CHAPTER NINETEEN

Ecology of a marine ectoparasite in farmed and wild salmon

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19.1 Introduction

The global demand for seafood has outpaced wild fisheries and led to a dramatic increase in aquaculture over the last 30 years (FAO, 2016). Fish that are farmed in open-net pens can exchange parasites and pathogens with wild fish populations (Bjørn et al., 2001; Krkošek et al., 2006), which can change the structure of host populations in coastal seas (Krkošek, 2016). The potential implications of this change for the health and conservation of wild fish populations has spurred research into the causes (Revie et al., 2003; Bateman et al., 2016) and consequences (Krkošek et al., 2011b; Thorstad et al., 2015; Vollset et al., 2015) of parasite transmission between farmed and wild fish, contributing to the general understanding of host–parasite ecology in wild and domesticated animals. This chapter describes the new theoretical insights that have been gleaned from the study of the factors influencing sea louse parasites shared by farmed and wild salmon. We focus on research in Pacific Canada, where wild salmon still outnumber their domesticated counterparts and are an important ecological, economic, and cultural resource.

Host migration and species diversity jointly influence sea louse dynamics on wild salmon. A natural separation of juvenile and adult wild salmon that results from extensive host migration reduces transmission of parasites to small, vulnerable juveniles (Krkošek et al., 2007b). However, the recent introduction of a domestic reservoir host – farmed salmon – has undermined the parasite-related benefits of migration for wild salmon (Krkošek et al., 2006). In Section 19.2 we discuss the roles of host diversity, migration, and reservoir hosts in parasite transmission, and how these factors have affected parasite abundances – and survival – of wild Pacific salmon.

The impact of sea lice differs between farmed and wild salmon, and this has fuelled controversy over how significant these parasites are to host survival (Marty et al., 2010; Krkošek et al., 2011b; Vollset et al., 2015). For wild salmon, ecological processes of the host – namely predation and competition –

https://doi.org/10.1017/9781316479964.019 Published online by Cambridge University Press

combine to influence parasite and host survival, sometimes in unexpected ways. In Section 19.3 we describe how inter- and intraspecific competition and predation mediate the effects of sea lice on wild salmon. Model analyses suggest that the impact of parasites on host populations may be more or less than previously thought, depending on the details of host ecology.

The impact of sea louse parasites on hosts has been the focus of the salmon-farming controversy, but in the last section of this chapter we consider how conditions in aquaculture and wild environments affect parasites. Farming practices generally promote the evolution of pathogen virulence, with high density, accelerated generation time, and low genetic variability in the host population – and aquaculture is no exception (Kennedy et al., 2016). Sea lice have also evolved resistance to chemotherapeutants in many regions, threatening the sustainability the industry (Aaen et al., 2015). The selection pressures for both virulence and drug resistance are influenced by the exchange of parasites between farmed and wild salmon, providing a case study of potential evolutionary ecosystem services that wild hosts may provide (Kreitzman et al., 2018).

Finally, we conclude the chapter with some lousy lessons that have been learned about the management of parasites for wildlife conservation. The potential for sea lice to affect wild salmon ecology and survival seems clear, but the relative importance of sea lice versus other environmental and human factors continues to be debated (Vollset et al., 2018). Theory suggests that the long-term coexistence of wild and farmed salmon hinges on our ability to manage parasites proactively and limit transmission to wild salmon (Orobko, 2016), and a precautionary approach is required to ensure the sustainability of wild salmon in perpetuity.

19.2 Anthropogenic changes to a host–parasite system

Pacific salmon (*Oncorhynchus* spp.) are anadromous fishes that hatch in freshwater rivers or lakes, migrate to spend most of their lives in the ocean, and return to spawn in their natal freshwaters. While in the ocean, Pacific salmon are susceptible to infection by sea lice (*Lepeophtheirus salmonis* and *Caligus clemensi*), which are native marine ectoparasitic crustaceans that are unable to survive in freshwater (Box 19.1). Nearly all Pacific salmon are semelparous and die soon after spawning, long before juveniles hatch and migrate to the open ocean. These life-history characteristics result in a natural separation between adult and juvenile subpopulations, termed migratory allopatry (Krkošek et al., 2007b), that limits transmission of sea lice from adults to vulnerable juveniles during their period of sympatry in the nearshore marine environment (Figure 19.1).

The parasite-avoidance benefits of migratory allopatry can be disrupted by the year-round presence of hosts in nearshore environments (Figure 19.1).

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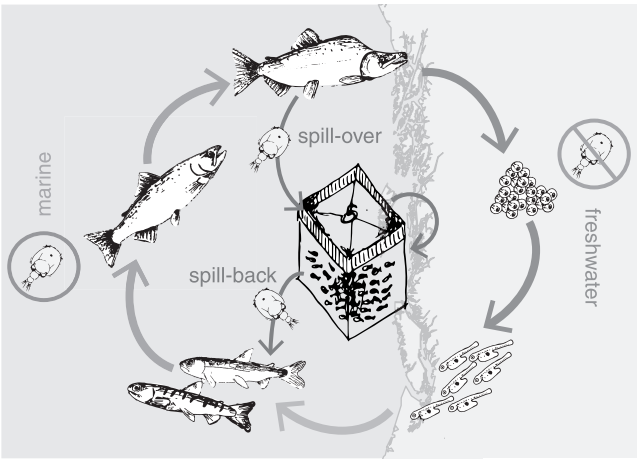

Figure 19.1 Spill-over and spill-back between wild and farmed salmon disrupts the natural separation of adults and juveniles, termed migratory allopatry, which normally minimises transmission of parasites such as sea lice to vulnerable juveniles. (A black and white version of this figure will appear in some formats. For the colour version, please refer to the plate section.)

Box 19.1: Natural history of sea louse parasites

Sea lice are naturally occurring marine ectoparasitic copepods that feed on the epidermis, musculature, and blood of host fish (Pike & Wadsworth, 2000). Two species of sea lice are common on Pacific salmon: *Caligus clemensi* is a generalist parasite of many fish species, including salmon, herring, and stickleback (Table 19.1). *Lepeophtheirus salmonis* (Figure 19.2d) is a salmon specialist and tends to be larger and more pathogenic to hosts (Costello, 2006).

Adult wild salmon often have a low infection intensity but high prevalence of sea lice, with low pathogenicity for adult hosts at endemic levels (Costello, 2006). High burdens of sea lice can cause host morbidity, and the control of sea lice is costly for salmon farms (Costello, 2009). Pathogenicity is highly dependent on the relative sizes of host and parasite, and even low burdens of *L. salmonis* can cause morbidity and mortality of juvenile salmon (Krkošek et al., 2006; Brauner et al., 2012).

As adults, sea lice are polygynous and mobile on the surface of their hosts and move among hosts to find mating opportunities or evade predation (Connors et al., 2011). Sea lice reproduce sexually, and male and female adult sea lice are easily distinguished (Johnson & Albright, 1991a). Males will also form pre-copulatory pairs with pre-adult females, waiting until they reach sexual maturity and are able to mate (Ritchie et al., 1996). These mate-searching and mate-guarding behaviours may increase the probability

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Box 19.1: (cont.)

of finding a suitable mate at low parasite densities, and potentially offset Allee effects in the parasite population (Krkošek et al., 2012).

Once mated, gravid females extrude eggstrings from which free-living nauplii hatch. Nauplii have the potential to disperse tens of kilometres with ocean currents before moulting into infectious copepodites. The development time is highly temperature-dependent (Johnson & Albright, 1991b), and thus the dispersal potential of nauplii may vary (Groner et al., 2014). Copepodites must attach to a host before moulting through two chalimus stages that are attached to the host by a central filament (Hamre et al., 2013), and two pre-adult stages that are mobile on the host. The generation time is highly temperapture-dependent, ranging from 16 weeks at 7°C to < 8 weeks at 12°C (Heuch et al., 2000).

Because *L. salmonis* are a salmon-specific parasite and the majority of salmonids in Pacific Canada are semelparous, there are few alternate hosts for *L. salmonis* over winter near river mouths (although cutthroat trout and winter-run Chinook may be present, their densities are typically very low; Krkošek et al., 2007b; Table 19.1). The generalist sea louse *C. clemensi* can have higher prevalence on young juvenile salmon than *L. salmonis* because *C. clemensi* has a broader range of hosts, including herring and stickleback (Table 19.1), which can be abundant in nearshore environments over winter and when juvenile salmon emerge from rivers in early spring (Jones et al., 2006; Krkošek et al., 2007a). However, *C. clemensi* tends to be less pathogenic than *L. salmonis* (Costello, 2006), and thus juvenile salmon mortality due to *C. clemensi* is generally low in natural systems (Krkošek et al., 2007b, 2011b).

In recent decades, reductions in the growth of fisheries coupled with aquaculture expansion (FAO, 2016) have led to large numbers of domesticated salmonids now inhabiting some coastal ecosystems, altering the dynamics of sea lice on wild salmonids (Thorstad *et al.*, 2015; Krkošek, 2016). Farmed Atlantic salmon (*Salmo salar*) raised in open-net pens (Figure 19.2a,b) can act as reservoir hosts that acquire sea lice from returning adult salmon in the fall (spill-over), harbour and amplify sea louse populations over the winter, and then transmit sea lice to out-migrating juvenile salmon in the spring (spill-back, Figure 19.1; Daszak et al., 2000). The presence of farmed salmon thereby disrupts migratory allopatry and can lead to *L. salmonis* infestations of vulnerable juvenile salmon (Krkošek et al., 2007b). Indeed, sea louse infestations of wild juvenile salmon along their migration tend to intensify once these salmon have passed salmon farms (Figure 19.2c,d; Krkošek et al., 2006), suggesting that farms can act as infestation hotspots. Fewer sea lice have been

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Table 19.1 *Host species for sea lice.**

Hosts of *Caligus clemensi* and *Lepeophtheirus salmonis*	
Chum salmon *Oncorhynchus keta*	Migrate to sea immediately after hatching Generation time of 3–6 years Direct mortality due to sea lice[1,2] No evidence of population-level declines correlated with sea louse infestations[3]
Pink salmon *O. gorbuscha*	Migrate to sea immediately after hatching Generation time of 2 years Direct mortality[1,2] and increased predation[4] due to sea lice Population-level declines correlated with sea louse infestations[5,6,7]
Coho salmon *O. kisutch*	Spend 1 year in freshwater before migrating to sea Generation time of 3–5 years Major predators of juvenile pink and chum salmon in freshwater and near-shore marine Show selective predation on pink salmon[8,9] and parasitised prey[4,9] Incur trophic amplification of sea lice[10] Population-level declines correlated with sea louse infestations[6,11]
Sockeye salmon *O. nerka*	Spend 1–2 years in freshwater before migrating to sea Generation time of 3–5 years Mainly infested with *C. clemensi* Impact of *C. clemensi* on competitive foraging[12] and growth Negative population-level impact of aquaculture production, mediated by competition with pink salmon[13]
Chinook salmon *O. tshawytscha*	Ocean-type may migrate to sea immediately; stream-type spend one year in freshwater Most spend 3–4 years in the ocean, but as few as one year and as many as 8 years Return to spawn relatively early, in June-July (hence the term 'spring salmon'), and may be the first adult salmon to transmit lice to juveniles in the absence of farms
Cutthroat trout *O. clarkii clarkii*	Do not migrate to the open ocean but reside in streams and brackish bays year-round Periodic forays into freshwater likely regulate infestations Predators of juvenile pink and chum salmon
Farmed Atlantic salmon *Salmo salar*	Non-native species in Pacific Canada Housed in open-net pens over an 18-month production cycle Can acquire sea lice from returning adult salmon, and transmit back to out-migrating juveniles in the spring Sea lice are managed above a threshold parasite burden by harvest and treatment with chemotherapeutants[7,14]
Hosts of *Caligus clemensi* only	
Pacific herring *Clupea pallasii pallasii*	Predominant forage fish in Pacific Northwest Aggregate nearshore for mass spawning in late winter/early spring, but migration patterns are not well described

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Table 19.1 (*cont.*)

	Large source population for *C. clemensi* during initial period of juvenile pink and chum out-migration
Three-spined stickleback *Gasterosteus aculeatus*	Year-round residents of nearshore and brackish waters Low abundance relative to herring and salmon Suitable hosts for *C. clemensi* Reported to have infestations of *L. salmonis*[15] but no evidence that the latter can complete its life cycle on stickleback[16]

* Not an exhaustive list of all host species, but covers the most common hosts mentioned in the chapter. Literature cited: (1) Morton and Routledge, 2005, (2) Krkošek et al., 2006, (3) Peacock et al., 2014, (4) Krkošek et al., 2011a, (5) Krkošek et al., 2007a, (6) Krkošek et al., 2011b, (7) Peacock et al., 2013, (8) Hargreaves and LeBrasseur, 1985, (9) Peacock et al., 2015, (10) Connors et al., 2010a, (11) Connors et al., 2010b, (12) Godwin et al., 2015, (13) Connors et al., 2012, (14) Saksida et al., 2010, (15) Jones et al., 2006, (16) Losos et al., 2010.

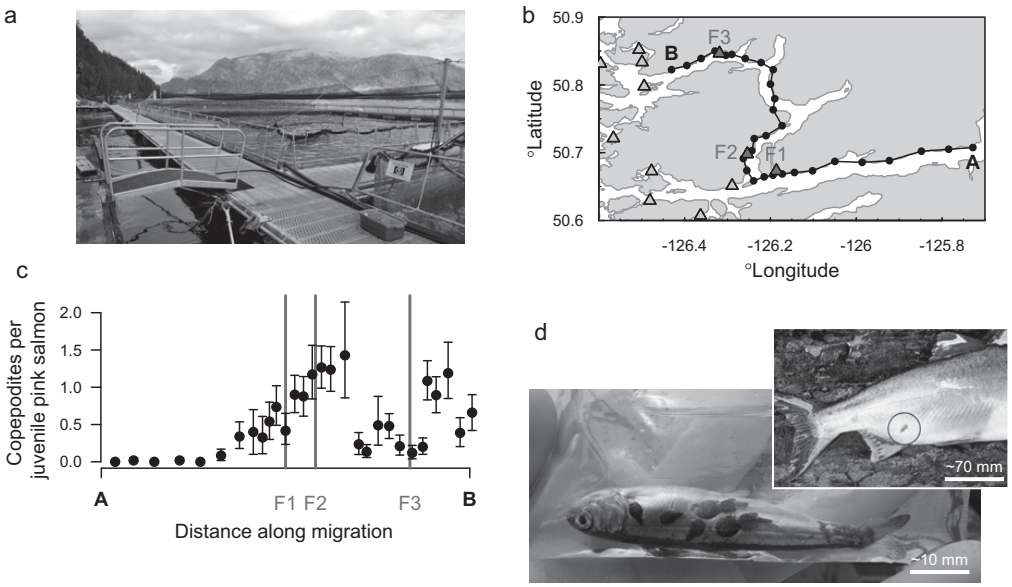

Figure 19.2 (a) An open-net salmon farm on the coast of British Columbia, Canada. (b) The migration route of juvenile salmon (black line; A to B) in the Broughton Archipelago, British Columbia, passes by several salmon farms (filled triangles). Black points are locations where juvenile pink salmon were captured and assessed for sea louse parasites (Krkošek et al., 2006). (c) The mean number of copepodid sea lice (±95% bootstrapped confidence intervals) per juvenile pink salmon sampled at black points in (b) from 18 to 28 April 2004 (Krkošek et al., 2006). (d) An infestation of motile sea lice on a juvenile pink salmon can have dramatic effects due to the large size of parasites relative to their host, while infections of adult salmon (inset) are much less pathogenic because hosts are larger and have developed protective scales. Photos: S Peacock, inset: C Miller. (A black and white version of this figure will appear in some formats. For the colour version, please refer to the plate section.)

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observed on out-migrating wild salmon in regions without salmon farming (Krkošek et al., 2007b) and in years when farms are fallow (Morton et al., 2005). Similarly, in Europe, sea louse numbers on wild trout are higher in regions of intensive salmon aquaculture than in areas without farms (reviewed by Thorstad et al., 2015).

The population-level impacts of parasites on wildlife can be exacerbated by the presence of reservoir hosts, such as domestic animal populations, because the usual density-dependent mechanisms that regulate epizootics do not apply (De Castro & Bolker, 2005). There is empirical evidence from other host–parasite systems that, in the absence of reservoir hosts, parasites may be at least as important as predation in regulating wild population dynamics (Watson, 2013). Theory predicts that this control of wild populations will cause the parasite populations to self-regulate: high parasite burdens result in parasite-induced host mortality, and for parasites with density-dependent transmission like sea lice (Frazer et al., 2012; Jansen et al., 2012), lower host population density leads to less transmission (De Castro & Bolker, 2005). However, in the presence of reservoir hosts, infestation pressure can remain high, even at low densities of wildlife hosts, potentially leading to host extinction (De Castro & Bolker, 2005; Krkošek et al., 2013a). Evolutionary similarity to domesticated animals has also been identified as an important factor increasing the potential risk of parasites to wildlife (Pedersen et al., 2007). Together, these factors highlight the potential for parasite-mediated declines of exposed wild salmon populations and heighten the conservation concern about sea louse transmission from farmed salmon to migrating wild juvenile salmon (Krkošek et al., 2007a).

The impact of farm-origin sea lice on wild Pacific salmon differs among host species, in part due to interspecific variation in life-history traits (Table 19.1). While some salmon species, including coho salmon and sockeye salmon, spend a year or two in freshwater before migrating to sea, pink salmon and chum salmon enter the marine environment immediately after hatching (Groot & Margolis, 1991). Pink and chum salmon are small and lack protective scales when entering the marine environment and are therefore expected to be the Pacific salmon species most vulnerable to farm-origin sea lice. Indeed, field-based experiments with wild-caught pink and chum salmon have demonstrated significant direct louse-induced individual mortality for both species (Krkošek et al., 2009; Figure 19.3a). Other salmonids, such as cutthroat trout and Dolly Varden, migrate in and out of coastal seas and estuaries on an annual basis and may moderate the impact of sea lice by periodically returning to freshwater, thereby reducing their louse loads. In Europe, where there is a longer history of sea louse transmission from salmon farms, heavily infested Atlantic sea trout (*Salmo trutta*) have been found to return early to freshwater

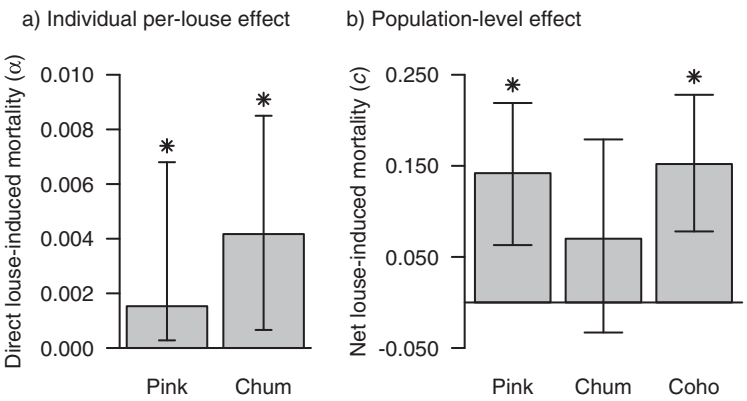

Figure 19.3 (a) The individual parasite-induced mortality (per sea louse per day) of captive pink and chum salmon estimated from field-based experiments (coho salmon estimate was not available) (Krkošek et al., 2009). (b) The population-level effect of farm-origin sea lice on wild salmon survival for pink and coho salmon (Krkošek et al., 2011a) and chum salmon (Peacock et al., 2014), shown as the parameter for louse-induced mortality (*c*) in an adapted Ricker population model for spawner–recruit data (Krkošek et al., 2011b). In both panels, bars are 95% confidence intervals on the estimates and stars indicate effects that are significantly different from zero.

(Thorstad et al., 2015), suggesting that life history and behaviour not only moderate the effects of sea lice, but may also be adaptive in response to them.

In Pacific Canada, much research has focused on whether direct parasite-induced mortality of host individuals results in reduced overall survival and recruitment for host populations. Studies in Europe have used an experimental approach to answering this question by tracking the oceanic survival of individual smolts that have been treated with anti-louse chemotherapeutants to that of untreated conspecifics. These studies have shown a significant decline in survival due to sea louse infection (Krkošek et al., 2013b; Vollset et al., 2015), but such an approach is more challenging for Pacific salmon due to their small size as juveniles (limiting marking options), variable age at maturity, and high natural mortality at sea. However, long-term data on spawner abundance in coastal British Columbia, Canada, allow for spatial and temporal comparisons of survival for populations exposed and unexposed to salmon farming, thus providing the opportunity for correlative examinations of the effect of sea lice from farmed salmon on wild salmon population dynamics. These comparisons have revealed a negative correlation between sea louse outbreaks on farmed salmon and survival of pink and coho salmon (Krkošek et al., 2007a, 2011b; Connors et al., 2010b). Similar analyses have identified aquaculture production, together with ocean climate and interspecific competition at sea, as factors associated with decline of sockeye salmon

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populations (Connors et al., 2012). Chum salmon populations, however, show no correlation between their survival rates and the magnitude of sea louse outbreaks on sympatric farmed salmon (Peacock et al., 2014; Figure 19.3b), despite estimates of direct parasite-induced mortality that are similar to pink salmon at the individual level. The data for chum salmon suggest that direct parasite-induced mortality (Figure 19.3a) is not the only factor determining the population-level impacts of parasites (Figure 19.3b). The discrepancy between individual- and population-level effects of sea lice has spurred further research into the ecological interactions of hosts that might mediate population-level effects.

19.3 Ecological factors mediating the impact of parasites

The true impact of parasites on wildlife populations can be difficult to estimate because animals rarely die from parasites alone. Before parasite burdens reach intensities that result in parasite-induced mortality, the *ecological* effects of parasites – that is, the ways in which parasites affect a host's ability to compete for food or avoid predators – become apparent. Thus, indirect ecological effects of parasites may determine how parasitism affects wildlife populations more than direct parasite-induced mortality (Ives & Murray, 1997; Hatcher et al., 2012).

19.3.1 Competition

Competition for resources is a fundamental challenge for all wildlife. Chief among these resources is food, as feeding determines the nutrients and energy an individual can put towards movement, growth, reproduction, and immune responses. For Pacific salmon, which migrate in large aggregations through variable prey densities, competition for food likely plays a large role in determining growth and survival (Groot & Margolis, 1991).

19.3.1.1 Intraspecific competition

Parasites can change the outcome of competitive interactions between individuals within a group, and these changes can have implications for host survival. Juvenile salmon may be particularly susceptible to parasite effects on competition, as they migrate in large schools to swamp and evade predators (Eggers, 1978; Furey et al., 2016). In environments with limited resources, parasitism can shift the competitive balance between individuals over food.

The influence of sea louse parasites on foraging in salmon has been best-studied in sockeye salmon (Table 19.1). Juvenile sockeye salmon feed primarily on zooplankton, which are spatially and temporally patchy (Chittenden et al., 2010; McKinnell et al., 2014). Some populations of juvenile sockeye salmon experience over 99% prevalence of *C. clemensi* sea lice while migrating through these patchy and food-limited environments (Price et al., 2011; Godwin et al.,

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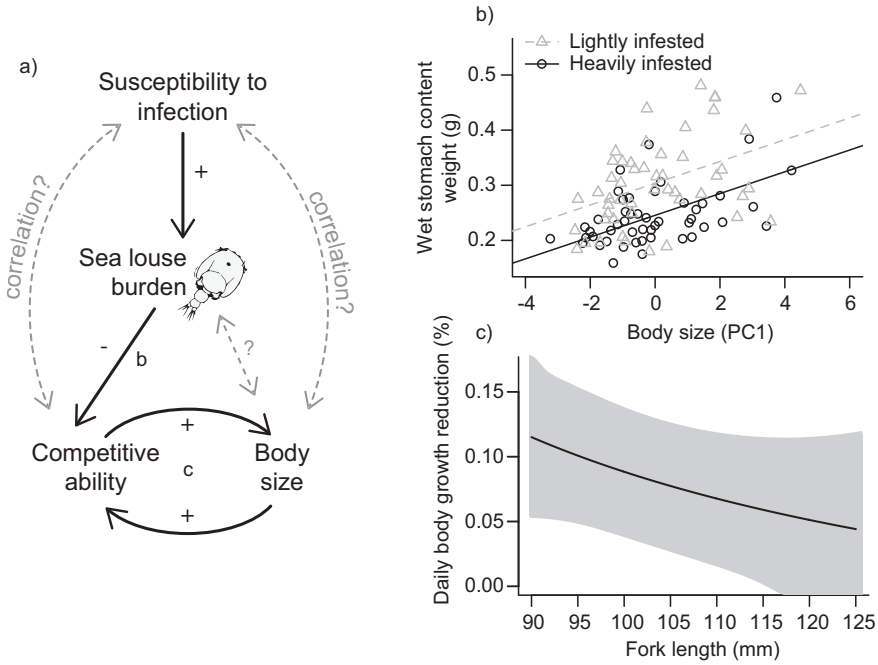

Figure 19.4 (a) The relationship between sea louse parasites, competitive ability, and body size for juvenile sockeye salmon has been investigated in field-based experiments, but the net impact on the host population may depend on whether susceptibility to infestation is biased towards individuals of a certain body size or competitive ability. (b) Comparison of body size and wet stomach content weigh between heavily infested (top ~10% of sample infection intensity) and lightly infested (bottom ~10% of sample infection intensity) juvenile sockeye salmon. Heavy infestation has been related to reduced competitive ability, but the competitive ability is also strongly related to body size. (c) Examination of growth increments for heavily infested and uninfested fish suggest that the percent daily reduction in body growth per sea louse (i.e. heavily infested – uninfested / uninfested) is a decreasing function of body size (line = mean, shaded region = ±95% CI). See Godwin et al. (2015, 2017) for details.

2015). *Caligus clemensi* tend to be less pathogenic than *L. salmonis* to juvenile fish because *C. clemensi* are smaller and more mobile and hence spend less time on individual hosts (Table 19.1; Costello, 2006), but high parasite burdens may nonetheless have ecological effects on host fitness (Figure 19.4a).

Field experiments suggest that heavily infested juvenile sockeye salmon have lower competitive foraging abilities than lightly infested or uninfested individuals (Figure 19.4b; Godwin et al., 2015), leading to reduced foraging success (Godwin et al., 2017) and body growth (Figure 19.4c; Godwin et al., 2017). Studies in Europe corroborate this finding, with scale analyses of sea trout showing reduced growth associated with intensive salmon farming and

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sea louse infestations (Thorstad et al., 2015). Furthermore, larger juvenile sockeye salmon also have higher rates of feeding (as measured by stomach content weight) than smaller conspecifics (Figure 19.4b), and daily growth increments on otoliths (ear bones) indicate larger fish have faster growth rates that are less-affected by parasites, thereby potentially amplifying divergent growth between infested and uninfested fish. Growth is a key component of fitness for many organisms, especially salmon, whose early marine growth is a determinant of overall survival (Beamish et al., 2004; Moss et al., 2005). Over time, these louse-associated growth effects may therefore ultimately affect survival.

While competitive outcomes can be understood at the level of individuals, ecologists are typically interested in population-level impacts. The net effect of parasite-modified competition on host populations may depend on whether the susceptibility to infestation is independent of both body size and competitive foraging ability of individuals (Figure 19.4a). If infestation is non-random such that individuals that are either smaller or less competitive are also more susceptible to infestation, then the impact of parasite-modified competition will reinforce existing differences in growth potential. In this case, population-level effects of reduced growth and survival due to sea lice may be compensatory, as those individuals would have been 'lost' from the population even in the absence of sea lice. However, if infestation is independent of body size or competitive ability, then 'healthier' individuals may also suffer infestations and a reduction in competitive ability, resulting in fewer highly competitive individuals. This may result in less equal resource allocation within the host population, and overall lower population health.

19.3.1.2 Interspecific competition

The previous section indicates that parasite-modified intraspecific resource competition occurs for juvenile sockeye salmon, but the influence of parasites can also include interspecific competition for resources (Hatcher et al., 2006). How parasite-modified competition affects communities depends on whether the parasite is shared among competitors, and whether the stronger or the weaker competitor is more affected by the parasite (Hudson & Greenman, 1998). If the stronger competitor is affected, then parasite-modified competition can promote coexistence, whereas if the weaker competitor is affected, then parasites may result in exclusion of the weaker competitor.

For Pacific salmon, interspecific competition can influence the growth, age-at-maturity, and survival of individuals as well as recruitment (Ruggerone & Connors, 2015). The number of recruits per spawner in sockeye salmon from the Fraser River, for instance, is negatively correlated with pink salmon abundance at oceanic scales due to competitive interactions between sockeye and pink salmon as adults in the Pacific Ocean (Figure 19.5; Connors et al.,

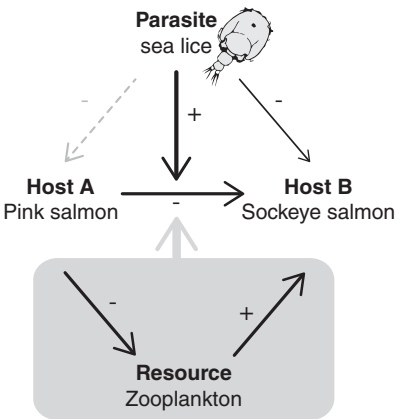

Figure 19.5 The impact of resource competition with pink salmon in the open Pacific Ocean on sockeye salmon survival may be amplified when farmed salmon (and their associated sea lice and other pathogens) are abundant (Connors et al., 2012). This indirect effect of parasite-modified competition is stronger than the direct effect of parasites alone. Sea lice also affect pink salmon survival at local scales (dashed light arrow), but competitive interactions with adult sockeye salmon occur at oceanic scales and thus the pink salmon competitors do not share the same infestation pressure as their sockeye salmon competitors.

2012). Aquaculture production occurring on wild juvenile sockeye migration routes, which is correlated with increased transmission of sea lice to juvenile salmon (Price et al., 2010, 2011), appears to amplify the negative impact of competition with pink salmon later in marine life (Connors et al., 2012). Thus, the reduced competitive foraging ability observed in small-scale, individual-level experiments (Figure 19.4b) may translate to lower survival of sockeye salmon populations. The interactive effect of pink salmon competition and aquaculture production is stronger than the effect of aquaculture production alone suggesting that in this case, the indirect effect of parasites via parasite-modified competition is more important than direct effects on sockeye salmon survival (Figure 19.5).

19.3.2 Predation

For most wildlife, predation affects individual survival and shapes the characteristics, including parasite loads, of surviving populations. For juvenile salmon, predation is the primary source of mortality, causing up to 90% mortality of pink salmon populations during the first three months of marine life (Parker, 1968). Mounting theory and evidence suggest that sea louse effects on predation may be a key determinant of the population-level impacts of epizootics.

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19.3.2.1 Parasite-mediated predation with a single prey species

The effect of predation on host–parasite dynamics depends on whether parasites make prey more or less susceptible to predation (Packer et al., 2003). In many species, infected prey are easier for predators to identify and catch, making those prey more susceptible to predation than uninfected conspecifics (Hudson et al., 1992; Johnson et al., 2006). However, if predators incur a cost for consuming infested prey, as is the case with trophically transmitted parasites (Lafferty, 1992), they may avoid parasitised prey.

Yearling coho salmon are one of the main predators of juvenile pink and chum salmon during their early marine life (Hargreaves & LeBrasseur, 1986). Experiments have shown that predators, including coho salmon smolts and cutthroat trout, preferentially target juvenile pink and chum salmon that are infested with sea lice (Krkošek et al., 2011a). Whether this selective predation results in increased mortality in the prey population depends on whether predation on parasitised prey is compensatory or additive to predation that would occur in the absence of parasites. If predators preferentially target parasitised prey but do not increase overall consumption, predation is compensatory and also leads to lower average parasite burdens among prey as heavily infested individuals are consumed.

Theory that merges host–macroparasite models with predator–prey models has helped to shed light on the conditions under which compensatory parasite-mediated predation occurs. Classical host–macroparasite models track the abundance of hosts (H) and the mean parasite burden (M; Box 19.2). These models have been adapted for juvenile salmon and sea lice to track a cohort of juvenile salmon that becomes infected and whose survival declines due to direct parasite-induced mortality *and* predation following a type II functional response (Figure 19.6a; Krkošek et al., 2011a).

The type II functional response describes a predation rate that increases with prey density until predators are limited by the time it takes to handle and digest prey. The predation rate is described by two parameters: a capture rate and a handling time (Holling, 1959). For juvenile salmon, experiments suggest that the capture rate increases with the prey's parasite burden (Krkošek et al., 2011a; Peacock et al., 2014). The type II functional response often applied to fish predation can be adapted to incorporate an increase in the capture rate with increasing mean parasite burden, M, such that the average predation rate within the prey population is:

$$f_{(H,M)} = \frac{\overbrace{(\theta + \sigma M)}^{\text{capture rate}} \; H}{1 + T_h(\theta + \sigma M)H} \tag{19.1}$$

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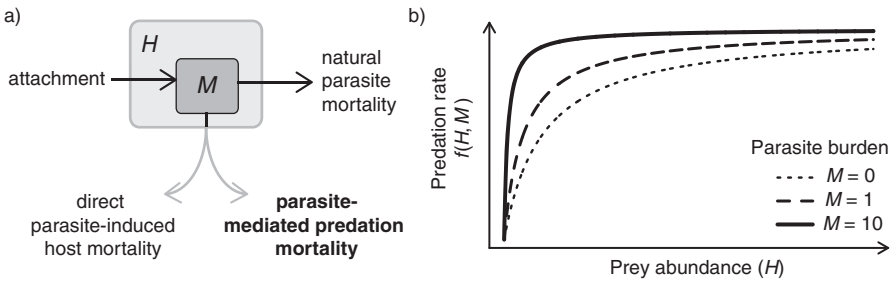

Figure 19.6 (a) Graphical description of a host–macroparasite model adapted to juvenile salmon and sea lice to include predation. The parasite load M increases due to attachment of free-living larvae and decreases due to natural parasite mortality. Juvenile salmon hosts H suffer mortality during their first few months of marine life due to parasites and parasite-mediated predation. (b) The rate of parasite-mediated predation is assumed to follow a type II functional response, with capture rates increasing with the mean parasite burden, M. As prey abundance increases, predators are limited by the time it takes to handle and digest prey, and the three lines eventually converge.

Box 19.2: Modelling host–parasite population dynamics

Mathematical models describing the growth and spread of infectious pathogens through a host population have been integral to the understanding of disease dynamics in both human and wildlife populations (May & Anderson, 1991; Hudson et al., 2002). In contrast to SIR models (Anderson & May, 1979), macroparasite models track the intensity of infection and the degree of parasite aggregation among hosts. Parasite aggregation is common (Shaw et al., 1998) and can fundamentally change how parasites influence host populations because mortality of heavily infected hosts will result in disproportionate mortality in the parasite population (Anderson & May, 1978). Explicitly considering the parasite burden and distribution among hosts is therefore important when the impact on hosts is proportional to parasite burden, as is the case of sea louse infestations of salmon.

Anderson and May (1978) developed a mathematical model for understanding host–macroparasite dynamics that consisted of two equations. The first equation describes the change in the host population, H:

$$\frac{dH}{dt} = \left(\underbrace{a}_{\textbf{birth}} - \overbrace{\underbrace{b}_{\substack{\text{natural}\\\text{mortality}}} - \underbrace{\alpha \sum_{i=0}^{\infty} i\, q(i)}_{\substack{\text{parasite-induced}\\\text{mortality}}}}^{\textbf{host mortality}} \right) H \tag{19.2}$$

where a is the rate of host birth and b is the rate of natural (background) host mortality. The rate of parasite-induced host mortality depends on the number of parasites per host, and is equal to αi for a host infected with

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Box 19.2: (cont.)

i parasites. The parasite-induced mortality of the host population is derived by considering the mortality of hosts with i parasites multiplied by the proportion of hosts that harbour i parasites, $q(i)$ and summed over all possible numbers of parasites. This term can be simplified to the expected parasite-induced mortality from the mean parasite burden, $M = P/H$:

$$\frac{dH}{dt} = (a - b - \alpha M)H \tag{19.3}$$

The total parasite population changes according to:

$$\frac{dP}{dt} = \underbrace{\beta LH}_{\textbf{transmission}} - \underbrace{\mu P}_{\textbf{natural mortality}} - \underbrace{\sum_{i=0}^{\infty} (b + \alpha i)\, i\, q(i)}_{\text{mortality due to host mortality}} \tag{19.4}$$

where β is the transmission coefficient, $L(t)$ is the density of infectious parasite larvae in the environment, μ is the mortality rate of parasites. The model assumes that parasites die when their host dies due to (1) natural mortality at rate b, and (2) parasite-induced host mortality at per-parasite rate α. The second component of this term depends on the aggregation of parasites among hosts; when parasites are highly aggregated, host mortality due to parasites will tend to result in higher rates of parasite death as many parasites die along with their heavily infested hosts. A negative binomial distribution, which is common for macroparasites (Shaw et al., 1998) is assumed.

Finally, to complete the parasite life cycle, the pool of infectious parasite larvae in the environment changes according to:

$$\frac{dL}{dt} = \kappa P - \gamma L - \beta LH \tag{19.5}$$

where κ is the rate of shedding of parasite larvae by attached parasites and γ is the per-capita mortality of parasite larvae in the environment. Often, the larval stage is short relative to the host and parasite dynamics, and a common assumption is that parasite larvae are at equilibrium ($dL/dt = 0$). This assumption simplifies the model to just two equations: one for the host population and one for the attached parasite population. In other cases, the density of infectious larvae is influenced by external forces and may be modelled separately. For example, the densities of larvae produced by sea lice on farmed salmon are much greater than the reproduction of lice on juvenile salmon themselves (Krkošek et al., 2005). Thus, models of sea louse dynamics on juvenile salmon have either included salmon farms as external sources of parasite larvae (e.g. Krkošek et al., 2005, 2006) or assumed that the density of larvae is constant (e.g. Krkošek et al., 2011a; Peacock et al., 2014).

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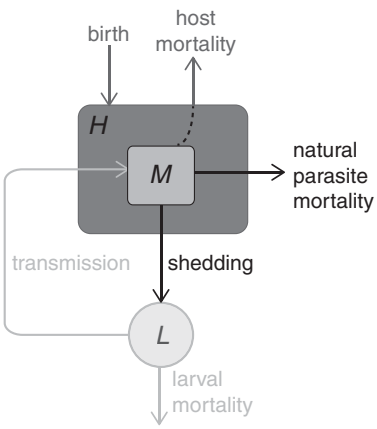

Figure B19.2.1 Model diagram for a host–macroparasite population model with three dynamic variables: the host population H, mean parasite burden M, and density of infectious larvae in the environment L. The model assumes that parasites die when their host dies (dashed line).

where H is the abundance of prey (i.e. hosts), θ is the baseline capture rate in the absence of parasites, σ is the per-parasite increase in the capture rate, and T_h is the handling time (Krkošek et al., 2011a, Peacock et al., 2014). The predation rate increases with the number of parasites at low prey abundance but not at high prey densities, where predators are limited by their handling time (Figure 19.6b). When predators are limited by prey handling time parasites alter *who* gets eaten, but not the overall *number* of prey consumed, and parasite-mediated predation is said to be compensatory. Analysis of the host–parasite population model (Figure 19.6a) also indicates that predation can amplify host mortality due to parasitism while simultaneously decreasing parasite burdens, creating a paradox that parasite-induced mortality may be high when observed parasite burdens are low (Krkošek et al., 2011a).

19.3.2.2 *Multi-host food webs*

Additional complexity may arise in multi-host systems if generalist parasites alter food web dynamics, for example by shifting predator pressure among prey species. Juvenile pink and chum salmon have similar early life histories (Table 19.1) and both are susceptible to sea louse infestations (Figure 19.3a) and experience high levels of predation during early marine life (Parker, 1969); however, coho predators seem to selectively consume pink salmon over chum salmon (Hargreaves & LeBrasseur, 1985; Peacock et al., 2015). The reasons for this prey preference are unknown, but it may be that preference increases when prey are infested and easier to catch (Peacock et al., 2014). This shift in predation would not only affect mortality

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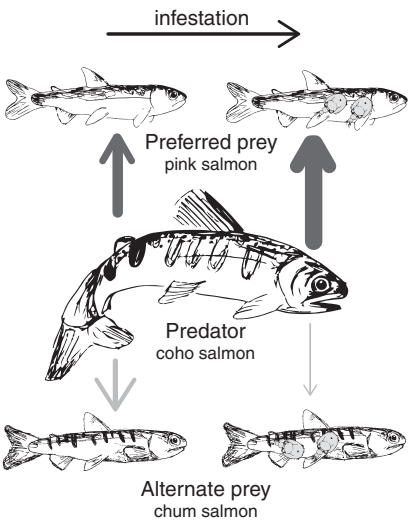

Figure 19.7 Sea lice make juvenile pink and chum salmon more vulnerable to predation by coho salmon, resulting in selective predation on infested prey. However, in this multi-prey system, predators preferentially consume pink salmon over chum salmon. Thus, when prey are infested and easier to catch, predation may focus on preferred prey (pink), which can result in a parasite-mediated release from predation for alternate prey (chum). Here, the thickness of the arrows represents the relative predation rates in this conceptual model.

of pink salmon, but could relieve mortality of the less-desirable prey, chum salmon (Figure 19.7).

Once again, formalising these ideas in a mathematical model helps to clarify assumptions and mechanisms, and allows for an exploration of the possible outcomes in the multi-prey scenario. The type II functional response in equation (19.1) can be adapted to allow for predation on multiple species with both selective predation on parasitised prey and preferential capture of one prey species over another (Figure 19.8a). The average predation rate on prey of species 1 with M_1 parasites, in the presence of an alternate prey, type 2, can be written:

$$f_1(H_1, H_2, M_1, M_2) = \frac{(\theta_1 + \sigma_1 M_1)H_1}{1 + T_h[(\theta_1 + \sigma_1 M_1)H_1 + (\theta_2 + \sigma_2 M_2)H_2]} \tag{19.6}$$

The definitions of parameters are as given for equation (19.1), but with subscripts 1 and 2 referring to the species-specific parameters and variables. The key difference from equation (19.1) is that the denominator in the multi-prey functional response considers the total time that each predator spends catching and consuming prey of both species.

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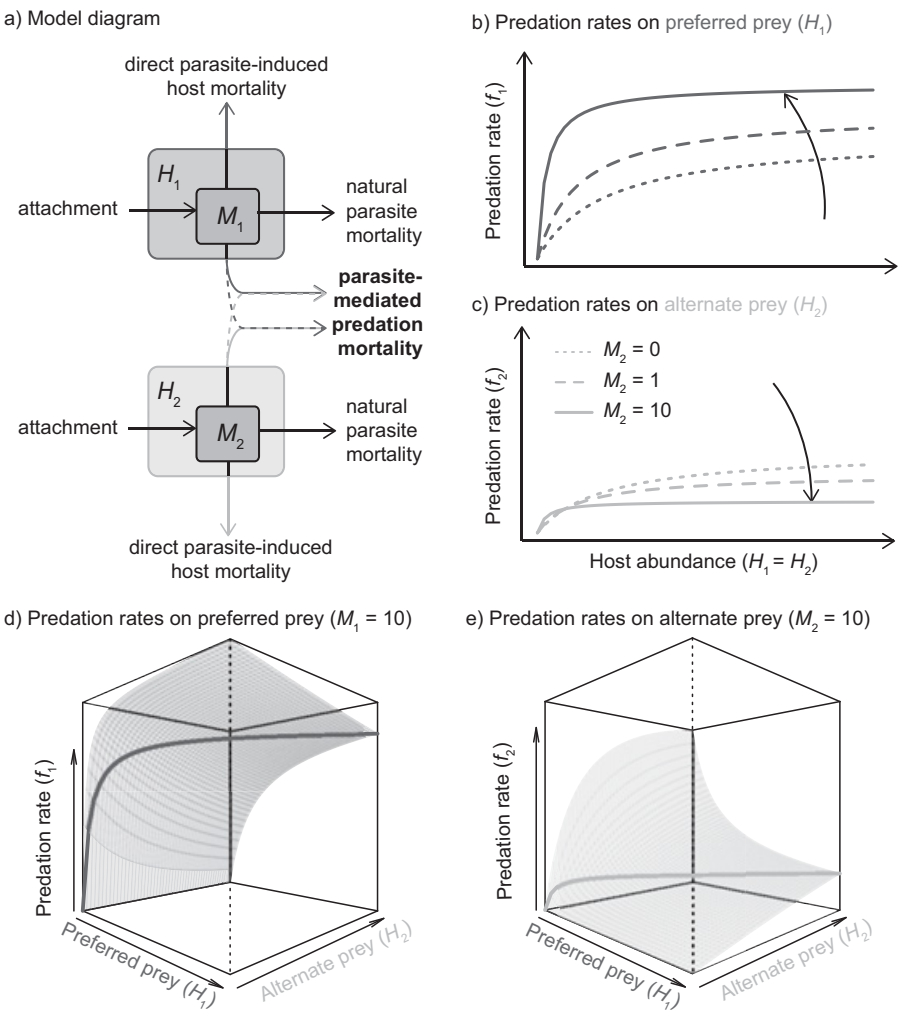

Figure 19.8 (a) Graphical description of a host–macroparasite model for parasite-mediated predation on two host species, H_1 and H_2, that interact via a shared predator (dashed lines; equation (19.5)). Predation rates on the two hosts may differ due to a predator preference. (b) Predation rates on preferred prey will tend to increase with parasite burden (Figure 19.6). (c) Predation rates on alternate prey will decline with parasite burden, but this decline depends on the abundance of prey (d,e; shown here for $M_1 = M_2 = 10$). Notably, if the abundance of preferred prey is low, predation rates on alternate prey will remain high (d). The lines in (d) and (e) correspond to the solid curves in (b) and (c), respectively, where $H_1 = H_2$.

In the case of juvenile salmon, the two prey species of interest are pink salmon (denoted by subscript 1) and chum salmon (denoted by subscript 2). Because coho salmon preferentially consume pink salmon

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over chum salmon, it can be assumed that $\theta_1 > \theta_2$. If predators not only show a base preference for pink salmon, but also increase that preference when prey is parasitised (Figure 19.8b,c), then $\sigma_1 > \sigma_2$. In this case, the capture rate of pink salmon increases with parasites more quickly than the capture rate of chum salmon increases with parasites. This model reveals that chum salmon may experience a 'parasite-mediated release' from predation (Peacock et al., 2014), whereas predation rates on preferred pink salmon increase with parasites regardless of prey density (Figure 19.8b,c). If the shift in predation pressure towards preferred prey is extreme and pink salmon are abundant enough to satiate predators (Figure 19.8d,e), then reduced predation on chum salmon may offset the direct effects of sea lice, or even result in a predation-mediated benefit of infestation. This may explain why there is no evidence of a population-level effect of sea lice on chum salmon, despite the significant declines in pink salmon correlated with the same sealouse epizootics (Figure 19.3b).

19.3.2.3 Parasites that escape predation

Although some parasites with complex life cycles rely on their hosts being eaten in order to complete their life cycle (Lafferty, 1999), other parasites – including sea lice – find the gut of a predator to be an inhospitable place. Sea lice have been observed to abandon their hosts during predation events (Connors et al., 2008); a substantial proportion of those that escape end up on predators (Connors et al., 2008). Trophic transmission of sea lice from prey to predators can amplify parasites on predators that are suitable hosts, such as coho salmon (Connors et al., 2010a). This trophic transmission may explain why coho salmon, although older, larger, and therefore less-vulnerable to the effects of sea lice when they enter the marine environment, seem to show reduced survival associated with sea louse epizootics (Figure 19.3b; Connors et al., 2010b).

19.4 Evolution of virulence and drug resistance

Host–parasite dynamics in wildlife are complicated by the ecological effects that parasites can have, but parasites of domesticated animals are subject to very different conditions. Domesticated hosts are often held in relatively high densities, and normally do not need to evade predators or compete for food to the same extent as their wild counterparts. They are often treated with drugs to reduce or eliminate parasite burdens (Van Boeckel et al., 2015). These conditions change the potential impact of parasites on hosts, but also affect the parasite traits – particularly virulence and drug resistance – selected for in agriculture and aquaculture (Mennerat et al., 2010).

19.4.1 Virulence

When hosts are abundant and at high density, parasites may invest in reproduction rather than survival, heavily exploiting individual hosts with little consequence for parasite fitness since the chance of infecting other hosts is high (Mennerat et al., 2010). Infestation with multiple species or strains of parasite can also lead to competition among parasites and select for virulent parasite strains (May & Nowak, 1995; van Baalen & Sabelis, 1995). Further, low genetic diversity typical of domesticated hosts may facilitate the spread and rapid evolution of parasites (Altizer et al., 2003). As described earlier in this chapter, farming practices serve to free parasites from natural regulation, a feature of natural systems that would normally reduce virulence (Lenski & May, 1994). Finally, a number of disease-management practices employed in aquaculture may themselves select for increased virulence (Kennedy et al., 2016).

Evidence from salmon farming indicates that predictions of increased virulence have been borne out, at least in part. For example, salmon aquaculture has been implicated in selecting for more virulent *Flavobacterium columnare* bacteria (Pulkkinen et al., 2010; Sundberg et al., 2016). Also, sea lice associated with salmon farms in Norway produce eggs faster than wild-type sea lice (Mennerat et al., 2010). Little work has been carried out in general on virulence evolution in macroparasites, however, and salmon farming may present a good test case. Unfortunately, the potential for pathogen transmission to wild fish species carries grave implications for populations unequipped to deal with pathogens that evolved in a domestic environment (Daszak et al., 2000).

19.4.2 Drug resistance

Salmon farmers have relied heavily on chemical treatments to manage sea lice at substantial annual cost (Costello, 2009), for example up to 23% of total production costs in Norway (Nilsen et al., 2017). A number of chemotherapeutants are used, the most common of which is emamectin benzoate (EB, trade name SLICE; Burridge et al., 2010). In most salmon farming regions of the world, sea lice have evolved resistance to EB due to strong selection pressure for mutations that confer resistance to chemotherapeutants and the relatively short generation time of sea lice that allows these mutations to spread quickly in the parasite population (Aaen et al., 2015). However, resistance has failed to emerge in the Pacific despite a similar duration of chemotherapeutant use (Saksida et al., 2010). Recent theory suggests this is because the large abundance of wild Pacific salmon provides a refuge from selection for drug resistance (Kreitzman et al., 2018).

Pest resistance to chemical treatment is a problem in agriculture generally, and avoiding its evolution has been the subject of much study. Theory suggests that the maintenance of an untreated 'refuge' pest population,

connected to a treated population via migration, can serve as a source of wild-type susceptible genes and avoid or delay treatment resistance (Comins, 1977). With aggressive treatment to ensure remaining resistance alleles are recessive, wild-type resistant hybrids that result from mating with immigrants are removed (the 'high-dose/refuge' strategy; Gould, 1998; Tabashnik et al., 2013).

Several models have explored how sea lice exchange between wild and farmed salmon may affect EB resistance (Ashander, 2010; Murray, 2011; McEwan et al., 2015; Kreitzman et al., 2018). Consistent with agricultural theory, these models indicate that a large untreated refuge population, connectivity between wild and domesticated hosts, and aggressive treatment oppose the evolution of resistance. A small refuge (e.g. North Atlantic salmon farming regions) appears to produce resistance in sea lice, whereas a large wild-host refuge (e.g. British Columbia) can preclude resistance (McEwan et al., 2015). Wild- and farmed-host populations of sea lice in British Columbia appear to be genetically connected (Messmer et al., 2011), and wild salmon spawning in the vicinity of salmon farms likely serve as an important link between these populations (Ashander, 2010). Thus, wild salmon may provide an ecosystem service by helping to maintain treatment susceptibility in sea lice on salmon farms (Ashander, 2010; Kreitzman et al., 2018).

19.5 Implications for conservation and management

The potential impact of parasites on wildlife species may be most pronounced in systems where wildlife are sympatric with domestic reservoir hosts (Tompkins et al., 2015). Evolution in domesticated species and the indirect ecological effects of parasites on host predation and competition may be the primary mode by which parasites affect wildlife populations in these situations (Hatcher et al., 2006; Krkošek et al., 2011a; Godwin et al., 2015). Further, indirect effects of parasites in multi-host communities may result in unexpected outcomes that are not obvious from pairwise interactions (Connors et al., 2012; Peacock et al., 2014).

Advice for conservation management can arise from host ecology such as migration timing (Krkošek et al., 2007b). Delousing can be effective for conserving wild salmon when treatment is coordinated to reduce parasite numbers during the migration of wild juvenile salmon (Peacock et al., 2013; Bateman et al., 2016). Not only has this strategy proven effective, it does not necessarily require additional treatment during a production cycle (Peacock et al., 2013), and so it does not lead to additional financial costs or elevated selection for drug resistance. However, environmental variability, such as anomalous ocean temperatures, can alter both the timing of wild salmon migrations and parasite development, leading to failure to control parasites (Bateman et al., 2016). These lessons may be increasingly applicable in other

systems as environmental change results in species range shifts, changes in parasite and host phenology (Kutz et al., 2014), and emerging infectious disease (Jones et al., 2008; Tompkins et al., 2015).

The timing of delousing treatments not only affects transmission of sea lice to juvenile wild salmon, but also transmission among farms. Coordinated area management involves the synchronised timing of treatments among farms at a regional scale, which may require cooperation of multiple aquaculture companies. Coordinated area management reduces reinfection from nearby farms (Murray & Salama, 2016; Peacock et al., 2016) and may also slow the evolution of drug resistance by reducing the chance that surviving lice can find mates (Krkošek et al., 2012; Groner et al., 2014). Other measures to minimise transmission among farms include stocking single age classes and fallowing to avoid perpetuating infection (Costello, 2004).

Global trends in salmon production from wild and farmed fish populations indicate fisheries stagnation and rapid expansion of domesticated fish will likely intensify sea louse spill-over and spill-back dynamics (Krkošek, 2016). Host density thresholds that arise due to density-dependent transmission occur at regional scales (Frazer et al., 2012; Jansen et al., 2012), and maintaining regional salmon aquaculture production below such thresholds may provide a means for avoiding costs of sea louse outbreaks. Alternatively, closed containment production of farmed fish is one way to eliminate the spill-over and spill-back dynamics of sea lice between wild and farmed salmon. The economic viability of such production systems is not clear (Liu et al., 2016), but trends are towards affordability as losses due to sea louse infestation of both wild and farmed fish continue to escalate and consideration of the external costs of open-net pen aquaculture are more fully deliberated.

Bioeconomic models that include the costs (e.g. parasite transmission) and benefits (reduced commercial harvest) of aquaculture for wild fish populations suggest stable coexistence of productive wild fisheries and aquaculture only if negative ecological feedbacks of domesticated animals on wildlife are kept below a threshold (Orobko, 2016). The definition of the threshold itself must include the reciprocal economic interactions between domesticated animals and wildlife, as well as the ecological effects of parasites on wildlife. The case study of sea louse parasites on farmed and wild salmon illustrates that the sustainability of both domesticated animals and wildlife depends on the broader anthropogenic and biological context of host–parasite interactions.

19.6 Conclusions

The ecological processes by which parasites affect domesticated versus wild animals differ, and the ecological context of host–parasite interactions can yield unexpected outcomes for wildlife health. For example, parasites can mediate food web dynamics of hosts, resulting in host resilience to epidemics

Table 19.2 *Conclusions (references: [1]Schumaker 2013; [2]Viana et al., 2015; [3]Pruvot et al., 2014, 2016; [4]Tian et al., 2015; [5]Dhondt et al., 2013; [6]Lafferty & Ben-Horin, 2013).*

Pathogen	Location(s)	Wild host	Domestic host	Spill-over/spill-back	Ecological effects	Ref
Brucellosis (*Brucella abortus*)	Greater Yellowstone Area, USA	Bison (*Bison bison*), elk (*Cervus elaphus*)	Cattle (*Bos taurus*)	Spill-over/-back has hindered the eradication of bovine brucellosis	*Migration* – Food supplementation in the park aims to reduce natural winter migration of bison and elk to outside areas *Competition* – Competition among ungulate species reduces habitat overlap and transmission	1
Canine distemper virus (morbillivirus)	USA, Africa	Wild canines, felids (e.g. lions (*Panthera leo*))	Dogs (*Canis lupus familiaris*)	Transmission among multiple domestic and wild species yields complex host community	*Evolution* – Host switching facilitates spread to new species	2
Giant liver fluke (*Fascioloides magna*)	Alberta, Canada	Elk (*Cervus elaphus*)	Cattle (*Bos taurus*)	Cattle are dead-end hosts, reducing prevalence in sympatric elk through a 'dilution' effect	*Migration* – Evidence of migratory escape by elk; non-migratory herds are significantly more likely to be infected	3
Avian influenza virus	Asia	Wild waterbirds	Poultry	Domestic waterbirds often grazed on rice paddy fields in contact with wild waterbirds	*Migration* – Migratory birds may be responsible for the spread of H5N1 in Asia *Evolution* – Mutation into highly pathogenic H5N1 strain	4
Mycoplasma gallisepticum	North America	House finch (*Haemorhous mexicanus*)	Poultry (Galliformes)	Spill-over to finches followed by spread to other Passerines, resulting in multi-host community	*Migration* – Spread from eastern to western North America via wild bird dispersal *Evolution* – increasing virulence in eastern North America, substantial spatial and temporal genetic variation in bacterium	5
Withering-Syndrome Rickettsia-Like Organism (WS-RLO)	California, USA	Black abalone (*Haliotis cracherodii*)	Red abalone (*Haliotis rufescens*)	Domestic abalone more resistant to the bacterium, acting as reservoir hosts		6

or high parasite-induced host mortality at low measured parasite burden. Untangling these indirect effects of parasites in ecosystems is critical to the conservation and sustainable management of wildlife, where parasites are a growing threat.

The spill-over and spill-back of parasites and pathogens between wild and domestic hosts is not limited to salmon aquaculture, nor is it a new phenomenon. There are other examples of pathogen transmission between domesticated hosts and wildlife that touch on the themes discussed in this chapter (Table 19.2). Studying these systems requires integrating information on both direct and indirect effects from multiple sources. Controlled laboratory experiments are well suited to determining the direct physiological effects of parasitism on individuals in an isolated and stable environment. Field-based behavioural studies can look at how parasites mediate isolated interactions such as predation or competition. Together, these sources can identify the range of possible effects, and inform broader analysis, but the specifics of any interaction can be heavily influenced by the black box of ecology. Which pathway is most important? As the case study of salmon and sea lice has exemplified, it depends: the highly contingent nature of the interactions means that it's important to assess net effects through large-scale manipulative experiments, if possible, or data on long-term population dynamics and health (e.g. parasite loads) in correlative analysis. Long-term ecological data are critical for untangling how and why parasites are maintained in cases where multiple domesticated and wild host species interact, such as with canine distemper in Tanzania's Serengeti ecosystem (Viana et al., 2015).

As the global human population grows, and with it the demand for protein, disease transmission between domesticated animals and wildlife is likely to increase. Emerging infectious diseases are limiting production in agriculture and aquaculture, threatening pollinator communities and the crops that depend on them, and bringing into question the sustainability of wildlife populations (Table 19.2; Tompkins et al., 2015). Finding solutions that minimise disease transmission and allow for sustainable coexistence of wildlife and domesticated animals is necessary both for the conservation of imperilled wildlife (such as Pacific salmon) and for feeding a growing human population. The theory and models inspired by the case of sea louse parasites on farmed and wild salmon may help understand and predict threats in other systems.

19.7 Acknowledgements

Much of the research described in this chapter was made possible by the hard work and dedication of those at the Salmon Coast Field Station, most notably Alexandra Morton, Scott Rogers, Coady Webb, Zephyr Polk, and Lauren Portner, and by the support of the Echo Bay community including Eric Nelson, Billy Proctor, Chris and Hannah Bennett, Yvonne and Al Maximchuck, and Pierre

Landry. We acknowledge research contributions from Larry Dill (Simon Fraser University), John Reynolds (Simon Fraser University), John Volpe (University of Victoria), Craig Orr (Watershed Watch Salmon Society), the Hakai Institute, and Fisheries and Oceans Canada. Funding was provided by the Natural Sciences and Engineering Research Council of Canada (NSERC), MITACS, ESSA Technologies Ltd, and the Killam Foundation.

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