Limnol. Oceanogr., 44(3, part 2), 1999, 925–931

How environmental stress affects the impacts of parasites

Kevin D. Lafferty and Armand M. Kuris
Marine Science Institute and Department of Biological Sciences, University of California, Santa Barbara, California 93106

Abstract

Parasites occur in nearly every population. They often interact in complex ways with other stressors. In some cases, the interaction may lead to a disproportionately negative effect on the host population. In other cases, the stressor may ameliorate the effects of parasitism. Here we illustrate intersections of four types of environmental stressors with infectious diseases. First, pollutants may increase parasitism by increasing host susceptibility or by increasing the abundance of intermediate hosts and vectors. Pollutants can also decrease parasitism if infected hosts suffer differentially high mortality, parasites are more susceptible to pollution than their hosts, or if pollutants negatively affect intermediate hosts or vectors. These effects vary depending on the particular parasite and pollutant that interact. Second, habitat alterations such as impounding water or development can affect both intermediate host and vector populations such that the abundance of their attendant parasites is either increased or reduced. Third, fisheries can impact populations already stressed by disease. However, they may act to lower the density of a host population below the threshold for sustained transmission to such an extent that the parasite population can no longer persist. Fourth, introduced species may introduce new diseases to susceptible native populations or they may gain an advantage if they invade without the parasites from their native range. The complexity and ubiquity of these interactions are good arguments for considering parasitism when evaluating stressors of aquatic systems.

In this review, we examine what may happen when stressors to a population overlap with parasitism—a ubiquitous stressor. There are several possible qualitative outcomes when parasites interact with other stressors. The most obvious is that some stressors may make hosts more susceptible to parasitism. Related to this are stressors that disproportionately increase mortality rates of infected hosts. Less obvious are stressors that decrease parasitism by killing parasites directly, by reducing the parasite's intermediate hosts or vectors or otherwise degrading their ease of transmission. By the same logic, some stressors may act to increase the abundance of intermediate hosts or vectors. Epidemiological theory points to another interaction: if a stressor reduces the abundance of a host, its parasites may not be able to persist. Finally, if the stressor is an introduced species, it may introduce new parasites to the native species it competes with or preys upon. Alternatively, and probably more commonly, an introduced species might gain a competitive advantage by being less susceptible to the parasites that are already present (Kuris and Gurney 1997; Lafferty et al. in prep.).

Water quality

Because many factors can alter water quality, it is unlikely that a generalization about the interaction between water quality and parasitism will emerge (Khan 1991; Khan and Thulin 1991; Poulin 1992; MacKenzie et al. 1995; Lafferty 1997). However, one obvious prediction is that pollutants may reduce the immunological capabilities of hosts, rendering them more susceptible to some parasites (*see* McDowell et al. 1999). Gill ciliates of fish are a good example of this effect. Intensities or prevalences of ciliates increase with oil pollution (Khan 1990), pulp mill effluent (Lehtinen 1989; Axelsson and Norrgren 1991; Khan et al. 1994), industrial effluent (Dabrowska 1974; Overstreet and Howse 1977; Vladimirov and Flerov 1975), and thermal effluent (Esch et al. 1976; Nilsen 1995). This appears to be due to an increase in susceptibility because toxic conditions compromise a fish's immune system (Khan 1990). The pathology that seems most directly causal to the success of these with ciliates is a toxicant's ability to impair mucus production—a fish's main defense against gill parasites (Khan 1987).

In contrast to the above example, altered water quality may improve conditions for parasites if their host's density increases (*see below*). Eutrophication and thermal effluent often raise rates of parasitism because the associated increased productivity can increase the abundance of intermediate hosts. Beer and German (1993) described how eutrophication improved conditions for snails that, when combined with a thriving population of urban-adapted mallards (escaped from local farms), fueled the life cycle of the digene, *Trichobilharzia ocellata.* This avian blood fluke causes outbreaks of swimmer's itch (schistosome dermatitis). Similarly, Valtonen et al. (1997) described how eutrophication among lakes and over time was associated with greater overall parasite species richness in two fish species.

Eure and Esch (1974) found that acanthocephalans were more common near thermal effluent because intermediate hosts were able to retain high densities in winter. Sankurathri and Holmes (1976) showed that thermal effluent altered the normal seasonal dynamics of a digenean community, essentially providing summer conditions so that transmission could continue uninterrupted year-round. Warm temperatures prevented icing over of the lake and fueled primary productivity. The warm water and increased vegetation extended the breeding season and provided extra food and habitat for snail populations that served as intermediate hosts for several species of digenes. Because it was ice-free, the area attracted migrating birds—the definitive hosts of most of the digenes. In addition, the warm water was unfavorable to a normally abundant oligochaete worm that is commensal with the snail host and interferes with the free-living stages of the digene's life cycle. The main consequence of this was an increase in the total prevalence of digenes in snails and (presumably) other hosts.

The effect of certain pollutants varies strongly among parasites. Crude oil increases parasitism by some species of ciliates, nematodes, and monogenes, yet decreases parasitism by acanthocephalans and digenes (Khan 1987; Lafferty 1997). Perhaps the negative effects of crude oil on some invertebrates that serve as intermediate hosts for these parasites reduced their abundance. The effects of sewage sludge are even more difficult to predict (Lafferty 1997). Acid rain may also affect parasites and their hosts in complex ways (*see* Leavitt et al. 1999; Niyogi et al. 1999). Marcogliese and Cone (1996) found that parasite species richness in yellow eels (*Anguilla rostrata*) from Nova Scotia was 4.0 per eel at buffered sites, about 2.5 per eel at moderately acidified sites, and 2.0 per eel at the most acidified sites. Separating this effect by parasite group indicated that while the prevalence of monogenes and digenes (and their snail hosts) decreased with acidity, acanthocephalans may have increased with acidity and tapeworms and copepods were relatively unaffected (Lafferty 1997).

Toxic chemicals and trace metals have a relatively consistent negative effect on intestinal helminths (Lafferty 1997). These pollutants may be analogous to a drug that kills the parasite but does not kill the patient. Selenium, for example, is more toxic to tapeworms than to their fish hosts (Riggs et al. 1987). A pollutant may also kill sensitive free-living stages of the parasite (Evans 1982). For example, trace metals in sewage sludge reduced survival of free-living cercariae and miracidia, leading to a lower prevalence of the digene, *Zoogonoides viviparous*, in snails (Siddall and Des Clers 1994). In other cases, parasitic infection may make the host more susceptible to toxins (Guth et al. 1977; Stadnichenko et al. 1995). Brown and Pascoe (1989) found that exposure to cadmium killed 94% of the amphipods infected with larval acanthocephalans compared to a 14% mortality rate of uninfected amphipods. Although the effect of trace metals is often to reduce parasitism, this does not imply that trace metals provide a benefit for aquatic populations.

Habitat alteration

Altering habitats can favor vectors and intermediate hosts and lead to an increase in human diseases. Several environmental disasters ensued after Egypt built the Aswan Dam in 1971 to provide inexpensive electricity for city dwellers and irrigation for greatly expanding farm land (Heyneman 1979). The annual floods along the Nile ceased, starving the river farmlands of fertile sediments, the Mediterranean sardine fishery collapsed, and most segments of the river slowed and offered better habitat for the snails that serve as the intermediate host for the flukes that cause human schistosomiasis. The snails and their flukes spread along the river and around the newly formed Lake Nasser and its associated irrigation canals, causing serious human morbidity. This epidemiological disaster has occurred repeatedly including Lake Volta in Ghana and most recently and spectacularly at Richard Toll in Senegal in 1988 (Gryseels et al. 1994), producing perhaps the most intense focus of transmission known. At some impoundments it is likely that the cold, clean running water at the dam outflow has created ideal conditions for blackfly larvae and attendant river blindness because the adult blackflies serve as filarial vectors.

Deforestation, damming, fish farming, and rice farming have increased malaria transmission. Malaria, one of the world's most important human diseases, increases with deforestation because this allows dense human habitation and also creates many puddles and ditches that make good habitat for some species of the *Anopheles* mosquitoes which serve as vectors. Some of these habitat alterations are ancient. The hunter-gatherer life style probably was not conducive to high levels of malaria. Several thousand years ago, Malayan swidden agriculture became widespread in Africa. The consequent deforestation and attainment of dense human populations provided much better conditions for transmission of malaria (Weisenfeld 1967; Desowitz 1991).

In tropical Africa, South America, and Asia, a variety of relatively recent habitat alterations have intensified the conditions for malaria transmission. For example, the Transamazon highway blocked numerous streams, creating small lakes favorable for mosquitoes. Mosquitoes then spread malaria from infected colonists to the rest of the population (Smith 1981); notably, the indigenous peoples suffered disproportionately. Solutions to this deadly problem exemplify the complexity of interactions between multiple stressors. The most effective approaches to mosquito control, pesticides and environmental engineering, serve as powerful stressors too. One of Benito Mussolini's biggest accomplishments was to rid the Pontina Valley of malaria by building canals to drain the marshes. Eventually, industrial pollution cleared out what mosquitoes remained. Other economically strong countries have followed suit and drained their swamps. This did successfully control malaria in key areas such as Israel and the Panama Canal Zone (Kitron and Spielman 1989; Desowitz 1991) but not without other severe ecological impacts.

Habitat alterations can also lead to decreases in parasitism. Populations of the horn snail, *Cerithidea californica*, in disturbed areas have a lower prevalence of digenes than do snail populations in adjacent undisturbed sites (Lafferty 1993). This is probably because birds—the definitive hosts—are less abundant in disturbed areas (Kuris and Lafferty 1994; Lafferty 1997). Others have also speculated that the prevalence of digenes declines with the degree of habitat degradation (Robson and Williams 1970; Pohley 1976; Hughes and Answer 1982; Granovitch 1992). Cort et al. (1960) pioneered this concept. They found that larval digene species richness had declined over a two-decade period in some Michigan lakes and was associated with an increase in human disturbance and a reduction in the shorebird population. Keas and Blankespoor (1997) recently resampled these sites and found continued declines in the digene fauna. However, a few digene species, presumably those that parasitize birds associated with human development, had increased. Because bird feces, beyond dispersing parasite eggs, are rich nutrient sources, spatial alteration of the defecation patterns of bird populations greatly alters aquatic communities in other ways (Kitchell et al. 1999).

Fisheries

Parasite transmission strongly interacts with fisheries. Interactions may be positive or negative with respect to the fishery. Whether inadvertent or not, fisheries manage the host parasite dynamic as well as the yield (Kuris and Lafferty 1992).

Fishing has clearly stressed populations of many species across the globe. Harvested species may also suffer from pathogenic parasites and diseases, making fisheries management very challenging. Parasites may contribute to the collapse of a fishery, or a fishery may act to eliminate a parasite from a fished stock. In addition, fisheries can spread parasites by discarding infected animals at sea (Petrushevski and Schulman 1958), as happens with the dinoflagellate that causes bitter crab disease in tanner crabs (Meyers et al. 1989).

Parasites that decrease host density have strong potential to synergistically exacerbate fishing mortality. We investigated the potential for interaction between parasites and fisheries when California's black abalone (*Haliotis cracherodii*) fishery, once the most lucrative abalone fishery in the state, began to collapse in the late 1980s. In a few years, densities of this once abundant species declined to 1% of historical values. The prevailing hypothesis at the time was that up to 16 stressors, including El Niño, pollution, loss of kelp beds, competition from sea urchins, fishing, and a disease that caused withering of the foot led to the collapse (Davis et al. 1992). We analyzed the spatial-temporal pattern of the spread of this marine mass mortality episode and deduced that there was empirical support only for an infectious disease process caused by a then unknown etiological agent (Lafferty and Kuris 1993). The disease, which spread throughout the Channel Islands from a central focus, has continued to expand its range up the mainland coast (Altstatt et al. 1996) and appears to be a rickettsia-like organism (Gardner et al. 1995). The withered foot syndrome did seasonally interact with warm temperatures that also may have stressed abalone populations (Lafferty and Kuris 1993). The California Department of Fish and Game eventually elected to close the fishery hoping that the surviving individuals might be resistant to the disease and provide sufficient broodstock to restore the dwindling populations.

Stepping further back in the history of human impact on the black abalone, it is pertinent that its historic abundance dated only to the 19th century and was a direct result of two early stressors. When the Spanish mission system evacuated Native Americans from the Channel Islands and Russian fur traders exterminated sea otters, the chief predators of the black abalone were removed. Under these conditions, black abalone on the Channel Islands attained extraordinary densities for an abalone, often being stacked upon each other in the mid-intertidal zone habitat. Peak host densities provided perhaps the single most important precondition to facilitate the epidemic spread of an infectious disease, particularly of a lethal but inefficiently transmitted agent. This was precisely the scenario for the spread of withered foot syndrome among black abalone and its resultant precipitous population decline that resulted in the long-term loss of a major fishery.

Theoretically, commercial fisheries that reduce abundance of a fished species should also reduce rates of parasitization on that species. This supposition comes from classic epidemiological theory. Kermack and McKendrick (1927) argued that there is a minimum density of hosts, or a ''host threshold,'' below which a disease would not invade or persist because transmission to new hosts must be greater than the loss of infected hosts by death or recovery. Low density populations may have too few host interactions for sufficient transmission to occur. Once a disease invades a host population, it can persist only as long as the density of susceptible hosts is high enough to maintain transmission (Hamer 1906; Black 1966). Models by Dobson and May (1987) showed that a fishery can ''fish out'' a parasite if the fishery takes the population below the host density threshold for the parasite. This can be an acceptable management strategy if the host threshold density is higher than the density for maximum sustainable yield (unlikely) or if host population recovery is anticipated from long-distance dispersal.

Kuris and Lafferty (1992) generalized the Dobson and May (1987) approach showing that fishing out a parasite is possible only for host–parasite interactions in which the parasite has a recruitment system that is relatively closed compared to the open recruitment of its host. One such fishery may be the red king crab (*Paralithodes camtschatica*) fishery in some areas of Southeast Alaska where nemertean worms living in the brooded egg mass of the female king crab consumed nearly all the eggs. These nemerteans evidently developed rapidly and were probably transmitted over very short distances among the aggregated female crabs in closed embayments and fjords with sills (Kuris et al. 1991). Since the king crab larvae are long lived and disperse widely, the heavily infested crab populations in certain fjords were candidates for the adaptive management of the fishery to fish the crabs intensively (including females) to break the host threshold density and remove this major source of crab mortality (Shields et al. 1990).

Long-term whole-lake experiments have shown that intensive fishing pressure significantly decreased the age-specific prevalence of the tapeworm, *Triaenophorus crassus*, in whitefish, *Coregonus lavaretus* (Amundsen and Kristoffersen 1990; *see also* Lawler 1970; Poole 1985). However, the manipulations did not reduce another tapeworm, *Diphyllobothrium ditremum*, probably because *T. crassus* uses pike (*Esox lucius*), which the investigators also fished intensively, as a definitive host, while *D. ditremum* is a parasite of birds. This has important economic implications as the visible cysts of *T. crassus* reduce marketability and, as a result, seriously impact whitefish fisheries in Canada and Scandinavia (Miller 1952; Vik 1965).

A similar example exists for marine fisheries. The presence of larval nematodes—sealworms—devalues the market price of cod and other fish. Cod serve as a second intermediate host for nematodes that normally use seals as definitive hosts. In addition to affecting the appearance of fillets, the worms can cause serious pathology if humans eat undercooked fish. The expense of removing nematode larvae from fillets may not be economically viable in areas where the parasite is common. Des Clers and Wootten (1990) concluded that if fish or seal populations drop below threshold

levels, the nematode population will go extinct, providing a large economic gain for the industry. However, because many fish species other than cod can serve as intermediate hosts, fishing to crash the transmission threshold would probably not eliminate the worm. The remaining option is to cull the seal population (Odense 1978; McClelland et al. 1983). This strategy, although conflicting with other cultural values, seems to be successful where it has been tried. The sealworm situation is becoming a more serious problem because in the U.S. and elsewhere regulations such as the Marine Mammals Protection Act of 1972 fully protect pinniped populations, which have soared. Not surprisingly, so have the prevalence and intensity of larval nematodes (Chandra and Khan 1988), with increasing risk to human consumers of fish.

The scallop fishery in Post Phillip Bay, Victoria, Australia, provides an interesting example of inadvertent parasite management by a fishery. Before they were heavily fished, most large scallops were infected with a bucephalid trematode parasitic castrator (Sanders 1966). With the development of a major fishery, parasitization rates plummeted (Sanders and Lester 1981; G. Parry pers. comm.) Either or both of two fishery interactions could be responsible: larger older scallops contained the bulk of the trematodes and these hosts have been fished out; the leatherjacket filefish is the final host for these worms. It is now fished so fewer trematode eggs are available to infect the scallops. The recent closure of the scallop fishery, but not the leatherjacket fishery, should enable the relative contributions of these factors to be estimated.

Avoidance of heavily infested stock, a common practice where the parasite directly affects market quality, can also exacerbate the host-parasite dynamics so as to intensify infection levels. By concentrating the fishery on the unparasitized stock, the parasites are, in effect, protected by the fishery. Thus, the threshold for transmission between metapopulations is lowered by artificially conserving the heavily infected stocks while the total host population is otherwise being sharply reduced. Examples where this could be occurring include the fisheries affected by sealworms (Young 1972; McClelland et al. 1983) and the Alaskan tanner crab fisheries afflicted with bitter crab disease (Meyers et al. abstr., 1990; Kuris and Lafferty 1992).

Some theory suggests that fishing out a parasite might not always be effective. For example, crab fisheries are often managed to protect crab brood stock by releasing trapped females. This inadvertently protects some types of parasites, causing such parasites to have a greater than expected effect on the crab population (Kuris and Lafferty 1992). It is also important to consider the scale of the fishery because fishing at a local scale will have little noticeable effect on a parasite or host that disperses widely (Kuris and Lafferty 1992).

Introduced species

Introduced species can affect native ecosystems strongly (Cohen and Carlton 1998) and often seem to be associated with other stressors (Ruiz et al. 1999). Exotic species may bring their parasites with them, intensifying their impacts if native species are susceptible to the introduced parasites. Scandinavians who colonized the Great Lakes region in the 19th century provide an interesting example. The colonists, many of whom were fishermen, brought the fish tapeworm, *Diphyllobothrium latum,* in their intestines and introduced it to North American fish populations. This tapeworm then became widespread in human and fish populations (Desowitz 1981). Although pathology in humans is rarely more than the shock of being infected by a worm 10 m long, fish growth, reproduction, and survival can be significantly impacted by larval tapeworms (Kuris 1997). This scenario is most likely in places where the parasite community is depauperate. Font and Tate (1994) found that native freshwater fish of the isolated Hawaiian Islands did not suffer from parasitism unless exotic fish had invaded their habitats and introduced exotic parasites.

Aquaculture and mariculture have recently become economically more attractive because fisheries have been mismanaged. Unfortunately, the globalization of these industries, coupled with woefully inadequate quarantine standards, has led to the repeated introduction of infectious diseases that are highly virulent to native organisms. Some recent examples include the myxozoan agent of whirling disease, brought from Europe with brown trout, spread through trout hatcheries, and now widespread in drainages of the western U.S. where it is devastating native salmonids (Hoffman et al. 1962; Hewitt and Little 1972). Bonamiasis, a protistan disease of oysters, has been distributed from its geographic origin in the western U.S. by the widespread movement of the European flat oyster, *Ostrea edulis* to Europe where it devastated the western European oyster fisheries (Chew 1990; Hedgecock 1993). Perhaps the most recent and potentially most serious of these introductions are several viruses of penaeid shrimps; these viruses build up under the high density of farming and seem to be spreading to native penaeids. This build up is associated with the increasingly predominant use of susceptible species, such as *Penaeus monodon,* throughout the world (*see* Lightner et al 1992*a*,*b*).

Occasionally an introduced species suffers markedly from native parasites. A good example is the pathology induced by larval trypanorhynch tapeworms (which mature in native sharks) in the introduced striped bass (*Morone saxatilus*) populations in San Francisco Bay (Sakanari and Moser 1990).

Introductions for freshwater fisheries enhancement or for aquaculture (marine and freshwater) generally involve entry of adult organisms with their attendant parasites. However, the most common mechanism for the introduction of marine species is as larvae via ballast water (Carlton 1985). Consequently, most introduced species arrive without their parasites, conferring upon them a competitive advantage over native species (Lafferty and Kuris 1996; Lafferty et al. in prep.). Calvo-Ugarteburu (1996) found that in South Africa the invasive mussel, *Mytilus galloprovincialis,* was not parasitized by trematodes which negatively affected the reproductive output and growth of the native mussel, *Perna perna.* Another example is the introduced European green crab, *Carcinus maenas.* We have sampled the crab's parasites in its native (Sweden, Netherlands) and introduced regions (California, eastern coast of the U.S., South Africa, Japan,

Victoria, and Tasmania). Nemertean worms, parasitic barnacles, larval acanthocephalans, and larval digenes were strikingly less common where the crab has been introduced than where it is native. This finding may explain why green crabs usually perform better in introduced locations (Torchin et al. unpubl. data), which is not to say that introduced species remain completely unparasitized. We also found that a native nemertean worm was able to colonize the European green crab in California by transferring from the native shore crab, *Hemigrapsus oregonensis* (Torchin et al. 1996). Studies show an accumulation of native natural enemies as a function of residence time of the introduced species (Blaustein et al. 1983; Cornell and Hawkins 1994), suggesting that the parasite-free advantage gained by a introduced species may lessen over time.

Conclusion

We have provided a number of examples of the interaction between parasites and other stressors in aquatic environments. Each parasite can interact differently with each stressor (Lafferty 1997). Although parasites may increase in association with other stressors, it seems that just as often, if not more often, they decrease. This is probably because many parasites have complex life cycles that make them susceptible to a wider range of disturbances than those to which most free-living species are subjected. Each host and life history stage of a parasite will usually have its own unique direct and indirect responses to a stressor. Thus, the probability that the overall impact with other stressors will be negative for the parasite is additive. A negative impact at but one of the stages in a host, or in transmission to that host, becomes increasingly likely the more complex the life cycle. Intensification of parasite effects will be expected most often when a new and more efficient host enters a system or when a host population is for some reason augmented, hence providing an expanded resource base for a parasite. For parasites with simple (one host) life cycles, the interactions are less predictable. If the stressor has a strongly negative effect on host defenses, then it will intensify the impact of the parasite. Such an effect will occur when stressors bring novel exogenous parasites to a native fauna. On the other hand, for the same reasons that stressors, by definition, degrade environments, reduce species diversity, or decrease host populations, their impact on parasites will also often be negative. The consequence of these complex interactions is always that alteration of infectious disease dynamics can have substantial impacts on a host community.

It is important to consider the full range of potentially interacting components of a system because the various direct and indirect effects that can impinge on a parasite's complex life cycle can make the effect of a stressor quite complex. Complexities aside, parasites can and do interact with other stressors. Parasites are also ubiquitous, suggesting that they require consideration if we desire a general understanding of anthropogenic impacts to aquatic ecosystems. Likewise, studies of parasite ecology should take into account how stressors in the form of changes in water quality, habitat alterations, fisheries, and exotic species affect parasite communities.

References

ALTSTATT, J. A., AND OTHERS. 1996. Recent declines of black abalone *Haliotis cracherodii* on the mainland coast of central California. Mar. Ecol. Prog. Ser. **142:** 185–192.

AMUNDSEN, P.-A., AND R. KRISTOFFERSEN. 1990. Infection of whitefish (*Coregonus lavaretus* L. s.l.) by *Triaenophorus crassus* Forel (Cestoda: Pseudophyllidea): A case study in parasite control. Can. J. Zool. **68:** 1187–1192.

AXELSSON, B., AND L. NORRGREN. 1991. Parasite frequency and liver anomalies in three-spined stickleback, *Gasterosteus aculeatus* (L.), after long-term exposure to pulp mill effluents in marine mesocosms. Arch. Environ. Contam. Toxicol. **21:** 505–513.

BEER, S. A., AND S. M. GERMAN. 1993. Ecological prerequisites of worsening of the cercariosis situation in cities of Russia (Moscow Region as an example). Parazitologiya **27:** 441–449.

BLACK, F. L. 1966. Measles endemicity in insular populations: Critical community size and its evolutionary implication. J. Theor. Biol. **11:** 207–211.

BLAUSTEIN, A. R., A. M. KURIS, AND J. J. ALIO. 1983. Pest and parasite species-richness problems. Am. Nat. **122:** 556–566.

BROWN, A. F., AND D. PASCOE. 1989. Parasitism and host sensitivity to cadmium: An acanthocephalan infection of the freshwater amphipod *Gammarus pulex*. J. Appl. Ecol. **26:** 473–488.

CALVO-UGARTEBURU, G. 1996. Parasitism and invasive species: An ecological study of mussel populations. Ph.D. thesis, Rhodes Univ.

CARLTON, J. T. 1985. Transoceanic and interoceanic dispersal of coastal marine organisms: The biology of ballast water. Oceanogr. Mar. Biol. Annu. Rev. **23:** 313–371.

CHANDRA, C. V., AND R. A. KHAN. 1988. Nematode infestations of fillets from Atlantic cod, *Gadus morhua*, of eastern Canada. J. Parasitol. **74:** 1038–1040.

CHEW, K. K. 1990. Global bivalve shellfish introductions. J. World Aquacult. Soc. **21:** 4–22.

COHEN, A. N., AND J. T. CARLTON. 1998. Accelerating invasion rate in a highly invaded estuary. Science **279:** 555–558.

CORNELL, H. V., AND B. A. HAWKINS. 1994. Patterns of parasitoid accumulation on introduced herbivores, p. 77–89. *In* B. A. Hawkins and W. Sheehan [eds.], Parasitoid community ecology. Oxford.

CORT, W. W., K. L. HUSSEY, AND D. J. AMEEL. 1960. Seasonal fluctuations in larval trematode infections in *Stagnicola emarginata angulata* from Phragmites flats on Douglas Lake. Proc. Helminthol. Soc. Wash. **27:** 11–12.

DABROWSKA, H. 1974. An attempt to evaluate the state of health of fish from the Lyna and Walsza Rivers in connection to their pollution. Przegl. Zool. **18:** 390–395.

DAVIS, G. E., D. V. RICHARDS, P. L. HAAKER, AND D. O. PARKER. 1992. Abalone population declines and fishery management in southern California, p. 237–249. *In* S. A. Shepard et al. [eds.], Abalone of the world: Biology, fisheries and culture. Blackwell.

DES CLERS, S. A. D., AND R. WOOTTEN. 1990. Modelling the population dynamics of the sealworm *Pseudoterranova decipiens*. Neth. J. Sea Res. **25:** 291–299.

DESOWITZ, R. S. 1981. New Guinea tapeworms and Jewish grandmothers. Norton.

———. 1991. The malaria capers. Norton.

DOBSON, A. P., AND R. M. MAY. 1987. The effects of parasites on fish populations—theoretical aspects. Int. J. Parasitol. **17:** 363–370.

ESCH, G. W., T. C. HAZEN, R. V. DIMOCK, JR., AND J. W. GIBBONS. 1976. Thermal effluent and the epizootiology of the ciliate *Ep-*

istylis and the bacterium *Aeromonas* in association with centrarchid fish. Trans. Am. Microsc. Soc. **95:** 687–693.

EURE, H. E., AND G. W. ESCH. 1974. Effects of thermal effluent on the population of helminth parasites in largemouth bass, p. 207–215. *In* Thermal ecology. U.S. AEC Symp. Ser. 32.

EVANS, N. A. 1982. Effects of copper and zinc upon the life cycle of *Notocotylus attenuatus* (Digenea: Notocotylidae). Int. J. Parasitol. **12:** 363–369.

FONT, W. F., AND D. C. TATE. 1994. Helminth parasites of native Hawaiian freshwater fishes: An example of extreme ecological isolation. J. Parasitol. **80:** 682–688.

GARDNER, G. R., AND OTHERS. 1995. Association of prokaryotes with symptomatic appearance of withering syndrome in black abalone *Haliotis crachrodii.* J. Invert. Pathol. **66:** 111–120.

GRANOVITCH, A. I. 1992. The effect of trematode infection on the population structure of *Littorina saxatilis* (Olivi) in the White Sea, p. 255–263. *In* Littorinid biology. Proc. 3rd Int. Symp. Malocol. Soc. Lond.

GRYSEELS, B., AND OTHERS. 1994. Epidemiology, immunology and chemotherapy of *Schistosoma mansoni* infections in a recently exposed community in Senegal. Trop. Geogr. Med. **46:** 209–219.

GUTH, D. J., H. D. BLANKESPOOR, AND J. CAIRNS, JR. 1977. Potentiation of zinc stress caused by parasitic infection of snails. Hydrobiologia **55:** 225–229.

HAMER, W. H. 1906. Epidemic diseases in England. Lancet **1:** 733–739.

HEDGECOCK, D. 1993. Human impacts on the biological diversity of sessile marine invertebrate populations: Introductions, invasions and artificial propagation, p. 125–150. *In* Human impact on self-recruiting populations. Int. Symp. R. Norw. Soc. Sci. Lett. Found.

HEWITT, G. C., AND R. W. LITTLE. 1972. Whirling disease in New Zealand trout caused by *Myxosoma cerebralis* (Hofer, 1903) (Protozoa: Myxosporida). N.Z. J. Mar. Freshwater Res. **6:** 1–10.

HEYNEMAN, D. 1979. Dams and disease. Human Nature **2:** 50–57.

HOFFMAN, G. L., C. E. DUNBAR, AND A. BRADFORD. 1962. Whirling disease of trouts caused by *Myxosoma cerebralis* in the United States, p. 1–15. *In* U.S. Fish Wildl. Serv. Spec. Sci. Rep. Fish. 427.

HUGHES, R. N., AND P. ANSWER. 1982. Growth, spawning and trematode infection of *Littorina littorea* (L.) from an exposed shore in North Wales. J. Molluscan Stud. **48:** 321–330.

KEAS, B. E., AND H. D. BLANKESPOOR. 1997. The prevalence of cercariae from *Stagnicola emarginata* (Lymnaeidae) over 50 years in northern Michigan. J. Parasitol. **83:** 536–540.

KERMACK, W. O., AND A. G. MCKENDRICK. 1927. A contribution to the mathematical theory of epidemics. Part 3. Further studies of the problem of endemicity. Proc. R. Soc. Lond. Ser. A **141:** 92–122.

KHAN, R. A. 1987. Crude oil and parasites of fish. Parasitol. Today **3:** 99–100.

———. 1990. Parasitism in marine fish after chronic exposure to petroleum hydrocarbons in the laboratory and to the *Exxon Valdez* oil spill. Bull. Environ. Contam. Toxicol. **44:** 759–763.

———. 1991. Influence of pollution on parasites of aquatic animals. Ann. Parasitol. Hum. Comp. **66** (suppl. 1)**:** 49–51.

———, D. E. BARKER, K. WILLIAMS-RYAN, AND R. G. HOOPER. 1994. Influence of crude oil and pulp and paper mill effluent on mixed infections of *Trichodina cottidarium* and *T. saintjohnsi* (Ciliophora) parasitizing *Myoxocephalus octodecemspinosus* and *M. scorpius.* Can. J. Zool. **72:** 247–251.

———, AND J. THULIN. 1991. Influence of pollution on parasites of aquatic animals. Adv. Parasitol. **30:** 201–238.

KITCHELL, J. F., AND OTHERS. 1999. Nutrient cycling at the landscape scale: The role of diel foraging migrations by geese at the Bosque del Apache National Wildlife Refuge, New Mexico. Limnol. Oceanogr. **44:** 828–836.

KITRON, V., AND A. SPIELMAN. 1989. Suppression of transmission of malaria through source reduction: Antianopheline measures applied in Israel, the United States, and Italy. Rev. Infect. Dis. **11:** 391–406.

KURIS, A. M. 1997. Host behavior modification: An evolutionary perspective, p. 293–315. *In* N. E. Beckage [ed.], Parasites and pathogens: Effects on host hormones and behavior. Chapman and Hall.

———, S. F. BLAU, A. J. PAUL, J. D. SHIELDS, AND D. E. WICKHAM. 1991. Infestation by brood symbionts and their impact on egg mortality of the red king crab, *Paralithodes camtschatica,* in Alaska: Geographic and temporal variation. Can. J. Fish. Aquat. Sci. **48:** 559–568.

———, AND R. GURNEY. 1997. Survey of Tasmanian crabs for parasites: A progress report, p. 92–94. *In* The demography, impacts and management of introduced populations of the European crab, *Carcinus maenas.* Proc. 1st Int. Workshop. Center Res. Introduced Mar. Pests Tech. Rep. 11.

———, AND K. D. LAFFERTY. 1992. Modelling crustacean fisheries: Effects of parasites on management strategies. Can. J. Fish. Aquat. Sci. **49:** 327–336.

———, AND ———. 1994. Community structure: Larval trematodes in snail hosts. Annu. Rev. Ecol. Syst. **25:** 189–217.

LAFFERTY, K. D. 1993. The marine snail, *Cerithidea californica,* matures at smaller sizes where parasitism is high. Oikos **68:** 3–11.

———. 1997. Environmental parasitology: What can parasites tell us about human impacts on the environment? Parasitol. Today **13:** 251–255.

———, AND A. M. KURIS. 1993. Mass mortality of abalone *Haliotis cracherodii* on the California Channel Islands: Tests of epidemiological hypotheses. Mar. Ecol. Prog. Ser. **96:** 239–248.

———, AND ———. 1996. Biological control of marine pests. Ecology **77:** 1989–2000.

LAWLER, G. H. 1970. Parasites of coregonid fishes, p. 279–309. *In* C. C. Lindsey and C. S. Woods [eds.], Biology of coregonid fishes. Univ. Manitoba.

LEAVITT, P. R., D. L. FINDLAY, R. I. HALL, AND J. P. SMOL. 1999. Algal responses to dissolved organic carbon loss and pH decline during whole-lake acidification: Evidence from paleolimnology. Limnol. Oceanogr. **44:** 757–773.

LEHTINEN, K.-J. 1989. Survival, growth and disease of three-spined stickleback, *Gasterosteus aculeatus* L., brood exposed to bleached kraft mill effluents (BKME) in mesocosms. Ann. Zool. Fenn. **26:** 133–144.

LIGHTNER, D. V., R. M. REDMAN, T. A. BELL, AND R. B. THURMAN. 1992*a*. Geographic dispersion of the viruses IHHN, MBV and HPV as a consequence of transfers and introductions of penaeid shrimp to new regions for aquaculture purposes, p. 155–173. *In* A. Rosenfield and R. Mann [eds.], Dispersal of living organisms into aquatic ecosystems. Md. Sea Grant.

———, R. R. WILLIAMS, T. A. BELL, R. M. REDMAN, AND L. A. PEREZ. 1992*b*. A collection of case histories documenting the introduction and spread of the virus disease IHHN in penaeid shrimp culture facilities in northwestern Mexico, p. 97–105. *In* Introductions and transfers of aquatic species. Mar. Sci. Symp. V. 194. ICES.

MCCLELLAND, G., R. K. MISRA, AND D. J. MARCOGLIESE. 1983. Variations in abundance of larval anisakines, sealworm (*Phocanema decipiens*) and related species in cod and flatfish from the southern Gulf of St. Lawrence (4T) and the Breton Shelf (4 Vn), p. 1–51. *In* Can. Fish. Aquat. Sci. Tech. Rep. 1201.

McDOWELL, J. E., B. A. LANCASTER, D. F. LEAVITT, P. RANTAMAKI, AND B. RIPLEY. 1999. The effects of lipophilic organic contaminants on reproductive physiology and disease processes in marine bivalve molluscs. Limnol. Oceanogr. **44:** 903–909.

MacKENZIE, K., H. H. WILLIAMS, B. WILLIAMS, A. H. McVICAR, AND R. SIDDALL. 1995. Parasites as indicators of water quality and the potential use of helminth transmission in marine pollution studies. Adv. Parasitol. **35:** 85–144.

MARCOGLIESE, D. J., AND D. K. CONE. 1996. On the distribution and abundance of eel parasites in Nova Scotia: Influence of pH. J. Parasitol. **82:** 389–399.

MEYERS, T. R., C. BOTELHO, T. M. KOENEMAN, S. SHORT, AND K. IMAMURA. 1990. Distribution of bitter crab dinoflagellate syndrome in southeast Alaska tanner crabs *Chionoecetes bairdi*. Dis. Aquat. Org. **9:** 37–43.

MILLER, R. B. 1952. A review of the *Triaenophorus* problem in Canadian lakes. Bull. Fish. Res. Bd. Can. 95.

NILSEN, F. 1995. Description of *Trichodina hippoglossi* n. sp. from farmed Atlantic halibut larvae *Hippoglossus hippoglossus*. Dis. Aquat. Org. **21:** 209–214.

NIYOGI, D. K., D. M. McKNIGHT, AND W. M. LEWIS, JR. 1999. Influences of water and substrate quality for periphyton in a montane stream affected by acid mine drainage. Limnol. Oceanogr. **44:** 804–809.

ODENSE, P. H. 1978. Some aspects of the codworm problem. Fish. Environ. Can. Fish. Mar. Serv. Ind. Rep. 106.

OVERSTREET, R. M., AND H. D. HOWSE. 1977. Some parasites and diseases of estuarine fishes in polluted habitats of Mississippi. Ann. New York Acad. Sci. **298:** 427–462.

PETRUSHEVSKI, G. K., AND S. S. SCHULMAN. 1958. The parasitic diseases of fishes in the natural waters of the USSR, p. 299–319. *In* V. A. Dogiel et al. [eds.], Parasitology of fishes. Leningrad Univ.

POHLEY, W. J. 1976. Relationships among three species of *Littorina* and their larval digenea. Mar. Biol. **37:** 179–186.

POOLE, B. C. 1985. Fish parasite population dynamics in seven small boreal lakes of central Canada. M.S. thesis, Univ. Manitoba.

POULIN, R. 1992. Toxic pollution and parasitism in freshwater fish. Parasitol. Today **8:** 58–61.

RIGGS, M. R., A. D. LEMLY, AND G. W. ESCH. 1987. The growth, biomass and fecundity of *Bothriocephalus acheilognathi* in a North Carolina cooling reservoir. J. Parasitol. **73:** 893–900.

ROBSON, E. M., AND I. C. WILLIAMS. 1970. Relationships of some species of digenea with the marine prosobranch *Littorina littorea* (L.) 1. The occurrence of larval digenea in *L. littorea* on the North Yorkshire coast. J. Helminthol. **44:** 153–168.

RUIZ, G. M., P. FOFONOFF, A. H. HINES, AND E. D. GROSHOLZ. 1999. Nonindigenous species as stressors in estuarine and marine communities: Assessing invasion impacts and interactions. Limnol. Oceanogr. **44:** 950–972.

SAKANARI, J. A., AND M. MOSER. 1990. Adaptation of an introduced host to an indigenous parasite. J. Parasitol. **76:** 420–423.

SANDERS, M. J. 1966. Parasitic castration of the scallop *Pecten alba* (Tate) by a bucephalid trematode. Nature **212:** 307–310.

———, AND R. J. G. LESTER. 1981. Further observations on a bucephalid trematode infection in scallops (*Pecten alba*) in Port Phillip Bay, Victoria. Aust. J. Mar. Freshwater Res. **32:** 475–478.

SANKURATHRI, C. S., AND J. C. HOLMES. 1976. Effects of thermal effluents on parasites and commensals of *Physa gyrina* Say (Mollusca: Gastropoda) and their interactions at Lake Wabamum, Alberta. Can. J. Zool. **54:** 1742–1753.

SHIELDS, J. D., D. E. WICKHAM, S. F. BLAU, AND A. M. KURIS. 1990. Some implications of egg mortality caused by symbiotic nemerteans for data acquisition and management strategies of red king crabs, *Paralithodes camtschatica*, p. 383–395. *In* King and tanner crabs. Proc. Int. Symp. Alaska Sea Grant Coll. Program Rep. 90–04.

SIDDALL, R., AND S. DES CLERS. 1994. Effect of sewage sludge on the miracidium and cercaria of *Zoogonoides viviparus* (Trematoda: Digenea). Helminthologia **31:** 143–153.

SMITH, N. J. H. 1981. Colonization lessons from a tropical forest. Science **214:** 755–761.

STADNICHENKO, A. P., AND OTHERS. 1995. The effect of different concentrations of nickel sulphate on the horn snail (Mollusca: Bulinidae) infected with the trematode *Cotylurus cornutus* (Strigeidae). Parazitologiya **29:** 112–116.

TORCHIN, M. E., K. D. LAFFERTY, AND A. M. KURIS. 1996. Infestation of an introduced host, the European green crab, *Carcinus maenas* by a native symbiotic nemertean egg predator, *Carcinonemertes epialti*. J. Parasitol. **82:** 449–453.

VALTONEN, E. T., J. C. HOLMES, AND M. KOSKIVAARA. 1997. Eutrophication, pollution and fragmentation: Effects on parasite communities in roach (*Rutilus rutilus*) and perch (*Perca fluviatilis*) in four lakes in central Finland. Can. J. Fish. Aquat. Sci. **54:** 572–585.

VIK, R. 1965. Studies of the helminth fauna of Norway. 6. An experiment in the control of *Diphyllobothrium* infections in trout. Nytt Mag. Zool. **13:** 76–78.

VLADIMIROV, V. L., AND B. A. FLEROV. 1975. Susceptibility to ichthyophthiriasis in fish following exposure to phenol and polychloropinene poisoning. Biol. Vnutr. Vod Inf. Byull. **25:** 35–37.

WEISENFELD, S. L. 1967. Sickle-cell trait in human biological and cultural evolution. Science **157:** 1134–1140.

YOUNG, P. C. 1972. The relationship between the presence of larval anisakine nematodes in cod and marine mammals in British waters. J. Appl. Ecol. **9:** 459–485.