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Fish farms, parasites, and predators: implications for salmon population dynamics

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Abstract. For some salmon populations, the individual and population effects of sea lice (*Lepeophtheirus salmonis*) transmission from sea cage salmon farms is probably mediated by predation, which is a primary natural source of mortality of juvenile salmon. We examined how sea lice infestation affects predation risk and mortality of juvenile pink (*Oncorhynchus gorbuscha*) and chum (*O. keta*) salmon, and developed a mathematical model to assess the implications for population dynamics and conservation. A risk-taking experiment indicated that infected juvenile pink salmon accept a higher predation risk in order to obtain foraging opportunities. In a schooling experiment with juvenile chum salmon, infected individuals had increased nearest-neighbor distances and occupied peripheral positions in the school. Prey selection experiments with cutthroat trout (*O. clarkii*) predators indicated that infection reduces the ability of juvenile pink salmon to evade a predatory strike. Group predation experiments with coho salmon (*O. kisutch*) feeding on juvenile pink or chum salmon indicated that predators selectively consume infected prey. The experimental results indicate that lice may increase the rate of prey capture but not the handling time of a predator. Based on this result, we developed a mathematical model of sea lice and salmon population dynamics in which parasitism affects the attack rate in a type II functional response. Analysis of the model indicates that: (1) the estimated mortality of wild juvenile salmon due to sea lice infestation is probably higher than previously thought; (2) predation can cause a simultaneous decline in sea louse abundance on wild fish and salmon productivity that could mislead managers and regulators; and (3) compensatory mortality occurs in the saturation region of the type II functional response where prey are abundant because predators increase mortality of parasites but not overall predation rates. These findings indicate that predation is an important component of salmon–louse dynamics and has implications for estimating mortality, reducing infection, and developing conservation policy.

Key words: aquaculture; behavior; fish farms; Lepeophtheirus salmonis; Oncorhynchus *spp.; parasites; population dynamics; predation; salmon; sea lice.*

Introduction

Transmission of native sea lice (*Lepeophtheirus salmonis*) from farmed salmon is a novel factor affecting the population dynamics of wild salmon (Krkošek et al. 2007*a*, Ford and Myers 2008, Costello 2009, Krkošek 2010). Sea lice are parasitic copepods, ubiquitous on farmed and wild adult salmonids in the oceans of the northern hemisphere, and capable of causing host morbidity and mortality (Boxaspen 2006, Costello 2006). In the absence of salmon farms, juvenile Pacific salmon are separated from sea lice during early marine life because most lice are on wild adult salmon that are located offshore when juvenile salmon first enter the sea and rear in coastal marine waters (Krkošek et al. 2007*b*). Although salmon farms may increase the exposure of wild juvenile salmon to sea lice, wild juvenile pink (*Oncorhynchus gorbuscha*) and chum (*O. keta*) salmon naturally experience high mortality in the absence of infestation owing to predation, particularly from coho salmon (*O. kisutch*) smolts (Groot and Margolis 1991). To understand how lice affect wild salmon population dynamics, it is therefore necessary to evaluate how

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parasitism interacts with predation. For example, parasitism could increase overall predation rates, leading to increased mortality. Alternatively, parasites could redistribute predation upon infected individuals without changing the overall predation rate on the prey population. In such a scenario, predators provide an ecosystem service by selectively removing infected fish from diseased wild fish stocks.

Predation and infectious disease can interact in complex ways and have important consequences for host and prey populations (Hudson et al. 1992, Packer et al. 2003, Hall et al. 2005, Hatcher et al. 2006). For juvenile pink and chum salmon, predation might mediate the ecological effects of increased parasite exposure via a variety of mechanisms. Parasitism may affect host behaviors such as leaping, foraging, and habitat selection in ways that have consequences for predation (Webster et al. 2007). Infected salmon may accept increased predation risk to meet increased foraging requirements to maintain physiological defenses to infection (Jones et al. 2007) or to replace resources that are either sequestered directly by parasites or lost to the environment through damaged surface tissues (Pike and Wadsworth 2000). Parasites can affect fish schooling behavior, which can interfere with the antipredator functions of diluting predation pressure and confusing predators (Landeau and Terborgh 1986). Infection could also directly affect the ability of a fish to evade a predatory strike by affecting the processing of sensory cues and associated rapid response in prey (Barber et al. 2000, Blake et al. 2006). In this paper we report on a series of field-based experiments and a mathematical model investigating the consequences of sea louse infection on juvenile pink and chum salmon tolerance of predation risk, schooling behavior, predation rates, and population dynamics.

The experiments involved either juvenile pink salmon or juvenile chum salmon, and sometimes both. During early marine life, these species are ecologically equivalent: they are similar in size, both migrate from freshwater to saltwater as fry, and they form mixed-species schools that occupy shallow (often intertidal) habitats (Groot and Margolis 1991). The particular species used in each experiment was determined by the availability of fish with appropriate louse burdens in the field. Although the primary focus of this work is on the effects of *L. salmonis* infestation on pink and chum salmon, a second species of louse, *Caligus clemensi*, was sometimes present on the juvenile salmon. We clarify our treatment of *C. clemensi* on a per experiment basis in the section *Experimental methods*. The mathematical modeling section focuses on *L. salmonis* and pink salmon.

The first experiment assessed whether lice increase the risk of predation that juvenile salmon will accept in order to obtain a foraging opportunity, as measured by the time to resume feeding following a simulated predatory strike. The schooling experiments used digital image analysis to investigate if lousy juvenile salmon exhibited deviant schooling behavior relative to uninfected conspecifics, specifically with regard to nearest-neighbor distances and positioning within schools. These first two experiments therefore tested whether lice affect juvenile salmon behavior in ways that may increase predation risk. The next two experiments directly tested whether predators preferentially select lice-infected prey in individual and group settings. In individual choice trials, one infected and one uninfected juvenile salmon were exposed to sea-run cutthroat trout (*O. clarkii*) predators. In addition to assessing differential survival, we also tested if differential survival was due to predators targeting infected fish and/or because infected fish were less likely to evade a predatory strike. In group predation experiments, we compared louse distributions among individuals within shoals of juvenile salmon before and after exposure to groups of coho salmon smolts in large net pens, to test for selective predation in more natural group interactions. To investigate the implications of the experimental results for salmon population dynamics, we developed a mathematical model of sea lice and pink salmon that combines classical theoretical approaches to modeling predator–prey with host–parasite dynamics. The empirical and theoretical results have important implications for understanding sea lice and salmon population dynamics that are also highly relevant to management and policy.

Experimental Methods

Louse life cycle

Lice have a life cycle that consists of free-swimming nauplii stages and an infective free-swimming copepodite stage. Once attached to a host fish, the copepodite develops through chalimus stages and then motile stages. Motile stages include pre-adult and adult stages, the latter of which reproduce sexually. Adult females extrude egg strings from which nauplii hatch, completing the life cycle.

Fish collection and handling

The experiments were conducted in the Broughton Archipelago, on Canada's west coast, between April and June 2004–2007. During this time a series of *L. salmonis* infestations of juvenile wild pink and chum salmon occurred and were linked to salmon farms (Krkošek et al. 2006). We used fish collected from these wild populations in a series of investigations into the effects of infection on schooling behavior and predation risk. We used a 35×3 m beach seine net with 4-mm mesh size to catch the fish and temporarily maintained them in 18.9-L buckets for ~30 minutes during transportation to a field laboratory consisting of floating docks and flow-through ocean enclosures. Once there, fish were either stored in ocean enclosures ($1.5 \times 1.5 \times 0.5$ m depth) for up to three days and maintained on commercial salmon feed or allowed to acclimatize to ocean enclosures (1 hour) before being used in an

experiment. During a set of experiments conducted simultaneously at the same facility in which naturally infected juvenile salmon were monitored daily for sea lice (Krkošek et al. 2009), we did not observe new copepodites on juvenile salmon, indicating that fish were unlikely to become infected during storage at the facility. However, some loss of lice may occur during storage or trials. We controlled for this when selecting uninfected fish for experiments by excluding those that had scars associated with lice. To separate the effects of natural louse mortality from those induced by predation, we included appropriate control replicates and statistical analysis. Details of individual experiments are provided.

Risk-taking

We used digital video analysis to investigate the effect of sea louse infection on the time it took juvenile pink salmon to resume feeding following a simulated predator strike. We used wild juvenile salmon and sorted them into infected and uninfected groups as previously described in (Krkošek et al. 2005*b*). Uninfected fish were those that had no lice (*L. salmonis* or *Caligus clemensi*), no evidence of past infection (no louse scars), and no evidence of other tissue damage (predation scars). Infected fish were those with one adult female *L. salmonis*, evidence of louse feeding (louse scars), no other louse species present, and no evidence of external scars or wounds from predators. At the lab, we placed groups of noninfected and infected fish into fiberglass ocean enclosures. There were 33–41 fish in each group, with infected and uninfected group pairs at matching densities. That is, infected and control pairs within a trial had the same density but the density varied slightly among trials. There were seven trials in total, each consisting of an infected and uninfected group held concurrently in separate experimental enclosures.

The experimental enclosures were 1.5 m × 1.5 m × 0.5 m deep fiberglass tubs with an open top and mesh windows cut in the submerged sides. In each corner, we placed artificial kelp made of floating strips of black plastic fastened to the floor. In the center of the floor of each tub, we fastened a 1 m diameter circle of white corrugated plastic to provide high contrast of the fish for video recording. There was also a floating ring (30 cm diameter) made of closed cell foam held in the center by fishing line, marking the location of food. The darker edges of the tub, where fake kelp was fastened, comprised a low-risk zone, whereas the open center with the white bottom and feeding ring was designated as a high-risk zone (Fig. 1). Each tub was outfitted with a fake camera model suspended centrally, about 1.75 m above the water, to habituate the fish to the overhead presence of a camera that would later be used to record each trial.

We prepared the fish by providing hourly feeding opportunities in the high-risk zone for 1.5 days before each trial when we simulated a predatory strike. After introducing the fish into each enclosure, we fed them

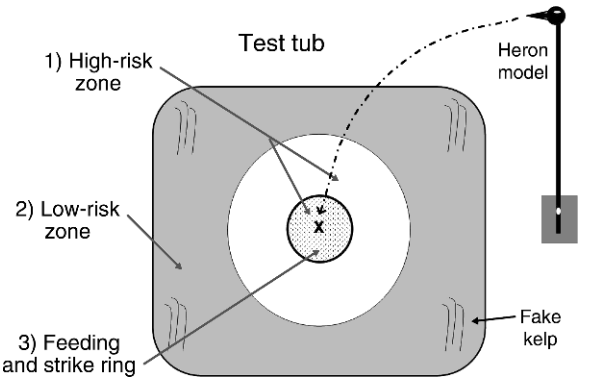

FIG. 1. Schematic drawing of the risky feeding experiment with wild juvenile salmon in 1.5 m × 1.5 m × 0.5 m deep fiberglass ocean tubs, showing the high-contrast area in the center defining the high-risk area, the central feeding ring where feed was introduced, the artificial kelp providing cover in the corners, and the model heron.

commercial fish food (EWOS micro #0–1; EWOS Canada, Surrey, British Columbia, Canada) by sprinkling an excessive quantity of food onto the water inside the ring each daylight hour (between 05:00 and 22:00 hours) during the day of capture and the first day following capture. Trials occurred on the second day following capture, during which fish were not fed until the trial commenced. For each trial, we replaced the fake camera model with a Sony Hi8 Handicam for video recording. We also fastened a wooden model of a heron head and beak to the adjacent dock such that it would strike the water inside the floating ring upon release (Fig. 1). We acclimated the fish to the heron and the presence of observers for 15 minutes before food was sprinkled in the floating ring, marking the start of a 15-minute trial. When >60% of the fish were feeding (judged by visual estimation), we released the model heron such that its beak struck the surface of the water, with the model then rebounding and assuming its original position. Each group was tested only once. After each trial, we reexamined each fish, measuring fork length and counting lice and scars, and then released the fish near their point of capture.

From the video recordings, we calculated the amount of time it took for 50% of the number of fish that were originally feeding to return to the central ring after they scattered into the artificial kelp during the strike. First, we converted the video to digital format using the software DazzleTM150 interface by Pinnacle Studio (Dazzle/Pinnacle Studio, Mountain View, California, USA, then burned the video to DVD, and analyzed it using LG Cyberlink™ PowerDVD 6 (Cyberlink, Fremont, California, USA) on a computer. At every five-second position in each trial, we calculated the percentage of fish in the center ring, and so could estimate when 50% of the fish returned to the high-risk area. To analyze these data, we applied a time to event (survival) analysis for censored data. The data are called

censored because in some trials fish did not resume feeding, so the time to resume feeding is not known but rather exceeds the time of observation. We obtained Kaplan-Meier (KM) estimates of the probability of not returning by using the observed return times. We fit parametric survival models with louse infection as a fixed factor to the KM survival estimates by maximum likelihood using exponential, Weibull, and lognormal error distributions. We used Akaike's information criterion corrected for small sample sizes (AIC_c) to select the distribution that best fit the data (Burnham and Anderson 2002). We used *t* tests to test for differences in fork length between infected and noninfected groups.

Schooling behavior

We used digital image analysis to observe the effect of infection on the positioning of juvenile chum salmon within a school of uninfected conspecifics. Uninfected fish had no motile or chalimus stage lice or other physical damage (based on observations of fish in seawater-filled plastic bags). Infected fish had one or more motile *L. salmonis* and some had physical damage to surface tissues caused by the lice. Schooling behavior was induced in a circular experimental pool (183 cm diameter × 39 cm deep with a 10-cm grid on the bottom) with a circular current produced by a central 500 GPH (gallons/h; 1 gallon = 4.55 L) bilge pump connected to a radial spray bar (2.5-cm PVC piping with 5 mm diameter holes at 15-cm intervals). Each trial consisted of three hours of observation during which we photographed schooling juvenile chum salmon (one infected and 29 uninfected individuals) every six minutes, totaling 30 observations per trial. We photographed the fish with a 3.1-megapixel digital camera mounted ~2 m above the pool, and simultaneously took a side photo using a 5.0-megapixel camera to identify the infected fish. If the fish were not schooling (approximately one-fourth of the trials), we induced schooling with a brief hand wave over the pool 10–30 seconds prior to a photo. We maintained the water height between 10 and 12 cm to minimize variation in vertical positioning of fish and maintained water temperature within 5°C through periodic exchange with fresh seawater. The fish used in each trial were of similar size and were selected from the same ocean enclosure where they had been held for at least 12 hours and up to one week. After each trial, we removed the fish from the pool using seawater-filled plastic bags and stored them in buckets while we inspected each fish for lice and made body depth and length measurements (Krkošek et al. 2005*b*). We released the fish near their location of capture following a minimum 30-minute recovery period.

To analyze the photographs, we used ImageJ 1.36b software (*available online*).[8] For each photo, we calculated (1) whether the infected fish was in the school center or periphery, (2) whether the infected fish was in the front or back of the school, and (3) nearest-neighbor distances (NNDs) for all schooling fish. Peripheral and central zones were defined by first delineating the school boundary with a polygon connecting the minimum number of heads necessary to circumscribe the school. This polygon was then reduced, while maintaining its center and rotation, until half of the fish heads were within the polygon and half of the fish heads were between the polygon and the school boundary. Front and back zones were delineated by a line, perpendicular to the school orientation axis, that divided the school in half. The school orientation axis was the mean of angles of the vectors connecting the tail to the head of individual fish. NND was the shortest head-to-head distance between a focal individual and adjacent fish (Barber and Huntingford 1996). We did not calculate NND for fish that were not schooling (NND > 3 body lengths), including the infected fish. We did not use photos when the infected fish's head was not visible. All analyses were done in two dimensions because the shallow depth (10–12 cm) minimized variation in vertical positioning.

There were 16 trials with 30 observations each. We analyzed the data at the among-trials level using: proportion periphery (the proportion of photos within a trial in which the infected fish was situated in the periphery of the school); proportion back (the proportion of photos within a trial in which the infected fish was situated in the back of the school); and NND differences (the mean, across observations within a trial, of differences in NNDs between the infected and the mean of uninfected fish. We used one-sample *t* tests on arcsine square-root transformed proportion data (to normalize variance) to test if proportion periphery or proportion back exceeded 0.5. We used a one-sample *t* test to test if the mean of NND differences differed from zero. We then used regression analysis to investigate if the body mass of the infected fish affected proportion periphery, proportion back, and NND differences. Body mass was calculated from body depth and length measurements using the allometric relations from (Krkošek et al. 2005*b*).

Predator choice

We observed predator–prey interactions in enclosures where individual cutthroat trout predators were provided a choice between one infected and one uninfected pink salmon prey. Infected fish carried, on average, 2.4 (range 1–5) motile *L. salmonis*, whereas uninfected fish did not carry a louse species and had no scarring that could be associated with previous infections or predators. We used five trout, averaging 22.6 cm fork length (length from mouth to fork in tail), caught by hook and line. The trout acclimated for 4 days, being fed juvenile pink salmon at ~2% body mass per day before trails began. The prey were housed in two enclosures, one for

[8] ⟨http://rsb.info.nih.gov/ij/⟩

infected fish and the other for uninfected fish (as per observations through seawater-filled plastic bags), and acclimated for a minimum of 24 hours. For half of the trials ($n = 30$), fish were examined for lice and morphometrics (as previously described in the schooling experiment), were size-matched, and then were allowed 15 minutes to acclimate before being exposed to a predator. Each trial was conducted in a $1.5 \times 1.5 \times 0.5$ m ocean enclosure and began with a mesh division separating the juvenile pink salmon from the cutthroat trout. After ~1 minute, the divider was lifted and the trial began and continued until one juvenile pink salmon was consumed. In order to test if the handling required for sea lice enumeration and fish morphometrics had an infestation-dependent effect on predation susceptibility the same experiment was replicated ($n = 30$) without handling the fish, by size-matching one infected and one uninfected fish size by eye in a shallow bucket by sequentially adding and removing uninfected fish. This procedure was always performed by the same person (B. M. Connors) and was found to match fork length to within ± 1 mm ($n = 10$). The unconsumed fish was then examined after the trial terminated. We collected data on the time to capture, handling time (time to consumption post capture), the total length of the trial, and the number of predation attempts (strikes) per prey fish. We used a binomial test to evaluate if infected and uninfected fish experienced the same predation risk, a t test to test for differences in the time to capture between infected and uninfected fish, and Wilcoxon rank sums tests to test if the number of strikes prior to capture differed significantly between infected and uninfected fish. We used linear regression to test for the effects of fork length and body depth on time to capture and handling time.

Group predation

We conducted a group predation experiment in which a random sample of 200 naturally infected pink or chum salmon were exposed to 50 coho smolt predators in a net pen for 36–48 h. There were five groups of 50 coho used during 13 predation trials conducted in 2005 and 2006. The coho salmon (90–140 mm fork length) were collected by beach seine and housed in ocean enclosures or net pens. There were four net pens used. Two were 7.5 m deep and 14.5×10.5 m across, and two were 9.5 m deep and 20×20 m across. The coho were not fed for three days before the trials began and for three days between each trial. For each trial, ~400 juvenile pink salmon were collected in one beach seine catch; 100 of these fish were assayed for sea louse infections and morphometrics (Krkošek et al. 2005*b*) and then released. A separate group of 200 fish from the same catch was used for the experiment. This protocol allowed us to indirectly estimate the sea lice and body size distributions of fish without subjecting the fish used in the experiment to the handling associated with sea lice identification and fish morphometrics. Of the remaining fish, 200 were introduced into a net pen using seawater-filled plastic bags and were allowed 1 hour to acclimate. The excess fish were released. During acclimation, the pen was divided in half to separate the juvenile pink or chum salmon from the coho smolts. Trials began by removing the barrier, which allowed the coho smolts to access the juvenile pink or chum salmon. After 36–48 hours, the trial was terminated and all juvenile pink or chum salmon and coho salmon were assayed for sea lice and (for pink and chum salmon only), morphometrics. We also conducted six control trials in which the same experiment was repeated, but the coho predators were replaced with uninfected juvenile pink or chum salmon (control fish were pinks when the infected fish were chums, and vice versa). The control trials were conducted to control for sea lice mortality not induced by predation. The uninfected fish were selected by examining them in seawater-filled plastic bags.

We analyzed the group predation data for each individual trial and also for the entire collection of control and treatment trials. We used bootstrapped two-sample t tests to test for significant differences in louse abundance between the start and end of individual trials (abundance defined as the average number of lice per fish). We used t tests on changes in abundance (posttrial minus pretrial abundance) within predation and control trials to test if there was a decline in louse abundance within the two treatment groups. We also used a generalized linear model with Poisson error on pretrial data to look for confounding relationships between louse abundance and fish size (fork length). To determine if changes in sea louse abundance during the trials could be attributed to predation, we used a generalized linear model with Poisson error and random effects to conduct an analysis of covariance between control and predation treatments. The random effects were assigned to individual trials to account for the nested structure of the data. The dependent variable was the number of sea lice on individual fish after a trial, and the independent variables included juvenile salmon species (pink or chum), the mean abundance of sea lice on the prey before the trial began, and a two-level treatment factor for control or predation trials. We tested whether predation contributed significantly to the abundance of sea lice at the end of the trials by including an interaction term between treatment and initial sea louse abundance.

Experimental Results

Risk-taking

Results of the experiment are summarized in Table 1. There was no difference in fork length between infected and noninfected groups (mean $\pm$ SD: noninfected 74.18 $\pm$ 1.52 mm, infected 75.6 $\pm$ 1.83 mm; $t = -1.57$, $\mathrm{df} = 12$, $P > 0.05$). Hence, we did not include fish fork length as a covariate in the survival analysis. Although the lognormal survival model was best supported by the data, there was little information in the data to

TABLE 1. Salmon risk-taking experiment: louse presence, number of fish per group, return time at which 50% of the fish resume feeding, fish fork length, louse prevalence by age and sex of the fish, and number of louse scars at the end of the trials.

Trial	Lice	Fish group size	Fish return time (s)	Fish length (mm)	Louse prevalence (%)				Motile scars
					Adult female	Adult male	Pre-adult	*Caligus* on juvenile	
1	yes	40	70	74.8 ± 6.4	100	32.5	0	5	8.3 ± 4.5
2	yes	41	210	73.6 ± 5.7	100	12.2	2.4	7.3	9.6 ± 5.8
3	yes	39	660+	73.0 ± 4.8	74.4	7.7	2.6	0	10.4 ± 7.1
4	yes	33	65	77.8 ± 5.3	60.6	9.01	12.2	0	14.4 ± 14.2
5	yes	35	150	77.0 ± 5.6	45.7	28.6	0	2.9	9.9 ± 8.2
6	yes	35	65	77.2 ± 5.5	74.3	14.3	2.9	2.9	13.0 ± 11.0
7	yes	34	115	75.7 ± 6.5	76.5	29.4	2.9	8.8	9.7 ± 7.4
1	no	37	90	72.3 ± 3.8					
2	no	40	130	73.1 ± 4.8					
3	no	38	295+	73.3 ± 3.7					
4	no	39	615	73.7 ± 4.9					
5	no	39	155	75.0 ± 4.2					
6	no	33	805+	75.2 ± 5.9					
7	no	36	315+	76.7 ± 7.8					

Notes: Values for fish fork length (length from mouth to fork in tail) and the number of louse scars (motile) are given as mean ± SD. Return times with "+" denote censored data (the true value is unknown but greater than shown). The experiment was conducted three times, with three replicate pairs for trial 1, three replicate pairs for trial 2, and one pair for trial 3. Although the primary focus of this work is on the effects of *Lepeophtheirus salmonis* infestation on pink and chum salmon, a second species of louse, *Caligus clemensi*, was sometimes present on the juvenile salmon.

distinguish among exponential, lognormal, and Weibull survival models (Table 2). Using each model, we therefore tested for an effect of lice on the time to resume feeding. The survival analysis indicated that infected groups returned to the high-risk area more quickly than noninfected fish (Table 3). The lognormal survival model showed a significant effect of louse infection on time to resume feeding at alpha = 0.05, whereas the exponential and Weibull models were marginally insignificant (Table 3). Overall, the lognormal survival model provided an excellent qualitative fit to the data (Fig. 2). Based on this model, the predicted average time for 50% of the fish to return to the high-risk area was 140.5 s for the infected group and 463.9 s for the uninfected groups (Fig. 2). In some trials, the total prevalence of lice as estimated after the trial was less than 100%, indicating some mortality and/or movement of lice.

Schooling behavior

In total, there were 480 photos, of which 18 photos were excluded because the infected fish was obscured. This yielded 462 observations on positioning within schools and 13 069 nearest-neighbor distances. The 480 juvenile chum salmon ranged in mass from 1.08 to 4.26 g, with a mean of 2.24 g. Infected individuals did not significantly differ in body mass from the average mass of uninfected individuals in the same trial (paired-sample t test, $t = 1.80$, df = 15, $P = 0.092$). Sea louse infection had a significant effect on schooling behavior. Infected fish were typically in the periphery (mean proportion of fish = 0.61; t test on arcsine square-root transformed data, $t = 2.20$, df = 15, $P = 0.044$) and back (mean = 0.66; t test on arcsine square-root transformed data, $t = 3.70$, df = 15, $P = 0.0021$) of the school. Furthermore, infected fish were more likely to have larger NNDs than uninfected fish (average 6.4 mm farther than uninfected fish; t test, $t = 2.18$, df = 15, $P = 0.045$). The effect of sea louse infection on schooling behavior decreased with increasing host body mass (Fig. 3). There was a strong negative relationship between fish mass and the proportion of photos in which the infected fish was situated in the periphery of the school ($y =$

TABLE 2. Summary of model selection statistics for the three survival models used to analyze the time-to-return data in the risky feeding experiment.

Model	k	AIC_c	ΔAIC_c	w_i
Lognormal	3	128.05	0	0.459
Exponential	2	128.07	0.023	0.454
Weibull	3	131.36	3.316	0.087

Note: Shown are the number of model parameters (k), Akaike information criterion (AIC), differences in AIC between each model and the best model (ΔAIC_c), and the Akaike weights (w_i) indicating the probability that each model is the best model within the set of models (Burnham and Anderson 2002).

TABLE 3. Statistical tests for the effect of lice on the time it took juvenile pink salmon to return to the high-risk feeding zone following a simulated predator attack in the risky feeding experiment.

Model	df	R	P
Lognormal	11	3.95	0.0468
Exponential	12	3.55	0.0594
Weibull	11	3.53	0.0601

Notes: Tests were conducted as likelihood ratio tests to determine if including lice presence in the survival model improved the fit of the model to the data. Results are shown for each of the three survival models. Shown are the degrees of freedom, the likelihood ratio statistic (R), and the P value from the likelihood ratio test (Hilborn and Mangel 1997).

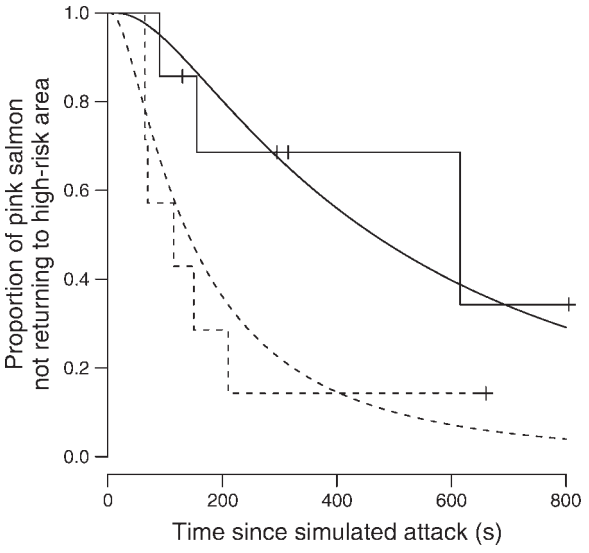

FIG. 2. Time spent by juvenile pink salmon (*Oncorhynchus gorbuscha*) infected (dashed lines) and uninfected (solid lines) by sea lice (*Lepeophtheirus salmonis*) before returning to the high-risk feeding zone following a simulated predatory strike in the risky feeding experiment. Shown are the proportions of trials in which fish did not return to the high-risk area for the corresponding time since the simulated predatory strike. Results are illustrated by the lognormal survival models (smooth curves) as well as Kaplan-Meier step plots, in which each step down represents the time at which a trial had fish return to the high-risk area. Hatch marks on the step plots indicate censored data (times at which the trial time of 15 minutes had ended).

$-0.32x + 1.28$; intercept $P < 0.001$ and slope $P < 0.001$). The relationship between fish mass and the proportion of photos in which the infected fish was situated in the back of the school was negative but weak ($y = -0.14x + 0.97$; intercept $P < 0.001$ and slope $P = 0.069$). Finally, there was a negative effect of mass on NND differences between infected and uninfected fish ($y = -12.0x + 31.7$; intercept $P = 0.0064$ and slope $P = 0.02$).

Predator choice

In each set of trials (i.e., prey handled before trials and not handled before trials), infected fish were eaten significantly more often than uninfected fish (22 trials out of 30 in each case; binomial test $P = 0.012$). The number of motile *L. salmonis* on infected fish was 2.3 ± 1.2 lice/fish (mean ± SD) and did not differ significantly between predation events when an infected fish was captured and those when an uninfected fish was captured (Kruskall-Wallis test, $P > 0.05$), meaning that the small variation in louse abundance on the infected fish did not affect the outcome of the experiment. Because there was no effect of handling (both sets of trials had the same results), we pooled the data for the following analyses. Time to capture did not vary between parasitized and unparasitized individuals (mean ± SD; for parasitized prey, 173 ± 435 minutes; for unparasitized prey, 173 ± 413 minutes; independent-samples t test, $P > 0.05$), nor was there a relationship between length or body depth and time to capture or handling time (linear regression; $P > 0.05$). The number of strikes (predation attempts) to capture was not significantly different between parasitized and unpara-

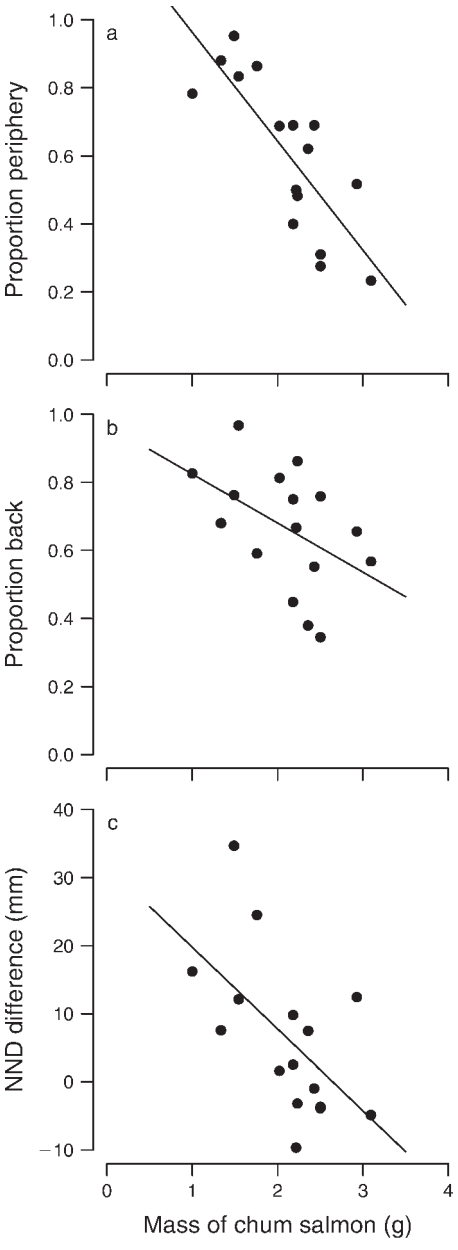

FIG. 3. Effect of body mass on schooling behavior of juvenile chum salmon (*Oncorhynchus keta*) infected with sea lice: (a) proportion periphery is the proportion of photos in which the infected fish was situated in the periphery of the school; (b) proportion back is the proportion of photos in which the infected fish was situated in the back of the school; (c) NND difference is the mean difference in nearest-neighbor distances between infected and uninfected fish. Each dot represents an individual trial, and the lines are linear regressions. See *Results* for detailed statistical results.

TABLE 4. Group predation experiment: number of juvenile pink or chum survivors (n) for each of 13 trials, before-trial mean abundance of chalimus and motile lice (B), and the difference (D) in mean louse abundance, with 95% CI, before and after exposure to predators.

		Chalimus lice (no./fish)				Motile lice (no./fish)			
Species	n	B	D	CI (L)	CI (U)	B	D	CI (L)	CI (U)
Pink	36	0.01	−0.01	−0.08	0.04	1.71	0.29	−0.20	0.72
Pink	76	0.14	0.07	−0.02	0.15	1.09	0.06	−0.29	0.44
Pink	58	0.14	0.05	−0.07	0.16	1.09	0.12	−0.27	0.46
Pink	68	1.33	0.80	0.41	1.23	0.96	0.45	0.17	0.73
Pink	116	0.73	0.49	0.23	0.78	0.96	0.55	0.25	0.85
Pink	124	0.55	0.34	0.08	0.65	0.82	0.62	0.38	0.85
Pink	98	0.10	0.05	−0.02	0.14	0.43	0.15	−0.05	0.36
Chum	72	0.05	0.05	0.01	0.11	1.25	0.09	−0.16	0.36
Chum	121	0.68	0.20	−0.02	0.39	0.34	0.09	−0.06	0.26
Chum	44	0.62	0.14	−0.12	0.41	0.34	0.00	−0.19	0.17
Chum	122	0.81	0.20	−0.03	0.41	0.24	0.04	−0.10	0.19
Chum	84	0.30	−0.08	−0.27	0.09	0.22	0.04	−0.09	0.19
Chum	43	0.40	−0.07	−0.33	0.17	0.16	0.02	−0.13	0.17

Notes: Bootstrapped 95% CI is shown with lower (L) and upper (U) limits. Exposure to predators (coho salmon smolts) lasted 36–48 h.

sitized individuals (parasitized, 2.1 ± 2.7 strikes; unparasitized, 1.7 ± 2.7 strikes; Wilcoxon rank sums test, $P > 0.05$).

Group predation

We conducted six control trials and 13 predation trials. There were four chum and two pink trials comprising the control data and seven pink and six chum trials comprising the predation data. At the end of each predation trial there were, on average, 81.7 juvenile pink or chum salmon remaining from the initial 200 that were introduced into the net pen (Table 4). Some trials demonstrated strong selective predation by coho salmon smolts on smaller and infected juvenile salmon (Fig. 4). There were five occurrences in which louse abundance was significantly related to fish size in the pretrial data, and of these there were two occurrences in which the relationship was negative (generalized linear models with Poisson error). Not all trials showed significant differences in mean louse abundances (for chalimi or motiles) before and after a trial (Table 5). A t test on the mean lice differences (posttrial minus pretrial louse abundance) in the predation trials revealed that there was a loss in lice during the predation trials (one-sample t test, $t = -3.12$, $\mathrm{df} = 12$, $P = 0.009$). There was also a decline in louse abundance during the control trials (one-sample t test, $t = -5.74$, $\mathrm{df} = 5$, $P = 0.0023$). The decline in louse abundance during the trials was significantly related to initial louse abundance for predation trials (linear regression, slope $= -6.4$, $P = 0.0016$), but not for control trials (linear regression, slope $= -0.035$, $P = 0.76$). The analysis of covariance using the generalized linear model with random effects found a significant interaction between treatment (predation or control) and initial louse abundance (Table 5), indicating that predation had a significantly negative effect on sea louse abundance at the end of a trial. Although there were only 13 trials involving coho predation, there was a detectable, but weak, relationship between the number of fish consumed in a trial and the average abundance of motile-stage lice at the beginning of the trial (linear regression, $P = 0.09$).

MODEL

The preceding experiments suggest that sea louse infection increases the predation risk of juvenile salmon. To evaluate the effects on salmon and louse population dynamics, we developed a mathematical model of *L. salmonis* and pink salmon that combines parasitism and predation. Previous modeling efforts have considered the effect of parasitism on predator–prey dynamics (Ives and Murray 1997) and the effect of predation on host–parasite dynamics (Hudson et al. 1992, Packer et al. 2003). These previous models have explicitly tracked host–parasite dynamics using the traditional Anderson-May macro-parasite model formulation (Anderson and May 1978, Grenfell and Dobson 1995) while coupling the host–parasite model to the dynamics of a predator population (Ives and Murray 1997) or incorporating predators as an exogenous fixed variable (Hudson et al. 1992, Packer et al. 2003). The details of predation have also varied among models, with functional responses taking on a type I (Hudson et al. 1992), type II (Ives and Murray 1997), or generalized (Packer et al. 2003) form. Here we build on previous modeling efforts but modify the details to better represent the biology of sea lice and salmon.

A main consideration of our model development is the separation of timescales for the parasite and the host. The period when juvenile salmon are exposed to increased sea lice from salmon farms is ~2–3 months (Krkošek et al. 2006, Krkošek et al. 2009), whereas the life cycle of *L. salmonis* is ~4–6 weeks (Stien et al. 2005). Therefore, the abundance of lice in the environment at the time of the early marine migration of juvenile pink and chum salmon will be largely controlled by external

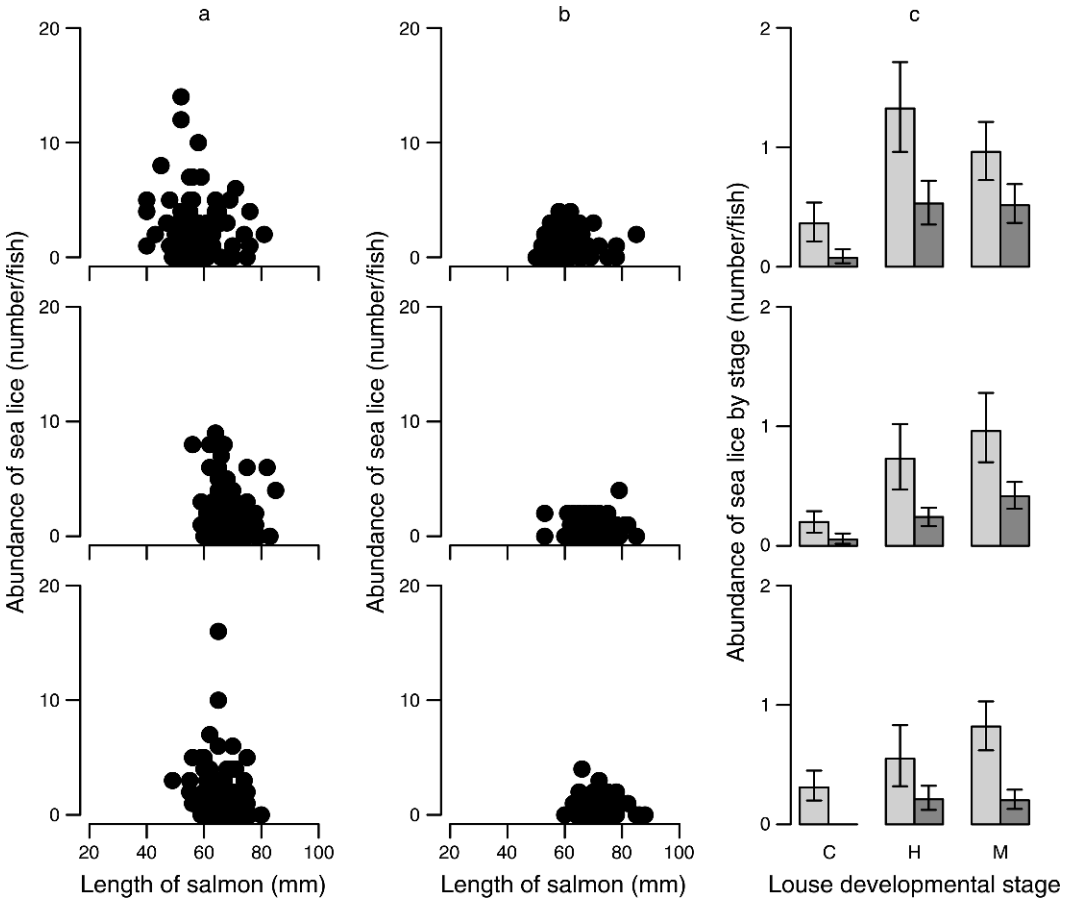

FIG. 4. Effects of predation on sea lice mean abundance and fish size distributions for three trials of the group predation experiment (each trial corresponds to one row of panels). Scatter plots show total lice vs. fish length for juvenile pink salmon (a) before and (b) after exposure to predatory coho salmon (*Oncorhynchus kisutch*) smolts for 36–48 h. (c) The barplots show changes in abundance (mean with 95% bootstrap CI) of different sea louse stages (C, copepodid; H, chalimus; M, motile) before (light bars) and after (dark bars) exposure to coho smolts for 36–48 h.

factors, e.g., management of salmon farms, rather than by reproduction of lice on juvenile salmon. Another separation of timescales occurs between the population dynamics of the parasite compared to the intergenerational population dynamics of its wild salmon host. Specifically, the dynamics of sea lice and juvenile salmon occur on a short and continuous timescale of 2–3 months over the juvenile portion of the host life cycle, whereas host dynamics occur in discrete generations that span two years. To represent this, the host–parasite dynamics occur as a continuous-time submodel that affects survival terms in a discrete-time model of salmon population dynamics. We begin by detailing the host–parasite submodel, which tracks salmon–louse population dynamics during the juvenile stage of a salmon cohort. We then incorporate the submodel into a discrete-time model for the intergenerational population dynamics of pink salmon.

The model for sea lice and juvenile salmon tracks the abundance of juvenile pink salmon, N, and the total number of lice on the juvenile salmon, P [the average abundance of lice per fish is $\bar{P}(t) = P(t)/N(t)$], giving

$$\dot{N} = -\phi(N, \bar{P})CN - \alpha\bar{P}N$$

$$\dot{P} = \beta LN - \mu P - N\sum_{p=0}^{\infty}\{pq(p)[\phi(N, p)C + \alpha p]\} \qquad (1)$$

where the dot on the left-hand side of each equation indicates differentiation with respect to time (i.e., d/dt). At time $t = 0$, juvenile salmon enter the marine environment at initial abundance $N(0) = N_0$ and are uninfected, with $P(0) = 0$, because lice are viable in marine conditions only. After entering the marine environment, lice begin to attach to the juvenile salmon and some of the salmon die due to predation and parasitism. The rate of direct parasite-induced mortality for an individual salmon infected with p lice is αp, where α is the per parasite rate of direct parasite-induced host mortality. The average rate of direct parasite-induced host mortality for a population of juvenile salmon is therefore $\alpha P/N = \alpha\bar{P}$. The mortality of juvenile salmon

TABLE 5. Analysis of covariance between control and predation trials using a generalized linear model with Poisson error and random effects.

Model	ΔAIC	Log likelihood	χ	df	*P*
S+L	15.1	−1082.1			
S+L+T	5.8	−1076.5	11.3	1	0.00079
S+L+T+(L×T)	0	−1072.5	7.9	1	0.0051

Notes: Model terms are fish species (S) for pink or chum salmon, mean louse abundance (L) preceding a trial, and treatment (T) for control or predation. All models contained random effects to accommodate the multiple trials.

due to predation is governed by the term ϕ, which is dependent on the abundance of juvenile salmon and the mean abundance of parasites. The abundance of predators, C, which represents coho salmon smolts, is uncoupled from the host–parasite dynamics and is instead controlled as an exogenous variable. Below, we detail ϕ for the case of a type II functional response where the capture rate is modified by parasite abundance. The total parasite population size, P, involves an immigration and death process whereby free-swimming lice at density L attach to host fish at rate β. Once attached to a host fish, lice then die at rate μ due to non-host mortality processes. Finally, we assume that lice die when their host dies, which leads to the final term in the second equation that sums the parasite mortality due to host mortality for all cases of p parasites per fish from zero to infinity. That is, the rate of parasite-induced host mortality for a fish with p parasites is $\phi\ (N, p)C + \alpha p$, where the first term is indirect mortality due to predation and the second term is direct parasite-induced host mortality. For each fish with p lice, the mortality rate of lice is $\phi(N, p)C + \alpha p$, which affects the portion of the parasite population that has p parasites per host, $Nq(p)$. Here, q is the probability density function that specifies the distribution of parasites on the host population. For a detailed derivation of a simplified form of model (1), where the host population has a constant birth rate and $\phi(N, p)C = 0$, see Anderson and May (1978).

The effect of parasitism on the survival of juvenile salmon depends on the rate of parasite-induced host mortality as well as on how parasitism affects predator–prey interactions. We assume that coho predators follow a type II functional response in relation to pink salmon abundance, which means that there is an average rate at which predators can capture pink salmon, but that predation rates are limited by the handling time required for prey processing. When parasite abundance is nil, the type II functional response governing the predation rate on juvenile pink salmon is

$$N\phi(N, P = 0) = \frac{\gamma N}{1 + \gamma T_h N} \tag{2}$$

(Holling 1959, Kot 2001), where γ is the rate at which predators capture prey and T_h is the handling time. The experimental data indicate that lice increase the rate at which predators capture juvenile pink salmon because infected juvenile salmon are more willing to accept increased predation risk when foraging, exhibit deviant schooling behavior that may increase predation risk, and are less able to evade a predatory strike. However, although lice may increase the rate at which predators can capture juvenile salmon, there is no basis to suggest that lice affect the time it takes a predator to consume a juvenile salmon once captured. Based on these assumptions, a suitable model has the form

$$N\phi(N, P = p) = \frac{(\gamma + p\sigma)N}{1 + (\gamma + p\sigma)T_h N} \tag{3}$$

where σ determines the per parasite increase in the rate of capture of juvenile pink salmon by a predator.

To continue with building the sea lice and juvenile salmon submodel, we must substitute the expression for predation ($N\phi(P, N)$; Eq. 3) into the sum in the host–parasite model (Eq. 1), which leads to a complex expression. To simplify the model, we used an approximation of the type II functional response given by

$$N\phi(N, P = p) \approx \begin{cases} (\gamma + \sigma p)N_c & \text{if } N > N_c \\ (\gamma + \sigma p)N & \text{if } N \le N_c \end{cases} \tag{4}$$

which splits the functional response into two linear functions corresponding to the limit $N \to 0$ of $N\phi(N, P)$ when $N \le N_c$ and the limit $N \to \infty$ of $N\phi(N, P)$ when $N > N_c$. This approximation means that parasites have a linear effect on host mortality, but that the linear effect is different depending on whether the abundance of juvenile pink salmon is greater than or less than the critical value of N_c. To complete the approximation, we observe that $\lim_{N\to\infty}[N\phi(N, \bar{P})] = 1/T_h$, and so estimate N_c by solving $(\gamma + \sigma\bar{P})N_c = 1/T_h$ to obtain

$$N_c = [T_h(\gamma + \sigma\bar{P})]^{-1}. \tag{5}$$

The model for sea lice and juvenile salmon population dynamics (Eq. 1) requires an assumption on the distribution of lice among individual juvenile salmon. That is, we must specify the distribution of q in Eq. 1. For sea lice and juvenile pink salmon in the Broughton Archipelago, previous studies have approximated $q(p)$ with a Poisson distribution (Krkošek et al. 2005*a*, 2006). Using a Poisson distribution and a little algebra (Appendix), the model for sea lice and juvenile pink salmon population dynamics becomes

$$\left.\begin{aligned} \dot{N} &= -(\gamma + \sigma\bar{P})N_c C - \alpha\bar{P}N \\ \dot{\bar{P}} &= \beta L - (\mu + \alpha + \sigma C N_c/N)\bar{P} \end{aligned}\right\} \quad \text{if } N > N_c$$
$$\left.\begin{aligned} \dot{N} &= -(\gamma + \sigma\bar{P})NC - \alpha\bar{P}N \\ \dot{\bar{P}} &= \beta L - (\mu + \alpha + \sigma C)\bar{P} \end{aligned}\right\} \quad \text{if } N \le N_c \tag{6}$$

where the parasite dynamics are given in terms of the average number of parasites per host, $\bar{P}(t)$.

Salmon population dynamics

To evaluate how sea lice infestation and predation affects pink salmon productivity, we begin with a Ricker model for salmon stock-recruitment population dynamics. The Ricker equation (Ricker 1954) is commonly used to estimate population growth rates (Myers et al. 1999) and density dependence (Brook and Bradshaw 2006) from abundance time series in fisheries and ecology. It is also used to understand the standard components of productivity, overcompensatory density dependence, and environmental variation that characterize fish population dynamics (Myers et al. 1999, Hilborn and Walters 2001), particularly for Pacific salmon (Dorner et al. 2008, Ford and Myers 2008). The model has the form

$$n_i(t) = n_i(t-2)\exp[r - bn_i(t-2)] \tag{7}$$

where $n_i(t)$ is the abundance of population i in year t, r is the population growth rate, and b determines density-dependent mortality. The Ricker equation is lagged two years, $n_i(t-2)$, to reflect the two-year life cycle of pink salmon. To include the submodel of sea lice and juvenile salmon, we must distinguish between components of the submodel that are already accounted for in the parameterization of the Ricker model. In particular, we need to remove mortality during early marine life from the Ricker model because that mortality will be replaced by the mortality that is modeled by the sea lice and juvenile salmon submodel. To do this, we assume that all the mortality rate of juvenile pink salmon during early marine life, ν, is due to predation. Assuming that the mortality rate as well as the juvenile salmon and sea lice submodel apply for a duration of three months ($T =$ 90 days), we remove early marine mortality from salmon productivity by calculating

$$r_1 = r + \int_0^T \nu d\tau \tag{8}$$

and rewriting the Ricker model for pink salmon population dynamics as

$$n_i(t) = n_i(t-2)\exp[r_1 - bn_i(t-2)]Q \tag{9}$$

where Q is the solution for juvenile salmon survival from Eqs. 6 after being normalized by the initial abundance of juvenile salmon when they enter the sea:

$$Q = N(T)/N_0. \tag{10}$$

Parameterization

Many of the parameters in the model are known from previous works. Our objective here is to use known parameter values from the literature in order to analyze the effects of lice and predation on salmon population dynamics. Specifically, we are interested in using βL and σ as control parameters in an analysis of their effects on sea lice dynamics ($\bar{P}$), juvenile salmon survival (N), and salmon population dynamics (n), while holding the other model parameters constant at values derived from the literature. Because many parameters are estimated indirectly and the uncertainty in many parameter values is not known, our objective is to explore the model dynamics in a qualitative sense to identify important implications for scientific understanding, management, and policy, rather than making quantitative predictions for salmon conservation and restoration. Below we detail how each parameter was estimated.

We begin by considering a juvenile salmon population that has an initial abundance of 100 000 fish at the time of sea entry. The subsequent population dynamics can be assessed on a normalized scale, $N(t)/N_0$, which is bounded between zero and one. At time $t = T = 3$ months, the endpoint of juvenile salmon exposure to sea lice from salmon farms in our model, we have $N(T)/N_0 = Q$, where Q is the survival of juvenile salmon in Eq. 9. For the mortality rate of juvenile salmon in the absence of sea lice, ν, we use the estimate for pink salmon in (Parker 1968), and reported also in (Heard 1991), which was 0.53 month^{-1} or, dividing by 30 days, $\nu = 0.017$ day^{-1}. We assume that ν is due to predation and that the effects of lice at natural abundances are negligible because louse prevalence is typically very low (<5%) (Krkošek et al. 2007*b*, Peet 2007), which leads to the following constraint:

$$\begin{aligned} N(T, L \to 0)/N_0 &= \exp\left[-\int_0^T \nu d\tau\right] \\ &= \rho. \end{aligned} \tag{11}$$

This constraint, when combined with the approximation of a type II functional response with zero parasite abundance given in Eq. 2, leads to a parameterization of the attack rate γ by solving the following relation:

$$\rho = \frac{1}{N_0}\exp\left[-\int_0^T \dot{N}_{N_0, P=0} dt\right]. \tag{12}$$

However, to do so, the other parameters in the equation for $\dot{N}$ must be constrained. We assume that the handling time T_h is equal to one because, on average, there was approximately one juvenile pink or chum salmon eaten per day per coho smolt in the group predation trials. Furthermore, we assume that the density of predators (coho smolts) is 5% of the initial density of juvenile pink salmon. After setting $T_h = 1$ and $C = 0.05N_0$, we solved Eq. 12 for the attack rate γ to obtain $\gamma = 3.4 \times 10^{-6}$. The remaining parameters for the juvenile salmon submodel (Eqs. 6), μ and α, have been estimated previously for motile-stage lice as $\mu = 0.24$ days^{-1} and $\alpha = 0.002$ (days/louse) in studies of juvenile salmon and salmon lice held in ocean enclosures (Krkošek et al. 2006, Krkošek et al. 2009). The parameters for the Ricker model for pink salmon (Eq. 7) have been estimated in previous work. The population growth rate is $r = 1.2$, as estimated from a meta-analysis of pink salmon stocks (Myers et al. 1999). The density-dependent parameter is $b = 0.64$, as estimated from an analysis of pink salmon escapement

data from the central coast of British Columbia (Krkošek et al. 2007*a*). These estimates for the Ricker model parameters come from pink salmon populations that were largely unexposed to salmon farms.

Analysis

Implementation and analysis of the model yielded important insights into the potential for interactions between parasitism and predation to affect salmon and louse population dynamics. Analysis of the model (Eqs. 6) reveals a compensatory mortality regime that is density dependent. To see this, we substitute Eq. 5 into Eqs. 6, giving

$$\left.\begin{aligned}\dot{N} &= -C/T_h - \alpha\bar{P}N \\ \dot{\bar{P}} &= \beta L - (\mu + \alpha + \sigma C N_c/N)\bar{P}\end{aligned}\right\} \quad \text{if } N > N_c$$
$$\left.\begin{aligned}\dot{N} &= -(\gamma + \sigma\bar{P})NC - \alpha\bar{P}N \\ \dot{\bar{P}} &= \beta L - (\mu + \alpha + \sigma C)\bar{P}\end{aligned}\right\} \quad \text{if } N \le N_c. \tag{13}$$

Eqs. 13 indicate that above the threshold density of juvenile salmon, N_c, the mortality of juvenile salmon due to predation, has no dependency on parasite abundance and is instead governed by the abundance of predators and their handling time of prey. Furthermore, when $N > N_c$, the mortality of parasites due to predation is dependent on parasite abundance, although the rate may be low when $N \gg N_c$. That is, when $N > N_c$, there is a region in model dynamics where there is a plateau in the mortality rate of juvenile salmon due to predation and that plateau does not increase with parasite abundance, whereas parasite abundance is in decline due to predation. The converse is true when $N < N_c$, where there is a regime in which the mortality of juvenile salmon due to predation is increased at a per parasite rate, as is the decline in parasite abundance due to predation. Thus, when $N < N_c$, there is an accelerated decline in juvenile salmon abundance due to the effects of parasitism on predation, and in addition, the rate of parasite mortality due to predation is also higher than in the compensatory regime because $\sigma C > \sigma C N_c/N$ when $N > N_c$. However, it is critical to note that the density-dependent threshold N_c that separates the regimes of compensatory mortality ($N > N_c$) and noncompensatory mortality ($N < N_c$) is dependent on parasite abundance. Recall that $N_c = [T_h(\gamma + \sigma\bar{P})]^{-1}$, which indicates that as the abundance of parasites increases, the threshold density, N_c, decreases. That is, as the abundance of lice increases, it is counterbalanced by a reduction in N_c, which limits the mortality of juvenile salmon due to predation while the increased mortality of lice due to predation is maintained.

One key unknown parameter in the model is σ, the per louse increase in the rate at which predators capture juvenile salmon. Analysis and simulations of the model indicated that the dynamics were sensitive to the strength of the effect of parasite-induced predation on pink salmon, σ, which increased the mortality of both sea lice and salmon (Fig. 5 and Eqs. 6). Sea lice population dynamics followed an immigration and death process characterized by an initial transient in which lice numbers increased, followed by an equilibrium in which lice numbers held steady, reflecting the balance between the rate of new infections and the mortality of lice from parasite mortality, parasite-induced host mortality, and mortality due to predation. In some model simulations, the initial transient in louse dynamics was characterized by an initial rise and then decline before equilibrating (Fig. 5b, c), which corresponds to a transition in the model where host density declined and crossed the N_c threshold. This pattern was not observed in some simulations (Fig. 5a) because the N_c threshold remained higher than the initial abundance of juvenile salmon due to reduced mortality rates when the infection pressure βL was low or there was a low rate of parasite-induced predation, σ. The equilibrium abundance of lice that was ultimately reached in each model simulation can be seen from Eqs. 6 when $N < N_c$ by solving $\dot{\bar{P}} = 0$ to obtain $\bar{P} = \beta L/(\alpha + \mu + \sigma C)$.

Not surprisingly, when the infection pressure, βL, or the rate of parasite-induced predation, σ, increased, there was a decline in juvenile salmon survival and a decline in the salmon population growth rate (Figs. 5 and 6). However, an important finding is that as the rate of parasite-induced predation increased, both louse abundance and salmon survival decreased simultaneously (Figs. 5 and 6). In particular, the interaction between parasites and predators can lead to a situation with low observed louse abundance on juvenile salmon and low productivity of wild salmon populations (Fig. 6). Such an effect could be generated not just by an increase in parasite-induced predation on juvenile salmon, but also by an increase in the abundance of predators. This can be seen from Eqs. 6, where the rate of parasite-induced predation, σ, and the abundance of predators, C, appear together multiplicatively, indicating that the effects of increasing σ in Figs. 5–6 are similar to what would occur with increasing C. It is therefore important to recognize that predators can mediate the relationship between louse parasitism and salmon productivity in complex ways; a simultaneous decline in parasite abundance and salmon productivity may be evidence of changes in predation, due to changes in either predator behavior or abundance, that is mediating the link between parasitism and salmon productivity.

Discussion

Parasites may affect host population dynamics in multiple ways, depending on how parasitism interacts with other sources of mortality. For example, parasites may increase mortality rates of infected prey, which could reduce prey abundance but also stabilize prey population dynamics (Hudson et al. 1992). Alternatively, parasites may have little effect on host population dynamics if mortality from parasites is compensated by a change in other mortality factors (Tompkins and Begon 1999). For example, if infected individuals are

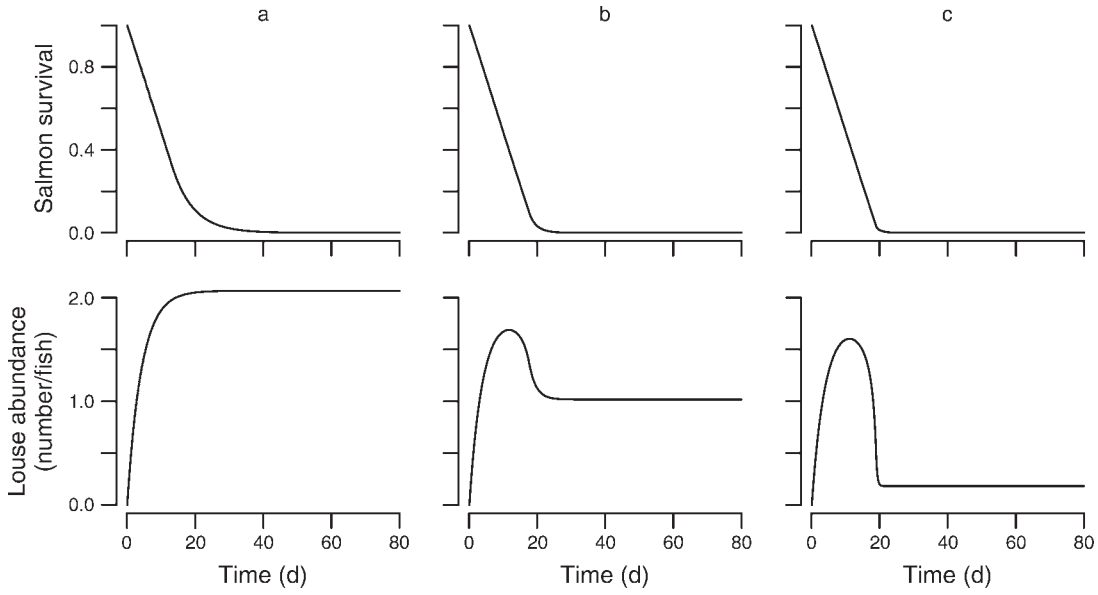

FIG. 5. Survival of juvenile salmon and abundance of lice at increasing rates of parasite-induced predation (σ, the per parasite increase in the rate of capture of juvenile salmon by a predator) according to model dynamics with (a) $\sigma = 0$, (b) $\sigma = 0.00005$, and (c) $\sigma = 0.0005$. The rate of new louse infections was held constant at infection pressure $\beta L = 0.5$ among all model results.

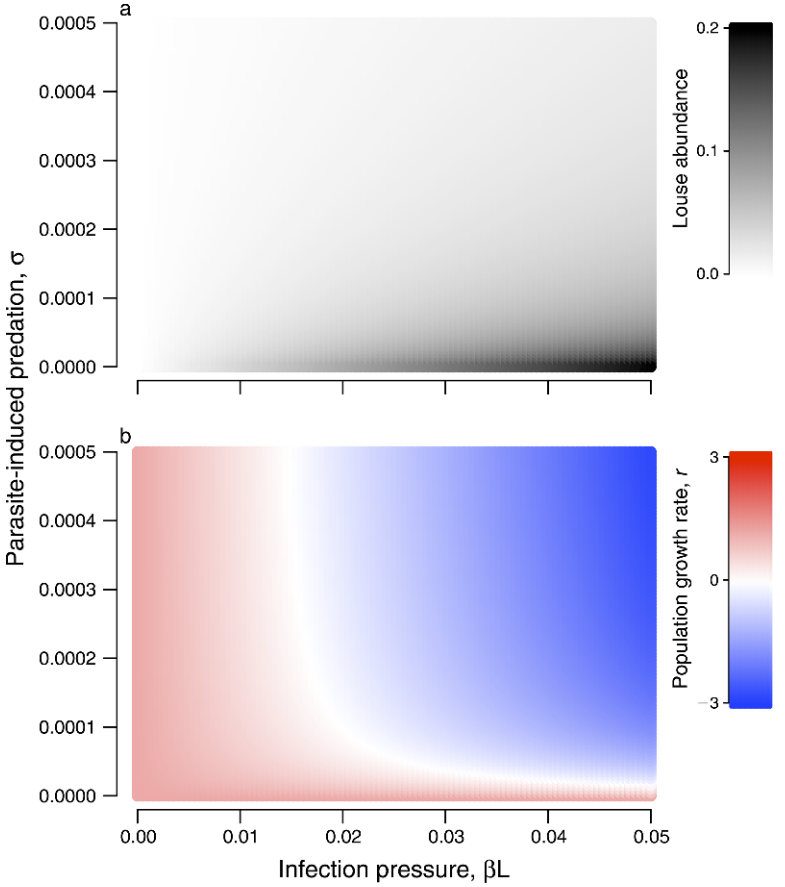

FIG. 6. (a) Equilibrium abundance of lice on juvenile pink salmon and (b) the corresponding population growth rate (r) for pink salmon according to model dynamics across parameter space defined by infection pressure (βL) and the rate of parasite-induced predation on juvenile pink salmon (σ).

also more likely to be eaten or competitively excluded, then uninfected individuals may be released from predation or competition. Although sea louse infestations of wild juvenile salmon are associated with declines in wild salmon stocks (Krkošek et al. 2007*a*, Ford and Myers 2008), these effects are probably mediated by predation because, in the absence of parasitism, predation is thought to be the primary cause of mortality (Heard 1991). In this way, predation may increase the mortality of wild juvenile salmon infested with sea lice beyond what would be predicted from experimental studies designed to estimate the pathogenicity of lice. Alternatively, selective predation may be compensatory, by removing infected prey, reducing subsequent transmission, and releasing uninfected prey from predation. Such compensatory predation amounts to an ecosystem service provided by predators by reducing or eliminating the effect of human-caused infestations of wild juvenile salmon. Our experimental results provide empirical support that lice make juvenile salmon more prone to predation, which, according to the model analysis, can have important implications for salmon population dynamics and conservation.

The risky feeding experiment evaluated whether louse parasitism increased predation risk-taking as measured by the time taken for juvenile salmon to return to an exposed feeding area following a simulated predatory strike. The infected groups of juvenile salmon returned to the high-risk area 3.3 times faster than did the uninfected groups, indicating that infection causes juvenile salmon to accept greater predation risk to obtain a feeding opportunity. The increased risk acceptance of infected prey when foraging may reflect increased nutrient and energy requirements to balance the costs of infection such as maintaining physiological defenses (Jones et al. 2007), increased stress and immune activity (Fast et al. 2006), loss of blood and osmoregulatory function (Dawson et al. 1999), and increased viral and bacterial infection (Pike and Wadsworth 2000). The results from this experiment were statistically significant using the best-fit survival model (the lognormal model). However, alternative survival models (exponential and Weibull) did not have a substantially lower level of support from the data (small AIC_c differences), and these models yielded P values that were marginally insignificant ($0.059 < P < 0.061$). Further replication of the trials would likely make all of the statistical tests significant. The presence of censored data (i.e., in some trials the fish did not return to the high-risk area) indicates that the trial time could have been extended. However, the censored data were well-accommodated using standard survival analysis methods. Despite these limitations, we regard the results from the risky feeding experiment as biologically significant.

The schooling experiment evaluated if lice affected the schooling behavior of juvenile salmon in ways that might make them more prone to predation. Schooling behavior is a primary defense against predators that dilutes predation pressure and confuses predators with multiple identical moving targets (Landeau and Terborgh 1986). Anything that isolates or makes an individual distinguishable from the others may increase its risk of attack and capture. In the schooling experiment, infected juvenile chum salmon exhibited deviant schooling behavior in a school of uninfected and size-matched conspecifics. Infected individuals were more frequently located in peripheral and rear portions of the school and were also more distant from their nearest neighbor than uninfected fish. The positioning in peripheral portions of the school could lead infected individuals to become isolated from the school following a predatory strike, thereby removing the protection of the school. Increased positioning in the rear of the school could increase the chances of being captured by a predator pursuing the school. Increased nearest-neighbor distances could provide an irregularity that gives predators a focal point for pursuit and capture (Barber and Huntingford 1996). These effects were size dependent: they declined as the size of the juvenile chum salmon increased. The experiment used shallow water to reduce the spatial analysis to two dimensions and also used a current to induce schooling. These conditions depart from natural predator–prey interactions, where schooling occurs in three dimensions and the presence of predators may cause schooling behavior to differ from that induced by water movement. Nevertheless, the results indicate that infection affects schooling behavior such that predation risk may be increased.

The predator choice experiment tested if infection increased the predation vulnerability of juvenile pink salmon, measured directly by providing a cutthroat trout predator a choice between one infected and one uninfected juvenile salmon. Here, schooling behavior was not a factor because there were only two prey fish for a predator to choose between, thereby isolating interactions between individual prey and predators. The results indicated that cutthroat trout predators captured infected prey more often than uninfected prey. They further identify that this was not due to predator preference, but rather to the reduced ability of infected prey to evade a predatory strike; the number of strikes was not different between infected and uninfected prey. While the preceding experiments indicate that infection may make juvenile salmon more prone to receiving a predatory strike, this experiment indicates that once a predator strikes at an infected juvenile salmon, there is a further reduced chance of evading the predatory strike relative to uninfected juvenile salmon. A side observation from this study was that in approximately three-quarters of the trials where the infected juvenile salmon was captured, the louse survived by swimming or moving directly onto the predator (Connors et al. 2008). This finding has implications for trophic transmission, predator health, and the behavioral ecology of lice (Connors et al. 2008). Together, the risky feeding, lousy schooling, and predator choice experiments

indicate that sea louse infection increased the predation vulnerability of juvenile salmon. All of these experiments nevertheless departed from natural field conditions, in which schools of predators and prey interact in a three-dimensional space. To resolve this we pursued group predation experiments.

The purpose of the group predation experiment was to mimic field conditions and evaluate if a group of predators selectively removed infected prey from prey schools that carried distributions of parasites representative of field conditions (some infected and some uninfected prey). We accomplished this by sampling prey (pink or chum salmon) from the field and directly transferring the group into the net pens. We used large net pens relative to the size of individual salmon and groups of salmon so that prey and predators could interact in natural-sized groups in a large, three-dimensional space. Some trials, where sea louse abundance was relatively high, showed a strong effect of selective predation on both smaller and more infected prey. However, several trials using fish with relatively lower louse abundances showed consistent, but not statistically significant, results. By synthesizing the data via meta-analysis of all trials, we found a significant effect of selective predation on infected juvenile salmon. The result of size-selective predation was consistent with previous investigations using a similar net pen design (Parker 1971, Hargreaves and Lebrasseur 1985). The results are also consistent with other work showing that infection of juvenile Chinook salmon (*O. tshawytscha*) with *Renibacterium salmoninarum*, the causative agent of bacterial kidney disease, increased predation by squawfish (*Ptychocheilus oregonensis*) and smallmouth bass (*Micropterus dolomieui*) predators in group predation experiments (Mesa et al. 1998). The mechanisms underlying the selective predation that we observed are probably a combination of the effects that we identified in the other experiments. Although we attempted to mimic field conditions, the net pens are nevertheless different due to a lack of habitat structure such as kelp, shallow intertidal areas, or sloping benthic habitat that may alter predator–prey interactions. It is worth noting that in parallel field-sampling programs studying juvenile salmon infection dynamics, as the salmon migrated past salmon farms (Krkošek et al. 2006), we occasionally observed coho salmon smolts and cutthroat trout feeding on juvenile pink and chum salmon in natural habitats (M. Krkošek, B. Connors, and A. Morton, *personal observations*).

The experimental results suggest that sea louse infection increases predation on infected individuals. However, the experiments do not indicate if the increased predation results in a population-wide increase in mortality or if the overall mortality remains unchanged while the distribution of predation pressure is shifted onto infected fish. In other words, is selective predation compensatory? In an attempt to resolve this, we developed a mathematical model to examine how increased predation risk due to sea louse infection manifests in salmon population dynamics. The model combined the Ricker model for pink salmon population dynamics with a submodel of sea lice and juvenile salmon population dynamics that contained elements of classical host–parasite and predator–prey models. A key component of the model was that predators followed a type II functional response in their predation rates on juvenile pink salmon. The type II functional response in the lice–salmon submodel separated the predator–prey interaction into two components: the rate at which predators capture juvenile salmon and the handling time required for a predator to consume and digest a juvenile pink salmon. The experimental work indicated that sea louse infection is likely to increase the rate at which predators capture juvenile pink salmon, but not the handling time needed by a predator to consume and digest an item of prey. We therefore modified the type II functional response such that there was a per parasite increase in the rate with which predators captured juvenile pink salmon, but handling time was constant. Using the model, we explored the effects of increasing parasite exposure as well as varying the rate of parasite-induced predation on sea louse abundance and salmon population dynamics.

Analysis of the salmon–louse model revealed a multitude of effects of sea louse infection on pink salmon population dynamics, including regions of parameter space that exhibited compensatory and non-compensatory mortality. The separation between the two mortality regimes was dependent on a critical threshold of juvenile salmon abundance, N_c, which itself was dependent on the abundance of lice. When the abundance of juvenile pink salmon, N, exceeded N_c, predation occurred in the saturation region of the type II functional response where the predation rate on juvenile salmon is determined by the handling time of the predator and is unaffected by sea louse abundance. In this regime where $N > N_c$, the predation rate on juvenile salmon is constant, whereas the mortality of sea lice due to predation remains proportional to sea louse abundance. Therefore, when $N > N_c$, compensatory mortality occurs and predators provide an ecosystem service by reducing parasite abundance without increasing the overall mortality of juvenile salmon. It is important to note that the critical threshold in juvenile salmon abundance, N_c, differentiating compensatory and non-compensatory mortality regimes is inversely related to parasite abundance. That is, as the abundance of lice increases, N_c decreases, thereby increasing the parameter space in which compensatory mortality occurs. This dynamic arises because as parasite abundance increases, so too does the accessibility of juvenile salmon to predators, thereby reducing the juvenile salmon abundance needed for predators to reach the saturation part of a type II functional response. It is also important to note that when the abundance of juvenile salmon decreases below N_c, parasitism becomes more influential

on predation rates, with concurrent per parasite increases in the rates of predation-related mortality of both juvenile salmon and lice. The sharp separation between compensatory and noncompensatory mortality regimes at the N_c threshold is due to our approximation of a type II functional response with a piecewise linear function. Similar dynamics would probably occur without the approximation, but the transition between compensatory and noncompensatory regimes would be more gradual.

The model indicates that changes in predation rates may compensate for increases in parasitism rates by limiting the overall predation-related mortality and increasing parasite mortality. However, it is important to differentiate this from the outcome that predation increases the overall mortality of juvenile salmon relative to previous analyses that considered only the direct effects of sea lice on salmon mortality and did not consider the ecological context of the host–parasite interaction (Jones et al. 2006, Krkošek et al. 2006, Jones and Hargreaves 2009, Krkošek et al. 2009). The sublethal effects of parasitism on juvenile salmon behavior indicate that predation rates on infected juvenile salmon populations should increase; it therefore follows that the effect of sea lice on juvenile salmon should be a decline in survival that is greater than previously thought. More troubling is the observation that changes in predator abundance or predator behavior can lead to a counterintuitive correlation between sea louse abundance and salmon productivity. Predation can lead to high mortality rates of sea lice and juvenile salmon, creating a situation of low sea louse abundance on juvenile salmon and poor productivity of salmon populations. The effects of predators may therefore mislead conservationists, managers, or regulators evaluating changes in parasite abundance and salmon productivity in the absence of other ecological information. A simultaneous decline in parasite abundance and salmon productivity may indicate a shift in predation that hides an underlying impact of sea lice on salmon populations.

The model of salmon–louse population dynamics provided important insights into the effects of predators in mediating salmon and louse mortality, but it also included many assumptions that must be considered, particularly in relation to predators. Importantly, we assumed that the abundance of predators was 5% of the abundance of juvenile pink salmon at the time of sea entry and that predator abundance did not vary within or among years. Although the abundance of predators is probably variable both within years and among years, our assumptions were necessary to assess the implications of predation for a typical average scenario. Another important assumption is that the abundance of predators was uncoupled from the prey population, whereas the population dynamics of coho predators may depend on the abundance of prey during early marine life. Indeed, coho survival may be linked to early marine growth, which would depend in part on the availability of prey (Beamish et al. 2004). That is, lice could simultaneously enhance the predator population and deplete the prey population, which could ultimately lead to collapse of both predator and prey populations. On the other hand, lice can be trophically transmitted during predator–prey interactions (Connors et al. 2008), leading to parasite accumulation on predators, which could depress predator populations and release prey populations. Although our model clarifies the basic processes and implications of predation on salmon–louse population dynamics, there is important further work necessary to understand the full dynamics of this predator–prey–parasite system.

Another important assumption of the salmon–louse model is that we did not consider the effects of environmental stochasticity. Salmon populations are notorious for high variation in population dynamics that is not easily attributable to deterministic processes. Environmental variation in the Ricker model is commonly modeled as a lognormal stochastic term, representing Gaussian random deviation from an average productivity determined by the population growth rate r (Hilborn and Walters 2001). Because we were interested in modeling and understanding the mechanistic underpinnings of predation in salmon–louse population dynamics, we did not consider environmental stochasticity. However, many departures from the model assumptions could be thought of as components of environmental stochasticity in the Ricker model as it relates to observed salmon–louse population dynamics. For example, the infection pressure experienced by juvenile pink salmon, βL, is unlikely to be constant within and among years, but rather dependent on the location of salmon farms, the louse population size on farmed salmon, the migration speed of juvenile salmon, and abiotic correlates such as temperature, salinity, and physical oceanography (Krkošek 2010). Such annual variation in infection pressure, as well as variation in predator abundance, could be thought of as a component of environmental stochasticity in the Ricker model. Understanding the components and effects of environmental stochasticity is an important line of further research that will be particularly important to evaluate if the model dynamics can be detected and estimated from data on sea lice and salmon abundances in the field.

The parameter values that we used in the salmon–louse model were taken primarily from the literature and we did not consider uncertainty in the estimates. Often, information on the uncertainty in parameter estimates was not available, or model parameters were calculated indirectly using point estimates of other parameters such as the mortality rate of juvenile pink salmon from (Parker 1968). Furthermore, we simplified the model by approximating a type II functional response with a piecewise linear function. The approximation simplified the analysis of the model dynamics and supported our assumption that lice are Poisson distributed on juvenile

salmon because the Poisson assumption is not violated if parasite-induced host mortality is linear (Rousset et al. 1996). Because of the uncertainty in parameter estimates and the simplifying approximation of a type II functional response, the results of the model analysis cannot yield detailed quantitative predictions. Rather, the utility of the model is in understanding how the underlying processes of predation and parasitism may interact to affect pink salmon population dynamics. This led to important insights, such as regimes of compensatory and noncompensatory predation as well as the potential for predation to lead to a simultaneous decline in sea louse abundance and salmon productivity. Although the model may not be useful for making quantitative predictions, it does point out to scientists, managers, and regulators the importance and implications of predation in mediating salmon–louse dynamics.

As management and policy progress toward reducing sea louse abundance on farmed salmon in order to protect wild salmon, they may be aided by the ecosystem service provided by predators. Our experimental results indicate that sea louse infection of prey probably increases the rate at which predators can access and capture them. Theoretically, we have shown that when the abundance of juvenile salmon is high, predators may be in the saturation region of a type II functional response in which predators may act to limit or reduce sea louse abundance on juvenile pink salmon without changing the overall predation rate. Furthermore, as sea louse abundance increases, the size of parameter space in which this ecosystem service occurs is increased. It follows that predators may play an important role in controlling the abundance of parasites and reducing the spread of infection. This has not been previously appreciated in studies of sea lice and salmon. Importantly, an implication of neglecting predation in previous analyses is that the mortality of juvenile salmon due to sea lice has probably been underestimated, possibly to a large degree, depending on predator abundance and behavior. Furthermore, if juvenile pink salmon abundance is low and predation rates are governed by the increasing (as opposed to saturation) region of the type II functional response, predation coupled with louse-induced mortality would accelerate population declines. The role of predators in mediating salmon–louse dynamics needs to be considered more thoroughly in analyses of the impact of salmon farms on wild salmon survival. Because predation can cause a simultaneous decline in parasite abundance and salmon productivity, scientists, managers, and regulators need to consider predators as a key component of the ecological context of salmon–louse dynamics when developing or revising management and policy to conserve and restore wild Pacific salmon populations.

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APPENDIX

Derivation of model for sea lice and juvenile salmon population dynamics (*Ecological Archives* A021-041-A1).