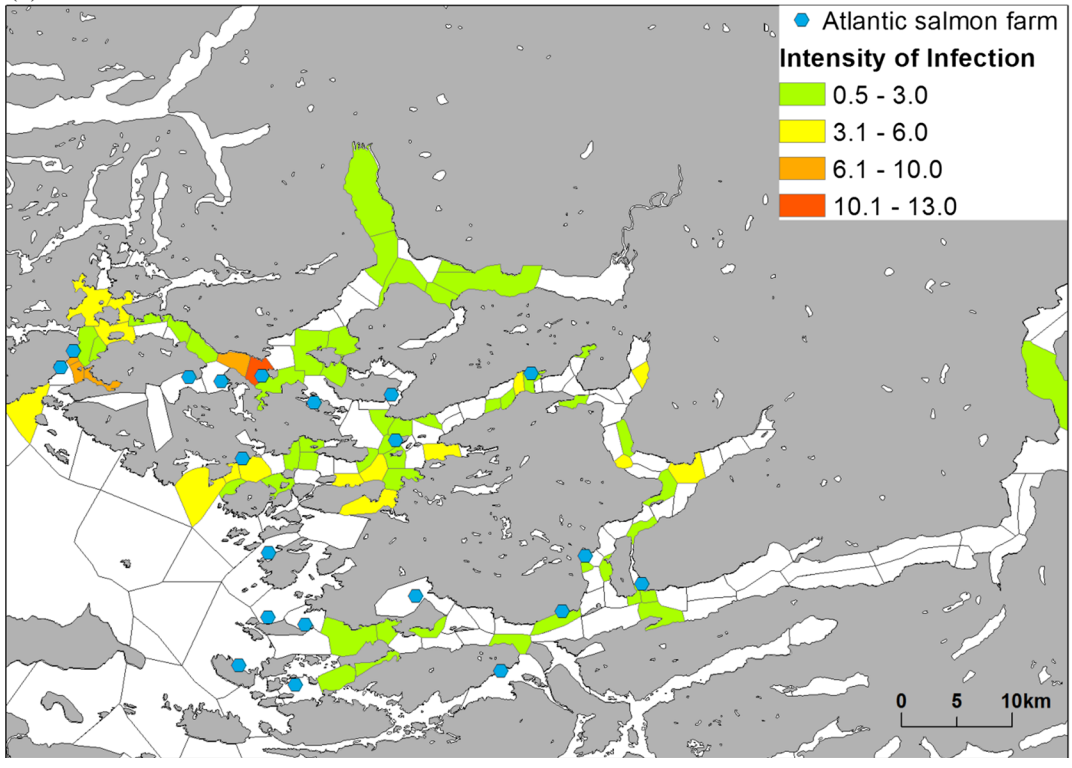
(b)


Atlantic salmon farm
Intensity of Infection
0.4 - 3.0
3.1 - 6.0
6.1 - 8.2
0 5 10km

(a)


Atlantic salmon farm
Probability of Infection
0.0 - 0.3
0.4 - 0.5
0.6 - 0.8
0.9 - 1.0
0 5 10km

(b)


Atlantic salmon farm
Intensity of Infection
0.5 - 3.0
3.1 - 6.0
6.1 - 10.0
10.1 - 13.0
0 5 10km

◀**Fig. 3** Model predictions of non-motile sea lice infection levels (*L. salmonis* and *C. clemensi*) on wild juvenile salmon (*chum* and *pink*) in the study area for favourable sea lice conditions, given simulated farm infection pressure capped at a maximum motile abundance of 3, for the **a** probability of infection (model Part I), and **b** intensity of infection (model Part II). *White* sampling zones had insufficient data to generate model estimates

juvenile salmon was most strongly associated with motile abundance on farms located within 30 km, over a 1-month period, as detected using our statistical approach. Biologically, this represents the space–time scale over which sea lice are produced and released into the environment at a sufficient dose to infect a measurable proportion of the host population. The key processes involved are: (a) the dispersion of the unattached sea lice stages given ocean circulation, (b) their survival and development rates given water salinity and temperature, and (c) the area over which wild fish are close enough (in space and time) to infected farms to be infected by sea lice originating on farms.

Similar statistical modelling approaches also found that farms exert sea lice infection pressure up to approximately 30 km over 2 weeks in Chile (Kristoffersen et al. 2012) and 31 km in Scotland (Middlemas et al. 2012). A mathematical modelling approach by Krkosek et al. (2005a) also indicated a positive association with sea lice infection levels on wild smolts within 30 km of one farm in a single channel of the Broughton Archipelago. These studies suggest that farms on average exert a sea lice infection pressure that is measurable up to 30 km, however, we caution upholding to this conclusion too strongly. The direction and speed of ocean currents are known to affect the spatial scale of disease transmission in other pathogen–host systems (Gustafson et al. 2007; Viljugrein et al. 2009). Within Knight Inlet of our study area, mooring stations recorded mean current speeds at a 5 m depth in early spring 2008 to range from approximately 0.02 to 0.18 m s^{-1} (Stucchi et al. 2011), which could move water, and potentially passively drifting planktonic sea lice, roughly 50–460 km over a 1 month period, assuming constant conditions. However, over these distances the study area varies from channels to more open areas, which we hypothesize will concentrate or dilute, respectively, the force of infection from farms. We accounted for this effect by standardising farm infection pressure (as defined by the kernel weighted measure) with the surface area of available water. Without standardisation in an alternate form of the model built over the study period 2004–2009 (output is available upon request), the footprint size was 10 km. With our current model and no standardisation we were unable to distinguish between a footprint size of 20 and 30 km (i.e. these candidate models had an AIC value within 2.0 of each other; Supplementary Material Table S2). Therefore, our accounting for the topography of the study area likely improves upon the estimated average reach of farm infection pressure from previous studies. Though beyond the scope of this study, accounting for the speed and direction of water currents should lead to even more accurate estimates for the spatiotemporal scales of pathogen transmission.

The environmental impact of marine Atlantic salmon aquaculture is often highlighted as a consideration in the conservation of wild salmon populations (Krkosek et al. 2007; Costello 2009). The number of lice harmful to the wild salmon hosts depends on their health, size and species; lice species and life stage; and the surrounding environment (Fast et al. 2002; Butterworth et al. 2008; Connors et al. 2008; Jones et al. 2008; Wagner et al. 2008). It is not clear how the effect of the pathogens may interact or be superseded by other mortality factors during the salmonid life history (Krkosek et al. 2013). In our model, when farm-level infection was set at the regulatory abundance of 3 motiles per fish and environmental conditions favoured sea lice survival, mean intensity of infection on wild salmon increased from an ambient level of 1.85 per fish to 2.72 per fish. Using an alternate form of the model built over the study period 2004–2009 (output is available upon request) we predicted for typical study period conditions (i.e. excluding 2004 data, the year of high infection) and found mean intensity of infection on wild salmon increased from an ambient level of 0.22 (SD = 0.08) per fish to 0.23 (SD = 0.09). Previous research indicated that risk of harm associated with *L. salmonis* is size dependent in juvenile pink salmon (Jones et al. 2008; Brauner et al. 2012). More modeling and controlled laboratory studies are required to assess the impacts of mixed *L. salmonis* and *C. clemensi* infections on juvenile salmon.

The observed and modelled variation in non-motile sea lice abundance given fish length provides insight on factors affecting sea lice infection of wild juvenile

chum and pink salmon over time. The data in our multivariable analysis were best fit using a quadratic expression of length such that mid-lengths had the highest infection levels of non-motile lice, as was also the result when modelling both sea lice species by an earlier study (Patanasatienkul et al. 2013). This relationship may result if the salmon accumulate lice for several weeks following entry into seawater, and then above mid-size lengths, have a decreased susceptibility to new infections, or that infections on larger fish tend to be with motile stages. A reduction in susceptibility may occur for larger (i.e. older) juvenile hosts because of developing immunity or resistance to re-infection (Jones et al. 2008; Sutherland et al. 2011) and added protection from increasing thickness of scales. It is also possible that mid-length juvenile chum and pink salmon with high infections do not survive, and thus are not represented at larger lengths. The interaction between fish length and species indicated differential levels of infection for chum and pink salmon relative to their lengths. Pink salmon had a lower intensity of non-motile sea lice than chum, and the proportion of new infections (non-motile sea lice) was less on larger fish for pink than for chum salmon. This interaction may reflect differential growth rates between juvenile chum and pink salmon (i.e. chum grow faster; Heard 1991; Salo 1991), and a faster and potentially stronger immune response (or resistance) to sea lice infection in pink than chum salmon (Jones et al. 2007).

Our study helps discern the multiple factors impacting dynamics of sea lice transmission in a wild-farmed salmon pathogen–host system. Previous studies have identified abiotic factors (Brooks 2005), farm activity (Peacock et al. 2013), or spatial processes of parasite dispersal and fish migration (Krkosek et al. 2005a). However, our study was the first to assemble all these factors into a single comprehensive analysis, and indicated that the factors examined herein influence transmission dynamics. Our multi-year analysis involved both fitting as well as validating a model, providing a robust tool for evaluating how Atlantic salmon marine aquaculture and parasite management policies influence spatial patterns of infection levels on wild juvenile chum and pink salmon. Our results suggest it is beneficial to maintain current policy as a precautionary measure to reduce exposure of wild juvenile chum and pink salmon to sea lice from Atlantic salmon farms.

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