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Interspecific Interactions Among Larval Trematode Parasites of Freshwater and Marine Snails[1]

WAYNE P. SOUSA

Department of Integrative Biology, University of California, Berkeley, California 94720

SYNOPSIS. Freshwater and marine snails serve as intermediate hosts for numerous species of larval trematodes. Any particular population of snails may be infected by several species. It is commonly observed that mixed-species infections are less frequent than expected by change in collections of host snails from natural populations. While several mechanisms might generate such negative associations, laboratory studies of freshwater snail-trematode associations have demonstrated the presence of strong antagonistic interactions between intramolluscan larval stages (rediae and sporocysts) of species that infect the same host individual. Both predatory and non-predatory antagonism has been observed, the former taking the form of predation by large, dominant redial forms on the sporocysts and rediae of subordinate species. These interactions are largely hierarchical, although in some systems priority effects have been observed, and in one case a sporocyst species replaced a redial species by strong non-predatory antagonism. Several instances of positive association between larval trematode species have also been observed. In such cases, interference with host defense mechanisms by the first parasite appears to enhance superinfection by the second. My own study of the larval trematode guild that infects the salt marsh snail, *Cerithidea californica,* has revealed patterns of association and interaction that are very similar to those demonstrated by laboratory studies of freshwater systems. Ultimately, the frequency of interactions among larval trematodes depends on the availability, relative to the numbers of susceptible snails, of infective eggs and miracidial larvae transmitted from definitive hosts.

INTRODUCTION

I will consider endoparasites as symbionts in this paper, and will focus on systems in which multiple symbionts potentially exploit the same host. These systems are the associations between gastropods and the parasitic larvae of digenetic trematodes. Populations of freshwater and intertidal marine snails are commonly infected by several species of trematode larvae (*e.g.,* James, 1969, 1971; Wright, 1971; Brown, 1978; Vaes, 1979; Rohde, 1981, 1982; Lauckner, 1980; Kuris, 1990; Sousa, 1990, 1993). Rather than discuss the impact of these symbionts on their snail hosts, I will examine the role that interspecific interactions play in structuring assemblages of larval trematodes that exploit the same host snail. Such assemblages have been termed infracommunities (Holmes and Price, 1986) or infraguilds (Sousa, 1990).

This paper summarizes published observations and experimental data bearing on several questions concerning potential interactions between larval trematodes: Does the presence of a prior infection affect the chances that a second species can become established? When multiple species of trematode larvae co-infect a snail, are there negative or positive consequences for the fitness of one or both parasites? Are these consequences asymmetrical in their impact on the species, and if so, how do they affect coexistence of the species within an individual host? If hierarchical relationships exist, are they consistent in space and time,

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and throughout larval ontogeny? Finally, by what mechanisms do larval trematode species interact?

Digenetic Trematode Life Cycle

At the outset, it is necessary to describe briefly the typical digenean life cycle. This cycle involves several parasitic stages that exploit an obligate sequence of intermediate and definitive hosts; transmission is effected either by free-living, motile larvae or by encysted larvae that are ingested by the host. Bear in mind, that there are numerous variants of the following generalized pattern (Shoop, 1988). Arbitrarily beginning with the egg stage, a trematode egg is deposited in the environment, commonly via the feces of the vertebrate definitive 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, that acquires nutrients by uptake 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. A mother sporocyst is common to all digenean life cycles, but the subsequent production of sporocysts versus rediae is a species and family-specific characteristic.

These daughter sporocysts or rediae migrate to their final sites of infection, commonly the gonad or digestive gland. Mature sporocysts or rediae of most species produce free-swimming cercarial larvae which leave the snail in search of a second intermediate host in or on 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 notable case of schistosome trematodes, 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: sporocyst and rediae. Within each of these larval categories, the sizes and morphologies of mature larvae vary among species of trematode.

Features that Promote Negative Larval Interactions

There are several *a priori* reasons to expect negative interactions among trematode larvae that exploit the same host species. First, intramolluscan larval stages proliferate rapidly by asexual reproduction; within a few weeks following infection, hundreds or even thousands of sporocysts or rediae may inhabit a snail's tissues (Lim and Heyneman, 1972). They grow rapidly, becoming comparatively large relative to the host (Kuris, 1974), and place a substantial demand on the host's metabolism. In heavily infected snails, tissues in and around the foci of infection are often heavily damaged and greatly reduced in biomass. Gonadal tissues, for example, are almost entirely absent in heavily infected snails, and permanent castration is common (*e.g.,* Lauckner, 1980; Curtis and Hurd, 1983; Sousa, 1983). Conditions of high parasite density and depleted host resource would seem to make negative interactions among co-occurring species likely. In addition, larval trematodes exhibit a limited amount of infection site specificity compared to the high degree of such segregation documented for adult stages in vertebrate hosts (*e.g.,* Crompton, 1973; Holmes, 1973). The partitioning of snail tissues by trematode larvae that does occur is coarse. For example, in the salt marsh snail *Cerithidea californica,* 3 trematodes infect exclusively the mantle wall while at least 13 species exploit the gonad and digestive gland to varying degrees, the former being the primary locus of infection (Yoshino, 1975; Sousa, 1993). Thus, the preferred infection sites of many larval trematodes overlap considerably, affording substantial opportunity for interspecific interaction. Even when two species occupy exclusive sites, this does not preclude interaction via released toxic chemicals, host-wide nutrient depletion, or other mechanisms. Certainly, the potential for negative

interspecific interactions exists. Does it occur?

EVIDENCE OF NEGATIVE ASSOCIATION AND INTERACTION AMONG LARVAL TREMATODES

A substantial body of circumstantial and direct evidence indicates that antagonistic interactions can and do occur between larval trematodes that exploit a common snail host. The primary circumstantial evidence consists of the common observation that mixed-species infections are less frequent than expected by chance in collections of host snails from natural populations. A statistical paucity of mixed infections of particular species-pairs has been demonstrated in a number of freshwater (*e.g.,* Cort *et al.,* 1937; Lie *et al.,* 1966; Anteson, 1970; Dönges, 1972; Goater *et al.,* 1989) and marine (*e.g.,* Martin, 1955; Vernberg *et al.,* 1969; Werding, 1969; Robson and Williams, 1970; Vaes, 1979; Lauckner, 1980; Kuris, 1990; Sousa, 1990, 1993) snail-trematode associations. This sort of evidence is obviously indirect, and should be interpreted with caution. Calculations of the expected number of mixed infections under the null hypothesis of independent association rest on the assumption that all snails in a sample have been equally exposed to the available trematode eggs or miracidial larvae. While the problem was clearly identified by Cort *et al.* (1937, pp. 516–517), the assumption of homogeneous exposure has seldom been evaluated or discussed in more recent analyses (but see Lauckner, 1980; Kuris, 1990; Sousa, 1990). It will often be violated when samples from different sites or times are pooled, since the prevalence of trematode infections is often heterogeneous in both space and time (*e.g.,* Bourns, 1963; DeCoursey and Vernberg, 1974; Cannon, 1979; Loker *et al.,* 1981; Rohde, 1981; Taraschewski and Paperna, 1981; Hughes and Answer, 1982; Appleton, 1983; Curtis and Hurd, 1983; Lauckner, 1988; Sousa, 1990, 1993; Fernandez and Esch, 1991*a, b*; Williams and Esch, 1991). The expected number of mixed infections will be artificially inflated by pooling such heterogeneous samples. This bias may lead one to conclude falsely that the species in question are negatively associated among individual hosts. Nevertheless, in some studies where mixed infection data are analyzed in such a way as to minimize the biasing influence of sample heterogeneity, simultaneous infections of more than one species remain unexpectedly rare (*e.g.,* for trematode parasites of *Cerithidea californica* [Sousa, 1990, 1993]). Even in studies where this has not been done, the observation that certain species pairs are never found together, even in large collections of infected snails, strongly suggests negative association. I hasten to add that not all species pairs are negatively associated; a fair number occur as often as would be expected by change, and a few species occur in mixed-species infections more often than would be expected. These interesting cases of positive association will be discussed later.

The strongest evidence for negative interactions among larval trematodes comes from the extensive experimental studies of K. J. Lie and his Hooper Foundation coworkers (see reviews of Lie *et al.,* 1968*b*; Lim and Heyneman, 1972; Combes, 1982; Kuris, 1990). These laboratory investigations of freshwater snail-trematode associations document not only that negative interactions occur, but the hierarchy of relationships among the different trematodes tested and some of the key mechanisms of interspecific antagonism. At the time of Lim and Heyneman's review, 24 combinations of 19 species and strains of Digenea in 5 species and strains of snail hosts had been studied. In many of the experimental designs, special attention had been given to the order of infection and the length of time between sequential infections. All four sequential combinations of redial and sporocyst species were tested.

A detailed description of these experiments and their outcomes is beyond the scope of this paper, and has already been presented in the above reviews. I will only summarize the major findings. Negative interactions can indeed be demonstrated in the laboratory either by challenging preexisting natural or experimentally established single-species infections with miracidia of a different species, or by exposing uninfected snails to miracidia of two different trema-

todes simultaneously. As first observed in natural infections by Wesenberg-Lund (1934), the rediae of several "dominant" echinostome species were shown to prey consistently on the rediae or sporocysts of other species that infect the same host (direct antagonism of Lie, 1967). The "subordinate" species is eliminated form the host's tissues, and it is often difficult or impossible to reinfect the host with the subordinate species. In most species combinations, the order of infection and the length of time between the introduction of the first and second species affect the rate of exclusion, but not the eventual outcome. If a subordinate species is established first and its larvae are allowed to mature before being challenged, a subsequent infection of a dominant redial species may develop more slowly than in a single-species infection, but in most cases it ultimately excludes the subordinate. When introduced simultaneously, the negative effect of subordinates on dominants is small or undetectable. There are, however, pairs of trematode species in which the eventual dominant depends on the order of establishment: whichever trematode becomes established first can prevent the invasion of the second (*e.g., Echinostoma audyi* and *Hypoderaeum dingeri,* both redia-producing echinostomes [Lie, 1969; see Lie *et al.,* 1966 and Anteson, 1970 for other examples]). Otherwise, the hierarchy of interactions appears to be quite fixed, even when the relative sizes of competing larvae change with ontogeny. For example, prior establishment of the subordinate echinostome, *Echinostoma paraensei,* slows the development of the dominant echinostome, *Paryphostomum segregatum,* but mature rediae of the former species do not prey on young rediae of the latter, which are smaller than they (Lie *et al.,* 1968*a*). To the contrary, immature rediae of a dominant species have been observed to attack larger, mature rediae of a subordinate species (Lie, 1973). The presence of a prior mature infection of a dominant species can, in some species combinations, prevent even the establishment of subordinate larvae.

There have been very few reports of larval cannibalism. Wesenberg-Lund (1934) reported an instance of daughter rediae of an echinostome preying on sibling larvae (rediae and cercariae) inside the mother redia, and Nasir (1962) found damaged rediae of *Echinostoma nudicaudatum* in the guts of conspecific rediae, and suggested that the rediae were preying on each other. Lie *et al.* (1975) demonstrated that an established infection by *Echinostoma lindoense* rediae reduced superinfection by the same trematode if the second exposure to miracidia occurred 3 or more days following the first. They attributed this reduction to predation by the established rediae on invading sporocyst stages from the second exposure, but such predation was never directly observed. On the other hand, Lie and coworkers have never observed cannibalism, nor suggested that it occurs, within a population of larvae established from a single exposure to miracidia (Lie *et al.,* 1975). While examining several thousand infected *Cerithidea californica,* I have never seen an instance of cannibalism.

Non-predatory interspecific antagonism (indirect antagonism of Lie, 1967) also occurs. 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 (Lie, 1967). As noted above, even dominant species can suffer a reduction in development rate when invading a well-established prior infection. Non-predatory antagonism is obviously the rule in negative sporocyst-sporocyst interactions, since these larvae cannot prey on one another. In experiments conducted by Basch *et al.* (1969) non-predatory antagonism alone was sufficiently strong that one sporocyst species, *Schistosoma mansoni,* excluded another, *Cotylurus lutzi.* The precise mechanism(s) of non-predatory antagonism remains 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 chemical compounds released by the larvae, or competition for nutrients or oxygen.

The relative and absolute strengths of predatory and non-predatory antagonism vary among tested combinations of parasites. In some interactions, particularly between large predatory, redia-producing species and sporocyst or small redia-pro-

ducing species, the dominant rapidly excludes the subordinate, while in others exclusion is a slow process or may never occur (*e.g., Schistosoma mansoni* and *Echinostoma barbosai* [Lie, 1966]). Although the combinations of species that have been tested is not exhaustive, most interactions appear to be hierarchical and transitive (*e.g., Paryphostomum segregatum* (redia) > *Ribeiroia marini* (redia) > *Schistosoma mansoni* (sporocyst); or *P. segregatum* > *Echinostoma barbosai* (redia) > *S. mansoni*; see Table I in Lim and Heyneman, 1972). Generally, rediae-producing species are dominant over sporocyst-producers; however, Ow-Yang and Lie (1972) have demonstrated an exceptional case in which the sporocyst-producing *Trichobilharzia brevis* eliminates the rediae-producing *Hypoderaeum dingeri* by strong non-predatory antagonism. In subsequent experiments a third trematode species, *Echinostoma audyi,* was added to this two-species system, with surprising results (Ow-Yang and Lie, personal communication in Lim and Heyneman, 1972; also Lie, 1973). *E. audyi* is a highly predatory echinostome that rapidly excludes *T. brevis* when paired with it. However, the presence of *H. dingeri,* even though suppressed by *T. brevis,* somehow inhibited the development of *E. audyi,* thereby preventing it from excluding *T. brevis.* Similarly, Lie *et al.* (1970) found that the presence of the parasitic microsporidian, *Perezia helminthorum,* can mediate interactions between a dominant echinostome and subordinate schistosome. In a number of trials, the echinostome became differentially infected by the microsporidian; this slowed echinostome development and prolonged the coexistence of the two trematodes. The existence of such complex, indirect interactions in naturally occurring guilds of larval trematodes has yet to be demonstrated.

Few natural snail-trematode systems have been studied in sufficient detail to evaluate the generality of the patterns of interspecific antagonism documented by the laboratory experiments of the Hooper Foundation and other workers (*e.g.,* Anteson, 1970; Dönges, 1972; Page and Huizinga, 1976). One such system is that involving the California salt marsh snail, *Cerithidea californica,* and more than 18 species of larval digenetic trematodes (Martin, 1955, 1972; Kuris, 1990; Sousa, 1990, 1993). As noted earlier, there is solid evidence from this system that many species-pairs occur less frequently in mixed infections than would be expected by chance. In Kuris' (1990) analysis of Martin's (1955) data from collections in Newport Bay, redia-producing species are very often negatively associated with both redia and sporocyst-producing species. Associations between sporocyst-producers are more variable in direction and strength; in some cases, the species are negatively associated, but in most cases the number of mixed-species infections is what would be expected by chance. Overall, the patterns suggest that redia-producing species are much stronger antagonists than species that produce sporocysts, a pattern that is consistent with the studies of Lie and co-workers.

My own eight-year study of the same trematode guild at Bolinas Lagoon, a more northern site, revealed very similar patterns (Sousa, 1993) briefly summarized below. Using a different statistical approach than Kuris (1990), I found that within a number of snail subpopulations in any given year, the frequency of snails infected by more than one species of larval trematodes is less than would be expected by chance. Out of a total of 5,025 infected snails examined over a seven-year period, only 128 (2.5%) were infected by more than one species (all but one were double infections, one was a triple). Two, more direct lines of evidence indicate that asymmetrical antagonism is in part responsible for the low number of mixed-species infections. First, while dissecting snails with mixed-species infections, I have observed rediae of one species preying on rediae, sporocyst, or cercariae of the other. These interactions are hierarchical; species with large rediae such as *Himasthla rhigedana* (Echinostomatidae) and *Parorchis acanthus* (Philophthalmidae) dominate other redial and sporocyst species. Species that have smaller rediae are, in turn, dominant over sporocyst-producing species. The second approach I have taken to evaluate the hierarchical nature of these interactions is to follow the temporal patterns of parasite species replacement in marked snails carrying known infections that I have released

in the field and recaptured at a later date. These sequences of replacement are also strongly hierarchical. *Himasthla* and *Parorchis* are the most common invaders of established infections of other species, which they inevitably replace. In turn, infections of these large redial species are very rarely invaded and, if so, only by the other member of the pair. Similar, but less extensive observations of larval predation and/or species replacement in the same system by Yoshino (1975) and Kuris (1990) are consistent with my own. Therefore, while it is premature to generalize, patterns of interspecific antagonism among members of the trematode guild that infects *Cerithidea* appear to be very similar to those observed in the much more extensively investigated freshwater trematode assemblages discussed above.

Fernandez and Esch (1991*a*) recently used methods similar to mine to investigate the processes structuring a guild of eight larval trematodes that infect the snail *Helisoma anceps* in the North Carolina pond. Field mark-recapture studies and laboratory challenges of established natural infections with infective egg stages of one of the species show that species with redial stages (Echinostomatidae and Hemiuridae) were generally dominant over species with only sporocyst stages in their life cycles. There was also evidence that priority effects were important for some pairs of redial species; whichever species was established first resisted superinfection by the other.

Why do dominant redial species exhibit predatory antagonism against co-occurring species? One possibility is that the interactions are accidental or facultative; the rediae are consuming host tissues, and simply ingest other trematodes that lie in their path. Several observations argue against this hypothesis. First, rediae seldom, if ever, prey on conspecific larvae, as might be expected if predation was indiscriminate. Second, predatory rediae appear to attach actively larvae of subordinate species in a directed fashion (Lim and Heyneman, 1972, pp. 205–206); the victim does not respond. Initiation of an attack is often sudden, as if elicited by some stimulus produced by the subordinate species. In addition, mobile, predatory rediae tend to aggregate in host tissues where the larvae of subordinates are concentrated (*e.g.*, Lie, 1967; Basch, 1970).

Lie (1969) suggested that larval predation may ultimately be a form of extreme interference competition over the limited host resource. At the same time, the dominant may receive the added benefit of consuming a more nutritious and easily assimilated food than the host itself, another trematode. Basch *et al.*'s (1970) observation that predatory rediae of *Paryphostomum segregatum* grow more rapidly in a mixed-species infection than when alone is consistent with this idea, but it may simply be that for a large, energy-demanding species such as *P. segregatum,* the negative impact of interspecific competition with a smaller, subordinate species is less than that which it suffers when competing exclusively with members of its own species. The patterns, origins, and consequences of the general phenomenon of intraguild predation are only beginning to be explored conceptually and experimentally (Polis *et al.*, 1989).

Two additional mechanisms might act to reduce the numbers of mixed-species infections. The presence of an already established infection by one species may somehow deter miracidia of other species from attempting to superinfect, or render such attempts unsuccessful. This mechanism was suggested by several early workers Sewell, DuBois, and Porter, as cited in Lie *et al.*, 1966). Direct observations of miracidial penetration behavior (Lie *et al.*, 1966) and detailed histological studies (Lim and Heyneman, 1972) lend no support to this proposal. Miracidia of subordinate species penetrate snails already infected by a dominant trematode at the same rate as they do uninfected controls, and vice versa. A deficit of mixed-species infections could also occur if such infection were more pathogenic than single-species infections. If this were the case, multiply infected snails would suffer higher rates of mortality and therefore be underrepresented in collections. In an extensive search of the literature, I found no hard evidence bearing on this idea.

OTHER PATTERNS OF INTERSPECIFIC ASSOCIATION

I have focused almost exclusively on negative associations and antagonism among

trematode symbionts. However, in most analyses of larval trematode associations, there are a fair number of species pairs that show no evidence of interaction; that is, they occur at frequencies that would be expected if the infections were distributed independently of one another. In some cases, this result is the product of small sample size and low statistical power (*i.e.*, large Type II error: failure to reject a false null hypothesis). In others, the species may simply not interact very strongly. More surprising is the observation that some species occur in mixed-species infections more often than would be expected by chance (*e.g.*, Bourns, 1963; Heyneman and Umathevy, 1968; James, 1969; Vernberg *et al.*, 1969; Robson and Williams, 1970; Kuris, 1990). Possibly the most extreme natural example is the estuarine schistosome, *Austrobilharzia terrigalensis*. Ewers (1960) first noted the high frequency with which this schistosome co-occurred with the heterophyid, *Stictodora lari*. Subsequent investigations by Walker (1979) and Appleton (1983) found the schistosome exclusively in mixed infections, almost all were double infections with one of three redia-producing species, but most frequently with *Stictodora*. The *Austrobilharzia* sp. that infects *Cerithidea californica* is similarly found primarily in mixed-species infections (Kuris, 1990; Sousa, 1993). As Kuris (1990) points out, positive associations of larval trematode species rarely imply mutual benefit. Usually, one species is suppressed; it produces fewer cercariae, its larval stages are damaged or killed, or it is displaced from its usual site of infection (*e.g.*, Robson and Williams, 1970; DeCoursey and Vernberg, 1974). Indeed, this is true in the case of mixed-species infections involving *Austrobilharzia*. Walker (1979) showed that the presence of the schistosome reduced cercarial production and redia size of co-occurring species, although the redia-producing species were not excluded as in the case of *Trichobilharzia* and *Hypoderaeum* (Ow-Yang and Lie, 1972) described earlier.

Walker (1979) hypothesized that the obligate association of *Austrobilharzia* with other trematodes was either a nutritional dependence in which the other trematodes somehow make host-derived nutrients more available to the schistosome, or the result of suppression of host immunological defense by a prior heterospecific infection. While the precise mechanisms operating in this particular system have yet to be investigated, the laboratory studies of K. J. Lie and co-workers on freshwater trematodes have provided very important insights as to why an infection of one trematode species might facilitate the establishment of an infection by another. A number of such cases have been documented in the freshwater trematode guild, including the following three examples. The rate of infection by *Echinostoma hystricosum* was found to be higher in snails previously infected by *Trichobilharzia brevis* than in uninfected control snails (Lie *et al.*, 1973). *E. hystricosum* eventually replaced the schistosome. Lie *et al.* (1976) documented longer survival of *Echinostoma lindoense* larvae if the snail had a prior infection of *E. lindoense, Paryphostomum segregatum,* or *Schistosoma mansoni*. Finally, Lie *et al.* (1977) found that a refractory strain of the host snail, *Biomphalaria glabrata,* became susceptible to *S. mansoni* if first infected by *E. lindoense, E. paraensei,* or *E. liei*. As in the first study, the schistosome was later excluded by the echinostomes. Subsequent research, reviewed by Lie (1982), Lie *et al.* (1987), Meuleman *et al.* (1987), and Bayne and Yoshino (1989), has revealed that in such cases the prior infection interferes with the snail's primary mechanism of resistance to infection, *i.e.*, snail hemocytes that phagocytose or encapsulate invading parasites. This interference is induced by substances secreted by the first parasite that are directly targetted at the hemocytes. Once the snail's innate defense is reduced or neutralized by the first infection, a second parasite can establish more easily.

CONCLUSIONS

Biotic interactions, both agonistic and facilitating, potentially play important roles in structuring the infraguilds of larval trematodes that infect gastropods. Overall, hierarchical negative interactions, involving either predation or various forms of non-predatory antagonism, seem to predominate. Assuming that the host snail is equally susceptible to the trematode species/strains

in question (*e.g.*, Lim and Heyneman, 1972; Richards, 1976; Rollinson and Southgate, 1985; Loker and Bayne, 1986), the frequency of such interactions depends ultimately on the availability of infective trematode stages relative to snail numbers. Temporal and spatial variation in the abundance of eggs and miracidia of different species determines how often a snail is infected simultaneously by two or more species, or established infections are challenged. Therefore, the diversity of trematodes within a population of host snails depends not only on the outcome of intramolluscan interactions, but on the transmission and establishment of new infections from the definitive vertebrate host (Sousa, 1990, 1993; Fernandez and Esch, 1991*a, b*; Williams and Esch, 1991). This particular dispersive phase of the trematode life cycle merits greater attention from ecological parasitologists, much as the phenomenon of larval recruitment has become a focus of investigations by marine ecologists of the population and community dynamics of free-living organisms with complex life cycles (*e.g.*, Sale, 1977; Underwood and Denley, 1984; Connell, 1985; Gaines and Roughgarden, 1985; Warner and Chesson, 1985).

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