

Epizootics of wild fish induced by farm fish

Martin Krkošek^{*†}, Mark A. Lewis^{*}, Alexandra Morton[‡], L. Neil Frazer[§], and John P. Volpe[¶]

^{*}Centre for Mathematical Biology, Departments of Mathematical and Statistical Sciences and Biological Sciences, University of Alberta, Edmonton, AB, Canada T6G 1G1; [†]Raincoast Research Society, Simoom Sound, BC, Canada V0P 1S0; [‡]Department of Geology and Geophysics, School of Ocean and Earth Science and Technology, University of Hawaii, 2525 Correa Road, Honolulu, HI 96822; and [§]School of Environmental Studies, University of Victoria, 3800 Finnerty Road, Victoria, BC, Canada V8P 5C2

Edited by Stephen R. Carpenter, University of Wisconsin, Madison, WI, and approved August 24, 2006 (received for review April 29, 2006)

The continuing decline of ocean fisheries and rise of global fish consumption has driven aquaculture growth by 10% annually over the last decade. The association of fish farms with disease emergence in sympatric wild fish stocks remains one of the most controversial and unresolved threats aquaculture poses to coastal ecosystems and fisheries. We report a comprehensive analysis of the spread and impact of farm-origin parasites on the survival of wild fish populations. We mathematically coupled extensive data sets of native parasitic sea lice (*Lepeophtheirus salmonis*) transmission and pathogenicity on migratory wild juvenile pink (*Oncorhynchus gorbuscha*) and chum (*Oncorhynchus keta*) salmon. Farm-origin lice induced 9–95% mortality in several sympatric wild juvenile pink and chum salmon populations. The epizootics arise through a mechanism that is new to our understanding of emerging infectious diseases: fish farms undermine a functional role of host migration in protecting juvenile hosts from parasites associated with adult hosts. Although the migratory life cycles of Pacific salmon naturally separate adults from juveniles, fish farms provide *L. salmonis* novel access to juvenile hosts, in this case raising infection rates for at least the first ≈ 2.5 months of the salmon's marine life (≈ 80 km of the migration route). Spatial segregation between juveniles and adults is common among temperate marine fishes, and as aquaculture continues its rapid growth, this disease mechanism may challenge the sustainability of coastal ecosystems and economies.

aquaculture | emerging infectious disease | migration | salmon | sea lice

Ocean fisheries and ecosystems are in decline (1, 2). The collapse and low resilience of exploited fish stocks (3) and declines in the abundance of global fishery landings (4) illustrate the challenges facing the sustainability of ocean fisheries (4, 5). These effects are partly mitigated by the rise of aquaculture, in which the farming of herbivorous fishes promises to improve global fish supplies (6). However, the decline of ocean fisheries and ecosystems can also be exacerbated by the industrial farming of carnivorous fishes such as salmon (6–8). By feeding farm salmon proteins and oils extracted from wild fish, there is a net loss of fish supply (6). Escaped farm salmon spread domesticated genes into wild populations (9) and have the potential to invade and displace native wild stocks (10–12). Finally, the spread of infectious pathogens from farm to wild salmon may also threaten wild stocks (13). Despite decades of work, the extent and impacts of parasite transmission from farm to wild salmon have remained contentious and unresolved (14, 15).

Most emerging infectious diseases in wildlife arise through complex interactions among humans, wildlife, and domesticated animals (16). The spread of nonnative parasites from livestock to wildlife has reduced the abundance (16–18) and resilience (19) of some wildlife populations and has challenged the conservation of other threatened or endangered species (16, 17, 20). For many marine fishes, aquaculture can change the dynamics of normally benign native parasites by providing parasites novel access to juvenile hosts. For Pacific salmon, juveniles are not sympatric with adults (and their parasites) for their first several months of marine life (21). However, salmon farms can undermine this temporal refuge from lice early in the salmon's life

cycle. During their first months at sea, wild salmon are sympatric with large abundances of domesticated salmon (and their parasites). This change in the timing and magnitude of parasite transmission in a host's life history may undermine a functional role of migration in protecting juvenile fish from parasites associated with adult fish.

The rise of salmon farming has coincided with the emergence of native sea lice (*Lepeophtheirus salmonis*) infestations of sympatric wild juvenile salmonids. Afflicted areas include Norway (22), Scotland (23), Ireland (24), and Canada (25). The infestations are concurrent with declines in affected populations, but the causal linkages are obfuscated by the myriad factors affecting wild fish populations, such as density dependence, climate, fishing, and habitat degradation. Here, we present extensive data sets and mathematical models that couple louse transmission and pathogenicity to estimate the impact of farms on the survival of wild juvenile pink (*Oncorhynchus gorbuscha*) and chum (*Oncorhynchus keta*) salmon migrating through an archipelago off the west coast of Canada. The analysis yields quantitative insights into the mechanisms and extent of impacts of aquaculture on disease dynamics in wild fish populations. More generally, the results inform the development of marine conservation and disease theory and its application in fisheries and aquaculture management.

Results

The transmission dynamics data set totaled 14,255 juvenile salmon nonlethally assayed for copepodid, chalimus, and motile stage lice at 1- to 3-km intervals along 40–80 km of three different migration routes containing two to three farms each (Fig. 1 and Fig. 4, which is published as supporting information on the PNAS web site). From among three candidate models, the data best supported a model that had a uniformly distributed ambient population of infectious planktonic larvae and point sources of planktonic larvae situated at the farms (likelihood ratio test, $P \ll 0.0001$; Akaike weights ≈ 1 ; Tables 1–3, which are published as supporting information on the PNAS web site). Across all data sets, this model fit the data well (Fig. 2 and Figs. 5 and 6, which are published as supporting information on the PNAS web site). The other models contained only farm- or ambient-origin lice. With the parameter estimates from the best-fit model, we reconstructed the spatial distributions of infective larvae originating from each source. Farm salmon were the primary source of lice, raising the density of infective parasite larvae above ambient levels for >80 km of the migration route (Figs. 2, 5, and 6).

Author contributions: M.K., M.A.L., and A.M. designed research; M.K., M.A.L., and A.M. performed research; M.K., M.A.L., and L.N.F. contributed new reagents/analytic tools; M.K. analyzed data; and M.K., M.A.L., and J.P.V. wrote the paper.

The authors declare no conflict of interest.

This article is a PNAS direct submission.

Freely available online through the PNAS open access option.

[†]To whom correspondence should be addressed. E-mail: mkrkosek@ualberta.ca.

© 2006 by The National Academy of Sciences of the USA

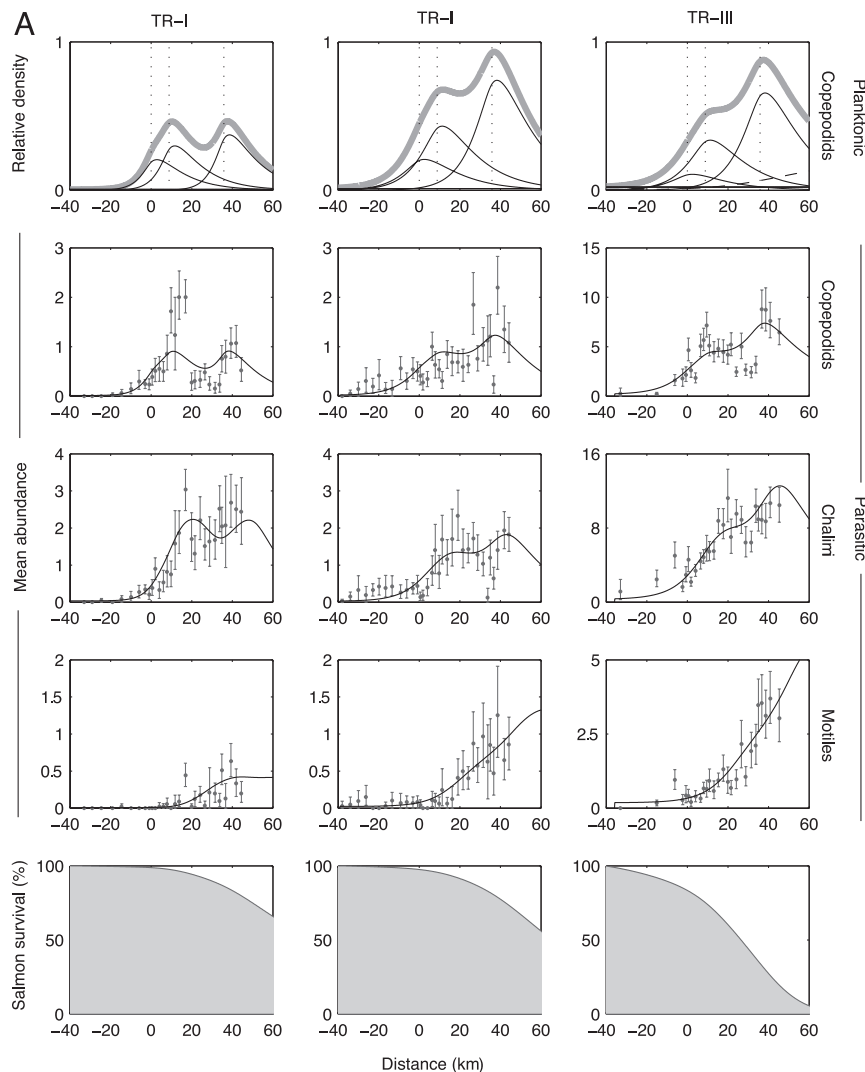


Fig. 2. (Figure continues on the opposite page.)

universities, conservation organizations, provincial and federal governments, and industry have not identified a prevalent viral or bacterial pathogen or other physical stressor.

The life cycles of most temperate marine fishes involve a period of spatial segregation between juveniles and adults. This segregation may protect juveniles from parasites associated with adults. For salmon, migration provides a temporal refuge from lice in early marine life. Farm salmon can eliminate this refuge, resulting in high epizootic mortality in wild salmon populations. These effects may not be limited to salmon but may extend to other species coming under culture around the world. Those that have migratory or highly dispersed life histories would be most at risk. As aquaculture continues its rapid growth into new regions, habitats, and species, this disease mechanism may challenge the sustainability of coastal ecosystems and economies.

Methods

To assess the impact of sea lice from salmon farms on the survival of wild juvenile salmon, we collected two types of data sets, one that tracks the infection dynamics of juvenile salmon migrating past salmon farms and another that tracks the survival of infested juvenile salmon collected from the field and reared in ocean enclosures. From the data sets, we used maximum-

likelihood and model selection statistics to select and parameterize mechanistic models of sea lice transmission and juvenile salmon survival. These models were then coupled to estimate the mortality of wild salmon caused by farm-origin lice. Below we offer a brief overview of the methods. Further details appear in *Supporting Text*.

Transmission Dynamics. For 2 years (2004–2005), we studied the infection dynamics of parasitic sea lice on juvenile pink and chum salmon as they migrated past active salmon farms, each containing $\approx 600,000$ Atlantic salmon (*Salmo salar*). Three migration routes containing two, two, and three farms were surveyed for 40, 60, and 80 km, respectively. At 1- to 3-km intervals, we sampled ≈ 100 juvenile salmon as they approached and passed the salmon farms (Figs. 1 and 4). We used a nonlethal sea lice enumeration technique (33) to count the number of copepodid, chalimus, and motile lice on each fish, thus capturing the developmental progression of lice.

To analyze these data, we extended an established spatial model of the stage-structured dynamics of sea lice infecting juvenile salmon migrating past salmon farms (13). The model uses advection–diffusion–decay equations to describe the dispersion of planktonic larvae. For parasitic stages, the infection dynamics on migratory juvenile salmon are described by the delay differential equations:

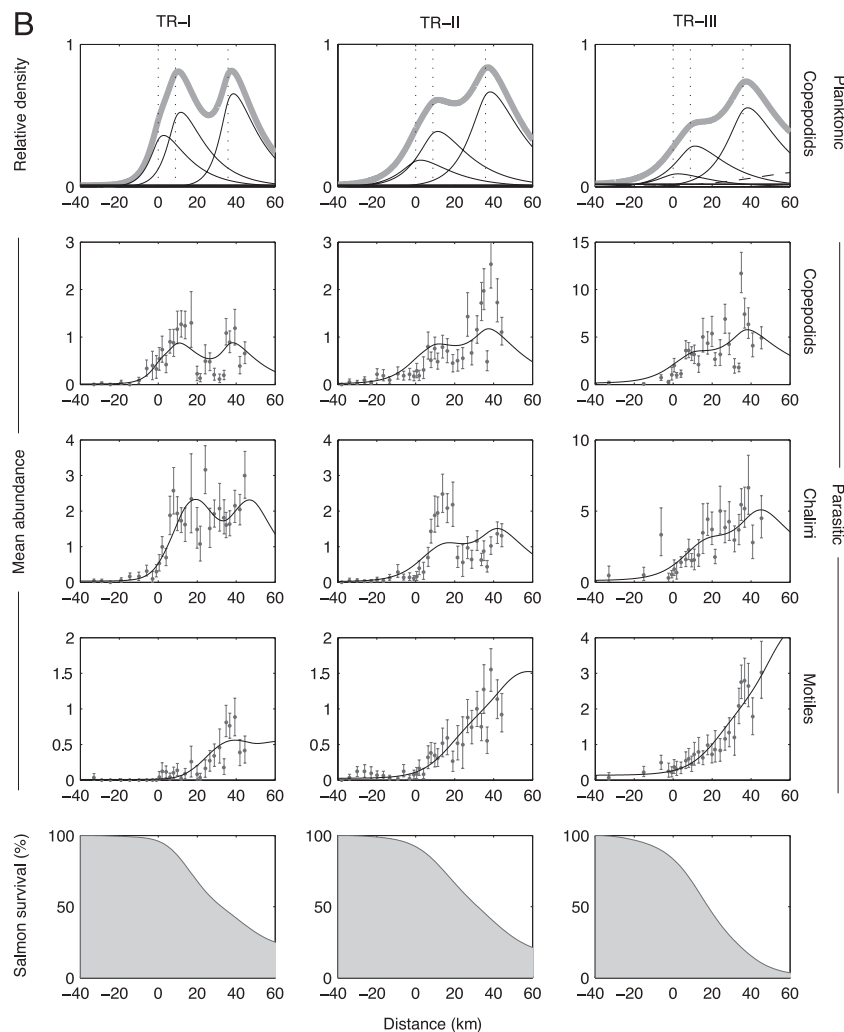


Fig. 2. Sea lice transmission dynamics and survival of juvenile chum salmon (A) and pink salmon (B) migrating past three active salmon farms. The seaward migration of salmon is from left to right, and the farm locations are shown by vertical dotted lines in the first row. The data were collected along the Tribune Channel migration corridor in 2004 (see Fig. 1). The three columns correspond to three replicate sets of samples taken April 18–28 (TR-I), April 28 to May 8 (TR-II), and May 21–29 (TR-III), 2004 (note the change in scale). The first row shows the estimated spatial distributions of planktonic copepodids originating from all sources (thick gray line), from farm salmon (three thin curves), from ambient sources (horizontal thin line), and the second generation of farm-origin lice (dashed curve, TR-III only). Reproduction of lice parasitizing the juvenile salmon was not considered in TR-I and -II because of the absence of gravid female lice in those data sets. The middle three rows depict the mean abundances of lice ($\pm 95\%$ bootstrap confidence interval) and maximum-likelihood model fits (black lines) along the migration route for the developmental progression through parasitic copepodid, chalimus, and motile stages. The bottom row depicts the estimated remaining juvenile salmon population that survived sea lice infestation. Temperature and salinity were measured at each site and averaged 9.0°C and 30.2‰ (TR-I), 10.4°C and 26.1‰ (TR-II), and 12.3°C and 22.2‰ (TR-III).

$$\begin{aligned} \frac{dC}{dx} &= \frac{\beta}{v} [L(x) - L(x - \lambda_c)] \\ \frac{dH}{dx} &= \frac{s_c \beta}{v} [L(x - \lambda_c) - L(x - \lambda_h)], \\ \frac{dM}{dx} &= \frac{s_c s_h \beta}{v} [L(x - \lambda_h) - L(x - \lambda_m)], \end{aligned} \quad [1]$$

which track the mean abundances of copepodid (C), chalimus (H), and motile (M) lice, respectively. Salmon migrate at an average velocity, v , and encounter local densities of infectious planktonic copepodids (L), which then attach to host fish at rate β . The proportions of surviving copepodids and chalimi are s_c and s_h , respectively. The λ are the cumulative distances salmon travel during successive louse developmental stages (C , H , and M).

We constrained the model by imposing independently estimated parameters for the advection, development, and mortality of planktonic larvae. Further, pink and chum data sets shared four parameters (larval dispersion, louse demographic rates, and ratios of farm and ambient louse production rates) in a composite likelihood function that spanned the data sets of both host species. We modeled the occurrence of infection events and subsequent louse survival as a Poisson-binomial process (ref. 13; Fig. 9, which is published as supporting information on the PNAS web site) and used maximum-likelihood and model selection statistics to fit and compare models. The models consisted of only ambient-origin lice, only farm-origin lice, and both.

Mortality Estimation. We analyzed survival data of infested and uninfested juvenile salmon collected from the same populations described in *Transmission Dynamics*. Details of the 2004 data can be found in ref. 34. The 2005 data were collected similarly. The

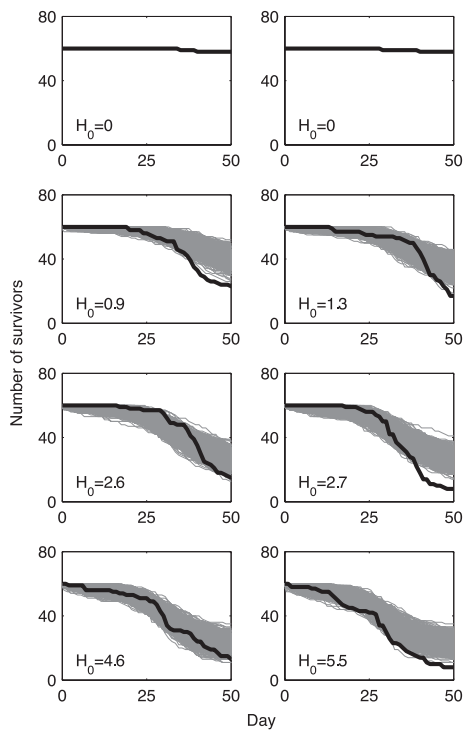


Fig. 3. Survival of juvenile chum salmon over a range of sea lice abundances. Sixty juvenile chum salmon initially infested with H_0 lice (all copepodids or chalimus I/II) were introduced into flow-through ocean enclosures and provisioned with salmon feed. Each image corresponds to an individual enclosure. The black line shows the trajectory for the daily number of survivors. The light-gray lines are the trajectories of 1,000 simulations of the best-fit model. The model was simulated as a Markov chain tracking the number of survivors in time. Each day, the number of mortalities was drawn from the number of survivors on the previous day using a binomial distribution with mortality probability calculated from the best-fit survival model. For all treatment replicates, the model has the same parameter values, except for H_0 , which is specific to each enclosure.

time-series analysis of mortality events consisted of a likelihood-based comparison of survival models that described how lice change in pathogenicity as they mature. The best-fit survival model contained two parasitic stages, a relatively benign pathogen (young chalimus lice) and a severe pathogen (motile stages). The two stages induce mortality in their hosts at rates α_1 and α_2 , respectively. The first stage is divided into a series of n identically and exponentially distributed substages, where n is an estimated constant from the gamma distribution (35).

The survival model was then coupled to the transmission

dynamics model by using the chain rule to map time to space. The coupled model tracks changes in the abundance of the two pathogenic stages of lice ($P_{1,i}$ and P_2) as juvenile salmon migrate to sea:

$$\begin{aligned} \frac{dP_{1,1}}{dx} &= \frac{s_c \beta}{\nu} L(x - \lambda_c) - \frac{1}{\nu} (n\mu_1 + \alpha_1) P_{1,1} \\ \frac{dP_{1,2}}{dx} &= \frac{n\mu_1}{\nu} P_{1,1} - \frac{1}{\nu} (n\mu_1 + \alpha_1) P_{1,2} \\ &\vdots \\ \frac{dP_{1,n}}{dx} &= \frac{n\mu_1}{\nu} P_{1,n-1} - \frac{1}{\nu} (n\mu_1 + \alpha_1) P_{1,n} \\ \frac{dP_2}{dx} &= \frac{n\mu_1}{\nu} P_{1,n} - \frac{\sigma}{\nu} P_2, \end{aligned} \quad [2]$$

where $1/\mu_1$ is the mean duration of the first pathogenic stage, which has variance $(n\mu_1)^{-1}$. Having arrived at the second pathogenic stage, lice die at rate σ , which represents the sum of natural parasite mortality and parasite-induced host mortality rates ($\sigma = \mu_2 + \alpha_2$), which were not separately identifiable. However, σ could be estimated directly from the transmission dynamics data as $\sigma = \nu \cdot (\lambda_m - \lambda_h)^{-1}$. The proportion of juvenile salmon at location x surviving sea lice infestation was then determined by:

$$\frac{dN}{dx} = -\frac{1}{\nu} \left[\alpha_1 \sum_{i=1}^n P_{1,i}(x) + p\alpha_2 P_2(x) \right] N, \quad [3]$$

where $N(x_0) = 1$, and x_0 is the landward extreme of the study area. The quantity p is the proportion of P_1 parasites that survive natural parasite mortality to reach the P_2 stage. There are four parameters ($\alpha_1, p\alpha_2, n, \mu_1$) that were estimated from the survival data and two parameters ($\beta s_c \cdot \nu^{-1}, \sigma$) estimated from the transmission dynamics data.

We thank E. Nelson, B. Connors, S. Rogers, and K. Assonitis, as well as the people of the Broughton Archipelago, for help in the field. R. Routledge (Simon Fraser University, Burnaby, BC, Canada) designed the 2004 survival studies. Comments from B. Anholt, M. Wonham, and two reviewers improved the manuscript. This work was supported by a Natural Sciences and Engineering Research Council Canada (NSERC) Canada Graduate Scholarship D and a D. Allen Birdsall Memorial Scholarship (to M.K.); a senior Canada Research Chair and NSERC Collaborative Research Opportunity grant (to M.A.L.); NSERC Discovery grants (to M.A.L. and J.P.V.); an NSERC/Social Sciences and Humanities Research Council of Canada Major Collaborative Research Initiative grant (to J.P.V.); a Tides Canada grant (to A.M.); and matching funds from the National Research Council of Canada Mathematics of Information Technology and Complex Systems program, the David Suzuki Foundation, the Canadian Sablefish Association, and the British Columbia Wilderness Tourism Association.

- Jackson JBC, Kirby MX, Berger WH, Bjørndal KA, Botsford LW, Bourque BJ, Bradbury RH, Cooke R, Erlandson J, Estes JA, et al. (2001) *Science* 293:629–638.
- Pauly D, Watson R, Alder J (2005) *Philos Trans R Soc London B* 360:5–12.
- Hutchings JA (2000) *Nature* 406:882–885.
- Pauly D, Christensen V, Guenette S, Pitcher TJ, Sumaila UR, Walters CJ, Watson R, Zeller D (2002) *Nature* 418:689–695.
- Pauly D, Alder J, Bennett E, Christensen V, Tyedmers P, Watson R (2003) *Science* 302:1359–1361.
- Naylor RL, Goldburg RJ, Primavera JH, Kautsky N, Beveridge MCM, Clay J, Folke C, Lubchenco J, Mooney H, Troell M (2000) *Nature* 405:1017–1024.
- Naylor R, Burke M (2005) *Annu Rev Environ Res* 30:185–218.
- Naylor RL, Goldburg RJ, Mooney H, Beveridge M, Clay J, Folke C, Kautsky N, Lubchenco J, Primavera J, Williams M (1998) *Science* 282:883–884.
- Fleming IA, Hindar K, Mjølnerod IB, Jonsson B, Balstad T, Lamberg A (2000) *Proc R Soc London Ser B* 267:1517–1523.
- Naylor R, Hindar K, Fleming IA, Goldburg R, Williams S, Volpe J, Whoriskey F, Eagle J, Kelso D, Mangel M (2005) *Bioscience* 55:427–437.
- Naylor R, Anholt BR, Glickman BW (2001) *Can J Fish Aquat Sci* 58:197–207.
- Volpe JP, Taylor EB, Rimmer DW, Glickman BW (2000) *Conserv Biol* 14:899–933.
- Krkošek M, Lewis MA, Volpe JP (2005) *Proc R Soc London Ser B* 272:689–696.
- McVicar AH (1997) *ICES J Mar Sci* 54:1093–1103.
- McVicar AH (2004) *Aq Res* 35:751–758.
- Daszak P, Cunningham AA, Hyatt AD (2000) *Science* 287:443–449.
- Hudson PJ, Rizzoli A, Grenfell BT, Heesterbeek H, Dobson AP (2001) *The Ecology of Wildlife Diseases* (Oxford Univ Press, Oxford).
- Hochachka WM, Dhondt AA (2000) *Proc Natl Acad Sci USA* 97:5303–5306.
- Jolles AE, Cooper DV, Levin SA (2005) *Ecology* 86:2258–2264.
- McCallum H, Dobson AP (1995) *Trends Ecol Evol* 10:190–194.
- Groot C, Margolis L (1991) *Pacific Salmon Life Histories* (UBC Press, Vancouver).
- Bjorn PA, Finstad B (2002) *ICES J Mar Sci* 59:131–139.
- Mackenzie K, Longshaw M, Begg GS, McVicar AH (1998) *ICES J Mar Sci* 55:151–162.
- Tully O, Gargan P, Poole WR, Whelan KF (1999) *Parasitology* 119:41–51.
- Morton A, Routledge R, Peet C, Ladwig A (2004) *Can J Fish Aquat Sci* 61:147–157.
- Nagasawa K (2001) *Hydrobiologia* 453:411–416.
- Beamish RJ, Neville CM, Sweeting RM, Ambers N (2005) *Fish Res* 24:198–208.
- Blazer VS (1992) *Annu Rev Fish Dis* 2:309–323.
- MacKinnon BM (1998) *ICES J Mar Sci* 55:188–192.
- Hudson PJ, Dobson AP, Newborn D (1992) *J Anim Ecol* 61:681–692.
- Mesa MG, Poe TP, Maule AG, Schreck CB (1998) *Can J Fish Aquat Sci* 55:1599–1606.
- Pike AW, Wadsworth SL (2000) *Adv Parasitol* 44:233–337.
- Krkošek M, Morton A, Volpe JP (2005) *Trans Am Fish Soc* 134:711–716.
- Morton A, Routledge R (2005) *Alask Fish Res Bull* 11:146–152.
- Lloyd AL (2001) *Proc R Soc London Ser B* 268:985–993.