AMER. ZOOL., 31:831–839 (1991)

# Consequences of Parasitism to Marine Invertebrates: Host Evolution?[1]

GREGORY M. RUIZ

*Smithsonian Environmental Research Center, P.O. Box 28, Edgewater, Maryland 21037*

SYNOPSIS. Parasitism among aquatic invertebrates is common, if not ubiquitous, and can be pathological to hosts. However, host evolution in response to parasitism has received little attention, particularly for marine invertebrates. Drawing on the rich literature demonstrating prey adaptations to predators, I develop analogous predictions for the ways in which host life histories may be molded by their parasites. Such adaptations are expected when the effects of parasites are severe and when the probability of infection is high. Predicted life history changes include the evolution of semelparity, reduced age at first reproduction and reduced size at first reproduction. Using Recent and fossil populations of two bivalves species in the genus *Transennella,* I show that the incidence of trematode parasites may explain a trend of reduced size through time and contribute to the maintenance of sexual dimorphism for size.

## INTRODUCTION

Parasites are common in marine communities and include a broad diversity of protozoan and metazoan species (for recent reviews see Kinne, 1980, 1983, 1984*a, b*; Muehlstein, 1989; Sindermann, 1990*a, b*). Furthermore, it is probable that all species in marine habitats have parasites (*e.g.,* Price, 1980).

For marine invertebrates, consequences of parasitism to the individual host have been described for a broad range of host and parasite taxa and can be severe. Parasitic infection often results in drastically reduced growth, fecundity, and survival and may alter behavior (for reviews see Holmes and Bethel, 1972; Kinne, 1980, 1983; Sousa, 1983; Sindermann, 1990*b*). In marine habitats, and more generally, the importance of these effects in population and community processes has received increasing attention (*e.g.,* Anderson and May, 1978, 1979*a*; May and Anderson, 1979; Rosenfield and Kern, 1979; May, 1983; Lessios *et al.,* 1984, 1985; Sousa, 1991).

This paper examines evolutionary consequences of parasitism to marine invertebrates. Despite recent interest in host-parasite evolution (*e.g.,* Holmes, 1983; Rollinson and Anderson, 1985; Freeland, 1986; Dobson, 1988), this topic has been virtually ignored for marine organisms. Drawing on available literature and original data, I develop some predictions for the evolutionary responses of marine invertebrate hosts to their parasites. The primary focus here is trematode parasitism of aquatic (*i.e.,* marine and freshwater) molluscs. The choice of these taxa, and the inclusion of freshwater studies in a symposium on marine ecology, was based on the limited literature available for host evolution of aquatic invertebrates.

## EVOLUTIONARY RESPONSES OF HOSTS TO PARASITISM

### *Background*

The interaction of parasite and host populations over time has been portrayed as an evolutionary arms race, with hosts evolving defenses against parasites that are evolving counter-adaptations to host defenses (Price, 1980; Holmes, 1983; Freeland, 1986; Seger and Hamilton, 1988). An analogous arms race between predator and prey populations (Gilbert, 1971; Dawkins and Krebs, 1979) has apparently led to the evolution of numerous anti-predator defenses or adaptations in aquatic invertebrates, including changes in behavior, morphology, life-history, and chemical composition that reduce the risk of predation (*e.g.,* Harvell, 1986; Kerfoot and Sih, 1987; Vermeij, 1987; Stearns, 1989; Crowl and Covich, 1990; Hairston and Dillon, 1990). While a similar suite of anti-parasite adaptations may be predicted for aquatic invertebrate hosts,

[1] From the Symposium on *New Perspectives in Soft-Sediment Ecology* presented at the Annual Meeting of the American Society of Zoologists, 27–30 December 1990, at San Antonio, Texas.

especially where the outcome of parasitism is death or castration (=reproductive death), these adaptations have not been reported (Holmes, 1983; Minchella, 1985; Dobson, 1988).

The literature on anti-parasite adaptations, or host evolution, for aquatic invertebrates is restricted primarily to immune responses of molluscs (Minchella, 1985). The immune system of freshwater gastropods plays an active role in defense against trematode infection and may exhibit local adaptation, having evolved greater resistance to local strains than to distant strains of the same trematode species (Wakelin, 1978, 1985; Lackie, 1980; van der Knaap and Loker, 1990). Parasite-resistant strains have also been reported in marine bivalves following epizootics or outbreaks of protozoan parasites (Rosenfield and Kern, 1979), presumably evolving in response to severe selection by the parasitism. Interestingly, sexual reproduction itself may have evolved in response to parasitism. Genetic recombination (meiosis) may produce an immune system more effective at meeting parasite counter-adaptations than that possible with asexual reproduction (Seger and Hamilton, 1988; Lively, 1987).

Besides immune responses, the only other anti-parasite adaptation reported for aquatic invertebrates has been termed fecundity compensation (Minchella and LoVerde, 1981; Minchella, 1985; Thornhill *et al.*, 1986). Exposure of the freshwater snail *Biomphalaria glabrata* to miracidia of the trematode *Schistosoma mansoni* has resulted in significantly increased rates of egg-laying relative to unexposed controls. The same result was obtained whether or not the snails actually became infected; this has been interpreted as a compensatory response to probable reduction in future reproductive success, because the infection usually results in castration.

*Theoretical considerations*

Parasites act as selective agents when they are common and cause a reduction in host fitness through lowered survival, growth, access to mates, or fertility (Minchella, 1985; Freeland, 1986). For molluscan hosts, the cost of infection by sporocyst and redia (*i.e.*, pre-cercarial) stages of digenetic trematodes can be very high, often including reduced fecundity, castration, and increased mortality (*e.g.*, Kuris, 1974; Lauckner, 1980, 1983). Nevertheless, if prevalence of infection is usually low, possibly due to immune defenses, selection for anti-parasite adaptations may be weak. Using this argument, Dobson (1988) suggested that low prevalence of trematode parasitism, usually near 1% and rarely exceeding 10% of a population, may explain the relative lack of anti-parasite adaptations for aquatic invertebrate hosts.

Although a review of trematode prevalence is not available, there are many molluscan species for which prevalence has been reported in excess of 10% (*e.g.*, Fraser, 1967; Køie, 1969; Swennen and Ching, 1974; Sanders and Lester, 1981; see Table 1 in Sousa, 1991). However, most prevalence data reported are difficult to interpret and compare; they are typically derived from few samples, taken over short temporal and undefined spatial scales. Parasitism can be highly seasonal and may exhibit patchiness or even predictable spatial pattern such as shore-level gradients (*e.g.*, Swennen and Ching, 1974; Lauckner, 1980, 1983). An additional complication exists where no distinction has been made among different sized/aged individuals, as trematode parasitism often increases with size/age (see below). With these caveats in mind, the current literature indicates many mollusc populations do not have the low prevalence suggested by Dobson (1988). Furthermore, Anderson and May (1979*b*) suggested that prevalence on a lifetime (or cohort) basis can be grossly underestimated with most available data, which do not include parasite-induced mortality.

Strong selection due to trematode parasitism, favoring the evolution of anti-parasite adaptations, apparently exists for some molluscan hosts. In particular, the prevalence and cost of pre-cercarial infection is often very high, despite the presence of immune defenses. Furthermore, post-cercarial stages (*i.e.*, metacercariae) can occur in virtually all individuals of a host population (*e.g.*, Lauckner, 1983; Fig. 3B; see also Table 3 in Sousa, 1991) and may have

further deleterious effects. Given the common occurrence of trematodes in molluscan populations (e.g., Lauckner, 1980, 1983), selection favoring anti-parasite adaptations may even be widespread. However, in addition to fecundity compensation by *Biomphalaria,* I am only aware of two cases (below) in which host evolution is hypothesized that does not involve the immune system.

Semelparity in freshwater temperate snails may be an evolved response to trematode parasitism. Semelparity in these snails is widespread, is derived from iteroparous ancestral marine or terrestrial species, and is probably a response to unpredictable or poor adult survivorship (Calow, 1978; Heller, 1990). Trematode parasitism is common among freshwater snails and frequently leads to castration. Because trematode prevalence commonly increases with host size and age (*e.g.,* Rothschild, 1941; Matthews, 1974; Sousa, 1983; Anderson and Crombie, 1984), Bayne and Loker (1987) proposed that trematode parasitism contributed to poor adult survivorship and the evolution of semelparity in the group. If parsitism is solely size-dependent, selection due to parasitism should favor the evolution of smaller size. Life history theory predicts that selection due to age-dependent parasitism should favor an earlier age at first reproduction (*e.g.,* Stearns, 1976; Reznick and Endler, 1982; Reznick, 1983; Harvell and Grosberg, 1988). Because of the positive correlation that often exists among size, age at first reproduction, and longevity (*e.g.,* Calow, 1983), each age- and size-dependent parasitism may result in decreased size, longevity, and age at first reproduction.

A similar change in life history due to trematode parasitism may have occurred in a marine bivalve (Ruiz and Lindberg, 1989). I next review the available data on life history and trematode parasitism for this clam host, considering a possible relationship between the two.

*A case history*

*1. Background.*—The venerid clams *Transennella tantilla* and *T. confusa* are abundant in sediments of bays and estuaries along the Pacific coast of North America (Abbott, 1974), where they can reach densities above 15,000 clams/$m^2$. Both species have direct development, live approximately one year, and attain maximum sizes of 5–6 mm in shell length (Obrebski, 1968; Asson-Batres, 1988; Russell and Huelsenbeck, 1989; Mottet, 1988; Ruiz, unpublished data). *Transennella tantilla* and *T. confusa* are both infected by digenetic trematodes.

The trematode *Telolecithus pugetensis* is described from *T. tantilla* (DeMartini and Pratt, 1964) and also occurs in *T. confusa* (personal observation). The pre-cercarial rediae infect *Transennella,* giving rise to cercariae that invade other bivalve species and mature in fish (*e.g.,* embiotocid surfperches). Of 200 female *T. tantilla* examined from False Bay, Washington, 39.5% were infected by *Telolecithus* in June and July (Kabat, 1986). For the size range of clams sampled, 3.5–5.9 mm, prevalence did not increase with size. Kabat (1986) found that female *T. tantilla* infected with *Telolecithus* had a lower fecundity (=brood size) compared to uninfected individuals of similar size (Fig. 1), concluding that infection resulted in "partial castration" and that hosts would remain reproductive. Not included in Kabat's figure were the 24 infected individuals (30.4% of his parasitized populations) that he found to be completely castrated, very likely the fate of all parasitized hosts at a more advanced stage of infection (*e.g.,* Lauckner, 1980, 1983; Anderson and Crombie, 1984). *Transennella confusa* infected with *Telolecithus* suffers a similar loss of fecundity (Ruiz, unpublished data).

Two other species of trematode, in addition to *Telolecithus,* were found in both species of *Transennella* at Bodega Bay, California. Of 184 *T. tantilla* examined from a single site (0.4 m above mean lower low water) in April 1990, 17.4% were infected with pre-cercarial trematodes. The prevalence of infection increased strongly with clam size (Fig. 2), from less than 10% of small (2.5–3.5 mm) clams infected to over 40% of large (>5.0 mm) clams infected. *Parvatrema borealis* was the most common pre-cercarial trematode found in *T. tantilla,* making up 56% of all infections. *Telolecithus* was only present in one individual (3%

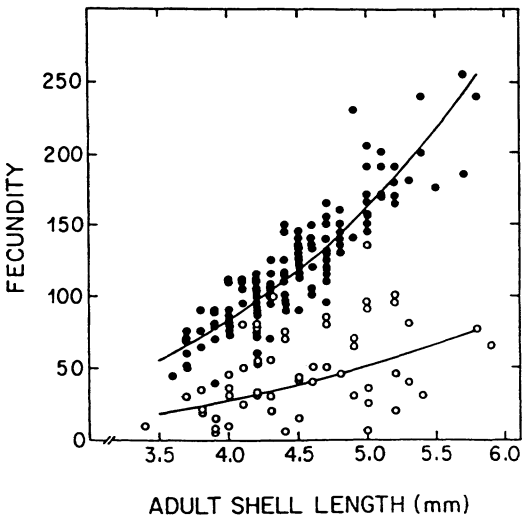


FIG. 1. Fecundity of female *T. tantilla* from False Bay, Washington in June and July 1984. Shown are the sizes of broods as a function of shell length for clams infected by pre-cercarial *Telolecithus* (○) and those not infected (●). (Fig. 1 from Kabat, 1986.)

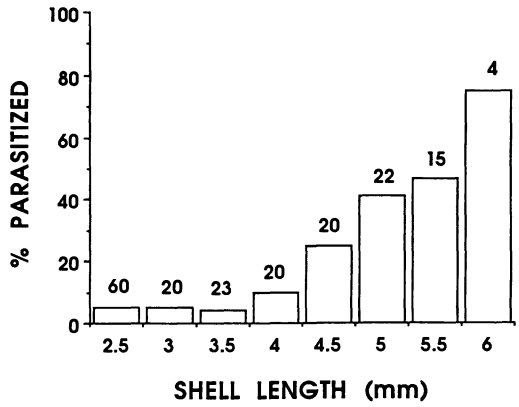


FIG. 2. Prevalence of pre-cercarial trematode infection for *T. tantilla* from Bodega Bay, California in April 1990. Prevalence is shown as a function of shell length, and sample sizes are indicated at the top of each size category.

of all infections). A third trematode species, not yet identified, was present in 5 individuals (16% of all infections), and the remaining pre-cercarial infections (25%) were at early stages of development that could not be identified to species. No multiple infections were present.

The prevalence of pre-cercarial infections for *T. confusa* at Bodega Bay in July 1986 had a very similar relationship with size (Ruiz and Lindberg, 1989, Fig. 4). Overall 14.2% of 161 clams were infected. Prevalence was approximately 0% for small (<3.5 mm) clams and over 30% for large (>5.0 mm) clams. Although the species composition was not quantified, all three trematode species occurred in this and subsequent samples (Ruiz, unpublished data).

In addition to False Bay and Bodega Bay, trematodes are common in other *Transennella* populations. Russell and Huelsenbeck (1989) reported the prevalence of unidentified trematodes to range from 2.9% to 67.9% for monthly samples of *T. confusa* from Half Moon Bay, California. Furthermore, one or more species of trematode have been present in all populations that I have examined of *T. confusa* (Half Moon Bay, California; Tomales Bay, California; Bodega Bay, California) and *T. tantilla* (Morro Bay, California; Elkhorn Slough, California; Bolinas Lagoon, California; Tomales Bay, California; Bodega Bay, California; Coos Bay, Oregon; False Bay and Wescott Bay, Washington).

As with *Telolecithus,* pre-cercarial infection by *Parvatrema* and the unidentified trematode, was associated with lowered fecundity and sterility for *T. tantilla* (Fig. 3A); a similar relationship was found for *T. confusa* (Ruiz, unpublished data). Other possible effects of pre-cercarial trematodes on behavior, growth, and survival of *Transennella* have not been explored. In addition to pre-cercarial infection, metacercariae of *Parvatrema* infect *T. tantilla* and *T. confusa* (Stunkard and Uzmann, 1958; James, 1964; Obrebski, 1968; personal observation). Virtually 100% of the *Transennella* examined from Bodega Bay were infected with metacercariae. The number of metacercariae increased markedly with clam size, as shown for *T. tantilla* (Fig. 3B). While Obrebski (1968) found mass mortality of *T. tantilla* at Tomales Bay, California associated with an increase in the number of metacercariae per clam, effects of *Parvatrema* metacercariae on *Transennella* have not been measured.

The metacercariae of *Parvatrema* are of further interest because they cause the formation of characteristic pits on the inside

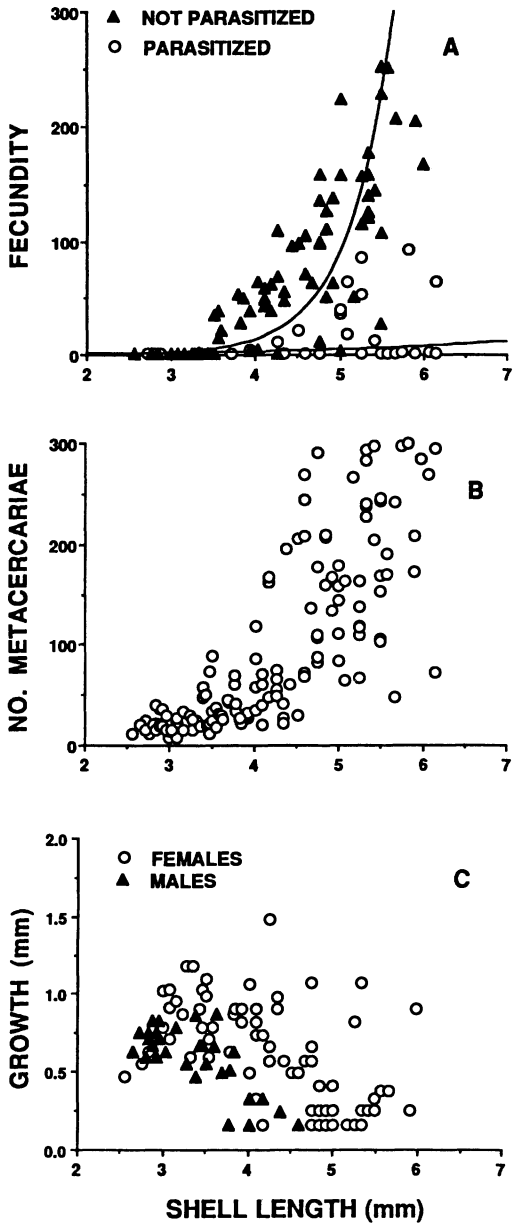


FIG. 3. Size-specific characteristics of *T. tantilla* at Bodega Bay, California in April 1990: (A) Fecundity of clams infected (○) and not infected (▲) by pre-cercarial trematode stages; (B) Number of *Parvatrema* metacercariae; (C) Growth in shell length plotted against final size for males (▲) and females (○).

shell surface of *Transennella* that are also present in fossils (Ruiz and Lindberg, 1989). The fossil record indicates that *Parvatrema* has infected *Transennella* in California since the Pliocene. Thus, *Transennella* has had at least 2 million years to evolve in response to interactions with trematodes.

*2. Evolutionary responses of hosts to parasites?*—In response to trematode parasitism, life history theory (*e.g.*, Stearns, 1976; Reznick and Endler, 1982; Calow, 1983) predicts changes in *Transennella* similar to those proposed by Bayne and Loker (1987) for their freshwater snails. In both cases, the prevalence and cost of pre-cercarial infection is high, and infection exhibits a strongly positive size-(age-)dependence. While Bayne and Loker (1987) suggested semelparity may have evolved in response to trematode parasitism, the general predictions of their argument include 1) size reduction in response to size-dependence of parasitism and 2) reduction in age at first reproduction in response to age-dependence that would likely result in reduced longevity and size (as above).

The fossil record for *Transennella* indicates a significant size reduction has occurred along the Pacific coast since the Pleistocene (Ruiz and Lindberg, 1989). The mean maximum size of clams from Pleistocene samples was 10.38 mm (SD = 1.30), and the same measurement from Recent populations was 6.63 mm (SD = 1.57). While many factors may have caused or contributed to changes in size, and correlated life history traits (*e.g.*, Ruiz and Lindberg, 1989), these data are consistent with the predictions of Bayne and Loker (1987) for the role of parasitism in host evolution.

An interesting pattern of parasitism emerges for *T. tantilla* when considering the sexes separately. The species has been described as a protandric hermaphrodite (Gray, 1982; Kabat, 1986; Asson-Batres, 1988). This conclusion was drawn from the observation that most males were smaller than females, never attaining the size of large females, and that some individuals appeared to be simultaneous hermaphrodites. However, Mottet (1988) has shown *T. tantilla* at False Bay, Washington are probably not protandric. It appears that only a small portion are hermaphroditic, as is occasionally found in other gonochoric bivalves (*e.g.*, Sastry, 1979). In the laboratory, Mottet (1988) showed that intersexual size differences can be attributed to differential growth

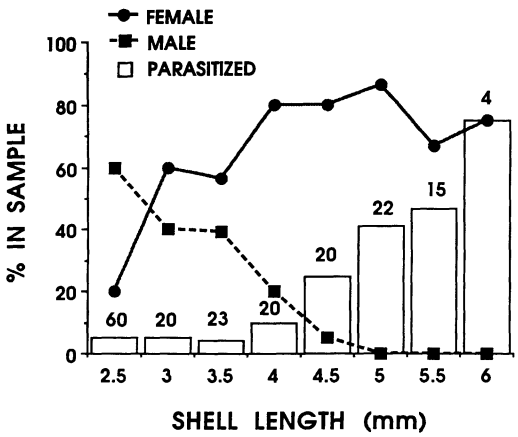


FIG. 4. Percentage of males (■) and females (●) in a sample population of *T. tantilla* (Bodega Bay, California; April 1990) shown with the prevalence of pre-cercarial trematode infection for each 0.5 mm size category. Males and females do not sum to 100% within each size because parasitized clams could not always be sexed.

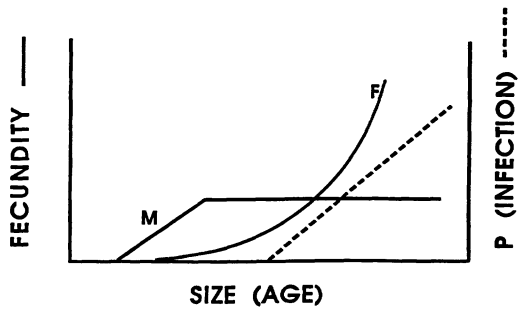


FIG. 5. Hypothetical relationship of fecundity and probability of pre-cercarial infection as a function of size for *T. tantilla*. Fecundity, or reproductive success, is shown separately for males (M) and females (F). These relationships are only defined qualitatively here (*i.e.*, without scale). Where probability of infection is high and strongly size-dependent, the different size-fecundity relationships between sexes may favor the evolution of sexual dimorphism.

rates. In the field, she found that sex ratio did not depart significantly from 1:1 and that growth beyond a winter growth check (ring) was much greater for females than males. Use of rings to infer growth rates (*e.g.*, Lutz and Rhoads, 1980; Tanabe, 1988) suggests similar intersexual differences exist at Bodega Bay (Fig. 3C) and many other locations (Ruiz, unpubl. data).

Sexual dimorphism is very rare among free-living bivalve molluscs (Sastry, 1979; Mackie, 1984), and no information is available on the prevalence of parasites for the few dimorphic species known. In the case of *T. tantilla,* males have a lower rate of pre-cercarial infection than the larger females (Fig. 4).

Parasitism may have played a role in the evolution of sexual dimorphism for *T. tantilla.* One interpretation of Figure 4 is that trematode prevalence for *T. tantilla* is positively size-dependent and that differences in age or sex have little or no effect on prevalence (independent of size). Thus, small males suffer a lower prevalence than would larger males, and selection due to parasitism has favored size reduction. The same is true for females. However, the relationship between size and reproductive success may differ for males and females. Brood size (fecundity) of female *T. tantilla* increases exponentially with size (Fig. 3A; Kabat, 1985, 1986), and brood production is probably a good measure of reproductive success. While the number of sperm produced or stored by male *T. tantilla* probably increases with size, just as female fecundity, male reproductive success may bear no such relationship (*e.g.*, Ghiselin, 1969). This may be especially true for species with direct development where males live in close contact with females and/or where males pass sperm to females (*e.g.*, O'Foighil, 1985) rather than broadcast spawn. Thus, selection for small size by parasitism may be counteracted to different degrees for males and females by selection due to size-dependent reproductive success (Fig. 5), resulting in sexual dimorphism for size.

## CONCLUSION

The goal of this paper is to examine the evolutionary responses of marine invertebrate hosts to parasitism, especially in soft-sediment habitats. It is clear that immune defenses exist for freshwater and marine invertebrate taxa. Despite the presence of immune defenses, selection for the evolution of anti-parasite defenses or adaptations should exist in many host populations and may even be common when trematodes are involved.

Unfortunately, there is more speculation than data available to evaluate the relation-

ship between trematode parasitism and host evolution. The examples presented for freshwater snails and the clams *Transennella* illustrate a relationship that may exist between parasitism and evolution of host life history traits. While both examples offer viable hypotheses, they are not intended to claim a causal relationship does exist.

Evaluation of these and other hypotheses regarding host evolution will often require intra- and interspecific comparisons of host traits as a function of parasitism (*e.g.*, Lively, 1987). Such comparisons rely on the quantity and quality of available data, which for the prevalence and effects of parasitism is still very limited in both regards. Collecting prevalence data in a standardized fashion, with attention to spacial and temporal scales and to host size, is essential for comparison. Furthermore, a strong need exists to test experimentally and verify the patterns (*e.g.*, size- or age-dependence) and effects of parasitic infection in field populations. Only after we have these data can the potential importance of parasitism in host evolution be evaluated.

## ACKNOWLEDGMENTS

I wish to thank W. H. Wilson, Jr. and B. Brown for organizing the symposium; along with R. Everett, E. Grosholz and W. Sousa, they provided critical comments that improved this paper. Development of many ideas presented here began while I was a postdoctoral fellow at the Friday Harbor Laboratories and benefited from early discussions with M. Mottet.

## REFERENCES

Abbott, R. T. 1974. *American seashells.* Nostrand Reinhold Company, New York.

Anderson, R. M. and J. Crombie. 1984. Experimental studies of age-prevalence curves for *Schistosoma mansoni* infections in populations of *Biomphalaria glabrata.* Parasitology 89:79–104.

Anderson, R. M. and R. M. May. 1978. Regulation and stability of host-parasite population interactions. I. Regulatory processes. J. Anim. Ecol. 47: 219–247.

Anderson, R. M. and R. M. May. 1979*a*. Population biology of infectious diseases: Part I. Nature 280: 361–367.

Anderson, R. M. and R. M. May. 1979*b*. Prevalence of schistosome infections within molluscan populations: Observed patterns and theoretical predictions. Parasitology 79:63–94.

Asson-Batres, M. A. 1988. Reproduction and growth of the brooding bivalve *Transennella tantilla.* Veliger 30:257–267.

Bayne, C. J. and E. S. Loker. 1987. Survival within the snail host. *In* D. Rollinson (ed.), *The biology of schistosomes: From genes to latrines,* pp. 321–346. Academic Press, New York.

Calow, P. 1978. The evolution of life-cycle strategies in fresh-water gastropods. Malacologia 17:351–364.

Calow, P. 1983. Life-cycle patterns and evolution. *In* W. D. Russell-Hunter (ed.), *The Mollusca. Volume 6. Ecology,* pp. 649–678. Academic Press, Orlando.

Crowl, T. A. and A. P. Covich. 1990. Predator-induced life-history shifts in a freshwater snail. Science 247:949–951.

Dawkins, R. and J. R. Krebs. 1979. Arms race between and within species. Proc. Roy. Soc. London B 205: 489–511.

DeMartini, J. D. and I. Pratt. 1964. The life cycle of *Telolecithus pugetensis* Lloyd and Guberlet, 1932 (Trematoda: Monorchidae). J. Parasit. 50:101–105.

Dobson, A. P. 1988. The population biology of parasite-induced changes in host behavior. Q. Rev. Biol. 63:139–165.

Fraser, T. H. 1967. Contributions to the biology of *Tagelus divisus* (Tellinacea: Pelecypoda) in Biscayne Bay, Florida. Bull. Mar. Sci. 17:111–132.

Freeland, B. 1986. Arms race and covenants: The evolution of parasite communities. *In* J. Kikkawa and D. J. Anderson (eds.), *Community ecology: Pattern and process,* pp. 289–304. Blackwell Sci. Publ., Victoria.

Ghiselin, M. T. 1969. The evolution of hermaphroditism among animals. Q. Rev. Biol. 44:189–208.

Gilbert, L. E. 1971. Butterfly-plant coevolution: Has *Passiflora adenopoda* won the selection arms race with heliconiine butterflies? Science 172:585–586.

Gray, S. 1982. Morphology and taxonomy of two species of the genus *Transennella* (Bivalvia: Veneridae) from western North America and a description of *T. confusa* sp. nov. Malacolog. Rev. 15:107–117.

Hairston, N. G., Jr. and T. A. Dillon. 1990. Fluctuating selection and response in a population of freshwater copepods. Evolution 44:1796–1805.

Harvell, C. D. 1986. The ecology and evolution of inducible defenses in a marine bryozoan: Cues, costs, and consequences. Am. Nat. 128:810–823.

Harvell, C. D. and R. K. Grossberg. 1988. The timing of sexual maturity in clonal animals. Ecology 69: 1855–1864.

Heller, J. 1990. Longevity in molluscs. Malacologia 31:259–295.

Holmes, J. C. 1983. Evolutionary relationships between parasitic helminths and their hosts. *In* D. J. Futuyma and M. Slatkin (eds.), *Coevolution,* pp. 161–185. Sinauer Assoc. Inc., Sunderland.

Holmes, J. C. and W. M. Bethel. 1972. Modification of intermediate host behaviour by parasites. *In* E. U. Canning and C. A. Wright (eds.), *Behavioral aspects of parasite transmission,* pp. 123–149. Zool. Jour. Linn. Soc. 51, suppl. 1.

James, B. L. 1964. The life cycle of *Parvatrema homeotecnum* sp. nov. (Trematoda: Digenea) and a review of the family Gymnophallidae Morozov 1955. Parasitology 54:1–41.

Kabat, A. R. 1985. The allometry of brooding in *Transennella tantilla* (Gould) (Mollusca: Bivalvia). J. Exp. Mar. Biol. Ecol. 91:271–279.

Kabat, A. R. 1986. Effects of trematode parasitism on reproductive output of the bivalve *Transennella tantilla.* Can. J. Zool. 64:267–270.

Kerfoot, W. C. and A. Sih. 1987. *Predation: Direct and indirect impacts on aquatic communities.* University Press of New England, Hanover.

Kinne, O. (ed.) 1980. *Diseases of marine organisms. Volume I. General aspects, Protozoa to Gastropoda.* John Wiley and Sons, Chichester.

Kinne, O. (ed.) 1983. *Diseases of marine organisms. Volume II. Introduction, Mollusca: Bivalvia to Scaphopoda.* Biologische Anstalt Helgoland, Hamburg.

Kinne, O. (ed.) 1984*a*. *Diseases of marine organisms. Volume IV, Part I. Introduction, Pisces.* Biologische Anstalt Helgoland, Hamburg.

Kinne, O. (ed.) 1984*b*. *Diseases of marine organisms. Volume IV, Part II. Introduction, Reptilia, Aves, Mammalia.* Biologische Anstalt Helgoland, Hamburg.

Køie, M. 1969. On the endoparasites of *Buccinum undatum* L. with special reference to the trematodes. Ophelia 6:251–279.

Kuris, A. M. 1974. Trophic interactions: Similarity of parasitic castrators to parasitoids. Q. Rev. Biol. 49:129–148.

Lackie, A. M. 1980. Invertebrate immunity. Parasitology 80:393–412.

Lauckner, G. 1980. Diseases of Mollusca: Gastropoda. *In* O. Kinne (ed.), *Diseases of marine organisms. Volume I. General aspects, Protozoa to Gastropoda,* pp. 311–424. John Wiley and Sons, Chichester.

Lauckner, G. 1983. Diseases of Mollusca: Bivalvia. *In* O. Kinne (ed.), *Diseases of marine organisms. Volume II. Introduction, Mollusca: Bivalvia to Scaphopoda,* pp. 477–962. Biologische Anstalt Helgoland, Hamburg.

Lessios, H. A., D. R. Robertson, and J. D. Cubit. 1984. Spread of *Diadema* mass mortality through the Caribbean. Science 226:335–337.

Lessios, H. A., J. D. Cubit, D. R. Robertson, M. J. Schulman, M. R. Parker, S. D. Garrity, and S. C. Levings. 1985. Mass mortality of *Diadema antillarum* on the Caribbean coast. Coral Reefs 3:173–182.

Lively, C. M. 1987. Evidence from a New Zealand snail for the maintenance of sex by parasitism. Nature 328:519–521.

Lutz, R. A. and D. C. Rhoads. 1980. Growth patterns within the molluscan shell. An overview. *In* D. C. Rhoads and R. A. Lutz (eds.), *Skeletal growth of aquatic organisms,* pp. 203–254. Plenum Press, New York.

Mackie, G. L. 1984. Bivalves. *In* A. S. Tompa, N. H. Verdonk, and J. A. M. van den Biggelaar (eds.), *The Mollusca. Part 7. Reproduction,* pp. 351–418. Academic Press, Orlando.

Matthews, R. A. 1974. The life-cycle of *Bucephaloides gracilescens* (Rudolphi, 1819) Hopkins, 1954 (Digenea: Gasterostomata). Parasitology 68:1–12.

May, R. M. 1983. Parasitic infections as regulators of animal populations. Amer. Sci. 71:36–45.

May, R. M. and R. M. Anderson. 1979. Population biology of infectious diseases: II. Nature 280:455–461.

Minchella, D. J. 1985. Host life-history variation in response to parasitism. Parasitology 90:205–216.

Minchella, D. J. and P. T. LoVerde. 1981. A cost of increased early reproductive effort in the snail *Biomphalaria glabrata.* Am. Nat. 118:876–881.

Mottet, M. G. 1988. Biology of the clam *Nutricola tantilla* (=*Transennella tantilla*) with emphasis on mass movements, selective-drift behaviors, and sex. Ph.D. Diss., University of Washington, Seattle.

Muehlstein, L. K. 1989. Perspectives on the wasting disease of eelgrass *Zostera marina.* Dis. Aquat. Org. 7:211–221.

Obrebski, S. 1968. On the population ecology of two intertidal invertebrates and the paleoecological significance of size-frequency distributions of living and dead shells of the bivalve *Transennella tantilla.* Ph.D. Diss., University of Chicago, Chicago.

O'Foighil, D. 1985. Sperm transfer and storage in the brooding bivalve *Mysella tumida.* Biol. Bull. 169: 602–614.

Price, P. W. 1980. *Evolutionary biology of parasites.* Princeton University Press, Princeton.

Reznick, D. 1983. The structure of guppy life histories: The tradeoff between growth and reproduction. Ecology 64:867–873.

Reznick, D. and J. A. Endler. 1982. The impact of predation on life history evolution in Trinidadian guppies (*Poecilia reticulata*). Evolution 36:160–177.

Rollinson, D. and R. M. Anderson. (eds.) 1985. *Ecology and genetics of host-parasite interactions.* Academic Press, Orlando.

Rosenfield, A. and F. G. Kern. 1979. Molluscan imports and the potential for introduction. *In* R. Mann (ed.), *Exotic species in mariculture,* pp. 165–191. MIT Press, Cambridge.

Rothschild, M. 1941. The effect of trematode parasites on the growth of *Littorina neritoides* (L.). J. Mar. Biol. Assn., U. K. 25:69–79.

Ruiz, G. M. and D. R. Lindberg. 1989. A fossil record for trematodes: Extent and potential uses. Lethaia 22:431–438.

Russell, M. and M. Huelsenbeck. 1989. Seasonal variation in brood structure of *Transennella confusa* (Bivalvia: Veneridae). Veliger 32:288–295.

Sanders, M. J. 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.

Sastry, A. N. 1979. Pelecypoda (excluding Ostreidae). *In* A. C. Geise and J. S. Pearse (eds.), *Reproduction of marine invertebrates. Volume V. Molluscs: Pelecypods and lesser classes,* pp. 113–292. Academic Press, New York.

Seger, J. and W. D. Hamilton. 1988. Parasites and sex. *In* R. E. Michod and B. R. Levin (eds.), *The*

*evolution of sex: An examination of current ideas,* pp. 176–193. Sinauer Assoc. Inc., Sunderland.

Sindermann, C. J. 1990*a*. *Principal diseases of marine fish and shellfish. I. Diseases of marine fish. Second edition.* Academic Press, San Diego.

Sindermann, C. J. 1990*b*. *Principal diseases of marine fish and shellfish. II. Diseases of marine shellfish. Second edition.* Academic Press, San Diego.

Sousa, W. P. 1983. Host life history and the effect of parasitic castration on growth: A field study of *Cerithidea californica* Haldeman (Gastropoda: Prosobranchia) and its trematode parasites. J. Exp. Mar. Biol. Ecol. 73:273–296.

Sousa, W. P. 1991. Can models of soft-sediment community structure be complete without parasites? Amer. Zool. 31:821–830.

Stearns, S. C. 1976. Life-history tactics: A review of the ideas. Q. Rev. Biol. 51:3–47.

Stearns, S. C. 1989. The evolutionary significance of phenotypic plasticity. Bioscience 39:436–445.

Stunkard, H. W. and J. R. Uzmann. 1958. Studies of the digenetic trematodes of the genera *Gymnophallus* and *Parvatrema.* Biol. Bull. 115:276–302.

Swennen, C. and H. L. Ching. 1974. Observations on the trematode *Parvatrema affinis,* causative agent of crawling tracks of *Macoma balthica.* Neth. J. Sea Res. 8:108–115.

Tanabe, K. 1988. Age and growth determinations of an intertidal bivalve, *Phacosoma japonicom,* using internal shell increments. Lethaia 21:231–241.

Thornhill, J. A., J. T. Jones, and J. R. Kusel. 1986. Increased oviposition and growth in immature *Biomphalaria glabrata* after exposure to *Schistosoma mansoni.* Parasitology 93:443–450.

van der Knapp, W. P. W. and E. S. Loker. 1990. Immune mechanisms in trematode-snail interactions. Parasitology Today 6:175–182.

Vermeij, G. J. 1987. *Evolution and escalation. An ecological history of life.* University Press, Princeton.

Wakelin, D. 1978. Genetic control of susceptibility and resistance to parasitic infection. Adv. Parasitology 16:219–308.

Wakelin, D. 1985. Genetics, immunity and parasite survival. *In* D. Rollinson and R. M. Anderson (eds.), *Ecology and genetics of host-parasite interactions,* pp. 39–54. Academic Press, Orlando.