Effects of Disturbance on Host-Parasite Interactions: A Case Study Involving the Pacific Giant Salamander

by

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ABSTRACT

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Impacts of parasitic diseases in wildlife scale from the individual to the community such that alterations in parasite-host interactions can cascade through the community. Recent studies suggest that anthropogenic disturbance significantly affects parasitic diseases in wildlife that could further exacerbate direct effects of disturbance on free-living species. Few studies rigorously evaluate the impact of disturbance on the incidence or abundance of parasitism in hosts. Even fewer assess mechanisms of disturbance that alter parasitism in wildlife. My work addresses the impacts of clearcut logging on parasitism by a digenetic trematode, *Cephalouterina dicamptodoni*, of three stream-dwelling hosts. The trematode obligately and sequentially infects the Pacific giant salamander, *Dicamptodon tenebrosus*, the stonefly, *Calineuria californica*, and the snail, *Juga silicula*. My goals were to 1) assess whether parasite density or prevalence in each host responded to logging, 2) evaluate the mechanistic relationship between logging-induced disturbances and parasitism, 3) experimentally evaluate the functional response of transmission to host density between each donor-target host pair, and 4) to

relate the functional response of transmission to the biotic changes in parasitism associated with logging-induced disturbances.

In 1998, I conducted censuses of each host in 7 unlogged basins, 7 logged basins and 4 logged basins that also experienced debris flows. All stream basins were located in the McKenzie or Little Fall creek watersheds, of the central Cascade Mountains, Oregon. During each of the 18 censuses, I measured geomorphic and other abiotic features of the creeks that responded to logging-induced disturbances. To evaluate whether parasitism responds to logging, I compared the prevalence and density for each parasite stage and host species between streams in the 3 forest types: unlogged, logged and logged-with-debris flows. I correlated parasite levels with abiotic and biotic features across forest types to assess whether the relationship between parasites and their hosts changed with disturbances. I conducted field experiments during 1995, 1996, and 1997 in which I manipulated donor and target host densities to test for the transmission response of parasitism to densities of each host.

The data reveal a clear and significant effect of logging on parasite population dynamics. Each stage in the life cycle of the parasite is decreased by disturbances associated with logging, but the strength of the effect is dependent upon severity of logging. In addition, the relative roles of abiotic and biotic variables in the parasite-host system are context dependent.

Logging has a significant effect on host-parasite relationships and on the density of each host, which suggests that logging alters the success and functional response of parasite transmission. Based on my experimental study, non-linearities in transmission exist in at least two of the three transmission stages. In addition, transmission is context

dependent in that limitations in transmission depend differentially upon reproduction and recruitment but in a predictable manner that is subject to the relative densities of donor and target hosts. According to epidemiological theory, non-linear transmission generates complex model behavior resulting in multiple stable states. The patterns of parasitism and host density that followed logging, coupled with the experimental results that non-linearities exist in transmission of *C. dicamptodoni*, suggest that logging causes this parasite-host system to shift into alternate states that depend upon the severity of logging-induced disturbances.

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- Anonymous NSF reviewer who helped fund this work

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CHAPTER 1 Introduction

"[Man] has created wonderful inventions to protect himself from his much less offensive and more vulnerable neighbors and has so far completely failed to grasp the significance of the vastly greater importance of these small animals which torment him openly, carry and transmit diseases which have done him to death, which have destroyed his timber and his homes, his crops and domestic animals, and have left him no place on earth to set his foot unmolested!"

- E.O. Essig (1929:499)

We still do not understand the full import of "these small animals" that carry disease, nor do we understand the full import of disease itself even though early observations mention the impacts of introduced pathogens on wildlife (e.g., Osborn 1906). We are just beginning to gather evidence that shows how the effects of parasites can scale to the community (Lessios 1988, Kohler and Wiley 1997). More limited than our knowledge of parasite-host interactions, is our understanding of anthropogenic impacts on parasitic diseases. Recent reviews on the subject demonstrate the necessity to understand how parasites respond to anthropogenic change (Harvell 1999, Daszak 2000), yet few studies rigorously assess the potential for anthropogenic disturbances to alter parasitic diseases of wildlife. My work addresses the impacts of clearcut logging on parasitism by a digenetic trematode, *Cephalouterina dicamptodoni*, of three stream-dwelling hosts. The trematode obligately and sequentially infects the Pacific giant salamander, *Dicamptodon tenebrosus*, the stonefly, *Calineuria californica*, and the snail, *Juga silicula*. My goals were to 1) assess whether parasite density or prevalence in each host responded to logging, 2) evaluate the mechanistic relationship between logging-induced disturbances and parasitism, 3) experimentally evaluate the functional response of transmission to host density between each donor-target host pair, and 4) to relate the

functional response of transmission to the biotic changes in parasitism associated with logging-induced disturbances.

In Chapter 2, I discuss the limitations in the current body of evidence to assess the response of parasitic diseases to anthropogenic disturbance. I provide a limited review of the literature and suggest some methods to detect impacts of disturbances on diseases.

In Chapter 3, I present the results from censuses I conducted on each host in 7 unlogged basins, 7 logged basins, and 4 logged basins that also experienced debris flows. All stream basins were located in the McKenzie or Little Fall creek watersheds, of the central Cascade Mountains, Oregon. During each of the 18 censuses, I measured geomorphic and other abiotic features of the creeks that responded to logging-induced disturbances. To evaluate whether parasitism responds to logging, I compared the prevalence and density for each parasite stage and host species between streams in the 3 forest types: unlogged, logged and logged-with-debris flows. I correlated parasite levels with abiotic and biotic features across forest types to assess whether the relationship between parasites and their hosts changed with disturbances.

Chapter 4 details analyses of field experiments I conducted during 1995, 1996, and 1997 in which I manipulated donor and target host densities to test for the transmission response of parasitism to densities of each host.

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CHAPTER 2 On the Necessity and Evidence to Detect Disturbance-Related Events in Parasite-Host Dynamics

Impacts of macroparasitic diseases can scale from the individual host to the community (e.g., Anderson and May 1978, 1982, Holmes 1982, Scott 1988, Scott and Dobson 1989), however our knowledge of the mechanisms that drive parasite-host dynamics in wildlife is still limited. Even more limited is our knowledge of how parasites of wildlife respond to anthropogenic disturbance. Many of the current emergent and resurgent parasitic diseases in wildlife appear to be associated with anthropogenic activities (Harvell 1999, Lafferty and Kuris 1999, Daszak et al. 2000), but the paucity of data on disease in general and ecological aspects of disease in particular limit our ability to assess causality. There are cases in which causality can be assigned, but these are generally limited to zoonoses (e.g. Lyme's disease, Spielman 1994) and introduced pathogens of endangered or charismatic species (e.g. upper respiratory disease in desert tortoises, Jacobson et al. 1991, malaria in Hawaiian birds, Van Riper et al. 1986, distemper in black-footed ferrets, Thorne and Williams 1988, whirling disease in trout, Hedrick et al. 1998). To date, there are no studies that rigorously assess the impacts of anthropogenic disturbances, such as habitat loss and degradation, on macroparasitic diseases in animal populations. Examples from human epidemiology do provide clues to the importance of anthropogenic disturbance in parasitic diseases (Patz et al. 2000). For example, deforestation is linked to increased prevalence of malaria in Brazil, Africa and Trinidad (for review see Walsh et al. 1993). Dam construction is implicated in the

emergence of Rift Valley Fever in Egypt (Shope and Evans 1993) and Senegal (Lederberg et al. 1992). But even for diseases in humans, the evidence needed to link disturbance with disease is often anecdotal (e.g., McSweegan 1996). Due to the extent and severity of anthropogenic disturbances and the likelihood that parasite-host dynamics will be significantly altered by them, it is essential that we begin to rigorously assess the response of different types of disease to disturbances and then apply this knowledge in restoration and conservation efforts. This chapter focuses primarily on macroparasites that, due to their complex life cycles, will be especially sensitive to disturbance due to the diversity of hosts and/or transmission stages necessary to complete their life cycles. In addition to their inherent sensitivity to disturbance, macroparasites can have significant, but difficult to detect effects on their host populations (Hudson et al. 1998). Indeed, recent work demonstrates that nematode parasites can cause population cycles in grouse (Hudson et al. 1998). Thus a change in macroparasitism in response to disturbance could greatly affect host populations, and the dynamics between hosts and parasites (May and Anderson 1979). This chapter provides a brief synopsis of the current status of knowledge on disturbance and parasitism, and considers some of the evidence necessary to assess the relationship between anthropogenic disturbances and parasite-host interactions.

POTENTIAL EFFECTS OF ANTHROPOGENIC DISTURBANCE ON MACROPARASITES

Anthropogenic disturbances are broadly defined here as factors that cause habitat loss and/or degradation, and include but are not limited to deforestation, development, agricultural practices, eutrophication, and pollution. I do not discuss effects due to

introduced pathogens or to climate change as these are discussed elsewhere (Daszak et al. 2000, Dobson and Carper 1992).

Macroparasites with complex life cycles are characterized by one or more parasitic stages that are dependent upon their hosts for food and shelter. A diversity of taxa are covered by the term "macroparasite", but here I focus on the higher-order taxa that are ubiquitous, abundant and likely to, or are known to, affect host dynamics. These taxa include nematodes, digenetic trematodes, cestodes and protozoans. The mode of transmission between hosts is quite diverse among the macroparasites. Transmission can be accomplished either through a trophic link, via a vector, or by one or more non-parasitic stages that are either free-living or resting stages in the environment (see Table 2.1). Anthropogenic disturbance can disrupt complex host-parasite systems directly by altering abiotic factors that impact the free-living stages of the parasite, or indirectly by affecting host abundance or distribution (in space or time) (Sousa and Grosholz 1991, Sapp and Esch 1994, Marín et al. 1998). The strength and direction of change in parasitism will depend upon the type of disturbance and the particular stage of the parasite that is affected.

Direct impacts of anthropogenic disturbance affect the ability of parasites to transmit between hosts. Alteration in temperature, moisture, light, and habitat structure all could affect the ability of transmission phases of the parasite to successfully enter and pass through the environment between hosts (e.g., Penner 1941, Sousa and Grosholz 1991, Esch and Fernández 1993). In the following paragraphs, I provide a brief discussion of known effects of environmental variables on parasite stages. For a more

thorough treatment of this subject, see Smyth and Halton (1983), Sousa and Grosholz (1991), and Esch and Fernández (1993).

For stages that transmit through aquatic environments, changes in abiotic factors such as temperature, stream velocity, habitat heterogeneity and possibly light penetration can directly affect reproduction and survival. Higher water temperatures often lead to increased rate of production of miracidial and cercarial stages of trematodes (Anderson et al. 1982, Evans 1985, Jewsbury 1985, Shostak and Esch 1990), but sometimes at the expense of survivorship of the free-living stages (Anderson et al. 1982, McKindsey and McLaughlin 1994). On the other hand, increases in stream velocity can affect free-living parasite stages by damaging individuals and causing a decrease in transmission success of parasite infective stages (Upatham 1974, Jewsbury 1985). Conversely, a significant decrease in the natural flow regime, as with river impoundments, could also inhibit migration of aquatic transmission stages causing parasites to find fewer hosts. Transmission of the free-living stages can also be directly affected by changes in geomorphology and habitat heterogeneity (Chernin and Perlstein 1969, for review see Sousa and Grosholz 1991). On the scale of the parasite, changes in habitat heterogeneity could include altered substrate composition, vegetation diversity, and even host density.

Most macroparasitic stages that transmit through terrestrial environments are encysted or encased within a desiccation-resistant mass (such as mucous or feces) (Table 2.1). These parasites are transmitted passively when a host serendipitously consumes parasitic larvae while grazing. However, high temperatures, if coupled with low humidity can cause desiccation of parasite eggs that are deposited in terrestrial environments (e.g., Penner 1941, Schmitz and Nudds 1994). Since free-living larvae in

terrestrial environments are generally transmitted passively, then a change in host foraging behavior due to disturbance could also affect parasite transmission.

Indirect effects of disturbance on parasitism are just as likely to occur as direct effects, and will be manifest through changes in host population characteristics. Natural disturbances can play a strong role in structuring patterns of species diversity and distribution (Sousa 1984, 2001, Pickett and White 1985). Although it is more difficult to assess effects on host species that result from anthropogenic disturbance, we know that the effects do occur (e.g., Crooks and Soulé 1999, Doak and Morris 1999). In fact, it was habitat fragmentation and loss of prey coupled with an outbreak of canine distemper that lead to the near extinction of Black-footed ferret populations (Thorne and Williams 1988).

Whatever direction of effect disturbance has on host populations, changes in the density, distribution, age structure or genetic diversity of hosts could have strong impacts on parasite populations. Based on basic mass-action models, a decrease in density of either infected or susceptible host would lead to a decrease in parasite transmission (e.g., May and Anderson 1979). If host density was forced below the threshold level, then the parasite could be completely lost from the system. Conversely, an increase in host density should lead to an increase in parasite transmission. In addition, disturbance-induced changes in host aggregations can affect the incidence of infection. The response of parasites to host density varies with scale. Small-scale aggregations of hosts can increase the probability of a parasite finding a host due to an increase in the chemical stimuli for parasite transmission (Esch and Fernández 1993). Large-scale aggregations that affect the habitat complexity perceived by the parasite can lead to lower levels of

host infection (Grosholz 1994). Habitat fragmentation could lead to lower immigration and emigration of hosts, causing a decrease in genetic diversity within populations. Lower diversity among hosts can lead to decreased resistance to parasitism (Lively 1995). Habitat fragmentation caused by roads can also increase the probability of parasites invading (e.g., Loye and Carroll 1995). For more extensive reviews of how direct and indirect effects alter parasite populations see (e.g., Dobson and Carper, 1992, McCallum and Dobson 1997, Real 1996).

EVIDENCE FOR ANTHROPOGENIC DISTURBANCE AND PARASITISM: HUMANS

To date, the best evidence for disturbance-induced changes in parasitism come from the epidemiological literature. Most of the evidence for change is based on long-term epidemiological databases that provide information on disease incidence before and after the disturbance. Because there are usually no controls for the disturbance, it can be difficult to assess the mechanism driving the change in parasitism by studying only prevalence of disease in humans. However, most epidemiological studies include information on vector or intermediate host densities associated with disturbance. An interesting example of this is the changing incidence of schistosomiasis in Egypt along the Nile River and delta from 1936 until the present. Schistosomes are trematodes that obligately and sequentially infect vertebrates and aquatic snails. There is no 3rd intermediate host. Before construction of the Aswan dam, prevalence of *S. haematobium* in humans along the northern section of the Nile River was between 50% and 75% in humans (El Alamy and Cline 1977, Abdel-Wahab et al. 1979). These data were collected by different researchers and in different geographic locations between 1937 and 1958

(Scott 1937, Weir et al. 1952, Van der Schalie 1958), but the general trend was for high prevalence of infection. Following construction of the dam, river flow downstream of the dam decreased and flow was regulated so that flood/drought periods were non-existent. This lead to a change in irrigation practices such that canals were flooded yearlong, instead of being closed for the flood season, as was the historical irrigation practice (Malek 1975). Malek (1975) assumed that this constant source of water in the canals lead to the increased densities of snails. An interesting twist on this example is that even though snail densities increased, prevalence of *S. haematobium* decreased to levels between 3.2% and 27% following construction of the dam (El Alamy and Cline 1977, Abdel-Wahab 1979). Without a thorough knowledge of parasite and snail systematics, a knowledge of parasite and snail densities before and after the dam construction, and new techniques to assess parasitemia in humans, the Aswan dam could have been lauded for its role in decreasing schistosomiasis. Although snail densities did increase, the relative densities of two snail genera switched following construction of the dam. *Bulinus* sp., the intermediate snail hosts of *S. haematobium*, were abundant prior to construction of the dam whereas *Biomphalaria* sp., which are intermediate hosts of *S. mansoni*, were rare. Concurrent with the changes in relative abundance of the snail genera and the decline in incidence of *S. haematobium*, was the increase in incidence of *S. mansoni* from around 4% to roughly 60% (El Alamy and Cline 1977, Abdel-Wahab 1979). From these data, some have concluded that the Aswan dam caused the shift from a *Bulinus* dominated system to a *Biomphalaria* dominated system, and that these changes lead to the shift in incidence of the schistosome species (e.g., Lafferty and Kuris 1999). However, a more careful review of the literature shows that the techniques used to detect schistosome

infection prior to construction of the dam were not as sensitive to infection by *S. mansoni* as they were to *S. haematobium* (El Alamy and Cline 1977). So the difficulty here is in interpreting whether the relative change in parasite species is real or artifactual. If the change is real, then the absolute prevalence of infection by schistosomiasis does not appear to increase, only the relative proportion of each schistosome species. Still, *S. mansoni* is more pathogenic than is *S. haematobium*, and the potential increase could have a significant effect on the morbidity and mortality of humans in the Nile Delta.

The point is, that detecting anthropogenic changes in complex parasite-host dynamics is in itself complex and even with a disease as well studied as schistosomiasis, the sampling regime and design to detect changes will inherently affect the ability to interpret the evidence. It is thus necessary to thoughtfully develop a rigorous sampling regime in order to detect not only the changes in parasitism associated with anthropogenic disturbances, but also the mechanisms driving those changes.

A limited survey of the literature on disturbance and human parasitic diseases revealed a diversity of epidemiological techniques to determine associations between parasites and disturbance. Data from most epidemiological studies could not definitively link parasitism with disturbance, but the data were used to infer that disturbance was the causative agent (E,H in Table 2.2). Some studies noted an increase in the incidence of disease through time and suggested hypotheses for the increase (H in Table 2.2). For the anisakis example, McCarthy and Moore (2000) suggested that anisakis was increasing due to the increased availability of the definitive hosts: e.g., whales and seals. The increase in density of definitive hosts is due to decreased hunting pressure on marine mammals. Epidemiological censuses conducted on several spatial and temporal scales,

as they were for malaria and Onchocerciasis, provided the best evidence for anthropogenically-induced changes in parasitic diseases of humans.

Although methodologies and sampling rigor varied dramatically among studies, there was a trend of increased human disease incidence associated with anthropogenic disturbances (Table 2.2). Of the 19 examples I report here, only 5 studies reported a decline in disease. Of these, the declines in malaria, onchocerciasis, and both species of schistosome were associated with declines in their intermediate hosts but with simultaneous increases in other species of the disease-causing parasite (Table 2.2). These increases were attributed to a switch in relative abundance of the intermediate or vector host of these parasites, just as in the schistosomiasis example from Egypt. The patterns in parasitic diseases that are associated with disturbances could be due to reporting bias, my review bias, or to actual effects. Since this is not an exhaustive review, I cannot make broad generalizations about the response of parasitism to the different modes of disturbance. It is interesting to note that of the few parasites that decreased following disturbance, all had indirect life cycles and were associated with a decreased abundance of their intermediate host. As described in the next section, the relationship between disturbance and wildlife diseases is more variable (Table 2.2).

Evidence for Anthropogenic Disturbance And Parasitism: Wildlife

In contrast to the nearly uniform increase of parasites in humans following disturbance, there was no general trend in response of parasites in wildlife, even within disturbance categories and studies (Table 2.2). Of the 18 examples, 4 found increased parasitism following disturbance, 8 found decreased parasitism, 4 found no effect, and 2

found mixed results depending on spatial (Bourque and Esch 1974) or temporal (Lahtinen 1989) differences between samples. Granted the data come from only 11 studies, but variability in the data in light of strong patterns in human disease is surprising. This could be due to the bias in sampling *for* increases in human diseases, instead of sampling *whether* humans have diseases. However, it is also important to assess the methods for testing the effects of disturbance on disease. Two studies were anecdotal. Cort et al. (1960) collected data on trematode parasites of snails for one locality in two years, 1936 and 1956/1957. He collected snails on 2 dates in the first year and 3 dates in the second. During the 20 years between sampling dates, the lakeshore apparently supported a "greatly increased number of summer cottages" (Cort et al. 1960) to which Cort et al. attributed a "decline", albeit unquantified, in vertebrates, especially birds. In comparing the prevalence of infection in snails between the two sample periods, Cort et al. found little evidence for decline. In 1936, prevalence for each date was 38.4% and 77.4% respectively. In 1956/1957, prevalence for each date was 15%, 12.4%, and 39.8% respectively. At the same time, Cort et al. claimed to see a decrease in the incidence of certain species. Although it is true that 5 rare parasites and 2 more abundant parasites were not detected in the 1956/1957 samples, it is also that fewer snails were collected in the second year. More importantly, parasites are extremely patchy in space and time and there is no reason to expect that parasite populations should stay stable over 20 years even in the absence of anthropogenic disturbance.

The second study is suggestive of Cort's, but less well-defined. Lafferty (1993) sampled a population of snails in a marsh near a "busy highway intersection" and compared prevalence of infection in this population to prevalence from a population of

snails that were in an adjacent marsh. No snails near the highway were infected whereas 25% of snails were infected in the adjacent marsh. In a later review (Lafferty and Kuris 1999), he concluded that the "disturbance" from the highway was the causative factor in the "decreased" parasitism. Neither Cort et al (1960) nor Lafferty (1993) provide the evidence necessary to assess 1) whether the differences between time or place are real, 2) what exactly the disturbance is, or 3) what exactly is **is**.

My review for effects of disturbance on parasites of wildlife, showed a number of unreplicated studies, in either space and/or time (C, CM(nr) in Table 2.2). Several studies were post-hoc, but included replicates in space (CM in Table 2.2). There was also one 20 year study that assessed whether the prevalence and density of a trematode parasite would respond to the loss of lake eutrophication (Marcogliese et al. 1990). There was no control for comparison, but the long time series of data before and 4 years after provide strong inference for effects of eutrophication on parasite levels. Clearly there are limitations to these types of studies.

DETECTING RESPONSE OF PARASITISM TO ANTHROPOGENIC DISTURBANCE

Clearly there is a need to understand how parasitism in wildlife is responding to anthropogenic disturbances. With limited resources available for research, it is important to set priorities for which species, both parasite and host, will be most important for a focus of study. Following this decision, it is essential to develop rigorous sampling and analytical protocols to provide the body of evidence necessary to 1) detect whether a change in parasitism has occurred and 2) assess whether anthropogenic disturbance is responsible for that change.

Since anthropogenic disturbances are likely to have effects on many parasite-host systems, it is important to carefully choose study species that will maximize our knowledge of these types of interactions and to minimize the effects of disturbances on communities. Responses of a parasite to disturbance will be best understood in a closed system, such as a lake or river, within which the parasite completes its life cycle. Parasites that infect only aquatic species, or only terrestrial species will provide the best models for understanding the relationships between disturbance and parasitism.

Host selection should be based on its importance in the community. Effects of disturbance on parasite-host interactions in which the host is a keystone species (Power and Mills 1995) or strong interactor (MacArthur 1972, Paine 1980) will have more effect on the community. Although anthropogenic disturbance was not the driving force, microsporidian infections caused mass mortality in the caddisfly, *Glossosoma nigrior*, a dominant grazer of trout streams (Kohler and Wiley 1997). Parasite-induced removal of the caddisfly from streams caused a significant increase in abundance in most grazers, filter-feeders and periphyton.

Once the species are selected, it is essential to design a study that will be able to assess whether parasitism has changed, and whether that change is caused by disturbance. The design will necessarily be dependent upon the species, habitat, and frequency and spatial extent of the disturbance. To narrow the scope of the project, hypotheses for how disturbance can affect parasitism could easily be based on the studies presented here or elsewhere in the literature.

Census data are likely to be a large part of understanding effects of disturbance on parasitism. For disturbances that are predictable, gathering data for Before After Control

Impact (BACI) studies would be an ideal way to assess whether parasitism changes with disturbance (Schmitt and Osenberg 1996). Lafferty (1997) suggests other sampling techniques for data that cannot fit into a BACI design. In many cases BACI designs will not be applicable due to the lack of "Before" data. For certain of these types of disturbances, it is possible to use mensurative experiments to assess the effect of disturbance on parasitism. For example, the response of parasitism to disturbance could be evaluated post-hoc for disturbances that are replicated in space, such as logging. The following chapters present the results of a study that uses post-hoc mensurative censuses and experimentation to assess the effects of clearcut logging on parasitism in stream communities.

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Table 2.1. Diversity of the major transmission stages of macroparasite taxa that are likely to respond to anthropogenic disturbances and have an impact on host dynamics. Definitive hosts (D) are infected by the sexually reproductive adult parasite stages whereas intermediate hosts (I) are infected by non-reproductive or asexually reproductive larval parasite stages. Parasites transmit between hosts via active (A) or passive (P) means. Taken in part from Esch and Fernández (1993). For a more detailed treatment of transmission stages and taxa, see Sousa and Grosholz (1991).

Taxa	# of obligate intermediate hosts	Modes of Transmission	Transmission Stage	Target Host	Environment
Digenetic trematodes	1 to 3[1]	A	miracidium	I	aquatic[3]
	generally 2	A	cercaria	D, I	aquatic[4]
		P	egg	I	aquatic, terrestrial
		P	metacercaria[2]	I	aquatic, terrestrial
Nematodes	1	P	egg	D	terrestrial
		P	microfilaria	I	terrestrial/vectored
		A,P	larva	D	aquatic
Cestodes	0 or 1	P	egg	I	aquatic, terrestrial
		P	cysticercus	D	aquatic, terrestrial
		A	coracidium	I	aquatic
Protozoa	0 or 1	P	gametocyte	D	terrestrial/vectored
		P	cyst	D	aquatic
		A	various forms	D	terrestrial/vectored

[1] e.g., *Halipegus occidualis* has 3 intermediate hosts (Goater et al. 1990)

[2] Metacercariae can encyst inside a host or on substrate that is grazed or consumed by the definitive host. Thus for species that encyst within hosts, this stage is an interesting combination of parasitic and transmission stage.

[3] Intermediate host is always a mollusk

[4] In some cases, as with *Schistosomiasis* and other blood flukes of terrestrial vertebrates, transmission is within an aquatic environment, but the target host is terrestrial.

Table 2.2 Some examples of the effects of anthropogenic disturbance on parasitism from human epidemiological and parasitological literature, and the evidence in support of these patterns.

Parasites of Humans

Disturbance	Host(s)	Parasite Taxon	Parasite Species	Transmission*	Effect	Evidence	Citation
Deforestation	humans, mosquitoes	Protozoa	*Plasmodium sp.* (malaria)	passive, vector	+	C,E,M	Walsh et al. 1993
	humans, mosquitoes	Protozoa	*Plasmodium sp.* (malaria)	passive, vector	-	E	Walsh et al. 1993
	humans, horseflies	Nematoda	*Loa loa* (loiasis)	passive, vector	-	H	Walsh et al. 1993
Development,	humans, sandflies	Protozoa	*Leishmania sp.* (Leishmaniasis)	passive, vector	+	E	Walsh et al. 1993
Urbanization, &	humans, livestock	Protozoa	*Cryptosporidium sp.* (Cryptosporidiosis)	passive, aquatic	+	E,H	Ambroise-Thomas 2000
Overpopulation	humans, other mammals	Protozoa	*Giardia sp.* (Giardiasis)	passive, aquatic	+	H	Ambroise-Thomas 2000
	humans, triatomine bugs	Protozoa	*Trypanosoma cruzi* (Chagas' Disease)	passive, vector	+	C,E	Forattini 1989
	humans, horseflies	Nematoda	*Loa loa* (loiasis)	passive, vector	+	H	Walsh et al. 1993
	humans, carnivora	Cestoda	*Echinococcus sp.* (Echinococcosis)	passive, terrestrial	+	E,H	Eckert et al. 2000
Logging	humans, blackfly	Nematoda	*Onchocerca volvulus*	passive, vector	-	E,H,M	Walsh et al. 1993
	humans, blackfly	Nematoda	(Onchocerciasis, River Blindness)	passive, vector	+	H	Walsh et al. 1993
	humans	Nematoda, Strongylida	hookworms	active, terrestrial	+	E,H	Lilley et al. 1997
	humans, snail	Trematoda	*Schistosoma haematobium*	active, aquatic	+	E,H	Walsh et al. 1993
	humans, snail	Trematoda	*Schistosoma intercalatum*	active, aquatic	-	E,H	Walsh et al. 1993
River Dams	humans, fish, birds	Nematoda	*Gnathostoma sp.* (Gnathostomiasis)	active, aquatic	+	H	McCarthy & Moore 2000
	humans, snails	Trematoda	*Schistosoma mansoni*	active, aquatic	+	E,H	El Alamy & Cline 1977,
	humans, snails	Trematoda	*Schistosoma haematobium*	active, aquatic	-	E	Abdel-Wahab 1979
	humans, snails	Trematoda	*Schistosoma sp.* (Schistosomiasis)	active, aquatic	+	E	Gryseels et al. 1994
	humans, snails	Trematoda	*Schistosoma sp.* (Schistosomiasis)	active, aquatic	+	E, BAI	Southgate 1997
Hunting	humans, fish, marine mammals	Nematoda	Anisakidae (Anisakidosis)	passive, aquatic	+	H	McCarthy & Moore 2000

Table 2.2 (cont.)

Parasites of Wildlife

Disturbance	Host (s)	Parasite Taxon	Parasite Species	Transmission*	Effect	Evidence	Citation
"Disturbance"	snails	Trematoda	various	various, aquatic	-	A	Lafferty 1993
	snails	Trematoda	various	various, aquatic	-	A	Cort et al. 1960
Eutrophication	mayfly nymphs	Trematoda	*Crepidostomum cooperi*	active, aquatic	+	BAI	Marcogliese et al. 1990
	fish	Trematoda	*Rhipidocotyle campanula*	passive, aquatic	+	C	Valtonen and Taskinen 1988
	fish	Helminth	various	various	- (int) / - (div)	C	Esch 1970
	snails	Helminth	various	various	- (int) / - (div)	C	Esch 1970
Logging	salamanders	Trematoda	*Cephalouterina dicamptodoni*	passive, aquatic	- (int) / 0 (prev)	C,F,M	Poteet, this study
	snails	Trematoda	*Cephalouterina dicamptodoni*	passive, aquatic	- (int) / - (prev)	C,F,M	Poteet, this study
	stonefly nymphs	Trematoda	*Cephalouterina dicamptodoni*	active, aquatic	0 (int) / - (prev)	C,F,M	Poteet, this study
Urbanization	Raccoon	Nematoda	*Baylisascaris columnaris*	passive, terrestrial	0	C,M(nr)	McComb 1981
	Raccoon	Cestoda	*Mesocestoides* sp.	passive, terrestrial	0	C,M(nr)	McComb 1981
Thermal effluents~	snail	Trematoda	various digeneans	active, aquatic	+	C, F	Sankurathri & Holmes 1976
	turtles	Trematoda	*various*	various, aquatic	0 (prev)	C,M	Bourque & Esch 1974
	turtles	Nematoda	*Camallus* sp.		+/- (prev)	C,M	Bourque & Esch 1974
	turtles	Nematoda	various	various, aquatic	- (int)	C,M	Bourque & Esch 1974
	turtles	Trematoda	various	various, aquatic	- (int)	C,M	Bourque & Esch 1974
Pollution~	fish	various	various	various, aquatic	+ / 0	L(P)	Lehtinen 1989
	Earthworm	Protozoa	Gregarines	passive, aquatic	+	C,M(nr)	Pi l & Sterzy ska 1991

~ A thorough review of thermal effects on parasites is available in Langford (1990). A more thorough review of pollution and eutrophication effects are in MaCKenzie et al. (1995).

* Refers to the mode of transmission to the stage that was sampled.

A = Anecdotal. This category is used for unqauntified or perceived disturbances

BAI = Data were collected before and after the anthropogenic disturbance but were not compared to a control site

C = Census data

E = Epidemiological evidence which includes long-term medical databases for geographic incidence of disease, and efficacy of control measures

F = Field Experiment

H = hypothesis

L = Laboratory Experiment

M = Multiple sites

(P) = Laboratory experiment not specifically designed to test for parasite levels, instead the results were discovered post-hoc

(nr) = no replicates

int = intensity, prev = prevalalence, div = diversity of parasites

CHAPTER 3 Logging-Induced Disturbances Alter Pattern and Process of Parasitism in Aquatic Communities

INTRODUCTION

A large body of work provides compelling evidence that anthropogenic impacts on the environment can alter patterns of parasitism in humans (e.g., Epstein et al. 1993, Haines et al. 1993, Hominick and Chappell 1993, Wilson et al. 1994, Colwell 1996, Epstein 1995, 1999). Although just as likely to occur, less is known about the impacts of anthropogenic disturbance on parasites in wildlife. Of the studies that do link changes in parasites of wildlife with anthropogenic effects, the most robust deal with high profile host species that are either endangered (Thorne and Williams 1988, Van Riper et al. 1986), human resources (Burkholder and Glasgow 1997), or charismatic (Harvell et al. 1999). Proposed reasons for the observed alteration in patterns of parasitism include climate induced alterations in parasite dynamics (Harvell et al. 1999), land-use changes altering host distributions and density (Walsh et al. 1993), anthropogenically altered levels of nutrients (Burkholder et al. 1995), and species introductions (Van Riper et al. 1986, review in Daszak et al. 2000). Given the ubiquitous nature of these anthropogenic disturbances, the effects they have on diseases in humans and certain high-profile species will almost certainly be manifest in other species of wildlife.

One of the main limitations in assessing impacts of anthropogenic disturbances on parasite populations in wildlife, or any population for that matter, is the ability to replicate environmental disturbances. We know that natural disturbances can play a

strong role in structuring patterns of species diversity and distribution (Sousa 1984, 2001, Pickett and White 1985). The effects of natural disturbances are often detected and assessed through replicated experimental manipulations within a designated temporal and/or spatial regime (e.g., Levin and Paine 1974, Sousa 1979, Hobbs and Mooney 1991). Unlike natural disturbances, the effects of anthropogenic disturbances on the environment can be difficult to assess as they are often generated by events that are unreplicated through time or space (for review see Schmitt and Osenberg 1996). In addition, understanding the effects of disturbance on epidemiology can be made more difficult due to migration of infected individuals, parasite stages or vectors away from or into the site of disturbance. Parasites with complex life cycles often infect hosts that cross ecosystem boundaries, such as schistosomes in humans, or migrate long distances, such as *Nanophyetus* (the trematode that transmits salmon-poisoning disease) in salmon. The difficulty then, is in assessing not only whether changes in epidemiological patterns occur, but also whether they are affected by anthropogenic impacts, natural causes, or sampling bias. In some cases, the evidence points compellingly towards anthropogenic effects as the force of change in disease patterns (Thorne and Williams 1988, Van Riper et al. 1986, Burkholder and Glasgow 1997). However, much of the current information on the effects of anthropogenic impacts on patterns of disease in wildlife is anecdotal or qualitatively inferential due to lack of spatial or temporal replication of impacted or control systems (see Harvell et al. 1999, Daszak 2000).

In this study, I focus on disturbances associated with clearcut logging and their effects on parasite-host dynamics in stream communities. To alleviate the problems inherent in an open parasite-host system, I focus on a trematode parasite, *Cephalouterina*

dicamptodoni, that obligately and sequentially infects three aquatic species: the Pacific giant salamander, *Dicamptodon tenebrosus*, the snail, *Juga silicula*, and the stonefly, *Calineuria californica*. By choosing small watersheds with similar amounts of clearcut forest, I can effectively replicate the same, or similar disturbances across space. In addition, similar small watersheds that support mature forests serve as multiple controls. In this study, I census host and parasite populations from 11 creeks in logged watersheds and 7 creeks in unlogged watersheds. I evaluate similarities among watersheds based on geomorphological features that do not respond, in the short term, to logging. In this way, I can control for cross-basin effects, such as elevation and watershed size, that are not affected by logging but that might alter parasite abundance. I also evaluate differences in watersheds based on geomorphological and biological features that do respond to logging. Differences in abiotic and biotic factors between logged and unlogged watersheds are used to measure their relative importance to parasite dynamics. My goals are to determine whether abundance and prevalence in each stage of the parasite respond to logging-induced disturbances. Further, I assess the mechanisms by which disturbance alters parasitism and ask whether the mechanisms themselves are altered by disturbance.

MATERIALS AND METHODS

Study System

Cephalouterina dicamptodoni is a trematode parasite that obligately and sequentially infects larvae of the Pacific Giant salamander, *Dicamptodon tenebrosus*, a common aquatic snail, *Juga silicula*, and nymphs of the stonefly, *Calineuria californica* (Senger and Macy 1953, Schell 1985) (see Fig. 3.1). Adult parasites live and sexually

reproduce in the intestines of their salamander hosts. Eggs of the parasite are deposited into the intestine of the host and released into the stream with the host's feces. When an egg is consumed by a snail host, the egg hatches into a miracidium that migrates to the gonad, and transforms into a mother sporocyst that then produces daughter sporocysts. Sporocysts can become so abundant in the gonad of the snail, that the snails are effectively castrated. Each of these sporocysts asexually produces thousands of virgulate cercariae that are then released through the snail's renal pore into the stream. The free-living cercariae transmit to the 2nd intermediate stonefly host, *C. californica*. Individual cercariae pierce the stonefly's exoskeleton with a scalpel-like stylet and penetrate the stonefly through the incision (Schell 1985). Cercariae encyst to form metacercariae in the muscle tissues of the stonefly at which time they begin to develop gonads and other features of adult anatomy. Cercariae do not appear to discriminate among muscle tissues within the stonefly host as they are found in all areas of the head, thorax and abdomen as well as the legs and cerci of the stonefly (M. Poteet, pers. obs.). The life cycle of the parasite is completed when a salamander eats a stonefly infected with metacercariae. The metacercariae excyst in the digestive system of the salamander, migrate to the intestine, and grow to sexual maturity.

Species Accounts – Larvae of *Dicamptodon tenebrosus* are abundant in cool streams ranging from northwestern California through southwestern British Columbia (Nussbaum et al. 1983, Good 1989). Larvae can occur at densities of up to 3 m^{-2} and often comprise the greatest vertebrate biomass in small streams throughout western Oregon and California (Murphy and Hall 1981, Corn and Bury 1989). In the streams sampled here, larval densities ranged between 0.01 and 0.16 m^{-2} with infection prevalence ranging from

0 to 100% (Table 3.1). During their aquatic stage, *D. tenebrosus* are strongly territorial and cannibalistic. With the exception of a few neotenic individuals, larval salamanders reside in streams for 2 to 3 years until metamorphosis to the terrestrial adult stage (Nussbaum and Clothier 1973).

The first intermediate host of *C. dicamptodoni*, the snail, *Juga silicula*, is abundant below 900m in elevation in streams ranging from Northern California north to southern Washington (Burch 1982). *J. silicula* lives from 3 to 6 years (Earnest 1967, as cited by Diamond 1982). These snails often comprise the majority of total invertebrate biomass in streams (Hawkins and Furnish 1987) and can reach densities of 1500 m^{-2} (Hawkins and Furnish 1987). Snail densities in my censuses ranged from 21 to 334 m^{-2} (Table 3.1). In the streams I sampled, ten species of trematodes infect *Juga* with the proportion of snails infected by *C. dicamptodoni* ranging from 0 to 14% (Table 3.1).

The second intermediate host of *C. dicamptodoni*, the stonefly nymphs of *Calineuria californica*, ranges from central California north through British Columbia and west to Montana (Stewart and Stark 1993). In the central Cascades, stonefly nymphs reside in the creeks for 2 years before metamorphosing to the winged adult stage. In the streams I sampled, stonefly densities ranged from 3 to 28 m^{-2} and infection prevalence averaged from 0 to 100% (Table 3.1).

Census Methods

During June and July of 1998, I conducted censuses of all host species in 18 creeks located in small basins with either heavily logged forests or mature forests (Table 3.2) to measure the relationship between logging, host density, and prevalence and intensity of parasitism within each host species. All basins were located in the McKenzie

River and Fall Creek watersheds in the western Cascade Mountains, Oregon. The USFS and several private timber companies control land ownership in these basins. All of the creeks I sampled were located in the USFS Blue River Ranger District, the USFS Lowell Ranger District or in timberlands owned by Weyerhaeuser Industries or Guistina Company. Basins with mature forests were rare and found exclusively on USFS land. I sampled 7 creeks in unlogged forests that were defined as basins with a forest stand age of at least 80 years. The other 11 creeks I sampled were in heavily logged basins and were all located on timberlands owned by Weyerhaeuser Industries, with the exception of South Fork Gate creek (SFG). The SFG watershed is mostly controlled by Guistina Co. with a small block of USFS owned land at the confluence of Hagan and SFG creeks. I sampled SFG just upstream of that confluence on USFS land. Heavily logged basins were defined as those with ≤ 20-year-old clearcuts covering at least 50% of the basin area. Four of the creeks in logged watersheds experienced mass wasting of more than one tributary that resulted in debris flows (as defined in Leopold et al. 1964) within the creek channels. Debris flows are characterized by the rapid movement of a slurry of soil, stone, and organic material down the stream channel. These intense flows can reconfigure channel morphology. In the streams I sampled the debris flows channelized the creeks by filling in pools and straightening the channel. The straightening is possible because the narrow valley floor limits creek meander such that the energy from the debris flows pushes through and wipes out bends in the creeks.

Debris flows that occurred in 1996 affected 4 streams that I censused in 1998. These debris flows were associated with a rain-on-snow event in February 1996 that

caused major flooding throughout the central Cascades. Timber harvest exacerbates the effects of rain-on-snow events causing increases in streamflow and landslides (Harr 1986, Harr and Coffin 1992). The 4 creeks that did experience debris flows in 1996 were in logged watersheds. Because of the significant effect that debris flows have on creek geomorphology and fauna, I created a separate "disturbance effect" category in my analyses to reflect creeks that were in logged watersheds that experienced debris flows. By separating logged sites without debris flows from those sites that experienced debris flows, I can assess the relationships between severity of disturbance associated with logging and host-parasite patterns. Thus I have three categories of disturbance: undisturbed creeks are those in watersheds without logged ("unlogged", n = 7), moderately disturbed creeks are those in watersheds with clearcut logging ("logged", n = 7), and severely disturbed creeks are those in watersheds with clearcut logging that also experienced debris flows ("logged-with-debris-flow", n = 4).

I selected creeks for the census based on creek bed gradient, basin size, amount of clearcutting, and elevation (see Table 3.2). Creeks ranged from pool-riffle to step-pool morphology depending on creek gradient. Precipitation in this area falls mainly between November and March and averages around 230cm annually (Bierlmaier and McKee 1989). Cobbly and stony loams developed from Tertiary Tuffaceous rocks characterize local soils. Vegetation in these basins is dominated by Western Hemlock (*Tsuga heterophylla*), Douglas Fir (*Pseudotsuga menziesii*), and Western Redcedar (*Thuja plicata*). Due to forest planting practices, Douglas Fir was the dominant tree species in the heavily logged basins.

Since the censuses were conducted over a two month period, there was a possibility that temporal changes in host densities and parasite prevalence could confuse effects of logging. To address this issue, I haphazardly chose sampling dates for each creek so that all creeks with the same forest type and elevation were not sampled during the same time period (see Table 3.2). Each creek was censused over a two day period to minimize the effect of researcher disturbance on faunal censuses. I collected surber samples and geomorphological measurements on the first day of sampling, followed by salamander censuses on the second day. Snails and stoneflies were collected using a 30 x 30 cm surber sampler with a 250 μm mesh size. In each creek, surber samples were taken in a stratified random design such that one randomly positioned sample was collected within every 5m segment along a 150m transect for a total of 30 surber samples per creek. The samples were preserved in 70% EtOH and all macroinvertebrates were sorted within 6 months of collecting. All snails greater than 5mm were dissected to determine prevalence and intensity of parasitism. Snails smaller than 5mm were counted but not dissected since prior dissections revealed that snails smaller than 5mm have undeveloped gonadal tissue so are never infected (unpub. data, M. Poteet). Since sporocysts in snails can number in the thousands, direct counts of parasite numbers per snail are prohibitive. Instead of directly measuring parasite intensity, I estimated it by multiplying the percent of the gonad infected by the length of the gonad. I visually determined the percentage of the gonad infected and assigned each snail to one of 5 categories: 0% (uninfected), 25%, 50%, 75% or 100%. Gonad length was not measured for all snails. Instead it was estimated based on shell length, which has a strong linear relationship with gonad length

(Fig. 3.2, based on data I collected from 1,510 snails in April-July 1995 from Cone, Mona, and Hagan Creeks).

Metacercariae in stoneflies are difficult to count since they are microscopic and distributed throughout the body of the host. To aid in finding the metacercariae, I dissolved the muscle tissues of each stonefly in a digestion bath of HCl and Pepsin for 2 to 4 days (Ash and Orihel 1991). The digestion bath does not dissolve parasite metacercariae if removed within that time frame. When the digestion was completed, I counted all metacercarial cysts from each stonefly using a dissecting microscope. Preserved total length and mass of each stonefly was recorded prior to digestion

I collected salamander larvae and neotenes from each creek along the 150m transect using a fish electroshocker. Seine nets were placed at least every 50m along the creek to block individuals that escaped the electroshocker. Shocking was always conducted from upstream to downstream with two or more people netting downstream of the electroshocker. Each stream segment was electroshocked twice. Salamanders were collected and placed in aerated buckets of stream water according to body size. If salamanders were not separated based on body size, then larger salamanders attacked and ate smaller salamanders. Salamanders were processed after the sampling of each 50m segment of stream was completed. During processing, all salamanders were anesthetized in an aerated solution of 0.02% Benzocaine. Each larva was then weighed and measured for total length and snout vent length (mm), TL and SVL respectively. A representative sample of larvae was euthanized and preserved in 70% EtOH. The remaining salamanders were placed in aerated buckets of fresh stream water and released after recovery. Upon dissection of euthanized larvae, all intestinal parasites were counted and

preserved in EtOH. Trematodes were identified, and encysted parasites in the musculature were noted. All prey items from the gut and intestine that were 50% or more intact were counted, identified, and preserved in EtOH. Stomach volume, stomach and intestinal lengths, and stomach mass were recorded.

Geomorphological data – Clearcut logging dramatically alters stream channel morphology, substrate composition, temperature, and scour regime (Dunne and Leopold 1978, Harr et al. 1979, Beschta et al. 1987, Bisson et al. 1987, Everest et al. 1987). Changes in such factors as stream channel morphology, substrate character, temperature, and light penetration could have strong direct effects on parasite dynamics by affecting parasite transmission success. These factors can also indirectly affect parasite dynamics by altering host population features (Chernin and Perlstein 1969, Upatham 1974, Anderson et al. 1982, Jewsbury 1985, Woolhouse and Chandiwana 1989, Shostak and Esch 1990, Sousa and Grosholz 1991, McKindsey and McLaughlin 1994). I measured a variety of abiotic factors associated with logging that are likely to affect parasitism. The factors were measured at two spatial scales, one directly associated with the surber samples, and another associated with the entire transect. At each surber sample site, I measured substrate embededness, dominant particle size, depth, thalweg velocity, and microhabitat. I generated the measure of embededness with the Bovee modification of the Brusven index (Gordon et al. 1992, Bovee 1982, Brusven 1977). The Bovee index is an integrated measure of substrate composition that uses a standardized index that runs from 0 to 9 for specific substrate size categories, and percent embededness.. Thus the index for one quadrat will be a 3 digit number in the form xx.x. The tenths position is an index, on a scale from 0 to 9, of % embededness with 9 being the highest amount of

embededness. The ones position is the dominant small particle size on a scale from 0 to 9, with 0 indicating no small particles, and 9 representing boulders. The tens position is the dominant large particle size and it has the same scale as the ones position. Thus the largest number possible is 98.9, since the dominant small particle size (in the ones position) will by definition always be smaller than the largest particle size (in the tens position). However, there were quadrats that were entirely bedrock; I gave these an index of 100.0.

I conducted one pebble count along the entire 150m transect to determine dominant particle size (Dunne and Leopold 1978). Thalweg velocity was measured every 5m with an electromagnetic current meter that was placed in the thalweg at a depth six tenths down from the surface of the water (Leopold et al. 1964). Thalweg velocity was recorded as the mean of 3 stabilized readings. A reading was considered stabilized when the digital reading of the meter remained constant ($\pm$ 0.1 m/s) for one minute. I defined microhabitat as pool, run, riffle, or cascade. A scale map was drawn of each stream so that habitat designations could be more specifically assessed if necessary.

Every 10m along the transect, I measured bank full width and depth to calculate an average width to depth ratio for the transect. Stream gradient was measured with rod and stadia along the transect at the water's edge. Percent canopy cover was taken every 20m along the transect using a concave spherical densiometer. Air temperature was measured in the morning. Water temperatures were measured with a Min/Max thermometer placed in the creek for the duration of the sample period. Basin area was calculated from USGS 7.5' topographic maps.

Statistical Analyses

Parasite responses to logging – Parasite populations are organized hierarchically within a community of hosts ranging from infection in a single individual of one host species to infection of all individual hosts of all host species. I tested for the effects of logging at three spatial scales: the infrapopulation, the metapopulation and the suprapopulation. An infrapopulation is the total number of conspecific parasites in an individual host (Esch et al. 1975, Margolis et al. 1982) and was measured here as the mean intensity (Margolis et al. 1982). A metapopulation is all the infrapopulations of one parasite species in all conspecific hosts in a community (Riggs et al. 1987) and was measured as density (# m^{-2} of creek) of each parasite stage. A suprapopulation is all stages of a parasite species within all host species in a community (Esch et al. 1975, Margolis et al. 1982). To assess effects of logging on parasite stage, I used mean densities per creek in one-way ANOVAs instead of nested ANOVAs of creek within forest. I did not use nested ANOVAs for parasite analyses due to the patchiness of parasitism and the frequency of 0 data. I conducted one-way ANOVAs for each parasite stage to determine whether the mean parasite intensity in streams of a particular forest type differed. One-way ANOVAs were also used to assess the differences in mean density in streams located in a particular forest type. Post-hoc comparisons were conducted using the Tukey method.

In addition to the two measures of parasite abundance, I tested for changes in parasite prevalence and aggregation in each host species by forest type. Mean parasite prevalence was analyzed by one-way ANOVA and Tukey post-hoc comparisons. I evaluated parasite aggregation among forest types by comparing the variance to mean

ratio for each stage of the parasite (Pielou 1969). All variables were transformed where necessary to meet the assumptions of homogeneity of variance and normality.

Host responses to logging – Prior studies show that clearcutting has strong effects on density, distribution, and size structure of many free-living aquatic species (e.g., Gibbons and Salo 1973, Swank and Crossley 1988, Hartman and Scrivener 1990, Growns and Davis 1991). Since environmentally driven changes in host species are likely to disrupt host-parasite interactions (Sousa and Grosholz 1991, Sapp and Esch 1994), I tested whether certain host population features associated with host-parasite dynamics were affected by logging. The host population features I focussed on were host density, host length, and salamander mass and diet. I assessed the effects of forest type and creek, on density and size of each host species in a nested ANOVA model. Salamander mass was treated similarly in a nested ANOVA model. Salamander diet was measured as the mean number of items found in all salamanders per creek. Differences in the mean # of prey per salamander across forest types were assessed in a one-way ANOVA. Post-hoc comparisons were conducted on forest type using Tukey multiple comparisons.

Parasite intensity is often correlated with host size and age (Pennycuick 1971, Dronen 1978, Anderson et al. 1982, Taskinen and Valtonen 1995). Logging was associated with significant increases in mean host size that could translate into an effect on parasite intensity. I conducted an ANCOVA to test whether parasite intensity was associated with host length across forest types. Slopes of the regression between host length and parasite intensity did vary across forest type. Multiple comparisons among slopes were calculated using the Tukey test (Zar 1984). All variables were transformed where necessary to meet the assumptions of homogeneity of variance and normality.

Logging affects physical attributes of streams – I selected basins for the census on the basis of stream gradient, elevation and basin size with the intent of controlling for factors not associated with logging. Using one-way ANOVAs, I tested whether these variables differed among forest types. Once I determined that these watershed characteristics did not differ among forest types, I ran a series of nested ANOVAs to test whether physical factors associated with the creek differed among forest type (see Table 3.11 for the list of factors). Of the abiotic factors that I measured, width to depth ratio, microhabitat, and pool to riffle ratio were calculated from data taken during the census. Width to depth ratios were calculated as the bank full width divided by the bank full depth. The microhabitat index was calculated based on the relative abundance of pool, run, riffle, and cascade habitats. Habitat types were transformed into a scalar by assigning them a number: pool = 1, run = 2, riffle = 3, cascade = 4. For each creek, the microhabitat scalars were summed and divided by the total number of observations to create an index of microhabitat availability. Thus a creek with a high density of pools relative to riffles or cascades would have a low microhabitat index as compared to a creek with a low density of pools relative to riffles or cascades. Pool to Riffle ratio was calculated as the total number of pools divided by the total number of riffles per creek transect. Three variables, pool to riffle ratio, air temperature, and maximum water temperature, had only one value per creek. These variables were analyzed with a single classification ANOVA. All variables were transformed as necessary to meet the assumptions of homogeneity of variance and normality. All post-hoc tests were conducted using Tukey multiple comparisons.

Many of the abiotic variables I measured were interdependent, so I conducted a Principal Components Analysis among all creeks (S-Plus 2000 1999, Selvin 1998) (see Table 3.3 for variables used in analysis). Before conducting the analysis, the data were transformed where necessary to meet the assumptions of normality and homogeneity of variance and all variables were standardized to a mean of 0 and unit variance to account for differing scales of measure. Upon plotting principal component scores against each other, there appeared to be no relationship among scores. I ran one-way ANOVAs to test for the effects of forest type on each principal component. Principal components 2, 3 and 4 were transformed to meet the assumptions of homogeneity of variance and normality.

Assessing effects on parasite suprapopulations – My data show that logging does alter intensity, density, and prevalence of parasitism in each of the hosts. These effects could be manifest through changes in biotic and/or abiotic factors that could alter availability of hosts, physical environment, or the transmission of parasites from one host to the next.

Stage to stage relationships among parasites provide a snapshot of the transmission relationship between parasites of one stage (e.g., adult parasites) and parasites in the subsequent stage (e.g., sporocysts). For example, if the density of adult parasites (donor stage) explains a significant amount of variation in the density of the sporocysts (target stage), then teasing apart effects of environmental change on parasite dynamics will be more straightforward than if there appears to be no relationship between donor and target parasites. I ask whether target parasite density can be explained by donor-target stage relationships among parasites in response to forest type or whether the relationship between logging and parasitism is more complex. Donor and target parasite

pairs are as follows: adult parasites to sporocysts; sporocysts to metacercariae; and metacercariae to adult parasites.

To determine whether stage to stage transmission explained a significant amount of variation in parasite density, I assessed the effects of donor parasite density by forest type on target parasite density in a single classification ANCOVA model. To compare slope and intercept values among forest types, I followed Sokal and Rohlf (1995) and Zar (1984) respectively. Common slopes and tests of these slopes were calculated based on analyses in Sokal and Rohlf (1995). Post hoc Tukey multiple comparisons were calculated following Zar (1984).

Donor-target relationships among parasites could not explain the variation in target parasite density. I expanded the analyses on parasite density to include other biotic variables and abiotic variables that are affected by logging. I assessed the effects of biotic and abiotic factors on parasite density and parasite prevalence in separate analyses. Biotic variables evaluated in the expanded analysis included target host length, target host density, donor parasite density, donor parasite prevalence, donor host density, donor host length, and, in the case of adult parasites, target host diet (i.e. # stoneflies eaten). Abiotic variables evaluated in the expanded analysis included the first 4 principal components of the PCA. Since the number of possible explanatory variables was greater than the number of samples, I conducted a Leaps and Bounds analysis using Mallows Cp statistic to evaluate all possible models derived from the abiotic and biotic variables (Selvin 1998, S-Plus 2000). I tested the best-fit models in an ANCOVA model with forest type as the covariate. However, since the best fit model for most of the donor-target pairs had at least 4 explanatory variables, I was unable to divide the forests into unlogged, logged and

logged-with-debris-flow for all of the analyses due to limitations in sample size. The final ANCOVA models used lumped logged and logged-with-debris-flow creeks in the "logged" variable. I compared slope and intercept values generated by the ANCOVA between forest types using Helmert contrasts as described in Venables and Ripley (2000).

RESULTS

Parasite responses to logging – Logging had a significant effect on infrapopulation intensity of the adult and sporocyst stages of the parasite (Table 3.1, 3.4). There was a significant decrease in intensity of adult parasites in logged as compared to unlogged creeks. Logged-with-debris-flow creeks had intensities of adult parasites that were intermediate between unlogged and logged creeks (Fig. 3.3). Sporocyst infrapopulations decreased significantly following logging, but only when coupled with debris flows (Fig. 3.3).

Results for parasite density were the same as those for intensity (Table 3.5, Fig. 3.4). Parasite prevalence was significantly affected by debris flows associated with logging for sporocyst and metacercarial stages of the parasite, but not for adult parasites (Table 3.5 and Fig. 3.5).

In addition to affecting abundance and prevalence of parasitism, logging had a strong effect on the distribution of parasites in salamander and stonefly hosts, but not in snail hosts (Fig. 3.6). Adult parasites are significantly more aggregated in salamanders from logged-with-debris-flow creeks than in unlogged or logged creeks (Table 3.6). In contrast, metacercariae are significantly less aggregated in their stonefly hosts following logging-with-debris-flows.

Host responses to logging – Density and length of each host differed significantly among creeks within the same forest type (Table 3.7a,b). Even with the significant within-creek variability, host density and length still varied among forest types (Table 3.7a.b). Salamander density was significantly lower in logged creeks as compared to unlogged creeks regardless of debris flows (Fig. 3.7). Even though salamander density was depressed in logged creeks, individual salamanders were significantly longer and heavier in logged-with-debris-flow creeks (Fig. 3.8, 3.9, Table 3.7b,c).

Snail density decreased significantly following logging-with-debris-flows (Fig. 3.7). Mean snail length was significantly greater in logged as compared to unlogged creeks (Fig. 3.8). However, mean snail length in logged-with-debris-flow creeks was not significantly different from unlogged or logged creeks (Fig. 3.8).

Stonefly density and length differed significantly following logging (Table 3.7), but post-hoc tests showed the opposite effect of logging-with-debris-flows on stoneflies as compared to snails (Fig. 3.7, 3.8). Stonefly density decreased significantly following logging, but then increased significantly after logging-with-debris-flows to above pre-logging levels (Fig. 3.7). Stonefly length also increased significantly following logging, but only when coupled with debris flows (Fig. 3.8).

Since parasite transmission depends upon salamander hosts eating infected stonefly hosts, a change in salamander diet following logging could affect parasite transmission. Logging had a significant effect on salamander diet (Table 3.8). The total number of prey eaten by salamanders was significantly lower in logged creeks, but not when coupled with debris flows (Fig. 3.10). However, logging did not affect the total number of stoneflies in the salamander diet (Table 3.8, Fig. 3.10).

The increases in host length in logged creeks could alter patterns of parasitic infection. I tested whether parasite intensity was correlated with host length, and whether the correlation differed with forest type. Parasite intensity was positively correlated with host length for each host-parasite pair except for the snail-sporocyst pair in logged-with-debris-flow creeks (Table 3.9a, Fig. 3.11). Regression slopes differed significantly among forest types for each host-parasite pair (Table 3.9b). Tukey post hoc tests could not detect differences among the forest types for regression of either salamander or stonefly parasites. The regression slope for sporocyst intensity on snail length in logged-with-debris-flow creeks differed significantly from unlogged and logged creeks. This effect might be due to the low sample size of infected snails in creeks that experienced logging-with-debris-flows.

Logging effects on physical attributes of streams – My goal was to sample creeks across a series of similar basins such that the majority of among creek variation was due to logging history and not to inherent geomorphological differences among basins. I controlled for geographic location, basin area, elevation, and stream gradient. All but three creeks were located in the McKenzie River watershed. The remaining three creeks, Jones, Slick and Sturdy, were in the Little Fall Creek watershed, which is the drainage immediately south of the McKenzie Rive watershed. There were no differences in basin area, elevation or stream gradient among streams in different forest types (Table 3.10).

Among creek variation in physical variables was high as revealed in the nested ANOVA (Table 3.11). Notably, width to depth ratios were significantly different among creeks, but did not differ among forest types (Table 3.11). Since the creeks share similar slopes, elevations and basin sizes, I expected that they would share similar geometries

within forest types, but differ across forest types. I suspect that the differences in width to depth ratios derived from local variation in geologic features and streamside vegetation. The differences did not derive from among creek variation in microhabitat, which did not differ among creeks within the same forest type (Table 3.11). However, there was a significant effect of forest type on microhabitat availability. Unlogged creeks had a significantly lower microhabitat index than did either logged or logged-with-debris-flow creeks (Fig. 3.12). Since cascade and riffle habitats were arbitrarily given a higher rank than pool habitats for the statistical analyses, this reflects the relative change from a more balanced pool-riffle morphology in unlogged creeks to a more riffle/run dominated morphology in logged and logged-with-debris-flow creeks (Fig. 3.13). Pool to Riffle ratios decreased from a ratio of approximately 26% in unlogged creeks to one of 11% in logged-with-debris-flow creeks (Fig. 3.13). The increase in riffle and run habitat following logging is favorable for stonefly hosts, but not for snail or salamander hosts.

Many of the differences that occurred in abiotic factors among forest types could be attributed to changes in microhabitat availability. For example, thalweg velocity increased significantly after logging (Table 3.11, Fig. 3.12) thus reflecting the increase in riffle and run microhabitats. Creek embededness (Brusven Index) also increased significantly following logging, but only when coupled with debris flows. The only physical factor that significantly decreased following logging was canopy cover. Surprisingly, canopy cover did not differ between logged and logged-with-debris-flow creeks. The decreased canopy cover in logged creeks did correspond to a pattern of increase in mean air temperature and maximum water temperature, though neither differed significantly among forest types (Table 3.11, Fig. 3.14).

Principal components analysis among all creeks (Table 3.3) resulted in the first four principal components explaining 76% of the variation in abiotic factors among all logged and unlogged creeks (Fig. 3.15). I interpreted the significant components based on variables with loadings greater than 0.40 or less than -0.40 (McGarigal et al. 2000, Hair et al. 1987).

The first principal component (PC1) is composed of changes in canopy cover and water temperature among the creeks (Fig. 3.16). As would be expected, the two factors are inversely related such that lower canopy cover above the creek results in higher water temperatures.

The second principal component (PC2) is composed largely of the changes in width to depth ratio and microhabitat availability across creeks (Fig. 3.16). These variables are directly related. Within this framework, the loadings for the second principal component are intuitive and represent a positive relationship between riffle habitat and width and an inverse relationship between riffle habitat and depth.

The third principal component (PC3) is composed largely of the changes in pool to riffle ratio and embededness (the Brusven Index). The two variables are inversely related such that increased stream embededness is related to a decrease in the ratio of pools to riffles. This result is consistent with the results of the ANOVA in which the Brusven Index increased significantly following logging-with-debris-flows whereas the pool to riffle ratio decreased significantly.

The fourth principal component (PC4) is composed largely of changes in air temperature and water temperature. As expected, the two are directly related.

Principal components 1 and 4 varied significantly with forest (Table 3.12). Creeks that had logging coupled with debris flows had significantly higher scores for PC 1 than creeks in unlogged and logged areas (Fig. 3.17) although the general trend was towards increasing scores with intensity of logging effect. Scores for PC 4, which were strongly dependent on air and water temperature, decreased significantly following logging.

In sum, canopy cover and water temperature were the most important differences among forest type. However, both the 2nd and 3rd principal components were chiefly composed of geomorphological features that reflected the significant decrease in the relative amount of pool habitat in creeks following logging.

Assessing effects on parasite suprapopulations – Donor parasite density was not related to target parasite density for any of the parasite pairs within forest type (Table 3.13a) except for the sporocyst-metacercaria pair in logged-with-debris-flow creeks. In addition, there was no change in the slope of the relationship between donor and target parasite density across forest types ($F_{(adult - sporocyst)} = 0.383$, $Df = (2,12)$, $p > 0.5$; $F_{(sporocyst - metacercaria)} = 2.278$, $Df = (2,12)$, $p = 0.328$; $F_{(metacercaria - adult)} = 1.278$, $Df = (2,12)$, $P = 0.568$). Common regression slopes for each donor-target pair (Table 3.13) were not significant ($F_{(adult - sporocyst)} = 2.040$, $Df = (1,14)$, $p = 0.392$; $F_{(sporocyst - metacercaria)} = 1.149$, $Df = (1,14)$ $p = 0.553$; $F_{(metacercaria - adult)} = 0.006$, $Df = (1,14)$, $p = 0.737$). Thus donor parasite density by itself cannot explain levels of target parasite density.

Although regression slopes did not differ between donor-target pairs, regression intercepts did differ significantly between the adult-sporocyst, and metacercaria-adult pairs (Table 3.13b). Tukey multiple comparisons revealed a strong negative effect of

logging-with-debris-flows on the intercept for the regression between adult parasites and sporocysts (Fig. 3.18) ($q_{(0.05, 14, 3)} = 3.702$; $q_{(\text{unlogged-logged})} = 0.228$, $q_{(\text{unlogged-logged with debris})} = 4.059$, $q_{(\text{logged-logged with debris})} = 4.690$). This effect is most likely driven by the significant reduction of sporocyst density in logged-with-debris-flow creeks.

Tukey Post hoc comparisons of the regression between metacercaria and adult stages showed a significant decrease in regression intercept following logging regardless of debris flows (Fig. 3.18) ($q_{(0.05, 14, 3)} = 3.702$; $q_{(\text{unlogged-logged})} = 70.089$, $q_{(\text{unlogged-logged with debris})} = 123.909$, $q_{(\text{logged-logged with debris})} = 0.580$). This result differs from the results of the one-way ANOVA for individual metapopulations (Fig. 3.4). ANOVAs reveal a significant decline in density of adult parasites after logging and no effect of logging on metacercarial density. However, the plot of metacercarial density clearly shows a pattern of decrease in parasite density with logging for both adult and metacercarial parasites (Fig. 3.4). The regressions seem to show the combined effects of the decreased mean density of adult and metacercarial parasites following logging and logging-with-debris-flows.

Since stage to stage comparisons of parasite density could not explain significant amounts of variability in target parasite density associated with logging, I expanded the analyses to include both abiotic and biotic explanatory variables. I also included separate analyses to assess which variables best explained changes in parasite prevalence following logging.

Best fit models for density or prevalence of each parasite , with the exception of metacercarial prevalence, ranked one of three principal components as the most important variable in the regression (Table 3.14, 3.15). Variation in adult parasite density was best explained by PC3, salamander density and salamander length (Table 3.14). Adult

parasite density was positively and significantly related to salamander density and length in both logged and unlogged creeks. Regression slopes of each variable did not differ among forest types ($F_{(PC3)} = 0.288$, $df = (1,15)$, $p = 0.603$; $F_{(Salamander\ Density)} = 0.489$, $Df = (1,15)$, $p = 0.578$; $F_{(Salamander\ SVL)} = 1.864$, $Df = (1,15)$, $p = 0.202$). Although logging had a significant effect on the overall density of adult parasites (Table 3.14), there was no significant difference in regression intercepts between unlogged and logged creeks ($F_{(\alpha = 0.05, 1,15)} = 0.845$, $p = 0.197$).

Variation in prevalence of adult parasites was best explained by a model that included only PC4 and salamander length. PC4 had a significant, negative effect on adult parasite prevalence in logged creeks, but not in unlogged creeks. In addition, salamander length had a significant positive effect on prevalence of adult parasites in logged creeks, but not in unlogged creeks (Table 3.15). However, regression slopes did not differ significantly between forest types ($F_{(PC4)} = 0.638$, $Df = (1,15)$, $p = 0.440$; $F_{(SVL)} = 0.01$, $Df = (1,15)$, $p = 0.923$). In addition, regression intercepts did not differ significantly between forest types ($F_{(\alpha = 0.05, 1,15)} = 0.085$, $p = 0.776$).

Variability in sporocyst density was best explained by a regression of PC1, adult parasite density, snail density, and salamander density. In unlogged creeks, sporocyst density was significantly and inversely associated with PC1 and positively associated with salamander density (Table 3.14). In logged creeks, the effect of salamander density on sporocyst density decreased and was not significant, although PC1 continued to have a significant inverse effect on sporocysts. Although PC1 significantly affected sporocyst density in both logged and unlogged creeks, the slope of the effect differed between forest

types ($F_{(\alpha = 0.05,\ 1,15)} = 6.764$, $p = 0.032$). The slope of the relationship between salamander density and sporocyst density was also affected by logging ($F_{(\alpha = 0.05,\ 1,15)} = 5.636$, $p = 0.045$). Neither adult parasite density nor snail density had a significant effect on sporocyst density (Table 3.15), and the slope of the relationships was not affected by logging ($F_{(\text{adult parasite})} = 1.233$, $Df = (1,15)$, $p = 0.299$; $F_{(\text{snail density})} = 0.000$, $Df = (1,15)$, $p = 0.993$).

Variation in sporocyst prevalence was best explained by a regression model of PC1, salamander density, and salamander size (Table 3.15). Sporocyst prevalence in unlogged creeks was inversely related to both PC1 and salamander length. In logged creeks, sporocyst density was also inversely related to PC1, but directly related to salamander density (Table 3.15). This suggests that abiotic factors have a strong effect in both logged and unlogged creeks, but that salamander density becomes limiting to sporocyst prevalence in logged creeks. As with sporocyst density, the slope of the relationship between sporocyst prevalence and PC1 was significantly affected by logging ($F_{(\alpha = 0.05,\ 1,15)} = 8.883$, $p = 0.014$). There was a similar effect of logging on the relationship between sporocyst prevalence and salamander length ($F_{(\alpha = 0.05,\ 1,15)} = 5.167$, $p = 0.046$). The slope of the relationship between salamander density and sporocyst prevalence was not significantly different between logged and unlogged creeks ($F_{(\alpha = 0.05,\ 1,15)} = 1.793$, $p = 0.210$).

The best fit model for variation in metacercarial density included PC1, sporocyst density and stonefly density (Table 3.14). Metacercarial density in unlogged creeks increased with PC1, but not significantly (Table 3.14). In logged creeks, metacercarial

density was significantly and positively related to sporocyst density. None of the slope or intercept comparisons were significant across forest type ($F_{(PC1)} = 0.963$, Df = (1,15), p = 0.350, $F_{(\text{sporocyst density})} = 1.328$, Df = (1,15), p = 0.276, $F_{(\text{stonefly density})} = 0.600$, Df = (1,15), p = 0.456, $F_{(\text{intercept})} = 0.071$, Df = (1,15), p = 0.796).

The best fit regression for metacercarial prevalence included only sporocyst density and sporocyst prevalence (Table 3.15). Metacercarial prevalence was significantly and positively associated with sporocyst density, but only in logged creeks. There was a strong, but nonsignificant inverse relationship between sporocyst prevalence and metacercarial prevalence in logged creeks, but not in unlogged creeks. The slope of the regression was not affected by logging for either of the explanatory variables ($F_{(\text{sporocyst density})} = 0.1.713$, Df = (1,15), p = 0.215, $F_{(\text{sporocyst prevalence})} = 0.079$, Df = (1,15), p = 0.784). However, there was a significant decrease in the regression intercept following logging ($F_{(\alpha = 0.05,\ 1,15)} = 5.861$, p = 0.784).

DISCUSSION

Logging was associated with significant changes in the patterns of parasitism and features that influence them. In general, logging had a significant negative effect on parasite intensity and density in salamander and snail hosts, but not in stonefly hosts. The strength of effect varied across forest type by parasite stage. Adult parasite densities declined following logging but recovered somewhat in creeks that experienced logging-with-debris-flows. The apparent recovery was due to an inexplicably high density of adult parasites in WF NF Gate Creek. The effect of logging was reversed in the

sporocyst stage. Sporocyst intensity and density decreased significantly only when logging was coupled with debris flows. Although the effects of logging on metacercariae were not significant, the pattern of decline in metacercariae with severity of logging mirrored that of the sporocyst stage. In addition to changes in abundance, parasite prevalence in logged creeks declined in all hosts, although the decline was significant only in snail and stonefly hosts from creeks that experienced logging-with-debris-flows. Since patterns of parasite intensity and density responded similarly to logging within each parasite stage, I combine discussions of the two measures and hereafter use the term abundance to refer to both intensity and density of parasites.

Altered levels of parasite abundance and prevalence in combination with the varied effects on different parasite stages, suggest that the host-parasite system is disrupted at multiple levels by logging-induced disturbances. Environmental disturbance can disrupt complex host-parasite systems directly by altering abiotic factors that impact the free-living stages of the parasite, or indirectly by affecting host abundance or distribution (in space or time) (Sousa and Grosholz 1991, Sapp and Esch 1994, Marín et al. 1998). For example, changes in abiotic factors such as temperature and velocity can directly affect reproduction and survival of transmission stages (Anderson et al. 1982, Evans 1985, Jewsbury 1985). Higher temperatures lead to increased reproductive rate, often at the expense of survivorship of the free-living stages (Anderson et al. 1982). On the other hand, increases in stream velocity result in damage to and decreased transmission success of parasite infective stages (Upatham 1974, Jewsbury 1985). Transmission of the free-living stages can also be directly affected by changes in geomorphology and microhabitat heterogeneity (Chernin and Perlstein 1969, for review

see Sousa and Grosholz 1991). Prior studies establish that logging dramatically alters stream channel morphology, substrate composition, temperature, and scour regime (Dunne and Leopold 1978, Beschta et al. 1987, Bisson et al. 1987, Everest et al. 1987). Creeks in this study are likewise affected and make direct impacts on transmission of the free-living stages of *C. dicamptodoni* very likely.

Indirect effects of logging on parasitism are just as likely to occur as direct effects, and will be manifest through changes in host population characteristics including host density and size structure. Logging does have a strong effect on density of each host species. In high gradient streams similar to those I censused, larval salamander and nymphal stonefly densities generally increase while snail densities generally decrease after logging (Murphy and Hall 1981, Murphy et al. 1981, Hawkins et al. 1982, 1983, but Corn and Bury 1989 found decreased salamander density in logged creeks). In this study, host density also responded strongly to logging and logging-with-debris-flows but the strength and direction of the effect was dependent upon host species and severity of logging. Salamander density decreased following logging, regardless of debris flows whereas snail density did not change with logging unless it was coupled with debris flows Stoneflies were the most sensitive to logging disturbance. Their population density decreased in logged creeks but rebounded above unlogged levels in creeks affected by logging-with-debris-flows. This increased abundance of stoneflies following debris flows is most likely due to the decrease in canopy cover and subsequent increase in light penetration to the stream. Increased sunlight penetration in streams leads to increased algal production (e.g., Gregory 1980, Hill and Harvey 1990), which translates into more herbivorous prey for the predaceous stoneflies (e.g., Power 1992, Hill et al. 1995).

Epidemiological models predict that parasite densities are strongly dependent upon densities of parasite transmission stages and susceptible (target) hosts (Crofton 1971, May and Anderson 1979, Anderson and May 1992). The response of parasite suprapopulations to host density changes will depend on the level of sensitivity of each parasite stage to the donor and target hosts (Marín et al. 1998). The sensitivity of each parasite stage to a particular host will depend upon the longevity and density of the host. Based on host life spans, densities and parasite prevalence, I make a series of predictions in the following section about how parasite stages will respond to changes in density of each host species (see May and Anderson 1979).

I predict that 1) parasite recruitment to snails will be more affected by the availability of parasite eggs in the environment than by the density of snail hosts, 2) parasite recruitment to stoneflies will be more affected by the density of stoneflies than by the availability of cercariae in the environment, and 3) parasite recruitment to salamanders will be dependent on the proportion of stoneflies infected and the density of salamanders. For the predictions of parasite recruitment into snails and stoneflies, I assume that the availability of eggs and cercariae are a function of the density of adult parasites and sporocysts, respectively.

Prediction 1) Parasite recruitment will depend upon egg availability and not snail density – Snails live for 3 to 6 years, have low parasite prevalence (roughly 5% in unlogged creeks), and are very abundant, which suggests that an increase, or decrease, in snail density associated with disturbance would not affect the density of parasites that recruit into the host. This does not suggest that extremely low densities of snails, such as those found in certain logged-with-debris-flow creeks, would not limit recruitment of

parasites into snails. In fact, mass action models predict that successful parasite transmission occurs only above a threshold host density (e.g., Anderson and May 1978). Thus parasite recruitment into snails might be limited by snail density in the logged-with-debris-flow creeks that have very low snail densities. However, in logged and unlogged creeks, the range in snail density is unlikely to affect recruitment of eggs into the snail host. Instead, recruitment is more likely to be driven by availability of eggs in the environment, which is related to the density of salamanders and adult parasites. Studies of similar snail-trematode associations in ponds (Fernández and Esch 1991) and salt marshes (Sousa 1993) also suggest that parasite transmission into snails is limited by availability of recruits, and not by snail density. Thus transmission of this stage is probably best modeled as density dependent upon egg availability. If this density-dependent model is correct, then my results should show a strong effect of adult parasite or salamander density on sporocyst density.

Prediction 2) Parasite recruitment to stoneflies will be affected by stonefly density and not the availability of cercariae – Unlike changes in snail host density, changes in stonefly density in response to environmental disturbances are likely to affect dynamics of *C. dicamptodoni*. Stoneflies live for only 2 years, have high prevalence of infection (roughly 92% in unlogged creeks), and are an order of magnitude less abundant than snails. The high prevalence in stoneflies suggests that increased stonefly density would benefit cercarial transmission by opening up more habitats (i.e. hosts) into which cercariae could recruit. The short lifespan of the stonefly, relative to snails and salamanders, indicates that parasite dynamics associated with the stonefly are on a shorter time scale than in either the snail or salamander host. This shorter temporal scale could

translate into more variable transmission into, or out of, the stonefly relative to the snail or salamander hosts. This suggests that transmission of *C. dicamptodoni* is limited by cercarial recruitment into the stonefly host and subsequent transmission of parasites from the stonefly to the salamander hosts. As with transmission of eggs, transmission of cercariae is likely to be best modeled by a density-dependent transmission function. In the case of the cercariae, the function will depend on density of stoneflies.

Prediction 3) Parasite recruitment to salamanders will be dependent on the proportion of stoneflies infected and the density of salamanders -- Predicting how changes in salamander density will affect parasite dynamics is not straightforward since salamanders acquire infections by eating infected stoneflies. Thus transmission of metacercariae will depend upon the salamander-stonefly functional response curve. May and Anderson (1979) suggest that transmission completed through a trophic link is extremely efficient and that the threshold density of predators (salamanders) required to maintain the adult parasite population is low (May and Anderson 1979). So even at extremely low salamander densities metacercarial transmission should be successful. However, the rate of transmission will vary with the predator-prey functional response, which will depend upon the density of salamanders and/or stoneflies. Pacific giant salamander larvae prey disproportionately on mayflies and large predaceous insects, including stoneflies (Parker 1994). The proportion of stoneflies relative to the other prey items preferred by salamanders is not constant across forest type (M. Poteet, unpub. data). Thus the functional response curve is likely to be non-linear with disturbance, either Type II or Type III, which would lead to non-linear parasite transmission and not the linear transmission function assumed by mass-action models (e.g., Crofton 1971, Anderson and

May 1978). In addition, since the salamander must eat an infected stonefly to become infected, then the rate of transmission will not only be affected by the functional response of the predator, but also by the proportion of stoneflies that are infected. This suggests that a transmission term that is dependent upon prevalence of infection in stoneflies would provide a better description of metacercarial transmission than would mass-action transmission. This is reminiscent of frequency-dependent models in which infection of the vector is dependent upon the frequency of infected non-vector hosts (e.g., Getz and Pickering 1983, Thrall et al. 1995, McCallum et al. 2001). One interesting outcome of frequency-dependent transmission is the lack of a host threshold, since transmission depends upon proportional infection. By this model, even if stonefly and salamander densities are extremely low, parasites should still be able to establish in salamanders. In either transmission model, decreased salamander density should result in decreased metacercarial transmission.

The difficulty in predicting the response of metacercarial transmission to salamander density is also exacerbated by the fact that salamander longevity and level of infection are intermediate between snail and stonefly hosts. Salamanders are resident in streams for 3 to 4 years, have intermediate levels of infection (82% prevalence in unlogged creeks), and are two orders of magnitude less abundant than stoneflies. Most likely, metacercarial transmission to salamanders will be sensitive to the relative densities of salamander and stonefly hosts and the proportion of infected stoneflies.

Abiotic and biotic controls on parasite populations – Thus far, I have considered direct (abiotic) and indirect (biotic) effects of logging on parasitism. But each plays a role in

the dynamics of this parasite-host system, and the relative roles of each factor often switch depending on the level of disturbance.

Adult parasites were significantly and positively correlated with salamander density and size in both logged and unlogged creeks. Salamander density decreased in logged creeks while salamander mean size increased. Since adult parasite abundance is lower in logged than in unlogged creeks, it appears that the negative effect of decreased salamander density on adult parasites in logged creeks is stronger than the positive effect of increased salamander size. The abiotic factor, PC3, selected by the best fit model did not have a statistically significant effect on adult parasite density in either logged or unlogged creeks. However PC3 did have a stronger positive effect in logged as compared to unlogged creeks. PC3 is a composite of pool:riffle ratio inversely linked with embededness. Adult parasites in logged creeks as compared to unlogged creeks are more positively correlated with P:R ratio and inversely correlated with embededness. This most likely has more to do with habitat availability for stonefly and salamander hosts than it does with parasite transmission, especially since transmission is via a trophic link and not a free-living stage. Salamanders and stoneflies inhabit interstitial spaces of the creek bed and populations of each respond negatively to embededness (Murphy et al. 1981, Parker 1992, Welsh 1993).

Interestingly, adult parasite density was not correlated with metacercarial prevalence in stoneflies, even though the prevalence declined significantly in logged and logged-with-debris-flow creeks as compared with unlogged creeks. If the frequency-dependent transmission model is correct, then the decline in metacercarial prevalence coupled with the decline in salamander density should lead to a significant decline in

adult parasite density. Following logging-with-debris-flows, even though metacercarial prevalence is lower, stoneflies are in general more abundant and are infected with similar levels of parasites as stoneflies in logged and unlogged creeks. Indeed, density of infected stoneflies is on average higher in logged-with-debris-flow creeks than in unlogged or logged creeks (Table 3.1). According to mass-action models, the increased availability of infective stages should lead to increased transmission of the parasite via the trophic link between stonefly and salamander hosts. Even though stonefly density increased following logging-with-debris-flows, stoneflies are not more abundant in the diet of salamanders from those creeks as compared to logged or unlogged creeks The prediction that adult parasite abundance is strongly dependent on density of the infective stage is incorrect due to the non-linear trophic response of salamanders to stonefly density. Thus the majority of variance in adult parasite density was explained by changes in salamander density and size structure. The relative strengths of these indirect variables were similar across forest types.

As with parasite abundance, salamander size has a significant positive effect on prevalence of adult parasites. Although adult parasite prevalence does not statistically decrease in logged as compared to unlogged creeks, it does decrease in logged-with-debris-flow creeks by 30% from unlogged creeks. This decrease is significantly correlated with PC4, which is principally composed of air and water temperature. As temperatures increase, adult parasite prevalence decreases. Whatever the mechanism for the negative effect of temperature on prevalence, it is ameliorated by a significant positive effect of salamander size.

Unlike adult parasite density, sporocyst density did behave according to the predictions based on host life span and parasite prevalence; but only in unlogged creeks. As predicted, sporocyst density was strongly correlated with the donor stage of the parasite. Since I could not directly measure the density of parasite eggs in the environment, I used adult parasite and salamander host density as proxies for availability of eggs. Sporocysts are not correlated with adult parasite densities, although they are positively correlated with salamander densities. Thus, there is a stronger effect of the donor stage than that of the target host density on sporocyst density in unlogged creeks. In addition to this strong biotic component, sporocyst density in unlogged creeks was significantly and negatively correlated with PC1, an abiotic component representing canopy cover and water temperature. Breaking the variable into its components, sporocyst density was positively correlated with canopy cover and negatively correlated with temperature in both logged and unlogged creeks. This effect is somewhat surprising since increased temperature is often associated with greater reproduction of the donor stage. It is possible that lower survival of transmission stages in warmer temperatures plays a role in the inverse relationship between sporocyst density and temperature (Anderson et al. 1982). However, the significant inverse correlation with PC1 could be indicative of other factors associated with canopy cover and logging, including changes in habitat complexity and embededness. This aspect of the life cycle needs more study to understand the mechanisms behind these patterns.

In logged creeks, the relative effects of abiotic and biotic factors switch. Variation in sporocyst density becomes decoupled from donor density and is largely explained by the abiotic variable, PC1. The relative effect of snail density on sporocysts

also increases in logged creeks. This effect is driven by the extreme declines in host and parasite populations in creeks that experienced logging-with-debris-flows (Fig. 3.19, Table 3.1). Snail density in logged-with-debris-flow creeks was on average half that of populations from unlogged and logged creeks. In two creeks, South Fork Gate (SFG) and North Fork of the North Fork Gate (NFNFG), snail densities dropped to 21 and 0 per m^2 respectively. Snail densities in SFG declined from an average of 73 per m^2 in 1995 to 0 per m^2 in the two years immediately following the 1996 debris flow (M. Poteet, unpub. data). Although NFNFG was not sampled prior to the 1996 debris flow, snails must have been present because salamander larvae are infected with the parasite. In addition other creeks in the Gate Creek watershed that were geomorphologically and biotically similar to NFNFG do support these snails. Recolonization by snails of SFG was probably quicker than in NFNFG due to migration from a source population in Hagan Creek, which is confluent with SFG just downstream of the censused transect.

Snail density, which in unlogged creeks does not control levels of sporocyst density, appears to limit sporocysts in logged-with-debris-flow creeks. In SFG, where snail density recovered to one third of its pre-debris flow density, no eggs had successfully transmitted to the snails that I collected. The other two creeks, East Fork North Fork Gate (EFNFG) and West Fork North Fork Gate (WFNFG), that experienced logging-with-debris-flows and supported snails had higher snail densities than 8 of the logged and unlogged creeks.. Even so, sporocyst density and prevalence were lower in all of the logged-with-debris-flow creeks as compared to all creeks in logged and unlogged watersheds (Table 3.1). This pattern of infection in snails suggests that the 1996 debris flows in EFNFG and WFNFG caused a dramatic reduction in snails similar

to that in NFNFG and SFG. but that recovery of these populations was more rapid. This also suggests that recovery of sporocyst populations was lagging behind that of the snail populations. In combination with the ANCOVA results, it appears that sporocyst density switches from a donor controlled system with abiotic inputs in unlogged creeks to an abiotically driven system that is more dependent upon target host density in logged-with-debris-flow creeks.

The abiotic and biotic factors that control metacercarial density also switched between forest types. There was a non-significant pattern of decline in metacercarial density as severity of logging disturbance increased. This did not reflect patterns in stonefly densities, which declined in logged creeks but rebounded to above pre-logging levels in logged-with-debris-flow creeks. Thus metacercarial density did not correlate with stonefly density as was predicted. Instead, in unlogged creeks metacercarial density was most affected by canopy cover and stream temperature (PC1). Unlike sporocysts, metacercariae were positively correlated with stream temperatures, suggesting that increasing temperatures enhanced cercarial transmission. If this relationship maintained in logged creeks, then a stronger positive response of metacercarial density to PC1 would be expected due to the decreased canopy cover and increased water temperature following logging. Instead, variation of metacercarial density in logged creeks is best explained by sporocyst density regardless of abiotic factors. Thus the controls on metacercarial density appear to switch from abiotic to biotic factors with logging-induced disturbance.

A comparison of the relationships between sporocyst and metacercarial densities within each forest type (Table 3.13) shows that this relationship is only significant following logging-with-debris-flows. This result suggests that the switch in control over

metacercarial density from abiotic to biotic factors occurs in logged-with-debris-flow creeks and not in logged creeks. Since sporocyst density in logged-with-debris-flow creeks is controlled by snail density, then it appears that the effect of sporocysts on metacercarial density in logged-with-debris-flow creeks is driven by the large reduction in snail density that occurs after debris flows (Fig. 3.20). Snails, which in unlogged creeks appear not to limit parasite populations, become limiting to both sporocyst and metacercarial stages in creeks that experienced severe disturbance.

In addition to limiting metacercarial densities, sporocyst density appears to limit metacercarial prevalence, but only in logged creeks. Metacercarial prevalence is reduced in stoneflies following logging, but only when coupled with debris flows. Parasite aggregations in stoneflies also decreased in logged-with-debris-flow creeks. The combined decline in metacercarial prevalence and aggregation in logged-with-debris-flow creeks is the result of low to no parasite recruitment from snails. By 1998, all stoneflies that survived the 1996 debris flows would have died or metamorphosed so that any stoneflies collected in the 1998 census must have been infected between late 1996 and the time of the census. As mentioned before, patterns of infection and recovery in snails and sporocysts suggest that the debris flows in 1996 caused a marked reduction if not local extinction of snails from debris flow creeks. Snail densities in some creeks recovered more quickly than in others while stonefly densities remained relatively constant. This provides an excellent opportunity to assess the relationships and recovery between snail-sporocyst and stonefly-metacercaria pairs. In those creeks where snail densities remain low and uninfected with sporocysts, metacercarial prevalence and density are low or nonexistent. Interestingly, in SFG where no censused snails were infected, there was a

low level of infection in stoneflies. This suggests that some of the snails in SFG must be infected, but at such a low level that I was unable to detect them in my samples. This, along with the fact that in logged creeks sporocyst prevalence and density affect levels of the metacercarial stage, provides good evidence that frequency-dependent transmission models are appropriate for understanding dynamics of the parasite between snail and stonefly hosts in logged-with-debris-flow creeks.

In logged-with-debris-flow creeks where snail densities have recovered and sporocyst densities are recovering, metacercarial prevalence and density have reached pre-logging levels (Fig. 3.20). In logged-with-debris-flow creeks, there is a clear trajectory of recovery in metacercarial density and prevalence that is based on recovery of the snail-sporocyst relationship. In unlogged creeks, there is no correspondence between sporocyst or snail densities and levels of the metacercarial stage (Fig. 3.19, Table 3.14, 3.15).

CONCLUSION

Pressures of land-use change and resource use by humans increasingly force species to cope with fragmented and/or degraded habitats. With these changes, has come an awareness of potential for changing dynamics of disease and parasitism that might affect these species (e.g., Dobson and Carper 1992). Although several reviews report that diseases or parasites of wildlife do respond to anthropogenic disturbances (Lafferty 1997, Daszak et al. 2000), most of the studies cited are either anecdotal or poorly studied due to restraints on time or replication (but see Burkholder and Glasgow 1997 for an exception).

In this study, I have the opportunity to study the effects of anthropogenic disturbance on a parasite-host system that has replicate impact and control sites.

The data reveal a clear and significant effect of logging on parasite population dynamics. Each stage in the life cycle of the parasite is decreased by disturbances associated with logging, but the strength of the effect is dependent upon severity of logging. In addition, the relative roles of abiotic and biotic variables in the parasite-host system are context dependent. Control over sporocyst densities switched from a combination of abiotic and donor stage factors in unlogged creeks to that of target stage and some abiotic input in logged creeks. Control over metacercarial abundance switched from abiotic factors in unlogged creeks to donor stage factors in logged creeks. Although adult parasite abundance did decrease, the relative effects of abiotic and biotic factors on this parasite stage were similar in logged and unlogged creeks. The switch from abiotic to biotic control in sporocyst and metacercarial stages both derive from the sharp decline in snail densities following logging-with-debris-flows that were associated with logging.

Environmental disturbances can thus not only affect the level of parasitism, but also the processes driving host-parasite dynamics. Understanding the implications of anthropogenically altered parasite dynamics will be important for minimizing effects of environmental disturbances on wildlife populations.

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Table 3.1 Mean densities and prevalences of hosts and parasite stages by creek. All creek-level data are back-transformed. All forest-level data are raw, untransformed data. "Log with Debris" are creeks that experienced debris flows following logging. See "Census Methods" in text for details of sampling protocols.

Creek	Forest Type	Salamander Density # / m^2	Snail Density # / m^2	Stonefly Density # / m^2	Adult Density # / m^2	Sporocyst Density # / m^2	Metacercaria Density # / m^2	Adult Prevalence (%)	Sporocyst Prevalence (%)	Metacercaria Prevalence (%)
Cone	Unlogged	0.09	78.40	12.27	0.73	1259.55	38.69	91.18	8.01	68.75
Elk	Unlogged	0.03	96.00	4.27	0.31	607.49	22.87	100.00	6.98	86.18
Hagan	Unlogged	0.11	58.13	22.40	0.20	177.43	456.17	58.70	2.61	97.35
Jones	Unlogged	0.16	160.00	23.47	0.24	3567.38	493.03	70.00	14.43	98.76
Mona	Unlogged	0.14	334.93	10.67	0.47	304.15	320.53	72.73	1.93	100.00
Simmonds	Unlogged	0.05	194.67	5.87	0.26	487.74	33.79	82.35	3.27	93.22
Slick	Unlogged	0.02	83.73	10.67	0.10	45.81	22.81	80.00	2.17	80.08
EF Deer	Logged	0.06	214.40	6.93	0.03	139.09	129.66	42.11	1.63	92.93
Finn	Logged	0.02	116.27	3.20	0.03	221.34	31.20	70.00	1.48	100.00
Holden	Logged	0.01	186.67	10.67	0.02	171.25	89.50	100.00	2.23	72.35
Osborne	Logged	0.10	60.27	10.13	0.19	720.10	68.79	75.00	8.01	93.04
Ritchie	Logged	0.07	44.80	3.73	0.26	411.16	0.76	71.43	8.25	43.38
Sturdy	Logged	0.01	203.73	7.47	0.00	394.54	50.54	0.00	4.30	87.48
WF Deer	Logged	0.05	307.73	8.53	0.03	112.50	78.39	39.13	1.83	92.27
EF NF Gate	Log with Debris	0.07	166.40	19.73	0.06	28.91	140.17	52.63	1.29	92.20
NF NF Gate	Log with Debris	0.03	0.00	27.20	0.00	0.00	0.00	27.27	0.00	0.00
SF Gate	Log with Debris	0.02	21.33	13.33	0.02	0.00	0.12	45.45	0.00	17.64
WF WF Gate	Log with Debris	0.05	164.27	27.73	0.48	11.53	16.18	82.35	0.90	64.63

Forest Type	Salamander Density # / m^2	Snail Density # / m^2	Stonefly Density # / m^2	Adult Density # / m^2	Sporocyst Density # / m^2	Metacercaria Density # / m^2	Adult Prevalence (%)	Sporocyst Prevalence (%)	Metacercaria Prevalence (%)
Unlogged	0.07	143.70	12.80	0.33	921.37	198.27	82.45	4.98	92.25
Logged	0.03	161.98	7.24	0.08	310.00	64.12	57.12	3.53	86.97
Log with Debris	0.04	88.00	22.00	0.14	10.11	39.12	52.42	0.27	37.96

Table 3.2 Physical description of creeks censused between June and August 1998. See "Census Methods" in text for details of sampling protocols. SF = South Fork. WF = West Fork. NF = North Fork. EF = East Fork.

Creek	Date Censused	Locality (T. R. sec.)	Ownership	Forest status*	% Canopy cover+	Basin Area (km^2)	Gradient (%)	Elevation (m)	Bank Full Width (m)
Cone	14-Jun-98	16S 4E sec. 19	USFS	mt	88.62	5.34	7.4	375	3.36
Elk	18-Jun-98	16S 4E sec. 19	USFS	mt	94.94	9.3	4.2	350	5.19
Jones	22-Jun-98	18S 2E sec. 25	USFS	mt	85.57	4.79	3	350	3.02
Slick	24-Jun-98	18S 2E sec. 27	USFS	mt	76.86	9.08	3.3	350	4.42
SF Gate	26-Jun-98	16S 3E sec. 15	USFS/Guistina	cc,df	75.43	17.6	1.7	425	7.08
Holden	29-Jun-98	17S 1E sec. 8	Weyerhaeuser	cc	90.12	7.29	3.6	250	4.95
Osborne	02-Jul-98	17S 1W sec. 34	Weyerhaeuser	cc	72.47	4.6	6.7	300	2.84
Ritchie	05-Jul-98	17S 1E sec. 14	Weyerhaeuser	cc	78.19	5.34	11.3	300	4.26
Finn	07-Jul-98	16S 2E sec. 19	Weyerhaeuser	cc	89.34	8.15	11.3	325	5.45
WF NF Gate	09-Jul-98	16S 3E sec. 7	Weyerhaeuser	cc,df	25.73	6.57	5.5	425	4.55
Hagan	11-Jul-98	16S 3E sec. 15	USFS	mt	57.62	13.9	2.6	425	6.05
EF NF Gate	14-Jul-98	16S 3E sec. 7	Weyerhaeuser	cc,df	45.92	12.98	2.4	450	4.79
Simmonds	17-Jul-98	16S 4E sec. 21	USFS	mt	87.68	12.3	3.8	375	5.36
WF Deer	20-Jul-98	17S 3E sec. 14	Weyerhaeuser	cc	75.79	10.16	2.6	360	5.77
EF Deer	22-Jul-98	17S 3E sec. 14	Weyerhaeuser	cc	70.13	12.73	2.2	360	4.60
NF NF Gate	24-Jul-98	16S 2E sec. 11	Weyerhaeuser	cc,df	25.44	16.24	2.6	475	5.03
Mona	27-Jul-98	15S 4E sec. 36	USFS	mt	85.96	3.55	6.8	425	2.39
Sturdy	30-Jul-98	18S 1E sec. 2	Weyerhaeuser	cc	86.51	6.42	6.9	480	4.05

* cc = clear cut; mt = mature; df = debris flow. + canopy cover calculated using spherical densiometer.

Table 3.3 Variables used in the principal components analysis.

PCA among creeks
Air Temperature
Water Temperature (Max)
Microhabitat
Canopy Cover
Basin Area
Gradient
Elevation
Pool to Riffle Ratio
Width to Depth Ratio
Brusven Index
Pebble Count

Table 3.4 Analysis of Variance for parasite intensity (mean # of parasites per infected host) by forest type for each parasite stage. Intensity data are means per creek. Forest types are unlogged, logged and logged with debris flows.

Parasite Stage	Df	SS	MS	F	p
Adult Parasite					
Forest	2	0.376	0.188	3.465	0.058
Residuals	15	0.815	0.054		
Sporocyst					
Forest	2	3.384	1.692	4.339	0.034
Residuals	14	5.459	0.390		
Metacercariae					
Forest	2	0.249	0.125	1.362	0.286
Residuals	15	1.374	0.092		

Table 3.5 Analysis of variance results for (a) parasite density and (b) prevalence by forest type. Parasite density is the per creek mean number of parasites per m^2 of creek. Parasite prevalence is the mean proportion of hosts infected per creek. Forest types are unlogged, logged and logged with debris flows.

(a) Density		Df	SS	MS	F	p
adult parasite						
	Forest	2	0.399	0.199	4.954	0.022
	Residuals	15	0.603	0.040		
sporocyst						
	Forest	2	126.853	63.426	8.823	0.003
	Residuals	15	107.830	7.189		
metacercaria						
	Forest	2	21.762	10.881	2.635	0.104
	Residuals	15	61.929	4.129		

(b) Prevalence		Df	SS	MS	F	p
adult parasite						
	Forest	2	0.388	0.194	1.558	0.143
	Residuals	15	1.864	0.124		
sporocyst						
	Forest	2	0.051	0.026	3.875	0.045
	Residuals	14	0.092	0.006		
metacercaria						
	Forest	2	1.078	0.540	4.900	0.023
	Residuals	15	1.650	0.110		

Table 3.6 Aggregation of each parasite stage by forest type. Means are of parasite intensity (# per infected host). V:M is the variance to mean ratio and is used as a measure of aggregation. When V:M > 1, parasite distributions are aggregated. N is the total number of hosts used for the analysis. Comparisons among forest types are calculated following Pielou (1969) with the critical value w, and the confidence limits (CL). An * is placed beside aggregations that are significantly different.

Parasite Stage	Mean	V:M	N
Adult Parasite			
Unlogged	5.34	6.58	130
Logged	2.40	6.89	101
Logged w/ Debris Flow	4.77	18.95	58
Sporocyst			
Unlogged	72.19	1243.81	1222
Logged	42.04	1319.07	1577
Logged w/ Debris Flow	13.50	1193.75	421
Metacercaria			
Unlogged	16.98	38.19	164
Logged	9.16	32.87	104
Logged w/ Debris Flow	2.33	9.99	122

Comparisons	w	CL	
Adult Parasite			
Unlogged : Logged	0.0224	+/- 0.2336	
Unlogged : Logged w/ Debris Flow	0.5284	+/- 0.2592	*
Logged : Logged w/ Debris Flow	0.5060	+/- 0.2822	*
Sporocyst			
Unlogged : Logged	0.0294	+/- 0.0668	
Unlogged : Logged w/ Debris Flow	-0.0205	+/- 0.0873	
Logged : Logged w/ Debris Flow	-0.0499	+/- 0.0791	
Metacercaria			
Unlogged : Logged	-0.0751	+/- 0.2168	
Unlogged : Logged w/ Debris Flow	-0.6710	+/- 0.2098	*
Logged : Logged w/ Debris Flow	-0.5960	+/- 0.2362	*

Table 3.7. Effects of forest type on a) host density (#/m^2), b) host length (mm), and c) salamander mass (gms). Analyses are ANOVAs of creek nested within forest type. N(unlogged) = 7, N(logged) = 7, N(logged w/ debris flows) = 4.

(a) Host Density	Df	SS	MS	F	p
Salamander					
Forest	2	51.75	25.87	55.900	0.000
Creek in Forest	15	268.31	17.89	38.648	0.000
Residuals	522	241.60	0.46		
Snail					
Forest	2	427480.00	213740.00	6.385	0.002
Creek in Forest	15	4006597.00	267106.50	7.979	0.000
Residuals	522	17474031.00	33475.20		
Stonefly					
Forest	2	16645.50	8322.76	19.965	0.000
Creek in Forest	15	15807.70	1053.85	2.528	0.001
Residuals	522	217600.00	416.86		

(b) Host Length	Df	SS	MS	F	p
Salamander					
Forest	2	35046.50	17523.26	25.419	0.000
Creek in Forest	15	78560.70	5237.38	7.597	0.000
Residuals	617	425349.10	689.38		
Snail					
Forest	2	110.43	55.21	5.035	0.007
Creek in Forest	14	741.72	52.98	4.831	0.000
Residuals	399	4375.69	10.97		
Stonefly					
Forest	2	312.39	156.19	18.001	0.000
Creek in Forest	15	500.78	33.39	3.847	0.000
Residuals	188	1631.31	8.68		

(c) Salamander Mass	Df	SS	MS	F	p
Salamander					
Forest	2	47.11	23.56	27.740	0.000
Creek in Forest	15	98.34	6.56	7.721	0.000
Residuals	607	515.42	0.85		

Table 3.8 Analysis of variance for salamander diet by forest type. Diet is analyzed for *Calineuria californica* and for the total number of prey found in individual salamander guts. Each measure of diet was averaged across all salamanders within each creek. Forest types are unlogged, logged and logged with debris flows.

Diet	Df	SS	MS	F	p
Calineuria					
Forest	2	0.806	0.403	2.646	0.104
Residuals	15	2.284	0.152		
All Prey					
Forest	2	4.594	2.297	6.174	0.011
Residuals	15	5.581	0.372		

Table 3.9 Analysis of covariance results to assess whether the relationship between parasite stage and host length varies by forest type. (a) Regression coefficients and significance for regressions by forest type. The first three coefficients of each regression are the intercepts while the last three are the regrsession slopes. (b) Analysis of differences among regression slopes by forest type (Zar 1984).

(a) Regressions by forest type	Coefficients	Std Err	t value	p
Adult Parasite				
Unlogged	0.054	0.149	0.360	0.719
Logged	-0.017	0.216	-0.079	0.937
Logged w/ Debris flow	-0.234	0.356	-0.656	0.512
SVL (Unlogged)	0.008	0.002	4.372	0.000
SVL (Logged)	0.006	0.002	2.246	0.026
SVL (Log w/ Debris)	0.009	0.004	2.616	0.010
Sporocyst				
Unlogged	2.356	0.091	25.856	0.000
Logged	2.314	0.113	20.504	0.000
Logged w/ Debris flow	2.348	0.288	8.143	0.000
Shell Length (Unlogged)	0.047	0.006	7.309	0.000
Shell Length (Logged)	0.047	0.008	5.978	0.000
Shell Length (Log w/ Debris)	0.028	0.018	1.589	0.114
Metacercaria				
Unlogged	-0.116	0.138	-0.842	0.401
Logged	-0.184	0.205	-0.896	0.371
Logged w/ Debris flow	-0.496	0.319	-1.556	0.121
Fly Length (Unlogged)	0.090	0.011	8.012	0.000
Fly Length (Logged)	0.080	0.017	4.710	0.000
Fly Length (Debris)	0.068	0.021	3.244	0.001

(b) Slope comparisons	Df	SS	MS	F	p
Adult					
Forest	2	-68561.508	-34280.754	-89.964	0.000
Residuals	180	68589.040	381.050		
Sporocyst					
Forest	2	-994.832	-497.416	-69.593	0.000
Residuals	140	1000.656	7.148		
Metacercaria					
Forest	2	-2055.560	-1027.780	-124.192	0.000
Residuals	255	2110.316	8.276		

Table 3.10 Creeks in this census were selected based on basin area (km2) above the sample transect, elevation (m), and stream gradient to control for creek features that do not vary with logging history. This table present ANOVA results that test whether the control variables differed among forest types.

	Df	SS	MS	F	p
Basin Area					
Forest	2	10.496	5.248	0.271	0.766
Residuals	15	290.174	19.345		
Elevation					
Forest	2	12864.680	6432.341	1.743	0.208
Residuals	15	55346.430	3689.762		
Gradient					
Forest	2	6.930E-05	3.470E-05	0.052	0.949
Residuals	15	9.990E-03	6.660E-04		

Table 3.11 Analyses of abiotic variables at the creek or basin level by forest type. creek level variables are those that describe within-creek variability. Basin level variables, such as pool:riffle ratio, are those that describe variability across creeks. Analyses for creek level variables are nested ANOVAs with creek nested in forest type. Basin level variables are analyzed with one-way ANOVAs.

	Df	SS	MS	F	p
Width to Depth ratio					
Forest	2	31.839	15.919	0.822	0.441
Creek in Forest	15	1940.659	129.377	6.679	0.000
Residuals	257	4978.224	19.370		
Microhabitat					
Forest	2	13.027	6.514	8.717	0.000
Creek in Forest	15	16.513	1.101	1.473	0.110
Residuals	520	388.551	0.747		
% Canopy Cover					
Forest	2	8746.240	4373.118	20.770	0.000
Creek in Forest	15	52047.190	3469.813	16.480	0.000
Residuals	127	26740.350	210.554		
Embededness (Brusven Index)					
Forest	2	43.208	21.604	5.391	0.005
Creek in Forest	15	120.761	8.051	2.009	0.013
Residuals	519	2079.852	4.007		
Thalweg Velocity					
Forest	2	0.888	0.444	28.276	0.000
Creek in Forest	15	1.087	0.072	4.616	0.000
Residuals	522	8.196	0.016		
Pebble Count					
Forest	2	1.048	0.524	0.417	0.659
Creek in Forest	15	201.337	13.442	10.682	0.000
Residuals	522	655.869	1.256		
Pool to Riffle ratio					
Forest	2	0.106	0.053	4.884	0.023
Residuals	15	0.164	0.011		
Air Temperature					
Forest	2	157.432	78.716	2.667	0.102
Residuals	15	442.679	29.512		
Water Temperature (max.)					
Forest	2	5.686	2.843	0.648	0.537
Residuals	15	65.759	4.384		

Table 3.12 ANOVA results of principal component scores by forest types. Forest types are unlogged, logged and logged with debris flow.

Principal Component	Df	SS	MS	F	p
PC1					
Forest	2	33.248	16.624	10.341	0.002
Residuals	15	24.115	1.608		
PC2					
Forest	2	2.207	1.014	0.375	0.69
Residuals	15	44.173	2.945		
PC3					
Forest	2	8.974	4.487	3.36	0.06
Residuals	15	20.034	1.336		
PC4					
Forest	2	7.182	3.591	5.288	0.02
Residuals	15	10.186	0.679		

Table 3.13 (a) Analysis of covariance to test whether forest type alters the relationship between donor parasite density and target parasite density. Regression slopes did not vary by forest type so common regression slopes, b_c, were calculated, but were not significant (see text). (b) Differences in regression intercepts by forest type were evaluated following Zar (1984).

(a) Regression by forest type	Slope	Intercept	Std Err	t value	p	b_c
Adult → Sporocyst						
Unlogged		1.113	5.316	0.209	0.838	
Logged		5.499	20.52	2.679	0.020	
Logged + Debris		-0.178	2.733	-0.065	0.949	
Adult (Unlogged)	10.863		7.846	1.385	0.191	4.766
Adult (Logged)	2.873		5.219	0.550	0.592	
Adult (Debris)	3.745		5.882	0.637	0.536	
Sporocyst → Metacercaria						
Unlogged		3.366	1.803	1.867	0.086	
Logged		6.051	3.38	1.790	0.099	
Logged + Debris		0.132	1.3	0.102	0.921	
Spor (Unlogged)	0.191		0.199	0.961	0.356	0.209
Spor (Logged)	-0.368		0.510	-0.721	0.485	
Spor (Debris)	1.443		0.682	2.114	0.056	
Metacercaria → Adult						
Unlogged		0.674	0.189	3.564	0.004	
Logged		0.628	0.238	2.643	0.022	
Logged + Debris		0.305	0.144	2.115	0.056	
Meta (Unlogged)	-0.002		0.035	-0.053	0.958	-0.002
Meta (Logged)	-0.078		0.061	-1.279	0.225	
Meta (Debris)	0.048		0.05	0.961	0.356	

(b) Intercept comparisons	Df	SS	MS	F	p
Adult → Sporocyst					
Forest	2	360.276	180.138	26.795	0.000
Residuals	14	94.118	6.723		
Sporocyst → Metacercaria					
Forest	2	6.062	3.031	0.741	0.697
Residuals	14	57.231	4.088		
Metacercaria → Adult					
Forest	2	0.774	0.387	8.985	0.006
Residuals	14	0.603	0.043		

Table 3.14 Results of ANCOVA to assess the relationship of biotic and abiotic variables with parasite density by forest type. Forest types are unlogged and logged. The first two coefficients listed for each parasite stage are regression intercepts. The following coefficients are regression slopes associated with either no logging (Unlog) or logging with or without debris flows (Log). PC = principal component; #/m^2 denotes host or parasite density, SVL = salamander snout vent length (mm).

Parasite Stage	Coefficients	SE	t	p
Adult parasite				
Unlogged	0.105	0.396	0.265	0.797
Logged	0.520	0.217	2.400	0.037
PC3 (Unlog)	0.054	0.071	0.755	0.468
PC3 (Log)	0.101	0.050	2.008	0.073
Salamander #/m^2 (Unlog)	0.290	0.122	2.366	0.040
Salamander #/m^2 (Log)	0.198	0.047	4.199	0.002
SVL (Unlog)	0.020	0.009	2.158	0.056
SVL (Log)	0.007	0.002	2.882	0.016
Sporocyst				
Unlogged	22.888	6.291	3.639	0.007
Logged	10.163	3.448	2.947	0.019
PC1 (Unlog)	-3.406	0.749	-4.546	0.002
PC1 (Log)	-1.338	0.267	-5.009	0.001
Adult #/m^2 (Unlog)	-12.014	6.264	-1.918	0.091
Adult #/m^2 (Log)	-4.360	2.874	-1.517	0.168
Snail #/m^2 (Unlog)	1.029	1.259	0.818	0.437
Snail #/m^2 (Log)	1.041	0.535	1.944	0.088
Salamander #/m^2 (Unlog)	4.481	1.021	4.388	0.002
Salamander #/m^2 (Log)	1.512	0.717	2.119	0.067
Metacercaria				
Unlogged	-1.036	3.026	-0.342	0.739
Logged	-2.237	3.355	-0.667	0.520
PC1 (Unlog)	1.532	0.762	2.011	0.072
PC1 (Log)	0.672	0.432	1.557	0.151
Sporocyst #/m^2 (Unlog)	0.340	0.218	1.559	0.150
Sporocyst #/m^2 (Log)	0.760	0.291	2.608	0.026
Stonefly #/m^2 (Unlog)	1.876	1.291	1.453	0.177
Stonefly #/m^2 (Log)	0.574	1.076	0.533	0.605

Table 3.15 Results of ANCOVA to assess the relationship of biotic and abiotic variables with parasite prevalence by forest type. Unlogged and Logged are regression intercepts. The remaining coefficients are regression slopes associated with each variable for either unlogged (Unlog) or logged with or without debris flow (Log) creeks. #/m^2 denotes host or parasite density, SVL = salamander snout vent length (mm), % = parasite prevalence.

Parasite Stage	Coefficients	SE	t	p
Adult parasite				
Unlogged	0.704	0.813	0.866	0.403
Logged	0.431	0.469	0.920	0.376
PC4 (Unlog)	-0.459	0.320	-1.433	0.177
PC4 (Log)	-0.845	0.362	-2.334	0.038
SVL (Unlog)	0.013	0.010	1.370	0.196
SVL (Log)	0.014	0.004	3.312	0.006
Sporocyst				
Unlogged	0.707	0.154	4.578	0.001
Logged	0.389	0.069	5.607	0.000
PC1 (Unlog)	-0.107	0.021	-5.192	0.000
PC1 (Log)	-0.042	0.006	-6.628	0.000
Salamander #/m^2 (Unlog)	-0.010	0.034	-0.307	0.765
Salamander #/m^2 (Log)	0.039	0.015	2.624	0.025
SVL (Unlog)	-0.009	0.003	-2.671	0.024
SVL (Log)	-0.001	0.001	-1.512	0.162
Metacercaria				
Unlogged	1.382	0.310	4.460	0.001
Logged	0.529	0.168	3.148	0.008
Sporocyst #/m^2 (Unlog)	0.099	0.091	1.089	0.298
Sporocyst #/m^2 (Log)	0.267	0.090	2.973	0.012
Sporocyst % (Unlog)	-4.098	3.743	-1.095	0.295
Sporocyst % (Log)	-5.408	2.785	-1.942	0.076

FIGURE CAPTIONS

Figure 3.1. Life cycle diagram of *Cephalouterina dicamptodoni*, a trematode parasite that infects the definitive host, *Dicamptodon tenebrosus*, the 1st intermediate host, *Juga silicula*, and the 2nd intermediate host, *Calineuria californica*. Parasites transmit between hosts via the parasite eggs and cercarial free-living stage. Arrows indicate direction of transmission for each stage of the parasite between hosts.

Figure 3.2. Relationship between shell length and gonad length in the snail, *Juga silicula*. Gonad length is $\log_e$ transformed.

Figure 3.3. Patterns of mean parasite intensity for *C. dicamptodoni* in each host from creeks in unlogged, logged, and logged-with-debris-flow watersheds. Values are averaged across creek means. Means and 95% confidence intervals are back transformed from $\log_e$. Letters indicate statistical significance within each parasite stage at the $\alpha = 0.05$ level. ns = non-significant. See text for statistics. Intensities vary across three orders of magnitude and are thus plotted on a log scale. $N_{(unlogged)} = 7$, $N_{(logged)} = 7$, $N_{(logged\ with\ debris)} = 4$.

Figure 3.4. Mean density for each parasitic stage of *C. dicamptodoni* in unlogged, logged and logged-with-debris-flow creeks. Values are averages of creek means. Error bars are 95% confidence intervals. Means and confidence limits are back transformed from the cube root. Letters indicate significance among forest types within one parasite

stage at the $\alpha = 0.05$ level. ns = non-significant. See text for statistics. Densities vary across four orders of magnitude and are plotted on a log scale to facilitate comparisons. $N_{(unlogged)} = 7$, $N_{(logged)} = 7$, $N_{(logged\ with\ debris)} = 4$.

Figure 3.5. Patterns of mean parasite prevalence for *C. dicamptodoni* in all hosts from creeks in unlogged, logged, and logged-with-debris-flow watersheds. Values are averaged across creek means. Error bars represent 95% confidence limits. Means and confidence limits are back transformed from the arcsine transformation for proportions. Letters indicate significance among forest types within parasite stage at the $\alpha = 0.05$ level. ns = non-significant. See text for statistics. $N_{(unlogged)} = 7$, $N_{(logged)} = 7$, $N_{(logged\ with\ debris)} = 4$.

Figure 3.6. Aggregation of each parasite stage in its host species by forest type. The graphs and coefficients were generated from data on individual salamanders, snails or stoneflies across all creeks from either unlogged, logged, or logged-with-debris-flow watersheds. * indicates significant difference at the $\alpha = 0.05$ level. For unlogged creeks: $N_{(salamanders)} = 131$, $N_{(adult\ parasites)} = 694$; $N_{(snails)} = 1886$, $N_{(sporocysts)} = 88217$; $N_{(stoneflies)} = 164$, $N_{(metacercariae)} = 2785$. For logged creeks: $N_{(salamanders)} = 101$, $N_{(adult\ parasites)} = 242$; $N_{(snails)} = 2126$, $N_{(sporocysts)} = 66299$; $N_{(stoneflies)} = 104$, $N_{(metacercariae)} = 953$. For logged-with-debris-flow creeks: $N_{(salamanders)} = 58$, $N_{(adult\ parasites)} = 277$; $N_{(snails)} = 661$, $N_{(sporocysts)} = 5682$; $N_{(stoneflies)} = 144$, $N_{(metacercariae)} = 284$.

Figure 3.7. Mean host density by forest type. Values are averaged across creek means. Error bars represent 95% confidence limits. Means and confidence limits for salamander hosts are back transformed from $\log_e$. Letters show significant differences among densities of a single host across forest types at the $\alpha = 0.05$ level See text for statistics. $N_{(unlogged)} = 7$, $N_{(logged)} = 7$, $N_{(logged\ with\ debris)} = 4$.

Figure 3.8. Comparisons of host length across forest types. Values are creek means averaged by forest type and 95% confidence limits. Letters show significant differences in length of individual hosts across forest types at the $\alpha = 0.05$ level. See text for statistics. Salamander length was measured from the snout to vent (SVL). SVL was measured in the field on all salamanders captured, regardless of whether they were released or collected. Snail length was measured on preserved specimens and was measure from the whorl apex to the outermost edge of the aperture. Stonefly length was measured on preserved specimens from the head to posterior of the abdomen. $N_{(unlogged)} = 7$, $N_{(logged)} = 7$, $N_{(logged\ with\ debris)} = 4$.

Figure 3.9. Differences in salamander mass across forest types. Mass was measured in the field on all salamanders captured, regardless of whether they were released or collected for the parasite study. Means are averages of creek means by forest. Letters show significant differences at the $\alpha = 0.05$ level. See text for statistics. $N_{(unlogged)} = 7$, $N_{(logged)} = 7$, $N_{(logged\ with\ debris)} = 4$.

Figure 3.10. Comparison of salamander diet by forest type. Diet was measured as the total number of prey found in the stomach and intestine of each salamander. Prey items were only counted and identified when more than 50% of the item was intact. Numbers of stoneflies (*Calineuria californica*) in the diet were assessed separately as an indication of differential parasite transmission across forest type. * indicates significance at the α = 0.05 level. ns = non-significant. See text for statistics. Means and 95% confidence limits are back transformed from the square root. $N_{(unlogged)} = 7$, $N_{(logged)} = 7$, $N_{(logged\ with\ debris)} = 4$.

Figure 3.11. Regressions between parasite intensity and host length for each parasite-host pair by forest type. Values on each y-axis are $\log_{10}$ transformed. Regressions were calculated for each host-parasite pair using ANCOVA techniques to determine whether regression coefficients varied by forest type. See Table 3.9 for results and text for description of technique. For unlogged creeks: $N_{(salamanders)}= 97$, $N_{(adult\ parasites)}= 694$; $N_{(snails)} = 79$, $N_{(sporocysts)} = 88217$; $N_{(stoneflies)} = 142$, $N_{(metacercariae)} = 2785$. For logged creeks: $N_{(salamanders)}= 57$, $N_{(adult\ parasites)}= 242$; $N_{(snails)} = 60$, $N_{(sporocysts)} = 66299$; $N_{(stoneflies)} = 80$, $N_{(metacercariae)} = 953$. For logged-with-debris-flow creeks: $N_{(salamanders)}= 32$, $N_{(adult\ parasites)}= 277$; $N_{(snails)} = 7$, $N_{(sporocysts)} = 5682$; $N_{(stoneflies)} = 51$, $N_{(metacercariae)} = 284$.

Figure 3.12. Comparisons of abiotic factors across forest type. See text for descriptions of methods, indices and statistics. Means for all factors except microhabitat and pool:riffle ratio were calculated per creek and then averaged by forest type. Microhabitat and pool:riffle ratios had single values per creek and were averaged by forest type.

Letters indicate significant differences among means. ns = non-significant. Pebble counts are in mm. Thalweg velocity is in m/s. All other measures are indices of abiotic factors as described in the methods section.

Figure 3.13. Comparisons of percent microhabitat by forest type. Percentages were averaged across creek by forest type. Bars are ± 1 standard error.

Figure 3.14. Comparisons of air and water temperatures across unlogged, logged and logged-with-debris-flow creeks. Errors are ± 1 standard error. Temperatures did not differ significantly across forest type for the 24h period measured.

Figure 3.15. Cumulative variance of principal components for analyses among creeks. The first four principal components were used in ANCOVA analyses.

Figure 3.16. Loadings for each principal component. Loadings were considered significant at ± 0.4

Figure 3.17. Trends in principal components (PC) by forest type. ANOVAs revealed significant differences (*) among means for PC1 and PC4 by forest type at an $\alpha = 0.05$ level. Values of PC2 and PC3 are $\log_e$ transformed. Values for PC4 are square root transformed.

Figure 3.18. Regressions between donor and target parasite density ($\#\cdot m^{-2}$). Data points are square root transformed mean densities of parasite stage per creek. See text and Table 3.13 for statistical analyses.

Figure 3.19. Mean a) parasite density ($\#\cdot m^{-2}$), b) parasite prevalence (% hosts infected), and c) host density ($\#\cdot m^{-2}$) per creek for each forest type. The first set of four creeks was in logged watersheds that experienced debris flows. The second set of seven creeks was in logged watersheds. The last set of seven creeks was in unlogged watersheds.

Figure 3.20. Mean a) parasite density ($\#\cdot m^{-2}$), b) parasite prevalence (% hosts infected), and c) host density ($\#\cdot m^{-2}$) per creek for logged-with-debris-flow creeks only.

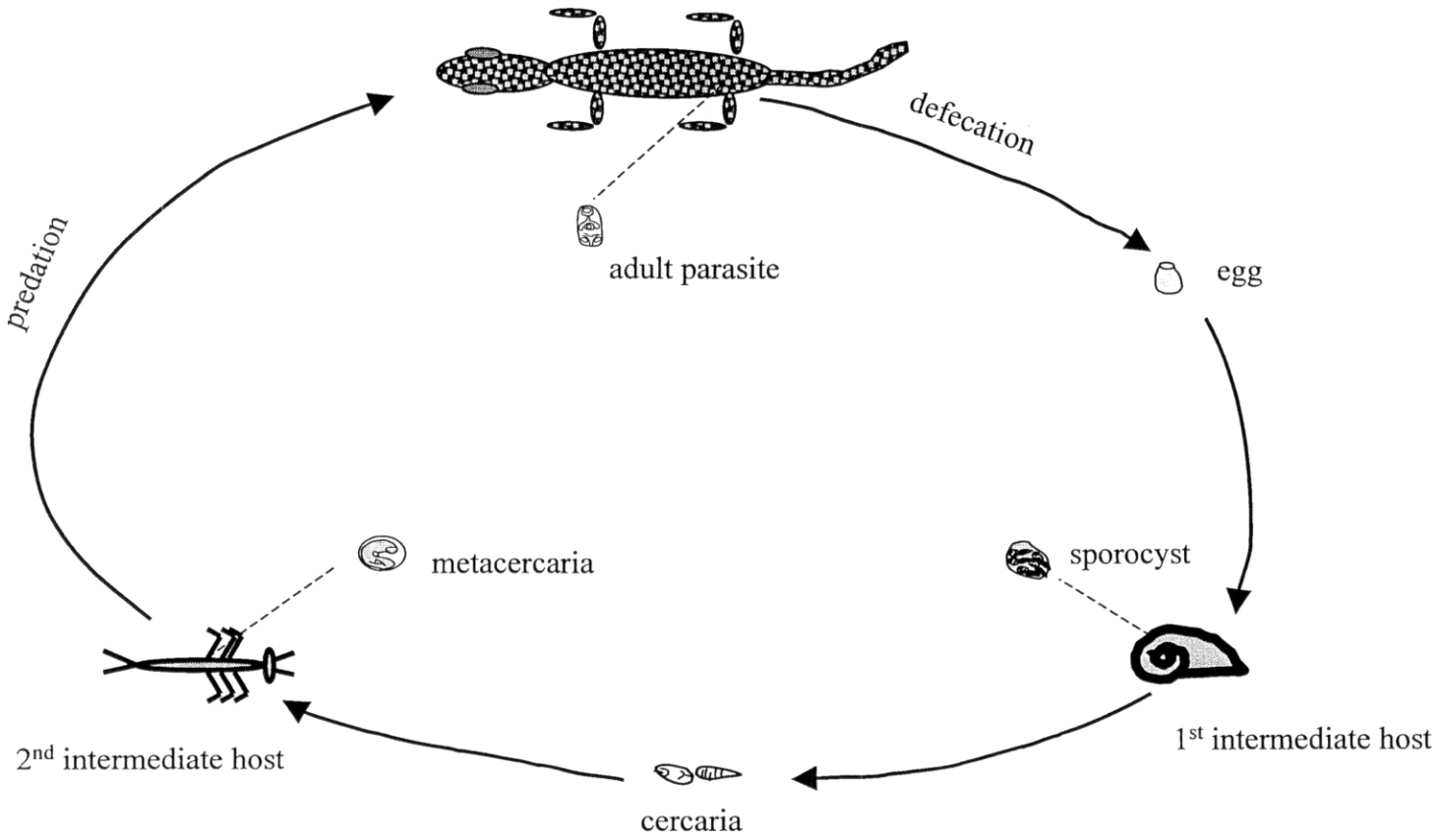

definitive host
defecation
adult parasite
egg
predation
metacercaria
sporocyst
2nd intermediate host
1st intermediate host
cercaria

Fig. 3.1

Fig. 3.2

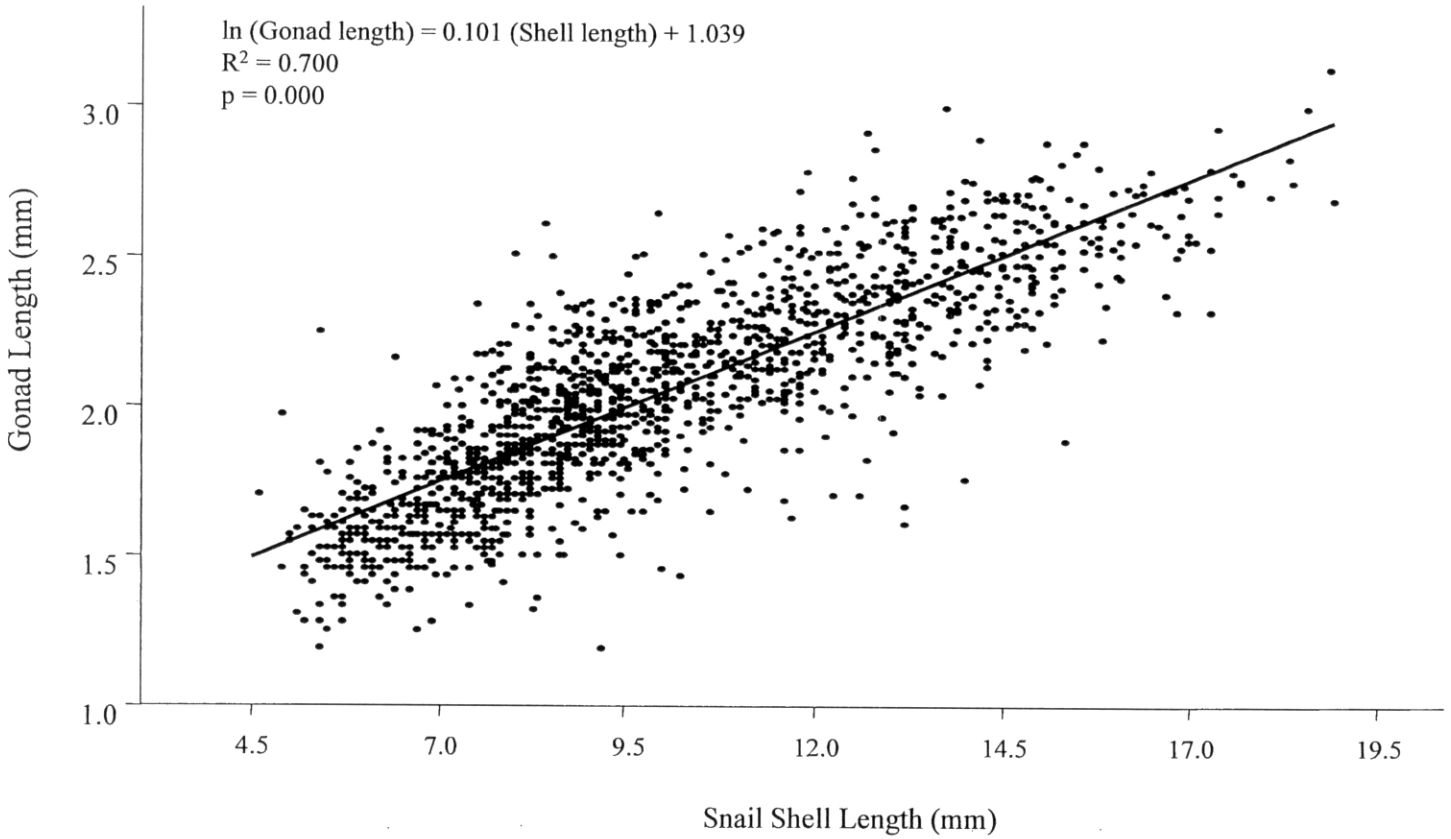

ln (Gonad length) = 0.101 (Shell length) + 1.039
R^2 = 0.700
p = 0.000
Gonad Length (mm)
3.0
2.5
2.0
1.5
1.0
4.5
7.0
9.5
12.0
14.5
17.0
19.5
Snail Shell Length (mm)

Fig. 3.3

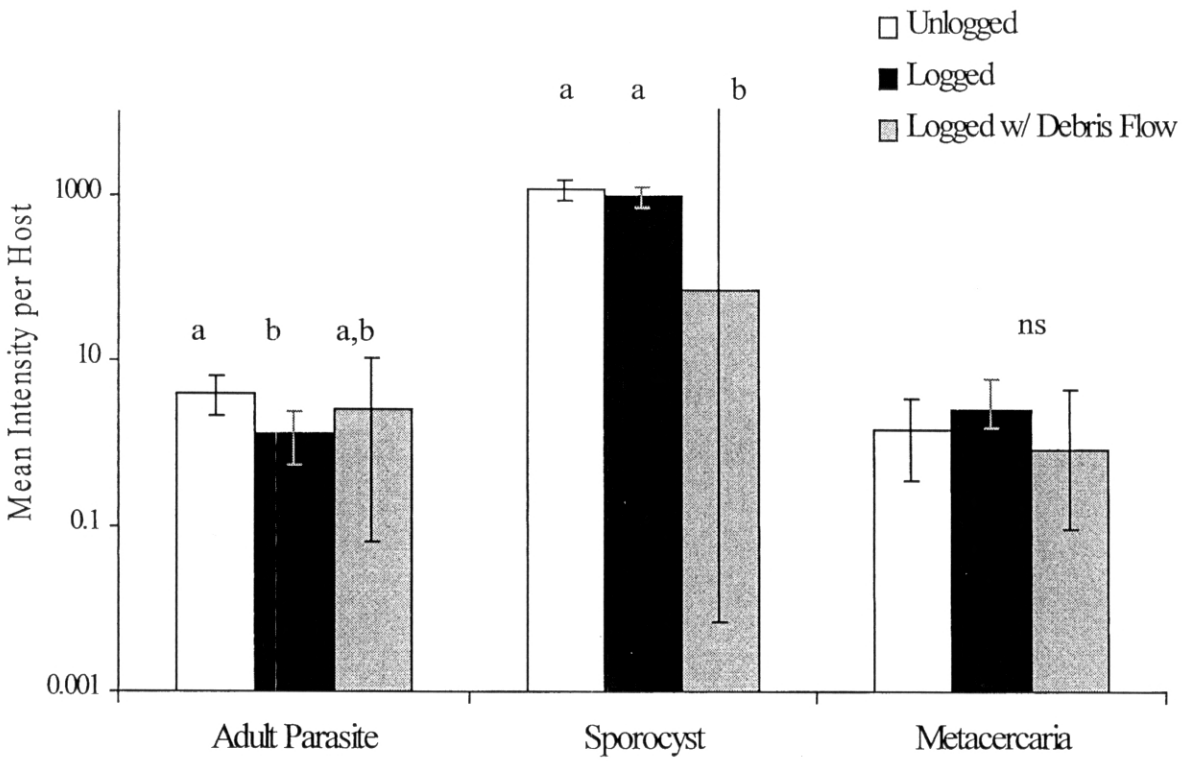

Unlogged
Logged
Logged w/ Debris Flow
Mean Intensity per Host
1000
10
0.1
0.001
a
b
a,b
a
a
b
ns
Adult Parasite
Sporocyst
Metacercaria

Fig. 3.4

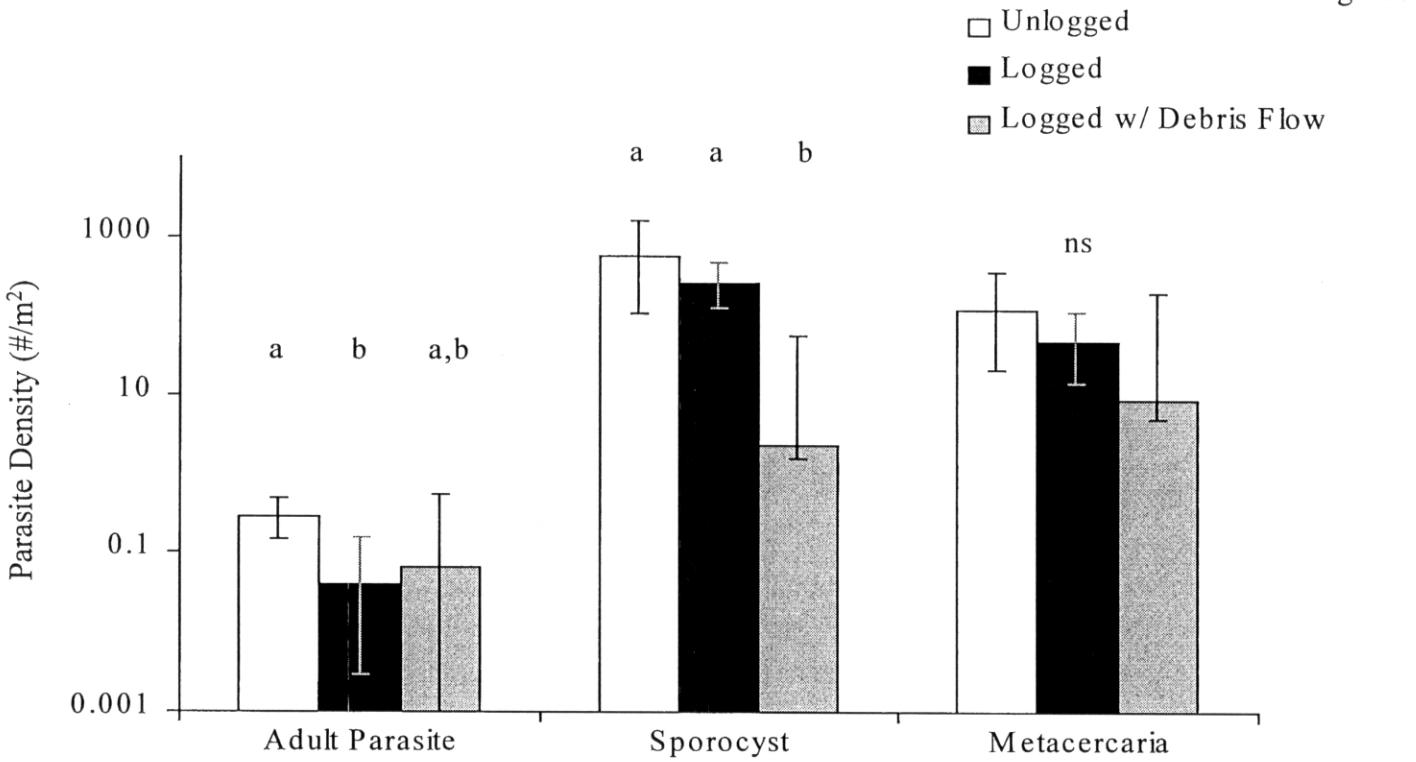

Unlogged
Logged
Logged w/ Debris Flow
Parasite Density (#/m²)
1000
10
0.1
0.001
a
b
a,b
a
a
b
ns
Adult Parasite
Sporocyst
Metacercaria

Fig. 3.5

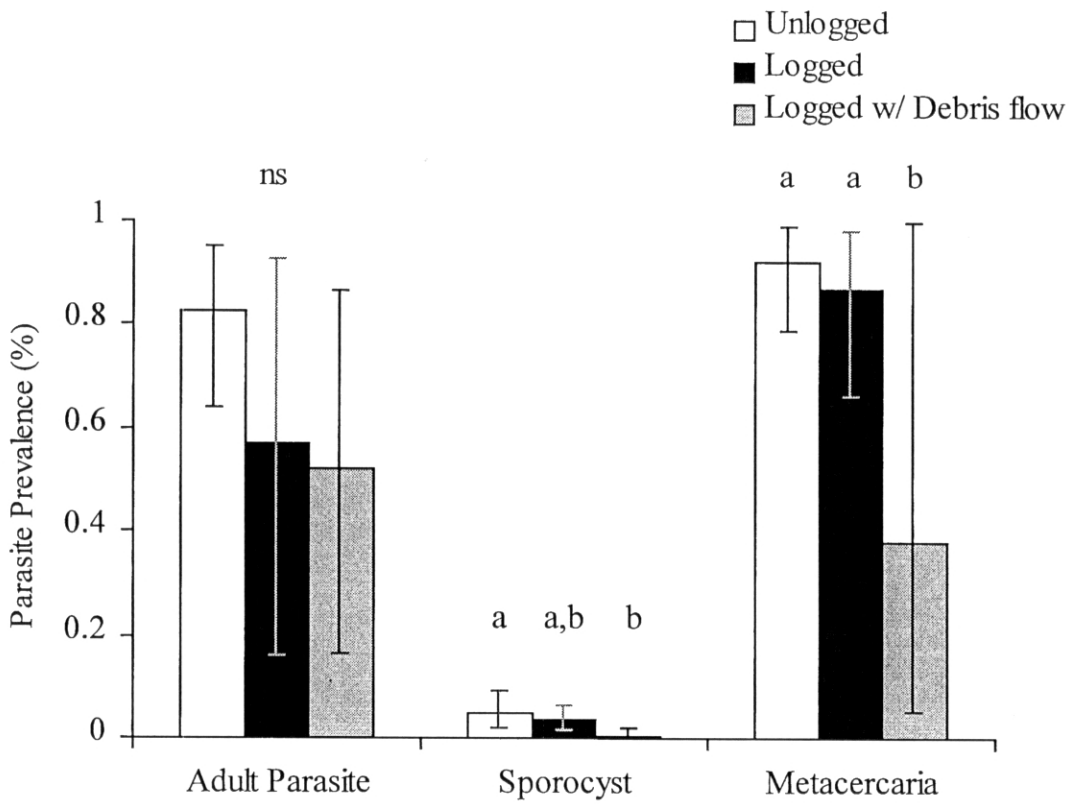

Unlogged
Logged
Logged w/ Debris flow
ns
a a b
a a,b b
Parasite Prevalence (%)
1
0.8
0.6
0.4
0.2
0
Adult Parasite
Sporocyst
Metacercaria

Fig. 3.7

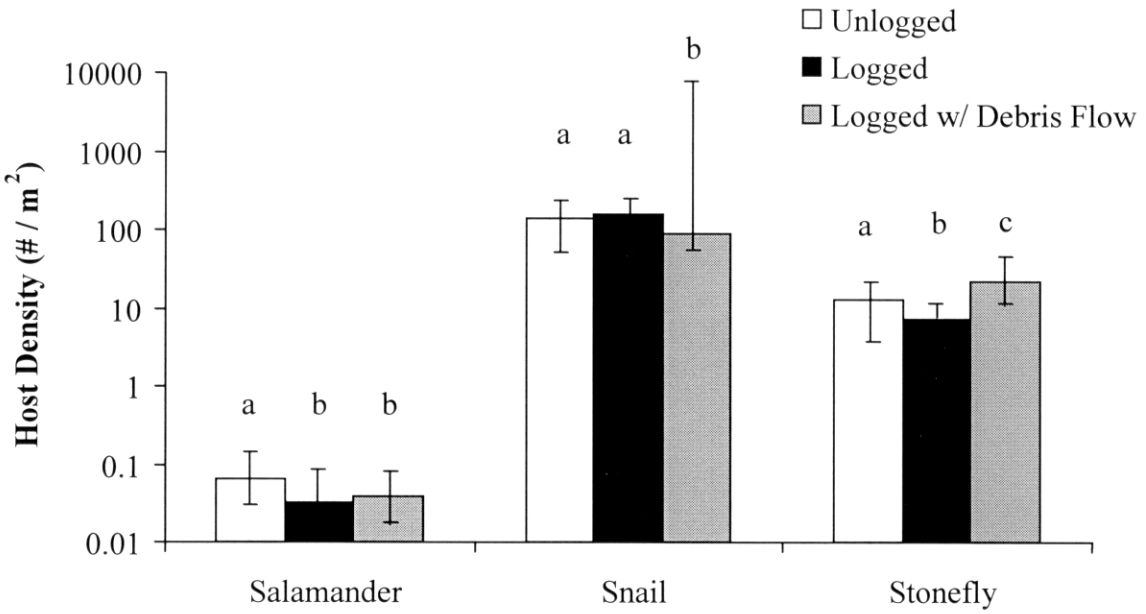

Unlogged
Logged
Logged w/ Debris Flow
Host Density (# / m2)
10000
1000
100
10
1
0.1
0.01
a
b
b
a
a
b
a
b
c
Salamander
Snail
Stonefly

Fig. 3.8

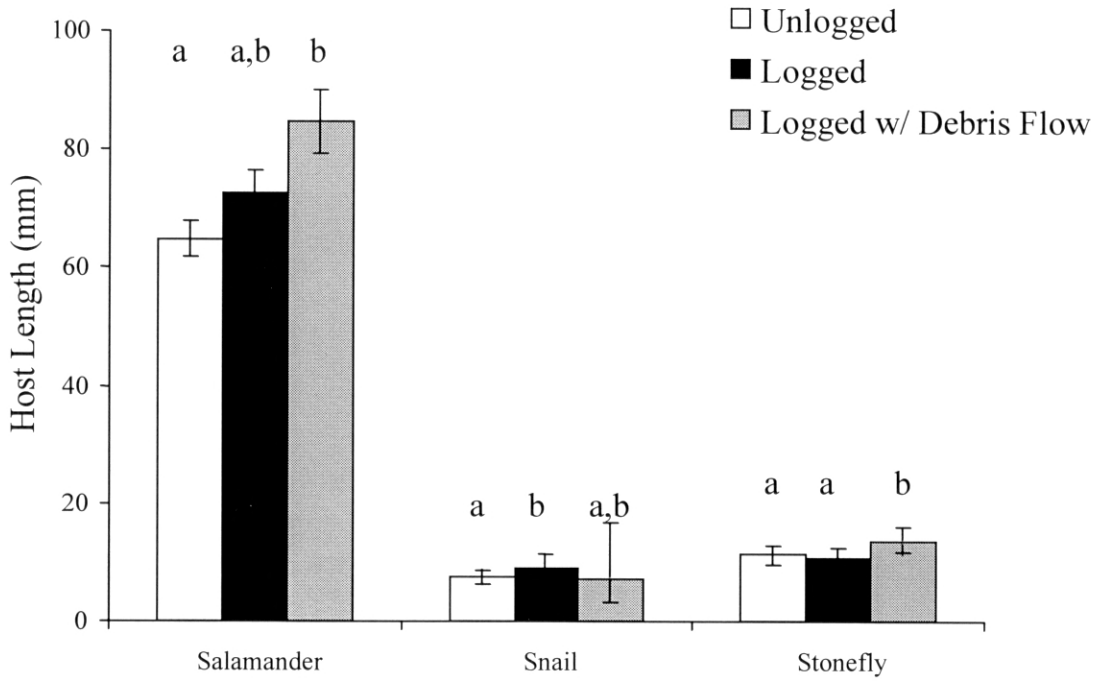

Unlogged
Logged
Logged w/ Debris Flow
Host Length (mm)
100
80
60
40
20
0
a
a,b
b
a
b
a,b
a
a
b
Salamander
Snail
Stonefly

Fig. 3.6

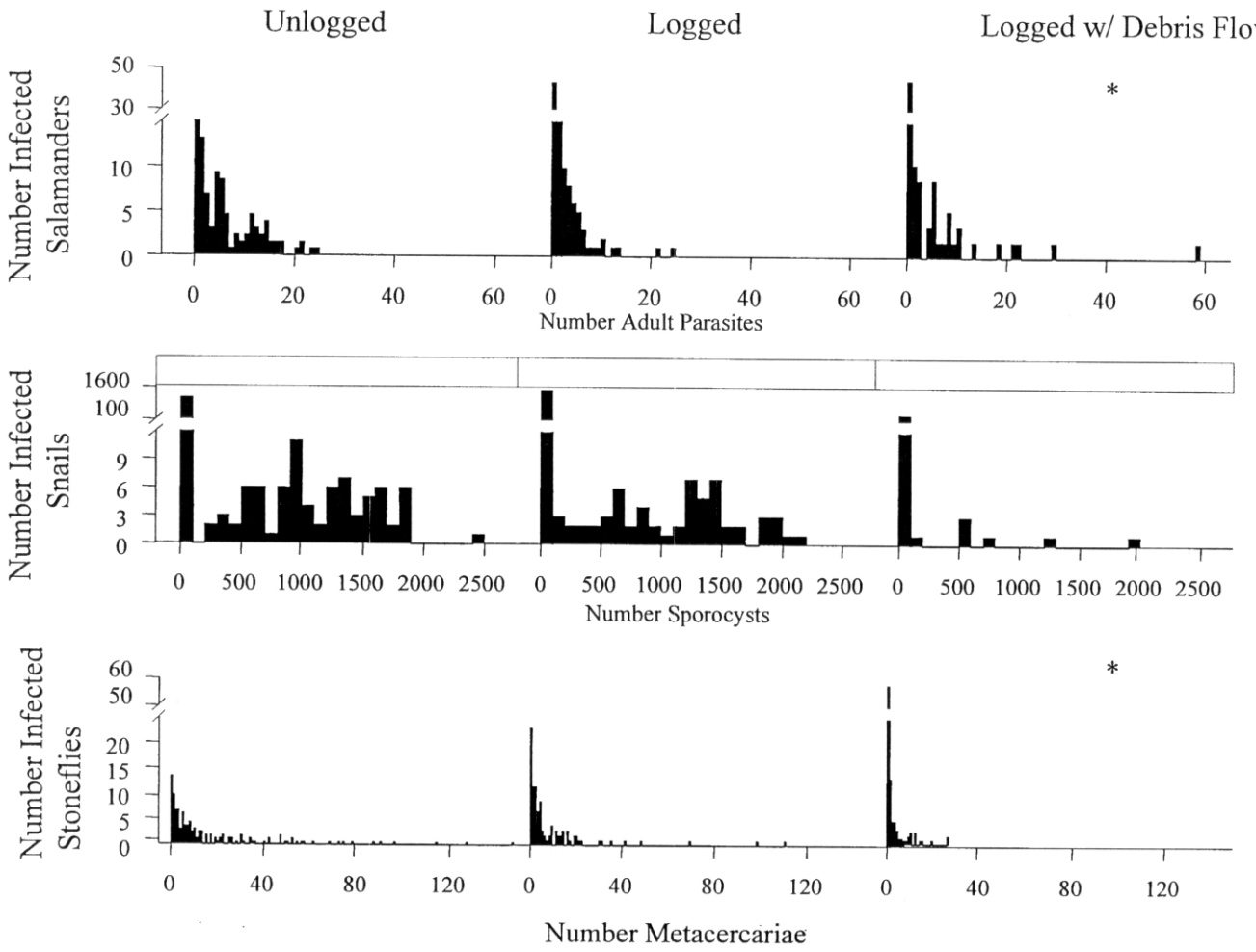

Unlogged
Logged
Logged w/ Debris Flow
Number Infected Salamanders
50
30
10
5
0
*
0 20 40 60 0 20 40 60 0 20 40 60
Number Adult Parasites
Number Infected Snails
1600
100
9
6
3
0
0 500 1000 1500 2000 2500 0 500 1000 1500 2000 2500 0 500 1000 1500 2000 2500
Number Sporocysts
Number Infected Stoneflies
60
50
20
15
10
5
0
*
0 40 80 120 0 40 80 120 0 40 80 120
Number Metacercariae

Fig. 3.9

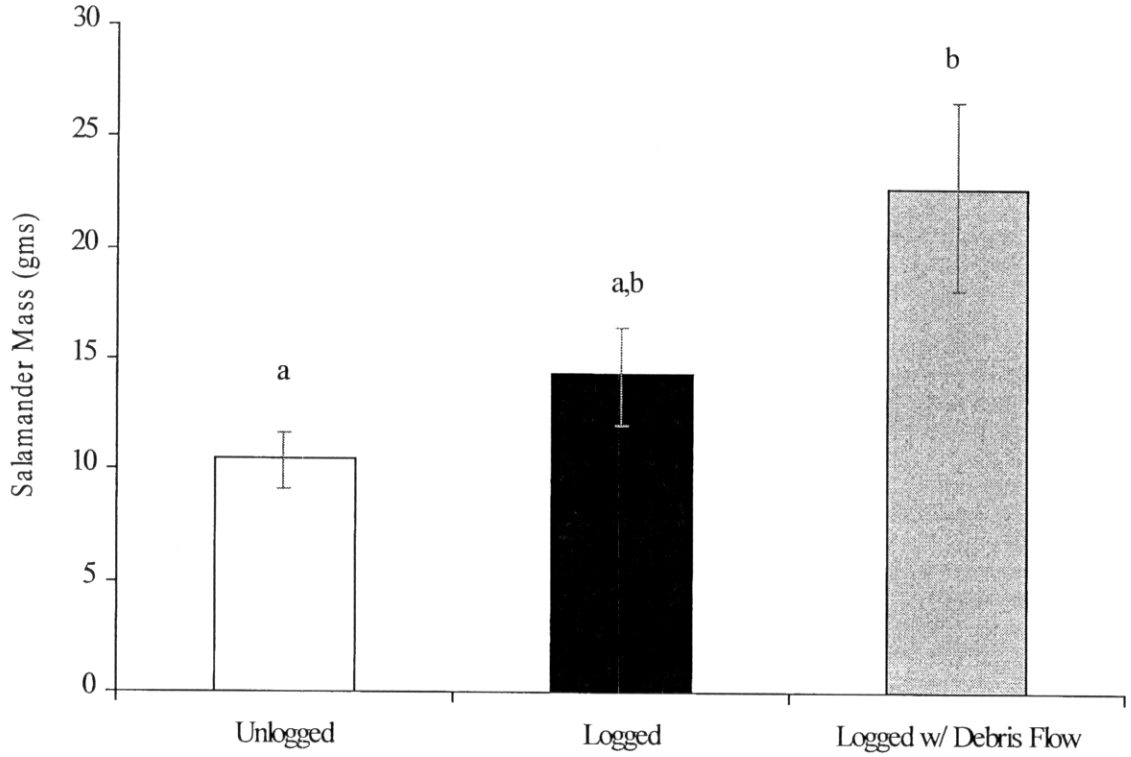

Salamander Mass (gms)
30
25
20
15
10
5
0
a
a,b
b
Unlogged
Logged
Logged w/ Debris Flow

Fig. 3.10

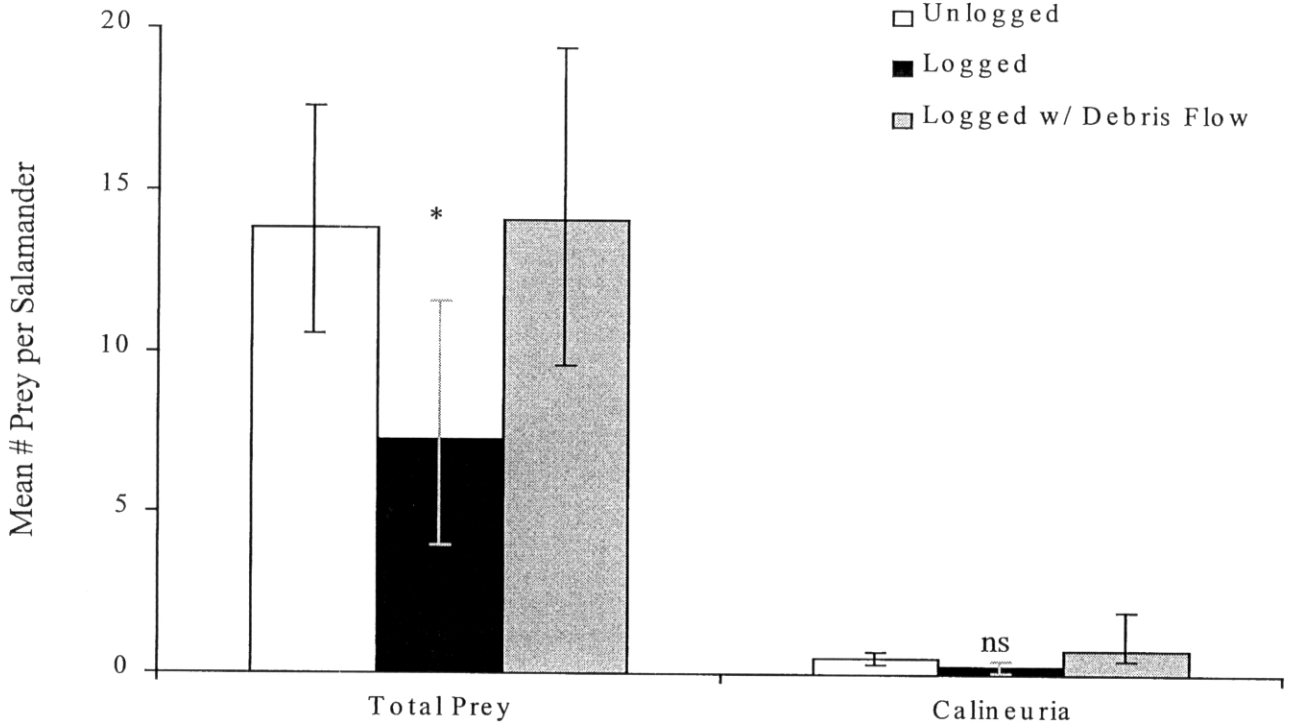

Unlogged
Logged
Logged w/ Debris Flow
Mean # Prey per Salamander
20
15
10
5
0
*
ns
Total Prey
Calineuria

Fig. 3.11

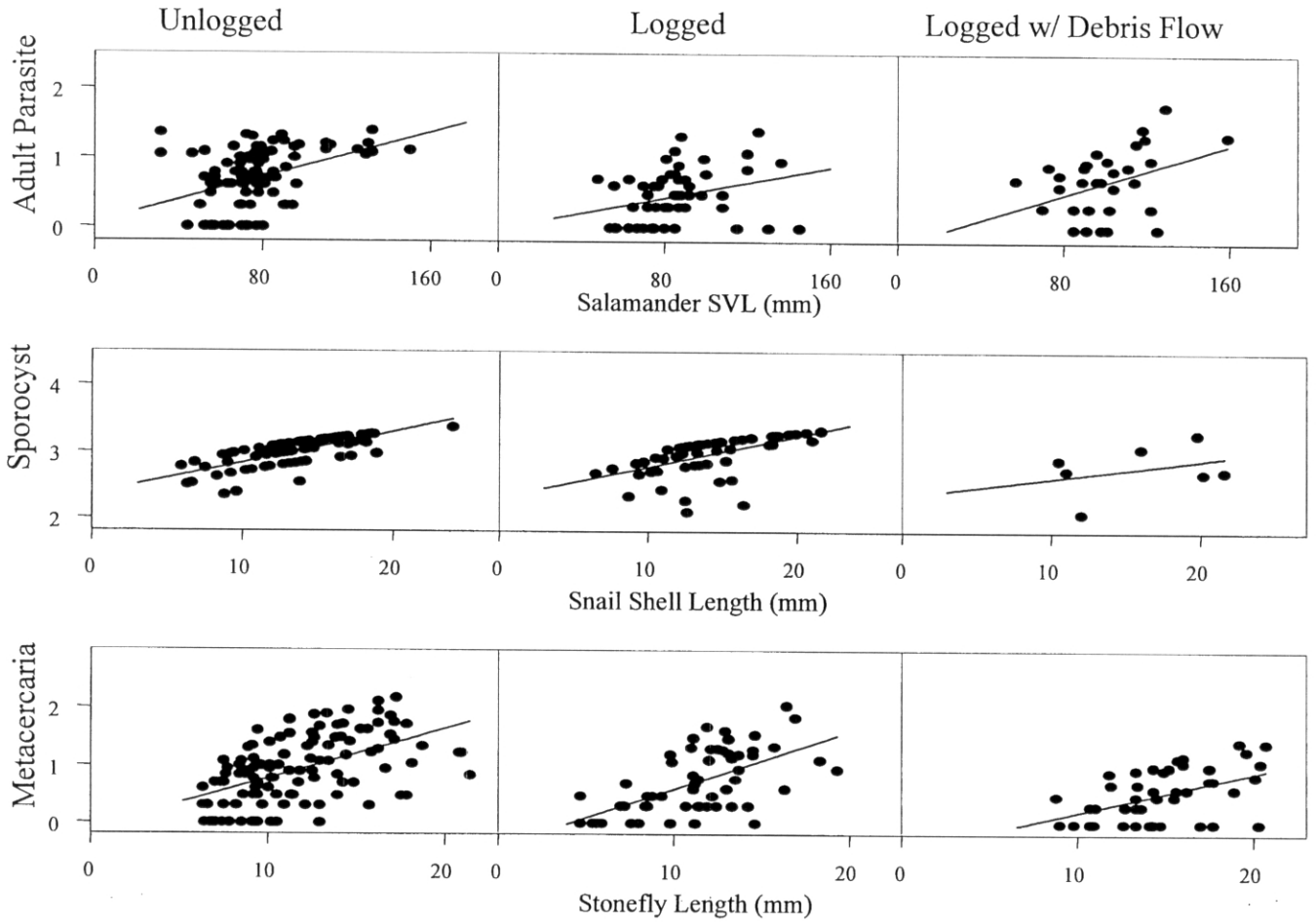

Unlogged
Logged
Logged w/ Debris Flow
Adult Parasite
Sporocyst
Metacercaria
Salamander SVL (mm)
Snail Shell Length (mm)
Stonefly Length (mm)

Fig. 3.12

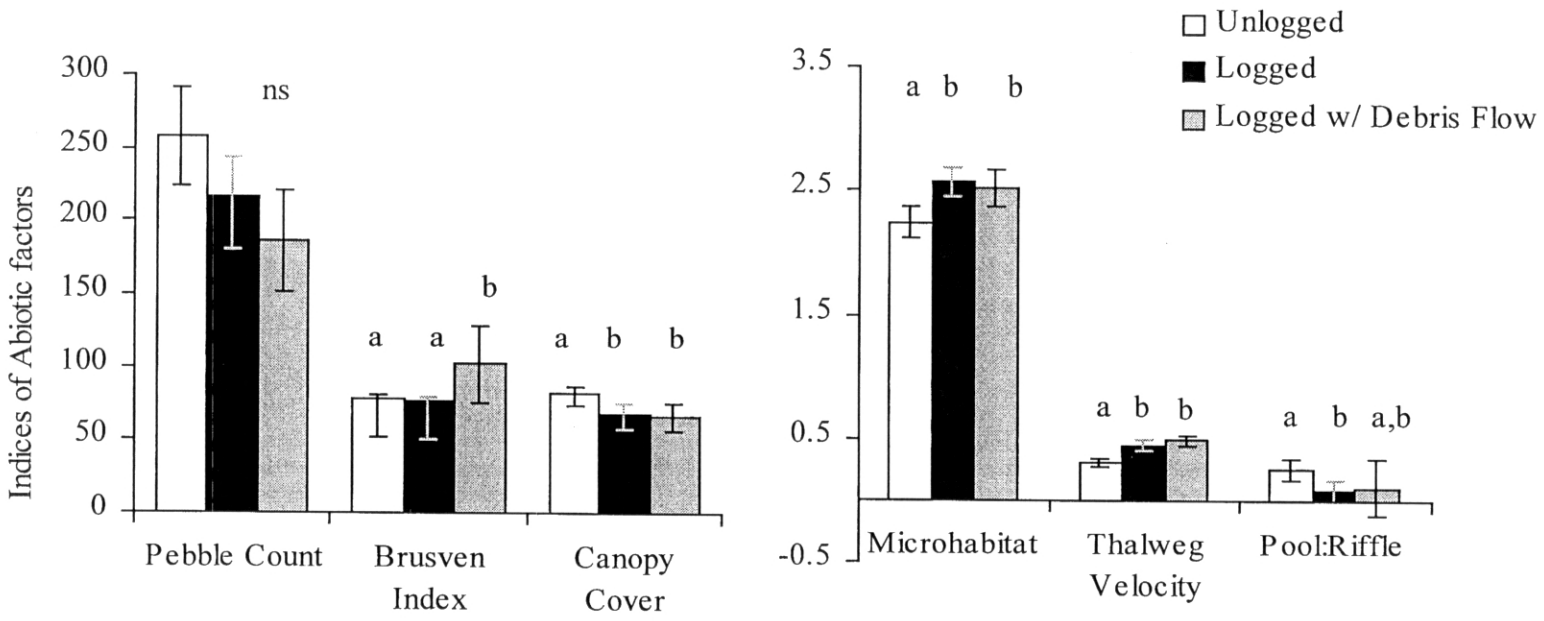

Indices of Abiotic factors
300
250
200
150
100
50
0
ns
a a b
a b b
Pebble Count
Brusven Index
Canopy Cover
Unlogged
Logged
Logged w/ Debris Flow
3.5
2.5
1.5
0.5
-0.5
a b b
a b b
a b a,b
Microhabitat
Thalweg Velocity
Pool:Riffle

Fig. 3.13

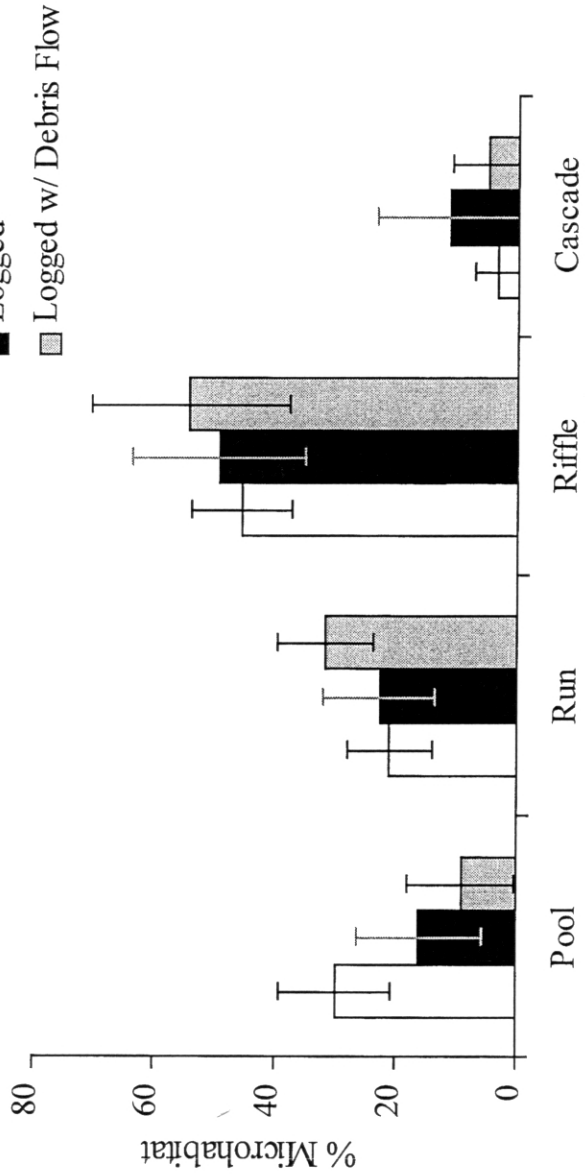

Unlogged
Logged
Logged w/ Debris Flow
% Microhabitat
0
20
40
60
80
Pool
Run
Riffle
Cascade

Fig. 3.14

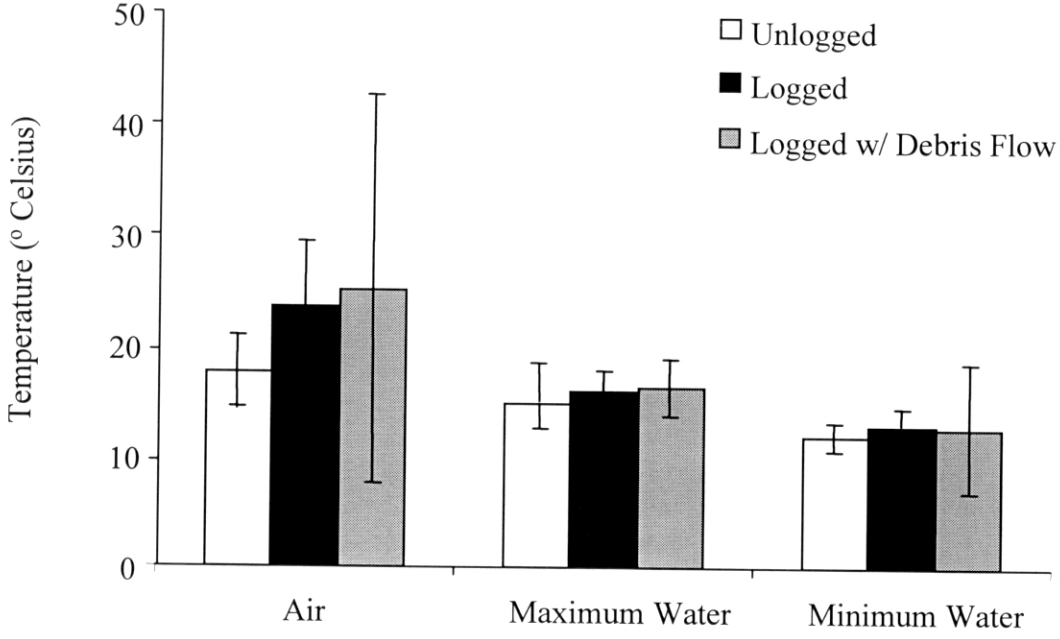

Temperature (° Celsius)
50
40
30
20
10
0
Unlogged
Logged
Logged w/ Debris Flow
Air
Maximum Water
Minimum Water

Fig. 3.15

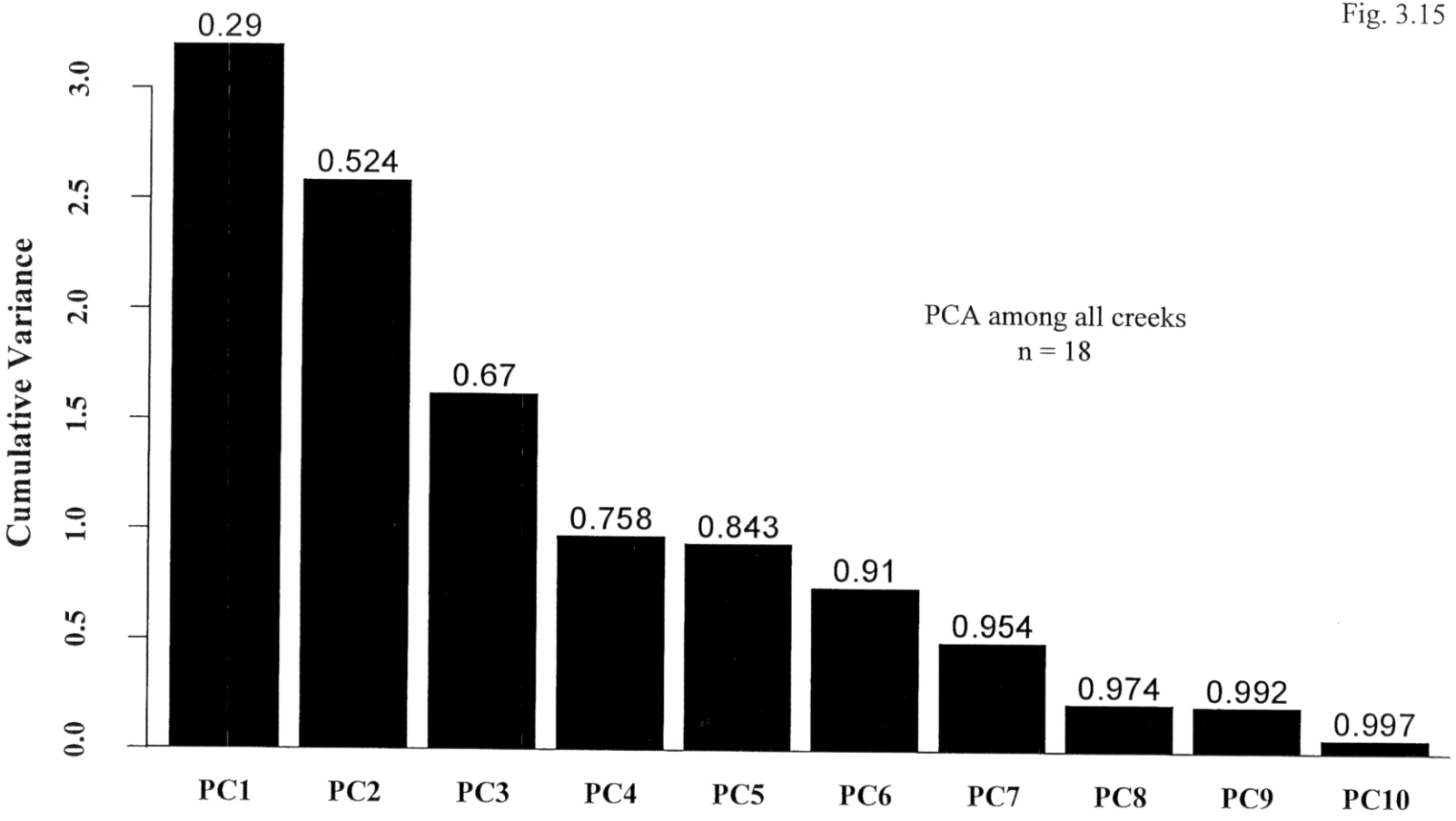
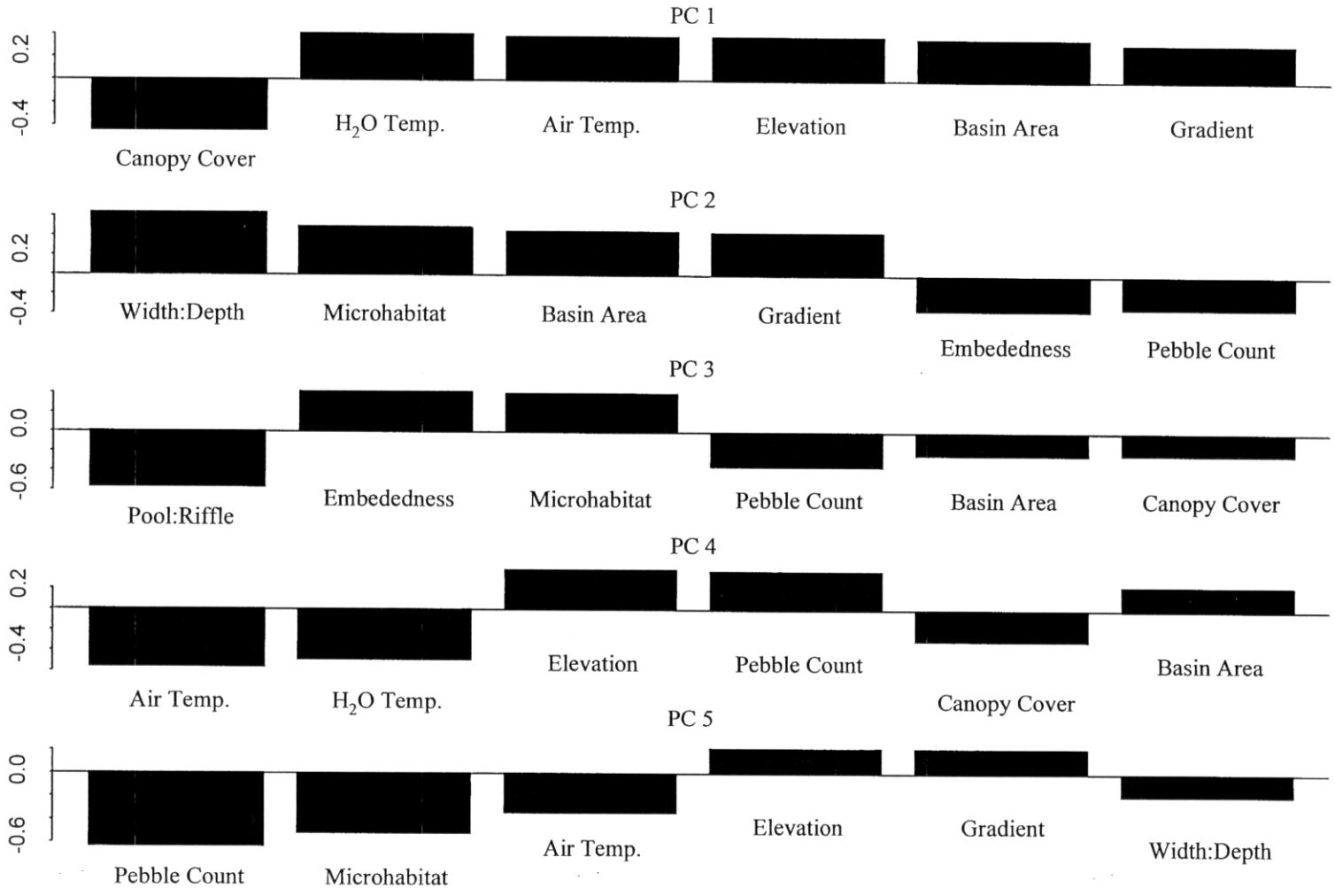

Cumulative Variance
0.0
0.5
1.0
1.5
2.0
2.5
3.0
0.29
0.524
0.67
0.758
0.843
0.91
0.954
0.974
0.992
0.997
PC1
PC2
PC3
PC4
PC5
PC6
PC7
PC8
PC9
PC10
PCA among all creeks
n = 18

Fig. 3.16



Principal Component Loadings

Fig. 3.17

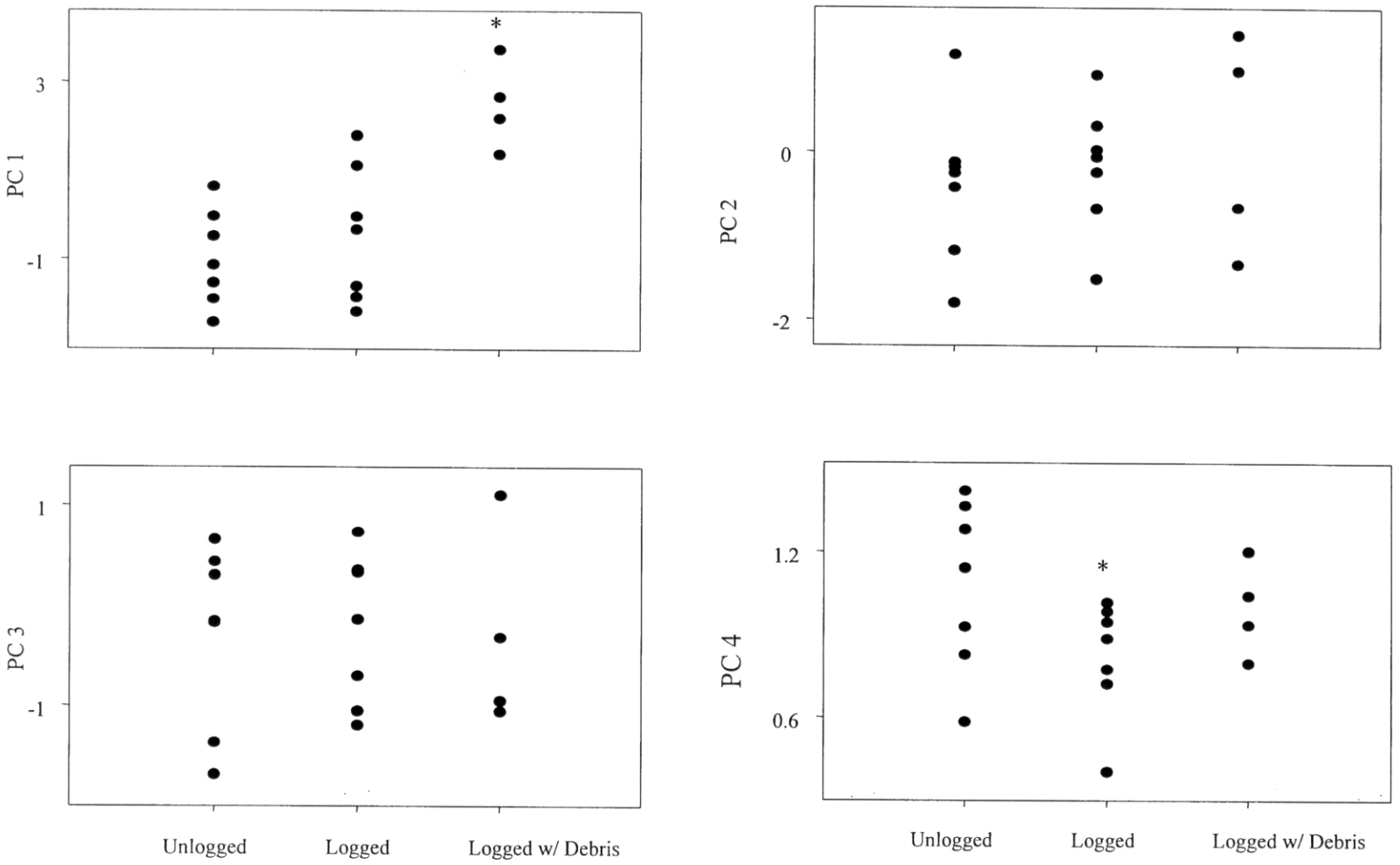

PC 1
3
-1
*
PC 2
0
-2
PC 3
1
-1
PC 4
1.2
0.6
*
Unlogged
Logged
Logged w/ Debris
Unlogged
Logged
Logged w/ Debris

Fig. 3.18

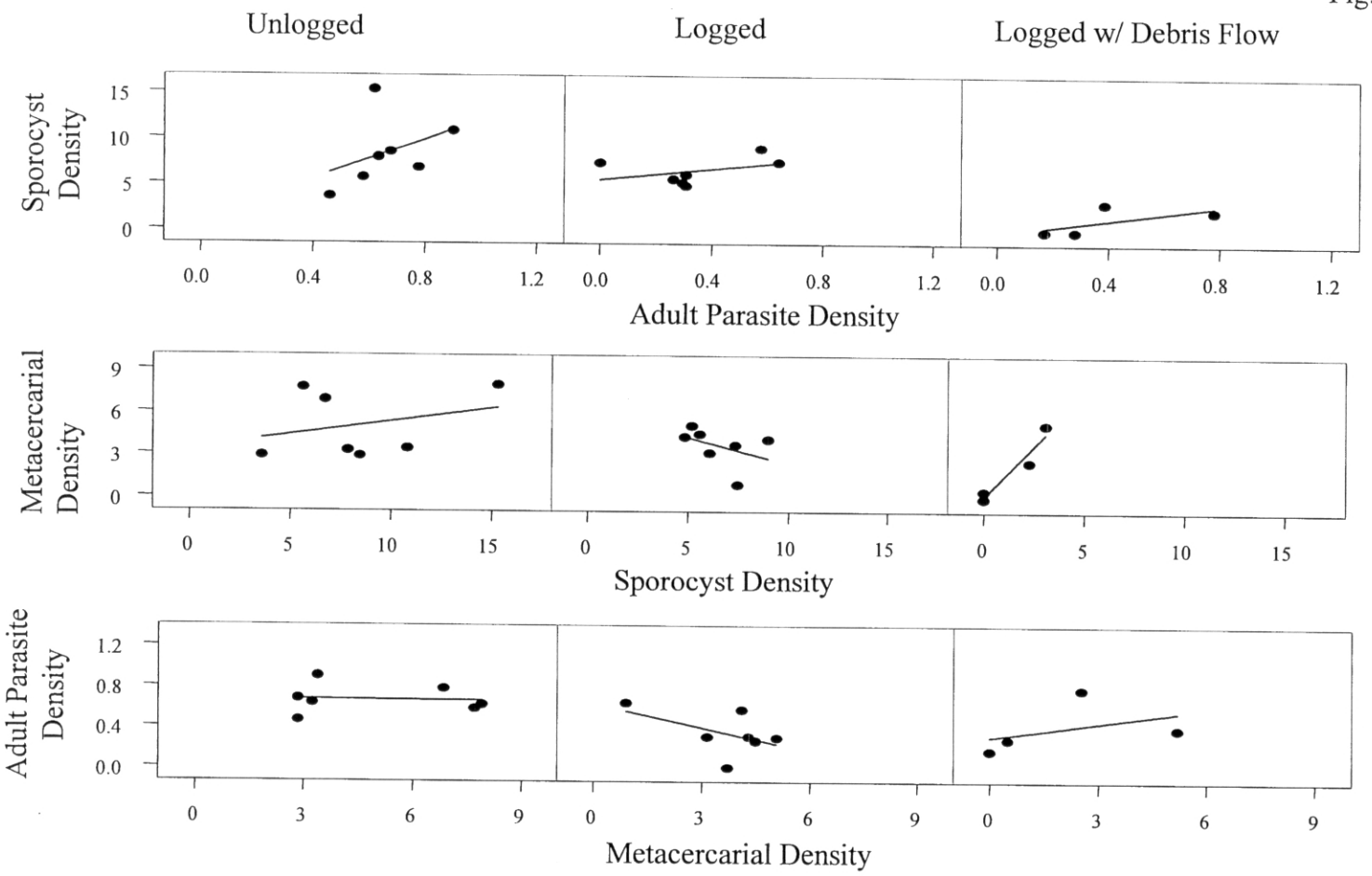

Unlogged
Logged
Logged w/ Debris Flow
Sporocyst Density
Adult Parasite Density
Metacercarial Density
Sporocyst Density
Adult Parasite Density
Metacercarial Density

Fig. 3.19

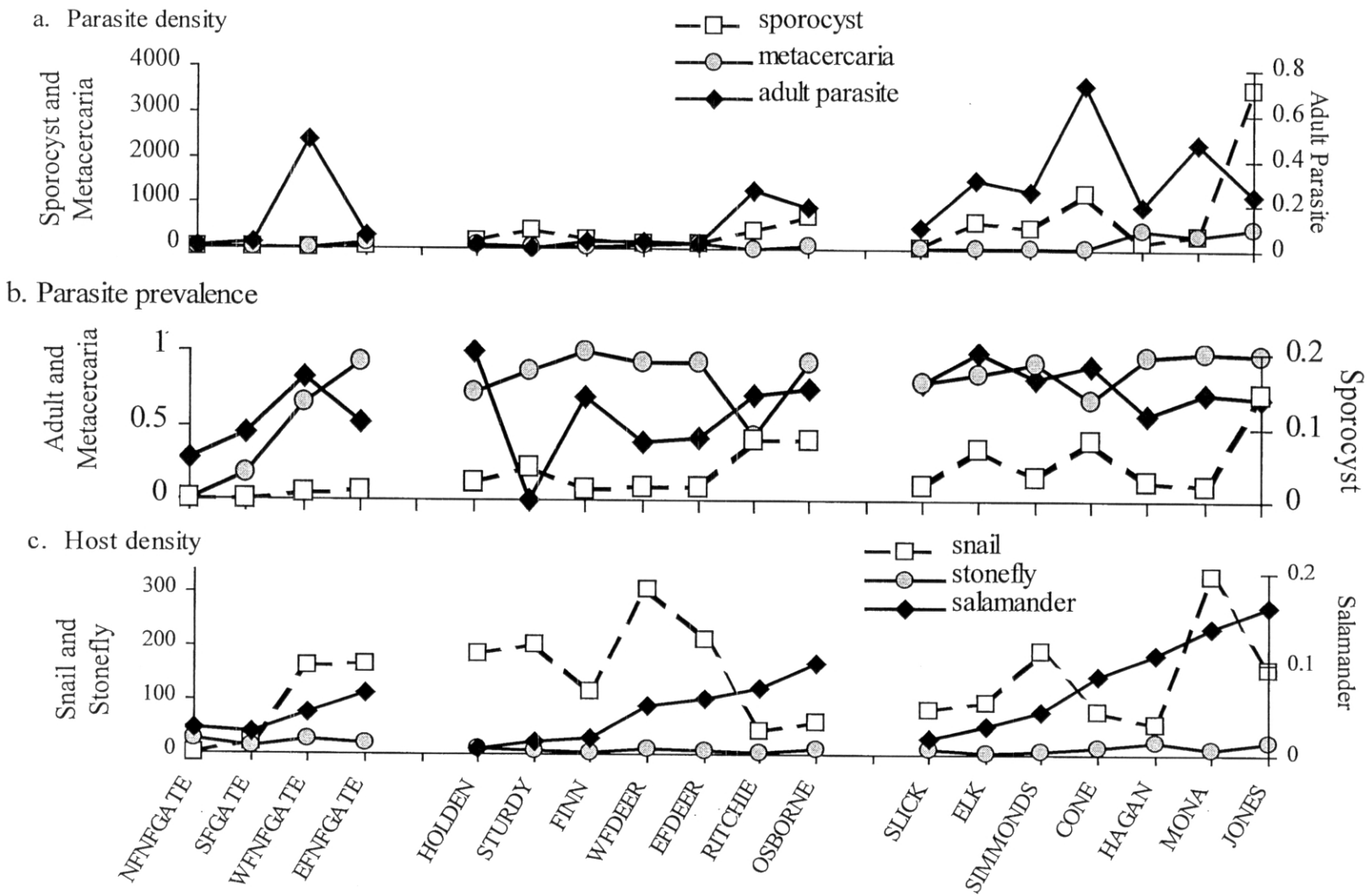
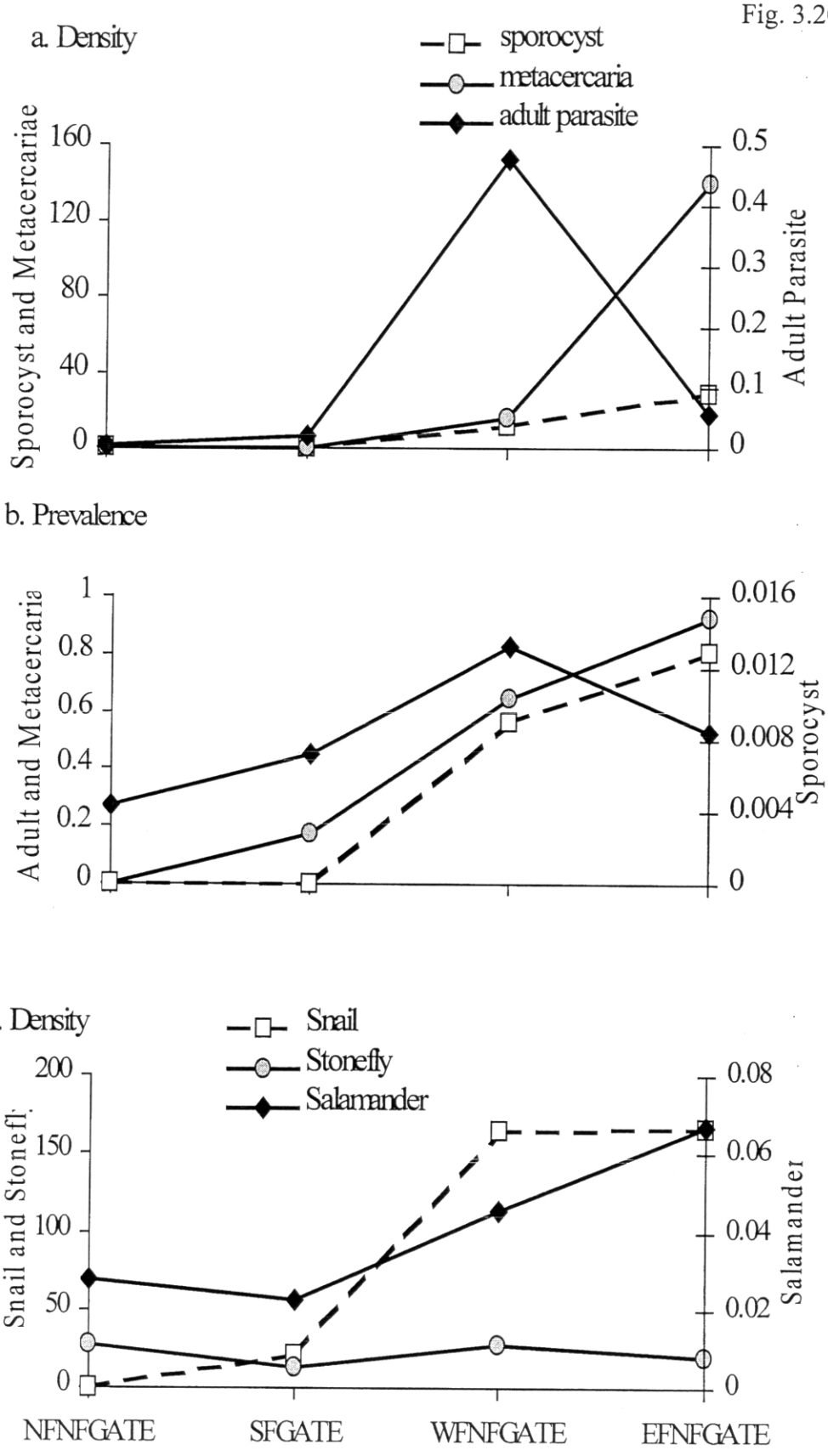

a. Parasite density
sporocyst
metacercaria
adult parasite
Sporocyst and Metacercaria
4000
3000
2000
1000
0
0.8
0.6
0.4
0.2
0
Adult Parasite
b. Parasite prevalence
Adult and Metacercaria
1
0.5
0
0.2
0.1
0
Sporocyst
c. Host density
snail
stonefly
salamander
Snail and Stonefly
300
200
100
0
0.2
0.1
0
Salamander
NFNFGATE
SFGATE
WFNFGATE
EFNFGATE
HOLDEN
STURDY
FINN
WFDEER
EFDEER
RITCHIE
OSBORNE
SLICK
ELK
SIMMONDS
CONE
HAGAN
MONA
JONES

Fig. 3.20


a. Density
sporocyst
metacercaria
adult parasite
Sporocyst and Metacercariae
160
120
80
40
0
0.5
0.4
0.3
0.2
0.1
0
Adult Parasite
b. Prevalence
Adult and Metacercaria
1
0.8
0.6
0.4
0.2
0
0.016
0.012
0.008
0.004
0
Sporocyst
c. Density
Snail
Stonefly
Salamander
Snail and Stonefl
200
150
100
50
0
0.08
0.06
0.04
0.02
0
Salamander
NFNFGATE
SFGATE
WFNFGATE
EFNFGATE

CHAPTER 4 Relative Strength of Parasite Transmission in an Indirect Life Cycle: Implications of Logging to Parasite Dynamics in Stream Communities

INTRODUCTION

Anthropogenic disturbance is widely implicated in altered patterns of parasitism in wildlife (Lafferty 1997, Harvell et al. 1999, Daszak et al. 2000). Few empirical studies evaluate this claim (e.g., Burkholder et al. 1995, Thorne and Williams 1998, Chapter 3) and even fewer assess the causal relationship between disturbance and parasitism (but see Burkholder and Glasgow 1997, Chapter 3). The lack of rigorous data is not surprising considering that many disturbances are unplanned and unreplicated, making evaluation of disturbance-induced changes in complex host-parasite systems difficult at best. That environmental disturbance could affect parasitism is intuitive since parasite dynamics are tightly linked to the density and distribution of their host species. Demonstrating the effects of disturbance on parasitism is more difficult, but by focusing on the aspects of parasite-host interactions that are likely to be sensitive to disturbances we can begin to predict the response of parasites to disturbances. Since the success of parasites hinges on transmission from one host to the next, disturbance-induced changes in host density are likely to have the greatest effect on the rate or functional response of transmission (e.g., May 1977, May and Anderson 1979, Schwartz 1985, Hochberg 1991, Woolhouse et al. 1991, Anderson and May 1992, Gubbins and Gilligan 1997, Hudson and Dobson 1997). The direction and strength of these effects would depend on characteristics of the

particular host-parasite association. For diseases induced by parasites with complex life cycles, parasite populations are dependent on successful transmission between multiple donor/target host pairs. Any or all of the transmission stages could respond to disturbance to affect parasite dynamics, however the strength and effect of those responses on parasite dynamics could vary. The best empirical example of this phenomenon is the work of Burkholder and Glasgow (1997) on the differential response of difference stages of the toxic dinoflagellate, *Pfiesteria piscicida*, to changing nutrient levels.

Theoretical models predict that parasite dynamics are sensitive to the functional form of transmission. Parasite-host models respond with strikingly different behaviors to variation in transmission (May and Anderson 1979, Gabriel et al. 1981, Getz and Pickering 1983, Liu et al. 1986, Hochberg 1991). The standard mass-action models assume that the rate of transmission is directly proportional to the densities of uninfected and infected hosts or parasitic stages (e.g., Crofton 1971, Anderson and May 1978, 1979, May and Anderson 1979). These linear transmission models generally predict a threshold host density below which parasites cannot persist and a stable equilibrium in which the disease is maintained. In comparison, models that assume some form of non-linear transmission exhibit more complex behaviors including unstable host-parasite equilibrium points, multiple stable equilibrium points, lack of host thresholds in some cases, and limit cycles (May and Anderson 1979, Gabriel et al. 1981, Getz and Pickering 1983, Liu et al. 1986, Hochberg 1991, Briggs and Godfray 1995, Dwyer et al. 1997, McCallum et al. 2001). The sensitivity of the models to transmission functions has implications for maintenance, epidemics and invasion of disease and parasitism. Since

changes will affect parasite transmission, but which phase of transmission is most affected will depend upon the interactions between each host-parasite pair.

I predict that sensitivity of parasite transmission will differ for each host population based on host life history and infection status. For example, snails are long-lived, have low parasite prevalence (1-4%), and their populations are typically abundant, which suggests that increased snail density would have little effect on either the number of parasites that recruit into the host or the number of reproductive stages dispersed from the host. Instead, parasitism in the snail would be most affected by the number of reproductive stages seeking to infect the snail as suggested by studies of similar snail-trematode associations in ponds (Fernandez and Esch 1991) and salt marshes (Sousa 1993). Alternatively, stoneflies are short-lived, more temporally variable in density than either snail or salamander populations and have a high prevalence of infection (40 - 90%) compared to snails. This suggests that stonefly density limits cercarial recruitment and that if the density of stoneflies increased, more cercariae (Fig. 4.1) would be able to find and recruit to stoneflies. The residence time of salamander larvae in streams is between that of the stoneflies and the snails, though the populations are not as temporally variable as populations of the stoneflies (M. Poteet, unpub. data). The prevalence of infection in larval salamanders is between 60 and 80%. Again, this suggests that parasite transmission might be sensitive to changes in salamander density.

To test the sensitivity of parasite transmission to changes in density of each host, I conducted a series of in-stream experiments in which I manipulated densities of each donor and target host to reflect the range of densities found across unlogged and logged streams. The experiments quantitatively test whether parasite transmission of the free-

living stages responds to 1) changes in donor host density, 2) changes in target host density, and 3) simultaneous changes in donor and target host density. In addition, the data revealed the functional response of transmission for each transmission stage across the natural range of host densities.

Based on host life history and parasite prevalence, I predict that 1) parasite transmission to snails will be limited by the reproduction of eggs, but not by the availability of snails, 2) parasite transmission to stoneflies will not be limited by the reproduction of cercariae but will be limited by the availability of stonefly hosts, and 3) parasite transmission to salamanders will be sensitive to availability of infected stoneflies, the trophic response of salamanders to stoneflies, and the density of salamanders. I expect that limitations in reproduction will be manifest as a saturating response of recruited parasite density to donor host density. Limitations in availability of target hosts will be manifest as a saturating response of recruited parasite density to target host density. Additionally, I discuss how changes in host density in response to disturbances from clear-cut logging could force the parasite-host system into alternate states.

MATERIALS AND METHODS

Study System

Species Accounts – The digenetic trematode, *Cephalouterina dicamptodoni*, obligately and sequentially infects larvae of the Pacific Giant salamander, *Dicamptodon tenebrosus*, a common aquatic snail, *Juga silicula*, and nymphs of the stonefly, *Calineuria californica* (Senger and Macy 1953, Anderson et al. 1966) (see Fig. 4.1). Transmission of the

parasite occurs through two free-living stages and one trophic interaction. Eggs produced by the adult parasite transfer out of the salamander host in the feces. The eggs hatch into a miracidium after a snail host eats them. The miracidium transforms into the mother sporocyst that then asexually produces multiple generations of daughter sporocysts. Daughter sporocysts are also asexual and produce multiple generations of the free-swimming cercarial transmission stage. Cercariae emerge from the snail, locate, and actively penetrate the 2nd intermediate stonefly host. Once inside the host, a cercaria encysts to form the metacercaria, a non-feeding larval stage. The life cycle of the parasite is completed when a salamander larva preys upon a stonefly infected by metacercariae. Once inside of the salamander gut, metacercariae excyst, migrate to the intestines and mature to the adult stage. Estimates of fecundity at each stage are unknown for this trematode species. In general, trematodes are exceptionally fecund with estimates for individual reproduction ranging from the thousands for egg or miracidial production into the hundreds of thousands for cercarial production (Dawes 1946, Kennedy 1975).

From 1995 through 1998, I censused the same 4 creeks to determine ambient density and levels of parasitism in each host population. In 1998, I censused host populations from 18 creeks in watersheds with varying severity of logging-induced disturbances (see Chapter 3). Densities of salamander larvae ranged between 0.01 and 3.5 m^{-2} depending on year and creek sampled. Prevalence of infection in salamanders averaged 82% and 57% in creeks from unlogged and logged watersheds respectively.

Of the three host species, *Juga silicula* was most abundant with densities that ranged between 11 and 969 m^{-2} for infected plus uninfected individuals, and between 2

and 20 m^{-2} for infected individuals. Prevalence of infection with *C. dicamptodoni* was relatively low in snails and ranged between 0.27% and 5% in unlogged and logged watersheds respectively. Once infected, these long-lived snails are likely to harbor the parasite for their entire 3-6 year lifespan. Since the snails do not occur in streams above 600m in elevation the parasite cannot complete its life cycle and thus do not infect salamanders of stoneflies above this elevation. This apparent elevational limit in snails, and thus parasites, means that there is a convenient source of uninfected stoneflies and salamanders upstream of this elevation that can be used in the following experiments.

Stonefly nymphs of *C. californica* serve as the 2nd intermediate host to this parasite. Stonefly density ranged between 7 and 77 m^{-2} with prevalence of infection averaging 92% and 38% in unlogged and logged creeks respectively.

Experiments

Since parasite dynamics are determined by successful transmission and host availability, elucidating the functional response of parasite transmission to host density provides the key to understanding the response of parasite populations to disturbance-induced availability of host species. Transmission of parasites by a free-living stage combines the processes of reproduction in and dispersal away from the infected or "donor" host, and location of and recruitment into the subsequent or "target" host. Changes in density of the infected host could thus alter the availability of parasite recruits, whereas changes in the density of target hosts could affect the probability of locating and recruiting to a host. Parasites that transmit through predator-prey interactions will also respond to altered host densities if the trophic functional response is altered as a result of the disturbance. I experimentally tested the response of parasite

transmission to changes in host density in a series of three experiments that were conducted in one stream. In recruitment experiments, I manipulated uninfected target host density to assess whether parasite recruitment responded to host availability. In reproduction experiments, I manipulated donor host density to assess whether parasite recruitment responded to availability of parasite recruits. The third experiment was designed to simultaneously test for the effects of reproduction and recruitment on the functional response of parasite transmission to host density. The experiments were conducted only on the egg and cercarial transmission stages. Target and/or donor host densities were manipulated in in-stream enclosures in various arrays depending on the experiment. Each experiment was replicated in a randomized complete block design. I measured three response variables for each experiment; mean density, total density and prevalence. Mean density was the mean number of parasites that recruited per target host in each replicate divided by the area of the experimental enclosure. Total density was the sum of parasites that recruited to all target hosts per replicate divided by the area of the experimental cage. Prevalence was the proportion of hosts infected. All experiments were completed during the summers of 1995, 1996, and 1997 in Lookout Creek in the H.J. Andrews LTER, Blue River Ranger District, OR.

Recruitment Experiments – Does parasite recruitment respond to changes in target host density and if so, does the direction of the response differ for recruitment of eggs compared to recruitment of cercariae? Parasite recruitment experiments tested the response of parasite recruitment to 3 treatment densities of target hosts. Target hosts were held in enclosures that were replicated across blocks and which were exposed to ambient levels of parasite eggs or cercariae. I tested the response of egg recruitment to

target host density by caging uninfected snails at three treatment densities. All snails were collected from Cone and Lookout creeks in 1995 and Hagan, Lookout, and North Fork Quartz creeks in 1997. Only uninfected snails between 12 and 20mm were used. I determined infection status by placing individual snails in vials of creek water for 48 hours. If no cercariae emerged within that time, snails were considered uninfected. I dissected a subset of 100 snails from those snails considered uninfected, and 1% of the "uninfected" snails had prepatent infections. However, since I randomly assigned snails to each treatment, the low level of infections should not bias treatment results. Snails that did shed cercariae were not used in the experiment.

I tested the response of cercarial recruitment to target host density by caging uninfected stoneflies at three treatment densities. Stoneflies were collected from Mack and McRea creeks in 1995 and Blue River, Lookout and Tidbits creeks in 1997. I used uninfected stoneflies between 13 and 20mm in length. I collected all the stoneflies upstream of snail habitat. Since snails, and thus parasites, do not occur above ca. 600 m, I collected stonefly nymphs at elevations $\geq$ 1000m. I assumed that stoneflies above $\geq$ 1000m would be uninfected. To be certain of this, I randomly subsampled and then dissected 100 stoneflies from those I collected for the experiments. As expected, none of those stoneflies were infected.

Target hosts from all creeks were mixed and then randomly assigned to one of three density treatments. I determined densities for each treatment based on the natural range of host density that I sampled during censuses of snail and stonefly hosts in 1994 and 1995. In 1995, experimental treatments consisted of 20, 40, or 60 uninfected snails for the egg recruitment experiments and 5, 15, or 25 uninfected stoneflies for the cercarial

recruitment experiments (Table 4.1). Experimental cages were constructed of a sewn cylinder of fiberglass windowscreen with 1 mm mesh. The screening was fitted over one end of a cylindrical wire frame of 75 cm diameter (Fig. 4.2). Treatments were randomly assigned within each of 7 blocks that were located in 2 pools of Lookout Creek, with 3 blocks in one pool and 4 blocks in the other. Cages were arrayed as in Fig. 4.2 to decrease parasite transmission among treatment cages (see caption for description of cage array). I placed a constant volume of cobbles inside each cage as an anchor and to provide a natural substrate. Each cage sat on end such that one end was submerged holding the snails or stoneflies and the other end was open above the waterline (Fig. 4.2). Experiments ran for 40 days.

In 1997, I constructed new cages from polyurethane oyster spat bags with a 2 mm mesh size. I used the new cages with the increased mesh size to reduce the effects of the cage on stream velocity. Two mm was the largest mesh size I could use since stoneflies could escape from larger mesh. Each cage was 80 x 33 cm, supported by a cylinder of wire fencing. The ends of the bags were folded and closed at each end by four 3" binder clips. Cages were completely submerged and anchored with a standardized volume of cobbles placed inside each cage (Fig. 4.2). I decreased the numbers of target hosts within each treatment in 1997 because of the smaller cage size so that treatment densities would be similar across years.

Treatments consisted of 12, 27, or 42 uninfected snails for the egg recruitment experiments, and 4, 18, or 12 uninfected stoneflies for the cercarial recruitment experiments (Table 4.2). Treatments were randomly assigned within each of the 6 blocks that were located in 6 pools of Lookout Creek. Experiments ran for 2.5 months. At the

end of each experiment, target hosts were preserved in 70% EtOH. I measured total length of each host, recorded presence of infection, species of parasite, and percent of gonad infected by sporocysts (for snails) or number of metacercariae per host (for stoneflies).

Reproduction Experiments – Does parasite reproduction respond to changes in donor host density and if so, does the direction and strength of those responses differ for egg as compared to cercarial recruitment? Recruitment experiments consisted of donor host density manipulated at 0, low, medium and high densities and target hosts held at low density. I measured the response as the prevalence and density of infections in target host at the end of the experiments. Although this response is a measure of the combined effects of reproduction, dispersal and recruitment of the parasite, I use it as a relative measure of reproductive success of the parasite since only donor host density is manipulated.

I tested the response of egg reproduction to donor host density by caging one of four densities of infected salamanders with one density of uninfected snails (Table 4.1). In 1996, salamanders were collected from Blue River, Mona and Cone creeks. Snails were collected from Lookout, Mona and Cone creeks. In 1997, salamanders were collected from N.F. Quartz, Mona, Florence, Lookout, Tidbits and Cone creeks. Snails were collected from N.F. Quartz, Lookout and Hagan creeks. Snail infection status was determined as described in "*Recruitment Experiments*". I assumed that salamanders were infected since salamanders from those creeks had high levels of infection (M. Poteet unpub. data).

I tested the response of cercarial reproduction to donor host density by caging one of four densities of infected snails with one density of uninfected stoneflies (Table 4.1). In 1996, snails were collected from Lookout, Mona and Cone creeks and stoneflies were collected from Lookout and McRea creeks. In 1997, snails were collected from Hagan, Lookout and N.F. Quartz creeks and stoneflies were collected from Blue River, Lookout and Tidbits creeks. Infection status of stoneflies was determined as described in "*Recruitment Experiments*".

Donor and target hosts from all creeks were mixed and then randomly assigned to one of the 4 density treatments. Cages were set out in a randomized complete block design with 5 replicates in 2 pools of Lookout Creek in 1996 and 6 replicates in 6 pools of Lookout Creek in 1997. In 1996, salamander to snail transmission experiments consisted of 4 treatments in which 0, 1, 2, or 3 infected salamanders were caged with 20 uninfected snails. I supplemented the food supply for salamanders to minimize aggressive behavior and competition for food among the salamanders. I added 10 earthworms per salamander to each cage every week during the experiments. The worms could survive for at least a week in the enclosures, and the salamanders readily ate them (M. Poteet, pers. obs.). Snail to stonefly transmission experiments consisted of 4 treatments in which 0, 4, 8, or 16 infected snails were caged with 10 uninfected stoneflies (Table 4.1). Cages were positioned to decrease cage to cage infection (Fig. 4.2). Experiments ran for 2.5 months.

In 1997, I used the new cage design, as described in "Recruitment Experiments". Densities of all hosts, except for salamanders, were adjusted to reflect the change in cage area. Salamander density could not be decreased in 1997 since the treatment levels for

1996 were 1, 2 or 3 salamanders per cage. Salamander to snail reproduction experiments consisted of 4 treatments in which 0, 1, 2, or 3 infected salamanders were caged with 12 uninfected snails. I supplemented the salamander diet with earthworms as described above. Snail to stonefly transmission experiments also consisted of 4 treatments in which 0, 1, 4, or 7 infected snails were caged with 8 uninfected stoneflies (Table 4.2). I recorded cage depth and relative position to other cages at the beginning of the experiments. Experiments ran for 2.5 months.

At the end of the experiments, target and donor hosts were collected and preserved in 70% EtOH. Target hosts were dissected and parasite presence and intensity were recorded.

Functional Response Experiments – All previously described transmission experiments were designed to test the effects of density changes in one host on parasite transmission and subsequent abundance. There is a strong possibility that changing the densities of both host taxa simultaneously could alter the magnitude and/or functional response of transmission. With this in mind, I designed a set of experiments in 1997 to test the effects of simultaneously altering donor and target host densities on parasite transmission success. The same transmission stages were tested as in the recruitment and reproduction experiments. Each experiment consisted of 9 treatments replicated 6 times with 3 densities each of donor and target hosts in a fully factorial design. A tenth treatment was added as a control to test for background levels of parasite infection in the stream and consisted of 0 donor hosts and the low density of target hosts. For egg transmission, 0, 1, 2, or 3 infected salamanders were caged with 12, 27, or 42 uninfected snails in a fully factorial design. For cercarial transmission, 0, 1, 4, or 7 infected snails and 4, 8, or 12

uninfected stoneflies were added to cages (see Table 4.2 for treatment designations). Experiments ran for 2.5 months. At the end of the experiments, target and donor hosts were collected and preserved in 70% EtOH. Target hosts were dissected and parasite presence and intensity were recorded.

Metacercarial Transmission – It is difficult to experimentally test the effects of host density on metacercarial transmission between stoneflies and salamanders since the metacercarial stage of the parasite is transmitted via a trophic link. Experimental manipulation of salamander and stonefly host density could, in the absence of natural prey availability, artificially alter the functional response of the salamander. If the full suite of natural prey were included in the experiment with the stoneflies, then problems of continuous depletion would occur that could alter relative abundance among the prey and alter the predator functional response. Instead of experimentally assessing metacercarial transmission through manipulations of host and parasite densities, I estimated transmission based on census data from 1998. I used the natural variation in host and parasite densities censused across creeks to assess whether patterns in adult parasite density were associated with either salamander density or metacercarial recruitment density. Data on density of infected salamanders and prevalence and density of adult parasites were generated directly from each of the 18 creeks that were censused in 1998 (see Ch. 3). I estimated the number of metacercariae recruiting to each salamander (E_{ij}, n = 1 to j) within each creek (n = 1 to i) by multiplying the number of stoneflies that I found in the salamander gut (S_{ij}), by the mean prevalence of infection in the stonefly population from that creek (P_i), and by the mean intensity of cysts in the

stonefly population (I_i). Thus metacercarial recruitment was estimated for each salamander from each creek as:

$$E_{ij} = S_{ij} * P_i * I_i.$$

I averaged the estimate of recruitment across all infected salamanders within each creek to get an estimate of mean metacercarial recruitment per creek ($\bar{E}_i$). I conducted an analysis of variance across creeks to assess the effect of metacercarial recruitment ($\bar{E}_i$) and mean infected salamander density on the mean intensity of adult parasites ($\bar{A}_i$).

In effect, the analysis was similar to that from the functional response experiments in that I analyzed the response of parasite density (adult parasites in this case) to variations in donor and target host densities. Although I had no control over host densities in the census data as I did in the experiments, the data did provide additional insight into transmission. The censuses were conducted in creeks from either logged or unlogged watersheds. Logging had a significant effect on the factors that correlate with parasitism (see Ch. 3), so I ran separate ANOVAs across creeks within each forest type. These analyses provided insight into how disturbance affected parasite transmission.

This analysis is a preliminary assessment of the metacercarial transmission behavior and necessarily includes several assumptions pertaining to host and parasite biology. The two major assumptions are that stoneflies are preyed upon independent of their infection status, and that a single temporal measurement of adult parasite intensity will reflect the current availability of metacercariae. Intermediate host behavior can be modified by infection with trematode parasites, presumably to increase the likelihood of transmission (for reviews see Holmes and Bethel 1972, Moore 1987). If metacercariae of *C. dicamptodoni* do modify stonefly behavior such that infected stoneflies are more likely

to be preyed upon, then this model would underestimate the number of metacercariae transmitting to salamanders since metacercarial transmission is based on a constant prevalence of stonefly prevalence per creek. Presumably, any behavioral modification of the stoneflies would not differ across creeks. Thus even if the estimate of transmission is low, the relative estimate of transmission across creeks would still be comparable such that the functional response of transmission should not be affected.

A more fundamental problem is raised by the second assumption that adult parasite intensity will reflect the current level of estimated metacercarial recruitment. Longevity of adult parasites is not known for *C. dicamptodoni*, however adults generally live longer than the larval stages (May and Anderson 1979). Thus, a single measure of adult parasite intensity will reflect multiple generations of metacercarial recruitment and subsequent sorting among adult parasite infrapopulations. A comparison between natural levels of metacercarial and adult intensities at one place and time would not reveal any functional aspect of transmission due to the different time scales over which each parasite stage operates. Data on metacercarial and adult intensity collected over multiple generations of adult parasites in one site would be ideal for evaluating the relationship between the two parasite stages. For most trematode parasites, including *C. dicamptodoni*, this would mean dissecting multiple host individuals over time, which is likely to impact the relationship being evaluated. To compromise between these two sampling regimes, I censused adult and metacercarial intensity in multiple sites during one time period. I average the estimate of metacercarial recruitment and adult parasite density within each creek to obtain an integrated estimate of metacercarial recruitment to salamanders within each creek. Since host and parasite densities vary across each creek,

I use this natural variability to estimate the relationship between metacercarial recruitment and adult parasite intensity, but recognize that interpretations of the data must include cautions regarding the above assumptions.

Statistical Analyses

Experiments – All experimental data that met or could be transformed to meet the assumptions of normality and homogeneity of variance were analyzed with ANOVA for randomized complete block designs. Density data were transformed with either square root or logarithmic transformations. Prevalence data were transformed with the arcsine transformation (for transformations see Krebs 1989). Following ANOVAs, I conducted Tukey multiple comparisons among treatment means (Zar 1984). In addition to Tukey unplanned comparisons, I conducted trend analyses to assess the polynomial relationship between transmission and host densities (Winer et al. 1991).

Donor and target host densities were manipulated in each treatment of the functional response experiments. This precluded analysis of the trends in donor and target hosts simultaneously, as in a factorial experiment, since there was no within block error. In addition, analysis for one trend across all treatments is not biologically meaningful since the response in recruitment to donor and target hosts will be three-dimensional. To analyze trends in the functional response experiments, I divided treatments based on either variation in donor host densities or variation in target host densities. For example, to analyze the trends related to changes in target host density, I conducted three separate trends analyses holding donor host density constant within each analysis. Thus trends in the data related to target hosts were conducted on treatments 1 through 4, 5 through 7, and 8 through 10 respectively (Table 4.2). To analyze trends

related to changes in donor host density, I conducted three separate trends analyses holding target host density within each analysis. Thus trends in data related to donor hosts were conducted on treatments 2,5,8; 3,6,9; and 4,7,10 respectively (Table 4.2).

Blocks 3 and 6 of the 1995 egg and cercarial recruitment experiments respectively were not used in analyses due to outliers. Treatment 1 in block 10 of the 1997 cercarial reproduction experiment was missing. I estimated the missing value following techniques for estimation of missing observations in Sokal and Rohlf (1995, p. 361). Prevalence of infection in the following experiments did not meet the assumptions of normality and homogeneity of variance for parametric tests: 1995 cercarial recruitment, 1997 cercarial recruitment, and 1997 egg reproduction. Differences among means in these three experiments were analyzed nonparametrically with the Friedman's X^2 method for randomized blocks (Sokal and Rohlf 1995).

Metacercarial Transmission – Data were analyzed in separate ANOVAs for creeks in logged and unlogged forests. Each ANOVA tested for differences in levels of adult parasites in response to the density of infected salamanders and the estimated level of metacercarial recruitment. Levels of adult parasites consisted of mean density, total density and prevalence. Mean density was the average number of parasites per infected salamanders per creek area sampled. Total density was the total number of adult parasites in all infected salamanders within the creek per creek sample area. Prevalence was the proportion of salamanders infected in each creek. Recruitment estimates and adult parasite intensity were square root transformed, and parasite prevalence was arcsine transformed to meet the assumptions of normality and homogeneity of variance.

RESULTS

Recruitment experiments – Is parasite recruitment limited by target host density?

Recruitment experiments tested the response in density and prevalence of recruited parasites to density of uninfected target hosts that were exposed to ambient levels of parasite transmission stages. Egg recruitment experiments measured the density and prevalence of sporocysts that established in three densities of snail hosts. Egg recruitment experiments in 1995 showed no effect of snail density on the mean density, total density or prevalence of recruiting parasites (Table 4.3). Even so, mean density of sporocysts decreased in a significant linear trend (Table 4.3, Fig. 4.3) by 49% from low to high snail densities. Prevalence of infection also decreased by 49% from low to high snail densities (Fig. 4.3) in a linear trend (Table 4.3). The large declines in mean density and prevalence suggest that recruitment was limited by availability of eggs. This is further supported by patterns in total density of sporocysts that increased by 41% to an asymptote from low to intermediate snail density (Fig. 4.3). Although 69% of the variation in total density was explained by the quadratic component, the trend was not significant (Table 4.3).

Even though low and intermediate treatment densities of snails were similar between the 1995 and 1997 egg recruitment experiments, the mean and total density of sporocysts that recruited in 1997 was much higher than in 1995 (Fig. 4.3, 4.4). Total density increased significantly between low and high snail densities by 79% (Table 4.4, Fig. 4.4). Where the majority of variance in total density for 1995 was explained by a quadratic trend, in 1997 the trend was significantly linear (Table 4.4). Neither mean density nor prevalence was significantly different across snail density treatments (Table

4.4). Mean sporocyst density did increase by 44% from low to intermediate snail densities then declined at high snail densities to only 26% above that found in the low snail density treatments. Prevalence increased by 46% and 34% respectively from low to intermediate and low to high snail densities (Fig. 4.4). Trend analyses were not significant, although 77% and 66% of the variation among treatments in mean density and prevalence respectively, was accounted for by a quadratic trend (Table 4.4). The magnitude of and significant increase in total density of sporocysts in 1997 suggests that availability of eggs was not limiting in these experiments.

Unlike recruitment of eggs, cercarial recruitment in 1995 increased significantly with stonefly density (Table 4.5). Mean density of metacercariae increased a significant 88% between low and medium stonefly densities. Mean density at the highest stonefly densities was not significantly different from that at either medium or low stonefly densities (Fig. 4.5). The asymptote in mean density was significant as revealed by the quadratic trend (Table 4.5).

Total density of metacercariae increased significantly between stonefly density treatments (Table 4.5). There was no significant difference in total metacercarial density between medium and high stonefly densities (Fig. 4.5) suggesting a decline in the rate of transmission with higher stonefly densities. The linear trend was significant and explained 89% of the variation among treatments (Table 4.5). However a test for nonlinearity was also significant ($F_{(1,10)} = 5.606$, $p = .042$) with the quadratic trend explaining the remaining 10% of the variation (Table 4.5). Thus total density, as with mean density, showed a slight decrease in the rate of transmission of cercariae at high densities of stoneflies.

Although the density of metacercariae increased significantly with stonefly density, prevalence of infection in stoneflies was not significantly affected by host density (Table 4.5, Fig. 4.5). These patterns suggest that the rate of increase in metacercarial density associated with stonefly density is constant.

Unlike experiments from 1995, there were no significant effects of stonefly density on cercarial recruitment in the 1997 experiments (Table 4.6). Although total density of metacercariae did not differ significantly among treatments, metacercarial density increased by 67% from low to high stonefly densities (Fig. 4.6). The trend was linear and significant (Table 4.6). Large block effects during the 1997 experiments likely decreased the power to detect differences among treatment means.

Reproduction Experiments – Is parasite recruitment limited by donor host density? – Reproduction experiments tested the response in density and prevalence of recruited parasites to density of infected donor hosts. Density of infected donor hosts was manipulated as a proxy for manipulating parasite reproduction. Egg reproduction experiments were conducted in 1996 and 1997 to test for the response of sporocyst recruitment to increased density of infected salamanders; a proxy for availability of parasite eggs. Response of sporocyst recruitment to infected salamander density was not statistically significant in either 1996 or 1997 (Table 4.7,8) even though density and prevalence of sporocysts increased by approximately 50% from low to high salamander densities. In 1996, transmission appeared to saturate at higher salamander densities (Fig. 4.7), but the linear trend component explained a significant amount of variation in each measure of parasitism (Table 4.7). A similar, but stronger asymptotic trend occurred in the 1997 experiments in all three measures of parasitism (Fig. 4.8). Although the

quadratic trend explained 67% and 71% of variation in sporocyst mean and total density respectively, the trends were not significant. Ability to detect non-linear trends in the data was probably limited by the significant among block effect. It is interesting to note that the densities of sporocysts that recruited to snails are strikingly similar between 1996 and 1997. If the experimental treatments did not have an effect on the availability of parasite eggs, sporocyst recruitment would be much more variable between years, as was demonstrated in the recruitment experiments. Thus manipulation of infected salamander hosts did successfully manipulate the availability of eggs in the cage environment in relation to the ambient availability of eggs in the environment. Also note that because cage size decreased in 1997, salamander density in the experiments increased from 1996 to 1997 while snail densities remained constant across years (Table 4.1a, 4.2b). Coincident with this increase in salamander density was a change in the amount of variation explained by the trend component. In 1996, 90% of the variation was explained by the linear trend whereas in 1997, 70% of the variation was explained by the quadratic trend. Thus the asymptotic trend in transmission that was suggested in the 1996 experiment was stronger with the increased salamander density in 1997. The rate of transmission of eggs decreases at high densities of infected salamanders. The low prevalence of infection in snails suggests that this trend is not due to a limitation in the availability of snail hosts. However, genetic and geographic heterogeneities in susceptibility of these snails to parasitism have not been investigated and could account for the low prevalence (e.g., Anderson 1978, Morand et al. 1996, Webster and Woolhouse 1998, Lively 1999, Knight et al. 1999).

Cercarial reproduction experiments were conducted in 1996 and 1997 to test for the response of metacercarial recruitment in stoneflies to increased availability of cercariae. Cercarial availability was manipulated by altering the density of infected snails which were a proxy for cercarial reproduction. The snails were placed in enclosures with a constant density of stoneflies. In 1996, neither mean nor total density of metacercariae that recruited to stoneflies changed significantly in response to infected snail density (Table 4.9). Although no significant trends were detected in the data, 90% and 72% of the variation in mean and total density respectively were explained by the combined variation in the quadratic and cubic trends components (Table 4.9). Tests for nonlinearity in these measures were not significant ($F_{(\text{mean density})} = 3.60$, $df = 1,12$, $p = 0.064$; $F_{(\text{total density})} = 0.921$, $df = 1,12$, $p = 0.390$).

Even though density of recruiting metacercariae was not affected, the proportion of infected stoneflies increased with infected snail density (Table 4.9, Fig. 4.9). The effect was attributed to a significant difference in prevalence between snail densities of 18 and 36 m^{-2}. In addition to the difference among treatment means, the response of parasite prevalence to snail density was significantly non-linear ($F_{(1,12)} = 4.926$, $p = 0.029$) (Fig. 4.9), with the cubic trend explaining 75% of the variation among treatments (Table 4.9).

The pattern of response in metacercarial density to infected snail density in 1997 was strikingly different from that in 1996 (Fig. 4.10), but there were no significant treatment effects (Table 4.10). Even so, mean density responded to snail density in a significant non-linear trend ($F_{(1,12)} = 3.815$, $p = 0.053$). The significant and increasing quadratic component explained 85% of the variability among treatments (Table 4.10) and

showed no sign of reaching an asymptote. Even though the quadratic component explained 77% of variation among treatments of total density (Table 4.10), there were no significant non-linear trends ($F_{(1,12)} = 1.113$, $p = 0.342$). Density of metacercarial recruits was higher in 1997 than in 1996, even though infected snail densities were lower. This probably reflects the increased stonefly density used in the 1997 experiments but does not explain the striking difference in transmission patterns between years (Table 4.1b, 4.2b).

Functional Response Experiments – Egg transmission. Egg transmission experiments tested the response of sporocyst density and prevalence in snails to changes in densities of infected salamander and uninfected snail hosts. The establishment of sporocysts in snails is the outcome of successful reproduction, dispersal and recruitment of eggs from salamanders to snails, so it provides a simultaneous evaluation of reproduction and recruitment. The mean density of sporocysts recruiting to snails did not respond significantly to densities of either salamander or snail hosts (Table 4.11), even though overall mean density of sporocysts declined by 43% in response to increasing levels of salamanders (Fig. 4.11a). This was reflected in the significant linear trend associated with changes in salamander density at high densities of snails (Table 4.12a). Mean sporocyst density at intermediate snail densities decreased by approximately 50% from that recruited to high and low snail densities (Fig. 4.11a). This was reflected in the significant quadratic trend associated with changes in snail density at low salamander densities (Table 4.13).

Total density of sporocysts responded significantly to salamander and snail hosts (Table 4.11), and the direction of the response switched between the two hosts (Fig. 4.11b). Total density of sporocysts increased significantly between the lowest and

highest snail density treatments (Table 4.11, Fig. 4.11b). The increase associated with snail densities was exponential at low salamander densities (Table 4.13b). While total density increased with availability of target hosts, it decreased with increasing salamander, or donor host density (Fig. 4.11b). Tukey multiple comparisons revealed a significant decrease in sporocyst total density at the highest levels of salamander density compared to both low and intermediate levels of salamander density (Fig. 4.11b). This decrease in total density across salamander densities was linear at high snail densities (Table 4.12b). The functional response in transmission showed consistent trends in both mean and total densities. Transmission of eggs responded in a non-linear fashion with snail density and linearly with salamander density.

Prevalence of infection was significantly affected by salamander host density but not by snail host density (Table 4.11). Tukey multiple comparisons found no significant difference among paired treatment means. However the response to salamander density was significantly non-linear and hump-shaped with respect to snail densities (Table 4.12, Fig. 4.11c). At high snail densities, prevalence declined linearly with increasing salamander density. In addition, prevalence responded to snail densities in a significant non-linear fashion at low densities of salamanders (Table 4.13c, Fig. 4.11c).

Functional Response experiments – Cercarial transmission. Cercarial transmission experiments tested the response of metacercarial density and prevalence in stoneflies to changes in densities of infected snail and uninfected stonefly hosts. Cercarial transmission experiments provide a simultaneous evaluation of transmission response to reproduction from the snail and recruitment to the stonefly. Unlike egg transmission, cercarial transmission responded positively to densities of snails and stoneflies (Fig.

4.12). ANOVA did not detect any effect of target or donor host densities on the mean density of metacercariae recruited to stoneflies (Table 4.14a,b). There was a very strong block effect that limited the power of the analysis (Table 4.14a,b), but trends analyses provided insight into the response of cercarial transmission to donor and target hosts. Mean metacercarial density saturated at high densities of snails and stoneflies (Fig. 4.12a) as revealed by the significant quadratic trend (Table 4.15a). At the lowest stonefly density, the majority of variation in mean metacercarial density associated with changing snail density was explained by the linear component. Mean metacercarial density increased by 30% and 40% from the low and intermediate snail densities to the highest snail density. At intermediate stonefly densities, the majority of variation in mean metacercarial density was explained by the quadratic component. Mean metacercarial density increased by 7.5% and 34% from the low and intermediate snail densities to the highest snail density. Since neither of these trends was significant, it suggests that cercarial transmission is not strongly affected by reproduction unless stonefly density is high.

Mean metacercarial density did not respond significantly to stonefly density (Table 4.14a). The proportion of variance in transmission explained by quadratic and linear trends did switch between low and intermediate snail densities (Table 4.16a, Fig. 4.12a).

The relative effects of snail and stonefly hosts on the response of cercarial transmission were more apparent for total density of recruited metacercariae than for mean density. Total density of metacercariae that recruited to stoneflies was significantly and positively affected by snail and stonefly density (Table 4.14b). Tukey multiple

comparison tests could not detect significant differences between means associated with snail density treatments. There were no trend effects associated with snail densities (Table 4.15b), although the largest proportion of variation in trends was explained by the quadratic component for different snail densities at intermediate and high stonefly densities. Indeed, at high stonefly densities, the total density of metacercariae increased by 45% from the lowest to the highest density of infected snails (Fig. 4.12b).

Stonefly density had a significant effect on total metacercarial density (Table 4.14). The effect was significant between low and intermediate, and low and high stonefly densities, but not between intermediate and high stonefly densities. The response of total metacercarial recruitment to stonefly density was significantly linear at intermediate and high snail densities (Table 4.16b). These patterns suggest that cercarial transmission is significantly limited by stonefly density when infected snail density is high. However, the rate of recruitment of metacercariae into stoneflies differs in response to levels of snail density. At low snail densities, total density of metacercariae increased 57% from low to high stonefly densities (Fig. 4.12b). At intermediate snail densities, total density of metacercariae increased 85% from low to high stonefly densities. Whereas at high snail densities, total density of metacercariae increased 73% from low to high stonefly densities (Fig. 4.12b). These patterns show that cercarial transmission does respond significantly to stonefly density, but the level of the response is dependent upon the density of snail hosts.

Although mean and total density responded positively to stonefly and snail host densities, prevalence of infection in stoneflies was high across all experimental

treatments (Fig. 4.12c) and did not respond significantly to density of either host (Table 4.14).

Metacercarial Transmission – The response of adult parasites to metacercarial recruitment and salamander density differed between forest types (Table 4.17a,b). In unlogged creeks, neither mean density nor parasite prevalence in adult parasites responded significantly to salamander density or metacercarial recruitment (Table 4.17a). Total adult parasite density increased significantly with salamander density and decreased with metacercarial recruitment (Table 4.17a). The nearly significant interaction term between salamander density and metacercarial recruitment reflected the different directions of response that each elicited in adult parasite density (Fig. 4.13).

Logged creeks had fewer metacercarial recruits ($t = -2.529$, $df = 16$, $p = 0.022$), total adult parasites ($t = -2.361$, $df = 16$, $p = 0.031$) (Fig. 4.14), and infected salamanders ($t = -2.217$, $df = 16$, $p = 0.041$) than unlogged creeks. Mean adult density and parasite prevalence decreased in logged as compared to unlogged creeks, but not significantly ($t_{(\text{mean adults})} = -1.894$, $df = 16$, $p = 0.076$, $t_{(\text{prevalence})} = -1.807$, $df = 16$, $p = 0.090$) (Fig. 4.13, 4.15). In addition to an overall decrease in parasites and hosts, transmission of metacercariae was not as strongly affected by metacercarial recruits as it was by the density of infected salamanders (Table 4.17b). Both mean and total density of adult parasites responded significantly to density of infected salamanders. The general three dimensional shape of the transmission function did not appear to differ significantly between logged and unlogged creeks (Fig. 4.13-4.15), but the effect of salamanders in logged creeks appears to have a much stronger effect on adult parasite recruitment than does the estimate of metacercarial recruitment.

DISCUSSION

In the following discussion, I explore the results in light of each prediction I made in the introduction.

Prediction 1: parasite transmission to snails will not be limited by the availability of snails but will be limited by the reproduction of eggs. If transmission of eggs is not limited by snail density, then neither sporocyst mean density nor prevalence should increase in response to snail density in the recruitment experiments. In 1995, trends in the recruitment experiment support the prediction that snail density does not limit parasite recruitment. Indeed, as snail density increases, mean sporocyst density and prevalence tend to decrease and total sporocyst density asymptotes. These data can clearly not be effectively modelled with mass-action transmission. Mass action transmission models predict that increased snail density coupled with a constant density of parasite eggs should lead to an overall decrease in the mean density of sporocysts per snail with a concomitant increase in sporocyst prevalence to unity (Anderson 1978, Wilson and Taylor 1978). Thus, based on the mass action models, total sporocyst density should be directly proportional to snail density. This is not the case for my experiments. Mass action models also provide that an increase in snail density exposed for a particular amount of time will have the same effect on transmission as a constant snail density exposed for a longer amount of time (Anderson 1978). This is true since mass action models assume that transmission behavior is not affected by host or parasite density. This is not the case for transmission of eggs in these experiments.

The patterns in sporocyst densities and prevalence in 1995 suggest that increased snail density is associated with a decline not only in the total sporocyst density, but also

in the rate of parasite transmission. Transmission rate is defined here as the density of new infections over the duration of the experiments (See McCallum et al. 2001). For a constant transmission rate, the total density of infections should increase linearly, the mean density of infections should remain constant and the prevalence of infection should rise to unity. Transmission rate will be sensitive to a variety of factors, including, for this phase of transmission, availability of eggs. If eggs are limiting transmission at high snail densities, then the rate of transmission would decline with increasing snail density.

Transmission rates of free-living *Schistosoma mansoni* miracidiae to the snail host, *Biomphalaria glabrata* (Carter et al. 1982), declined in a similar manner to the transmission of eggs for *C. dicamptodoni*. Although no experimental evidence existed to explain the lower transmission rate, Carter et al. (1982) suggested that increased snail density impacted miracidial searching efficiency resulting in a decline in transmission rate. Since sporocyst density was not measured, it is not known whether the decline in parasite prevalence represents an overall decline in transmission rate, or merely an increase in parasite aggregation associated with an overall increase in total sporocyst density. The latter is most likely to be the case given that snails release a water-soluble substance that attracts miracidiae (Chernin 1970) and more recent work shows a strong effect of host spatial aggregation on trematode infections (Grosholz 1994). Thus the results of Carter et al. (1982), that transmission declines with snail density, is likely due to increased aggregation and not a decrease in transmission. In the transmission experiment I conducted, the combined evidence of saturation in total sporocyst density and decline in prevalence provide a clear indication that transmission rate declines with snail density. This suggests three hypotheses: 1) availability of parasite eggs limits

transmission of the parasite to snails, or 2) mutual interference among snails at high densities causes a decline in per capita number of parasite eggs consumed and thus transmitted (e.g., Hassell and Varley 1969, Hassell 1971, Beddington 1975, Sih et al. 1985). In the following text, I discuss these hypotheses in light of evidence from the remaining experiments.

The 1997 recruitment experiment results conflict with those from the 1995 experiments in that in 1997 total sporocyst density increases linearly and significantly with snail density as is predicted by mass action models. Sporocyst prevalence and mean density increase, albeit nonsignificantly, with snail density and both metrics trend toward saturation (Table 4.4, Fig. 4.4). Increased sporocyst mean density and prevalence suggest that an increase in snails translates into a higher rate of parasite transmission; thus snails would be considered limiting to sporocyst density. Increases in sporocyst prevalence in response to snails were also found in laboratory experiments (Kuris 1973, Anderson 1978, Anderson and May 1979, Carter et al. 1982) except that the increase in prevalence was accompanied by a decrease in mean density of sporocysts.

The density of sporocysts that recruited in 1997 was more than twice that in 1995. This might be explained by increased ambient levels of infected salamanders between 1995 and 1997, which could result in higher availability of eggs to the cages. This is unlikely considering the severe scouring floods that occurred in 1996, which tended to have a negative impact on the local aquatic fauna (Ch. 3, M. Poteet, unpub. data). It is more likely that the high density of sporocysts in 1997 was due to the increased mesh size of the enclosures. The larger mesh size was associated with an increased water velocity within the enclosures. The higher velocity would be more likely to sweep eggs into the

enclosures and would pass a higher volume of water through the enclosures thus increasing the probability of moving eggs into the cage. Whatever the mechanism, the availability of eggs in recruitment enclosures was much higher in 1997 than in 1995. Also note that the treatment levels were similar in 1995 and 1997 for low host densities, but lower in 1995 than 1997 for high host densities (Fig. 4.3, 4.4). If hypothesis 2 is correct, that high densities alter snail behavior and cause a decline in transmission, then the strongest negative impact of snail density on parasite transmission should be seen in 1997. This is obviously not the case and mutual interference among snails is unlikely to operate when egg densities are high. Instead, sporocyst density and egg transmission rates appear to increase at low snail densities in 1997, suggesting that when egg density is high, low snail densities limit parasite transmission. There is a striking similarity in total sporocyst density between years at the lowest snail density. At higher snail densities, the density of sporocysts rises much faster in 1997, when egg availability was apparently high, than in 1995. These patterns provide further support that low densities of snails limit parasite transmission when parasite densities are high. Since it appears that limitation in transmission switches depending on availability of eggs, these data cannot refute the hypothesis that mutual interference may occur when egg densities are low.

The data from 1997 suggest another hypothesis that 3) susceptibility of snails to infection is heterogeneous. Even though the maximum mean and total sporocyst densities increase respectively by 63% and 55% in 1997, sporocyst prevalence increases by only 27% (Fig. 4.3, 4.4) and is still quite low with only 9% of snails infected. This suggests that even when eggs are not limiting, the proportion of snails infected will never reach unity. Heterogeneity in susceptibility to infection is common in snail hosts and

arises from genetic factors, spatial distribution, and behavior (Lie et al. 1975, Anderson 1978, Grosholz 1994, Morand et al. 1996, Webster and Woolhouse 1998, Lively 1999, Knight et al. 1999). Further study of heterogeneity in these snail hosts is necessary to understand the mechanisms driving transmission.

Although the recruitment experiments *suggested* that availability of parasite eggs could limit transmission, reproduction experiments specifically *tested* for effects of egg production on transmission in 1996 and 1997. Sporocyst densities and prevalence in 1996 did increase linearly with density of infected salamanders. The relationship was not statistically significant even though mean and total sporocyst densities respectively increased 3 and 4 fold with salamander density and sporocyst prevalence increased 5 fold with salamander densities (Fig. 4.7). Results in 1997 were similar but with a more pronounced saturation in transmission at the highest salamander densities (Fig. 4.7, 4.8). The magnitude and pattern of increase in sporocysts were strikingly similar across years. Data from both years tended to asymptote between 6 and 8 salamanders per m^2 and a combined graph of data across years reveals saturation in each term around 7 salamanders per m^2 (Fig. 4.16). Patterns in the data from both years suggest that transmission is linear at low densities of salamanders since the total density of sporocysts increases linearly. Declines in the total density and prevalence of infection at high salamander densities suggest a decline in the rate of egg transmission. Saturation in egg transmission at high salamander densities suggests two more hypotheses: 4) saturation in the functional response of snails to parasite eggs at high densities of eggs leading to a decrease in parasite transmission (Type II functional response, sensu Holling 1959) or 5) production of parasite eggs decreases at high salamander densities due to competition

among salamanders. As for the type of competition, I cannot definitively state that interference competition is more important than exploitation competition. However, since I did supplement food for the salamanders each week, and since these salamanders are aggressively territorial and cannibalistic (Parker 1992), I suggest that interference competition would be more significant than would exploitation competition.

Competitive interference at high densities would translate into less time foraging and more time fighting which could negatively impact the production and distribution of eggs by adult parasites. However, if that was the case, then an increase in salamanders should translate into an inverse relationship between salamander and sporocyst density; the opposite trend was observed.

The evidence points toward saturation in egg predation by snails. Even though salamander density in the treatments increased from 1996 to 1997, snail density remained constant at $45 \cdot m^{-2}$. At the same time, the density and prevalence of sporocysts that recruited to snails remained strikingly similar across years, even though densities of sporocysts in the 1997 recruitment experiments clearly showed that there were more eggs available for transmission in the enclosures. However, the high sporocyst densities in the 1997 recruitment experiments were only achieved at high snail densities whereas transmission was limited at low snail densities ($45 \cdot m^{-2}$). Since the reproduction experiments, which held stonefly density constant while varying snail density, tested the transmission response of increased egg production at only the lowest snail density, the experiment was limited and could not assess the full response range of transmission to increased egg densities. However, at low snail densities and high egg production, it appears that hypothesis 1 is incorrect and hypothesis 4 is correct such that snail density

does limit parasite transmission. Again, the assumption of mass action transmission could not accurately model this relationship.

Transmission experiments in 1997 assessed the functional behavior in transmission over the full range of ambient donor and target host densities with surprising results. As salamander density increases, the numerical response in transmission declines across all snail densities while the functional response of transmission to snail density becomes more linear. The trend of increase in transmission with snail density is in general agreement with results from the 1997 recruitment studies, except for the lack of saturation at high snail densities. Instead of saturating at high snail densities, the rate of transmission across snail densities increases significantly at low salamander densities and remains constant at high salamander densities. Since sporocyst density declines significantly with salamander density, then the lowest salamander density in these experiments appears to represent the highest availability of parasite eggs. Thus transmission of parasites at high egg densities responds significantly and non-linearly to snail density.

The decline in sporocysts with salamander density might partly be explained by saturation of the snail predators at high egg densities. But if snail saturation is the only mechanism driving the decline in sporocyst density, then the mean and prevalence of sporocysts at each snail density should remain constant while the total density of sporocysts should continue to increase. Instead, there is a significant inverse relationship between salamander density and sporocyst density. If the inverse relationship was limited to response in total density alone, this pattern could be explained by variation in susceptibility of snail hosts (Anderson 1978). However, sporocyst prevalence is also

inversely related to salamander density, so the rate of transmission also declines. If heterogeneity were the only factor driving declines, then total sporocyst density should increase and prevalence should plateau. One explanation for lower transmission at higher salamander densities is competitive interference among salamanders at high densities; hypothesis 5. Interestingly, the inverse relationship between salamander and sporocyst densities is most apparent at high snail densities when egg availability is most likely to limit transmission. Thus egg transmission is a complex and non-linear function of the relative abundance of salamander and snail densities.

Prediction 2: cercarial transmission will not be limited by reproduction from snails, but will be limited by the availability of stonefly hosts. Cercarial transmission is directly proportional to stonefly density in both years in metacercarial recruitment experiments. The trends in total metacercarial transmission were similar between 1995 and 1997. However mean density and prevalence in 1995 were asymptotic and quite low compared to mean density and prevalence in 1997. The significant quadratic form in mean density coupled with the asymptotic prevalence suggests that cercarial transmission declines with snail density in 1995. This is not the case in 1997, where total density and prevalence increase with snail density. The low density of metacercariae that recruited to stoneflies could cause the patterns in 1995 or they might be the result of heterogeneities in host susceptibility. The low metacercarial densities could reflect a very limited pool of cercariae possibly due to the small mesh size of the enclosures, as suspected in the recruitment experiments. Extremely low parasite densities tend to cause under-dispersion at high host densities due to the limited availability of parasites (Anderson et al. 1978b). On the other hand, the low prevalence in 1995 could also be driven by host heterogeneity

in susceptibility (Anderson et al. 1978b) possibly related to behavioral differences in habitat use or foraging at high stonefly densities. However, behavioral heterogeneities do not explain the extremely low metacercarial densities in 1995 as compared to 1997. Genotypic differences among stoneflies between years cannot be ruled out as a factor that drives both heterogeneity to susceptibility (e.g. Grosholz 1994, Knight et al. 1999) and the low level of metacercarial recruitment. Stoneflies used in the 1995 experiments were collected from two 1st order creeks in the Lookout Creek drainage. Neither of these creeks supports snails along their entire length. Stoneflies used in the 1997 experiments were collected from three 3rd order creeks, all of which support snails downstream from where stoneflies were collected. The naiveté of and the probable lower genetic diversity in stoneflies used during the 1995 experiments could explain the low metacercarial recruitment (e.g. Lively 1989). However, since resistance to parasitism is heritable in certain species (e.g. Grosholz 1994), it could also be argued that the naïve hosts could be more susceptible to parasitism. Whatever the mechanism, cercarial transmission is directly proportional to stonefly density across a wide range of ambient densities. Thus linear transmission models would be able to accurately predict cercarial transmission for ambient stonefly densities.

I predicted that cercarial transmission would respond to stonefly densities, but not to snail densities. Cercarial transmission, however, does respond to snail density and with very complex behavior. The rate of transmission in 1996 increases from ambient to low snail densities, decreases at intermediate densities and increases again at high snail densities. This complex response does not occur in 1997 where metacercarial density is directly proportional to density of infected snails. The measurable differences between

1996 and 1997 could explain the variation in response. Once again, metacercarial densities in 1997 were markedly higher than in 1996. As described earlier, the decline in metacercariae could be due to a cage effect, or possibly to genetically controlled heterogeneities in susceptibility. The cage effect argument is unlikely to be valid in reproduction experiments except for treatments in which no snails were added. If the mesh size of the cage did have an effect on cercarial availability in treatments with infected snails added, the cage in 1996 would be more likely to sequester cercariae and transmission should be higher than in 1997. This is not the case. In addition, stonefly densities were lower in the 1996 experiments than in the 1997 experiments, so any behavioral effect due to crowding would be manifest first in the 1997 experiments. The only other measured difference between treatments in 1996 and 1997 is metacercarial dispersion. In 1996, metacercarial dispersion was constant across treatments (constant variance to mean ratio), whereas in 1997, metacercariae became more under-dispersed with increases in snail density (decreasing variance to mean ratio). Again, this pattern is significant for interpreting results in 1997 only and suggests that the non-significance of the large treatment effects across snail densities was due to the rate of increase in variance compared to the mean (see Anderson et al. 1978a).

Another possible explanation is based on the differing levels of infected snails and uninfected stoneflies across years. In 1996, snail treatment densities were higher than in 1997, but the stonefly density was lower. If the functional response of transmission is dependent on the relative densities of infected snails to uninfected stoneflies, then interpretations of the response based on two snapshots of relative donor to target abundance are likely to prove futile. Indeed, the cercarial transmission experiments, in

which snail and stonefly host densities were simultaneously manipulated, resulted in very similar patterns to both the 1996 and the 1997 reproduction experiments. A trough in transmission occurs from low snail/high stonefly densities to intermediate snail/low stonefly densities (Fig. 4.12). This trough corresponds with densities in the 1996 experiments that elicited low metacercarial prevalence. Peaks above this trough are associated with 1) high stonefly densities at intermediate and high snail densities, and 2) low and intermediate stonefly densities at low snail densities (Fig. 4.12).

These patterns suggest that transmission is limited by the reproduction of cercariae from snails only at high stonefly densities. Thus the response of transmission to reproduction in 1997, which varied snail densities at one high stonefly density. At low snail densities, cercarial transmission is limited by density of stoneflies. At high stonefly and high snail densities, cercarial transmission saturates. The combined interactions between snail and stonefly densities can explain the complex patterns in the data without invoking complex hypotheses of heterogeneities in host genotype or behavior.

Prediction 3: metacercarial transmission will be sensitive to availability of infected stoneflies, the functional trophic response of salamanders, and salamander density. Densities of adult parasites respond differentially to salamander and metacercarial availability and the strength of the response depends on the relative abundance of each. At low salamander densities, adult parasite densities increase significantly in response to salamanders and not metacercarial recruitment. At high salamander densities, adult parasites are directly related to salamander density, but inversely related to metacercarial recruitment which results in an overall decline in adult parasite density at high salamander, high recruitment densities. Since logged creeks have significantly fewer

salamanders and less per capita recruitment than unlogged creeks, metacercarial transmission is limited by different mechanisms depending on forest type. In logged creeks, metacercarial transmission is limited by salamander density. In unlogged creeks, metacercarial transmission is limited by salamander density, while it appears to be decreased by metacercarial recruitment. Because of the differential timing and effects of salamander density and metacercarial recruitment on transmission, mass action models would not be able to effectively model transmission of the metacercarial stage.

CONCLUSION

Transmission of *C. dicamptodoni* is not a simple mass action process. Non-linearities in transmission exist in at least two of the three transmission stages. In addition, transmission is context dependent in that limitations in transmission depend upon reproduction and recruitment differentially but in a predictable manner based on relative densities of donor and target hosts.

Non-linear transmission generates complex model behavior resulting in multiple stable states (Anderson and May 1978, Getz and Pickering 1983, Liu et al. 1986, Briggs and Godfray 1995). If a parasite-host system is subject to large disturbances, the system could be forced into a different state (May and Anderson 1979). This potential outcome is especially significant to this system in light of the prevalent source of frequent and intense anthropogenic disturbances: clear-cut logging. Indeed, logging does have a significant and differential negative effect on host density (Ch. 3). Based on the results provided here, the relative change in host densities could alter the functional response of transmission, thus forcing the system into an alternate, disturbance-induced state. For

example, logging in these creeks negatively affects snail and stonefly densities (Ch. 3). Referring to the functional response results for cercarial transmission (Fig. 4.12), one can develop *hypotheses* about how parasite densities will change based on the three apparent equilibrium points in parasite density. One potential equilibrium occurs in a trough at low metacercarial densities that occurs from low snail/high stonefly densities to intermediate snail/low stonefly densities. Because this equilibrium is in a trough within the functional response plane, it suggests that parasites in this state would be resistant to disturbance (Connell and Sousa 1983) such that the force of disturbance required to move the parasite-host system out of the "trough" state and up the functional response plane would necessarily be large. An unstable equilibrium occurs at high stonefly, high snail densities, which leads to high densities of metacercariae. This suggests that the force of disturbance required to move the parasite-host system from this state, would be lower than that in the "trough". Another unstable equilibrium occurs at low snail/intermediate stonefly densities. If the parasite-host system is in the upper, unstable equilibrium, then a decline in snail density would precipitate a dramatic decline in parasites that would cascade through the parasite host system. This would be the case for Jones and similar creeks (Table 4.18), which support very high snail, stonefly and metacercarial densities. However, if the parasite-host system already existed in the "trough" equilibrium area, then any disturbance-induced change in snail densities would be unlikely to have large effects on the parasite-host system. This would be the case for Hagan and similar creeks (Table 4.18), which support very low densities of infected snails, high densities of stoneflies, and low densities of metacercariae. However, if infected snail density is low and stonefly density is intermediate, and change in snail or stonefly density could

precipitate a large change in the parasite-host system. This is the case for both debris flow creeks that support snails and it suggests that parasite populations could be made unstable following severe disturbances (Table 4.18). Thus it appears that altering host densities and causing changes in the transmission function, can cause this parasite-host system to shift into alternate states.

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Table 4.1 Treatment levels of target and donor hosts for experiments conducted in 1995 and 1996. Tables present the absolute numbers (#) and the densities (#/m^2) of hosts at three treatment levels. a) Levels of snails and stoneflies used in egg and cercarial recruitment experiments respectively. b) Levels of infected donor and uninfected target hosts used in egg and cercarial reproduction experiments in 1996. Enclosure area = 0.442 m^2.

a. Recruitment Experiments, 1995

	Egg		**Cercarial**	
	Snail		Stonefly	
Treatment	#	#/m^2	#	#/m^2
1	20	45	5	11
2	40	90	15	34
3	60	136	25	57

b. Reproduction Experiments, 1996

	Egg Reproduction				**Cercarial Reproduction**			
	Salamander		Snail		Snail		Stonefly	
Treatment	#	#/m^2	#	#/m^2	#	#/m^2	#	#/m^2
1	0	0	20	45	0	0	10	23
2	1	2	20	45	4	9	10	23
3	2	5	20	45	8	18	10	23
4	3	7	20	45	16	36	10	23

Table 4.2 Treatment levels of target and donor hosts for experiments conducted in 1997. Tables present the absolute numbers (#) and the densities (# / m^2) of hosts at each treatment level. a) 1997 recruitment experiments. b) 1997 reproduction experiments, c) 1997 functional response experiments.

a. Recruitment Experiments

	Egg		**Cercarial**	
	Snail		Stonefly	
Treatment	#	#/m^2	#	#/m^2
1	12	45	4	15
2	27	102	8	30
3	42	159	12	45

b. Reproduction Experiments

	Egg Reproduction				**Cercarial Reproduction**			
	Salamander		Snail		Snail		Stonefly	
Treatment	#	#/m^2	#	#/m^2	#	#/m^2	#	#/m^2
1	0	0	12	45	0	0	8	30
2	1	4	12	45	1	4	8	30
3	2	8	12	45	4	15	8	30
4	3	11	12	45	7	27	8	30

c. Reproduction combined with recruitment

	Egg Transmission				**Cercarial Transmission**			
	Salamander		Snail		Snail		Stonefly	
Treatment	#	#/m^2	#	#/m^2	#	#/m^2	#	#/m^2
1	0	0	12	45	0	0	4	15
2	1	4	12	45	1	4	4	15
3	1	4	27	102	1	4	8	30
4	1	4	42	159	1	4	12	45
5	2	8	12	45	4	15	4	15
6	2	8	27	102	4	15	8	30
7	2	8	42	159	4	15	12	45
8	3	11	12	45	7	27	4	15
9	3	11	27	102	7	27	8	30
10	3	11	42	159	7	27	12	45

Table 4.3 Results of egg recruitment experiments from 1995 for sporocyst mean density, sporocyst total density and sporocyt prevalence. Trend analyses partitioned the treatment SS into linear and quadratic components. The proportion of the treatment SS explained by each trend is listed in the column %SS Explained.

Mean Density	*df*	SS	% SS Explained	MS	F	p
Block	5	6.958		1.392	0.349	0.871
Treatment	2	22.007		11.004	2.759	0.111
Linear	1	19.156	87.04	19.156	4.804	0.055
Quadratic	1	2.851	12.96	2.851	0.715	0.444
Error	10	39.878		3.988		

Total Density	*df*	SS	% SS Explained	MS	F	p
Block	5	601.893		120.379	1.024	0.453
Treatment	2	470.012		235.006	2.000	0.186
Linear	1	145.376	30.93	145.376	1.237	0.312
Quadratic	1	324.636	69.07	324.636	2.762	0.144
Error	10	1175.288		117.529		

Prevalence	*df*	SS	% SS Explained	MS	F	p
Block	5	0.0034		0.0007	0.1938	0.958
Treatment	2	0.0166		0.0083	2.3446	0.146
Linear	1	0.01659	99.94	0.017	4.686	0.058
Quadratic	1	0.00001	0.09	0.000	0.004	
Error	10	0.0354		0.0035		

Table 4.4 Results of egg recruitment experiments from 1997 for sporocyst mean density, sporocyst total density and sporocyst prevalence. Analyses of trend are reported for linear and quadratic polynomials. "%SS Explained" reports the proportion of treatment SS explained by each trend..

Mean Density	*df*	SS	% SS Explained	MS	F	p
Block	5	108.974		21.795	1.255	0.377
Treatment	2	24.477		12.239	0.705	0.521
Linear	1	5.416	22.13	5.416	0.312	0.617
Quadratic	1	19.061	77.87	19.061	1.098	0.413
Error	10	173.650		17.365		

Total Density	*df*	SS	% SS Explained	MS	F	p
Block	5	2020.080		404.016	1.160	0.413
Treatment	2	3985.849		1992.925	5.722	0.023
Linear	1	3329.809	83.54	3329.809	9.561	0.012
Quadratic	1	656.040	16.46	656.040	1.884	0.218
Error	10	3482.627		348.263		

Prevalence	*df*	SS	% SS Explained	MS	F	p
Block	5	0.108		0.022	1.521	0.276
Treatment	2	0.023		0.012	0.810	0.477
Linear	1	0.008	35.43	0.008	0.574	0.479
Quadratic	1	0.015	66.21	0.015	1.072	0.354
Error	10	0.142		0.014		

Table 4.5 Results of cercarial recruitment experiments from 1995 for metacercarial mean density, total density and prevalence. Analyses of trend are reported for linear and quadratic polynomials. "%SS Explained" reports the proportion of treatment SS explained by each trend..

Mean Density	*df*	**SS**	**% SS Explained**	**MS**	**F**	**p**
Block	5	0.701		0.140	1.277	0.346
Treatment	2	0.987		0.493	4.492	0.040
Linear	1	0.414	41.97	0.414	3.770	0.086
Quadratic	1	0.573	58.03	0.573	5.214	0.046
Error	10	1.098		0.110		

Total Density	*df*		**% SS Explained**	**MS**	**F**	**p**
Block	5	1.034		0.207	0.351	0.870
Treatment	2	31.664		15.832	26.866	0.000
Linear	1	28.361	89.57	28.361	48.126	0.000
Quadratic	1	3.303	10.43	3.303	5.605	0.042
Error	10	5.893		0.589		

Prevalence	*df*		**% SS Explained**	**MS**	**F**	**p**
Block	5	0.3440		0.0688	0.7739	0.589
Treatment	2	0.4940		0.2470	2.7784	0.110
Linear	1	0.176	35.59	0.176	1.978	0.210
Quadratic	1	0.318	64.35	0.318	3.576	0.092
Error	10	0.8890		0.0889		

Table 4.6 Results of cercarial recruitment experiments from 1997 for metacercarial mean density, total density, and prevalence. Analyses of trend are reported for linear and quadratic polynomials. "%SS Explained" reports the proportion of treatment SS explained by each trend..

Mean Density	***df***	**SS**	**% SS Explained**	**MS**	**F**	**p**
Block	5	18.296		3.659	6.449	0.006
Treatment	2	0.497		0.249	0.438	0.657
Linear	1	0.337	67.81	0.337	0.594	0.474
Quadratic	1	0.160	32.19	0.160	0.282	0.636
Error	10	5.674		0.567		

Total Density	***df***	**SS**	**% SS Explained**	**MS**	**F**	**p**
Block	5	111.328		22.266	5.480	0.011
Treatment	2	26.100		13.050	3.212	0.084
Linear	1	26.096	99.98	26.096	6.423	0.034
Quadratic	1	0.004	0.01	0.004	0.001	
Error	10	40.630		4.063		

Prevalence	***df***	**X^2**	**p**
Treatment	2	4.133	0.127

Table 4.7 Results of egg reproduction experiments from 1996 for sporocyst mean density, total density, and prevalence. Analyses of trend are reported for linear, quadratic and cubic polynomials. "%SS Explained" reports the proportion of treatment SS explained by each trend..

Mean Density	*df*	SS	% SS Explained	MS	F	p
Block	4	25.804		6.451	0.284	0.883
Treatment	3	147.869		49.290	2.168	0.145
Linear	1	142.108	96.10	142.108	6.251	0.017
Quadratic	1	1.391	0.94	1.391	0.061	ns
Cubic	1	4.370	2.96	4.370	0.192	0.693
Error	12	272.793		22.733		

Total Density	*df*	SS	% SS Explained	MS	F	p
Block	4	428.025		107.006	0.236	0.912
Treatment	3	2550.410		850.137	1.878	0.187
Linear	1	2388.101	93.64	2388.101	5.275	0.043
Quadratic	1	66.253	2.60	66.253	0.146	0.724
Cubic	1	96.056	3.77	96.056	0.212	0.680
Error	12	5432.791		452.733		

Prevalence	*df*	SS	% SS Explained	MS	F	p
Block	4	0.0230		0.0058	0.3286	0.858
Treatment	3	0.1080		0.0360	2.0571	0.160
Linear	1	0.0995	92.13	0.0995	5.6857	0.037
Quadratic	1	0.0067	6.16	0.0067	0.3803	0.574
Cubic	1	0.0020	1.81	0.0020	0.1114	0.746
Error	12	0.2100		0.0175		

Table 4.8 Results of egg reproduction experiments from 1997 for sporocyst mean density, total density, and prevalence. Analyses of trend are reported for linear, quadratic and cubic polynomials. "%SS Explained" reports the proportion of treatment SS explained by each trend..

Mean Density	*df*	**SS**	**% SS Explained**	**MS**	**F**	**p**
Block	4	425.457		106.364	5.264	0.011
Treatment	3	66.258		22.086	1.093	0.390
Linear	1	20.226	30.53	20.226	1.001	0.367
Quadratic	1	44.806	67.62	44.806	2.218	0.184
Cubic	1	1.225	1.85	1.225	0.061	ns
Error	12	242.467		20.206		

Total Density	*df*	**SS**	**% SS Explained**	**MS**	**F**	**p**
Block	4	5607.45		1401.86	5.83	0.008
Treatment	3	892.04		297.35	1.24	0.339
Linear	1	230.05	25.79	230.05	0.96	0.379
Quadratic	1	636.40	71.34	636.40	2.65	0.146
Cubic	1	25.59	2.87	25.59	0.11	0.750
Error	12	2883.83		240.32		

Prevalence	*df*	$\mathbf{X^2}$	**p**
Treatment	3	4.378	0.223

Table 4.9 Results of cercarial reproduction experiments from 1996 for metacercarial mean density, total density, and prevalence. Analyses of trend are reported for linear, quadratic and cubic polynomials. "%SS Explained" reports the proportion of treatment SS explained by each trend..

Mean Density	*df*	**SS**	**% SS Explained**	**MS**	**F**	**p**
Block	4	2.700		0.675	5.606	0.009
Treatment	3	0.964		0.321	2.669	0.096
Linear	1	0.098	10.15	0.098	0.813	0.416
Quadratic	1	0.462	47.92	0.462	3.836	0.079
Cubic	1	0.405	42.01	0.405	3.363	0.094
Error	12	1.445		0.120		

Total Density	*df*	**SS**	**% SS Explained**	**MS**	**F**	**p**
Block	4	29.124		7.281	3.741	0.034
Treatment	3	10.458		3.486	1.791	0.202
Linear	1	2.889	27.62	2.889	1.485	0.248
Quadratic	1	3.982	38.08	3.982	2.046	0.199
Cubic	1	3.587	34.30	3.587	1.843	0.217
Error	12	23.353		1.946		

Prevalence	*df*	**SS**	**% SS Explained**	**MS**	**F**	**p**
Block	4	0.1640		0.0410	0.8039	0.546
Treatment	3	0.6670		0.2223	4.3595	0.027
Linear	1	0.1334	20.00	0.133	2.615	0.150
Quadratic	1	0.0314	4.70	0.031	0.615	0.481
Cubic	1	0.5023	75.31	0.502	9.850	0.014
Error	12	0.6120		0.0510		

Table 4.10 Results of cercarial reproduction experiments from 1997 for metacercarial mean density, total density, and prevalence. Analyses of trend are reported for linear, quadratic and cubic polynomials. "%SS Explained" reports the proportion of treatment SS explained by each trend.

Mean Density	*df*	SS	% SS Explained	MS	F	p
Block	4	1105.231		276.3078	0.8667	0.516
Treatment	3	2249.795		749.9317	2.3524	0.143
Linear	1	19.896	0.88	19.89585	0.0624	ns
Quadratic	1	1923.118	85.48	1923.118	6.0324	0.034
Cubic	1	306.781	13.64	306.7808	0.9623	0.379
Error	11	3506.792		318.7993		

Total Density	*df*	SS	% SS Explained	MS	F	p
Block	4	0.0093		0.0023	0.156	0.947
Treatment	3	0.0628		0.0209	1.402	0.318
Linear	1	0.0069	11.03	0.0069	0.464	0.506
Quadratic	1	0.0483	76.86	0.0483	3.233	0.100
Cubic	1	0.0076	12.14	0.0076	0.511	0.494
Error	11	0.1642		0.015		

Prevalence	*df*	X^2	p
Treatment	3	3.577	0.311

Table 4.11 Results of egg transmission experiments from 1997 that tested the response of egg transmission to different densities of donor and target hosts in a total of 10 treatments. The response is measured in terms of sporocysts that established in the snail hosts. Data were analyzed in randomized complete block ANOVAs on a) overall treatment effects and b) separate effects of donor (infected salamander) host and target (uninfected snail) host densities.

a. Sporocyst density and prevalence for treatments.

Mean Density	***df***	**SS**	**MS**	**F**	**p**
Block	5	312.622	62.524	2.443	0.049
Treatment	9	361.554	40.173	1.570	0.336
Error	45	1151.806	25.596		
Total Density					
Block	5	5018.633	1003.727	1.873	1.873
Treatment	9	14921.840	1657.982	3.094	0.006
Error	45	24111.350	535.808		
Prevalence					
Block	5	0.299	0.060	3.037	0.021
Treatment	9	0.328	0.036	1.851	0.088
Residuals	45	0.886	0.020		

b. Sporocyst density and prevalence for donor and target hosts

Mean Density	***df***	**SS**	**MS**	**F**	**p**
Block	5	312.665	62.533	2.475	0.045
Donor	3	160.745	53.582	2.120	0.110
Target	2	114.348	57.174	2.262	0.115
Residuals	49	1238.273	25.271		
Total Density					
Block	5	5017.940	1003.588	1.847	0.121
Donor	3	5208.830	1736.277	3.196	0.031
Target	2	7203.370	3601.685	6.629	0.003
Residuals	49	26621.290	543.292		
Prevalence					
Block	5	0.293	0.059	2.931	0.022
Donor	3	0.162	0.054	2.711	0.055
Target	2	0.074	0.037	1.850	0.168
Residuals	49	0.979	0.020		

Table 4.12 Analyses of trend for the 1997 egg transmission experiments for a) mean density, b) total density, and c) prevalence of sporocysts in snails. Trends were analyzed across salamander treatment densities at each treatment den of snails (45, 102 or 159 / m^2). "% SS Explained" is the proportion of variation explained by the trend SS. * denotes significance at the = 0.025 level for 2 trends and = 0.0125 for 3 trends.

a. Trends in response of mean sporocyst density to salamander density.

Snail Density		*df*	SS	% SS Explained	F	
45 / m^2	Linear	1	19.066	5.273	0.451	
	Quadratic	1	15.777	4.364	0.373	
	Cubic	1	26.186	7.243	0.620	
102 / m^2	Linear	1	10.570	2.923	0.651	
	Quadratic	1	61.104	16.900	3.756	
159 / m^2	Linear	1	103.150	28.530	10.870	*
	Quadratic	1	0.008	0.002	0.001	

b) Trends in response of total sporocyst density to salamander density.

Snail Density		*df*	SS	% SS Explained	F	
45 / m^2	Linear	1	196.42	1.316	0.318	
	Quadratic	1	446.473	2.992	0.724	
	Cubic	1	122.241	0.819	0.198	
102 / m^2	Linear	1	274.401	1.839	0.589	
	Quadratic	1	1860.281	12.467	3.994	
159 / m^2	Linear	1	4296.372	28.793	10.865	*
	Quadratic	1	15.828	0.106	0.040	

c) Trends in response of sporocyst prevalence to salamander density.

Snail Density		*df*	SS	% SS Explained	F	
45 / m^2	Linear	1	0.0612	18.6509	1.8595	
	Quadratic	1	0.0019	0.5854	0.0585	
	Cubic	1	0.0136	4.1524	0.4142	
102 / m^2	Linear	1	0.0015	0.4494	1.2287	
	Quadratic	1	0.0504	15.3659	41.9960	*
159 / m^2	Linear	1	0.0643	19.6006	7.8503	*
	Quadratic	1	0.0015	0.4451	0.1788	

Table 4.13 Analyses of trend for the 1997 egg transmission experiments for a) mean density, b) total density, and c) prevalence of sporocysts in snails. Trends were analyzed across snail treatment densities at each treatment density of salamanders (4, 8, or 11 / m^2). "% SS Explained" is the proportion of variation explained by the trend SS. * denotes significance at the = 0.025 level.

a. Trends in response of mean sporocyst density to changes in snail density.

Salamander Density		*df*	SS	% SS Explained	F	
4 / m^2	Linear	1	2.261	0.625	0.099	
	Quadratic	1	144.934	40.086	72.233	*
8 / m^2	Linear	1	0.072	0.020	0.003	
	Quadratic	1	0.146	0.040	0.006	
11 / m^2	Linear	1	19.995	5.530	1.458	
	Quadratic	1	33.396	9.237	2.436	

b) Trends in response of total sporocyst density to changes in snail density.

Salamander Density		*df*	SS	% SS Explained	F	
4 / m^2	Linear	1	3536.276	23.699	7.808	*
	Quadratic	1	3201.369	21.454	7.069	*
8 / m^2	Linear	1	2030.356	13.607	2.526	
	Quadratic	1	6.409	0.043	0.008	
11 / m^2	Linear	1	450.337	3.018	1.161	
	Quadratic	1	488.521	3.274	1.260	

c) Trends in response of sporocyst prevalence to changes in snail density.

Salamander Density		*df*	SS	% SS Explained	F	
4 / m^2	Linear	1	0.0019	0.5793	0.1840	
	Quadratic	1	0.1190	36.2805	11.3610	*
8 / m^2	Linear	1	0.0002	0.0610	0.0082	
	Quadratic	1	0.0034	1.0366	0.1266	
11 / m^2	Linear	1	0.0183	5.5793	1.3096	
	Quadratic	1	0.0224	6.8293	1.6047	

Table 4.14 Results of cercarial transmission experiments from 1997 that tested the response of cercarial transmission to different densities of donor and targe hosts in a total of 10 treatments. The response is measured in terms of metacercariae that established in the stonefly hosts. Data were analyzed in randomized complete block ANOVAs on a) overall treatment effects and b) separate effects of donor (infected snail) host and target (uninfected stonefly) host densities.

a. Metacercarial density and prevalence for treatments

Mean Density	***df***	**SS**	**MS**	**F**	**p**
Block	5	56.075	11.215	10.695	0.000
Treatment	9	11.296	1.255	1.197	0.290
Error	45	47.189	1.049		
Total Density					
Block	5	2.077	0.415	8.953	0.000
Treatment	9	1.356	0.151	3.247	0.004
Error	45	2.088	0.046		
Prevalence					
Block	5	2.847	0.569	6.037	0.000
Treatment	9	1.062	0.118	1.251	0.573
Error	45	4.244	0.094		

b. Metacercarial density and prevalence for donor and target hosts

Mean Density	***df***	**SS**	**MS**	**F**	**p**
Block	5	56.075	11.215	10.881	0.000
Donor	3	5.733	1.911	1.854	0.150
Target	2	2.248	1.124	1.091	0.344
Error	49	50.504	1.031		
Total Density					
Block	5	2.077	0.415	9.090	0.0000
Donor	3	0.411	0.137	2.998	0.0393
Target	2	0.795	0.398	8.699	0.0006
Error	49	2.239	0.046		
Prevalence					
Block	5	2.847	0.569	6.074	0.000
Donor	3	0.196	0.065	0.695	0.559
Target	2	0.518	0.259	2.765	0.073
Error	49	4.593	0.094		

Table 4.15 Analyses of trend for the 1997 cercarial transmission experiments for a) mean density, b) total density, and c) prevalence of metacercariae in stoneflies. Trends were analyzed across stonefly treatment densities at each treatment density of snails (4, 15, or 27 / m^2). "% SS Explained" is the proportion of variation explained by the trend SS. * denotes significance at the = 0.025 level.

a) Trends in response of mean metacercarial density to levels of stonefly density.

Snail Density		*df*	SS	% SS Explained	F	
4 / m^2	Linear	1	0.047	0.419	0.039	
	Quadratic	1	2.057	18.210	1.680	
15 / m^2	Linear	1	2.856	25.283	1.833	
	Quadratic	1	0.074	0.655	0.048	
27 / m^2	Linear	1	0.248	2.195	0.232	
	Quadratic	1	0.280	2.479	0.262	

b) Trends in response of total metacercarial density to levels of stonefly density.

Snail Density		*df*	SS	% SS Explained	F	
4 / m^2	Linear	1	0.081	5.973	1.430	
	Quadratic	1	0.123	9.071	2.177	
15 / m^2	Linear	1	0.457	33.680	7.295	*
	Quadratic	1	0.000	0.028	0.116	
27 / m^2	Linear	1	0.220	16.224	5.060	*
	Quadratic	1	0.064	4.720	1.470	

c) Trends in response of metacercarial prevalence to levels of stonefly density.

Snail Density		*df*	SS	% SS Explained	F
4 / m^2	Linear	1	0.000	0.0000	0.001
	Quadratic	1	0.046	4.3315	0.824
15 / m^2	Linear	1	0.575	54.1431	4.180
	Quadratic	1	0.172	16.1959	1.250
27 / m^2	Linear	1	0.055	5.1789	0.898
	Quadratic	1	0.018	1.6949	0.294

Table 4.16 Analyses of trend for the 1997 cercarial transmission experiments for a) mean density, b) total density, and c) prevalence of metacercariae in stoneflies. Trends were analyzed across snail treatment densities at each treatment density of stoneflies (15, 30, or 45 / m^2). "% SS Explained" is the proportion of variation explained by the trend SS. * denotes significance at the = 0.025 level for 2 trends and = 0.0125 for 3 trends.

a) Trends in response of mean metacercarial density to levels of snail density.

Target Density		*df*	SS	% SS Explained	F
15 / m^2	Linear	1	1.846	16.342	1.780
	Quadratic	1	0.006	0.049	0.005
	Cubic	1	0.591	5.235	0.571
30 / m^2	Linear	1	0.019	0.167	0.024
	Quadratic	1	1.063	9.410	1.376
45 / m^2	Linear	1	1.636	14.483	8.275 *
	Quadratic	1	1.249	11.057	6.318 *

b) Trends in response of total metacercarial density to levels of snail density.

Target Density		*df*	SS	% SS Explained	F
15 / m^2	Linear	1	0.032	2.360	0.742
	Quadratic	1	0.003	0.192	0.060
	Cubic	1	0.016	1.164	0.365
30 / m^2	Linear	1	0.002	0.145	0.050
	Quadratic	1	0.059	4.344	1.510
45 / m^2	Linear	1	0.049	3.636	1.159
	Quadratic	1	0.063	4.653	1.486

c) Trends in response of metacercarial prevalence to levels of snail density.

Target Density		*df*	SS	% SS Explained	F
15 / m^2	Linear	1	0.004	0.3766	0.021
	Quadratic	1	0.017	1.6008	0.093
	Cubic	1	0.194	18.2674	1.067
30 / m^2	Linear	1	0.002	0.1883	0.090
	Quadratic	1	0.025	2.3540	1.184
45 / m^2	Linear	1	0.055	5.1789	1.390
	Quadratic	1	0.084	7.9096	2.110

Table 4.17 Estimated metacercarial transmission response to salamander and metacercarial densities. The estimate is based on natural variation of salamander and parasite densities. Ambient host and parasite densities were determined from censuses conducted in a) 7 creeks in unlogged watersheds and b) 11 creeks in logged watersheds. "Salamander" is the density of infected salamanders per creek. "Cyst estimate" is the estimate for the mean number of metacercariae that recruited to salamanders via predation of infected stoneflies. "Salamander * Cyst" is the interaction term between salamander density and metacercarial recruitment.

a. Unlogged

Mean Density	*df*	**SS**	**MS**	**F**	**p**
Cyst Estimate	1	2.44E-05	2.44E-05	3.169	0.173
Salamander	1	1.64E-05	1.64E-05	2.130	0.240
Cyst*Salamander	1	1.21E-05	1.21E-05	1.571	0.299
Residuals	3	2.31E-05	7.70E-06		
Total Density					
Cyst Estimate	1	0.0018	0.0018	7.200	0.075
Salamander	1	0.0186	0.0186	74.400	0.003
Cyst*Salamander	1	0.0021	0.0021	8.280	0.064
Residuals	3	0.0008	0.0003		
Prevalence					
Cyst Estimate	1	0.0762	0.0762	0.994	0.392
Salamander	1	0.0017	0.0017	0.022	0.891
Cyst*Salamander	1	0.0031	0.0031	0.041	0.853
Residuals	3	0.2299	0.0766		

b. Logged

Mean Density	*df*	**SS**	**MS**	**F**	**p**
Cyst Estimate	1	2.72E-07	2.72E-07	0.035	0.857
Salamander	1	4.25E-05	4.25E-05	5.459	0.052
Cyst*Salamander	1	1.47E-05	1.47E-05	1.888	0.212
Residuals	7	5.45E-05	7.79E-06		
Total Density					
Cyst Estimate	1	8.82E-05	8.82E-05	0.070	0.798
Salamander	1	1.14E-02	1.14E-02	9.102	0.019
Cyst*Salamander	1	4.64E-03	4.64E-03	3.708	0.096
Residuals	7	8.76E-03	1.25E-03		
Prevalence					
Cyst Estimate	1	0.155	1.55E-01	1.128	0.324
Salamander	1	0.409	4.09E-01	2.976	0.128
Cyst*Salamander	1	0.037	3.67E-02	0.267	0.621
Residuals	7	0.962	1.37E-01		

Table 4.18. Densities of infected hosts and parasites by creek. Data are from a census of 18 creeks that I conducted in 1998. See Chapter 3 for descriptions of the census.

Creek	Forest Type	Infected Salamander # / m^2	Infected Snail # / m^2	Infected Stonefly # / m^2	Adult Density # / m^2	Sporocyst Density # / m^2	Metacercaria Density # / m^2
Cone	Unlogged	0.0778	6.28	8.43	0.73	1259.55	38.69
Elk	Unlogged	0.0308	6.70	3.68	0.31	607.49	22.87
Hagan	Unlogged	0.0647	1.52	21.81	0.20	177.43	456.17
Jones	Unlogged	0.1143	23.09	23.18	0.24	3567.38	493.03
Mona	Unlogged	0.1014	6.46	10.67	0.47	304.15	320.53
Simmonds	Unlogged	0.0389	6.36	5.47	0.26	487.74	33.79
Slick	Unlogged	0.0145	1.82	8.54	0.10	45.81	22.81
EF Deer	Logged	0.0256	3.49	6.44	0.03	139.09	129.66
Finn	Logged	0.0120	1.72	3.20	0.03	221.34	31.20
Holden	Logged	0.0054	4.16	7.72	0.02	171.25	89.50
Osborne	Logged	0.0757	4.83	9.43	0.19	720.10	68.79
Ritchie	Logged	0.0514	3.69	1.62	0.26	411.16	0.76
Sturdy	Logged	0.0000	8.77	6.53	0.00	394.54	50.54
WF Deer	Logged	0.0208	5.63	7.87	0.03	112.50	78.39
EF NF Gate	Debris	0.0352	2.15	18.19	0.06	28.91	140.17
NF NF Gate	Debris	0.0076	0.00	0.00	0.00	0.00	0.00
SF Gate	Debris	0.0103	0.00	2.35	0.02	0.00	0.12
WF WF Gate	Debris	0.0374	1.48	17.92	0.48	11.53	16.18

FIGURE CAPTIONS

Figure 4.1. Life cycle diagram of *Cephalouterina dicamptodoni*, a trematode parasite that infects the definitive host, *Dicamptodon tenebrosus*, the 1st intermediate host, *Juga silicula*, and the 2nd intermediate host, *Calineuria californica*. Parasites transmit between hosts via the egg and free-living cercarial stages. Arrows indicate direction of transmission for each stage of the parasite between hosts.

Figure 4.2. Generalized schematic of enclosure positions in one block for transmission experiments conducted in 1997. All enclosures were placed not less than 0.5m from each other. Recruitment experiments, which had three treatment levels, were positioned as in numbers 1,2,4, or 1,3,4. Reproduction experiments, which had four treatment levels, were positioned as in numbers 1,2,3,4, or 1,2,4,5. Enclosure positions were altered in order to maintain constant depth, flow, substrate, and light environment within a block. a) Enclosures in 1995 and 1996 were open-ended cylinders with small mesh. b) Enclosures in 1997 were fully submerged and closed cylinders with larger mesh size. See text for full descriptions.

Figure 4.3. 1995 egg recruitment experiments. Mean density, total density and prevalence of sporocysts that established in snail hosts in the 1995 egg recruitment experiments. Treatments were low ($45 \cdot m^{-2}$), medium ($90 \cdot m^{-2}$) and high ($136 \cdot m^{-2}$) densities of snails and were replicated in 6 blocks. Each point represents the mean across all blocks.

Figure 4.4. 1997 egg recruitment experiments. Mean density, total density and prevalence of sporocysts that established in snail hosts in the 1997 egg recruitment experiments. Treatments were low ($45 \cdot m^{-2}$), medium ($102 \cdot m^{-2}$) and high ($159 \cdot m^{-2}$) densities of snails and were replicated in 6 blocks. Each point represents the mean across all blocks.

Figure 4.5. 1995 cercarial recruitment experiments. Mean density, total density and prevalence of metacercariae that established in stonefly hosts in the 1995 cercarial recruitment experiments. Treatments were low ($11 \cdot m^{-2}$), medium ($34 \cdot m^{-2}$) and high ($57 \cdot m^{-2}$) densities of stoneflies and were replicated in 6 blocks. Each point represents the mean across all blocks.

Figure 4.6. 1997 cercarial recruitment experiments. Mean density, total density and prevalence of metacercariae that established in stonefly hosts in the 1997 cercarial recruitment experiments. Treatments were low ($15 \cdot m^{-2}$), medium ($30 \cdot m^{-2}$) and high ($45 \cdot m^{-2}$) densities of stoneflies and were replicated in 6 blocks. Each point represents the mean across all blocks.

Figure 4.7. 1996 egg reproduction experiments. Mean density, total density and prevalence of sporocysts that established in snail hosts in the 1996 egg reproduction experiments. Treatments were ambient ($0 \cdot m^{-2}$), low ($2 \cdot m^{-2}$), medium ($5 \cdot m^{-2}$), and high ($7 \cdot m^{-2}$) densities of infected salamanders, each caged with 20 ($45 \cdot m^{-2}$) uninfected snails. Each treatment was replicated 5 times. Each point represents the mean across all blocks.

Figure 4.8. 1997 egg reproduction experiments. Mean density, total density and prevalence of sporocysts that established in snail hosts in the 1997 egg reproduction experiments. Treatments were ambient ($0 \cdot m^{-2}$), low ($4 \cdot m^{-2}$), medium ($8 \cdot m^{-2}$), and high ($11 \cdot m^{-2}$) densities of infected salamanders, each caged with 12 ($45 \cdot m^{-2}$) uninfected snails. Each treatment was replicated 5 times. Each point represents the mean across all blocks.

Figure 4.9. 1996 cercarial reproduction experiments. Mean density, total density and prevalence of metacercariae that established in stonefly hosts in the 1996 cercarial reproduction experiments. Treatments were ambient ($0 \cdot m^{-2}$), low ($9 \cdot m^{-2}$), medium ($18 \cdot m^{-2}$), and high ($36 \cdot m^{-2}$) densities of infected snails, each caged with 10 ($23 \cdot m^{-2}$) uninfected stoneflies. Each treatment was replicated 5 times. Each point represents the mean across all blocks.

Figure 4.10. 1997 cercarial reproduction experiments. Mean density, total density and prevalence of metacercariae that established in stonefly hosts in the 1997 cercarial reproduction experiments. Treatments were ambient ($0 \cdot m^{-2}$), low ($4 \cdot m^{-2}$), medium ($15 \cdot m^{-2}$), and high ($27 \cdot m^{-2}$) densities of infected snails, each caged with 8 ($30 \cdot m^{-2}$) uninfected stoneflies. Each treatment was replicated 5 times. Each point represents the mean across all blocks.

Figure 4.11. 1997 egg transmission experiments to assess the response of sporocysts that establish in snails to different densities of infected salamander and uninfected snail hosts.

Actual data points are designated by a '●'. The 3 dimensional plane represents the hypothetical functional response plane across all salamander and snail densities within the range of the experiment. a) Mean density shows the response of egg transmission to changing host densities as the mean density of sporocysts that established in snails during the experiment averaged across all 6 blocks. b) Total density shows the response of transmission as the total density of sporocysts that established in all snails from one replicate of one treatment, averaged across all 6 blocks. The total density axis is divided by 1000. c) Prevalence is the response in the percentage of snails infected per treatment for each combination of salamander and snail host. Salamander and snail densities used in the experiment are given in Table 4.2. All densities are reported as $\#\cdot m^{-2}$.

Figure 4.12. 1997 cercarial transmission experiments to assess the response of metacercariae that establish in stoneflies to different densities of infected salamander and uninfected snail hosts. Actual data points are designated by a '●'. The 3 dimensional plane represents the hypothetical functional response plane across all snail and stonefly densities within the range of the experiment. a) The response in mean density of metacercariae that established in stoneflies to changing densities of snail and stonefly hosts. b) The response in total density of metacercariae that established in stoneflies to changing densities of snail and stonefly hosts. c) The proportion of stoneflies infected for each combination of snail and stonefly host. Snail and stonefly densities used in the experiment are provided in Table 4.2. All densities are reported as $\#\cdot m^{-2}$.

Figure 4.13. Mean density of adult parasites ($\#\cdot m^{-2}$) as affected by the density of infected salamanders ($\#\cdot m^{-2}$) and the estimate of metacercarial recruitment ($\bar{E}_i$). Data were analyzed separately for unlogged and logged creeks.

Figure 4.14. Total density of adult parasites ($\#\cdot m^{-2}$) as affected by the density of infected salamanders ($\#\cdot m^{-2}$) and the estimate of metacercarial recruitment ($\bar{E}_i$). Data were analyzed separately for unlogged and logged creeks.

Figure 4.15 Proportion of salamanders infected by adult parasites (%) as affected by the density of infected salamanders ($\#\cdot m^{-2}$) and the estimate of metacercarial recruitment ($\bar{E}_i$). Data were analyzed separately for unlogged and logged creeks.

Figure 4.16. Combined data for the egg reproduction experiments in 1996 and 1997.

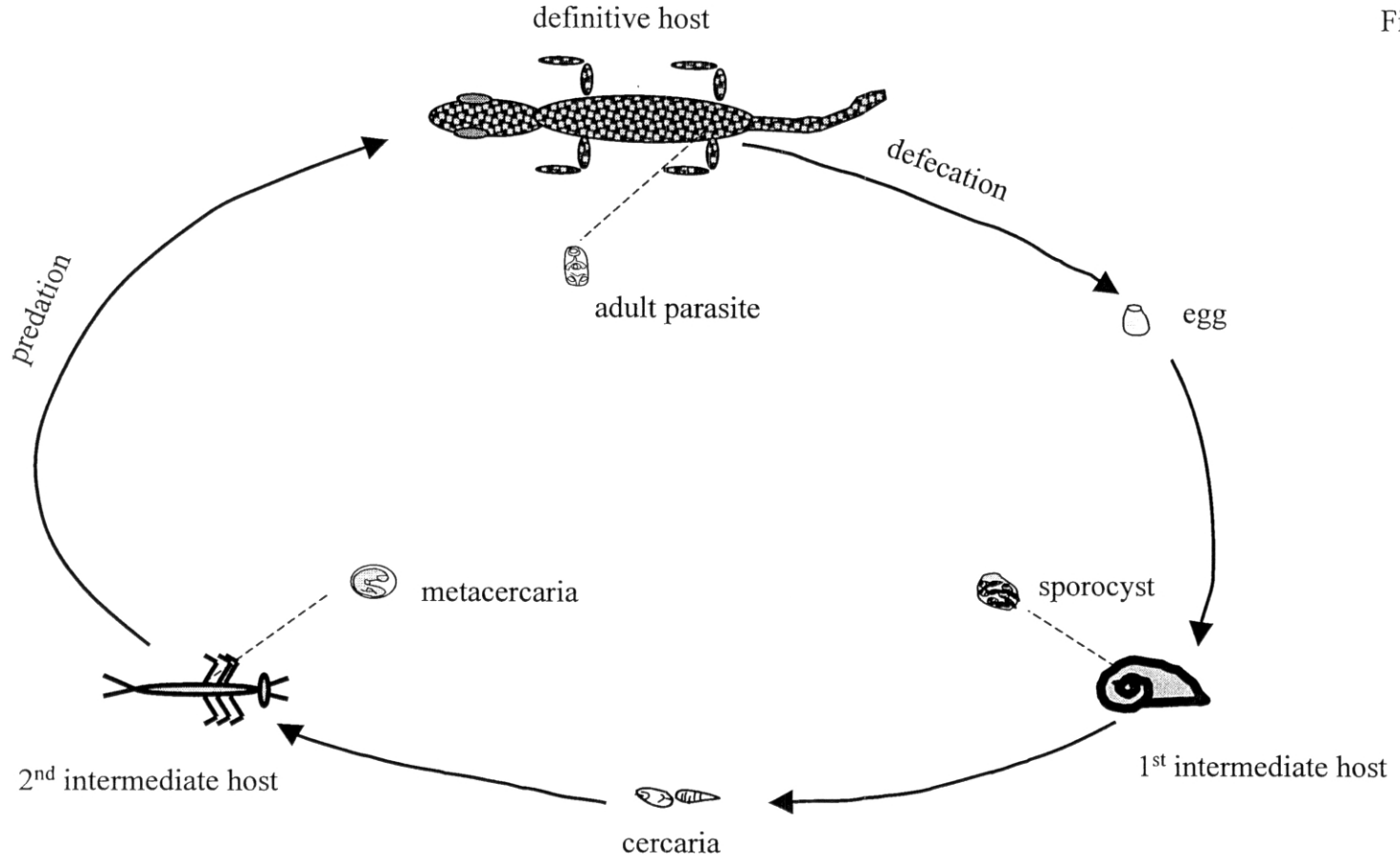
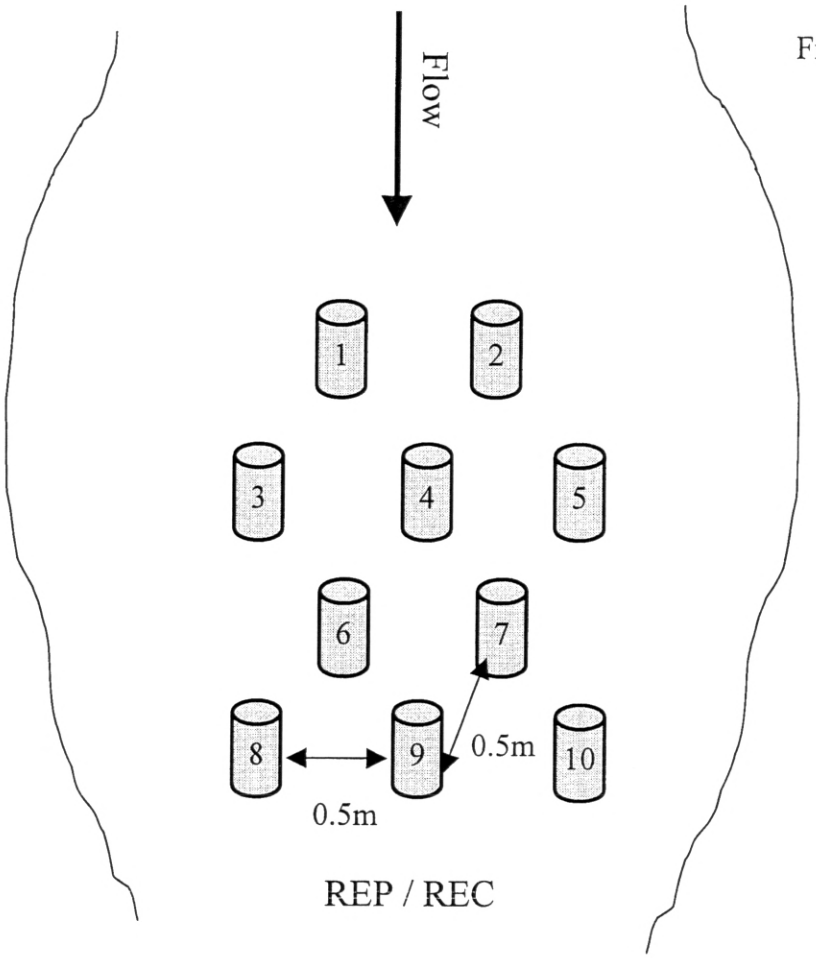

definitive host
defecation
adult parasite
egg
predation
metacercaria
sporocyst
2nd intermediate host
1st intermediate host
cercaria

Fig. 4.1

Fig. 4.2



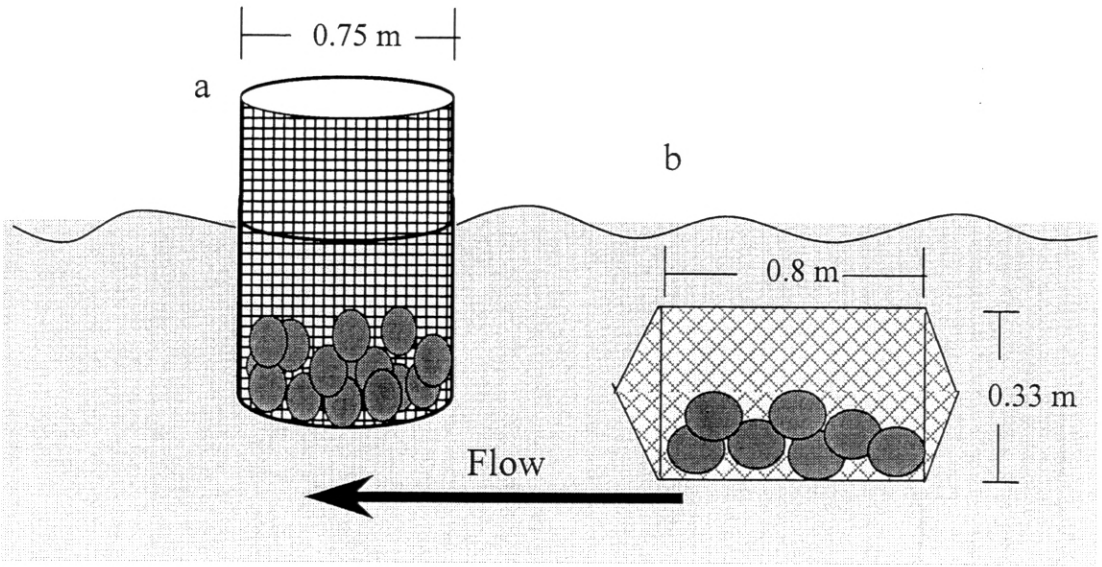


Fig. 4.3

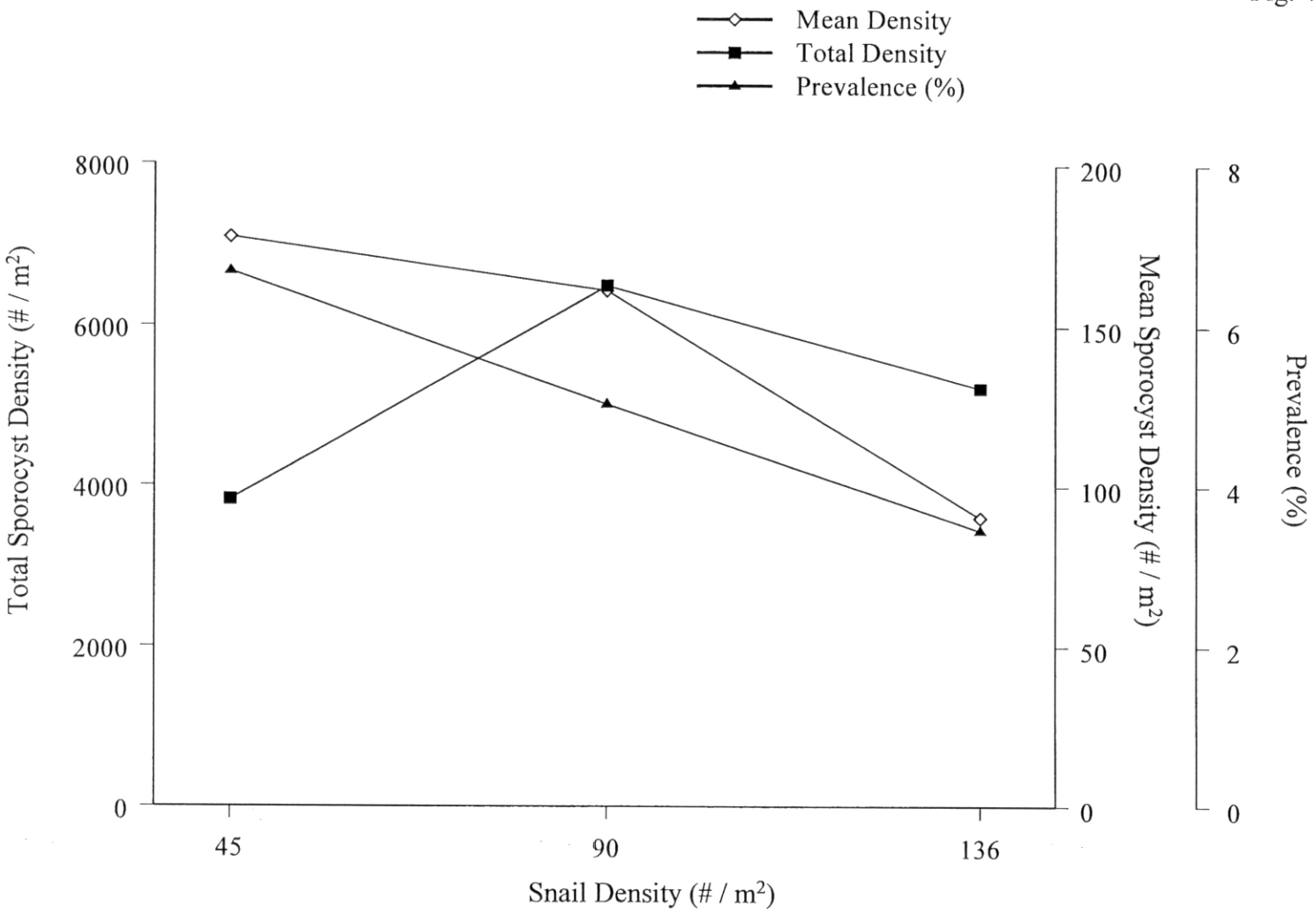

Mean Density
Total Density
Prevalence (%)
Total Sporocyst Density (# / m2)
Mean Sporocyst Density (# / m2)
Prevalence (%)
8000
6000
4000
2000
0
200
150
100
50
0
8
6
4
2
0
45
90
136
Snail Density (# / m2)

Fig. 4.4

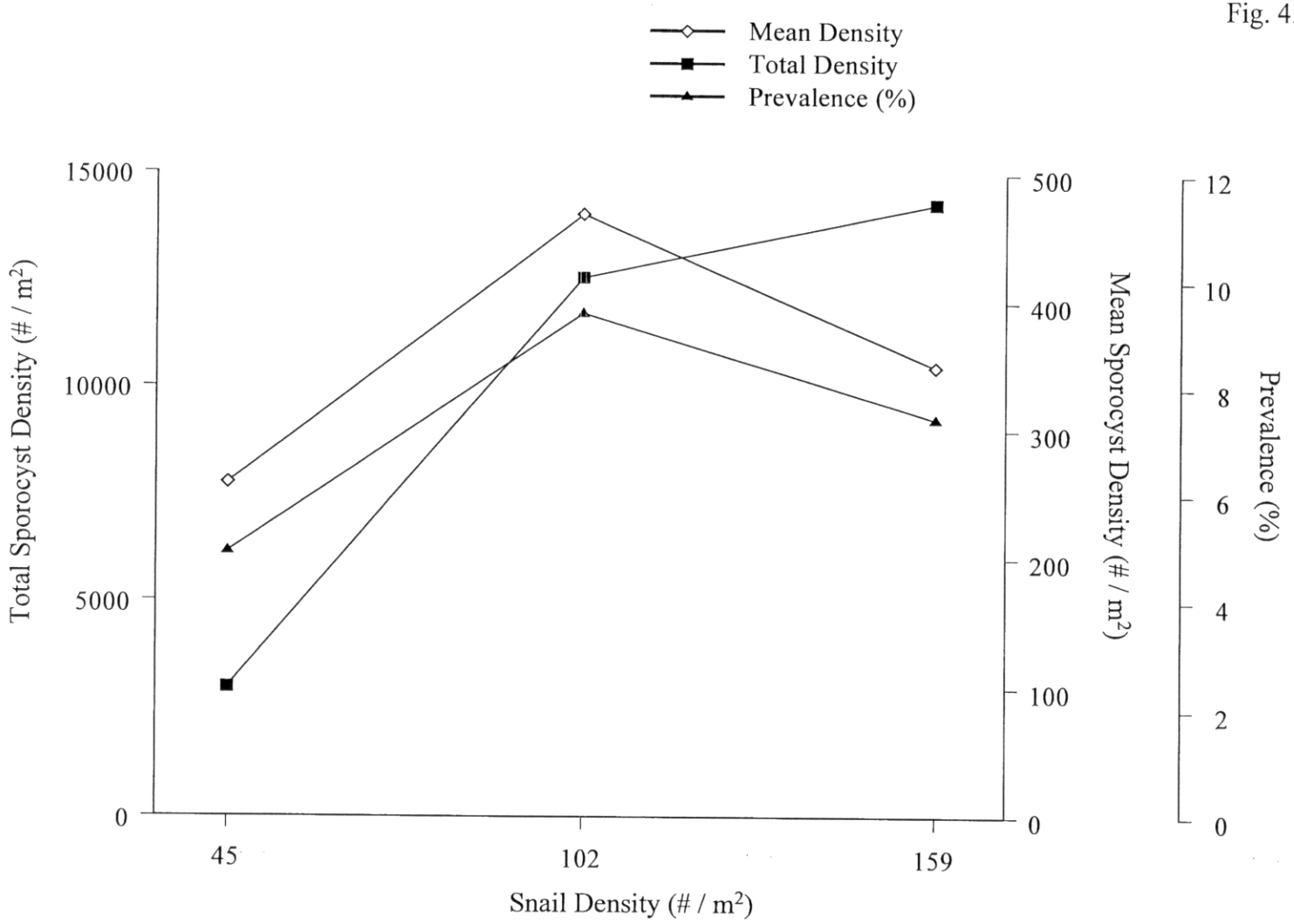

Mean Density
Total Density
Prevalence (%)
15000
10000
5000
0
Total Sporocyst Density (# / m2)
500
400
300
200
100
0
Mean Sporocyst Density (# / m2)
12
10
8
6
4
2
0
Prevalence (%)
45
102
159
Snail Density (# / m2)

Fig. 4.5

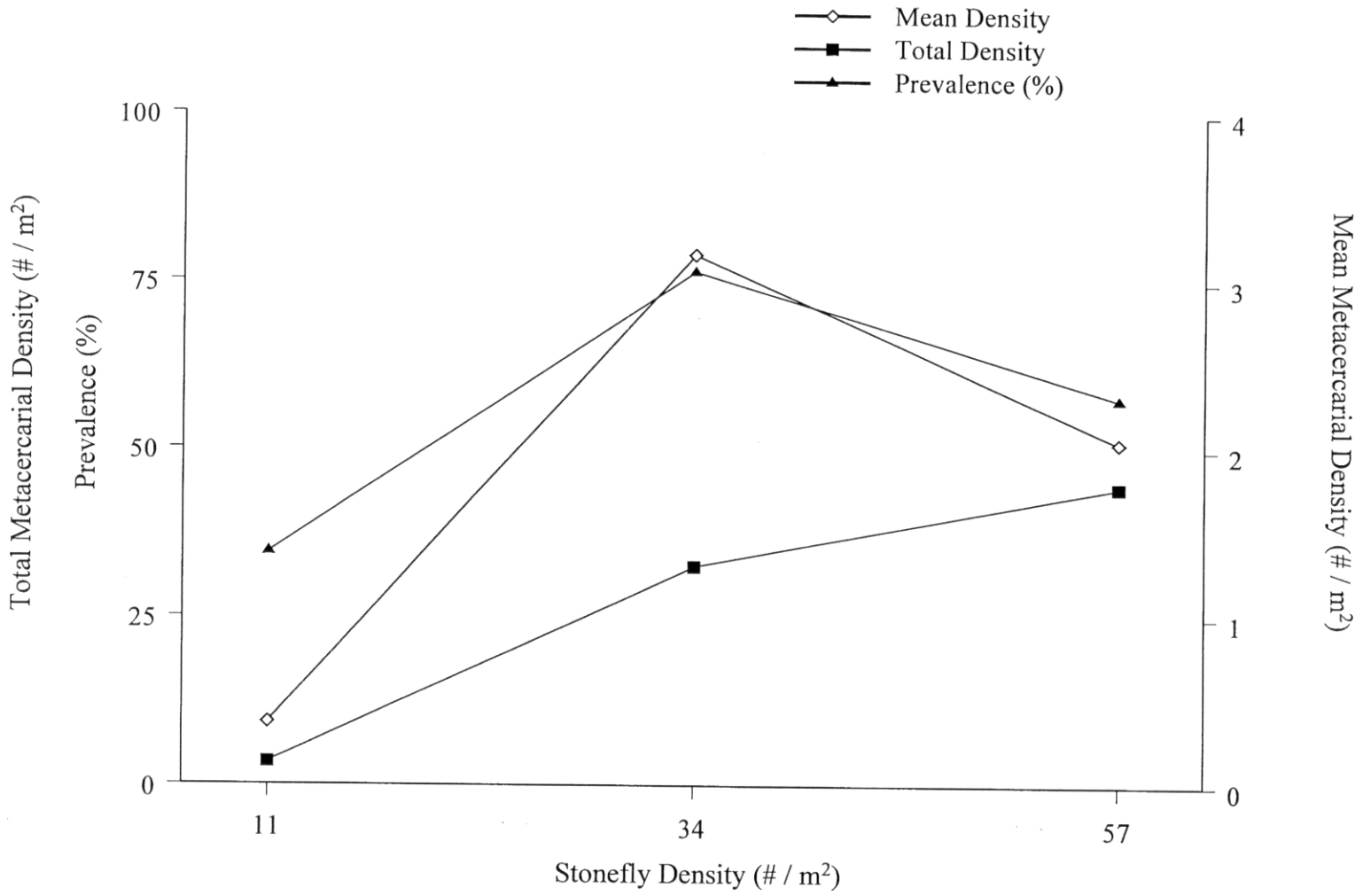

Mean Density
Total Density
Prevalence (%)
100
75
50
25
0
4
3
2
1
0
11
34
57
Total Metacercarial Density (# / m2)
Prevalence (%)
Mean Metacercarial Density (# / m2)
Stonefly Density (# / m2)

Fig. 4.6

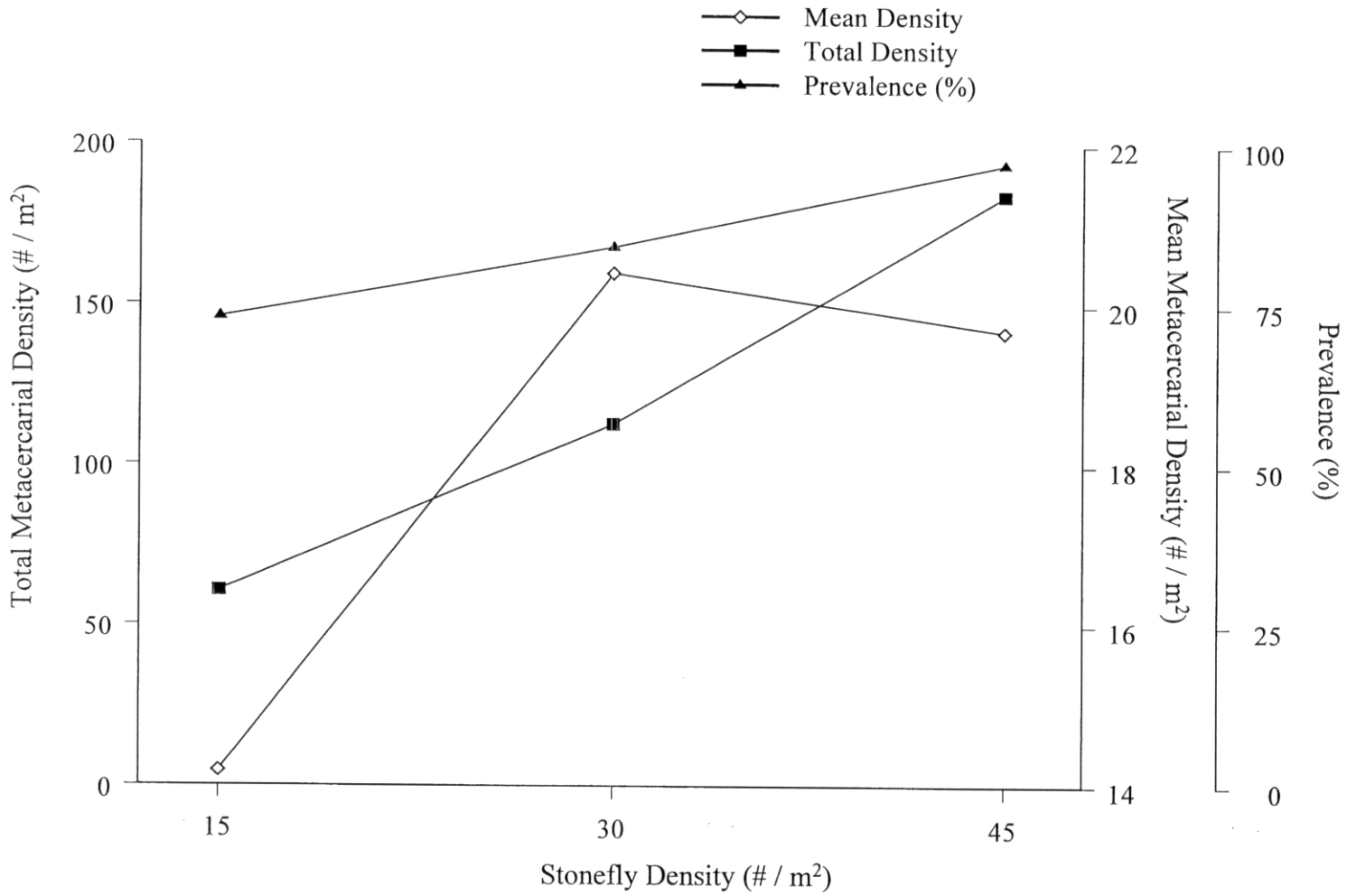

Mean Density
Total Density
Prevalence (%)
Total Metacercarial Density (# / m²)
Mean Metacercarial Density (# / m²)
Prevalence (%)
200
150
100
50
0
22
20
18
16
14
100
75
50
25
0
15
30
45
Stonefly Density (# / m²)

Fig. 4.7

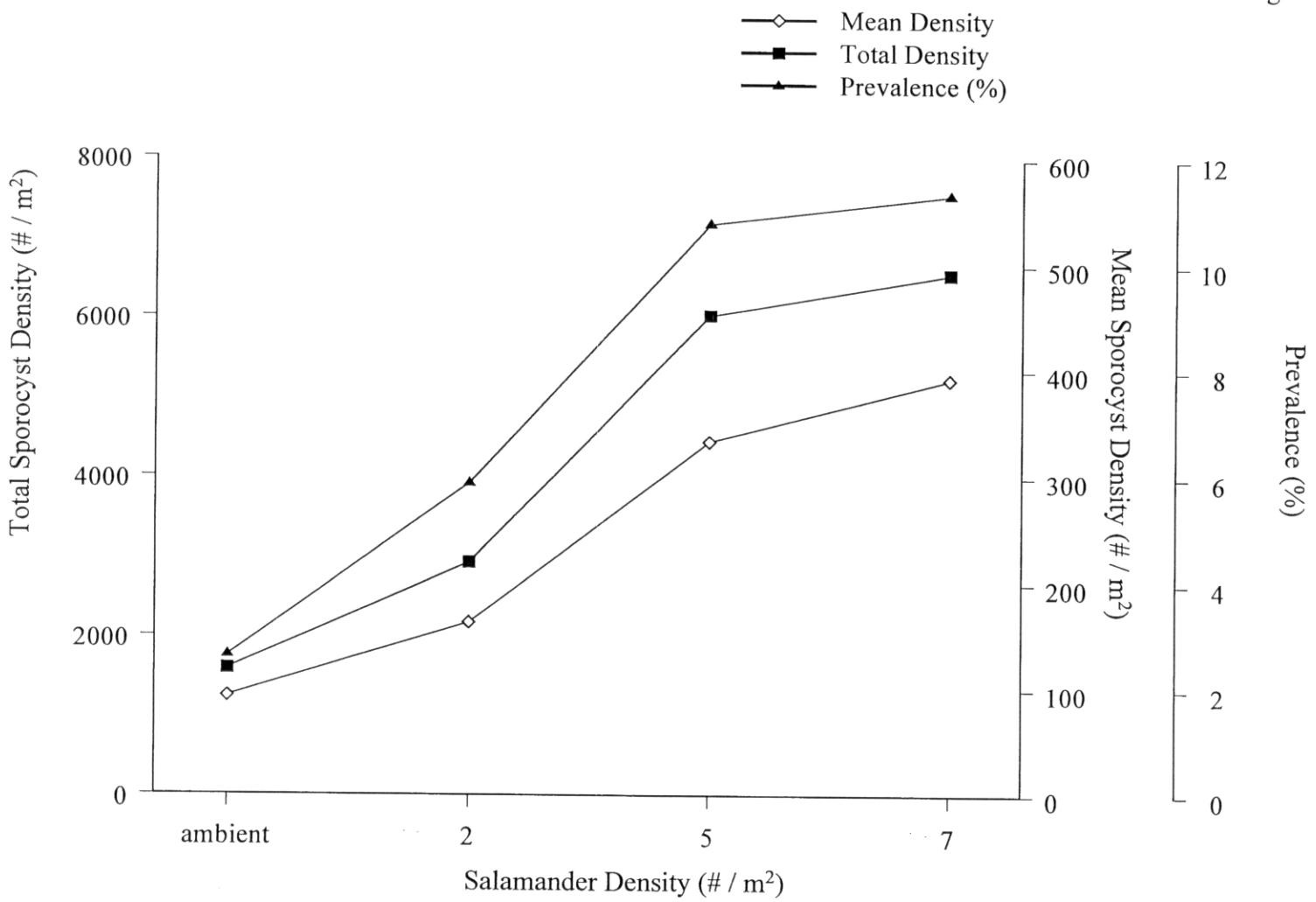

Mean Density
Total Density
Prevalence (%)
Total Sporocyst Density (# / m2)
Mean Sporocyst Density (# / m2)
Prevalence (%)
8000
6000
4000
2000
0
600
500
400
300
200
100
0
12
10
8
6
4
2
0
ambient
2
5
7
Salamander Density (# / m2)

Fig. 4.8

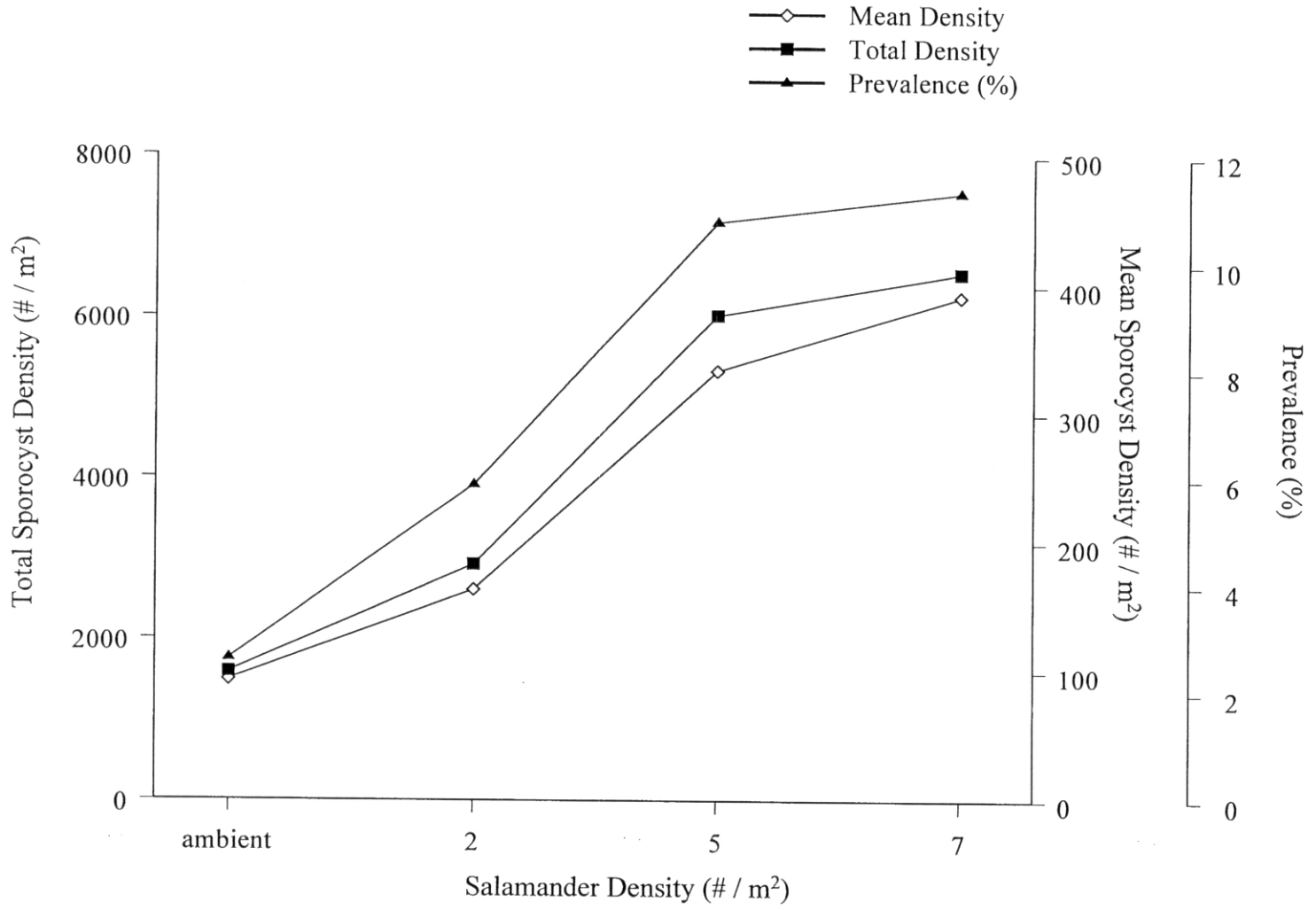

Mean Density
Total Density
Prevalence (%)
Total Sporocyst Density (# / m2)
Mean Sporocyst Density (# / m2)
Prevalence (%)
8000
6000
4000
2000
0
500
400
300
200
100
0
12
10
8
6
4
2
0
ambient
2
5
7
Salamander Density (# / m2)

Fig. 4.9

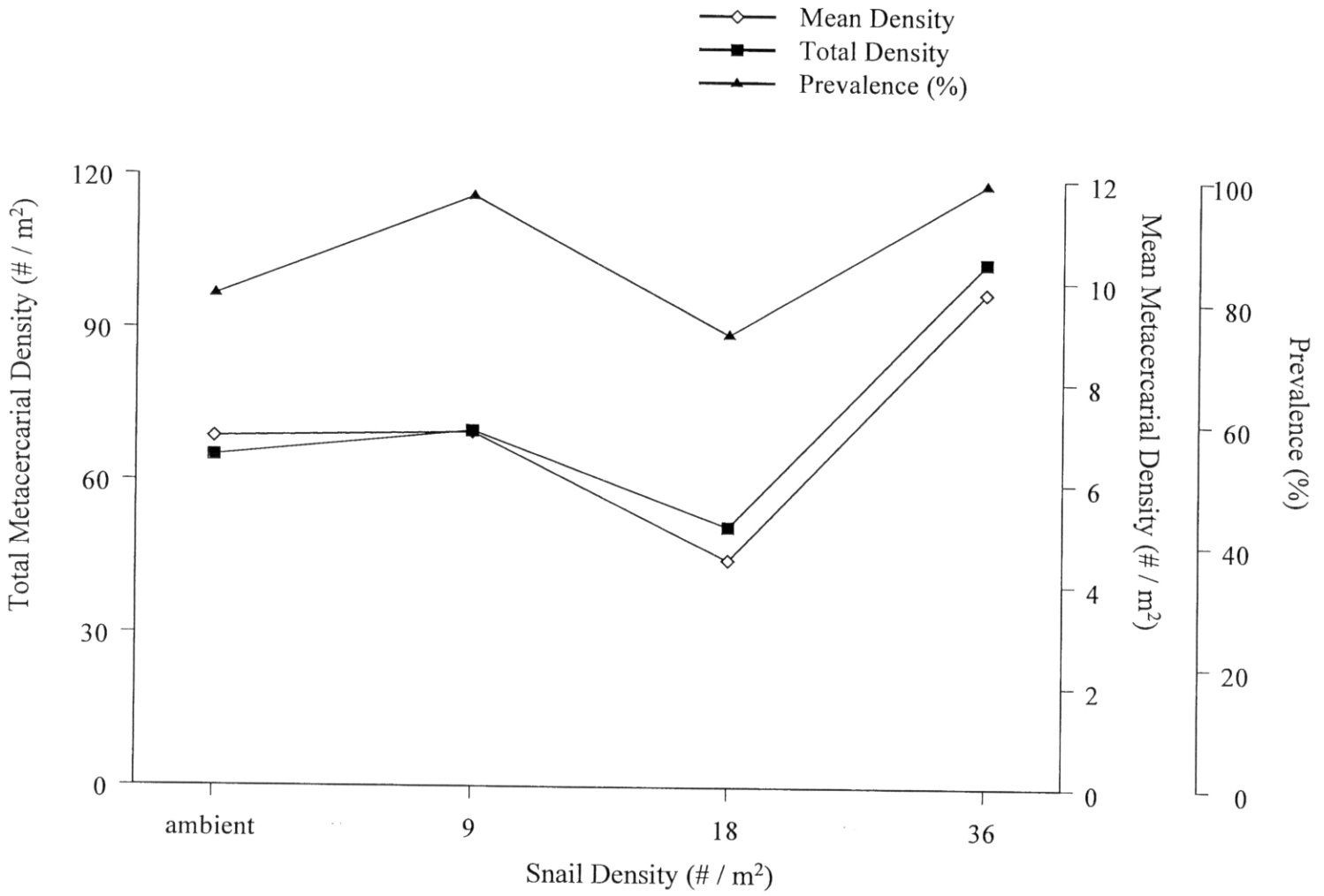

Mean Density
Total Density
Prevalence (%)
Total Metacercarial Density (# / m2)
Mean Metacercarial Density (# / m2)
Prevalence (%)
120
90
60
30
0
12
10
8
6
4
2
0
100
80
60
40
20
0
ambient
9
18
36
Snail Density (# / m2)

Fig. 4.10

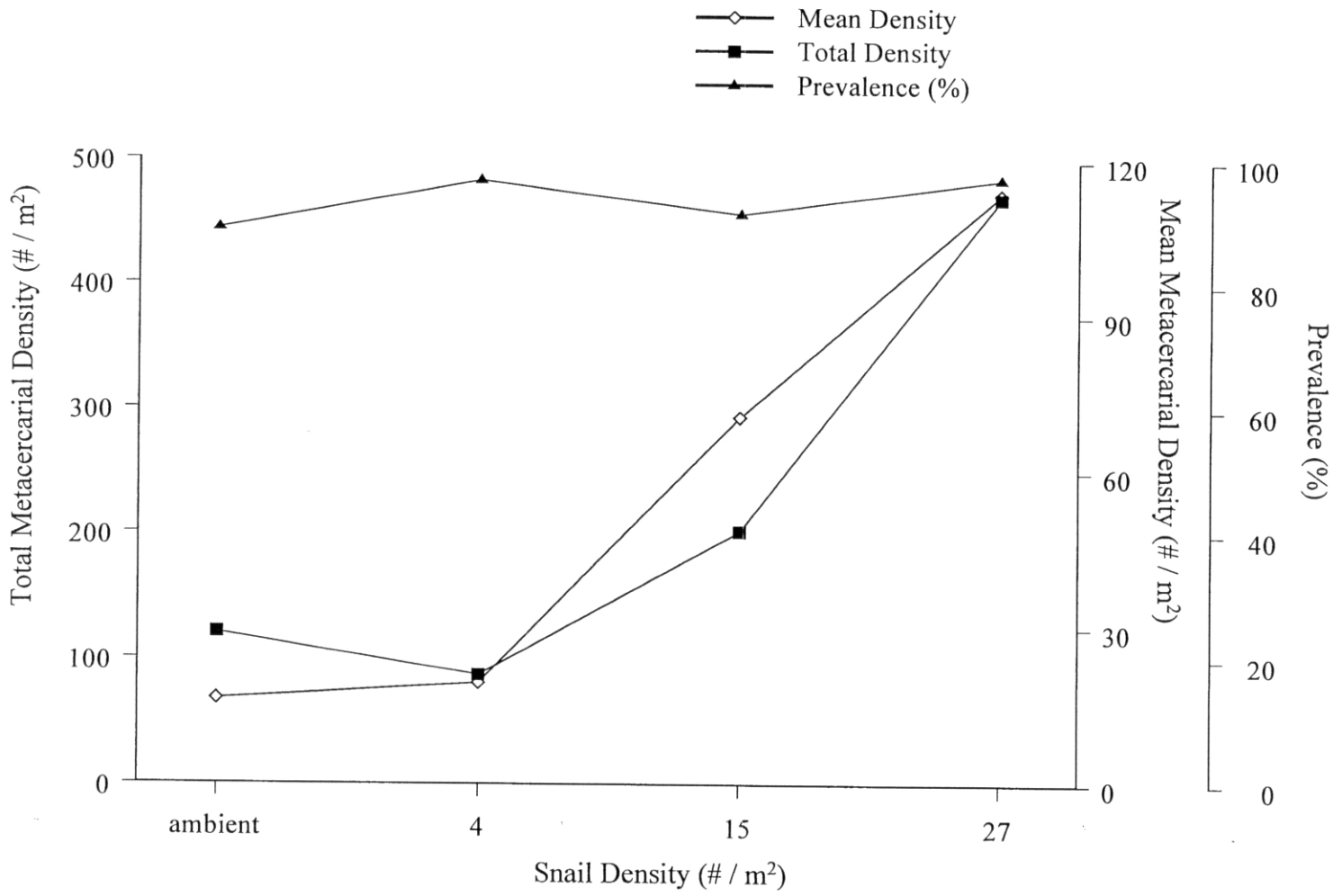
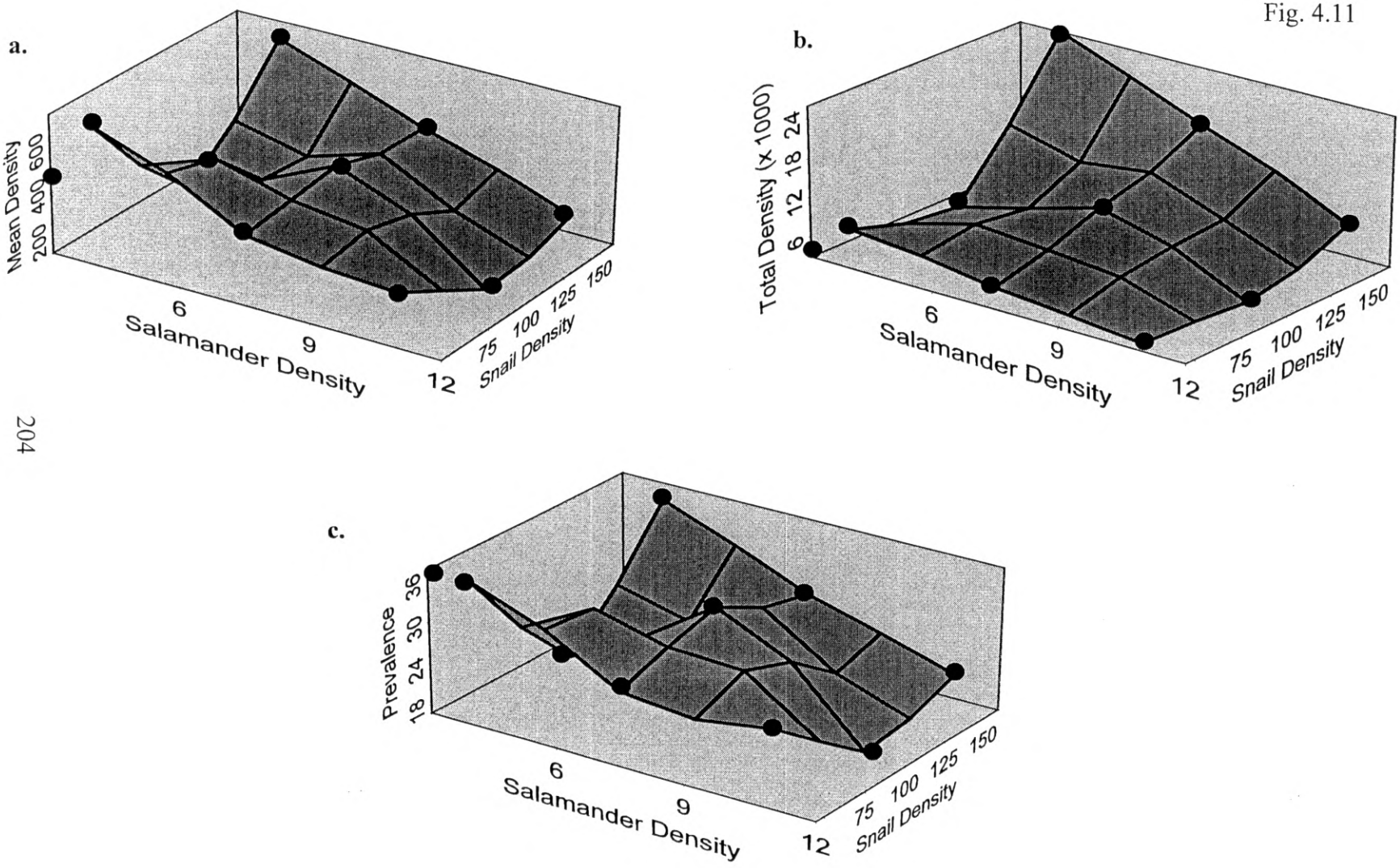

Mean Density
Total Density
Prevalence (%)
Total Metacercarial Density (# / m2)
Mean Metacercarial Density (# / m2)
Prevalence (%)
500
400
300
200
100
0
120
90
60
30
0
100
80
60
40
20
0
ambient
4
15
27
Snail Density (# / m2)

Fig. 4.11


a.
Mean Density
200 400 600
6
9
12
Salamander Density
75 100 125 150
Snail Density
b.
Total Density (x 1000)
6 12 18 24
6
9
12
Salamander Density
75 100 125 150
Snail Density
c.
Prevalence
18 24 30 36
6
9
12
Salamander Density
75 100 125 150
Snail Density

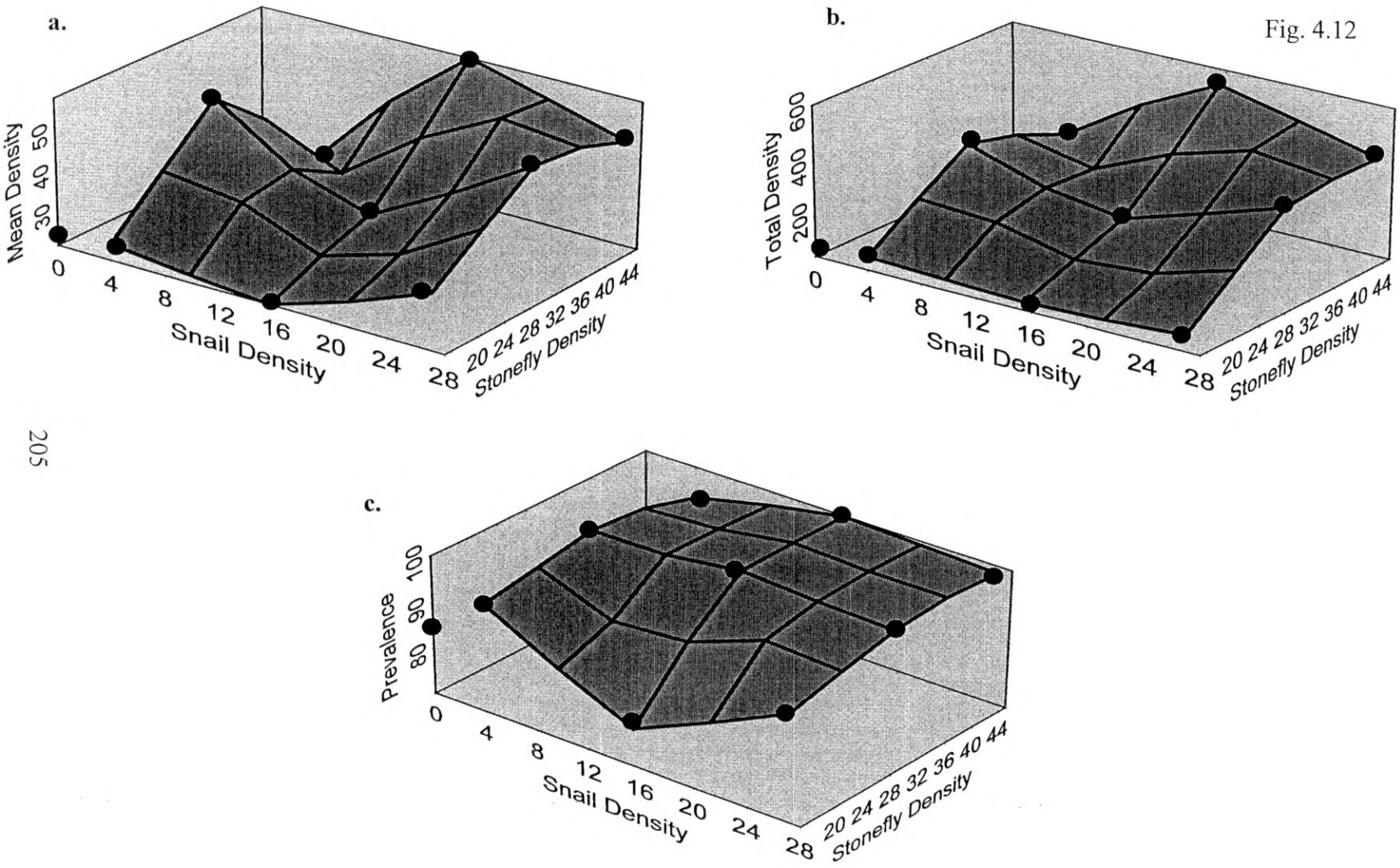
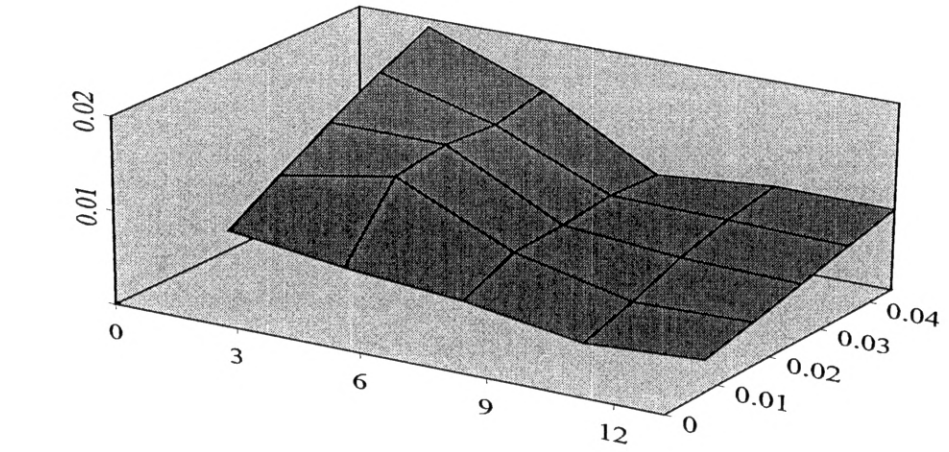
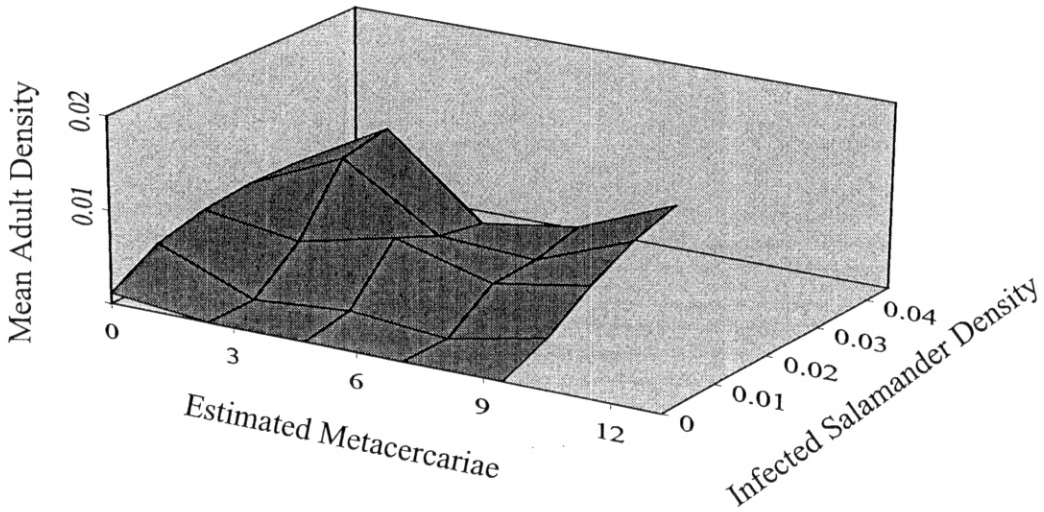

a.
Mean Density
30 40 50
0 4 8 12 16 20 24 28
Snail Density
20 24 28 32 36 40 44
Stonefly Density
b.
Total Density
200 400 600
0 4 8 12 16 20 24 28
Snail Density
20 24 28 32 36 40 44
Stonefly Density
c.
Prevalence
80 90 100
0 4 8 12 16 20 24 28
Snail Density
20 24 28 32 36 40 44
Stonefly Density

Fig. 4.12

Fig. 4.13



Unlogged



Logged

Fig. 4.14

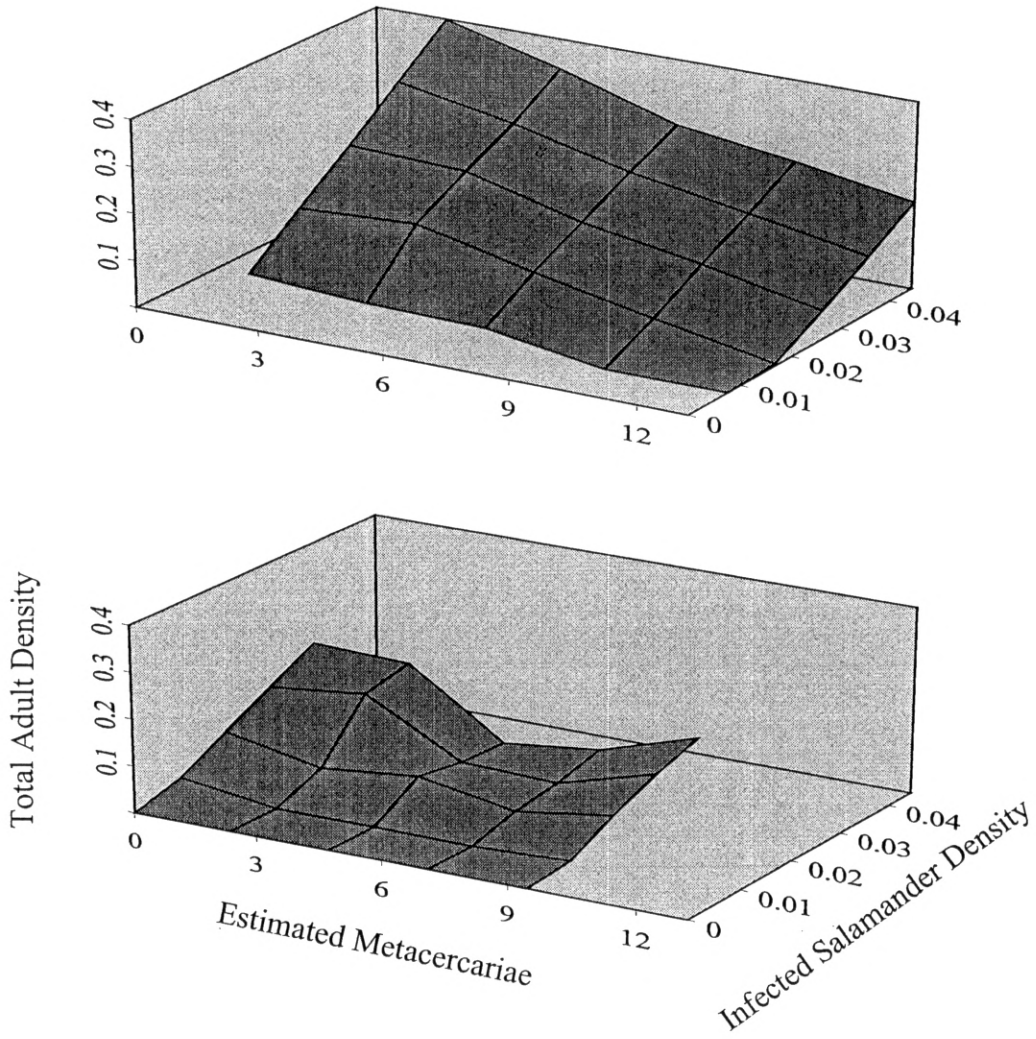
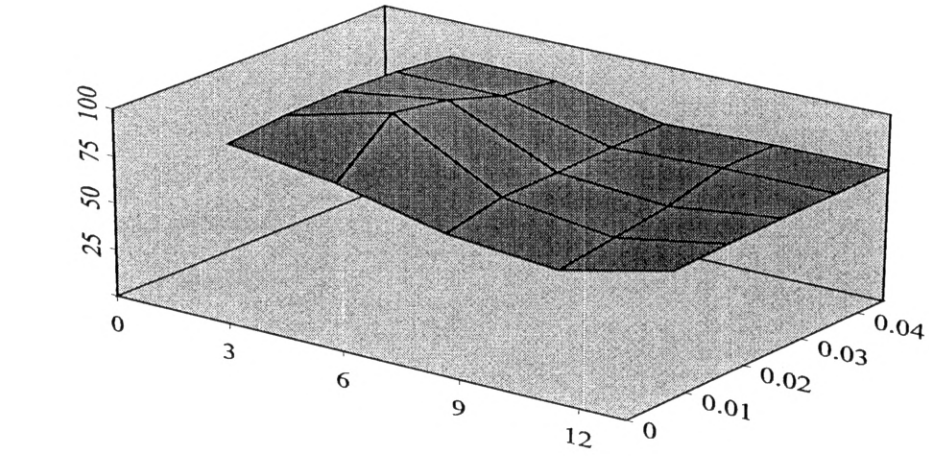
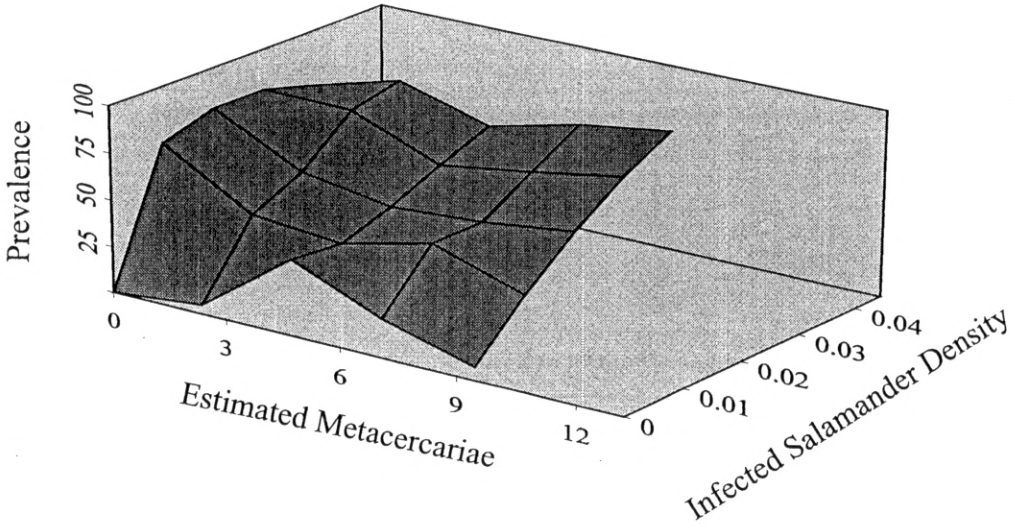

Unlogged
Logged
Total Adult Density
0.1
0.2
0.3
0.4
0
3
6
9
12
Estimated Metacercariae
0
0.01
0.02
0.03
0.04
Infected Salamander Density

Fig. 4.15

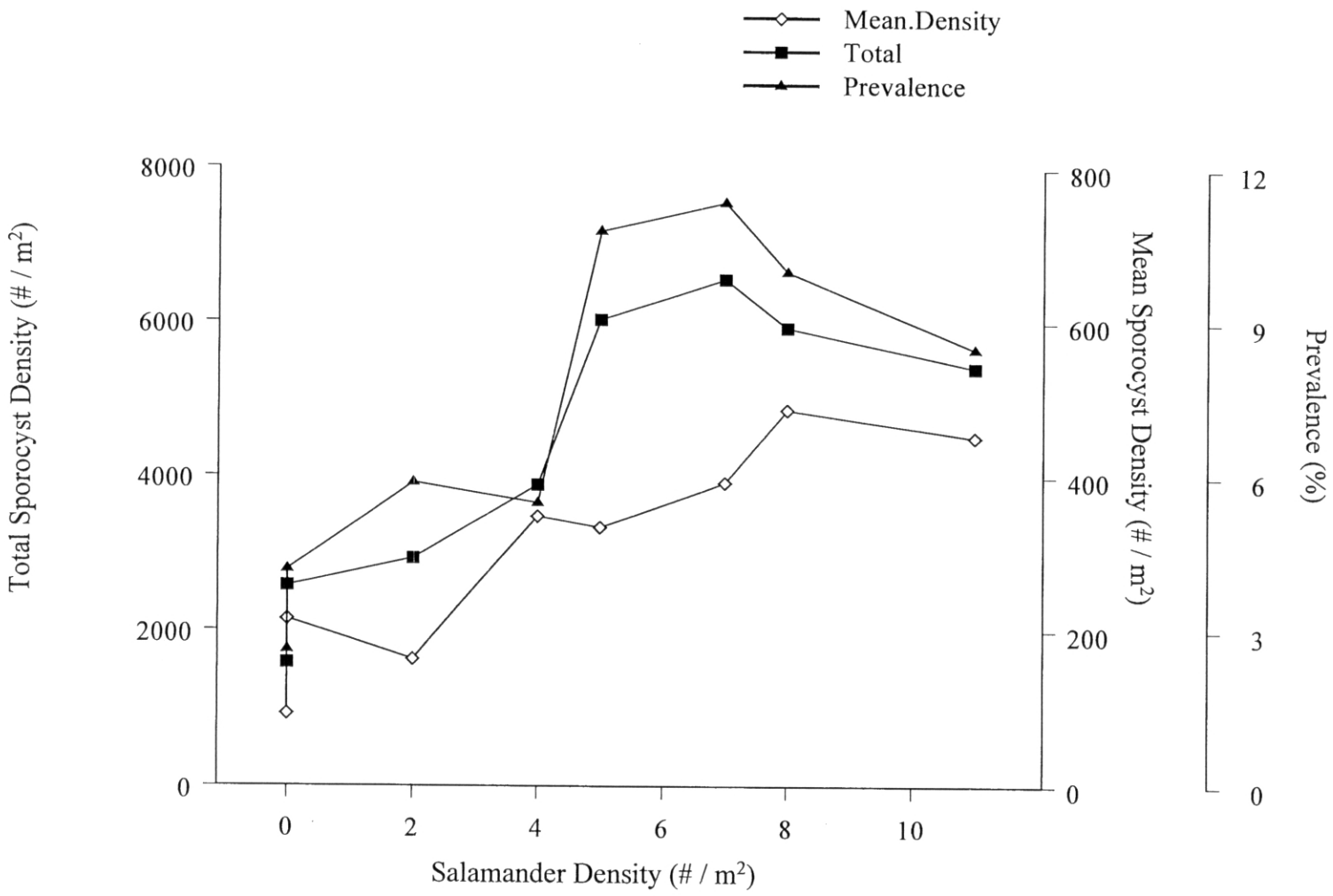


Unlogged



Logged

Fig. 4.16


Mean.Density
Total
Prevalence
Total Sporocyst Density (# / m2)
Mean Sporocyst Density (# / m2)
Prevalence (%)
Salamander Density (# / m2)
0
2000
4000
6000
8000
200
400
600
800
3
6
9
12
0
2
4
6
8
10