7 Unveiling the Hidden Players: Exploring the Intricate Dance of Aquatic Parasites, Host Biodiversity and Ecosystem Health

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Abstract

This chapter delves into the multifaceted roles of aquatic parasites within natural ecosystems. It highlights both the negative and positive impacts these parasites can have on individual hosts, host populations, biodiversity and overall ecosystem health. The discussion covers how parasites influence various levels of biological organisation and ecosystem functions. It also explores how healthy ecosystems are defined and maintained, emphasising the roles of vigour, organisation and resilience. The complex interactions between parasites and their hosts are illustrated through numerous examples, spanning cases of behavioural modification, host–parasite coevolution, and broader ecological consequences stemming from those interactions. Understanding the interplay between parasites, hosts and ecosystems is presented as crucial for a comprehensive view of ecosystem dynamics.

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7.1 Introduction

The impact of aquatic parasites on their hosts has been relatively well studied, especially for parasites of veterinary and medical importance (see Chap. 19) and for pathogens whose impact on aquaculture threatens food security (see Chap. 23). Within natural and modified aquatic ecosystems, parasites impact all levels of biological organisation (see Chap. 20). Although many of these effects are considered negative, research over the past three decades has also shed light on the positive impacts of aquatic parasites on individual host health and, ultimately, on the functioning and maintenance of ecosystems.

N. J. Smit, B. Sures (eds.), *Aquatic Parasitology: Ecological and Environmental Concepts and Implications of Marine and Freshwater Parasites*, https://doi.org/10.1007/978-3-031-83903-0_7

The aim of this chapter is to introduce the reader to the various ecological roles and impacts (positive and negative) of aquatic parasites in natural ecosystems. We focus on the effects of parasites on individual hosts, host populations, biodiversity and ecosystem health in general (see Chap. 8 for a detailed discussion on the impact of aquatic parasites on host community structures). To demonstrate the importance of aquatic parasites for ecological processes, we will first introduce the concept of a healthy ecosystem followed by how aquatic parasites both drive and bear the brunt of the state of ecosystems.

7.2 What Is a Healthy Ecosystem?

All systems, whether simple or complex, have a finite lifespan. They evolve as they age and as their smaller components are replaced (Costanza and Patten 1995; Costanza and Mageau 1999). A healthy system is thus a system *predicted* to be on track to achieving a full natural lifespan, with the outcome of that prediction being visible only retrospectively (Costanza and Patten 1995). Therefore, any process that prematurely reduces the predicted lifespan of a system beyond its normal evolution due to ageing can be considered detrimental to the health of that system (Costanza and Mageau 1999).

Historically, the health of an ecosystem was implicitly understood in light of the values promoted in human health, in a human-centred attempt to manage the environment (Science Advisory Board 1990). Since then, the definition of "ecosystem health" has in turn included notions of balance, complexity, stability and growth potential. Nowadays, a healthy ecosystem is defined as 'stable and sustainable' (Costanza et al. 1992), meaning it shows 'the ability to maintain its structure (organisation) and function (vigour) over time in the face of external stressors (resilience)' (Costanza and Mageau 1999) throughout its full predicted natural lifespan (Costanza and Patten 1995). These three attributes of ecosystems (i.e. organisation, vigour and resilience) have been characterised by various authors. The organisation of an ecosystem is a qualitative and quantitative measure of the interactions between species and with the surrounding habitat (Costanza and Mageau 1999). An ecosystem's organisation depends on its richness and diversity, the level of ecological specialisation of each of its species and the number of unique interactions between the ecosystem's components. Network analyses have been used to quantify this factor (see Leontief 1941; Ulanowicz 1986). The vigour of an ecosystem is defined by Costanza and Mageau (1999) as 'a measure of its activity, metabolism or primary productivity' that is reflected in a variety of quantifiable factors, for example, gross primary production (see also Odum 1971). Finally, an ecosystem's resilience is defined as its ability to keep its organisation intact when exposed to perturbations of biotic or abiotic origin (Costanza and Mageau 1999).

In twentieth-century ecology, these characteristics were mostly envisioned in relation to predator–prey relationships or interspecific competition. Parasites, whose biomass was deemed insignificant, were long excluded despite their extraordinary success (Horwitz and Wilcox 2005; Hudson et al. 2006b). Research has since demonstrated parasites can drive, as well as be a consequence of, the state of ecosystems (reviewed by Selbach et al. 2022) and contribute more to the biodiversity of an ecosystem than free-living organisms (see Fig. 7.1).

7.3 A Parasite's Effect Is Context Dependent

Contrary to popular belief, parasites are not always detrimental to their hosts (see Selbach et al. 2022; Chaps. 13 and 20) and their effects on any ecosystem vary over time. Their influence on both hosts and communities, positive or negative, depends on a wide range of environmental factors.

Parasitism usually incurs a cost to the hosts in terms of growth, survival and/or reproduction. The scale of damage from parasitic infections depends on factors related to the host–parasite relationship such as the life history of the para-

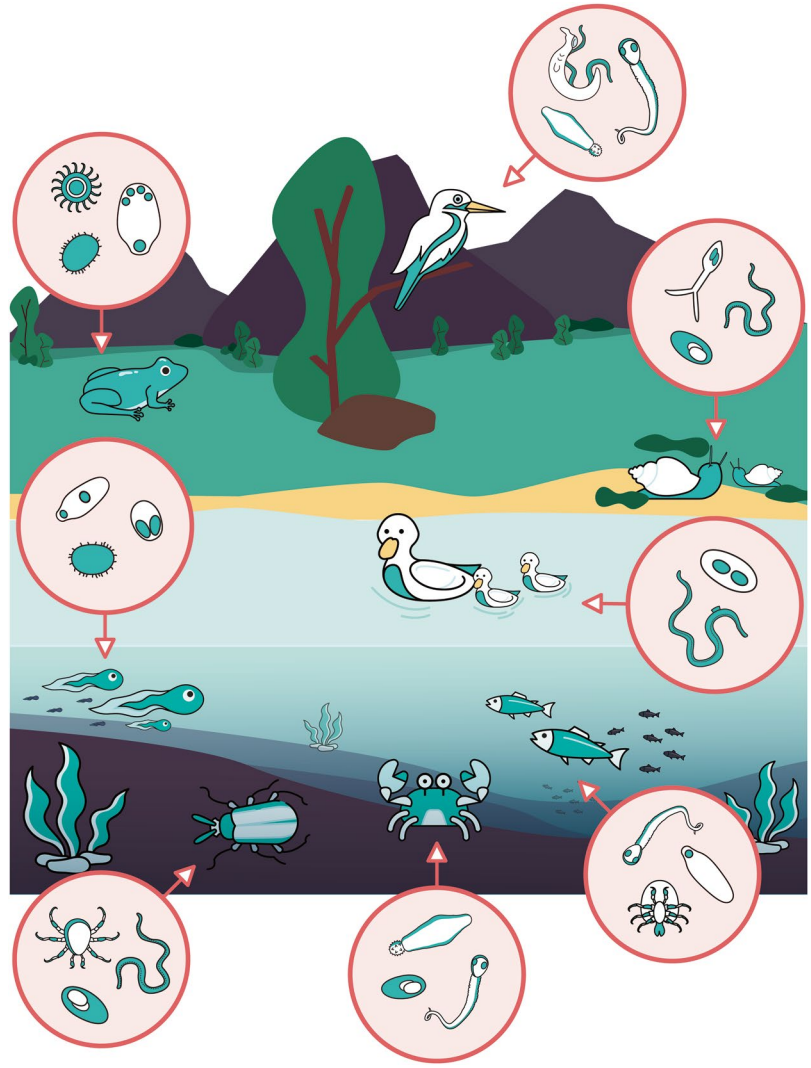

Fig. 7.1 An illustration of the diversity within an aquatic ecosystem highlighting the presence of parasites. All animal species living in this ecosystem serve as potential hosts for one or more parasite species at various developmental stages

site, site of infection, parasite load or efficiency of the host's immune response and on factors related to the ecosystem such as host population density, resource availability or overall parasite presence in the community (see Sects. 7.4 and 7.5). The interaction between these intrinsic and extrinsic factors determines the outcome of infections for the host, which can range from weight loss to heightened sensitivity to opportunistic diseases, reduced lifespan or parasitic castration (see Sect. 7.4.1).

In contrast, some studies suggest that phenotypic alterations provoked by parasite presence are not detrimental to hosts under specific environmental conditions. In these cases, the damage incurred by parasite presence is offset by the benefits of being infected, leading to increased net fitness and/or survival for the host and ensuring its reproductive success (see Chap. 21). For example, Richardson's ground squirrels (*Urocitellus richardsonii*) infected with *Trypanosoma otospermophili* show significantly higher mass gain when allowed to feed *ad libitum* under a vitamin B6-deficient diet compared to uninfected controls in laboratory conditions (Munger and Holmes 1988) because trypanosomes produce that vitamin for the host (Stoffel et al. 2006). Although trypanosomes are preva-

lent in aquatic habitats, no similar cases of function rescue are known from trypanosomes of aquatic hosts. The nature and extent of the effects a parasite exercises on its host also heavily depend on the characteristics of the surrounding biotope. Resource availability has been proposed to play a key role in how hosts manage their parasite loads. For instance, individuals of the limpet *Fissurella crassa* infected by metacercariae of *Proctoeces humboldti* in rich upwelling areas of the Chilean coast display a significantly higher gonadosomatic index (excluding parasite biomass in the gonads) than both infected and uninfected hosts in nutrient-poor areas. Moreover, infected hosts in upwelling areas consume significantly less oxygen than their uninfected counterparts, whereas those consumption rates are equivalent for both parasitised and unparasitised hosts in oligotrophic waters (high oxygen consumption indicates physiological stress) (Aldana et al. 2020). This example shows that access to resources (in this case: nutrient availability) mediates the physiological response of a host individual to parasite infection.

Historically, parasitism was described as a form of symbiosis (i.e. a long-term, intimate ecological relationship between individuals of distinct species; Combes 2001; Rózsa and Garay 2023) wherein one of the species involved incurs some form of fitness cost. However, the recognition of the existence of "conditionally helpful" parasites through the examples above has blurred the lines between mutualistic and parasitic relationships. This recognition forced changes to the definition of parasitism (e.g. Fellous and Salvaudon 2009; Parmentier and Michel 2013; Weinersmith and Earley 2016). If the host–parasite relationship between populations of two species is long-lasting enough, that relationship can evolve through evolutionary arms race and population dynamics to a more balanced interaction and, sometimes, a mutualistic one, thus reducing the cost of virulence for the parasite and that of resistance for the host (Antia et al. 1994; Combes 1997).

The benefits of parasitism can also extend to host populations. In ecosystems featuring species consuming potentially parasitised prey, selective feeding based on the probability of infection of a particular cohort of prey has been observed as a means to avoid parasites. Such behavioural adaptation tends to spare the whole prey cohort, including both parasitised and healthy animals. For example, in the UK, depending on the season, oystercatchers (*Haematopus ostralegus*) feeding on cockles infected by metacercariae of bird trematodes tend to maximise energy intake while minimising parasite loads by preying preferentially on the middle-sized cockle cohort, individuals of which are more nutritious than small cockles and statistically less infected than large (i.e. old) ones (Norris 1999). Consequently, both healthy and parasitised individuals from the two remaining cockle size cohorts may benefit from decreased predation rates and enhanced chances of reproduction (Thomas et al. 2000). In addition, the infection of cockles with echinostome trematodes can change the functional role of this dominant benthic organism, which in turn influences the surrounding benthic community (see Chap. 8 for a detailed discussion on a case study from the sand flats of Otago Harbour, New Zealand).

7.4 Parasites as Drivers of Ecosystem Processes

All ecosystem resources are finite. For example, *access* to sunlight is limited even if *sunlight* is not (Darlington Jr 1972). Thus, for a community of several species occupying distinct ecological niches and sharing common resources (e.g. space) to remain in equilibrium, each of these species must be limited in its growth by at least one biotic or abiotic factor to avoid complete resource depletion and subsequent biodiversity loss (MacArthur 1958; Levin 1970). Consequently, the population dynamics of each species in any ecosystem both affect and are affected by the dynamics of all other species within that ecosystem through mechanisms of direct or indirect species interactions: the presence of each species generates limiting factors

affecting all the others (Paine 1966; Levin 1970). Parasites, like predators, prey or resources, provide limiting factors regulating species within ecosystems (Darlington Jr 1972). Limitation of species' fitness and population densities by parasitism operates either directly on their hosts or indirectly on non-host species, via density-mediated indirect effects (DMIE) and trait-mediated indirect effects (TMIE) (Figs. 7.2, 7.3) (see also Chaps. 8 and 18 for examples of DMIEs and TMIEs on host community structures).

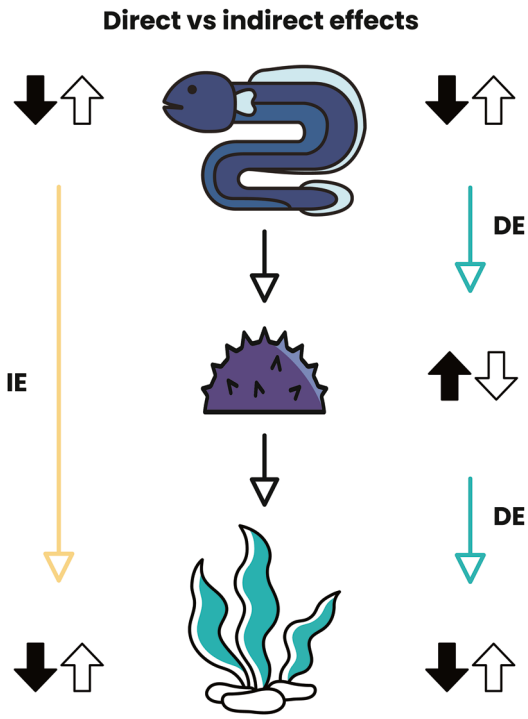

Fig. 7.2 The difference between direct and indirect effects. Wolf eels (top) feed on urchins (middle), which feed on macroalgae (bottom). Wolf eel populations thus have a direct effect (DE) on urchin populations, and urchins on algae populations. When wolf eel populations increase, urchin populations decrease, consequently favouring algal growth (white arrows). The opposite effect is observed in the case of a wolf eel population reduction (black arrows). Thus, wolf eel populations have an indirect effect (IE) on algae populations, mediated by urchins

7.4.1 Direct Effects on Hosts and Parasites

7.4.1.1 Parasitic Castration

An extreme example of parasitism-associated cost directly affecting fitness is parasitic castration (Fig. 7.3), a phenomenon recorded in a wide range of parasitic groups (reviewed by Lafferty and Kuris 2009b). Examples include digenean asexual stages castrating their first-intermediate mollusc (e.g. Sousa 1983) and polychaete hosts (Køie 1982; Cribb et al. 2011) (see Chap. 5); rhizocephalan cirripeds (e.g. species of *Sacculina*) castrating various decapod crustacean genera (e.g. Toyota et al. 2023; Chap. 6); some diphyllobothrium cestodes (e.g. *Schistocephalus solidus*) castrating fishes via nutrient deprivation (Heins 2017; Chap. 5); epicarid (e.g. *Hemioniscus balani*) and cymothoid isopods (e.g. *Riggia paranensis*) castrating cirripeds (Blower and Roughgarden 1988) and fishes (Azevedo et al. 2006), respectively (see Chap. 6); and pearlfishes of the genus *Encheliophis* (Ophidiiformes: Carapidae) permanently castrating their holothuroid hosts (Parmentier and Vandewalle 2005). At the individual level, the infected hosts usually become permanently unable to transmit their genetic material, although some host species have been shown to enhance their reproductive output before complete castration is attained (Minchella et al. 1985; Sorensen and Minchella 2001) or regain the ability to reproduce if they succeed in killing the parasite (Kuris et al. 1980). Following castration, the only genome from the host–parasite entity participating in natural selection (i.e. transmitted to the next generations) will be that of the parasite; from the point of view of natural selection, the castrated host is no more than a 'shell' (O'Brien and Van Wyk 1985).

At the population level, parasitic castration has been shown to significantly reduce host density, effectively removing part of the gene pool. However, the effects of castrators on host populations depend on the nature of the castrators themselves. In the case of trematodes, the main

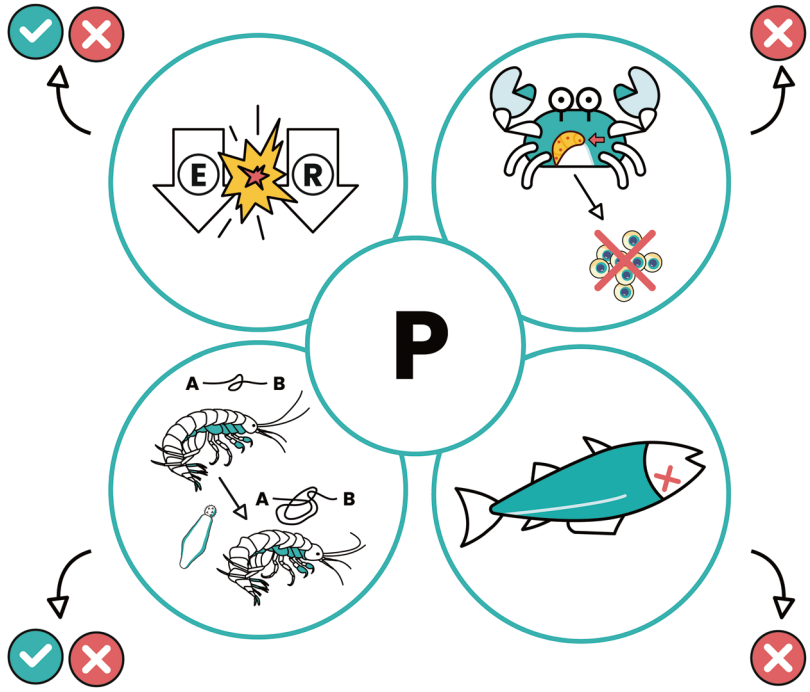

Fig. 7.3 The four types of parasite-induced direct effects on hosts. (Top left) Direct parasite competition (spark) or synergy directly affects hosts; for example, the competition between digeneans *Ribeiroia ondatrae* ("R") and *Echinostoma trivolvis* ("E") decreases both their populations. (Top right) Parasitic castration, here of a crab by a rhizocephalan parasite (yellow crescent). (Bottom left) Parasite (acanthocephalan)-induced direct modification of a host's (amphipod) physiological, physical, or behavioural traits, here deformed exoskeleton and increased erratic activity in the movement from A to B. (Bottom right) Host morbidity or mortality. These four types of direct effects can have positive (green tick) and/or negative (red cross) outcomes for host fitness

castrators of molluscs, the effects of castration on intermediate-host population densities are mitigated by the complexity of trematode life cycles, the rates of parasite recruitment from definitive hosts (which depends on definitive-host mobility) and environmental conditions, so that rate of parasitic castration and intermediate host population density cannot be inferred from each other (Lafferty 1993). Host population densities might be more directly affected when confronted with castrators having direct life cycles. Castration impacts host populations on two levels. First, as unparasitised individuals compete with castrated hosts for resources and the energy consumed by castrated hosts is at least partly confiscated by parasites, competition for resources instead takes place between healthy snails and the parasites themselves, significantly affecting host populations (Lafferty 1993). Second, castration progressively reduces gamete production. This effect has been recorded in several host–parasite combinations, e.g. polpulations of the California horn snail *Cerithideopsis californica* parasitised by various trematode species (Lafferty 1993). The effect of castration on host populations is generally believed to be enhanced in the case of non-random selection of host individuals by parasites and depends on factors such as host size structure and sex ratios. For example, the isopod *Hemioniscus balani* preferentially targets the oldest and most fertile individuals of the barnacle *Chthamalus fissus* because more energy is allocated by older hosts to the reproduction effort, meaning greater gains for the castrator but significant reduction in barnacle reproductive output at the population level (Blower and Roughgarden 1988).

7.4.1.2 Host Morbidity and Mortality

Whether a host will die from being parasitised, or not, is hard to predict. Generally, parasitised individuals are significantly more likely to die prematurely than uninfected ones (Robar et al. 2010). Parasites can kill their hosts directly (e.g. through excessive infection burden) (Fig. 7.3) or indirectly (e.g. by making hosts more likely to be predated upon). Parasitic infections can also decrease host resistance to environmental stressors: in the study of Lafferty (1993), trematode-infected snails of *C. californica* subjected to an unidentified environmental stressor died significantly more than did unparasitised snails exposed to the same stressor. On the contrary, parasitism often does not affect host mortality rates (e.g. Friesen et al. 2017) and can even benefit the host. For example, in the laboratory, freshwater clams *Pisidium amnicum* parasitised by the trematode *Bunodera luciopercae* survive significantly longer to lethal pentachlorophenol (PCP) concentrations than unparasitised ones do, possibly because trematodes may sequester PCP in their fatty tissues to the benefit of their host (Heinonen et al. 2001).

A meta-analysis by Robar et al. (2010) showed that globally, infected-host mortality varies widely according to many factors, the most influential of which are parasite life-cycle characteristics (i.e. direct or non-predation-mediated trophic transmission vs. predation-mediated trophic transmission regardless of parasite lineage), host lineage and latitude.

Intermediate hosts of trophically transmitted parasites requiring predation at some stage of their life history (i.e. most acanthocephalan, trematode, nematode and cestode species) are 2.4 times more likely to die compared to hosts harbouring parasites not depending on predation-mediated transmission (Robar et al. 2010). In addition to the increased predation likelihood induced by physical damage to the hosts (e.g. Jonsson and Andé 1992), a high diversity of adaptive strategies of host morphological, sensorial and behavioural manipulation by parasites has been evolutionarily selected to ensure predation on infected hosts and successful trophic transmission (Fuller et al. 2003; Seppälä et al. 2004; Poulin et al. 2005) (see Sect. 7.4.2). Of the three parasite categories considered by Robar et al. (2010) (i.e. helminths, arthropods and microparasites), helminths were shown to have the strongest influence on host survival and microparasites the weakest, independently of transmission mode.

Globally and after correction for other significant factors, infected molluscs seem at increased risk of mortality directly or indirectly caused by parasites compared to other invertebrate groups (e.g. arthropods) as well as some vertebrate groups. This difference is explained in part by the commonly deleterious effect of digenean intermediate stages on mollusc first-intermediate hosts, as well as phenotypic modifications by parasites that render the host more vulnerable to predation (Robar et al. 2010) (see *Parasite-induced direct trait modification* below). An illustration of digenean-induced morbidity is given by Jonsson and Andé (1992), wherein a *Cerastoderma edule* cockle population parasitised by an unknown digenean in Sweden underwent a mass mortality event putatively caused by extensive tissue damage (also see Chap. 8). However, not all mollusc species display the same sensitivity to their digenean parasites, and not all digeneans have the same effects on their hosts. For example, Marchand et al. (2020) recorded a higher mortality (putatively caused by the parasite) in parasitised snails of *Ladislavella elodes* than in non-parasitised individuals, but equal rates of mortality in parasitised and non-parasitised snails of *Planorbella trivolvis*, when these two species were infected by *Echinostoma* spp. (Digenea: Echinostomatidae).

Infected fishes and amphibians are also more likely to die (directly or indirectly from their parasites) than parasitised invertebrates, birds and mammals. This discrepancy in mortality between vertebrate lineages might be linked, in part, to differences in host immune response to the presence of non-self across these lineages (Robar et al. 2010).

As vertebrates tend to feature sophisticated immune systems relying on highly efficient feedback loops, excessive triggering of their immune defences can induce deleterious immune cas-

cades, ultimately resulting in organ damage and compromised host survival. Examples include chronic granulomatous inflammations in fishes following infection by metazoan endoparasites (Feist and Longshaw 2008) and cytokine storms in other vertebrate lineages (e.g. Tong et al. 2021). These processes may kill a host even if its parasite is not lethal on its own. Thus, a parasite tends to be the main cause of death only when the vertebrate host's immune response to that parasite is weak (Casadevall and Pirofski 1999).

Lastly, host mortality tends to vary on a latitudinal gradient. The meta-analysis of Robar et al. (2010) suggests that overall, and when accounting for all intrinsic host and parasite characteristics, host mortality tends to be significantly higher in equatorial areas compared to subpolar regions. Moreover, the influence of temperature on host survival is understood to add up to the effects of host taxon and predation-mediated transmission. In all ecosystems, parasites tend to be highly sensitive to abiotic factors, which partly depend on latitude (e.g. temperature, moisture and rainfall) (e.g. Thieltges and Rick 2006). Significant variations of these factors could be associated with parasite outbreaks, although the directions of trends appear different in aquatic and terrestrial systems (Harvell et al. 2002; Hudson et al. 2006a; Torchin et al. 2015). In migrating smolts of Atlantic salmon (*Salmo salar*), for example, sea temperature increase is predicted to accelerate the development of parasitic copepods *Lepeophtheirus salmonis* and *Caligus* spp., leading to higher levels of infestation and increased mortality (Vollset 2019). Outcomes of parasitism vary widely between host–parasite systems, rendering further generalisation difficult.

7.4.1.3 Parasite-Induced Direct Trait Modification

Many parasites can directly affect their host's physiological, physical or behavioural traits (Fig. 7.3). A "trait" is understood here as a specific phenotype, for example, fecundity level, body shape or size or swimming behaviour. In freshwater, an interesting example of parasite-induced trait modification is seen in the infection of the three-spined stickleback, *Gasterosteus aculeatus* by plerocercoid larvae of *Schistocephalus solidus*. Laboratory experiments demonstrated that fish infected by the plerocercoids spent significantly more time foraging near the surface than their uninfected counterparts (Quinn et al. 2012; Talarico et al. 2017), possibly because of an increased need for oxygen (Lester 1971). These trait modifications (i.e. higher oxygen consumption and higher occupation rates of surface layers) could potentially increase predation risk from piscivorous birds, helping the parasite complete its life cycle (Quinn et al. 2012). Procercoids of the same parasite also affect the behaviour of their first intermediate host, the copepod *Macrocyclops albidus*. Transmission of procercoid larvae from first to second (stickleback) intermediate hosts happening between 11 and 31 days post-copepod infection ensures the parasites have attained the critical mass necessary for optimal fitness inside the bird definitive host (Hammerschmidt et al. 2009). Procercoids were experimentally shown to manipulate their copepod hosts in order to minimise predation risk before Day 11 post-infection (by keeping the hosts less active and immobile for longer after a simulated "predator attack"), and maximise predation risk after Day 17 post-infection (by reducing immobility time after attacks and enhancing activity) (Hammerschmidt et al. 2009). *Schistocephalus solidus* is thus able to directly alter the behavioural traits of both of its intermediate hosts.

Significant differences seem to exist in the nature and degree of parasite-induced behavioural alterations across host types (i.e. vertebrate or invertebrate), parasite lineages and transmission strategies (i.e. trophic vs. non-trophic) (Lafferty and Shaw 2013). For instance, trematode and nematode parasites might induce analgesia in terrestrial vertebrates (Kavaliers et al. 1984; Pryor et al. 1998). In contrast, the trematode *Microphallus papillorobustus* supposedly alters serotonergic pathways and neuron morphology in the brain and optic neuropils of the amphipod *Gammarus insensibilis* (Helluy and Thomas 2003), provoking surface-seeking behaviours (Helluy 1983). As illustrated in the

latter case, the infection site seems to play a critical role. Parasites invading a host's brain and nervous system (Klein 2003) or organs involved in hormone production, modulation of neuronal activity and immunity seem to have considerable effects on behaviour [see reviews by Beckage (1993), Adamo (2002), Thomas et al. (2005) and Helluy (2013)]. An instance of behavioural modification through alteration of a vertebrate's central nervous system is that of fathead minnows (*Pimephales promelas*) by the brain fluke *Posthodiplostomum ptychocheilus*. When cercariae of that species encyst at low intensities on the surface of the optic lobe of their hosts' brains, the infected fish follow moving objects significantly less and take significantly more time to respond to changes in the direction of those objects (Shirakashi and Goater 2001). Unlike many other cases [see Poulin (1994a) for review], such changes likely result from the impairment of visual organs rather than from any parasite-induced lethargy. Slow response to moving objects almost certainly has significant implications for fish survival, as it directly relates to predator avoidance (Shirakashi and Goater 2001).

The study of Acanthocephala-gammarid amphipod systems has led to particularly interesting observations on the evolution of host-behaviour manipulation strategies in parasite and anti-parasite counter reactions in hosts. Cystacanths of *Pomphorhynchus laevis* induce a strong attraction to light and the water surface in the amphipod *Gammarus pulex* by directly or indirectly manipulating serotonin levels in its brain (Tain et al. 2006, 2007), which supposedly increases the infected individuals' vulnerability to predation. However, infected individuals of the sympatric *Gammarus roeselii* are not significantly more photophilic than their uninfected counterparts and do not show any significant serotonin response to parasite presence (Bauer et al. 2000; Tain et al. 2007). In contrast, both *G. pulex* and *G. roeselii* infected by *Polymorphus minutus* swim significantly more often at the surface and cling significantly more to floating objects than do their uninfected conspecifics; however, in that case again, the magnitude of behaviour alteration is stronger in *G. pulex* than in *G. roeselii* (Bauer et al. 2005). Importantly, *G. roeselii* is an invasive species (Jażdżewski 1980) whereas *G. pulex* is native to the study areas investigated in the aforementioned studies. Thus, the lack of evolved ability of *P. laevis* to alter *G. roeselii*'s behaviour has been interpreted as a sign of maladaptation of *P. laevis* to that host (Bauer et al. 2005; Tain et al. 2007). Conversely, the strong effects of *P. laevis* on *G. pulex* in the above study areas [but not in water bodies where *P. laevis* was recently introduced; see Kennedy et al. (1989) and Kennedy (1996)] hint at the evolution of hyperspecialised manipulative abilities (Bauer et al. 2005; Tain et al. 2007) in a fish parasite with reduced dispersion potential (Kennedy 1996) [but see alternative explanation in Tain et al. (2007) and Poulin et al. (2005)]. Behaviour alteration by *P. minutus* in both native and invasive hosts may come from that parasite's adoption of birds as definitive hosts: high dispersal range of the birds might put the parasite in contact with a broader range of intermediate hosts and favour the evolution of host-manipulation abilities for a broader range of gammarids (Bauer et al. 2005).

The above example illustrates the evolution in host and parasite populations of specific phenotypes called "adaptive traits". These adaptations represent the visible outcome of the natural selection pressure applied by each antagonist against the other (see Chap. 11). An evolutionary adaptation refers to 'a genetically determined feature that has become or is becoming prevalent in a population because it confers a selective advantage to its bearer through an improvement in some function' (Poulin 1995). Importantly, the resulting physical, physiological or behavioural modification cannot be a by-product of the adaptation process, but must result directly from selection pressure to be called 'adaptive' (Ridley 1993; Poulin 1994b). As such, even beneficial changes to host or parasite phenotypes could be side effects of parasite infestation, not adaptations (Poulin 1995). First, changes in a parasite's trait (or, by extension, changes in its host) are more likely to be genuinely adaptive if they evolved independently more than once in distantly related parasite lineages (Poulin 1995) sharing closely related hosts (Cézilly and Perrot-

Minnot 2005). Second, true evolutionary adaptation is more likely if the trait under scrutiny predictably corresponds to the most efficient way of performing a task, for example, infecting the next host in line (for the parasite) or resisting a parasite (for a host) (Poulin 1995). One of the best examples of a host–parasite system fulfilling this requirement is that of the digenean *Dicrocoelium dendriticum* inducing ant intermediate hosts to remain out of the nest at night, climb up to the top of grass blades, clamp their mandibles shut around the stems and tetanise until morning, and to do so repeatedly, for the parasite to increase the likelihood of encounter with the definitive sheep hosts (Hohorst and Graefe 1961; Anokhin 1966; De Bekker et al. 2018). Third, an adaptive modification must confer fitness benefits to the species (Poulin 1995). Fourth, modifications imposed on the host are more likely to be truly adaptive if they target not only one but several host traits at once (Cézilly and Perrot-Minnot 2005). For example, *P. laevis* induces a wide range of behavioural and physical modifications in the intermediate host, *G. pulex*: infected individuals are significantly less fecund (females) (Bollache et al. 2002), significantly more active (Dezfuli et al. 2003), asymmetrical (Alibert et al. 2002) and phototactic (Cézilly et al. 2000). The presence of these four characteristics above in a host–parasite system is usually hard to prove (Poulin 1995).

In parasite populations, adaptive changes can happen to counter hosts' phenotypical or behavioural innovations; they are often expressed through the hosts as extended parasitic phenotypic traits, meaning natural selection pressure on a parasite is expressed in the effects induced on the hosts (Dawkins 1982). In host populations, adaptive changes can happen to counter the loss of fitness induced by parasite infection, or in reaction to parasite threat. Possible examples of behavioural adaptations by the host could be the premature egg production by young individuals of the snail *Biomphalaria glabrata*, the first-intermediate host of the blood fluke *Schistosoma mansoni*, following non-infective exposure to that parasite in the wild in order to compensate for the perceived imminent risk of castration (Minchella and Loverde 1981), or preference for middle-sized cockles by oystercatchers in order to maximise energy intake while minimising infection risk when the birds are not restricted by the nutritiousness of the prey (Norris 1999; see Sect. 7.3).

Adaptive changes in host behaviour in response to parasitism can go as far as influencing the outcomes of sexual selection. In vertebrates, sexual selection is based on behavioural and physical adaptations, sometimes taken to extravagant levels of sophistication. Tentative hypotheses from several angles have been provided to explain female choice (e.g. Ryan 1990; Nowicki and Searcy 2004). One of the most interesting hypotheses regarding reasons for male display is based on host–parasite coevolution and sexual selection of genetically resistant hosts: in natural ecosystems where parasites are assumed to be ubiquitous, males displaying the most attractive physical characters are energetically able to do so because their parasite burden is minimal, implying that they possess alleles conferring resistance to long-term parasite infections, acquired as part of an evolutionary arms race (Hamilton and Zuk 1982). Parasite-induced mate selection may thus, in some species, drive evolution within species and host populations, although mate selection has been proven to operate independently from parasite presence in some cases (Aguilar et al. 2008).

7.4.1.4 Direct Parasite Competition or Synergy Through Co-Infection

Just like many predator species can hunt the same prey, many parasite species or strains frequently share the same host individual (Pedersen and Fenton 2007). For hosts, the impacts of direct interactions between parasites depend on parasite life history, strain, virulence and population density, as well as on individual host immunity (Woolhouse et al. 2015). For parasites, sharing a host can result in benefits for one or more of the species involved or, on the contrary, in active hindrance of the establishment of a species by another via various forms of direct competition [see Mideo (2009) for details] (Fig. 7.3). An example of interaction with benefits for patho-

gens is found in aquaculture. The open wounds inflicted on the rainbow trout *Oncorhynchus mykiss* by the fish louse *Argulus coregoni* significantly facilitate colonisation by *Flavobacterium columnare*, leading to increased fish mortality (Bandilla et al. 2006).

Contrary to beneficial interactions, direct competition can lead to unpredictable outcomes for the host, ranging from survival due to strong interference competition between parasite species to death from excessive parasite burden, damage inflicted by each parasite species (Johnson and Buller 2011) or immunosuppression (Cox 2001). An example of direct parasite competition with positive outcomes for the host is that of co-infections by entomopathogenic enterobacteria like *Photorhabdus asymbiotica* and *Xenorhabdus nematophila* in the caterpillar *Galleria mellonella* (Massey et al. 2004). In vitro cultures of mixed infections on agar plates showed mutual growth inhibition of bacterial species on each other, confirming their ability for mutual allelopathic interference.[1] When inoculated together in *G. mellonella*, both strains together were less virulent than each species would have been if inoculated alone, resulting in significantly decreased host mortality.

The effects of co-infection are often more nuanced, especially when metazoan parasites able to modulate host immune defences are involved (see Maizels and Yazdanbakhsh 2003; Maizels et al. 2004). Importantly, a strong antagonistic interaction between infectious agents does not necessarily restore host fitness, as each parasite species can inflict its own damage in addition to that incurred by the host due to competing pathogens. For example, co-infections of *Echinostoma trivolvis* and *Ribeiroia ondatrae* in the Pacific chorus frog *Pseudacris regilla* in Californian wetlands result in direct negative effects on each parasite's survival (Fig. 7.3), but also in more damage to the host population than either species could inflict on its own (Johnson and Buller 2011).

[1] "Allelopathic interferenc" refers to a widely reported type of interference competition in which a species inhibits the development of another through the release of harmful chemical components. 'Interference competition' is a type of direct competition in which a parasite's establishment and/or life-cycle completion are directly impeached by mechanical or chemical attacks from another pathogen (Mideo 2009).

7.4.2 Indirect Effects on (Other) Species and on Ecosystems

The presence of parasites in ecosystems affects not only their hosts but also the species linked to their hosts through predation or resource competition. For example, the local extirpation of a species (i.e. a 'resource') by parasitism will affect all the other species routinely interacting with that resource. These ripple effects, which can also be induced in predator–prey and competitive interactions, are classified as density- (DMIE) or trait-mediated indirect effects (TMIE). Both can coexist in the same trophic web.

The nature and intensity of indirect effects depend on the mode of parasite transmission (i.e. direct or indirect), parasite life stage, intrinsic and parasite-induced rates of host mortality and the efficiency of host immunity or behavioural adaptation against that parasite (see below). The level of complexity and the unpredictability of these interactions in any given ecosystem grow significantly with the number of parasite species and interactions between hosts, non-hosts and the environment (Hochberg et al. 1990; Hatcher et al. 2006; Keesing et al. 2006).

7.4.2.1 Parasite-Induced Density-Mediated Indirect Effects (DMIE)

The term "population density-mediated indirect effect" refers to cases where induced variations in the population density of a species in a system impact the population densities of other species that do not directly interact with it, for example, by propagation through the food web (trophic cascades) (Abrams et al. 1996). Population density variations can be mediated by parasites in various ways (see also Chaps. 8 and 18).

Parasite-Induced Apparent Competition When a parasite is shared by two or more host species,

these species have the potential to up or down-regulate each other's population densities via parasite-induced apparent competition (Fig. 7.4). For apparent competition to take place, one of the species must have some competitive advantage over the other(s) regarding tolerance to, or population recovery from, parasitism. Such competitive advantages can range from more efficient immune systems to comparatively faster growth in uninfected individuals (Holt 1977; Holt and Pickering 1985). Apparent competition, often difficult to disentangle from interspecies direct resource competition (see below), is most obvious in the laboratory experiment of Bonsall and Hassell (1997): when two non-competing caterpillar populations of pyralid moths *Plodia interpunctella* and *Ephestia kuehniella* are parasitised by the parasitoid wasp *Venturia canescens*, *E. kuehniella* is always eradicated (Fig. 7.4). This three-species system systematically fails to reach equilibrium even while *P. interpunctella*-wasp and *E. kuehniella*-wasp systems perdure individually. Similarly, the success of clones of *Plasmodium chabaudi* of differing virulence inoculated together in mice depends on the hosts' immunity: the avirulent strain is immunosuppressed via heterologous reactivity[2] in immunocompetent mice and least affected in immunodeficient mice, implying that immunity against the weakest clone could be mediated by host immunity (Råberg et al. 2006). No such examples are known from aquatic host–parasite systems. However, as many cases exist for terrestrial hosts, similar rules are expected to apply in freshwater- and marine environments.

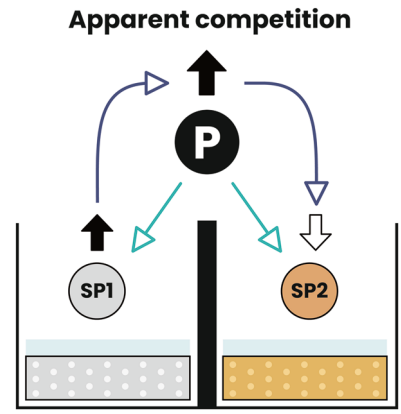

Fig. 7.4 The experiment of Bonsall and Hassell (1997) proves the existence of apparent competition. When two caterpillar populations of moths *Plodia interpunctella* ("SP1") and *Ephestia kuehniella* ("SP2"), feeding on independent unlimited resources (grey and orange substrates), are parasitised by the wasp *Venturia canescens* ("P" and green arrows), *E. kuehniella* (SP2) is always eradicated. Through its more efficient response to parasitism (black arrow), SP1 favours an increase in the population density of P (black arrow), which reduces the population density of SP2 (white arrow). The effect of SP1 on SP2 mediated by P is shown with long grey arrows

Parasite-Mediated Direct Competition Parasite-mediated direct competition has a profound influence on the respective population densities of both infected and non-infected species (Price et al. 1986). It can provoke the local extirpations of many native species populations, but also maintain balance between species that could not coexist otherwise (MacNeil and Dick 2011). This effect exists in contrast with apparent competition (see above) as host species or individuals actively compete for the same resource. In parasite-mediated direct competition, a parasite species can be shared by one (host-specific parasitism) or more host species. In the first case, depending on environmental conditions, the parasite can directly influence the fitness of its host and thus tip the scale in favour of or to the disadvantage of its non-infected competitors. For example, the native South African mussel *Perna perna* is easily outcompeted for space by the introduced mussel *Mytilus galloprovincialis*, potentially because the trematodes infecting the former significantly reduce its population density, hydration levels and growth while they do

[2] "Heterologous reactivity", also called heterologous- or cross-immunity, is a type of immune-cell cross-reactivity towards two or more antigens from different pathogen species or strains. In this phenomenon, a parasite species induces attacks against another species by triggering an immune response able to target both pathogens. Through this process, hosts can develop immunity towards many parasites sharing similar antigenic signatures if at least one of them triggers an effective immune response [see Agrawal (2019)]. On the contrary, a strong immune response against a single pathogen can be modulated by the presence of other parasites able to alter the host's immunity (Graham et al. 2005; Hardisty et al. 2022).

not infect the latter (Calvo-Ugarteburu and McQuaid 1998a, b) (Fig. 7.5). In the second case (i.e. that of a parasite shared by several host species), the parasite alters the resource exploitation efficiency of each host differently at the population level. The outcomes of parasite-mediated direct multi-host competitions depend on several factors intrinsic to both hosts and parasites, such as growth rate, magnitude of aggregation, or pathogenicity (Yan 1996). Parasite effects can be so strong as to suppress the competitive edge of more efficient, but more sensitive, species (see Chap. 8 for examples).

If the infected host species are predators of the same guild, parasite-mediated direct competition then becomes a case of parasite-mediated intraguild predation. For instance, in the British Isles, the crangonyctid amphipod *Crangonyx pseudogracilis* co-occurs significantly more often with two of its predators, the gammarid amphipods *Gammarus duebeni* and *G. pulex*, when they are parasitised by the microsporidian *Pleistophora mulleri* and the acanthocephalan *Polymorphus minutus* (see MacNeil and Dick 2011).

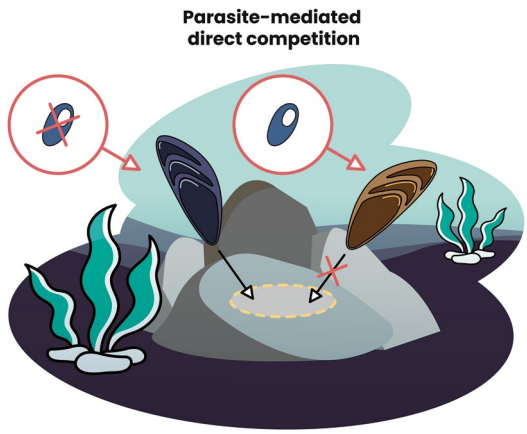

Fig. 7.5 An example of parasite-mediated direct resource competition. Here, competition for space (dashed circle) between the South African native mussel *Perna perna* (brown mussel), routinely affected by trematode larvae (white bubble), and the introduced mussel *Mytilus galloprovincialis* (black mussel), unaffected by trematodes (white bubble with crossed parasite), results in the competitive exclusion of *P. perna* by *M. galloprovincialis* (red cross on arrow)

Phenological and Population Synchrony The indirect effect of seasonal climatic fluctuations on parasite transmission can add up to the direct effects of climate on host populations, leading to synchrony in host abundance across those populations. An example is the spatial synchrony induced in populations of red grouse (*Lagopus lagopus*) by the terrestrial nematode *Trichostrongylus tenuis*, a one-host parasite with density-dependent transmission (Cattadori et al. 2005) and deleterious effects on brood production (Hudson 1986), when the effects of the parasite on grouse fertility and chick survival are enhanced by seasonal climatic conditions (Cattadori et al. 2005). A different phenomenon occurs in Californian aquatic systems (Fig. 7.6). Tadpoles of *P. regilla* at risk of developing limb malformations from infections by the frog flatworm *R. ondatrae* in the early stages of their development are significantly less likely to develop such malformations if they escape infection before or during the critical limb-development period (Johnson et al. 2011). Crucially, the risk of contracting infections starts rising in early spring in low-altitude ponds compared to mid-year in high-mountain ponds whereas tadpoles start developing at similar times in both habitats. At equal mean infection intensities, this difference results in 100 times more tadpoles malformed in earlier-warming ponds, where parasite- and host populations are most synchronised in their development and cercariae infect younger tadpoles, compared to later-warming ponds where most tadpoles have developed enough to avoid malformations by the time cercarial production takes off (McDevitt-Galles et al. 2020). This difference is particularly important in the context of climate change (see Chap. 22), for habitats might experience shifts in their temperature profiles that can synchronise parasite- and endangered-host populations, as with frogs (Yang and Rudolf 2010).

Parasite-Mediated Trophic Interactions Both predators and parasites consume other species and use the energy stolen from their 'prey' for their own survival and reproduction, leading to

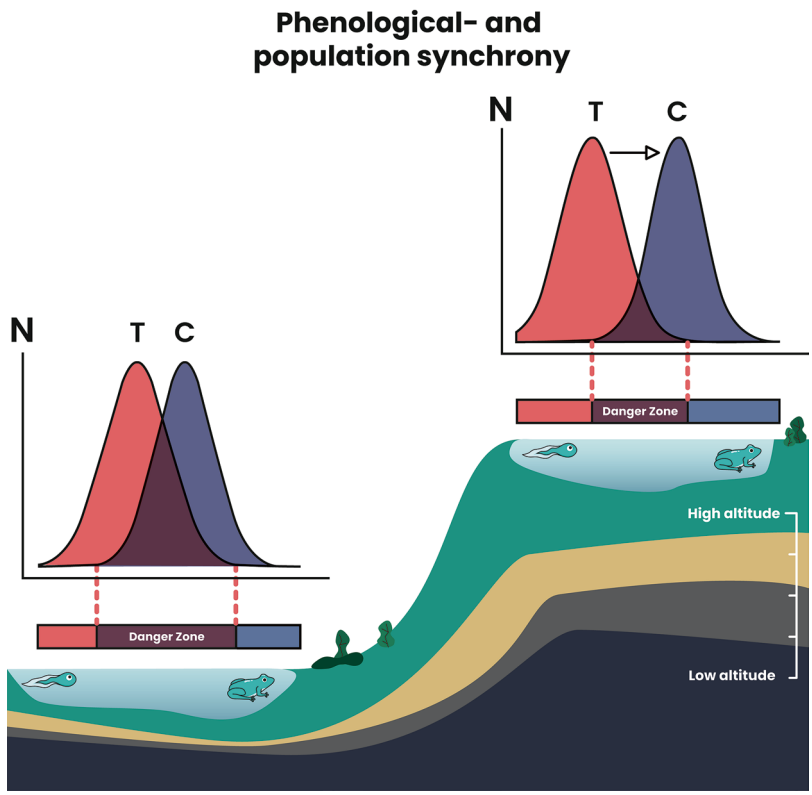

Fig. 7.6 An example of population synchrony adapted from McDevitt-Galles et al. (2020). Graphs represent population density curves of tadpoles of *Pseudacris regilla* and cercariae of the trematode *Ribeiroia ondatrae* in low- (bottom left) and high-altitude ponds (top right) (X-axis: time in a year; Y-axis: numbers of cercariae and tadpoles in each pond; C: cercariae; T: tadpoles). The purple area in each graph represents the area of synchrony between tadpole- and cercarial populations; the larger the area, the more tadpole and cercarial populations overlap and the higher the risk for tadpoles to encounter cercariae at any point during their development. Each purple area in the graphs is reported as a purple rectangle in the horizontal bar below; the longer that rectangle, the more likely tadpoles are to become infected before or during their critical window of limb development and to bear malformations as adults. Young tadpoles of *Pseudacris regilla* from high-altitude lakes (top graph and pond) avoid infection during their early lives and subsequent malformations due to a cold-induced delay in trematode emergence (arrow between F and C curves). In contrast, tadpoles of *P. regilla* from low-altitude lakes are more frequently infected because cercarial emergence occurs earlier in the year (population curves closer to each other). In low-altitude lakes, frog and trematode populations are more synchronised in their dynamics than in high-altitude lakes

an increase in consumer population (Hall et al. 2008) (but see Chap. 16). Just as in predation, consumption by parasites induces complex patterns of host population dynamics (Anderson and May 1979; May and Anderson 1979) with, in some cases, significant impacts on host population density (e.g. Hudson et al. 1998; Lafferty 2004) (Fig. 7.7). However, contrary to predators, each parasite individual can target only a single host individual per life stage. Moreover, whereas predation can only take three forms (micropredation, social predation and solitary predation) (Lafferty and Kuris 2002), host–parasite and host–parasitoid interactions are much more diverse (Hall et al. 2008). As every host–parasite system sits on a parasitism–predation gradient, each relationship will influence the surrounding ecosystem in a unique manner (Hall et al. 2008). Parasite-induced DMIEs on trophic interactions can take various forms. The hosts can be prey, predators (i.e. free-living carnivorous species) or both, in ecological assemblages incorporating

many parasite species infecting animals at all trophic levels. The effects of infections sometimes cascade from predator level to resource level (Fig. 7.7).

Natural systems in which a parasite infects prey but not its predators result in highly diverse outcomes. Indeed, the effects of consumption of prey by both predators and parasites on prey, parasite and predator population dynamics differ depending on the system under study. The more species interact in that system, the more unpredictable the overall effects of parasitism will be (Hochberg et al. 1990; Banerji et al. 2015). In this context, the fact that a large part of the knowledge on parasite-induced DMIEs on prey populations has been obtained through mathematical modelling (e.g. Chattopadhyay and Arino 1999; Greenhalgh and Haque 2007) is problematic. The strength of the links between parasite and host population dynamics is hard to assess in real-world prey populations because of the presence of many confounding factors, like predation and co-occurring diseases. An example of such difficulty is seen in the *L. lagopus–T. tenuis* system. *Trichostrongylus tenuis* has a significant destabilising effect on red grouse populations in the UK and is at least partly responsible for marked cycles (Hudson et al. 1992, 1998, 2002). Additionally, red grouse are predated upon by, among others, the hen harrier (*Circus cyaneus*) (Thirgood et al. 2000), thus imposing a density-dependent limiting and stabilising effect on those populations (Redpath and Thirgood 1999; Thirgood et al. 2000). When culling of harriers was stopped in 1992 in Langholm Moor, Scotland, the red grouse population missed a cycle and kept declining. Although there is evi-

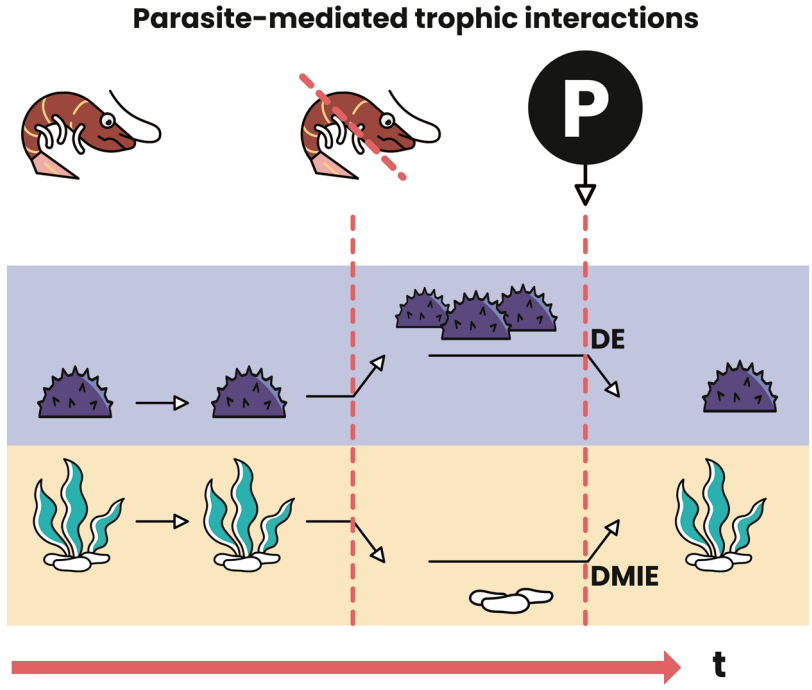

Fig. 7.7 An example of parasite-mediated trophic interactions on prey following the removal of natural predators, based on the study of Lafferty (2004). Lobsters (top) regulated urchin populations (middle) *via* predation, ensuring the stability of kelp populations (bottom) in the Channel Islands. The removal of lobsters through overfishing provoked urchin multiplication to the point that kelp forests became urchin barrens (three urchins, bare rocks). The emergence of disease (circled 'P' and downward arrow) in areas where urchin populations reached critical density levels became the main regulator of urchin populations in the absence of their natural predator, allowing kelp regrowth (one urchin, kelp). The direct effect of the parasite on urchin population density led to a density-dependent, parasite-induced density-mediated indirect effect (DMIE) on kelp forests

dence that combined effects of parasite-induced population cycles and increased hen harrier predation alone prevented the grouse population from recovering after its cyclic decline, other factors were identified as possibly being involved in the failed red grouse population recovery in the studied area (Hudson et al. 2002), those being louping ill virus (Reid et al. 1978; Hudson et al. 2002) and habitat loss (Thirgood et al. 2000). Unfortunately, no similar examples exist for aquatic ecosystems. The disappearance of predators and the subsequent rise in prey populations can itself be the source of increased density-dependent parasite-induced DMIEs on prey (see Chap. 8 for examples).

Parasite-mediated DMIEs on trophic webs tend to be strong but subtle, with complex outcomes. In contrast, predator-induced DMIEs are immediately visible, to the point that most known trophic cascades recorded in natural ecosystems are induced by predator pressure (Buck and Ripple 2017). When DMIEs induced by both parasites and predators are present in ecosystems, powerful prey- and resource-population responses can be observed (Fig. 7.8). Parasite–predator–prey systems in which a parasite infects a predator but not its prey are predicted to evolve into a state of equilibrium where (a) all predators have been eradicated by the pathogen, (b) the pathogen has been eradicated from the predator population, or (c) all the species perdure (Auger et al. 2009). By regulating predator population densities, and thus the strength of predator-induced DMIEs, parasites alone can alter the outcomes of trophic cascades for the whole ecosystem. Through parasite-induced DMIEs and direct consumptive effects, the equilibria of populations of prey and infected predators will be distinct from those they would reach in the absence of a parasite (Auger et al. 2009). A real-world illustration of this principle was observed in the Isle Royale National Park, USA. Following a canine parvovirus-induced crash of the wolf population in that area, the Western moose (*Alces alces*) population became primarily regulated, not by predation as before, but by resource availability. This change shifted the point of equilibrium of the moose population and rendered the latter vulnerable to changes in the Northern-Atlantic Oscillation (Wilmers et al. 2006, 2007).

When a parasite affects a predator, the strength of its top-down effects on the trophic web depends on two factors (Buck and Ripple 2017; Anaya-Rojas et al. 2019). First, the intensity of its top-down effects depends on how strongly this parasite regulates the predator's population density, which in turn depends on (1) the level of host resistance to parasitic invasion (Hudson et al. 1998); and (2) the extent of parasite-induced host trait alterations (see section below) (Anaya-Rojas et al. 2016). Second, a parasite's top-down effects depend on the trophic position of the predator it infects (Lafferty et al. 2006). Thus, parasite-induced DMIEs on the trophic chain are strongest when the parasite's direct impact is strongest (Anaya-Rojas et al. 2019). For example, mesocosm experiments on three-spined stickleback (*G. aculeatus*) infected by a natural combination of various parasites showed that the probability of stickleback-induced trophic cascades was significantly increased when stickleback parasite load was reduced. Moreover, fishes with natural parasite loads had much weaker direct and indirect effects on the zooplankton grazer population and the phytoplankton resource, respectively, than fishes with artificially reduced parasite loads (Anaya-Rojas et al. 2019).

While parasites that infect predators but not their prey are mainly non-trophically transmitted, parasites infecting both often exploit the trophic chain to reach their definitive hosts, in which they reproduce (see Chaps. 2–6). These parasites often depend on the death of their intermediate hosts, typically when consumed by the next host, to ensure successful transmission and reproduction. Interactions between prey and predators mediated by a shared parasite have also been mathematically modelled, allowing for the prediction of complex population dynamics with a wide variety of possible outcomes (e.g. Hsieh and Hsiao 2008). Importantly, the outcome of a trophically transmitted infection for host populations, predator or prey, is heavily dependent on the parasite's nature and life stage. Thus, although digenean intermediate-stage larvae are produced more numerously and longer-lived in healthy

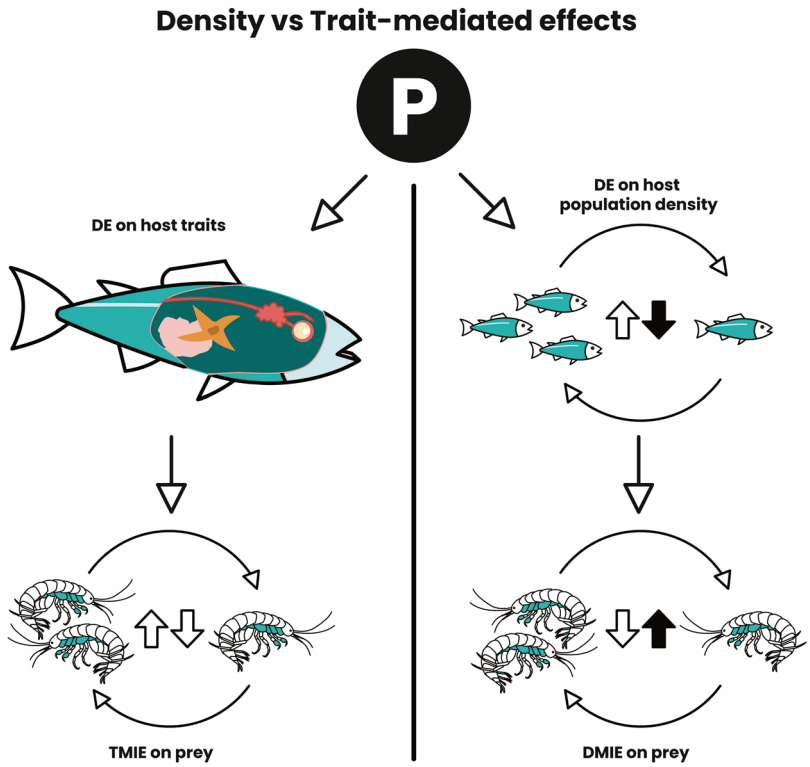

Fig. 7.8 Difference between trait- (TMIE) and density-mediated indirect effects (DMIE). (Left) Direct parasite (circled "P") effects on a species' (fish) behaviour (symbolised by brain, brainstem and eye), immunity (symbolised by a dendritic cell), other organs (symbolised by muscle tissues) or life history induces TMIEs impacting prey (amphipods) population density (thick white arrows). These TMIEs can in turn trigger more TMIEs in the ecosystem. (Right) Direct parasite effects on a species' (fish) population density induce population increases (white arrow) or decreases (black arrow) in that species, which decrease (white arrow) or increase (black arrow) prey populations, respectively, via DMIEs

first-intermediate hosts (Seppälä et al. 2008), parasite-induced adaptive phenotypic changes (see section below) in digenean second-and-above intermediate hosts often tend to increase the likelihood of death by predation (e.g. Lafferty and Morris 1996; Seppälä et al. 2004; Seppälä et al. 2005; reviewed in Lafferty and Shaw 2013).

DMIEs can be manifested by all types of parasites but are strongest when parasites kill their hosts (Buck and Ripple 2017). Thus, the outcomes of both effects on the ecosystem depend on the parasite lineage and life stage (see Sect. 7.4.1). A widely known example of a parasite inducing strong consumptive and indirect effects is chytrid fungus (*Batrachochytrium dendrobatidis*) (see Chap. 4). Where chytridiomycosis outbreaks collapsed frog populations in Rio Guabal, Panama (i.e. direct consumptive effect), the absence of tadpoles resulted in a sharp increase in chlorophyll *a* and inorganic matter, and a drastic shift in the periphyton community composition from small diatom-dominated to larger diatom- and cyanobacteria-dominated (i.e. parasite-induced DMIE on the trophic web) (Connelly et al. 2008; Chap. 4).

7.4.2.2 Parasite-Induced Trait-Mediated Indirect Effects (TMIE) or Interactions (TMII)

The term "trait-mediated indirect effect" (TMIE) is used when alterations of a species' behaviour (see Sect. 7.4.1), phenotype or life history,

because of the presence of another species, impact the population densities of still other species that do not directly interact with it (Abrams 1995; Werner and Peacor 2003, also see Chap. 8) (Fig. 7.8). The latter species become impacted by the propagation of TMIEs through the food web via predation or competition (Abrams 1995). TMIEs are understood to be at least as strong as DMIEs (Werner and Peacor 2003).

Predator-induced TMIEs stem from prey modifying their behaviour or physiology in order to remain alive. In contrast, most parasite-mediated TMIEs on the ecosystems happen when host populations have already been infected, that is, 'consumed' (Fig. 7.8). Thus, parasite-induced TMIEs can arise from both consumptive and non-consumptive effects depending on whether a host has been successfully infected (Buck 2019). An example of consumptive TMIE, the most frequent type of parasite-induced TMIE (Buck and Ripple 2017), is described by Toscano et al. (2014): crabs of *Eurypanopeus depressus* conspicuously infected by the castrating rhizocephalan barnacle *Loxothylacus panopaei* consumed significantly fewer mussels (*Brachidontes exustus*) than uninfected or inconspicuously-infected crabs, partly because they reacted significantly more slowly to the introduction of mussel prey and because conspicuously infected crabs seemed to become more inactive. The mussel population is thus indirectly affected by the rhizocephalan parasites. Although seemingly less common than in predator-prey dynamics (Buck and Ripple 2017), parasite-induced non-consumptive TMIEs can be observed when potential hosts modify their habits or phenotype to avoid infection, which in turn affects other species in their environment (Koprivnikar et al. 2021). Preventative self-defence, widely present in predator-prey interactions (e.g. Peacor et al. 2020), is termed risk-induced trait response (RITR). In host–parasite interactions, RITRs are expressed through five strategies that may incur associated fitness costs (Rigby et al. 2002; Daversa et al. 2021): (1) avoidance of diseased conspecifics (as seen in lobsters; Behringer et al. 2006); (2) active avoidance of infective parasites upon detection (e.g. tadpoles swimming explosively to avoid cercariae; Taylor et al. 2004); (3) avoidance of places or items where parasites tend to become concentrated (see Hutchings et al. 2000); (4) the use of specific counterattack behaviours when those items, or the parasites, cannot be avoided (e.g. fishes soliciting the services of cleaners against gnathiid isopods, parasitic copepods and monogeneans; Grutter 1999; Becker and Grutter 2004); and (5) the development of immune resistance (Råberg et al. 2009). Importantly, parasite- and predator-induced indirect effects are not mutually exclusive and can act on the same trophic chain (see Banerji et al. 2015).

Through prey, parasites can trigger bottom-up (from prey on predator) or top-down (from prey on resource) TMIEs at the same time in the same trophic chain. Whether more than one TMIE occurs in any predator-prey-resource system depends on the diversity of direct effects a parasite can induce in its prey host. For example, when moderately infected by the bacterium *Holospora undulata*, individuals of the ciliate *Paramecium caudatum* swim faster and feed more often than non-infected ones, thus impacting the primary resource more strongly; this impact reflects parasite-induced top-down TMIEs on the resource. In addition, a parasitised *P. caudatum* population induces lower peak population densities and higher mortality rates in the ciliate *Didinium nasutum*, the predator of *P. caudatum*. This population decline can reflect either a parasite-induced bottom-up DMIE on the predator mediated by higher prey mortality, or a parasite-induced TMIE on the predator mediated by the compromised nutritional value of the infected prey (Banerji et al. 2015).

Both TMIEs and DMIEs can also affect the traits or population densities of species outside of the direct trophic chain of the parasitised host. This type of interspecies link, first described in plant-insect systems and not usually included in trophic web models, is termed an "indirect interaction web" (Utsumi et al. 2010). An example in aquatic systems is the link between infestation of the cricket *Nemobius sylvestris* by parasitic larvae of the hairworm *Paragordius tricuspidatus* on land and benthic algal density in fresh water. Larval nematomorphs nearing maturity manipu-

late their cricket hosts' behaviour to jump into water, where they exit the host, mature and become free-living adults (Thomas et al. 2002). In season, the water-trapped crickets form most of the diet of Kirikuchi char (*Salvelinus leucomaenis*) (Sato et al. 2011). Here, "consumption" of *N. sylvestris* by *P. tricuspidatus* induces *S. leucomaenis* to temporarily shift its diet, thus amounting to a parasite-induced TMIE on the fish mediated by behavioural change in the cricket. In turn, preferential feeding on crickets by the fish reduces predation on invertebrate grazers, thus reducing benthic macroalgae density via predator-induced DMIE on the resource mediated by grazers (Sato et al. 2012).

When it acts on pathogens, host immunity amounts to predation in terms of trophic level (Pedersen and Fenton 2007). Unlike predation, however, both host immunity and host resource (i.e. energy) are contained within the ecosystem of the host's body (i.e. the 'inner' ecosystem), so that the impairment of one results in the impairment of the other (Johnson and Buller 2011). In turn, any failure of immunity or energy depletion can affect host behaviour and mortality, and thus the entire ecosystem. Therefore, host immunity is a major way through which parasite-induced TMIEs act on ecosystems at large. This is especially true in cases of co-infection. As parasite interactions with each other and their use of the host resource are mediated in part by host immunity, parasites induce TMIEs on each other and the host as well as direct effects [see Sect. 7.4.1 and review by Johnson and Buller (2011)]. Trait-mediated apparent competition between parasite species (top-down effect) takes place when one of the parasites stimulates host immune defences, hampering the growth and reproduction of the others through immune cross-reactivity (Christensen et al. 1987; Balmer et al. 2009) or cross-immunity between pathogens with similar antigenic signatures (see Curry et al. 1995). In contrast, host immune alteration induced by one of the parasites can also incidentally benefit co-infecting agents (Cattadori et al. 2008; Su et al. 2005). Thus, parasite assemblages in hosts tend to be non-random and not merely the consequence of parasite accumulation over time (Thomas et al. 1997; Dezfuli et al. 2000; Poulin and Valtonen 2001). In fact, parasites that are able to modify host traits can condition the successful invasion of a chain of other parasite species extending in both space (i.e. infection sites) and time (i.e. sequential infections throughout a host's life) via parasite-induced TMIEs on other parasites mediated by changes in host traits (Christensen et al. 1987; Poulin and Valtonen 2001; Jackson et al. 2006; Karvonen et al. 2019). Some parasites that are unable to manipulate those traits even depend on these associations to complete their life cycles. For example, the digenean *Maritrema subdolum* infecting the amphipod *Gammarus insensibilis*, its second intermediate host, benefits from coinfection with *Microphallus papillorobustus* as this parasite induces the amphipod to swim closer to the surface in order to increase transmission probability whereas *M. subdolum* cannot (Thomas et al. 1997). These TMIEs can in turn trigger more TMIEs in the other species in the ecosystem. The outcomes of TMIEs on both parasites and hosts depend on a wide variety of biotic (i.e. intrinsic to the pathogens, the hosts or the surrounding free-living species interacting with the host) and abiotic factors (reviewed by Herczeg et al. 2021). For that reason, synergistic relationships between parasites can be tricky to ascertain and difficult to disentangle from individual-host immunity and environmental variations (Karvonen et al. 2009).

7.4.2.3 Parasite-Induced Indirect Alteration of Habitat

In aquatic ecosystems, the influence of parasites extends not only to their hosts but also, indirectly, to the whole community via TMIEs. By altering their hosts' ability to perform their usual ecosystem functions through normal behaviour, parasites indirectly alter the characteristics of the surrounding physical habitat, influencing the living conditions of the other species and leading to profound changes in community composition and function (Mouritsen and Poulin 2010). Such influence on physical habitat characteristics is most visible in the case of the himasthlid trematode *Curtuteria australis* infecting the foot of the cockle *Austrovenus stutchburyi*, an ecosystem

engineer of muddy substrates in New Zealand (see Chap. 8 for details). The influence of parasite presence on habitat and community structure is on par with those of more studied interspecific interactions such as competition and predation, especially when parasites affect keystone species. Parasites' effects are so important for ecosystems some authors consider them as ecosystem engineers in their own right (Hatcher et al. 2012).

7.5 Parasites Are Affected by Ecosystem Processes

7.5.1 A Parasite's Fate Is Linked to Those of Its Hosts

Parasites, like all other species in natural ecosystems, are affected by the processes arising from species and habitats interacting with each other. If we consider parasitism as a form of predation (see Hall et al. 2008 but see also Chap. 16), then the loss of a host species to a parasite amounts to the loss of a prey species to a predator. However, given parasitism is a long-term relationship, the "consumption" of the host by the parasite happens over a much longer time. Thus, and contrary to free-living species, parasites can be positively or negatively affected by what happens to them directly [e.g. predation on free-living stages or on ectoparasites; see Sect. 7.5.2 and also Rohr et al. (2008)], what happens to uninfected host populations [e.g. in case of local extirpation or extinction of a host species, thus preventing life-cycle completion; see below and Sures et al. (2023)], and what happens to the individual host while the parasite is consuming it [see Raffel et al. (2008) on cercarial encystment].

Broadly speaking, the more stressed an ecosystem is, the more affected parasite biodiversity is and *vice versa* (Huspeni and Lafferty 2004; Pérez-del Olmo et al. 2007). However, predicting how a parasite species will react to any specific environmental stressor applied to it or its hosts is challenging. The effects of stressors greatly depend on the life-cycle characteristics of each parasite species (e.g. heteroxenous or monoxenous, internal or external stages, trophic or non-trophic transmission, level of host-specificity, number of host species used); in particular, a parasite species' survival in a stressed environment is determined by the resilience of its most sensitive life stage (Sures et al. 2023). Parasites with indirect life cycles (i.e. heteroxenous) are thought to be more at risk than monoxenous ones (Wood and Lafferty 2015). Nevertheless, individual parasite groups exhibit varying sensitivities, even among monoxenous species (see Lafferty 1997), resulting in a complex scenario. For example, contrasting results have been obtained in studies on gyrodactylid monogeneans (Poléo et al. 2004; Pravdová et al. 2023) depending on the type of environmental damage. In addition, a parasite's resilience depends heavily on the tolerance level of each of its hosts towards environmental stressors (Lafferty and Kuris 2009a). As any species on Earth can have one or more symbionts (including parasites), parasites might be the group disappearing at the highest rate through mechanisms of co-extinction and extinction cascades (Koh et al. 2004; Dunn et al. 2009; Lafferty 2012) as well as the group for which extinctions might be the most underreported (Dunn et al. 2009). As parasites are seen as broadly undesirable by the wider community despite their vital importance (Stork and Lyal 1993; Dougherty et al. 2016), conservation efforts on these species are rare (see Chap. 13).

The threats to aquatic ecosystems are many, ranging from water pollution (Chap. 20) to the impacts of climate change (Chap. 22). When environmental damage leads to a significant population decrease of one link in a parasite's host chain, then that parasite may become extinct. Several cases have illustrated this point, such as that of the nematode *Cystidicola stigmatura*, pronounced locally extinct following a crash in the abundance of its definitive host, the lake trout *Salvelinus namaycush* in the North American Great Lakes (Black 1983). Furthermore, if host density decreases below a sustainable transmission threshold in an area where all of a parasite's life-stages are concentrated, then that parasite will also be at risk even if the host population is not itself threatened with extirpation (Lafferty and Kuris 1999). As such, a parasite is predicted

to disappear before any of its hosts (De Castro and Bolker 2005). Parasite survival and host population fluctuations are so intricately linked that parasites can be used in wild commercial fish stock monitoring (Williams et al. 1992; Marcogliese 2002; Chap. 17).

7.5.2 Parasites as Prey for Incompatible Hosts

Parasites are commonly consumed as prey at both intermediate and adult stages by incompatible hosts that arrest trophic transmission (Johnson et al. 2010). Parasites can be detrimentally consumed in four different ways, namely, through concomitant predation, targeted predation on free adult or larval stages, grooming and hyperparasitism (Fig. 7.9) (reviewed by Johnson et al. 2010).

7.5.2.1 Concomitant Predation

Adaptive changes of parasites in response to an evolutionary arms race with their hosts can, in turn, induce profound changes in host phenotype (see Sect. 7.4.2). Such changes, however, are not always to the advantage of the parasite. Indeed, a behaviourally impaired, weakened or more conspicuous host is rendered vulnerable, not only to the next compatible host species in line, but also to the rest of the animal community, thus leading to concomitant predation and subsequent reduction in parasite transmission. A typical example of this kind of parasite consumption is that of trematode parasitism in the cockle *A. stutchburyi*. Although the definitive hosts of the trematode are birds, cockles stuck on the sediment surface are predated upon by fishes significantly more often. As the great majority of metacercariae are consumed by unsuitable hosts or lost to the environment (Mouritsen and Poulin 2003), the value of metacercarial accumulation in the cockle foot as an evolutionary adaptation facilitating transmission has been questioned (see Chap. 8).

7.5.2.2 Grooming

Whole groups of animals specialise in feeding on parasites already infecting their hosts. One of the most representative examples of this phenomenon is predation on gnathiid isopods on coral reefs by obligate cleaner species like cleaner wrasses (*Labroides* spp.) and shrimps [*Lysmata* spp. (Grutter 1999; Becker and Grutter 2004)] and facultative consumers like species of *Diproctacanthus* and *Thalassoma* (Labridae), *Saurogobio* (Cyprinidae) and *Gramma* (Grammatidae) (Randall 1967; Grutter and Feeney 2016). Interspecies grooming in reaction to parasitism has conditioned the adaptive behavioural complexification of a wide range of species towards investing time in queueing to get cleaned rather than in feeding or mating (Grutter 1995, 1996; Bshary and Grutter 2002). This phenomenon has important short- and long-term implications for parasite burdens both in the environment and on the host. As both host- and non-host species benefit from parasite removal (Bshary 2003; Waldie et al. 2011), the presence of cleaners regulating parasite populations is critical for maintaining free-living species biodiversity and abundance on coral reefs (Grutter et al. 2003).

7.5.2.3 Predation on Free-Living Life Stages

In some ecosystems, free-living stages of parasites (i.e. eggs, larvae, juveniles or adults) are purposely or incidentally consumed by predators (Thieltges et al. 2008a, b). For example, cercariae of the marine trematode *Himasthla elongata* targeting the keystone cockle *C. edule* as second-intermediate host are hunted by crabs, accidentally ingested by other compatible hosts not consumed by the definitive hosts (thus not allowing life-cycle completion), or incidentally consumed as planktonic food by incompatible filter-feeders. The dilution of cercarial output between all these non-target species significantly reduces metacercarial infection intensity in the whole cockle population (Thieltges et al. 2008a). In coral reef ecosystems, the free-living adults of gnathiid isopods are targeted by nocturnal predatory soldierfishes of the genera *Myripristis*, *Holocentrus* and *Sargocentron*. As these fishes are more numerous than specialist cleaner gobies (*Elacatinus evelynae*) in their

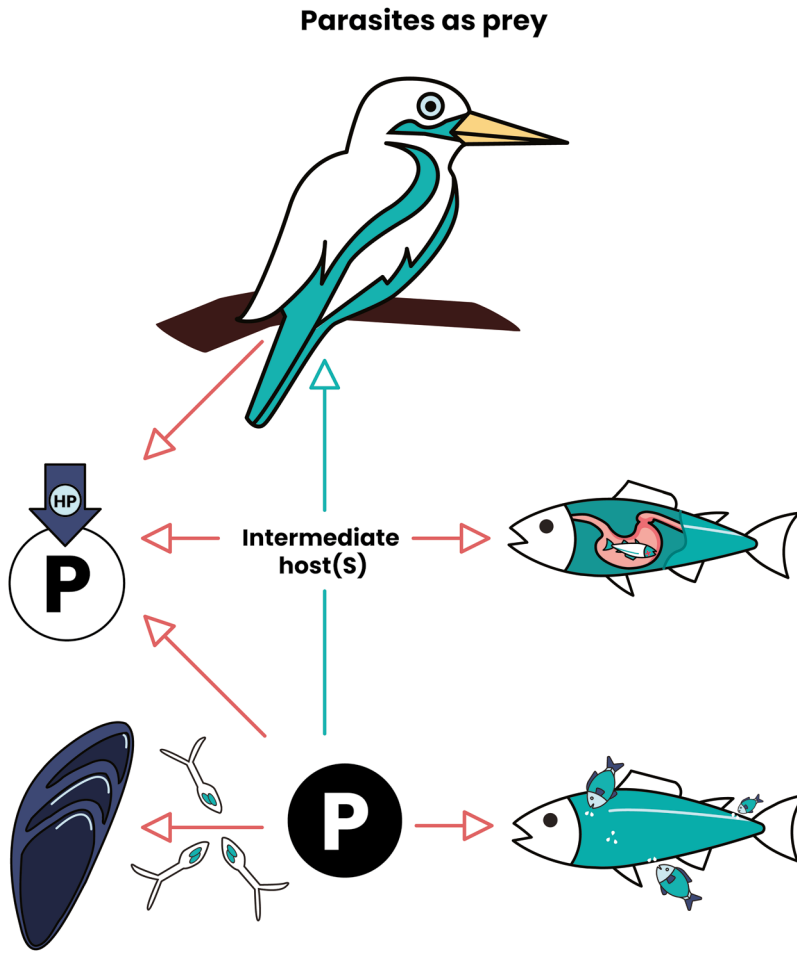

Fig. 7.9 An illustration of how parasites can become prey for incompatible hosts. A parasite individual (black-circled 'P') can fail to reach and reproduce in its final host (bird) in various ways (red arrows). (Bottom right) It can be directly eliminated from the intermediate (or definitive) host through consumption by cleaner species. (Bottom left) Its infective swimming stages (cercariae) can be consumed incidentally by filter feeders (mussel) or intentionally by other predators. (Top left) It can be killed by a hyperparasite at any stage of its development ('HP' in thick arrow). (Top right) It can be incidentally consumed by a predator along with its host. The green arrow symbolises the only pathway through which a parasite will successfully reach and reproduce in the definitive host

studied system, Artim et al. (2017) hypothesise that these facultative predators significantly affect larval gnathiid populations at ecosystem level. For incompatible predators targeting them, parasites are valuable prey (see Schultz and Koprivnikar 2019) with high reproductive output (Lafferty et al. 2006; Kuris et al. 2008). Thus, the consumption of parasitic free-living stages decreases the predation burden on other prey (Schultz and Koprivnikar 2019), triggering TMIEs and DMIEs on trophic chains (Lafferty et al. 2006).

7.5.2.4 Hyperparasitism

Hyperparasitism refers to the consumption of parasites by other parasites. One of the most studied cases of hyperparasitism is that of bacteriophages (Węgrzyn 2022) infesting pathogenic bacteria (reviewed by Ye et al. 2019). However, hyperparasitism encompasses many more inter-

action types often involving metazoan organisms. In aquatic systems, ciliates of the subclass Peritrichia (phylum Ciliophora) infect parasitic crustaceans (reviewed by van As 2019), while parasitic nematomorph larvae parasitise trematode rediae in their snail hosts (Hanelt 2009). In the marine environment, the cryptoniscid isopod *Liriopsis pygmaea* attaches to the rhizocephalan barnacle *Briarosaccus callosus*, which in turn infects the false king crab *Paralomis granulosa* (Peresan and Roccatagliata 2005); many more cryptoniscid genera are known to parasitise rhizocephalans (van As 2019). Aquatic microsporidians (Fungi) and myxosporeans (Cnidaria: Myxozoa) also include several hyperparasitic species. For instance, microsporidians of the genus *Unikaryon* parasitise species of *Microphallus* Ward, 1901 (Digenea: Microphallidae), the trematodes whose metacercariae encyst in the shrimp *Panopeus herbstii* (Sokolova et al. 2021); and the myxozoan *Myxidium giardi* parasitises the monogenean *Pseudodactylogyrus bini* which in turn infects the European eel *Anguilla anguilla* (Aguilar et al. 2004). Hyperparasitism contributes significant direct and indirect effects on parasite- and host populations, respectively, regulating the proliferation of the former and favouring the growth of the latter (Gleason et al. 2014). This phenomenon is also predicted to drive the evolution of both hyperparasite and parasite virulence (Parratt and Laine 2016; Northrup et al. 2024), with potentially significant consequences for host–parasite relationships (Springer et al. 2013).

7.5.3 High Biodiversity Favours Parasite Dilution Effects

A host–parasite relationship never exists in a void. It is surrounded by a multitude of other species that enact a wide range of direct and indirect effects on both the host and the parasite. In many ecosystems, those species that are not part of the parasite's life cycle can hinder transmission to definitive hosts and the spread of a disease in a host population. This phenomenon is called a "dilution effect" (Ostfeld and Keesing 2012; see also Chap. 18).

Dilution effects are enabled by dead-end predation on free-living stages, competition dynamics between compatible and incompatible hosts, and downregulation of compatible host populations via predation (Hall et al. 2009). In addition, the more the obligate hosts are regulated by other incompatible species, the more likely the dilution effects are (Ostfeld and Keesing 2012). For example, the targeted removal of sick and weakened hosts by predator populations (e.g. Genovart et al. 2010) contributes to the health of the host population through concomitant parasite predation (see Sect. 7.5.2) (Packer et al. 2003). For example, freshwater crayfish (*Faxonius limosus*) and water scorpions (*Nepa cinerea*) catch significantly more individuals of *G. pulex* infected by the acanthocephalan *P. laevis* than they do uninfected ones, destroying those parasites and preventing their transmission to the fish definitive hosts (Kaldonski et al. 2008). Thus, the higher the biodiversity of an ecosystem is, the less likely it is that parasites will be able to reach their definitive hosts. The inverse is also true: in prey populations mainly regulated by disease outbreaks, factors negatively affecting predator populations could also favour parasite transmission (Packer et al. 2003). The lack of dilution effects has been most apparent in recent decades as human disease emergence has intensified together with habitat- and biodiversity loss (Civitello et al. 2015). Indeed, reservoir-species of pathogens shared by both humans and wild animals, notably rats, bats and birds, are more present and abundant in habitats heavily damaged by humans (Gibb et al. 2020).

Natural ecosystems are delicate, intricate webs. Dilution effects can appear or disappear, not only because of the removal of predators but also *via* the introduction of invasive competitors. An example of such is the sharp decline in infections of native St Lawrence River fishes by trematodes of the genus *Diplostomum* following the introduction of the Eurasian round goby, *Neogobius melanostomus*. This invasive species is thought to act as a dead-end host for the trematodes, thereby reducing the numbers of cercariae

entering the intermediate hosts that would allow infection of the definitive host, the ring-billed gull *Larus delawarensis*. In addition, a shift in the gull's diet towards invasive gobies may have reduced predation on native fishes and thus trematode transmission (Gendron and Marcogliese 2017).

7.6 Conclusion and Future Direction

The intricate interactions between aquatic parasites, their hosts and the ecosystems they inhabit reveal a complex web of ecological dynamics. Aquatic parasites play multifaceted roles, influencing individual host health, population dynamics, biodiversity and overall ecosystem health. These roles often manifest in both direct and indirect ways, shaping ecosystems in subtle yet profound manners.

The exploration of terrestrial examples within this chapter highlights the paucity of comparable data in aquatic environments. While terrestrial ecosystems provide numerous case studies of parasite-induced ecological interactions, aquatic systems remain underexplored. This disparity underscores the critical need for targeted research in aquatic environments to uncover the processes and interactions analogous to terrestrial systems.

Future research should prioritise filling this knowledge gap by investigating the specific ways in which aquatic parasites influence their ecosystems. This includes studying parasite-induced TMIEs and DMIEs within aquatic food webs, as well as the broader ecological consequences of parasite-host dynamics in aquatic settings. Understanding these interactions is essential for developing comprehensive models of aquatic ecosystem health and resilience. Additionally, future studies should focus on the role of parasites as ecosystem engineers in aquatic environments. Given their potential to alter ecosystem characteristics and community structures, understanding how parasites interact with key species in aquatic ecosystems will provide deeper insights into their ecological significance. By advancing research in these areas, it will be possible to develop a more holistic understanding of aquatic parasitology, contributing to the conservation and management of aquatic ecosystems at large. The insights gained will not only enhance scientific knowledge but also inform practical approaches to maintaining ecosystem health in the face of environmental changes and challenges.

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