CHAPTER 12

# How Predators Affect Marine Parasites and Diseases

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## 1. Introduction

The pivotal role of predators in driving population dynamics of marine organisms and structuring marine communities is a cornerstone of marine ecology. Since the early experimental work by pioneers such as Bob Paine and Robert Connell (Paine 1966; Connell 1970), a plethora of studies have identified strong direct consumptive effects of predators that have disproportionally large impacts on the structure of entire communities ("keystone species") (Paine 1969; Mills et al. 1993). These direct effects of predators on the density of their prey can also have indirect effects on the prey's resources and thus lead to trophic cascades (Pace et al. 1999; Terborgh and Estes 2010; Eriksson et al. 2024). A seminal example of these density-mediated indirect effects comes from sea otters in Pacific kelp forests, where the direct predation of otters on herbivorous sea urchins indirectly leads to an increase in kelp biomass by reducing urchin grazing pressure. When sea otter predation pressure is reduced, such as in regions where they have been hunted to extinction, massive increases in sea urchin abundance can intensify grazing pressure on kelp to the extent that former kelp forests turn into sea urchin barrens (Estes and Palmisano 1974; Estes and Duggins 1995).

Alongside these consumptive effects, predators can also exert non-consumptive effects. In this case, predators do not consume their prey, but the predation risk perceived by their prey can lead to morphological, physiological, or behavioral changes in prey individuals in an effort to avoid predation but also to prepare for it through a "flight or fight" response (Lima 1998; Sheriff and Thaler 2014; Werner and Peacor 2003). As with consumptive effects, these predator-induced alterations of prey traits can lead to trait-mediated indirect effects involving other species that may modify population dynamics and community structure (Peacor et al. 2020). For instance, in marine systems, the presence of sharks can cause herbivorous dugongs to reduce their grazing pressure on seagrass while engaging in predator avoidance. Thus, the mere presence of sharks can indirectly affect the standing stock of local seagrass meadows, even without consuming any prey (Wirsing and Ripple 2011).

When considering how predators may affect prey relationships with other species through consumptive and non-consumptive effects, it has become increasingly recognized that such predator effects include parasite–host interactions and thereby alterations of infectious disease dynamics. For instance, when predators preferentially consume or avoid infected hosts, this can alter parasite infection levels in the host population. Likewise, changes in host behavior in response to predation risk can change the likelihood of parasite–host encounters. Understanding such predator effects on parasites and diseases is important given the pivotal role of predation in marine systems as well as large-scale marine predator declines and rewilding initiatives that often consist of reintroducing predators (Myers and Worm 2003; Myers et al. 2007; Heithaus et al. 2008; Pettorelli et al. 2019). In this chapter, we provide a conceptual framework for how the consumptive and non-consumptive effects of predators influence

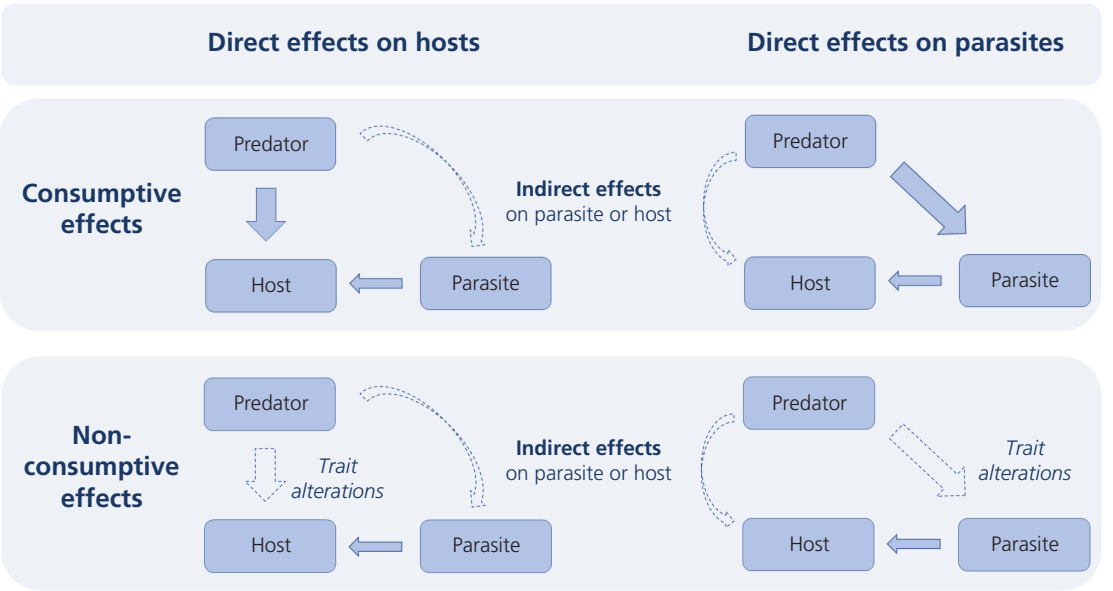


**Figure 12.1** Conceptual framework of consumptive and non-consumptive effects of predators on parasite–host interactions and diseases. Both result from direct effects of predators on either hosts or parasites, which in turn can lead to indirect effects on parasites or hosts in parasite–host interactions.

parasite–host interactions and diseases by affecting hosts or parasites (Figure 12.1). We build on previous reviews (e.g., Lopez and Duffy 2021; Shaw and Civitello 2021; Richards et al. 2023; Thieltges et al. 2024) and illustrate predator effects with empirical examples from marine ecosystems. We first discuss consumptive effects, via predation either on hosts or on parasites, before exploring non-consumptive effects of predators. Finally, we identify gaps in our current knowledge and highlight open questions for future research.

## 2. Consumptive Effects of Predators

Predators can have consumptive effects on parasites and diseases by either consuming prey that serve as hosts to parasites or consuming parasites themselves (Figure 12.1). These direct effects of predators on hosts or parasites by reducing their numbers can lead to indirect effects on either player in the parasite–host interaction and thus alter disease dynamics (Figure 12.1). While those indirect effects are largely density-mediated (i.e., caused by a reduction of host or parasite density through predation), there can also be other side effects of predation, such as the release and spread of parasite stages during what is known as "partial predation" (i.e., when predators inflict wounds on prey that can become infection portals) (Lopez and Duffy 2021; Richards et al. 2023). In the following section, we outline the main mechanisms behind consumptive effects of predators on host–parasite interactions and illustrate them with examples from marine ecosystems.

### 2.1. When Predators Consume Hosts

There are several ways the consumption of hosts by predators can affect parasite–host interactions. First, a reduction in the density of hosts through predation can affect infectious disease dynamics because host population density is often a strong driver of parasite population size (Arneberg et al. 1998; Grenfell and Dobson 1995). This stems from the basic epidemiological principle that transmission among hosts is expected to increase with increasing host density because encounters of susceptible individuals with infected hosts or free-living infective stages of parasites become more frequent, thereby facilitating transmission. In contrast, when the density of hosts falls below the threshold level for a specific parasite population, transmission becomes compromised and can cause local parasite extinction. In

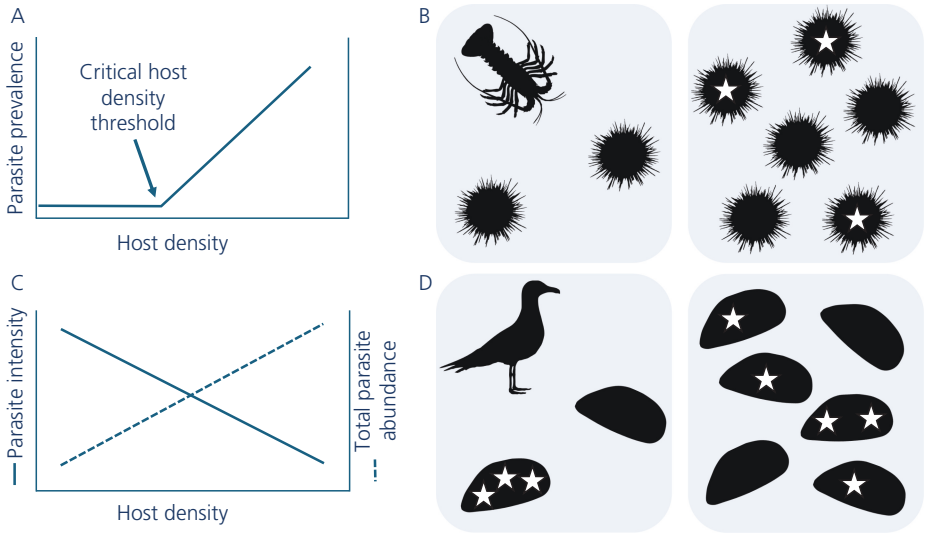


**Figure 12.2** By reducing host density, predators can affect parasites and diseases. (*A*) Below a critical host density threshold, transmission may be impaired, leading to local parasite extinction, for example, (*B*) observed in lobsters preying on sea urchins that are affected by a bacterial disease (Lafferty 2004). (*C*) Host density can have the opposite effect on macroparasite intensity in individual hosts through dilution effects, where infection levels in hosts are driven by exposure to infective stages; however, the total number of parasites in the population (total parasite abundance) still increases with host density. (*D*) Both effects can, for example, be observed in mussels infected by trematode metacercarial (cyst) stages (Levakin et al. 2023).

marine systems, support for such "critical host density thresholds" (Figure 12.2A) is evident in systems such as sea lice epidemics in salmon farms, where high stocking densities above thresholds facilitate disease outbreaks (Krkošek 2010; Frazer et al. 2012).

The existence of critical host density thresholds suggests that predators can initiate indirect effects on parasite populations and disease dynamics by altering host densities through predation. In marine systems, the removal of predators through human activities such as hunting or fishing can reveal these indirect effects. For example, in the California Channel Islands, lobsters control the populations of herbivorous sea urchins through predation, which allows the kelp forest to grow in a classic trophic cascade fashion (Lafferty 2004). However, in areas where lobsters are removed by fishing, this eliminates the top-down control and leads to an increase in sea urchin populations and a subsequent overgrazing of local kelp forests. Data from kelp forest communities at 16 sites over the course of 20 years indicates that the removal of top-down control can cause local urchin populations to exceed the critical host density threshold for a bacterial pathogen of sea urchins, resulting in disease outbreaks (Figure 12.2B). As expected for directly transmitted microparasites that have simple one-host life cycles, the associated mortalities of sea urchins are also density-dependent, with high-density populations showing the highest mortalities (Lafferty 2004). This example illustrates the potential for predators to control diseases by reducing host density. It also illustrates that fisheries can indirectly affect diseases of non-target organisms by eliminating the top-down control of host densities through the removal of their predators.

While the positive relationship between host density and parasite prevalence is a general epidemiological pattern (Arneberg et al. 1998; Grenfell and Dobson 1995), host density can have the opposite effect on infection levels in individual hosts of macroparasites. The latter have complex life cycles without direct transmission, such that host infection levels are driven by the exposure to infective stages (in contrast to microparasites that multiply within their hosts). In this case, high host densities can lead to lower infection intensities in hosts as the pool of infective stages becomes divided among all host individuals (Figure 12.2C). These encounter-dilution effects are considered to be one of the adaptive advantages of living in groups ("safety in numbers")

(Mooring and Hart 1992; Côté and Poulin 1995). However, the total number of parasites in a local host population ("total parasite abundance") can still be expected to increase with host density as chances of host encounter increase with host density (Figure 12.2C). In marine systems, such encounter-dilution effects have been reported from ectoparasitic copepods (sea lice) in Atlantic salmon (Samsing et al. 2014) and infective stages of trematodes in mollusks (Mouritsen et al. 2003; Thieltges and Reise 2007; Buck et al. 2017; Magalhães et al. 2017; Levakin et al. 2023). Although predator effects on parasites have not explicitly been studied in these systems, it is reasonable to assume that predator-driven reductions of host density would lead to an increase in infection intensities in individual hosts while total parasite abundance decreases (Figure 12.2D).

In all these cases, predators usually do not distinguish between infected and non-infected prey. However, predation is often non-random, with infected and weaker individuals in a host population potentially experiencing higher predation pressure than do uninfected conspecifics. Such selective predation on infected hosts can occur as a result of altered behaviors (e.g., decreased activity levels as a result of sickness behavior) or other traits (e.g., appearance) associated with disease. When predators selectively remove infected individuals from a population, this can amplify their effects on parasites and disease beyond host density reductions alone. As a result, the removal of sick or moribund individuals by predators can lower transmission and disease risk in a host population ("keeping the herds healthy hypothesis") (Packer et al. 2003). However, in some cases, selective predation on infected hosts can also increase disease risk when parasite-induced manipulations of host behaviors or appearance facilitate transmission to the next host in complex life cycles (e.g., making prey easier to find and catch for predators that serve as their hosts) (Poulin 2010; Poulin and Maure 2015). Examples of increased trophic transmission in marine systems include trematode infections in killifish brains that alter their behavior in a way that increases predation by bird definitive hosts (Lafferty and Morris 1996) and trematode infections in the feet of clams that inhibit their burrowing capability and result in higher rates of predation by downstream hosts compared to uninfected burrowed individuals (Thomas and Poulin 1998). The increased transmission rates resulting from higher predation on infected prey can be expected to have positive effects on parasite population dynamics and to increase disease risk.

However, in many other cases, selective predation on infected hosts does not lead to infections of predators; instead, the removal of infected individuals can reduce parasite population size and disease risk (Packer et al. 2003). In principle, it is also possible that some predators preferentially consume uninfected hosts (e.g., to avoid infection or because of potentially higher nutritional value of uninfected prey), but this aspect has been generally little studied (Gutierrez et al. 2022). In marine systems, studies on preferential predation by predators on infected or uninfected prey are surprisingly rare, as indicated by recent meta-analyses on predation and parasitism interactions (Richards et al. 2021; Hasik et al. 2023). The existing studies on selective predation on infected prey indicate that there is no universal response. For example, in prey choice experiments, blue crabs did not show a preference for oysters infected with a protozoan parasite (*Perkinsus marinus*) (Malek and Byers 2016). In contrast, gastropods and bivalves infected with shell-boring polychaetes are preferentially predated by crabs in choice experiments (Kent 1981; Ambariyanto and Seed 1991; Buschbaum et al. 2007). Likewise, infections with blood-feeding monogeneans lead to elevated predation on juvenile flounders (Shirakashi et al. 2008), and infections with parasitic barnacles increase predation on mud crabs by larger non-host crabs (Gehman and Byers 2017; Brothers and Blakeslee 2021). Preferential predation can also be more complex, as in the case of coho salmon that preferentially prey on pink salmon over chum salmon (Peacock et al. 2014). When both prey species are parasitized by sea lice, the prey preference increases, resulting in higher predation pressure on pink salmon and a reduced predation pressure on chum salmon (Peacock et al. 2014, 2015). Sea lice infections can thus lower the predation pressure on chum salmon, which in turn reduces the negative population-level impact of lice infections (Peacock et al. 2014, 2015).

While all the effects discussed above are largely density-mediated and result from the complete consumption of prey, predators can also affect parasites and diseases through side effects of the predation process itself. For example, when predators do not swallow prey in one piece but instead disrupt prey tissue and release bits and pieces

into the environment during consumption ("sloppy predation"), this may release infective stages of parasites into the water and lead to increased transmission (Cáceres et al. 2009; Lopez and Duffy 2021; Richards et al. 2023). This type of predator effect may be very common in marine systems given that predation by shrimps, crabs, sharks, and other marine predators usually releases significant amounts of blood, tissue, and viscera into the water. However, specific studies and quantifications of the ramifications for marine parasites and diseases are lacking. Apart from sloppy predation on whole prey individuals, "partial predation" of prey by predators (i.e., non-lethal attacks that may involve a small amount of consumption) can also affect parasites and diseases (Richards et al. 2023). One way in which this occurs is through inflicting wounds on prey that create entry portals for infections from the water column. Another way is the uptake of infectious stages by predators from prey during partial predation and subsequent transfer to other prey that can lead to increased transmission. Predators can thus act as mechanical vectors without becoming infected themselves This effect is probably very common given that a certain level of parasite specificity will often prevent infections of predators. However, in some cases predators can also act as alternate hosts for parasites from their prey, such that the distinction between mechanisms may not always be clear. For example, harbor porpoises can acquire lethal bacterial infections with *Neisseria animaloris* following traumatic injuries by predatory gray seals (Foster et al. 2019). These infections are most likely acquired from the gray seals, whose oral cavities generally harbor a diverse microbiome that includes potential pathogens for porpoises and can be found in wounds inflicted by seals (Gilbert et al. 2020). As not all of these bacteria are pathogenic for seals, the latter will function as vectors, reservoirs, or alternate hosts with respect to infections in harbor porpoises, depending on the type of bacteria. Similar complexities exist in corallivore fish that can transmit infectious agents among corals through partial predation and increase diseases in corals; however, the exact mechanisms are not always identified and can range from mechanical vectoring to alternate hosts as a function of the specific corallivore fish species (Clemens and Brandt 2015; Renzi et al. 2022). It has been suggested that it may be the physical degree of damage inflicted by different fish species (shallow vs. deep wounds) that dictates this for various corallivores (Nicolet et al. 2018).

Finally, parasites from infected hosts may sometimes survive gut passage in the predator and spread to new hosts or habitats as a result (Duffy 2009; Reilly and Hajek 2012; Morley 2022). Reports for gut passage survival from marine systems are few (Morley 2022), but given the diversity of records from terrestrial and freshwater systems, the phenomenon may be widespread and could facilitate both local transmission and large-scale dispersal. For example, eggs of didymozoid trematodes were reported to survive the gut passage in marine predatory fish that consume the flatfish hosts of the parasites (Lester 1980). Likewise, eggs of zoonotic nematodes (*Ascaris*, *Trichinella*) have been recorded to survive the gut passage of predatory marine fish, potentially contributing to dispersal and human exposure (Ono 1958; Bukina 2014). In some cases, entire hosts may survive the gut passage together with the parasites infecting them. For instance, hydrobiid snails can survive the passage through the intestines of shelducks feeding on tidal flats (Cadée 2011). The snails themselves are known to harbor a high diversity of trematode species (Thieltges et al. 2009a), such that the survival of hosts in the gut of shelducks may lead to increased long-distance dispersal of the parasites.

## 2.2. When Predators Consume Parasites

Predators can also affect parasites and diseases by consuming parasites. This can happen in various ways (Johnson et al. 2010), and studies on parasite-inclusive food webs from estuarine and coastal ecosystems suggest that between 30% and 66% of all trophic links in these food webs are predator–parasite links (Thieltges et al. 2013). Most of these links relate to cases where predators eat infected prey and consume parasites in the latter at the same time without becoming infected themselves ("concomitant predation"; Figure 12.3). In some cases, predation on infected hosts leads to "trophic transmission" when the predator serves as host for the parasites (Figure 12.3). Although concomitant predation appears very common in marine food webs, the magnitude of the phenomenon and its consequences for parasite populations and disease dynamics are largely unknown. Modeling work on sea lice infecting pink and chum salmon indicates

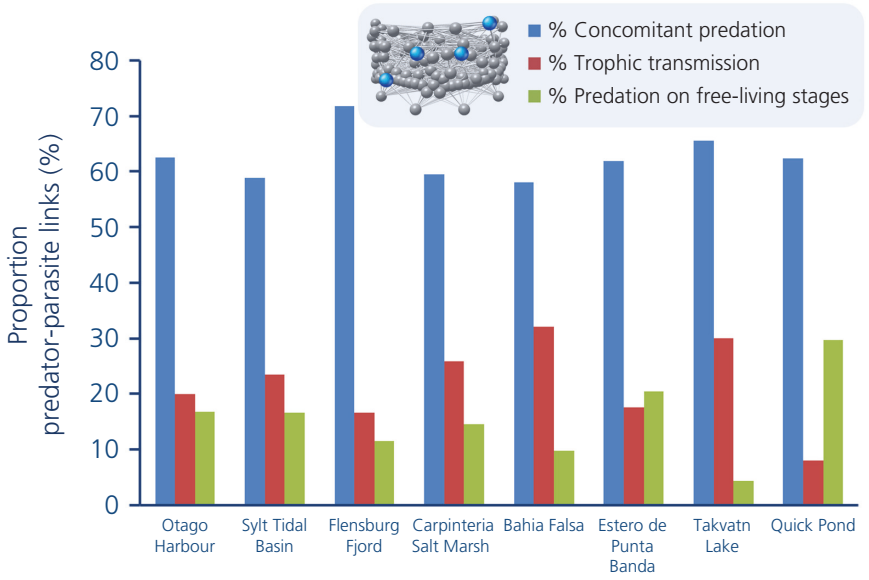


**Figure 12.3** Proportion of different types of predator–parasite links in estuarine and coastal food webs from Europe, New Zealand, and North America. For details, see text. Data from Thieltges et al. (2013).

that concomitant predation on both host species by coho salmon can affect both the parasite and the prey/host populations through the preferential predation on one of the two host species, as discussed in the previous section (Peacock et al. 2014). Sometimes concomitant predation can also interfere with trophic transmission, as in the case of the New Zealand cockle. As mentioned in the previous section, trematode infections in the feet of the bivalves impair their burrowing behavior so that they lay on the sediment surface, which leads to increased trophic transmission to bird definitive hosts. However, this parasite manipulation also makes cockles more prone to partial predation by benthic fish, which only consume part of the foot and thus reduce infection levels in the cockles (Mouritsen and Poulin 2003). Concomitant predation can probably often lead to similarly complex effects in many other systems, but this deserves more research.

Another common type of predation on parasites is "predation on free-living infectious stages" (Figure 12.3). Many aquatic parasites (especially macroparasites) have one or more infectious stages found in the environment which may be vulnerable to consumption, especially if they are similar in size to zooplankton or other regular prey. In parasite-inclusive food webs from estuarine and coastal ecosystems, this type of predation on parasites is thus relatively high, overall responsible for 4%–30% of all predator–parasite links (Thieltges et al. 2013). As in the case of concomitant predation, predation on infective stages of parasites does not lead to infections in the predator but instead removes infective stages from the environment, interfering with parasite transmission (Johnson et al. 2010; Johnson and Thieltges 2010). Many types of parasites are known to be affected by this type of predation (Thieltges et al. 2008). Probably the best-studied group are the motile larval (cercarial) stages of trematodes; a large range of vertebrate and invertebrate non-host predators are known to remove them from the environment through consumption (Koprivnikar et al. 2023). However, predation on free-living parasite stages also occurs in many other parasite groups and can affect other helminths, viruses, and bacteria (Thieltges et al. 2008; Burge et al. 2016; Welsh et al. 2020). While many different types of non-hosts, such as predators, grazers, and scavengers, can remove infective stages, the effect is particularly strong for filter feeders. They can filter large volumes of water and can thus capture a large number of infective stages from various parasite groups (Burge et al. 2016). For example, single Pacific oysters can remove

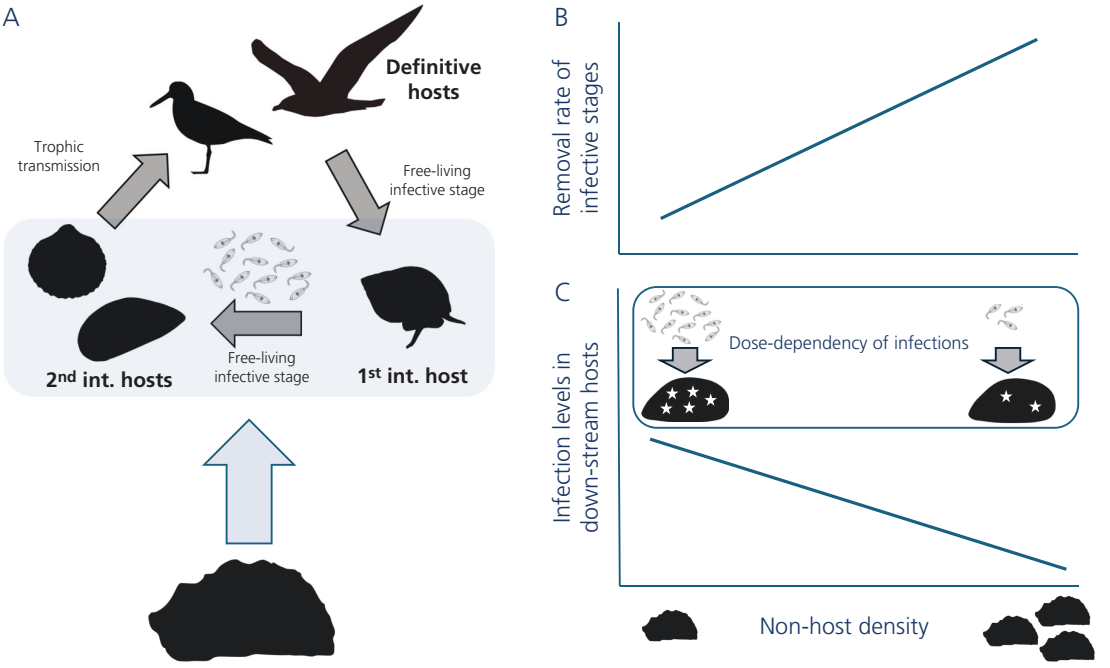


**Figure 12.4** (*A*) Example of the consumption of infective stages (trematode cercariae) by a non-host (oyster) that can lead to transmission interference between first and second intermediate (int.) hosts through (*B*) the removal of infective stages from the environment, which (*C*) results in lower infection levels in the downstream second intermediate hosts due to the dose dependency of infections in second intermediate hosts. Both removal rates of infective stages and infection levels in downstream hosts are correlated with oyster density. For details, see text.

large amounts of cercariae (>30%) from the water and thus reduce trematode transmission from first to second intermediate hosts (Figure 12.4A). This effect increases with oyster density (Figure 12.4B) (Welsh et al. 2024) as well as with oyster size (Welsh et al. 2019). As infections in downstream second intermediate hosts are dose-dependent (i.e., higher cercarial doses will lead to higher infection levels) (Liddell et al. 2017), the removal of cercarial stages from the environment by oysters leads to lower infection levels in downstream hosts, as observed in laboratory and field experiments (Figure 12.4C) (Thieltges et al. 2009b). As the effects of filter feeders on parasite transmission are often strong, they are also discussed as a potential measure to reduce disease risk in an aquaculture context (Burge et al. 2016).

Finally, predators can also consume parasites without consuming the host or the infective free-living stages by removing ectoparasites from the host. This type of predation on parasites is a form of grooming that can occur both intra- and interspecifically (Johnson et al. 2010). In marine systems, interspecific grooming in the form of cleaning symbioses has received considerable attention (Côté 2000; Vaughan et al. 2016). In these mutualistic interactions of small "cleaner" fishes and shrimps with larger fish "clients," ectoparasites are removed from the fishes' skin, which can significantly reduce parasite intensity on the cleaned fish and thus also potentially affect parasite populations as a whole. For example, cleaner fish specialized on ectoparasites can remove two-thirds of all gnathiid isopods that locally infect client fish (Grutter 1996, 2002, 2008). Also, various groups of shrimps have been reported to be involved in cleaning activities; however, this has been much less studied than in their fish counterparts (Vaughan et al. 2016). Like filter feeders, cleaner fish and shrimps have received increasing attention as potential biological control agents in aquaculture (González and de Boer 2017; Vaughan et al. 2018). However, in some cases cleaning organisms may also facilitate the transmission of parasites and increase disease risk, similar to the corallivore fish discussed in the previous section (Narvaez et al. 2021, 2022).

## 3. Non-consumptive Effects of Predators

Beyond consuming hosts or parasites, predators can also have non-consumptive effects on their prey that can affect parasites and diseases (Figure 12.1). These effects can occur when prey that serve as hosts to parasites, or parasites themselves, react to predation risk cues with changes in behavioral, physiological, or morphological traits in an effort to avoid or prepare for predation. These direct predator effects on host or parasite traits can lead to "trait-mediated indirect effects" that can alter parasite–host interactions and disease dynamics (Figure 12.1). In the following section, we outline the main mechanisms of non-consumptive effects of predators in this context and illustrate these with examples from marine ecosystems.

### 3.1. When Predators Scare Hosts

Many species that are preyed upon by predators can react to predation risk cues by inducing behavioral, physiological, or morphological trait changes to evade predation or mitigate its effects (Figure 12.5) (Lima 1998; Sheriff and Thaler 2014; Werner and Peacor 2003). These changes are phenotypically flexible (i.e., they are not genetically fixed but instead induced by predation risk cues). Some of these trait changes can be short-term responses (e.g., elevated levels of particular stress-associated hormones, such as glucocorticoids) in prey to increase vigilance and escape behaviors (Hawlena and Schmitz 2010). Other trait changes occur on longer timescales (e.g., in the form of increasing body size or growing thicker shells such as in bivalves exposed to crab predator cues) (Freeman and Byers 2006). Likewise, many organisms have a diverse set of defense mechanisms against parasites to avoid the usually negative fitness effects of infections (Figure 12.5). These anti-parasite defenses include various pre-infection mechanisms such as changes in movement, habitat choice, or foraging to avoid parasite contact (Behringer et al. 2018; Buck et al. 2018; Koprivnikar et al. 2021). In addition, hosts can defend themselves against parasites and their negative effects post-exposure by resistance mechanisms, such as immune responses and parasite removal (grooming), or by tolerance to infections, for example, through illness-mediated anorexia or behavioral fevers (Råberg et al. 2009; Sheldon and Verhulst 1996; Hite et al. 2020). These pre- and post-infection

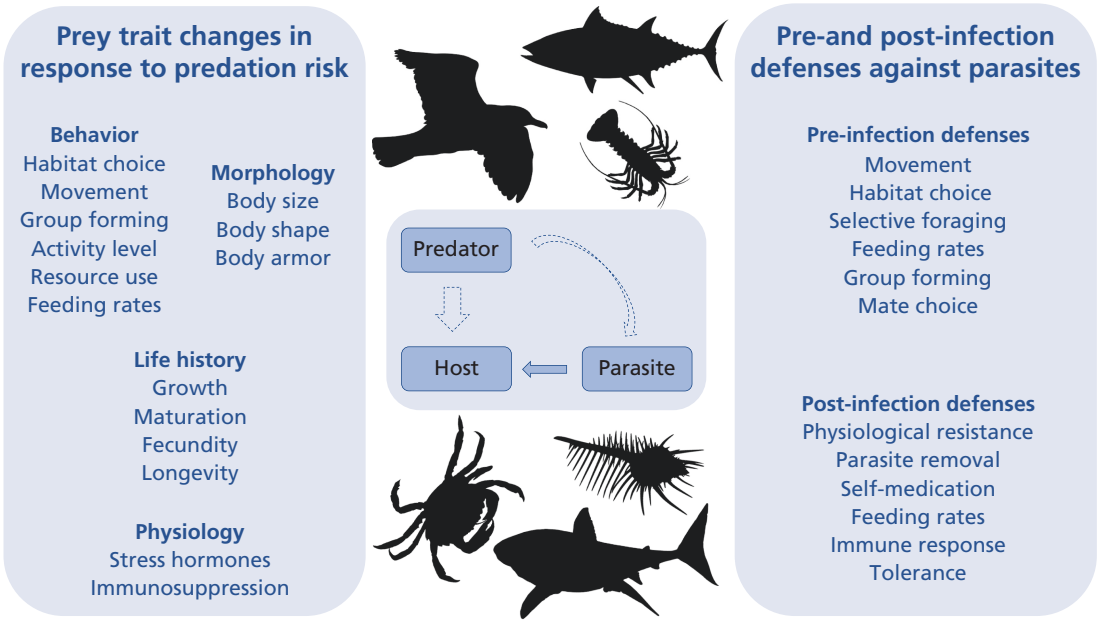


**Figure 12.5** Examples of prey trait changes in hosts in response to predation risk (*left*) that may affect pre- and post-infection defenses of prey that serve as hosts to specific parasites (*right*). This can lead to indirect effects of predators on parasite–host interaction (*middle*). Based on Thieltges et al. (2024).

defenses of hosts against parasites may be altered by some of the trait changes induced in response to predation risk cues. In addition, the suitability of prey as host for parasites may change through these trait changes. As a result, predation risk–induced trait changes in hosts can have indirect effects on parasite–host interactions and diseases (Figure 12.5) (Thieltges et al. 2024).

Trait changes in hosts in response to predation risk can affect parasite–host interactions at four steps in the life cycles of parasites: (1) during host encounter, (2) when parasites establish infections after host encounter, (3) infection persistence and effects on infected hosts, and (4) parasite propagule production (Thieltges et al. 2024).

Many hosts have developed strategies to avoid encounters with parasites, driven by the fact that such avoidance strategies are probably less costly than infection resistance or tolerance (Gibson and Amoroso 2022; Hart 1990; Hart and Hart 2018). Any trait changes in potential hosts induced by predation risk that alter the likelihood of parasite–host encounters can thus affect parasite–host interactions. A common predator avoidance strategy of prey is to move to different habitats; however, this habitat change may increase exposure of prey to parasites. For example, gastropods that move to the intertidal to avoid predation pressure by subtidal crabs and sea stars are exposed to trematodes and incur higher infection levels than in the subtidal (Byers et al. 2015). Seeking safety in numbers through shoaling is another common antipredator behavior of organisms in marine systems that may, in turn, increase exposure to directly transmitted parasites, as observed with sea lice in salmon farms (Krkošek 2010; Frazer et al. 2012). In contrast, in the case of indirectly transmitted parasites, shoaling could reduce individual host exposure and lead to lower infection levels (see Figure 12.2).

Small-scale, refuge-seeking behaviors in response to predation risk can also indirectly alter parasite–host encounter probabilities. For example, mussels residing in the matrix created by intertidal oyster beds experience lower crab and bird predation but at the same time endure increased infections with trematode in response to higher exposure to cercariae emerging from gastropods also living inside the oyster bed matrix (Goedknegt et al. 2020). However, the same mussel hosts experience lower infections with endoparasitic copepods as oysters probably dilute the settlement of infective stages from the water column (Goedknegt et al. 2020). This example shows that predation risk effects on parasite–host encounter probabilities are unlikely to be universal but rather context-dependent.

After the initial encounter of parasites and hosts, trait changes in hosts in response to predation risk can affect the establishment success of parasites. Exposure to stress factors, such as fear of predators, often initiates up-regulation of specific hormones (particularly cortisol and corticosterone); long-term elevations of these can in turn lead to immunosuppression that makes hosts more susceptible to infections (Romero 2004; Sapolsky et al. 2000; Adamo et al. 2017). Behavioral changes in hosts in response to predation risk can also affect parasite establishment. For example, sessile marine bivalves cannot easily move away from predators but instead quickly close their valves when they perceive predator presence to avoid predation. As a consequence of this behavior, filtration of the bivalves ceases. Infective stages of trematode parasites can then no longer infect the bivalves after encounter, and the predator avoidance response thus results in reduced infection levels in the bivalve hosts (Figure 12.6A, C) (Cornelius et al. 2023).

Trait changes in hosts in response to predation risk can affect the persistence and pathogenicity of infections by altering host physiological resistance or tolerance. While stress due to fear of predation can often lead to immunosuppression (especially over prolonged periods), it can sometimes also increase immune function (Adamo et al. 2017; Duong and McCauley 2016). This, in turn, can affect the resistance of hosts to infections and could also reduce the persistence of infections. Hence, the indirect effects of predation risk responses on parasites may not always be beneficial for parasites. When parasites are able to persist in hosts, trait changes of their host in response to predation risk may alter the effects of parasites on their hosts. For example, in marine gastropods infected by trematodes, predation risk cues can amplify personality effects, in the form of high intra-individual repeatability of behaviors such as climbing height, time spent out of water, and time spent out of refuge, which can all be considered to be related to predation risk (Salerno et al. 2023).

Finally, parasite propagule production can be affected by predation risk–induced trait changes of their hosts. Stress responses of hosts under

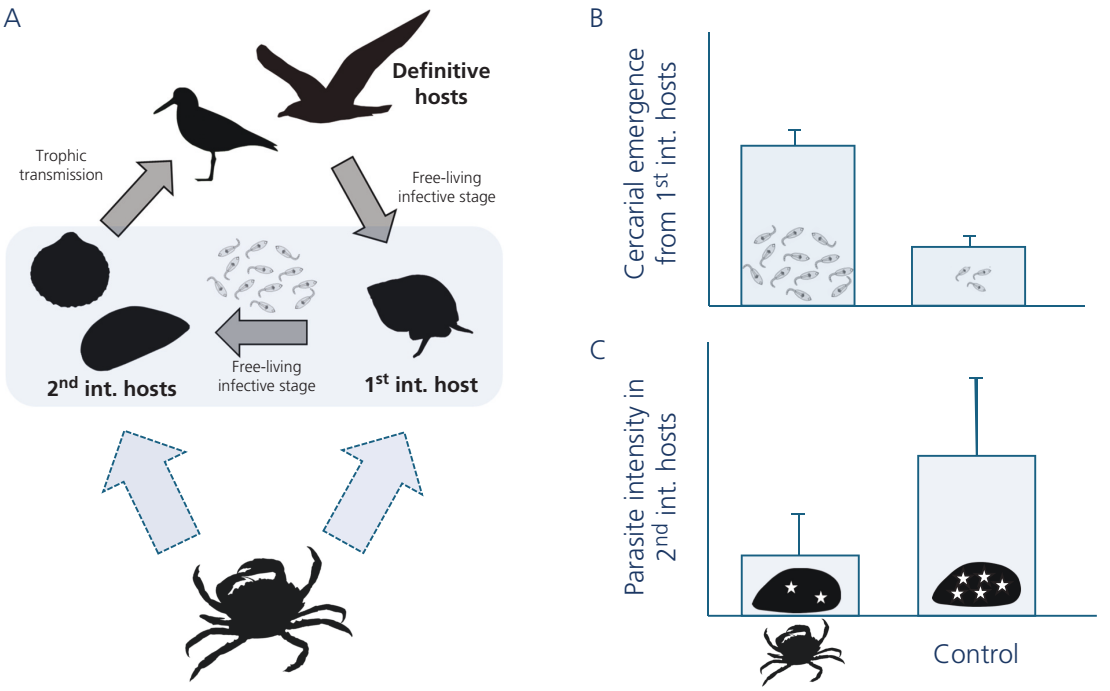


**Figure 12.6** (*A*) Example of how crab predation risk can affect parasite transmission by (*B*) altering traits in first intermediate (int.) hosts leading to higher cercarial emergence and (*C*) inducing trait changes in second intermediate hosts that lead to reduced infection levels in the presence of crab cue. For details see text. Based on Cornelius et al. (2023).

perceived predation risk could compromise the condition of hosts so that fewer resources are available for parasite propagule production, leading to lower transmission. In contrast, the opposite effect can also occur when propagule production is elevated under predation risk. For example, the emergence of infective stages of trematodes from their intertidal gastropod hosts increases when crab predation cues are present (Figure 12.6B) (Cornelius et al. 2023). The latter causes the gastropods to be more active, and the associated elevated metabolism may facilitate increased propagule production.

### 3.2. When Predators Scare Parasites

In principle, parasites might also respond to predator presence to avoid predation if they regularly experience consumption by non-hosts (see above; Johnson et al. 2010; Johnson and Thieltges 2010; Koprivnikar et al. 2023). However, to the best of our knowledge, this has not been investigated in marine or other ecosystems to date. The only reasonable line of support right now may be that the circadian rhythms of trematode cercarial emergence (Combes et al. 1994) could reflect avoidance of predation as well as maximizing encounter with the next host. It is not unlikely that parasites may be able to perceive predation cues given that many free-living infective stages of parasites possess a diversity of sensory mechanisms (e.g., mechanisms responsible for chemical taxis, phototaxis, geotaxis) to facilitate host finding (Chaisson and Hallem 2012; Haas 2003). In the case that parasites are indeed able to perceive predation cues and mount avoidance responses, this could be expected to lead to similar indirect effects on parasite–host interactions as the other consumptive and non-consumptive effects of predators (Figure 12.1).

## 4. Knowledge Gaps and Future Research Areas

While various consumptive and non-consumptive effects of predators on parasites and diseases have been reported from marine systems, as discussed above, research on the matter is still in its infancy. Observational and experimental studies are needed

to identify and quantify the diversity of predator effects in specific parasite–host systems. In particular, potential non-consumptive effects of predators on parasites and their infective stages deserve more research effort as it is currently completely unknown whether parasite infectious stages can perceive predation cues and exhibit anti-predator defense mechanisms. Our knowledge is still very limited also for non-consumptive effects on parasite–host interactions through predation risk–induced trait changes of hosts (Thieltges et al. 2024). In addition, there are currently clear taxonomic and habitat biases in research as most of our knowledge stems from invertebrates (mollusks, crustaceans) in coastal and intertidal habitats. Hence, extensive progress in the field could be gained from expanding the scope to open pelagic habitats and/or other animal taxa and from conducting experimental studies that identify non-consumptive effects via predation risk–induced trait changes in hosts or in parasites.

As most parasite–host systems are probably subjected to various consumptive and non-consumptive effects of predators at the same time, it will be relevant to quantify the relative importance of the different mechanisms for specific parasite–host systems. In some cases, consumptive effects of predators on parasite–host interactions are likely to dominate, in particular when hosts or parasites do not show strong trait changes in response to predator cues. In contrast, strong trait changes in hosts or parasites in response to predator cues could result in non-consumptive effects overriding consumptive effects. Disentangling the relative importance of consumptive and non-consumptive effects in specific parasite–host systems will require more complex experimental approaches, which may pose challenges for some systems (e.g., ethics around experiments involving vertebrate predators).

Related to the relative importance of consumptive and non-consumptive effects of predators on parasite–host interactions is the question of whether the individual-level responses of parasites and hosts to predators observed in small-scale experiments also scale up to population-level consequences (i.e., whether they will ultimately affect host and disease dynamics). While it is easy to see that consumptive effects of predators may indeed affect population dynamics of hosts and parasites by reducing their density, this is less obvious for non-consumptive effects of predators because these only affect trait expression in parasites or hosts but not their densities. In general, evidence for population-level consequences of non-consumptive predator effects is poor, not only with respect to parasite–host interactions but also for predation risk effects on predator–prey and herbivore–plant interactions (Sheriff et al. 2020). One can speculate that trait changes with strong, direct consequences on growth, fecundity, or survival of parasites or their hosts have a greater chance of affecting populations relative to those with only subtle effects (Sheriff et al. 2020; Thieltges et al. 2024). The population-level importance of non-consumptive effects can also be expected to depend on parasite pathogenicity and the intensity dependence of the pathology for a specific parasite–host system (Thieltges et al. 2024). The strongest population effects can be expected when parasites exhibit high pathogenicity and when pathology is strongly intensity-dependent. In these cases, changes in host encounter, parasite establishment, or infection persistence due to predation risk–induced trait changes are likely to lead to population-level effects. A combination of experimental and modeling approaches will be needed to explore the population-level importance of consumptive and non-consumptive predator effects on parasites and diseases. In addition, it will be interesting to explore the extent to which anti-parasite and anti-predator defenses may be in conflict with one another. If so, this could result in poor prey/host outcomes if one type of natural enemy becomes more dominant than the other.

Finally, while these consumptive and non-consumptive predator effects act on ecological timescales (i.e., on parasite and host populations and disease dynamics), changes in predation pressure over time may exert selective pressures on parasites to adapt in the long run. This evolutionary timescale may be relevant in regard to range shifts of marine predators in response to climate change (Pinsky et al. 2020; Braun et al. 2023) that will introduce new selective predation pressures for hosts and parasites. This could affect parasites not only through host density changes but also through alterations of host species richness and evenness. For example, an introduced or newly stocked predator may preferentially consume one type of prey such that it changes community composition and thereby the balance of competent versus less competent hosts (biodiversity-dilution effects).

In addition, future temperature increases in the ocean are likely to intensify predation pressure (Schemske et al. 2009; Englund et al. 2011; Ashton et al. 2022), which may, in turn, also alter ecological and evolutionary responses of parasites and hosts. Selective landscapes of predation pressure can also change through conservation measures and increasing rewilding initiatives, especially when reintroducing predators to marine systems (Pettorelli et al. 2019; Clover 2023). The opposite effect of relaxed predation pressures may result from large-scale predator declines in the ocean, mainly due to fisheries (Myers and Worm 2003; Myers et al. 2007). The removal of top predators from the oceans not only reduces predation pressure and weakens trophic cascades but can also alter the magnitude of non-consumptive effects from predators (Heithaus et al. 2008). These indirect effects will also affect the selective pressures for parasites and hosts.

In conclusion, this chapter discussed the diversity of mechanisms through which consumptive and non-consumptive effects of predators can affect parasite–host interactions and disease dynamics in marine ecosystems. At the same time, it highlighted large gaps in our understanding of the magnitude and relative importance of these effects for marine parasites and diseases. A holistic approach, combining empirical data, experimentation, and modeling scenarios, is most promising to further explore consumptive and non-consumptive effects of predators on marine parasites and diseases.

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