

Fish Farms and Sea Lice Infestations of Wild Juvenile Salmon in the Broughton Archipelago—A Rebuttal to Brooks (2005)

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*Contrary to several recent studies, a review (Brooks, 2005) of sea lice (*Lepeophtheirus salmonis*) interactions between wild and farm salmon in the Broughton Archipelago, British Columbia, Canada, concluded that there is little potential for sea lice transmission from farm to wild salmon. In this rebuttal, we show that this conclusion was based on a flawed interpretation of how salinity affects louse development, a misunderstanding of how the timing of salinity changes corresponds to the timing of the juvenile salmon migration, models of larval dispersion that overestimate the transport of louse larvae, and a selective and misleading assessment of the literature. We analyze and extend the current models of larval dispersion and demonstrate the (perhaps counter-intuitive) result that sustained high abundances of infectious larvae should be expected near lice-infested salmon farms. We also highlight important studies overlooked in Brooks (2005) and clarify some misinterpretations. Counter to the conclusions in Brooks (2005), the modeling and empirical work to date on sea lice interactions between wild and farm salmon are consistent and point to a strong association between salmon farming and recurrent infestations of wild juvenile salmon in the Broughton Archipelago.*

Keywords sea lice, aquaculture, salmon, parasite, dispersion, transmission dynamics, reservoir host, emerging disease

Introduction

There can be little doubt that temperature, salinity, and site-specific oceanographic features affect the spread of planktonic larval sea lice (*Lepeophtheirus salmonis* and *Caligus* spp.) released from salmon aquaculture installations in the nearshore environment. In a recent review of sea lice interactions between wild and farm salmon in the Broughton Archipelago, British Columbia, Brooks (2005) argues that these effects combine to prevent the transmission of lice from farm salmon to sympatric wild juvenile pink (*Oncorhynchus gorbuscha*) and chum (*O. keta*) salmon. The conclusions in Brooks (2005) conflict with

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several empirical studies conducted on the effect of salmon farms on sea lice infestations of wild juvenile salmon in the Broughton Archipelago. These studies clearly demonstrate the transmission of lice from farm salmon to wild juvenile salmon (Morton and Williams, 2004; Morton et al., 2004; Krkošek et al., 2005a; Morton et al., 2005). Here we attempt to reconcile this discrepancy by assessing the salinity data, larval dispersion models, and arguments used by Brooks (2005) to arrive at his conclusions. We show where Brooks (2005) goes wrong and how the models, data, and literature point to a strong association between salmon farms and recurrent sea lice infestations of juvenile salmon in the Broughton Archipelago.

Salinity

Salinity is known to have a large influence on the survival of sea lice (Johnson and Albright, 1991). Brooks (2005) claims that salinities less than 30‰ are hostile to the development and survival of copepodids. Based on this cutoff value and an assessment of salinity regimes in the Broughton Archipelago, Brooks (2005) concludes that salinities between June and November produce a natural control mechanism against sea lice proliferation. However, the critical period for juvenile salmon migrating through these waters is March through June. Furthermore, a more careful assessment of the literature shows that salinities between 25‰ and 30‰ are suitable for copepodid development and survival (see below). Between March and June, this is the historical salinity range in Knight Inlet (Brooks, 2005) and this has been the observed salinity range in Tribune Channel, where recurrent infestations of juvenile pink and chum salmon have occurred (Figure 1).

Experimental work by Johnson and Albright (1991) has shown that at 10°C the development of *L. salmonis* larvae into copepodids is severely limited at salinities less than or equal to 25‰. At salinities of 30‰, 35.2% of eggs in static water developed into active copepodids. The developmental success in flowing water at 10°C and 30‰ was 26.8%. We could not find information on how developmental success changes between 25‰ and 30‰. When newly moulted copepodids were introduced to different salinities at 10°C, optimal survival occurred at 25‰ with reduced survival at 15, 20, and 30‰ (Johnson and Albright, 1991). These data do not suggest that 30‰ is a critical salinity threshold below which copepodids do not develop and survive. Rather, these data suggest salinities ranging 25–30‰ are suitable for copepodid development and survival.

Models of Larval Spread

Brooks (2005) presents the results of a complex circulation and particle-tracking model of the Broughton Archipelago, British Columbia. That model, developed by Dario Stucchi (Institute of Ocean Sciences, Department of Fisheries and Oceans, Canada) predicts copepodids are carried long distances from their farm sources. In contrast, a competing model for louse dispersion around salmon farms predicts copepodids will remain near their source (Krkošek et al., 2005a). Consistent with Krkošek et al. (2005a) and in contrast to Brooks (2005), empirical studies from the Broughton Archipelago repeatedly show increased copepodid infections of juvenile pink and chum salmon near active salmon farms (Morton and Williams, 2004; Morton et al., 2004; Krkošek et al., 2005a; Morton et al., 2005). Below, we reconcile the discrepancies in model predictions between Brooks (2005) and Krkošek et al. (2005a). Errors in parameter values and subtle inaccuracies in model structure produced overestimates of larval transport in Brooks (2005). In contrast, an independent parameterization of the larval dispersion model in Krkošek et al. (2005a), using experimental data of larval developmental and survival rates and field measurements of surface current flows, predicts a copepodid distribution that agrees well with field observations of louse abundance.

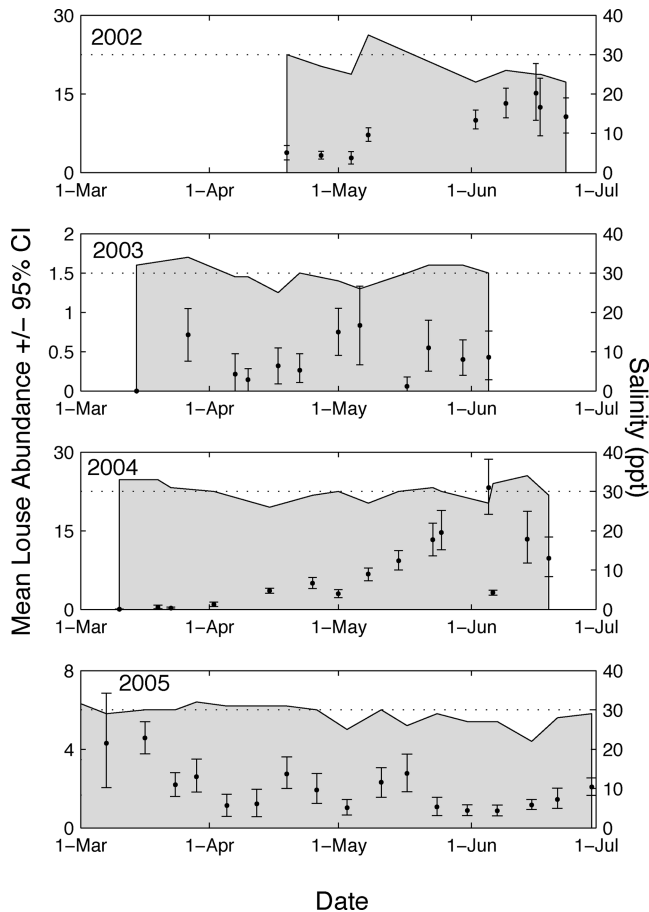


Figure 1. Salinity measurements (shaded regions and right axes) and mean louse abundance ($\pm 95\%$ CI) per juvenile pink and chum salmon during the juvenile salmon migration at the Glacier Falls site, Tribune Channel, Broughton Archipelago in 2002–2005. Salinity measurements were taken with a salinity refractometer at the location where juvenile salmon were collected. Sea lice and salinity data were collected by AM as part of a long-term research program. The 2002 data were published in Morton et al. (2004), the 2002–2004 data were published in Morton et al. (2005), and the 2005 data are previously unpublished but gathered according to the same protocols.

Influence of Parameter Estimates

Both Stucchi's model (Brooks, 2005) and Krkošek et al. (2005a) focus on the dispersion of sea lice from a single farm in Knight Inlet—the Doctor Islet farm. Stucchi's model predicts louse larvae released from the Doctor Islet farm will be carried approximately 40 km away before developing into copepodids. The analysis in Krkošek et al. (2005a) of larval spread from this same farm estimated the mode of the resulting copepodid distribution was situated ~ 0.5 km from the farm. As we will see, this difference is largely due to a poor correspondence in parameter values between Stucchi's model and empirical measurements. To fully understand this effect, it is useful to derive an analogue to Stucchi's model within a more tractable mathematical framework.

Stucchi's model is a numerical solution to the three-dimensional Navier-Stokes equations with tidal and freshwater forcing. It includes bathymetric structure at a resolution of

up to 50 m. It does not include the effect of wind on surface currents. Overlaid on this circulation model is a particle-tracking model that tracks the spread of particles released from two farm locations in the Broughton Archipelago (Doctor Islet and Glacier Falls). The inert buoyant particles were released every hour for 25 hours and their final locations were recorded after 5 and 10 days (in their model, nauplii develop into copepodids on day 5 and copepodids die on day 10).

Knight Inlet is ~ 100 km long and ~ 2 km wide. The simplest possible model of such habitat is a one-dimensional domain. In this framework, an analogue of the Stucchi model is simply the solution of the advection-diffusion equation

$$\frac{\partial \eta}{\partial t} = D \frac{\partial^2 \eta}{\partial x^2} - \gamma \frac{\partial \eta}{\partial x} \quad (1)$$

after $t = 5$ or $t = 10$ days following a pulse release of particles at $x = 0$ (the farm location) at $t = 0$. D is the diffusion coefficient and represents randomness in larval movement—the combined effects of bathymetry and tides. For illustrative purposes we will take $D = 1$ (Krkošek et al. (2005a) estimated $D = 0.26$ and $D = 0.95$ $\text{km}^2 \cdot \text{day}^{-1}$ for the Knight Inlet in April and May of 2003, respectively). The advective flow is γ and represents the average seaward flow of currents. The solution to this problem is well known; it is a traveling Gaussian with mean γt and variance $2Dt$.

The advective flow required by equation 1 to displace the mode of the copepodid distribution by 40 km is $\gamma = 8$ $\text{km} \cdot \text{day}^{-1} = 9.3$ $\text{cm} \cdot \text{s}^{-1}$. The flow measured at the Doctor Islet farm reported by Brooks (2005) is 1.4 $\text{cm} \cdot \text{s}^{-1} = 1.21$ $\text{km} \cdot \text{day}^{-1}$. This results in an overestimate of larval displacement by 6.6 times (Figure 2). It appears that this overestimate

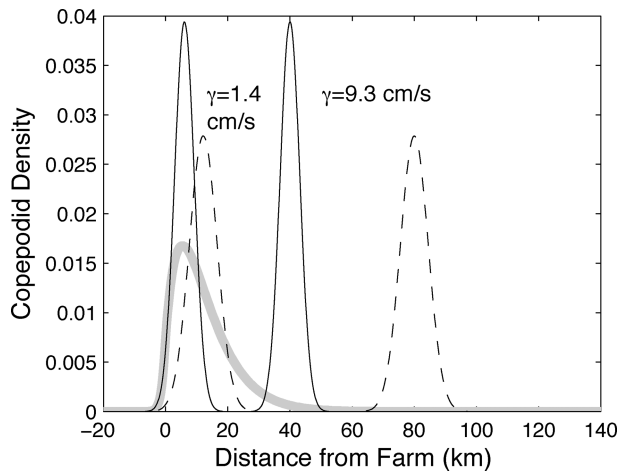


Figure 2. The effect of the advection parameter on predicted copepodid distributions around the Doctor Islet salmon farm, Broughton Archipelago, situated at $x = 0$. Distributions for copepodids are plotted using our analogue of the Stucchi model (equation 1) at times $t = 5$ d (emerging copepodids, thin solid lines) and $t = 10$ d (end of the copepodid lifespan, thin dashed lines). The leftward pair of thin lines result from empirically measured advective speeds ($\gamma = 1.4$ $\text{cm} \cdot \text{s}^{-1}$) and the rightward pair of curves result from advection speeds necessary to transport larvae 40 km in 5 days ($\gamma = 9.3$ $\text{cm} \cdot \text{s}^{-1}$). The diffusion coefficient was $D = 1$. For comparison the copepodid distribution predicted by the larval distribution models from Krkošek et al. (2005a) are also plotted (thick grey line) for the empirical advection parameter estimate, $\gamma = 1.4$ cm/s .

of advection occurs because Stucchi's model does not have the spatial resolution necessary to capture the combined effects of small scale bathymetry, tidal forcing, and wind. For the nearshore environment—where juvenile salmon and sea lice interact—this limitation will underestimate diffusive components of larval movement and overestimate advective flow.

The level of resolution required to model the detailed effects of wind, tide, currents, and small-scale bathymetry may be much higher than currently used in Stucchi's model. An alternative is to group advective and diffusive components of movement and estimate these parameters directly from field data. This was the approach of Krkošek et al. (2005a), which used a slightly different model formulation. That model had the form

$$\frac{\partial \eta}{\partial t} = D \frac{\partial^2 \eta}{\partial x^2} - \gamma \frac{\partial \eta}{\partial x} - \mu \eta \quad (2)$$

where the mean nauplii period of the louse lifecycle is μ^{-1} ; nauplii develop into copepodids at a constant rate μ . This model assumes the farm releases nauplii at a constant rate (so $\eta(x = 0, t) = \alpha$ is the density of nauplii adjacent to the farm) and that larvae cannot travel infinitely far ($\lim_{x \rightarrow \pm\infty} \eta = 0$). To find the resulting spatial distribution of nauplii we set equation (2) equal to zero and find the spatial steady state

$$\eta(x) = \alpha \begin{cases} \exp(a_1 x), & x \leq 0 \\ \exp(a_2 x), & x > 0 \end{cases}, \quad a_2 < 0 < a_1 \quad (3)$$

where $a_{1,2} = [\gamma \pm (\gamma^2 + 4\mu D)^{0.5}]/(2D)^{-1}$. We can scale this distribution into a probability density function (PDF) so it integrates to unity. This distribution now becomes a source distribution for copepodids. For simplicity and illustrative purposes, we make the same assumptions on larval movement and development as Stucchi's model in Brooks (2005): copepodids are subject to the same movement rules as nauplii and the lifespan of planktonic copepodids equals that of nauplii. Therefore, equation (2) also applies to copepodids. The predicted spatial distribution of copepodids is then found by convolving the scaled (PDF) version of equation (3) with itself once.

Krkošek et al. (2005a) coupled this model to a model of juvenile salmon migration and estimated the model parameters directly from field data of sea lice infecting juvenile pink and chum salmon as they migrated past the Doctor Islet salmon farm. That analysis estimated the seaward advective flow in Tribune Channel to be 1.11 and 3.4 $\text{cm}\cdot\text{s}^{-1}$ in April and May of 2003, respectively (Krkošek et al., 2005a). Similarly, the estimated seaward advective flow in Knight Inlet was $-0.0056 \text{ cm}\cdot\text{s}^{-1}$ (counter-clockwise around Gilford Island) in April 2003 and $4.64 \text{ cm}\cdot\text{s}^{-1}$ (seaward down Knight Inlet) in May 2003.¹

Effect of Larval Mortality

We have not yet not fully reconciled Brooks (2005) and Krkošek et al. (2005a). Figure 2 shows that copepodids are predicted to occur near the Doctor Islet farm. However, the mode of the copepodid distribution predicted by the Krkošek et al. (2005a) larval dispersion model is displaced about 5 km from its source. The fits of Krkošek et al. (2005a) to field data yield a mode in the planktonic copepodid distribution that is displaced about 0.5 km from this farm. What are we missing?

¹The Knight Inlet values differ from Krkošek et al. (2005a) (-0.1 km/day) because in that analysis the parameters were constrained to produce a counter-clockwise prevailing current around Gilford Island. This constraint was based on preliminary results from D. Stucchi's model in 2003 which predicted this flow.

Both equations (1) and (2) make the same assumptions on movement and mean developmental rates. The only difference is how they treat variance in developmental rates. Equation (1) assumes there is no variation in the durations of nauplii and copepodid stages, whereas the second model assumes these parameters are exponentially distributed. The latter assumption implies the probability a nauplius moults into a copepodid and the probability a copepodid dies are independent of age. Both models make biologically problematic, but different, assumptions. In the appendix we show how these models are related—they occupy two extreme endpoints of a single more general model that tracks nauplii age. The subtle difference in structure between equations 1 and 2 has little effect on predicted copepodid distributions (Figure 2). However, when one considers the effect of larval mortality the difference in model structure has an important effect. To incorporate mortality into equation (2) we introduce an additional term

$$\frac{\partial \eta}{\partial t} = D \frac{\partial^2 \eta}{\partial x^2} - \gamma \frac{\partial \eta}{\partial x} - \mu \eta - \phi \eta \quad (4)$$

where ϕ is the instantaneous death rate of larval lice. Johnson and Albright (1991) report that in flowing water at 10°C and 30‰, approximately 1/4 of larvae develop into active copepodids. This suggests that larvae are three times more likely to die than survive and so $\phi = 3\mu$.

Perhaps surprisingly, this brings the expected distribution of copepodids nearer to the source. This effect arises because variability in the nauplii period allows longer living individuals to travel farther. Including mortality and variability in louse developmental rates reduces the number of long distance dispersers. With empirical estimates for advection, developmental rates, and mortality rates we find that equation (4) yields a predicted copepodid distribution with a mode displaced about 1 km from the farm location (Figure 3). This agrees well with the estimate of 0.5 km obtained from the same model independently fit to field data (Krkošek et al., 2005a).

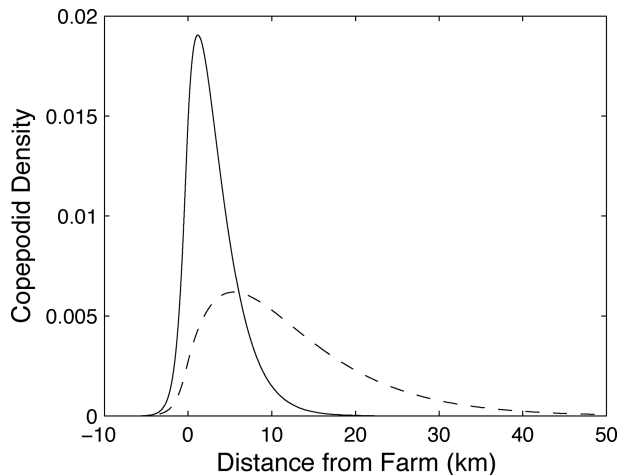


Figure 3. The effect of larval mortality on the expected distribution of copepodids around the Doctor Islet salmon farm located at $x = 0$. Both lines are steady state solutions to equation (4) for nauplii and copepodid movement with $\phi = 0$ (dashed line, no mortality) and $\phi = 3\mu$ (solid line, 3/4 of larvae perish). Solutions are found by convolving the scaled steady-state solution of equation 4 with itself once. Both curves are scaled to integrate to unity.

In contrast, incorporating mortality into our analogue of Stucchi's model (equation 1) has no effect on the expected copepodid distributions. The density is of course reduced, but the shape is the same. This is because the model implicitly assumes there is no variability in louse developmental rates.

Reconciling Model Predictions

Models for the spread of sea lice larvae around salmon farms reported by Brooks (2005) and Krkošek et al. (2005a) yield very different predictions of copepodid transport. Stucchi's model (Brooks, 2005) predicts long-distance transport of copepodids from their farm source—nauplii are transported 40 km from the Doctor Islet farm before they become infective copepodids. Krkošek et al. (2005a) fit a larval dispersion and salmon migration model to field data and found copepodid lice were distributed near their farm source—the mode of the distribution was ~ 0.5 km from the Doctor Islet Farm. An independent and empirical parameterization of the Krkošek et al. (2005a) larval dispersion model predicts a copepodid distribution with a mode displaced 1 km from the Doctor Islet Farm. This is consistent with other empirical studies in the Broughton Archipelago that have repeatedly observed high abundances of copepodids on juvenile pink and chum salmon near active salmon farms (Morton and Williams, 2004; Morton et al., 2004; Krkošek et al., 2005a; Morton et al., 2005). Brooks (2005) overestimates the transport of copepodids primarily because Stucchi's model overestimates advective flow (Figure 2). In addition, the inclusion of variability in louse development rates and larval mortality brings the copepodid distribution closer to its source (Figure 3).

Literature

Some thorough and conclusive studies were omitted from the literature review in Brooks (2005) while emphasis was placed on a few inconclusive studies. The interpretation of some studies—including several by the authors of this article—were sometimes mistaken and misleading. In this section, we correct these misinterpretations and highlight some important results in the literature overlooked in Brooks (2005).

European Studies

Many studies have correlated salmon farming with *L. salmonis* infestations of juvenile salmonids (e.g., Scotland: Mackenzie et al. (1998); Ireland: Tully et al. (1999); Norway: Bjørn and Finstad (2002) and Bjørn et al. (2001)). Perhaps the most thorough study was Tully et al. (1999), which sampled a total of 3166 sea trout from 22 bays throughout the entire Irish coastline over 5 years. This study found significantly higher sea lice infestations of juvenile sea trout in bays containing lice-infested farm salmon. Genetic analyses have found a lack of differentiation of *L. salmonis* between wild and farm populations indicating high inter-transmission rates between wild and farm salmon (Todd et al., 2004). Brooks (2005) omits these studies and instead focuses on a single inconclusive study that attempted to temporally correlate production stages of a fish farm with infestation levels of adjacent wild sea trout post-smolts (Marshall, 2003). In that study, only a weak relationship was found and most variation was attributed to seasonality. This is to be expected because such study designs import large seasonal variations in confounding factors (e.g., temperature and

salinity) that affect the dynamics of all lice and obfuscate the interactions between wild and farm salmon.

The review in Brooks (2005) of temporal and spatial patterns in nauplii and copepodid dispersion from salmon farms is more inclusive. Studies that found spatial and temporal correlations between salmon farming and nauplii and copepodid dispersion were included (Costelloe et al., 1998; McKibben and Hay, 2004; Penston et al., 2004). However, the interpretation of Costelloe et al. (1998) and its relation to the work of Krkošek et al. (2005a) was mistaken. Brooks (2005) emphasizes the finding of Costelloe et al. (1998) that wild adult sea trout likely produced the observed sporadic high abundances of larvae at river mouths 10 and 14 km from the studied farm. However another major finding by Costelloe et al. (1998) was omitted—that sustained high abundances of sea lice larvae occurred near the farm. This finding is consistent with the models of Krkošek et al. (2005a).

Studies in the Broughton Archipelago

Brooks (2005) focuses on empirical sea lice studies conducted by the Canadian Department of Fisheries and Oceans (DFO) but fails to mention that these studies were not designed to test the effect of salmon farms on sea lice infestations of wild juvenile salmon (Jones and Nemeč, 2004). Other studies have been optimally designed to test these effects. Morton and Williams (2004) and Morton et al. (2004) found that juvenile salmon were more highly infested near to than distant from salmon farms. Further, Morton et al. (2004) found that sea lice infections approached nil in regions without any salmon farms. Morton et al. (2005) found sea lice infestations of juvenile salmon dropped significantly when farms were fallowed and increased again when farms were stocked. Krkošek et al. (2005a) estimated that the transmission of lice from an isolated salmon farm to migratory sympatric wild juvenile salmon peaked near the farm at 73 times ambient levels and exceeded ambient levels for 30 km of migration route.

Krkošek et al. (2005a) presented evidence that successive generations of farm-origin lice spread within the juvenile salmon populations. Brooks (2005) suggests this was implausible because it implies juvenile salmon would have begun migrating already between January 22 and February 1. This argument, however, is based on the developmental rates of *L. salmonis*. The dominant louse observed in 2003 was *C. clemensi* (Jones and Nemeč, 2004). The developmental rates of *C. clemensi* are unknown, but based on the results from Krkošek et al. (2005a) these rates may be faster than for the larger *L. salmonis*.

Brooks (2005) suggests Krkošek et al. (2005a) were unaware of the production status of farms adjacent to Doctor Islet in their study of the impact of that farm on sea lice infections of sympatric migratory juvenile pink and chum salmon. This somehow leads to a conclusion that the results of Krkošek et al. (2005a) were implausible. In the field, the production status of most farms is plainly observable. Krkošek et al. (2005a), clearly stated that these smolt farms were grouped into a fallow/smolt category. This grouping was based on the assumption that smolts would not be major producers of larval lice, and could therefore be neglected as contributors of lice to the sympatric wild juveniles. This grouping is supported by the low levels of gravid lice reported on these farms in Brooks (2005).

Brooks (2005) challenges the credibility of Morton and Williams (2004), Morton et al. (2004), and Krkošek et al. (2005a) by alleging a lack of “quality control” in sea lice identification. In Morton and Williams (2004) and Morton et al. (2004), sea lice were identified using standard laboratory methods. Correspondences between the lead author of these studies and experts in DFO and Europe have confirmed the accuracy of the species identifications. The accuracy of nonlethal sampling methods used in Krkošek et al. (2005a) has been evaluated

in Krkošek et al. (2005b), but was overlooked in Brooks (2005). Brooks (2005) emphasizes the reduced taxonomic resolution of the nonlethal methods but fails to note that this does not affect the results and conclusions in Krkošek et al. (2005a).

Brooks (2005) emphasizes the DFO finding that sea lice were not associated with reduced growth or condition of their juvenile salmon hosts (Jones and Nemec, 2004) but fails to consider the several other factors that may produce this result. Morton and Routledge (2005b) found sea lice can be lethal to juvenile pink and chum salmon and Morton and Routledge (2005a) found high condition factor values were maintained by lice-infested fish for a time, but declined rapidly just prior to death. Fish with declining condition factor values not only died rapidly, but they exhibited behavior that would increase their risk of predation. Furthermore, sea lice are known to reduce the swimming performance of other salmonid hosts (Wagner et al., 2003). There is a high likelihood that these moribund fish are rapidly removed from the population by predators.

Conclusions

In a recent review of sea lice interactions between wild and farm salmon in the Broughton Archipelago, Brooks (2005) argues that the effects of temperature, salinity, and hydrodynamics combine to prevent the transmission of lice from farm salmon to sympatric wild juvenile salmon. The conclusions reached in Brooks (2005) were based on a flawed interpretation of how salinity affects louse development, a misunderstanding of how the timing of salinity change corresponds to the timing of the juvenile salmon migration, models of larval dispersion that overestimate the transport of copepodids, and a selective and misleading assessment of the literature. Our analysis of larval dispersion models has shown the (perhaps counterintuitive) result that sustained high abundances of copepodids should be expected near lice-infested salmon farms. Counter to the conclusions in Brooks (2005), the modeling and empirical work on sea lice interactions between wild and farm salmon are consistent and point to a strong association between salmon farming and recurrent sea lice infestations of juvenile pink and chum salmon in the Broughton Archipelago.

Acknowledgments

We are grateful to Allen Gottesfeld, Neil Frazer, and Larry Dill for comments on the article. This work was supported by an NSERC Canada Graduate Scholarship D and a D. Allen Birdsall Memorial Scholarship to MK; an NSERC Discovery grant and Canada Research Chair to MAL; and an NSERC Discovery grant to JPV.

References

- Bjørn, P., and B. Finstad. Salmon lice, *Lepeophtheirus salmonis* (Krøyer), infestation in sympatric populations of arctic char, *Salvelinus alpinus* L., and sea trout, *Salmo trutta* (L.), in areas near and distant from salmon farms. *Ices J. Mar. Sci.*, **59**: 131–139 (2002).
- Bjørn, P., B. Finstad, and R. Kristoffersen. Salmon lice infection of wild sea trout and arctic char in marine and freshwaters: the effects of salmon farms. *Aquac. Res.*, **32**: 947–962 (2001).
- Brooks, K. The effects of water temperature, salinity, and currents on the survival and distribution of the infective copepodid stage of sea lice (*Lepeophtheirus salmonis*) originating on Atlantic salmon farms in the Broughton Archipelago of British Columbia, Canada. *Rev. Fish. Sci.*, **13**: 177–204 (2005).

- Costelloe, M., J. Costelloe, G. O'Donohoe, N. Coghlan, M. Oonk, and Y. V. D. Heijden. Planktonic distribution of sea lice larvae, *Lepeophtheirus salmonis*, in Killary harbour, west coast of Ireland. *J. Mar. Biol. Ass. U.K.*, **78**: 853–874 (1998).
- Johnson, S., and L. Albright. The developmental stages of *Lepeophtheirus salmonis* (Krøyer, 1837) (Copepoda, Caligidae). *Can. J. Zool.*, **69**: 929–950 (1991).
- Jones, S., and A. Nemeč. Pink Salmon Action Plan: Sea lice on juvenile salmon and on some non-salmonid species caught in the Broughton Archipelago in 2003. Canadian Science Advisory Secretariat. Research Document. www.dfo-mpo.gc.ca/csas/ (2004). Last accessed Nov. 21, 2005.
- Keeling, M., and B. Grenfell. Understanding the persistence of measles: reconciling theory, simulation and observation. *Proc. Roy. Soc. Lond. B.*, **269**: 335–343 (2002).
- Krkošek, M., M. Lewis, and J. Volpe. Transmission dynamics of parasitic sea lice from farm to wild salmon. *Proc. Roy. Soc. Lond. B.*, **272**: 689–696 (2005a).
- Krkošek, M., A. Morton, and J. Volpe. Non-lethal assessment of juvenile pink and chum salmon for parasitic sea lice infections and fish health. *Trans. Am. Fish. Soc.*, **134**: 711–716 (2005b).
- Lloyd, A. Destabilization of epidemic models with the inclusion of realistic distributions of infectious periods. *Proc. Roy. Soc. Lond. B.*, **268**: 985–993 (2001).
- Mackenzie, K., M. Longshaw, G. Begg, and A. Mcvicar. Sea lice (Copepoda : Caligidae) on wild sea trout (*Salmo Trutta* L.) in Scotland. *ICES J. Mar. Sci.*, **55**: 151–162 (1998).
- Marshall, S. The incidence of sea lice infestations on wild sea trout compared to farmed salmon. *Bull. Eur. Ass. Fish Path.*, **23**: 72–79 (2003).
- McKibben, M., and D. Hay. Distributions of planktonic sea lice larvae *Lepeophtheirus salmonis* in the inter-tidal zone in Loch Torridon, Western Scotland in relation to salmon farm production cycles. *Aquac. Res.*, **35**: 742–750 (2004).
- Morton, A., and R. Routledge. Fulton's condition factor: is it a valid measure of sea lice impact on juvenile salmon? *N. Am. J. Fish. Man.* (in press). 2005a.
- Morton, A., and R. Routledge. Mortality rates for juvenile pink (*Oncorhynchus gorbuscha*) and chum (*O. keta*) salmon infested with sea lice (*Lepeophtheirus salmonis*) in the Broughton Archipelago. *Alaska Fish. Res. Bull.* (in press). 2005b.
- Morton, A., R. Routledge, C. Peet, and A. Ladwig. 2004. Sea lice (*Lepeophtheirus salmonis*) infection rates on juvenile pink (*Oncorhynchus gorbuscha*) and chum (*Oncorhynchus keta*) salmon in the near shore marine environment of British Columbia, Canada. *Can. J. Fish. Aquat. Sci.*, **61**: 147–157 (2004).
- Morton, A., R. Routledge, and R. Williams. Temporal patterns of sea lice infestation on wild Pacific salmon in relation to the fallowing of Atlantic salmon farms. *Am. J. Fish. Man.*, **25**: 811–821 (2005).
- Morton, A., and R. Williams. First report of the sea louse, *Lepeophtheirus salmonis*, infestation on juvenile pink salmon, *Oncorhynchus gorbuscha*, in nearshore habitat. *Can. Field. Nat.*, **117**: 634–641 (2004).
- Penston, M., M. McKibben, D. Hay, and P. Gillibrand. Observations on open-water densities of sea lice larvae in Loch Shiel, Western Scotland. *Aquac. Res.*, **35**: 793–805 (2004).
- Todd, C., A. Walker, M. Ritchie, J. Graves, and A. Walker. Population genetic differentiation of sea lice (*Lepeophtheirus salmonis*) parasitic on Atlantic and Pacific salmonids: analyses of microsatellite DNA variation among wild and farmed hosts. *Can. J. Fish. Aquat. Sci.*, **61**: 1776–1191 (2004).
- Tully, O., P. Gargan, W. Poole, and K. Whelan. Spatial and temporal variation in the infestation of sea trout (*Salmo trutta* L.) by the Caligid Copepod *Lepeophtheirus salmonis* (Krøyer) in relation to sources of infection in Ireland. *Parasitology*, **119**: 41–51 (1999).
- Wagner, G., R. McKinley, P. Bjørn, and B. Finstad. Physiological impact of sea lice on swimming performance of Atlantic salmon. *J. Fish. Biol.*, **62**: 1000–1009 (2003).

Appendix

Equations (1) and (2) arise as special cases of a more general model of larval dispersion that tracks nauplii age. The general model divides the nauplii period into a sequence of

m consecutive substages, where the duration of each substage is equal and exponentially distributed. With this framework, the nauplii period, τ , is gamma distributed with probability density

$$f(\tau) = \frac{(m\mu)^m}{\Gamma(m)} \tau^{m-1} e^{-m\mu\tau} \quad (5)$$

and variance $\sigma^2 = [m\mu^2]^{-1}$ (Lloyd, 2001; Keeling and Grenfell, 2002). Notice that when $m \rightarrow \infty$, then $\sigma^2 \rightarrow 0$ and the model converges to our analogue of Stucchi's model. When $m \rightarrow 1$ τ becomes exponentially distributed as in Krkošek et al. (2005a).