Ecological Monographs, 63(2), 1993, pp. 103–128

INTERSPECIFIC ANTAGONISM AND SPECIES COEXISTENCE IN A DIVERSE GUILD OF LARVAL TREMATODE PARASITES[1]

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Abstract. The salt marsh snail *Cerithidea californica* is first intermediate host to a diverse guild of larval trematode parasites. In Bolinas Lagoon, in central California, the site of this study, at least 15 species of trematodes infect snail populations. This study investigated patterns of interspecific association and interaction among members of this parasite guild.

Seven to 19 host subpopulations were sampled annually at each of two sites in the lagoon from 1981 to 1988. Mixed-species infections constituted only 2.5% of the 5025 infections examined in the study. A Monte Carlo simulation procedure demonstrated that the numbers of such infections were often less than would be expected by chance, especially when the overall prevalence of infection was high. Patterns of association between particular pairs of species depended on whether the species' life histories include redial or only sporocyst larvae. Species that develop as rediae were predominantly negatively associated with other redial species and with most species that develop only as sporocysts. There was weak evidence of positive interspecific association between a few redial and sporocyst-only species, while members of other such pairs were distributed independently. Associations between sporocyst-only species were either weakly positive or neutral.

Snails carrying known infections were marked, released, and recaptured at both study sites. During their exposure in the field, some initial infections were invaded by another parasite species that often excluded the first parasite. The vulnerability of a parasite species to invasion and replacement by another differed among the tested species. Infections of the largest redial species, *Parorchis acanthus,* were especially resistant to replacement, while those of the smallest redial species, *Euhaplorchis californiensis,* were the most frequently excluded. Four other species were invaded or replaced at intermediate rates. The two largest redial species, *P. acanthus* and *Himasthla rhigedana,* were responsible for >90% of the invasions or exclusions. Direct observations showed that the rediae of these species prey on the larval stages of other species, as do the rediae of *Echinoparyphium* sp. This direct form of interspecific antagonism is probably the primary mechanism by which such species exclude others from host snails, as has been widely demonstrated in similar freshwater snail–trematode systems.

While hierarchical, negative interactions prevent the coexistence of species at the level of the individual host, the mark–recapture study showed that rates of exclusion are low for most subordinate species, with the exception of *Euhaplorchis californiensis.* At the level of the host subpopulation, the assemblage of larval trematodes is diverse, and its composition is temporally and spatially variable. There is no trend toward dominance of the assemblage by large redial species as the level of infection rises within aging cohorts or subpopulations of hosts. These patterns of guild structure within host cohorts and subpopulations are consistent with the hypothesis that recruitment processes rather than interspecific interactions primarily determine the composition and relative abundance of species at this regional level.

Several characteristics of snail–trematode systems that may promote regional coexistence of such a large number of potentially interacting parasite species are the isolated and subdivided nature of the host resource, the aggregated distributions of larval stages, and the differential exploitation of different-sized hosts. Many features of this system are consistent with Price's (1980) non-equilibrial view of parasite communities.

Key words: Cerithidea californica; *complex life history; digenetic trematode; guild structure; host–parasite association; interspecific association; interspecific competition; intraguild predation; larval predation; larval recruitment; non-equilibrium; parasite; salt marsh; snail.*

[1] Manuscript received 16 September 1991; revised 25 June 1992; accepted 1 July 1992.

Introduction

Debate over the importance of competition in natural populations and communities has surfaced repeatedly during the past half-century. The latest exchange of views, beginning in the late 1970's, focuses on the role of interspecific competition in structuring natural communities. While the existence of interspecific competition is not disputed, there is strong disagreement about (1) what constitutes "structure" and the degree to which it is exhibited, (2) the relative merits of different types of evidence for competition's role, and (3) the ubiquity of past and present competition (e.g., Diamond 1978, Conner and Simberloff 1979, 1986, Strong et al. 1979, Connell 1980, 1983, Lawton and Strong 1981, Harvey et al. 1983, Schoener 1983, Strong 1983, and related chapters in Strong et al. 1984).

Parasitic organisms have taken a "back seat" during much of this discussion (but see Strong 1983), despite a strikingly similar, but less vociferous, debate that has ensued among investigators of parasite assemblages. The reasons for this oversight are a matter of conjecture, but it is unfortunate because several characteristics of parasite–host systems, particularly the high level of replication they afford and the relative ease with which the use of space as a resource can be quantified (Holmes and Price 1986), make them especially suited for studies of competition. Nevertheless, during the late 1970's and early 1980's several divergent and rather polarized views emerged concerning the ecological and evolutionary role of interspecific competition in assemblages of parasites.

Holmes (1973) concluded from an extensive review of the literature on assemblages of intestinal helminth parasites of vertebrates that the high degree of infection site specificity exhibited by these parasites (Crompton 1973) was primarily a result of strong interspecific competition for such sites in the past, which selected for genetically based niche diversification over evolutionary time ("selective site segregation"). He found little evidence that present-day antagonistic interactions ("interactive site segregation") were responsible for patterns of site occupation. Competitive exclusion of one parasite from the host by another was more common. Based on these observations, Holmes hypothesized that helminth assemblages in vertebrate guts were "mature communities whose diversity was established to an important extent through biotic interactions" (Holmes 1973: 344).

Price (1980) put forward the alternative view that most parasite assemblages are non-equilibrial in nature and that competition, where it occurs, is transient, and has played only a minor role in the organization of parasite communities. Equilibrium is prevented by the inherent complexity of most parasitic life cycles and the associated spatial and temporal variability in host population size and parasite transmission rates. He argued that these conditions should produce low rates of colonization and high rates of extinction, characteristic of non-equilibrial conditions where resources remain unsaturated. He further argued that infection sites are no more specialized than would be true if colonization was random and species did not interact. This is because the organ and tissue systems of vertebrate hosts offer steep resource gradients, and parasitic organisms have independently evolved a relatively high degree of specialization for reasons other than interspecific competition.

A third alternative, coined the "population concentration hypothesis" by Holmes and Price (1986), is really an amalgam of the following two related hypotheses. Rohde (1979, 1982, 1991) suggested that site segregation by helminth parasites of vertebrate hosts simply reflects aggregative behavior that enhances the likelihood of finding mates in low-density populations, and that interspecific competition has played a minor role. Earlier, Sogandares-Bernal (1959) and Martin (1969) suggested that site segregation evolved as a means of avoiding hybridization.

Two related, synthetic theories of parasite community structure have evolved from this debate. Agreeing that evidence exists for both of their earlier views of parasite community structure, Holmes and Price (1986) erected a dichotomous categorization of parasite infracommunities: interactive vs. isolationist. The population of a particular parasite species that infects an individual host has been termed an infrapopulation (Esch et al. 1975, Margolis et al. 1982); the collection of populations of different parasite species within a single host is an infracommunity (Bush and Holmes 1986). Interactive communities are composed of species that readily colonize the host, thereby developing dense infrapopulations that frequently interact with those of co-infecting species. Consistent with the perspective of Holmes (1973), the structures of interactive assemblages are predicted to be in equilibrium, fully saturated with species that are evenly dispersed in resource space. In contrast, isolationist communities correspond to Price's (1980) and Rohde's (1979, 1991) visions of parasite assemblages as composed of species with low probabilities of transmission. As a consequence, their infrapopulations are small and seldom interact with those of other species. Such assemblages are non-equilibrial, lacking a persistent structure. Member species are dispersed individualistically in resource space, and their use of resources is relatively unaffected by the presence of similar species. Holmes and Price (1986) acknowledged the oversimplification of their dichotomous scheme, and soon thereafter Goater et al. (1987) interpreted interactive and isolationist communities as extremes of a continuum.

Kennedy et al. (1986) discerned fundamental differences in the parasitic helminth communities of endothermic and ectothermic vertebrates, based primarily on data from birds and freshwater fishes. They argued that endotherms support more diverse, and potentially

more interactive, assemblages than ectotherms because the former possess more differentiated intestinal tracts, providing more infection sites, consume food at a greater rate due to a higher metabolic rate, and have higher vagility, both of which increase the rate of exposure to infective stages of parasites. In addition, the broad diet, characteristic of many, though not all, endotherms, enhances the diversity of larval parasites that they consume.

Recent research on parasite infracommunities provides mixed support for these synthetic theories (e.g., Goater et al. 1987, Moore and Simberloff 1990, relevant chapters in Esch et al. 1990, Downes 1991, Lotz and Font 1991). As Moore and Simberloff (1990) suggest, the one-dimensional constructs predicted by current theories may be too simple to explain the wide diversity of patterns exhibited by parasite communities of vertebrates. With the limited data available, particularly of an experimental nature, attempts to formulate general theory may well be premature (Simberloff 1990).

It should be apparent from the above remarks that efforts to conceptualize parasite community structure have focused almost exclusively on adult stages of helminth parasites (a notable exception is Price 1980). This is understandable given that adult stages of helminths such as trematodes, cestodes, nematodes, and acanthocephalans are relatively large and therefore easily identified and counted as individuals, as compared, for example, to protozoans or bacteria. Yet even while restricting our attention to helminth parasites, it is important to recognize that their life cycles are often complex, involving not only the definitive vertebrate host but one to several intermediate hosts that commonly are invertebrates. Biotic interactions that exert a strong effect at one stage in such a life cycle may have little or no influence at others.

Several unique features of the association between intermediate invertebrate hosts and larval helminths are likely to promote patterns and processes of infracommunity organization distinct from those that structure assemblages of adult helminths in definitive vertebrate hosts. As patches or islands of habitat for parasites, invertebrate hosts are smaller (both absolutely and relative to the size of infective stages) and less structurally diverse than vertebrate hosts. Invertebrates not only offer fewer target organs to parasites, but the organs that serve as the main foci of infection in invertebrates (e.g., gonad or digestive gland of molluscs) are often more spatially homogeneous than those of vertebrate hosts (e.g., differentiated gut, gills). As a result of this lower habitat diversity, one might expect less partitioning of infection sites among the larval stages of different species than is typically observed among adult helminths in vertebrate hosts. This difference alone suggests that assemblages of larval helminths in invertebrate hosts are potentially more interactive (sensu Holmes and Price 1986) than assemblages of adult forms. Interaction is made all the more likely in the case of digenetic trematodes, the subjects of this study, because redial and sporocyst larval stages reproduce parthenogenetically within the molluscan intermediate host. Thus, one or a few individuals of a particular species can rapidly monopolize host resources (available infection sites or nutrients) and thereby exclude another species or preempt its colonization. In contrast, infrapopulations of adult helminths in vertebrate hosts grow more slowly by recruitment of individual parasites from the external environment.

Populations of invertebrates that serve as intermediate hosts are commonly infected by several species of parasitic larval helminths (Denny 1969, Wright 1971, Brown 1978, Rohde 1982, Lauckner 1980, 1983). To understand better the processes that structure such assemblages, I conducted a long-term study of the diverse guild of larval digenetic trematodes that exploit the salt marsh snail, *Cerithidea californica,* as first intermediate host in their life cycles. In referring to this assemblage as a guild, I am using the term more liberally than Root (1967) may have originally intended when he defined a guild as "a group of species that exploit the same class of environmental resources in a similar way." I have elected to adopt the broader definition of Polis et al. (1989): a guild includes "all taxa in a community that use similar resources (food or space) and thus may compete, regardless of differences in the tactics of resource acquisition." The latter definition seems especially applicable to the assemblage of larval trematodes that infect *Cerithidea.* In spite of some differences in the primary sites of infection and the mechanisms of resource acquisition (see *Materials and methods: Study organisms*), observations made in this study indicate that the species exploit overlapping portions of the pool of available resources (nutrients or infection sites) within the host. Therefore, the potential for direct or indirect negative interaction among the species exists.

The results reported here address three questions: (1) Are the parasite species distributed randomly and independently among available hosts?, (2) What patterns and mechanisms of biotic interaction, if any, occur among species that infect the same host?, and (3) To what degree do such interactions account for guild structure at different hierarchical levels of the host–parasite association? In regard to this third question, an earlier report (Sousa 1990) examined variation in guild structure among host subpopulations, while this paper investigates patterns at two lower levels: within individual hosts and within even-aged cohorts of hosts.

Materials and Methods

Study organisms

Dense populations of the deposit-feeding gastropod *Cerithidea californica,* commonly exceeding 500 individuals/m^2 (McCloy 1979, Race 1981; W. Sousa,

TABLE 1. Species of larval trematodes infecting the salt-marsh snail *Cerithidea californica* in Bolinas Lagoon, Marin County, California.*

Larval type and digenean family	Species code	Species	Primary foci of infection†
		Redial	
Echinostomatidae	ACAN	*Acanthoparyphium spinulosum*	G
	ECHI	*Echinoparyphium* sp.‡	G
	HIMA	*Himasthla rhigedana*	G
Heterophyidae	EUHA	*Euhaplorchis californiensis*	G
	PHOC	*Phocitremoides ovale*	G
Notocotylidae	CATA	*Catatropis johnstoni*	M
Philophthalmidae	PARO	*Parorchis acanthus*	G
		Sporocysts only	
Cyathocotylidae	MESO	*Mesostephanus appendiculatus*	M
	CYA2	small cyathocotylid‡	G/D
Microphallidae	MIC1	small microphallid‡	G/D
	MIC2	large microphallid‡	G/D
Renicolidae	RENB	*Renicola buchanani*	M
	REN2	small renicolid (with stylet)‡	G/D
	REN3	small renicolid (no stylet)‡	M
Schistosomatidae	AUST	*Austrobilharzia* sp.‡	G/D

* List compiled from dissections of over 25 000 snails from eight annual samples, 1981–1988. Species are grouped according to the type of precercarial, intramolluscan larval stage in the life cycle, i.e., those with redial stages and those lacking rediae, having sporocyst stages only (see *Materials and methods: Study organisms*). The four-character codes will designate the species in subsequent tables and figures.

† G: ≥95% of larvae in gonad; G/D: 70% of larvae in gonad, 30% in digestive gland; M: 100% of larvae in mantle region (mean values of replicate visual estimates).

‡ The taxonomic identity of the species has yet to be determined.

unpublished data), inhabit pickleweed (*Salicornia virginica*) marshes and adjacent high intertidal (+1.2–2.1 m above mean lower low water) mudflats and tidal creeks in protected bays and estuaries along the Pacific coast of North America. The species' range extends from Tomales Bay (Marin County, California) to central Baja California, Mexico (Macdonald 1969*a*, *b*). Details of the snail's life history are summarized in Sousa (1983). Sexes are separate and reproduction is iteroparous. Snail larvae undergo direct development within benthic egg strings; there is no planktonic dispersal stage. The maximum life-span is at least 7 yr, based on mark–recapture studies (W. Sousa, *unpublished data*).

Cerithidea is first intermediate host to at least 18 species of digenetic trematodes in California (Martin 1955, 1972, Yoshino 1975, Sousa 1983, 1990, Kuris 1990). In Bolinas Lagoon (Marin County, California), the site of this study, 15 species of trematodes were found in the eight annual samples of snail populations on which this paper is based (Table 1). The life cycles of all but one of these species (Fig. 1) appear to follow the typical digenean sequence (Shoop 1988). The following description of a generalized life cycle begins arbitrarily with the trematode egg, which is deposited in the environment, commonly via the feces of the vertebrate definite host. A free-living miracidium larva hatches from the egg, contacts a snail, and penetrates its epithelium. In some trematode species, the egg is ingested by the snail, hatches in the gut, then the miracidium penetrates the gut wall. Upon penetrating the snail, the miracidium changes into a mother sporocyst, from which either a daughter sporocyst generation, or two or more generations of rediae, are produced asexually. A sporocyst is essentially a closed sac with limited mobility, which absorbs nutrients through its body wall. In contrast, a redia has a well-developed mouth, muscular pharynx, and gut with which it feeds on host tissue directly. In addition, rediae are actively mobile within the tissues they infect. A mother sporocyst is common to all digenean life cycles, but the subsequent production of sporocysts vs. rediae is a species- and family-specific characteristic (Table 1).

The daughter sporocysts or rediae migrate to their final sites of infection, e.g., the gonad, digestive gland, or mantle (Table 1). When mature, they produce free-swimming cercarial larvae, which generally exit the snail in search of a second intermediate host in which they encyst as metacercariae. When an infected second intermediate host is consumed by a definitive vertebrate host, the adult worm develops from the ingested metacercariae, and the cycle is completed. In the exceptional case of schistosome trematodes such as *Austrobilharzia* sp., the metacercarial stage is bypassed; the definitive host is infected directly by the free-living cercariae.

This paper is concerned primarily with the precercarial, intramolluscan stages: sporocysts and rediae. To reiterate, these larval stages of the parasite are not transmitted between snails; a snail only acquires them when penetrated by free-living miracidial larvae or, less commonly, by consuming eggs of the parasite. Trematode eggs, and ultimately miracidia, are carried to the vicinity of the snail population by the definitive

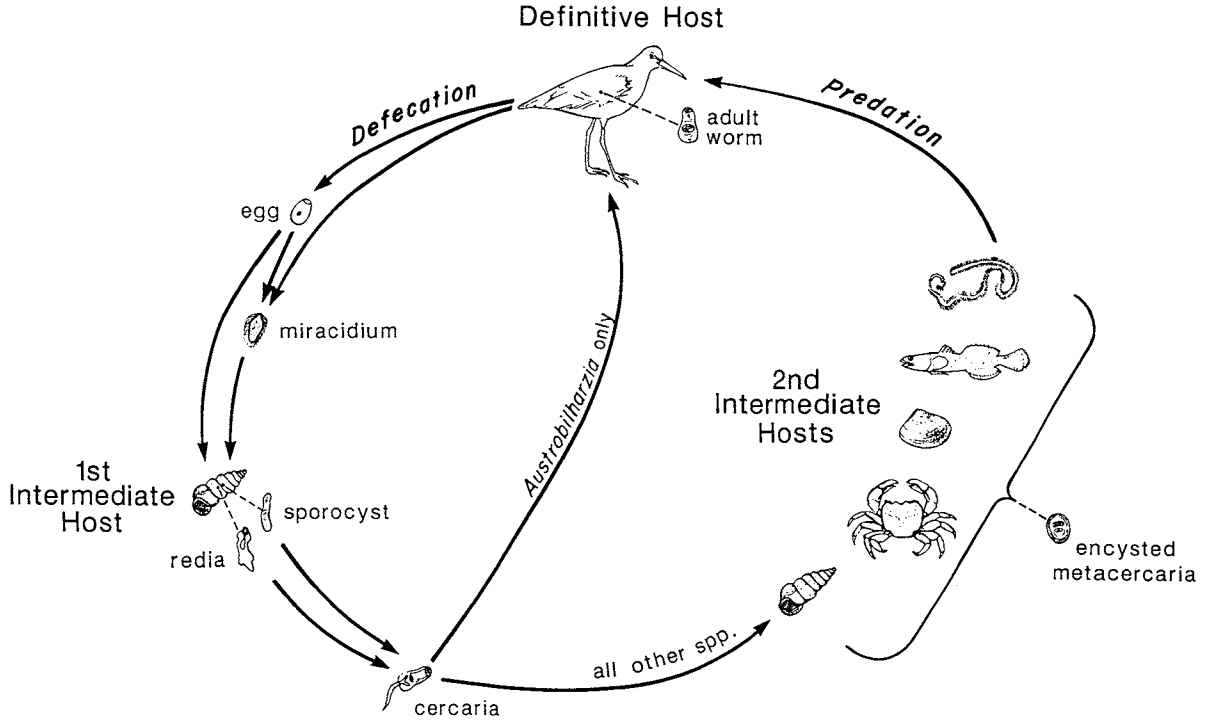

FIG. 1. Generalized life cycle of digenetic trematodes for which the salt-marsh snail *Cerithidea californica* serves as first intermediate host.

host (Fig. 1). Dense infrapopulations of rediae or sporocysts propagate asexually from a single miracidium, often consuming or otherwise destroying most tissue (i.e., resources) in the target organ (see *Materials and methods: Analysis of mixed-species infections*).

The definitive hosts for most of the trematodes that infect *Cerithidea* are probably birds, including shorebirds, waterfowl, gulls, and wading birds, but there is as yet little information concerning the distribution of adult trematodes among potential avian or mammalian hosts that inhabit the study area.

Study sites

The study was conducted in Bolinas Lagoon, located 24 km northwest of San Francisco, California (37°55′ N, 122°41′ W). *Cerithidea* populations and their parasites were sampled at two sites within the lagoon. One site is adjacent to the mouth of Pine Gulch Creek (hereafter PGC site), which flows into the lagoon on its western edge, and the other is at the northeast corner of Kent Island (hereafter KI site). The sites are designated "B" and "C," respectively, on the map of the lagoon that appears in Stenzel et al. (1976), and are ≈750 m apart. The surface sediment at PGC is a poorly sorted, very fine sandy mud and has a considerably higher organic content than the surface sediment at KI, which is a well-sorted, fine to medium sand (Ritter 1969; W. Sousa, *unpublished data*). This variation in sediment quality is related to hydrological and biological differences between the sites (Sousa 1990).

The differential use of lagoon habitats by definitive avian hosts and variation in the abundances of second intermediate hosts between the sites may combine to produce spatial differences in the abundance of infective stages of different trematodes. The freshwater flowing from the creek at PGC is an important resource for birds. Densities of wintering, migrant waterfowl and roosting gulls and terns are much higher at this site than at KI, which lacks a source of freshwater (Point Reyes Bird Observatory and W. Sousa, *unpublished data*). The species composition and abundance of foraging shorebirds and wading birds also differs between the sites, apparently due to the difference in sediment and in the associated benthic invertebrates and fishes on which the birds feed (Stenzel et al. 1976, Page et al. 1979, Quammen 1984). As noted above (see *Materials and methods: Study organisms*), these birds are definitive hosts, and many of their prey are second intermediate hosts, for some of the parasites that infect *Cerithidea* (Robinson 1952, Russell 1960, Badley 1979, Bush 1990, Ching 1990; W. Sousa, *unpublished data*). Since estuarine bird species differ in diet, they harbor different assemblages of parasites (e.g., Russell 1960).

At both study sites, snails are distributed as a series of subpopulations occupying shallow (5–15 cm deep) depressions (hereafter called pans) in the surface of the mudflat that hold standing water at low tide. These pans range in size from slightly less than 1 m^2 to 20 m^2. During most months of the year they are flushed daily by high tides. At PGC, the pans are located along

the interface of the tidal mudflats and the higher elevation, *Salicornia*-dominated marsh. Snails rarely move between them (W. Sousa, *unpublished data*), presumably because the emergent mudflats (which often consist of hard, dried plates of sediment) and the dense stands of pickleweed that border the pans act as physical barriers to snail movement. As a consequence of this lack of adult migration and the absence of a planktonic stage in the snail's life history, the demographies of different subpopulations of snails at PGC vary greatly. The subpopulations differ in size distribution, rate of recruitment, density, and rate of parasitic infection (W. Sousa, *unpublished data*).

At KI, pans occur across the upper tidal mudflat as well as along the mudflat–marsh boundary, as at PGC. Areas between the pans are pockmarked with the conical sediment mounds that mark the burrow openings of a dense population of the ghost shrimp, *Callianassa californiensis*. Snails are present in these surrounding areas, but at much lower densities than in the pans, which are foci of snail feeding and reproduction. The *Callianassa*-dominated areas between the pans remain moist during most low tides, and few of the KI pans have vegetation around their edges. These features, as well as the fact that the average distance between neighboring KI pans is only half that of PGC pans (8 vs. 16 m), probably account for the fact that rates of snail movement between subpopulations is at least three times greater at KI than PGC (W. Sousa, *unpublished data*). As an apparent consequence of this greater exchange of migrants, demographic characteristics of different subpopulations at KI are very similar within any particular year, and they change quite synchronously over time (W. Sousa, *unpublished data*). The influence of habitat structure, migration, and parasitism on the demography of snail populations will be considered in a separate paper.

Sampling procedures

In 1980, 36 subpopulations of *Cerithidea* were selected for long-term monitoring of demographic parameters and parasitic infection, 21 at PGC and 15 at KI. The chosen pans comprised almost all of those that contained snails along the 315 m and 70 m of marsh edge habitat studied at PGC and KI, respectively. Two subpopulations at PGC (Pans 3 and 6) went extinct in the winter of 1980/1981 before the regular sampling program had begun.

Subpopulations at both sites were sampled each August from 1981 through 1987. The PGC pans, but not those at KI, were also sampled in October 1988. The snails were sampled with a 225-cm^2 scoop core that collected all sediment and benthic invertebrates to a depth of 2 cm, sufficient to collect all snails in the area. The contents of each scoop were sieved through 1-mm mesh in the field and returned to the laboratory for analysis. The length (apex to aperture) of each snail was measured to the nearest 0.01 mm; then it was dissected to determine its sex and what species of trematode, if any, infected it.

Five or 10 scoop samples were collected from each pan, depending on the density of snails; the greater number was taken in sparser populations. The scoops were made at regularly spaced intervals along the length of a metric tape transecting each pool, parallel to its long axis. These samples provided estimates of snail density (both young-of-the-year and older individuals) and biomass. To increase the statistical power of tests for interspecific association among larval trematodes, an additional sample of snails was collected from those pans for which the total number of snails ≥ 1 yr old in the scoop samples numbered < 100. This supplemental sample was collected from one to three haphazardly chosen locations within a pool. Starting from the position at which the first individual was collected, all snails (excluding new recruits) from the immediate area were collected until the total sample numbered ≥ 100 individuals; a few collections fell short of this goal. For each pan sampled in a given year, I tested for differences between the size distributions of ≥ 1-yr-old snails in these supplemental collections and in the pooled scoop samples with two-sample Kolmogorov-Smirnov tests (Sokal and Rohlf 1981:440; also known as the Smirnov test, Lindgren 1968:335). In most cases, no significant difference was detected, and the scoop and supplemental samples taken in a particular pan and year were pooled for the analyses described in this paper. In the few cases where the size distributions differed ($P < .05$), only the scoop samples were used. Young-of-the-year snails collected in the scoop samples were never found to harbor trematode infections and are not included in the analyses.

In most cases, snail subpopulations were dense enough that sampling was unlikely to have an impact on population dynamics. However, because of a variety of factors, but especially storm-related disturbance, the density of snails in certain pans sometimes fell to such low levels that I chose not to sample a pan for fear of affecting subsequent dynamics. Also, over the course of the 8 yr, a number of snail populations at both sites did go extinct (along with their parasite assemblages) owing to physical and biological disturbances, including flooding, sedimentation, and alteration of the habitat by burrowing ghost shrimp. For both these reasons, the number of subpopulations sampled at each site varied from year to year, and gradually diminished over the course of the study. The numbers of snails ≥ 1 yr old examined from each site in a given year are listed in Table 2.

Analysis of mixed-species infections

Several statistical procedures were used to evaluate whether different species of larval parasites simultaneously infected the same snail as often as would be expected under a null hypothesis of random and independent assortment of parasites among snails. The

analyses were focused at the level of the host subpopulation within a given year, rather than examining some aggregate sample of pans or years. While this procedure resulted in smaller sample sizes, it avoided the potential bias inherent to analyses of pooled data sets in which the individual samples are heterogeneous with regard to parasite species composition or abundance (see discussions in Cort et al. 1937, Lauckner 1980, Kuris 1990, Sousa 1990, 1992). The expected number of mixed-species infections would be artificially inflated by pooling such heterogeneous samples (analogous to the Wahlund Effect in population genetics, Futuyma 1979: 279), leading one, in some cases, to conclude falsely that the species in question are negatively associated. Assemblages of larval parasites infecting subpopulations of *Cerithidea* are quite variable in space and time (W. Sousa, *unpublished data*), and therefore calculations of the expected number of mixed-species infections are best made on the scale of the individual pan.

The following analyses treat the intensity of infection qualitatively, i.e., a snail was either infected or it was not. While quantitative information on the intensity of infection, e.g., the number of parasite larvae per host, would be valuable, its collection was impractical and unnecessary for the purposes of this study. The redial and sporocyst larval stages that infect *Cerithidea* proliferate rapidly by asexual reproduction within a host. An infected snail may harbor hundreds or even thousands of larvae, too many to count given the number of infected individuals that had to be examined for this study. More importantly, because of the high rate of asexual reproduction, intensity of infection is practically a dichotomous variable; snails were either uninfected or heavily infected; in more than 70% of the infections examined, 70% or more of the target tissues were affected (W. Sousa, *unpublished data*).

As documented below (see *Results: Frequency of mixed-species infections*), few snails in the annual samples were found to be infected by more than one species of trematode. To evaluate the statistical likelihood of this apparent rarity of mixed-species infections, I used a Monte Carlo simulation procedure (Sokal and Rohlf 1981:794) to estimate the one-tailed probability of the observed number of such infections, disregarding the species involved. This probability was determined for each annual sample from a pan. In each replicate simulation, the observed number of infections by each parasite was randomly cast into N snails, where N equalled the size of the sample. The number of simulated hosts that became "infected" by two or more parasite species was counted. This procedure was repeated 10 000 times for each pan. The fraction of replicate simulations in which the number of snails assigned ≥ 2 parasites was $\leq$ the observed number of mixed-species infections is an estimate of the one-tailed probability of the observation. In a small number of snails, the identity of the infecting parasite could not be unambiguously determined (Table 2); these cases were excluded from the analysis.

I then asked whether individual parasite species were distributed independently among hosts. The pattern of association between a particular pair of species, based on their presence/absence in individual snails, can be tested as a 2×2 contingency table. However, assigning probabilities to the outcomes of simultaneous tests of pairwise association among more than two species is difficult for two reasons. Such tests are not independent (Schluter 1984), and the probability of committing type I errors (falsely rejecting a null hypothesis of no association) increases with the number of simultaneous tests (Pielou 1974). While recognizing these limitations, I undertook an exploratory analysis of pairwise associations among larval trematode species, adopting a two-stage statistical procedure that reduced the influence of the above problems. First, for each annual sample from a pan, I tested patterns of pairwise association between all species that were represented by at least one infection in the sample. A Fisher exact test was performed on each 2×2 table. While infections by different species of larval trematodes have most commonly been found to be negatively associated in previous studies of intramolluscan trematode guilds (especially in redial–redial and redial–sporocyst pairs), there are some well-documented cases of transient or persistent positive associations between certain species (Lim and Heyneman 1972, Kuris 1990, Sousa 1992). As this analysis was of an exploratory nature, I applied two-tailed tests.

As discussed above, probabilities generated from simultaneous tests of pairwise association are suspect due both to the lack of independence among the tests and the increase in the experimentwise error rate with the number of tests performed. The second stage of the test procedure (developed by E. Adams, Department of Biology, University of Rochester, Rochester, New York) determined which of the pairwise associations within a given sample are significant when the experimentwise error rate (α) is held at .05. This second stage of the analysis was based on a simulation procedure identical in its initial steps to the Monte Carlo simulations described above. For each annual sample from a pan, the observed number of infections by each parasite was randomly cast into the observed number of snails. Then, for parasite species represented by at least one infection in the sample, all possible pairwise associations were tested with two-tailed Fisher exact tests. For each simulation, a cumulative frequency distribution of the resulting pairwise probabilities, ordered from smallest to largest, was constructed. This procedure was repeated 1000 times for each sample to generate a set of 1000 cumulative frequency distributions of the probabilities of pairwise association under the null hypothesis that parasites are assorted randomly and independently among snails within the sample.

Tests of association for observed distributions of

TABLE 2. Counts of uninfected and infected snails for each census year and site, pooled across pans. PGC = Pine Gulch Creek; KI = Kent Island. See Table 1 for key to species codes.

Type of parasite infection	1981		1982		1983		1984		1985	
	PGC	KI	PGC	KI	PGC	KI	PGC	KI	PGC	KI
Uninfected	3451	1588	2500	1506	992	457	1736	885	1654	1219
Single-species infections: life history and species of parasite										
Redial										
ACAN	33	9	24	12	118	37	31	32	9	14
ECHI	205	17	71	2	13	2	148	2	45	0
HIMA	54	80	48	28	92	32	59	40	51	9
EUHA	28	106	27	37	6	22	6	18	4	9
PHOC	0	0	0	0	0	0	0	1	0	0
CATA	59	30	13	7	4	0	15	5	9	6
PARO	134	97	125	32	58	16	97	64	69	7
UNID*	6	2	1	2	2	2	7	2	3	0
Sporocysts only										
MESO	0	0	0	0	1	0	1	0	0	0
CYA2	3	2	4	0	55	1	18	5	46	2
MIC1	56	146	18	20	5	12	1	10	3	2
MIC2	0	0	0	0	0	0	0	0	0	0
RENB	1	4	1	1	1	0	0	0	0	0
REN2	13	52	14	20	13	5	3	2	39	3
REN3	0	0	1	0	0	0	0	0	0	0
AUST	0	0	0	1	0	0	0	0	0	0
UNID*	7	0	2	0	0	0	2	0	3	1
Total single-species infections	599	545	349	162	368	129	388	181	281	53
Multiple-species infections: life history and species of parasites										
Redial–redial										
ACAN-EUHA	0	0	0	1	0	0	0	0	0	0
ACAN-HIMA	0	0	0	0	2	0	0	0	0	1
ECHI-HIMA	1	0	1	0	0	0	0	0	0	0
ECHI-PARO	0	0	0	0	0	0	0	0	0	0
HIMA-EUHA	0	0	0	0	7	0	0	0	0	0
HIMA-PARO	0	0	0	0	0	0	0	0	0	0
EUHA-PARO	0	1	0	0	0	1	0	0	0	0
CATA-PARO	0	0	1	0	0	0	0	0	0	0
Sporocyst–sporocyst										
CYA2-MIC1	0	0	0	0	1	0	0	0	0	0
CYA2-RENB	0	0	0	0	0	0	0	0	0	0
MIC1-REN2	1	5	0	0	0	0	0	0	0	0
MIC1-AUST	0	0	0	0	0	1	0	0	0	0
RENB-REN2	0	0	0	0	0	0	0	0	1	0
Redial–sporocyst										
ACAN-REN2	0	0	0	0	0	1	0	0	0	0
ACAN-AUST	0	0	0	0	1	0	1	0	0	0
ECHI-MESO	0	0	2	0	0	0	0	0	0	0
ECHI-MIC1	1	0	1	0	0	0	0	0	0	0
ECHI-RENB	0	0	0	0	0	0	0	0	0	0
ECHI-REN2	0	0	0	0	0	0	1	0	0	0
ECHI-AUST	0	0	0	0	0	0	0	0	0	0
HIMA-CYA2	0	0	0	0	0	0	0	0	0	0
HIMA-MIC1	0	1	0	0	0	1	0	0	0	0
HIMA-RENB	1	0	0	0	0	0	0	0	0	0
HIMA-REN2	0	1	0	0	0	1	0	0	0	0
HIMA-AUST	0	0	0	0	1	0	0	0	1	0
EUHA-MIC1	0	0	0	2	0	0	0	0	0	0
EUHA-REN2	0	2	0	0	0	0	0	0	0	0
EUHA-AUST	0	0	0	1	0	0	0	0	0	0
CATA-MIC1	1	3	0	0	0	0	0	0	0	0
CATA-REN2	1	2	0	2	0	0	0	0	0	0
PARO-CYA2	0	0	0	0	0	0	0	0	0	0
PARO-MIC1	0	0	0	0	0	0	0	0	0	0
PARO-REN2	0	0	0	0	0	0	0	0	0	0
PARO-AUST	0	0	0	0	0	0	1	0	0	0
CATA-MIC1-REN2	0	1	0	0	0	0	0	0	0	0
Total multiple-species infections	6	16	5	6	12	5	3	0	2	1
Total infected	605	561	354	168	380	134	391	181	283	54
Total snails sampled	4056	2149	2854	1674	1372	591	2127	1066	1937	1273

* Species identification uncertain.

TABLE 2. Continued.

1986		1987		1988	Total	
PGC	KI	PGC	KI	PGC	PGC	KI
1346	958	1022	780	735	13436	7393
37	14	9	8	6	267	126
146	6	296	2	320	1244	31
55	14	46	12	125	530	215
0	3	3	0	2	76	195
0	0	0	0	0	0	1
11	5	29	5	48	188	58
72	10	102	1	167	824	227
1	0	2	0	0	22	8
1	0	0	0	1	4	0
55	1	14	1	35	230	12
6	2	5	7	43	137	199
0	0	1	0	0	1	0
4	0	3	0	0	10	5
22	4	13	1	63	180	87
0	0	0	0	0	1	0
0	0	0	0	2	2	1
1	0	0	0	0	15	1
411	59	523	37	812	3731	1166
0	0	0	0	0	0	1
0	0	0	0	0	2	1
0	0	0	0	0	2	0
0	0	1	0	0	1	0
1	0	0	0	1	9	0
0	0	2	0	1	3	0
0	0	0	0	0	0	2
0	0	0	0	0	1	0
0	0	0	0	10	11	0
0	0	1	0	0	1	0
0	0	0	0	0	1	5
0	0	0	0	0	0	1
0	0	0	0	0	1	0
0	0	0	0	0	0	1
0	0	0	0	0	2	0
0	0	0	0	0	2	0
0	0	3	0	18	23	0
1	0	0	0	0	1	0
0	0	5	0	2	8	0
0	0	1	0	2	3	0
0	0	0	0	1	1	0
0	0	0	0	9	9	2
0	0	0	0	0	1	0
0	0	1	0	1	2	2
0	0	0	0	0	2	0
0	0	0	0	0	0	2
0	0	0	0	0	0	2
0	0	0	0	0	0	1
1	0	1	0	4	7	3
0	0	1	0	1	3	4
1	0	0	0	0	1	0
0	0	0	0	1	1	0
1	0	0	0	0	1	0
0	0	0	0	0	1	0
0	0	0	0	0	0	1
5	0	16	0	51	100	28
416	59	539	37	863	3831	1194
1762	1017	1561	817	1598	17267	8587

parasites were judged to reject the null hypothesis if they yielded cumulative frequency distributions with an excess of small probabilities. The smallest P value from tests of association with the observed data was judged significant if it was smaller than the smallest P value from all but the most extreme 5% of simulations. If this smallest value was significant, then the second smallest P value from tests on the observed data was examined. It was judged significant if smaller than the second smallest P value from all but the most extreme 5% of null simulations. If this second smallest P value was significant, the next smallest P value was evaluated, and so forth. In identifying the 5% tail of extreme values, if two cumulative distributions tied for the smallest P value, they were compared by reference to the next smallest P value. This process was continued until the tie was resolved. The test procedure produced a list of associations among parasites confirmed to be significant under this more conservative community-wide test.

Mark–recapture of infected snails

To evaluate both the invasiveness and resistance to invasion of infections by particular parasites, I conducted a mark–recapture study of snails carrying known infections. The general procedure was as follows. Approximately 2 wk prior to a release, a large sample of snails was collected from the site at which the release was to be made. The snails were then screened in the laboratory for the presence of mature infections by inducing cercarial larvae to shed (see Sousa 1983 for details). The cercariae of different trematodes have distinctive morphologies allowing identification of the resident parasite(s). Each snail that shed cercariae was individually marked with both a numbered and a colored adhesive tag, which were glued to the shell with clear waterproof epoxy. Within a week after tagging, the snail was released in the field. A series of such releases was made at both study sites from 1981 to 1987; totals of $\approx$1600 and 400 infected snails, respectively, were marked and released at PGC and KI. These releases were made into 15 pans at PGC and 11 at KI. The pans were carefully searched for marked snails at irregular intervals, ranging from $\approx$3 mo to 1 yr. Recaptured, living individuals were returned to the laboratory, where they were dissected and their parasites identified.

To maximize the likelihood that a given infection was challenged by miracidial larvae of another species, the analysis reported here includes only those individuals that spent at least 100 d in the field, including a minimum of 60 d between September and February when numbers of migratory shorebirds and waterfowl on the lagoon are high (Page et al. 1979; W. Sousa, *personal observations*). A total of 946 snails at PGC and 224 snails at KI met these criteria (Table 3).

In this kind of study, one has little control over either the length of time that any particular snail is exposed

TABLE 3. Summary statistics on periods of exposure (release to recapture) in the field for marked snails known to be infected with trematode parasites. See Table 1 for key to species codes. Total snail days is the sum of all individual exposure periods for snails infected with the indicated parasite species.

Site	Parasite	N	No. days of exposure in field					Total snail days
			Mean	SD	Median	Min.	Max.	
Pine Gulch Creek	ACAN	88	363.3	181.4	379.0	122	1136	31 968
	ECHI	102	359.8	136.9	410.0	105	736	36 702
	HIMA	96	375.3	199.8	372.0	122	1121	36 028
	EUHA	24	369.9	132.3	359.0	122	754	8878
	PHOC	1	372.0	...	372.0	372	372	372
	CATA	2	366.5	140.7	366.5	267	466	733
	PARO	314	395.6	168.1	372.0	122	1152	124 204
	CYA2	315	367.5	162.5	372.0	122	1413	115 752
	MIC1	3	750.7	653.3	375.0	372	1505	2252
	RENB	1	323.0	...	323.0	323	323	323
Kent Island	ACAN	36	318.0	167.8	344.0	128	745	11 448
	ECHI	8	419.5	126.7	373.0	373	733	3356
	HIMA	46	355.5	167.4	355.0	128	802	16 353
	EUHA	33	347.8	196.3	338.0	128	745	11 477
	PHOC	1	373.0	...	373.0	373	373	373
	PARO	78	439.8	175.2	373.0	128	746	34 307
	CYA2	22	321.0	180.8	338.0	128	723	7063

in the field or the number of snails carrying a particular infection that is recaptured in a search. In addition, as noted earlier, the prevalence of different parasites varies widely in space and time so it was difficult to obtain balanced sample sizes. Given these complications, the mean and median days of exposure were remarkably similar across parasite species and sites (Table 3).

Differences among parasite species in the percentages of infections that changed to another species were examined separately for each study site. First I evaluated, with a χ^2 test for goodness of fit, whether variation in the observed percentages of changed infections among parasite species simply reflected differences in the length of time that snails infected by the different species were exposed in the field. The expected number of changes for a particular parasite was computed as the proportion that species comprised of the total snail-days that infected snails were exposed in the field (last column of Table 3) times the total number of observed changes. If this hypothesis was rejected, I tested the independence of parasite species and rate of change with χ^2 tests and Tukey-type multiple comparison tests for proportions (Zar 1984: 401). Then, for each species, I tested with χ^2 tests the null hypothesis that the percentage of infections that changed did not differ between sites.

My interpretation of the observed patterns of species replacement could be questioned on two grounds. First, some mixed-species infections may go undetected in the initial laboratory screening if one or more infections are immature and do not shed cercariae. In those instances in which a mixed infection was initially overlooked (and for the sake of discussion involves no more than two species, which is typically the case, see *Results: Frequency of mixed-species infections*), one of the following two misinterpretations would be made: (1) if the undetected (nonshedding) species has been excluded by the detected (shedding) species prior to recapture, only the latter will be found when the snail is dissected, and the case will be misidentified as a persistent, single-species infection of the shedding species, and a case of antagonistic exclusion will be missed; or (2) if the overlooked mixed-species infection persists, it will be detected upon dissection, and the case will be identified as an invasion by a new species of a single-species infection. Fortunately, such errors will be infrequent since mixed-species infections are rare (see *Results: Frequency of mixed-species infections*).

Second, my interpretation of the changes in parasite species composition within individual snails also assumes that snails do not lose their infections in the field and become reinfected prior to recapture. In other words, I assume that the loss of one species is a consequence only of the invasion of a second. This assumption seems valid given the rarity with which recaptured, supposedly infected snails have been found to be uninfected upon dissection. Of the 1174 "infected" snails recaptured in this study, only 4 were uninfected when dissected. Similarly, I only observed 1 "lost" infection out of 188 parasitized snails in an earlier study of the influence of parasites on snail growth (Sousa 1983). In related experimental field studies involving several hundred infected snails (to be reported separately) I did not record any cases of parasitized snails losing an infection. It is quite possible that the few cases in which an infection appeared to have been lost were either tagging errors or situations in which one or a few free-swimming cercariae of a parasite had entered the mantle cavity of an uninfected snail and then emerged when the snail was submerged in seawater during the shedding procedure. Consequently, the 4 cases of lost infections have been dropped from the analyses reported in this paper. Observations from this and other studies (Kuris 1990) strongly support

the assumption that larval trematode infections persist for the life of the snail, changing only when invaded by another parasite species.

Direct observations of interspecific interactions

While dissecting snails for the studies described here and in related experimental studies mentioned above, I encountered mixed-species infections in which redial larvae of one species were preying on rediae, sporocysts, or cercariae of the other. All such encounters were recorded and many were photographed.

Temporal changes in the assemblage of parasites within a cohort of snails

The results of the mark–recapture study and direct observations of interspecific interactions identified two species, *Parorchis* and *Himasthla,* as dominants in antagonistic interactions with other species of larval trematodes (see *Results*). If these two species exclude infections of other species at a higher rate than the latter establish new infections, the assemblage of parasites within a cohort of snails should come to be dominated by *Parorchis* or *Himasthla* as the cohort ages.

To evaluate whether the relative abundance of these two species increased with time, I first divided the annual samples of snails from each pan into age classes. Size/age classes within a sample of snails were distinguished by constructing normal probability plots of the cumulative distribution of snail lengths and visually determining points of inflection between adjacent size classes as described by Harding (1949) and Cassie (1954). A variety of computer-based algorithms for separating a polymodal size distribution into component size classes is available; however, such programs could not easily be applied to *Cerithidea* size distributions because the shapes of the component sub-distributions were highly variable, making it difficult to apply standard curve-fitting procedures. In addition, snail growth rate varies with sex, presence vs. absence of parasitic infection, and species of infecting trematode (W. Sousa 1983, and *unpublished data*). Consequently, final assignments to age class were only made after inspecting separate probability plots for uninfected male, uninfected female, and parasitized snails. Analysis of the plotted distributions by eye proved the most effective and efficient means of separating size classes. The correspondence between size class and age class was based on independent measurements of snail growth rates (W. Sousa, *unpublished data*).

Next, for each age class distinguished within a given annual sample from a pan that contained at least 10 infections, I calculated the proportion of the infections that were by *Parorchis* or *Himasthla.* To determine if this proportion varied with the age of the cohort, the data were examined in two ways. First, I tested for a significant difference in the mean proportion of infections that were by *Parorchis* or *Himasthla* among age classes, disregarding the census year. Second, for individual cohorts that could be followed for at least three consecutive years, I plotted the proportion against cohort age.

Results

Frequency of mixed-species infections

Very few snails examined in the annual samples were infected by more than one species of parasite (Table 2). Of the total of 5025 infections, 4897 (97.45%) were single-species infections, 127 (2.53%) were double infections, and 1 (0.02%) snail was infected by three species. As determined by Monte Carlo simulation, the number of mixed-species infections was significantly fewer than expected by chance ($P \leq .05$) in a number of the pan samples from PGC in each year of the study (Table 4). At KI, mixed-species infections were shown by simulation to be significantly fewer than expected only in seven pan samples taken in 1981 (Table 5). The observation that mixed-species infections are often less common than one would expect by chance is not unique to samples collected in the summer and fall. Such infections were chronically rare in monthly samples from four snail subpopulations collected over a 2-yr period (W. Sousa, *unpublished data*). Infected snails were present in each of the 96 monthly samples, but mixed-species infections were found in only 25 (26.0%) of the samples and 36 (1.7%) of the 2066 infected snails examined in the study.

Monte Carlo probabilities were inversely related to the prevalence of infection: the higher the rate of infection in a sample of snails, the lower the probability that the deficit of mixed-species infections was due to chance (Fig. 2). At both sites, as the infection rate approached 30%, this probability tended to drop below .05. In each year at PGC, several pans had rates of infection that exceeded 30%, while at KI this only occurred in a few pans in two years (Fig. 3). In general, the prevalence of infection was lower at KI than PGC, and consequently negative associations were detected less often at the former site.

Pairwise tests of interspecific association showed that in a number of samples individual parasite species were not distributed independently of others. Two-tailed Fisher exact tests yielded probabilities $\leq .05$ for the pairs of parasites listed in Table 6. Fewer such pairs were identified at KI than PGC due to the lower infection rates at the former site. The simulation procedure that evaluated the statistical likelihood of these probabilities showed that 26 of the 46 would be obtained $\leq 5\%$ of the time by chance, and can therefore be considered statistically significant. Lack of independence and the large number of pairwise tests conducted on each sample could account for the low exact test probabilities in the remaining 20 cases. Additional study is needed to determine whether the 11 associations

TABLE 4. Ratios of observed/expected numbers of mixed-species infections for each annual sample from a host subpopulation (pan) at PGC. The expected value is the median number of mixed-species infections obtained in 10 000 Monte Carlo randomizations (see *Materials and methods: Analysis of mixed-species infections*). The probability associated with each ratio is the proportion of total simulations that yielded ≤ the observed number of mixed-species infections. Subpopulations in pans 3 and 6 went extinct in the winter of 1980/1981 before the annual sampling program had begun.

Pan no.	Year							
	1981	1982	1983	1984	1985	1986	1987	1988
1	0/10***	2/14***	···†	···	···	···	···	···
2	0/7***	1/0	···	···	···	···	···	···
4	0/1	0/3*	2/5*	0/0	0/1	0/3*	1/3	9/21***
5	0/11***	···	···	···	···	···	···	···
7	0/1	0/1	1/3	0/0	0/0	···	0/1	0/5***
8	0/2	···	0/1	0/0	0/0	1/6**	1/0	0/1
9	0/0	···	0/0	0/0	0/0	1/1	0/2	23/34***
10	0/0	0/0	0/4**	0/0	1/5*	0/0	1/2	2/5
11	0/1	0/0	0/2	0/0	0/4**	0/0	0/1	0/12***
12	0/3*	1/0	1/6***	0/1	0/2	0/0	0/2	0/11***
13	0/2	0/0	1/5**	1/2	0/0	1/4*	2/9**	···
14	1/5*	0/1	3/6	0/3*	0/0	0/5**	0/2	8/28***
15	2/4	0/1	···	···	···	···	···	···
16	1/2	0/1	2/2	0/5**	0/2	0/0	0/0	0/0
17	1/3	1/4*	2/6*	0/3**	0/0	0/0	1/1	4/28***
18	1/1	0/0	···	···	···	···	···	···
19	0/0	0/0	0/2	0/3*	0/1	0/5**	2/16***	1/9***
20	0/0	0/0	0/1	1/8***	1/1	0/3*	4/10**	3/17***
21	0/0	0/0	0/0	1/1	0/0	2/8**	4/12***	1/18***

* $P < .05$, ** $P < .01$, *** $P < .001$.
† Ellipses (···) indicate snail subpopulations that went extinct or were not sampled.

represented by these 20 ambiguous low probabilities are anything other than spurious.

In all, 13 distinct interspecific associations were represented among the 26 statistically unlikely probabilities; some associations were demonstrated to be significant in more than one year or site (Table 6). Ten of these 13 associations were consistently negative, including all 4 associations between redial and redial species, and 6 out of 9 of those between redial and sporocyst species. The other 3 redial–sporocyst associations were positive: *Acanthoparyphium–Austrobilharzia, Himasthla–Austrobilharzia,* and *Catatropis*–small renicolid with stylet. In each of these positive associations, one member of the pair was represented solely in 1 or 2 double infections in the sample, so the test is rather weak. For none of the sporocyst–sporocyst pairs at either site was the null hypothesis of independent distribution convincingly rejected.

TABLE 5. Ratios of observed/expected numbers of mixed-species infections for each annual sample from a host subpopulation (pan) at KI. The expected value is the median number of mixed-species infections obtained in 10 000 Monte Carlo randomizations (see *Materials and methods: Analysis of mixed-species infections*). The probability associated with each ratio is the proportion of total simulations that yielded ≤ the observed number of mixed-species infections.

Pan no.	Year						
	1981	1982	1983	1984	1985	1986	1987
1	0/1	0/0	2/3	0/1	0/0	0/0	0/0
2	0/5**	2/0	1/3	0/2	0/0	0/0	0/0
3	1/3	0/0	0/2	0/2	0/0	0/0	0/0
4	2/5	0/0	1/1	0/1	0/0	0/1	0/0
5	1/3	0/0	···†	···	0/0	···	···
6	2/6*	0/0	···	0/1	0/0	0/0	0/0
7	0/5**	0/0	0/0	0/1	0/0	0/0	0/0
8	1/4	1/0	···	0/1	1/0	0/0	0/0
9	2/2	0/0	0/1	···	0/0	0/0	···
10	0/5**	1/2	···	0/1	0/0	···	···
11	0/3*	0/0	···	···	···	···	···
12	1/5**	0/0	···	···	···	···	···
13	0/4**	1/0	···	···	···	···	···
14	3/5	0/0	1/0	···	···	···	···
15	3/5	1/3	···	···	···	···	···

* $P < .05$, ** $P < .01$.
† Ellipses (···) indicate snail subpopulations that went extinct or were not sampled.

Mark–recapture of infected snails

Ninety-seven of the 1170 initial infections in marked and recaptured snails were replaced or invaded by another species during their periods of release in the field. Sample sizes for six of the initial species released at PGC were sufficiently large to ensure expected values ≥1.0 for χ^2 statistical analysis: *Acanthoparyphium, Echinoparyphium, Himasthla, Euhaplorchis, Parorchis,* and the small cyathocotylid. At KI, the same was true for all but *Echinoparyphium.*

At PGC, patterns of species replacement did not reflect the proportions of total snail days that the six species were exposed in the field (Fig. 4, $\chi^2 = 138.69$, df = 5, $P < .001$). At KI the patterns of replacement also did not match those expected solely on the basis of exposure times ($\chi^2 = 11.03$, df = 4, $P = .026$). When data from both sites are combined, the null hypothesis that days of exposure explained species replacement rates was strongly rejected ($\chi^2 = 105.72$, df = 5, $P < .001$).

The percent of infections that changed (Fig. 4) differed strongly among trematode species at PGC ($\chi^2 =$

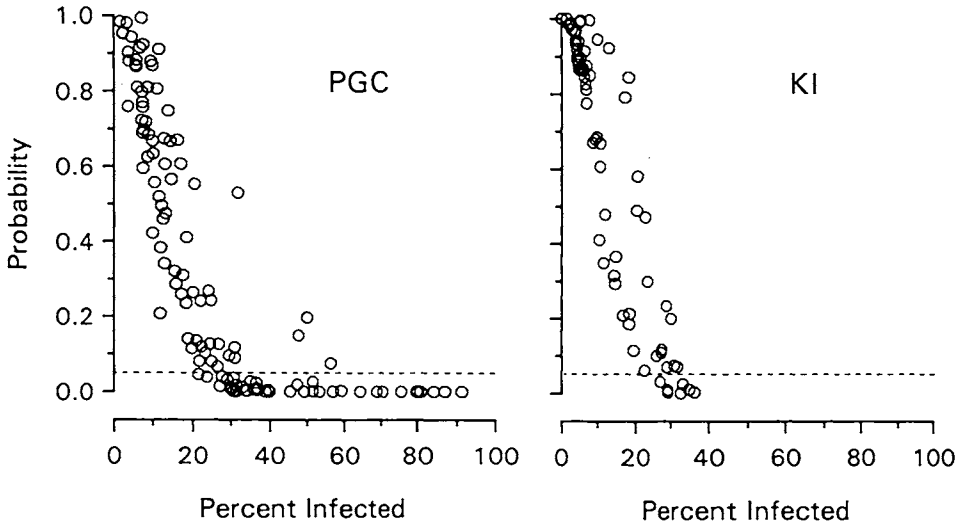

FIG. 2. Relationship between the probability of obtaining ≤ observed number of mixed-species infections by chance and the percent of the host subpopulation that was infected by larval trematodes. Probability was estimated by Monte Carlo simulation for each subpopulation sampled at each of the two sites (as described in *Materials and methods: Analysis of mixed-species infections*). PGC = Pine Gulch Creek; KI = Kent Island. Dashed horizontal lines indicate $P = .05$.

146.29, df = 5, $P < .001$), and to a lesser degree at KI ($\chi^2 = 9.46$, df = 4, $P = .051$). Multiple comparison testing (Table 7) showed infections of *Euhaplorchis* to be the most vulnerable to invasion at PGC, and those of *Parorchis* most resistant. There was no detectable difference in the likelihood of change among the other four species. *Euhaplorchis* infections were also most frequently invaded at KI, more so than all the species except *Himasthla*. No difference was detected among the replacement rates of the other five species. When data from both sites were combined the statistical pattern of differences among species was identical to that at PGC.

When rates of change for each species were compared between study sites, *Euhaplorchis* infections were replaced or invaded by other species more often at PGC than KI ($\chi^2 = 16.00$, df = 1, $P < .001$). Difference in exposure (snail days) between sites does not explain this pattern since rates of change did not match those expected based on the number of days that snails infected with this species were exposed at the two sites ($\chi^2 = 8.58$, df = 1, $P = .003$). For none of the other

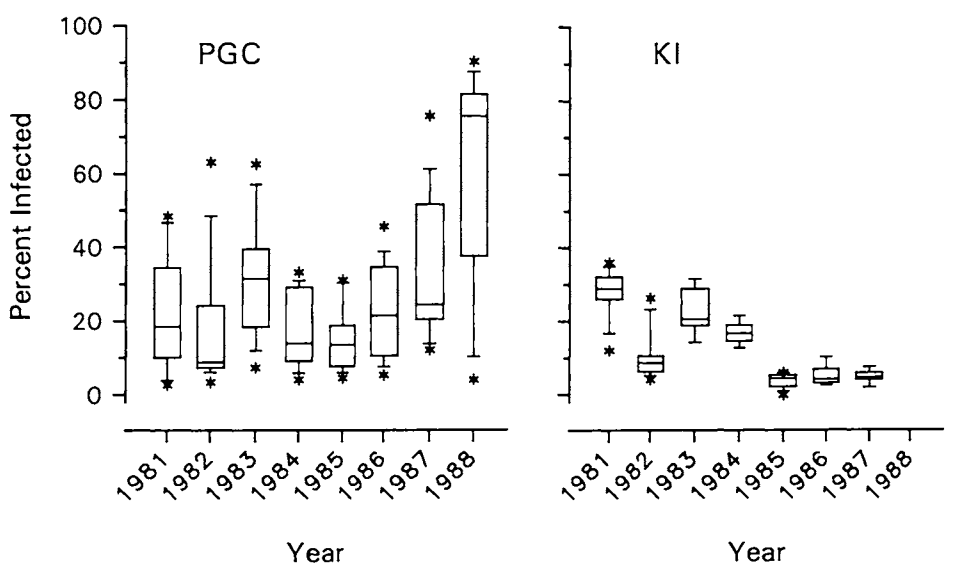

FIG. 3. Prevalence of larval trematode infections in host subpopulations at each site over the course of the study. Data are medians (horizontal line inside box), 25th and 75th percentiles (lower and upper limits to the box), 10th and 90th percentiles (caps on vertical bars), and 5th and 95th percentiles (asterisks). See Tables 4 and 5 for numbers of pans (shallow depressions) sampled in each year at PGC and KI, respectively. KI was not sampled in 1988.

TABLE 6. Tests of pairwise associations among larval trematode species parasitizing *Cerithidea californica* snails. Fisher exact tests were performed for all pairs of species in each pan sampled in the annual collections at the two study sites (PGC and KI). Data show the expected numbers of double infections (Exp) under the null hypothesis of random association, the observed number of such infections (Obs), and the probability levels. Exp = $N_{Sp1} \times N_{Sp2}/N_{total}$, where N_{Sp1} and N_{Sp2} are the numbers of infections of Species 1 and 2, respectively, in the sample, and N_{total} is the total number of snails in the sample. A simulation procedure identified those exact test probabilities that would occur <5% of the time by chance, i.e., when the experimentwise error rate was held at 0.05 (see *Materials and methods: Analysis of mixed-species infections*). Tests of association yielding probabilities >.05 are not reported. See Table 1 for key to species codes.

Parasite pair (Sp1–Sp2)	Year	Pan	N_{total}	Single infections		Double infections	Fisher exact two-tailed *P*	Total number of tests per pan	Experimentwise error rate
				N_{Sp1}	N_{Sp2}	(Obs/Exp)			
Pine Gulch Creek (PGC)									
				Redia–redia associations					
ACAN–ECHI	1981	5	160	19	38	0/4.5	.0075	45	*
ECHI–HIMA	1988	4	79	27	31	0/10.6	.0000	15	*
		9	122	54	7	0/3.1	.0171	45	*
		14	136	37	32	0/8.7	.0000	28	*
		17	180	35	20	0/3.9	.0151	28	*
ECHI–PARO	1981	1	207	64	16	0/4.9	.0034	10	*
	1982	1	112	45	13	0/5.2	.0015	28	*
	1984	20	267	39	25	0/3.7	.0323	28	NS
	1987	13	126	42	14	1/5.1	.0188	18	NS
		19	119	70	23	0/13.5	.0000	10	*
		20	130	59	11	0/5.0	.0010	7	*
		21	131	30	19	0/4.4	.0068	21	*
	1988	9	122	54	16	0/7.1	.0001	45	*
		12	121	35	11	0/3.2	.0324	28	NS
		14	136	37	18	0/4.9	.0033	28	*
		17	180	35	25	0/4.9	.0050	28	*
		19	69	44	7	0/4.5	.0004	15	*
		20	103	42	12	0/4.9	.0013	36	*
		21	94	25	23	0/6.1	.0003	21	*
HIMA–EUHA	1983	16	66	8	0	2/0.3	.0210	10	NS
	1986	21	155	5	0	1/0.0	.0387	36	NS
HIMA–PARO	1988	11	74	15	24	0/4.9	.0016	15	*
				Redia–sporocyst associations					
ACAN–AUST	1984	20	267	1	0	1/0.0	.0075	28	*
ECHI–CYA2	1988	9	122	54	9	0/4.0	.0045	45	*
ECHI–MIC1	1982	1	112	45	7	0/2.8	.0403	28	NS
		2	278	6	1	1/0.1	.0498	15	NS
	1988	19	69	44	4	0/2.6	.0146	15	*
ECHI–REN2	1988	17	180	33	29	2/5.3	.0473	28	*
HIMA–RENB	1981	16	490	11	0	1/0.0	.0245	36	NS
HIMA–REN2	1988	17	180	20	31	0/3.4	.0270	28	*
HIMA–AUST	1985	20	182	1	0	1/0.0	.0110	10	*
CATA–MIC1	1981	18	169	1	3	1/0.0	.0469	15	NS
	1986	9	153	0	1	1/0.0	.0131	28	NS
	1987	21	131	2	1	1/0.0	.0454	21	NS
CATA–REN2	1981	14	106	4	0	1/0.0	.0472	28	NS
PARO–MIC1	1988	9	122	16	36	0/4.7	.0029	45	*
PARO–REN2	1988	17	180	25	31	0/4.3	.0090	28	*
PARO–AUST	1984	21	194	8	0	1/0.0	.0464	10	NS
				Sporocyst–sporocyst associations					
CYA2–MIC1	1988	14	136	7	7	3/0.7	.0264	28	NS
CYA2–RENB	1987	8	86	2	0	1/0.0	.0349	21	NS
RENB–REN2	1985	10	134	0	3	1/0.0	.0229	21	NS
Kent Island (KI)									
				Redia–redia associations					
ACAN–HIMA	1985	8	134	2	1	1/0.0	.0444	6	NS
				Redia–sporocyst associations					
HIMA–MIC1	1983	4	82	2	0	1/0.0	.0366	6	NS
CATA–REN2	1981	8	121	2	1	1/0.0	.0492	21	NS
	1982	2	114	0	1	2/0.1	.0005	10	*
				Sporocyst–sporocyst associations					
MIC1–AUST	1983	14	83	1	0	1/0.0	.0241	6	NS

* $P < .05$, NS $P > .05$.

species was there a significant difference in rate of species replacement between sites (range of χ^2: 0.24–1.28, df = 1, $P > .25$).

Two redial species, *Himasthla* and *Parorchis,* were responsible for >90% of the invasions or exclusions in mark–recaptured, infected snails (Table 8). Infections of these species, in turn, were invaded or replaced almost exclusively by each other. Patterns were similar at both study sites.

Direct observations of interspecific interactions

In 41 mixed-species infections involving the species *Himasthla, Parorchis,* or *Echinoparyphium,* I observed rediae of these species consuming larvae (rediae, sporocysts, or cercariae) of the co-occurring parasite (Table 9, Fig. 5). *Himasthla* accounted for 61%, *Parorchis* 17%, and *Echinoparyphium* 22% of these attacks. *Parorchis* was only observed preying on redial species, *Echinoparyphium* only on sporocyst species, and *Himasthla* on several species of each life history. I also observed one incidental case of *Catatropis* rediae catching and consuming cercariae of *Euhaplorchis* when both were immersed in vitro in seawater within a glass petri dish containing two snails singly infected with the two species. No cases of cannibalism were observed.

Temporal change in the assemblage of parasites within a cohort of snails

At neither site was there a detectable difference among age classes of snails in the mean proportion of infections that were by *Parorchis* or *Himasthla* (Table 10). The same result obtained when the proportions of infections attributable to each species were analyzed separately (W. Sousa, *unpublished data*). To examine temporal trends within individual cohorts, I plotted the proportion of infections composed of *Parorchis* or *Himasthla* against age for eight cohorts that could be followed for at least three consecutive years (Fig. 6). All such cohorts were from PGC. Again, there was no consistent temporal trend in the relative abundances of these antagonistically dominant species. If anything, the combined relative abundance of these species tended to peak within the first 2 yr and gradually decline thereafter. There was no evidence that *Parorchis* or *Himasthla* comes to dominate the assemblage of parasites within a cohort of snails as the cohort ages.

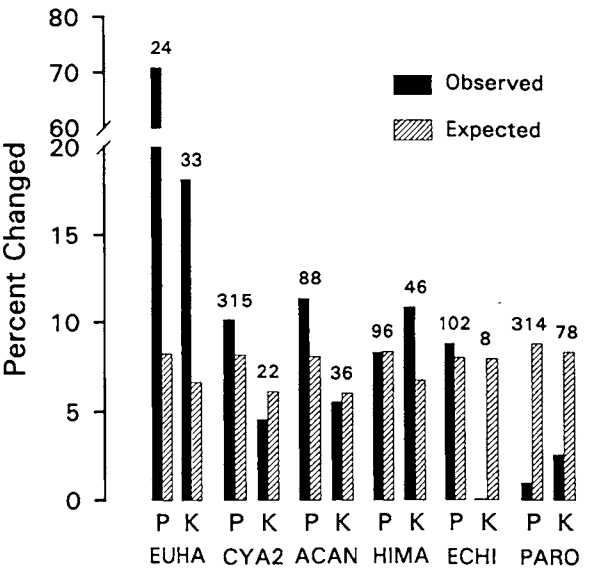

FIG. 4. Observed and expected percentages of initial infections that changed (were invaded or replaced by another species) during field exposure of mark–recaptured snails. Expected values are based on the null hypothesis that species-specific rates of change are strictly proportional to the number of snail-days that each species was exposed in the field; see text for method of calculation. Sample sizes for each parasite species and site appear above each pair of bars. P = Pine Gulch Creek, K = Kent Island; see Table 1 for key to species codes.

DISCUSSION

Patterns of interspecific association and interaction

Larval trematode species do not randomly and independently infect available host snails. Negative associations predominate and infraguilds are depauperate, rarely consisting of more than a single species. Mixed-species infections of larval trematodes are often less frequent than expected by chance in collections from natural populations of freshwater and marine snails (e.g., Cort et al. 1937, Martin 1955, Lie et al. 1966, Vernberg et al. 1969, Werding 1969, Anteson 1970, Robson and Williams 1970, Dönges 1972, Vaes 1979, Lauckner 1980, Goater et al. 1989, Fernandez and Esch 1991*a*, Williams and Esch 1991).

Detailed analysis of pairwise associations between species is compromised by fairly small sample sizes and lack of statistical independence (Simberloff 1990:

TABLE 7. Multiple comparison tests of the proportions of infections that changed during field releases of snails carrying known infections of trematode species. Underlines indicate proportions that are not significantly different at $P < .05$ using the Tukey-type test described in Zar (1984: 401). Species are ordered (left to right) from highest to lowest proportionate change; see Fig. 4 for values. See Table 1 for key to species codes.

Site	Initial parasite					
PGC	EUHA	ACAN	CYA2	ECHI	HIMA	PARO
KI	EUHA	HIMA	ACAN	CYA2	PARO	ECHI
Combined	EUHA	ACAN	CYA2	ECHI	HIMA	PARO

TABLE 8. Initial and final species of parasites in field-released and recaptured snails. Numbers of exclusions or invasions by a particular parasite species (see column headings) are summarized at bottom of table. In an exclusion, the final parasite has completely replaced the initial parasite. In an invasion, a second parasite species has superinfected, but has not excluded

Initial parasite	Site	Final parasite: Single infection MIC1	EUHA	CYA2	ACAN	HIMA	ECHI	PARO	PHOC	CATA	RENB
MIC1	PGC	*1*									
EUHA	PGC		*7*			9		6			
	KI		*27*			3		3			
CYA2	PGC			*283*	1	19		4			
	KI			*21*		1					
ACAN	PGC				*78*	1		8			
	KI				*34*			1			
HIMA	PGC					*88*		7			
	KI					*41*		5			
ECHI	PGC						*93* *	8			
	KI						*8*				
PARO	PGC					2		*311*			
	KI					2		*76*			
PHOC	PGC								*1*		
	KI								*1*		
CATA	PGC									*2*	
RENB	PGC										*1*
Exclusions		0	0	1	1	37	0	43	0	0	0
Invasions*		2	0	0	0	11	1	1	0	0	0

* Not included in the summary table is one invasion by AUST.

294), but the body of results from several statistical procedures is convincing evidence that most species with redial stages in their life histories are negatively associated with other redial and sporocyst species. Associations among sporocyst species are less clear, due in part to small sample sizes. Kuris (1990) detected similar patterns of interspecific association in Martin's (1955) collections of *Cerithidea* from Newport Bay in southern California.

There are a number of potential causes of negative associations among larval trematodes. As discussed earlier (see *Materials and methods: Analysis of mixed-species infections*), violation of the assumption that all snails in a sample have been equally exposed to available miracidia is one artifactual cause. I have attempted to minimize the influence of different histories of exposure by analyzing associations on a pan-by-pan basis. However, any heterogeneity in the distribution of parasite species among host phenotypes or genotypes would contribute to a pattern of negative association. Such heterogeneity does exist in the *Cerithidea*–trematode system. Infections by different species of larval trematodes are differentially distributed among size classes of *Cerithidea* (Sousa 1983, Kuris 1990). This was true for each of the annual samples from both study sites; however, the size distribution of snails that a particular parasite infected changed from year to year, as did the rank order of the median sizes infected by different species (W. Sousa, *unpublished data*). There is currently no information on genetic variability in susceptibility to infection within *Cerithidea* populations. A deficit of mixed-species infections would also occur if such infections were more lethal than single-species infections. I do not have the appropriate data to test whether this possibility applies to the *Cerithidea*–trematode system, but I have found no evidence in the literature to support the idea for any snail–trematode association.

The mark–recapture study and direct observations of interspecific interactions provide compelling evi-

TABLE 9. Observed acts of predation by rediae on larval stages (cercariae, rediae, or sporocysts) of co-occurring species. See Table 1 for key to species codes.

Predator species	Prey species: Redial HIMA	PARO	ECHI	ACAN	EUHA	Sporocysts-only REN2	MIC1	CYA2	AUST
HIMA	···	1	1	3	12	4	1	2	1
PARO	3	···	1	2	1				
ECHI			···			7	2		
CATA					1*				

* CATA rediae caught EUHA cercariae in vitro, while both were immersed in seawater within a glass petri dish. All other observations were in vivo.

the initial parasite; the two species co-occurred at the time of dissection. Italic values on diagonal are numbers of infections that did not change. See Table 1 for key to species codes.

Final parasite					
Double infection of initial parasite and:					PARO-
ECH1	HIMA	PARO	MIC1	AUST	CYA2
1	1				
	1				1
	7	1			
			1		
				1	
			1		
	1				
	1				

dence that negative biotic interactions occur. The patterns of species replacement were hierarchical; the two largest redial species, *Himasthla* and *Parorchis* (Table 11), accounted for most of the observed cases of exclusion or invasion. Predation by rediae on other larvae in mixed-species infections is a primary mechanism of interspecific antagonism employed by large redial species, including *Echinoparyphium*. These forms prey on a wide array of smaller redial and sporocyst species. Yoshino (1975) and Kuris (1990) have observed larval predation by the same three species: *Himasthla* preying on *Echinoparyphium* and *Euhaplorchis, Parorchis* preying on *Echinoparyphium* and *Euhaplorchis,* and *Echinoparyphium* preying on *Euhaplorchis*. The relative rankings of *Himasthla* and *Parorchis* in this hierarchy are roughly equivalent. *Himasthla* with its larger pharynx is a more aggressive predator, judging by the greater number of observed acts of predation. *Parorchis* infections, however, are more resistant to invasion and replacement. Each species was observed to prey on, and exclude, the other, although *Parorchis* appeared to be slightly more successful at both.

The interactions I observed are remarkably similar to those first observed in nature by Wesenberg-Lund (1934) and further documented in the classic experimental studies of freshwater snail–trematode associations by K. J. Lie and co-workers (see reviews by Lie

TABLE 10. Proportions of infections within different age classes of snails composed of the agonistically dominant trematode species *Parorchis* and *Himasthla*.*

		Age class (years)				One-way ANOVA		
Site		1	2	3	4/5	*F*	df	*P*
PGC	$\bar{X}$	0.424	0.392	0.389	0.415	0.140	3,105	0.936
	SD	0.282	0.219	0.220	0.255			
	N	33	42	22	12			
KI	$\bar{X}$	...	0.275	0.346	...	1.403	1,39	0.243
	SD		0.184	0.200				
	N		21	20				

* Sample sizes were adequate for statistical comparison of four age classes at PGC (1, 2, 3, and a combined class of 4 and 5 yr olds) and two age classes at KI (2 and 3 yr olds). For each site, differences among means were tested with one-way ANOVA; homogeneity of variance was confirmed with Bartlett's test (Sokal and Rohlf 1981).

TABLE 11. Mean dimensions of redial larval stages for trematode species collected from *Cerithidea* snails in annual samples from Bolinas Lagoon.* See Table 1 for key to species codes.

	Body				Pharynx		
Parasite species	*N*	Length (mm)	Width (mm)	Volume (mm^3)	*N*	Length (mm)	Width (mm)
PARO	13	2.09	0.37	0.222	20	0.045	0.038
HIMA	15	1.51	0.38	0.175	20	0.096	0.058
ECHI	6	1.04	0.38	0.133	20	0.075	0.064
CATA	5	0.60	0.30	0.043	21	0.044	0.037
ACAN	12	0.51	0.16	0.010	20	0.054	0.051
PHOC	16	0.60	0.13	0.008	16	0.023	0.023
EUHA	7	0.51	0.10	0.004	20	0.026	0.024

* Body measurements were made by the author on living larvae, without a coverslip. Body volume was estimated assuming a cylindrical shape. All pharynx dimensions, with the exception of CATA, were measured from separate collections of heat-killed, unmounted larvae, under light coverslip pressure, by H. Ching. Pharynx dimensions for CATA are those measured by Martin (1956) from stained, mounted specimens.

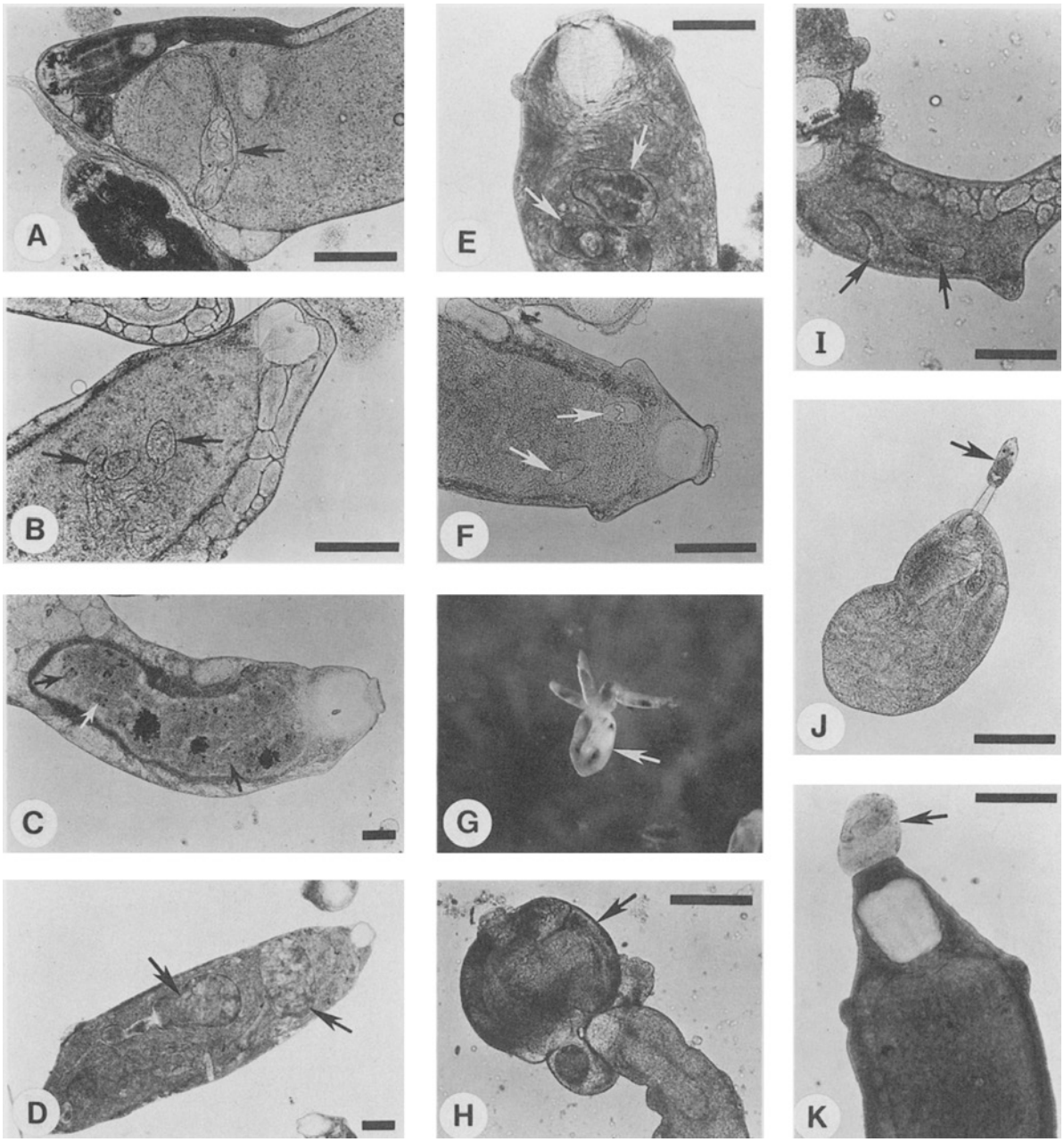

FIG. 5. Photomicrographs of rediae preying on larvae of co-occurring species. Arrows indicate prey. Scale bars equal 200 μm. Predator–prey species pairs are: (A) *Himasthla*–cercaria of *Austrobilharzia*; (B) *Himasthla*–cercariae of small cyathocotylid; (C) *Himasthla*–cercariae of *Euhaplorchis* (note paired eyespots throughout gut); (D) *Himasthla*–sporocysts of small cyathocotylid; (E) *Himasthla*–cercariae of *Acanthoparyphium*; (F) *Echinoparyphium*–cercariae of small microphallid; (G) *Parorchis*–redia of *Himasthla* (note 3 attacking rediae at top); (H) *Parorchis*–redia of *Echinoparyphium* (note attacking redia in lower right); (I) *Echinoparyphium*–cercariae of small renicolid with stylet; (J) *Catatropis*–cercaria of *Euhaplorchis*; (K) *Himasthla*–redia of *Euhaplorchis*.

et al. 1968, Lim and Heyneman 1972, Kuris 1990, Sousa 1992). Additional laboratory demonstrations of interspecific antagonism were provided by the studies of Anteson (1970), Dönges (1972), and Page and Huizinga (1976). While most interactions among freshwater larval trematodes are hierarchical, transitive, and dominated by large predatory redial forms, these experimental studies have revealed a rich variety of interactions, both positive and negative. For some species pairs, order of infection determines the eventual winner. Remarkably similar antagonistic interactions occur between the larvae of co-infecting species of insect parasitoids (e.g., Salt 1961, Wylie 1972, Cruz 1981).

Indirect antagonism also occurs in these freshwater

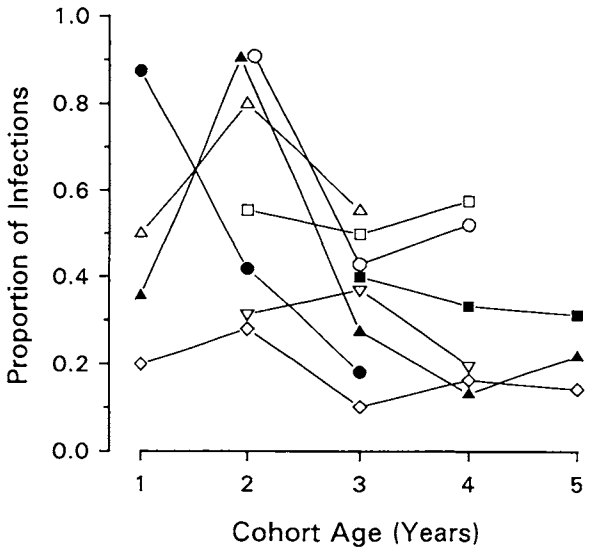

FIG. 6. Temporal change in the proportion of larval trematode infections that were by *Parorchis* or *Himasthla* within individual cohorts of snails at PGC. Each symbol represents a distinct cohort within one of the PGC pans that could be followed for at least three consecutive years.

snail–trematode associations. Subordinate rediae or sporocysts often show abnormal or slowed development in mixed infections, even when not in direct contact with larvae of co-occurring dominant species. In the experiments of Basch et al. (1969), indirect antagonism alone was sufficiently strong that one nonpredatory sporocyst species excluded another. The precise mechanisms of indirect antagonism remain poorly understood (Lim and Heyneman 1972). Possibilities include immunological responses or cellular tissue reactions in the snail host induced by a prior or concurrent heterospecific infection, toxic chemicals released by the larvae, or exploitation competition for nutrients or oxygen. Such indirect interactions may account for some of the negative associations among members of the guild of *Cerithidea* parasites, but this remains a matter of speculation until comparable experiments are conducted in that system.

The two-tailed Fisher exact tests gave weak statistical evidence of positive associations among some guild members. In most cases, these associations are probably spurious consequences of small sample size; only three remained significant when the experiment-wise error rate was held at .05. That *Catatropis* and *Renicola buchanani* were members of several of these potentially positive associations is interesting, since these species infect the mantle and not the gonad or digestive gland occupied by the co-infecting species. *Catatropis* was also positively associated with the small microphallid in Martin's data (Kuris 1990). This suggests that negative interactions may be weaker if larval populations are spatially separated within the host. Even so, indirect antagonism in the form of exploitation competition for nutrients seems possible because gonadal development is often visibly diminished in snails parasitized by the mantle-infecting species (W. Sousa, *personal observation*).

Mixed-species infections involving the schistosome *Austrobilharzia* may represent true cases of positive association. This species was rare in the Bolinas collections, but 10 out of its 13 infections were with other species, nine redial and one sporocyst (Table 2). In Martin's (1955) collections, 67 of 162 *Austrobilharzia* infections were mixed-species. The Australian *Austrobilharzia terrigalensis* is very frequently, sometimes exclusively, found in mixed-species infections with redial species in its estuarine snail host (Ewers 1960, Walker 1979, Appleton 1983). As Kuris (1990) points out, such positive associations are usually not mutually beneficial. In many cases, cercarial production of one of the species is reduced, or its larval stages are damaged, killed, or displaced from their usual site of infection. This is true in the case of mixed infections involving *A. terrigalensis*; the presence of the schistosome reduced the cercarial production and the size of rediae of co-occurring species. This represents one of only a few known cases of a sporocyst species suppressing a redial species by indirect antagonism (Sousa 1992). Mixed-species infections involving *Austrobilharzia* from Bolinas were not observed in sufficient detail to detect such patterns.

Laboratory experiments with freshwater larval trematodes have demonstrated cases in which a prior infection by one trematode species facilitates the establishment of an infection by another; one species is usually later excluded by direct antagonism (reviewed in Sousa 1992). Subsequent studies have shown that substances secreted by the first parasite interfere with the actions of snail haemocytes that defend against infection (see reviews by Lie 1982, Lie et al. 1987, Meuleman et al. 1987, and Bayne and Yoshino 1989).

Effect of interspecific antagonism on parasite guild structure

That the members of an assemblage interact says little about the influence of such interactions on the structure of the assemblage in nature, e.g., the number and relative abundance of species. Indeed, without manipulating the abundance of putative interacting species in controlled field experiments, it is difficult to address the issue in a definitive way. Due to the complexity of digenetic trematode life cycles and the minute size of trematode eggs and infective miracidial larvae, such experiments are very difficult to conduct in natural snail–trematode systems. To my knowledge, the only experimental field studies of larval trematode interactions are those of K. J. Lie and coworkers in Malaysia (Lie et al. 1970, 1971, Lie 1973, Combes 1982). They introduced laboratory-reared eggs of freshwater echinostome trematodes (redial forms) into small ponds that contained snails infected by larval schistosomes (sporocyst forms), with the aim of de-

TABLE 12. Projected annual rates at which established infections of different parasites were invaded or excluded by other species at the two study sites, as estimated from mark–recapture results. See Table 1 for key to species codes.

Parasite species	Study site	
	PGC	KI
	Annual rate (%)*	
ACAN	10.9	5.9
ECHI	7.9	0.0
HIMA	8.2	11.2
EUHA	72.0	19.6
PARO	0.9	2.5
CYA2	10.0	4.9

* Annual rate of invasion or exclusion = percent of initial infections that changed (see Fig. 4) divided by median period of field exposure per snail in years (sixth column of Table 3/365).

creasing the prevalence of the latter infections. The experiments met with various logistical and design problems, but in at least two of the three trials the echinostomes eliminated the schistosomes by predatory antagonism. The experiments were meant to evaluate a potential method of biologically controlling an agent of human disease, and do not address the issue of whether dominant trematode species affect the abundance of subordinate species under natural conditions. To do so would require the even more difficult task of manipulating, with appropriate controls, the natural influx of eggs and/or miracidia. This has yet to be done in any snail–trematode system.

Given these constraints, the following arguments are best considered hypotheses. I have found it useful to view the snail–trematode system from a patch dynamics perspective (Sousa 1990). Each host snail is a patch or island of parasite habitat. Empty patches are born via the recruitment of susceptible offspring to the snail population, increase in size and change in a variety of other characteristics as snails age, and disappear (along with their resident parasites) as snails die. Each patch is effectively isolated from others by the complex life cycle of the parasite. Redial and sporocyst larval stages cannot move directly between snails; they recruit to the host patch only via infection by miracidia dispersed by the definitive vertebrate host.

The question of the impact of interspecific antagonism on guild structure can be addressed at several different scales. At the level of the individual host, the infraguild or within-patch scale, if more than one species of parasite successfully recruits to the same snail, deterministic and hierarchical antagonistic interactions almost always result in the exclusion of one of the two species. In addition, prior infections by certain species may preempt the resources and prevent successful establishment by others. Such exclusions and preemptions contribute to the deficit of mixed-species infections.

Established infections of the common trematode species were invaded or excluded at the projected annual rates listed in Table 12. Such antagonism may be a substantial threat to the persistence of *Euhaplorchis* infections, especially at PGC. However, the remaining parasites lose only between 0.9 and 11.2% of existing infections to interspecific antagonism each year.

It is more difficult to estimate the number of infections that failed to become established because an existing infection by a dominant species preempted host resources. The highly significant differences in rates of replacement among the parasites indicate that their infrapopulations differ in susceptibility to invasion. What such rates do not indicate is the degree to which variation in relative invasibility among parasite species is attributable to (1) differences in the levels of direct or indirect resistance to invasion, or (2) differences in the extent to which host defenses against infection are suppressed, and invasion is facilitated, by the initial infection (see examples in Sousa 1992). One approach to quantifying the cost to inferior species of prior monopolization of host resources by dominants is to simultaneously expose uninfected snails and snails known to be infected by various species of parasites in the field and compare the rates at which new infections become established in the different groups of snails. One practical problem with this experiment is that it is difficult to obtain a sample of known uninfected snails from the field; snails carrying mature infections can be detected by inducing them to shed cercariae, but to be certain that a *Cerithidea* does not carry an immature infection, one must dissect it. It might suffice simply to dissect an initial subsample of the snails that have not shed cercariae to determine the ambient level of immature infections, then adjust the number of "new" infections observed at the end of the exposure period for the number of immature infections that were present at the outset. The mark–recapture study reported here did not include uninfected snails and consequently does not yield this information. A study of the design just described is in progress.

A prior infection by one species may preclude establishment of another by several means. Immature larvae of the invading species may be consumed or otherwise killed, but more subtle forms of negative interaction could also occur. For example, the presence of an already established infection by one species may somehow deter miracidia of other species from attempting to superinfect. Similar avoidance behaviors are exhibited by planktonic larvae of some sessile marine invertebrates (Grosberg 1981), and this form of interaction has been suggested by several early workers (see references in Lie et al. 1966) as an explanation for the rarity of mixed-species infections of larval trematodes. It has not been looked for in the *Cerithidea*–trematode system, but direct observations of miracidial penetration behavior (Lie et al. 1966) and detailed histological studies (Lim and Heyneman 1972) indi-

cate that the phenomenon does not occur in similar freshwater systems. Miracidia of subordinate species penetrate snails already infected by a dominant trematode at the same rate as they do uninfected controls, and vice versa.

Do the patterns and rates of interspecific interaction observed at the infraguild level influence the structure of the assemblage at higher levels? To answer this question, I have examined guild structure at two nested scales above that of the individual host. The first of these levels was that of an even-aged cohort of snails within a host subpopulation. This scale is nested within the second level, that of the host subpopulation itself (the component community or guild, sensu Holmes and Price 1986). The analysis of cohort-level patterns is reported in this paper, while the structure of the component guild within host subpopulations is examined in detail in Sousa (1990).

The structure and dynamics of parasite guilds at both these levels seem to be little affected by the outcomes of antagonistic encounters that occur between parasite species within individual hosts. As a snail cohort ages or the mean age of a host subpopulation increases, the prevalence of infection rises, the number of susceptible, uninfected hosts declines (Sousa 1990), and the frequency of interspecific interactions among parasites should increase. In time, if the host resource becomes sufficiently limiting, the hierarchical antagonistic interactions described (see above, *Patterns of interspecific association . . .*) would be expected to drive trematode diversity downward. There is no evidence for such a pattern within parasite assemblages at either the level of a *Cerithidea* cohort or subpopulation. The parasite assemblages of neither snail cohorts nor subpopulations suffer a decline in diversity as they age. While the relative abundances of a few subordinate species such as *Euhaplorchis* may be measurably reduced within the larger host population by such interactions, there is little evidence that the antagonistically most aggressive species are able to numerically dominate the parasite assemblage. In fact, just the opposite pattern was observed for component parasite guilds: diversity, both in terms of species richness and equitability, rose monotonically with the average age of the host subpopulation (Sousa 1990). The rate at which subordinate species are being excluded from individual patches or snails, relative to the rate at which new infections of these species are being established in uninfected snails, is not high enough to cause a decline in diversity at the regional level of the host subpopulation. The host resource will remain unlimiting as long as rates of parasite recruitment and infection are low relative to the rate at which uninfected, susceptible new hosts recruit. Further, mortality of old, infected hosts may limit the rate of accumulation of infections by dominant species. Under these conditions, an equilibrium assemblage dominated by a few species would seldom if ever develop, and parasite diversity will only increase as infections accumulate. Guild structure, therefore, would be determined primarily by spatial and temporal patterns of parasite recruitment.

On the other hand, diversity at the regional level might remain high despite frequent negative interactions if snails infected by the dominant species died at higher rates than those infected by subordinate species. At present, I do not have the appropriate data to make a direct comparison of the survivorship schedules of snails infected by different parasites, but snails infected by each of the species evaluated in the mark–recapture study can survive well over a year in the field (Table 3), and laboratory stress trials showed that the species of infecting parasite had no effect on survival rates, except under relatively rare, anoxic conditions (Sousa and Gleason 1989). Even under these conditions, there was no consistent trend for snails infected by more dominant trematodes to die at higher rates.

As I have argued elsewhere (Sousa 1990), patterns of guild diversity at the level of a *Cerithidea* subpopulation are consistent with the alternative hypothesis that recruitment processes rather than interspecific interactions primarily determine the composition and relative abundance of species in component parasite guilds. Fernandez and Esch (1991*b*) reached a similar conclusion concerning the processes that structured an assemblage of larval trematodes infecting a population of the pond snail, *Helisoma anceps*. In Bolinas Lagoon, the relative prevalences of different trematode species differ markedly between sites, and from year to year (Table 2), despite the fact that individual infections can persist for years. The structures of parasite assemblages within individual pans exhibit large shifts from year to year, with no trend toward dominance by large redial species (W. Sousa, *unpublished data*). The likely cause of this variation in parasite guild structure is spatial and temporal variability in the abundance, survival, or infectivity of eggs and miracidial larvae dispersed by definitive vertebrate hosts, primarily birds. At the same time, if two or more trematode species co-occur as adults in the same vertebrate host and tend to shed eggs synchronously, intramolluscan interactions between these particular species could be frequent despite spatial and temporal variation in the abundance of the definitive host (e.g., Fernandez and Esch 1991*a*). I am currently examining census data for lagoon bird populations collected by the Point Reyes Bird Observatory to determine if bird numbers and/or species composition are correlated with rates of parasitism.

Several features of this system promote the regional coexistence of such a large number of potentially interacting parasites at the level of the host subpopulation. The discrete, divided nature of resources provided by hosts and the complex life cycles of trematodes preclude the direct spread between snails of asexual

parasitic stages, including the predatory rediae of dominant species. In effect, this limited interpatch mobility provides a temporary spatial refuge for subordinate species, much as it did in Huffaker's (1958) classic study of predator–prey interactions among mites in a patchy, experimental system. Asexual reproduction within colonized hosts generates a pattern of independent aggregation that promotes coexistence (Shorrocks et al. 1979, Atkinson and Shorrocks 1981, Hanski 1981, 1990, Dobson 1985, Ives and May 1985, Ives 1988, Dobson and Keymer 1990, Shorrocks 1990). Differential exploitation by the parasite species of different-sized hosts described above may also reduce the rate of interspecific antagonism. However, while there are differences in the mean sizes of hosts infected by the different species, there is also much overlap in the size ranges they infect. I found no evidence that host subpopulations with greater variation in individual host length supported more diverse assemblages of larval trematodes (Sousa 1990) as would be expected if such partitioning was reducing the level of negative interspecific interaction. Finally, the high rate at which new patches of resource are made available by host reproduction affords ample opportunities for colonization by subordinate species (Price 1990).

Some, but not all (e.g., hierarchical interspecific antagonism), of the features of this system are consistent with those required by "lottery models" of species coexistence (Chesson and Warner 1981, Chesson 1985, Warner and Chesson 1985, Dobson 1990). It is an open system in which local recruitment of miracidia to snail populations is very likely independent of the prevalence of infection in the snail population, since adult flukes reproduce in, and are dispersed by, highly mobile vertebrate hosts that acquire infections while foraging over areas much larger than that occupied by a snail subpopulation. Recruitment of new infections to snail populations varies temporally and probably stochastically, with no apparent correlation among species. However, much more detailed information is needed to determine the extent to which a lottery model applies (Warner and Chesson 1985).

Finally, there are strong parallels between the apparent role of larval recruitment in this system and the role some investigators attribute to this process in assemblages of sessile, free-living, marine invertebrates with planktonic larval phases (e.g., Sale 1977, Underwood and Denley 1984, Connell 1985, Gaines and Roughgarden 1985, Warner and Chesson 1985, Hughes 1990). Rates of interspecific antagonism should be higher when and where miracidial stages and extant infections of different trematode species are abundant. The observations that the probability of detecting a negative association among species increased with infection rate (within and among years), and was higher at PGC with its higher abundance of definitive hosts, are consistent with this prediction. Alternatively, this may simply be the result of the increased statistical power associated with larger samples of infections. More direct evidence comes from the mark–recapture results. The rate of exclusion of subordinate *Euhaplorchis* infections by large redial species was more than three times higher at PGC where the density of definitive hosts is higher, and so presumably is the input of miracidia of predatory species, than at KI.

Models of parasite community structure: do they apply to invertebrate hosts?

There are unique characteristics of the intermediate invertebrate host–larval trematode association that are not considered in current models of parasite community structure, which focus largely on the adult phase of helminth life cycles. As a result of these differences, the relative contributions of particular biotic processes to parasite community structure may differ between these two kinds of host–parasite association. First, rediae and sporocysts, as opposed to the adult stage, reproduce asexually within the invertebrate host. Asexual propagation of larval stages causes more rapid increase in infrapopulation density than does the recruitment of individual adult parasites to a vertebrate host. Second, larval trematodes exhibit very limited differentiation of infection sites compared to adult helminths in the vertebrate host. Not only do molluscan hosts offer fewer target organs to parasites, but the tissues of the primary organs infected by larval trematodes, the gonad and digestive gland (Wright 1971, Kuris 1974, Brown 1978, Lauckner 1980, 1983, Sousa 1983), are relatively homogeneous and not as easily partitioned as are the complex organ systems of vertebrate hosts. Third, intraguild predation (Polis et al. 1989) is a widely documented form of negative interaction among trematode larvae that is uncommon, if it occurs at all, among vertebrate gut parasites (Holmes et al. 1977). These life history and morphological traits of larval trematodes and their invertebrate hosts should promote stronger interactions, negative or positive, among members of larval trematode infraguilds than among adult helminths in vertebrate hosts.

Expanding our focus to the assemblage of larval trematodes infecting a subpopulation of snails, many features of this system are consistent with Price's (1980) non-equilibrium view of parasite community structure. At this component community scale, the larval trematode–snail system lies more towards the isolationist than interactive end of the continuum erected by Holmes and Price (1986) and Goater et al. (1987). While negative interspecific interactions occur within infraguilds, their occurrence appears to be intermittent and infrequent for most species. The structure of the assemblage is in a state of flux and changes appear to be driven largely by the external process of larval recruitment (parasite transmission), which varies substantially in space and time. In fact, one might expect that the influence of small-scale spatial variation in the availability of infective stages on infracommunity

structure should be more marked for invertebrate than vertebrate hosts because invertebrate hosts are generally less mobile, and "absorb" parasites that are carried to localized populations, while vertebrate hosts commonly move over greater distances, "collecting" parasites, usually as a result of consuming infected intermediate hosts.

The ultimate challenge will be to wed the dynamics of adult phases in vertebrates with those of larval stages in intermediate hosts, with attention to the intervening transmission processes. While this will not be accomplished easily, it is the only means of developing a comprehensive understanding of the relative contributions of biotic and abiotic processes to the structures of assemblages of helminth parasites.

ACKNOWLEDGMENTS

I am indebted to many people for their assistance and support during this study. I owe many thanks to the eight annual "snail crews" without whose help in the field and laboratory this study would not have been possible. E. Adams wrote excellent, tailor-made programs for the statistical simulation analyses. E. Adams, J. Addicott, S. Allison, A. Bush, B. Downes, G. Esch, J. Fernandez, M. Gleason, J. Holmes, P. Kotanen, B. Mitchell, P. Price, D. Simberloff, and J. Watanabe made many helpful comments on the manuscript. Parasite species identifications were expertly confirmed by H. Ching. I thank R. Paine and the Department of Zoology at the University of Washington for providing a wonderful sabbatical refugium in which to work on the manuscript. Access to the study sites was kindly allowed by the Marin County Department of Parks and Recreation. This research was supported by National Science Foundation grants DEB-8004192, BSR-8300082, BSR-8505871, and BSR-8516522, and grants from the Committee on Faculty Research at U. C. Berkeley.

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