# Behavioral and Ecological Relationships of a Parasite and Its Hosts within a Coral Reef System[1]

GRETA S. AEBY[2]

ABSTRACT: The life cycle of the digenetic trematode *Plagioporus* sp. includes an intermediate stage that encysts in the scleractinian coral *Porites compressa* and an adult stage that probably resides in a coral-feeding fish. Coral polyps infected with metacercariae of *Plagioporus* appear as swollen nodules ranging in color from bright pink to white and have lost their ability to retract into their calices. The polyps' altered appearance and behavior was thought to increase their vulnerability to predation. This study investigated the effect of parasite encystment on coral growth and the effect of fish predation on both coral growth and on the parasites' rate of transmission. Parasitized *P. compressa* showed a 50% reduction in growth when compared to nonparasitized *P. compressa*. No significant differences were found in growth of corals kept in predator exclusion cages and that of corals left exposed to fish predation in either group, parasitized or nonparasitized. Uncaged parasitized *P. compressa* showed a marked reduction in number of parasitic cysts, with the infected polyps being replaced by healthy ones. The regeneration of healthy polyps suggests that parasite removal is beneficial to the coral, and the reduction in cyst number suggests that the parasites' rate of transmission was enhanced by exposure of infected corals to fish predation.

THE LIFE CYCLES OF MOST digenetic trematodes include one or more intermediate hosts and involve many intimate inter-relationships with hosts that can lead to interesting behavioral adaptations. Of particular interest is the suggestion that some parasites alter the appearance or behavior of their intermediate host to facilitate transmission of the parasite to its final host (Holmes and Bethel 1972). For example, snails infected with the broodsacs of *Leucochloridium* have tentacles that pulsate in response to light (Cheng 1986), and fish infected with certain heterophyid or strigeid trematodes develop "black spots" caused by host response to larval trematode invasion (Rothschild 1962). In both cases, it has been suggested that the increased conspicuousness of the intermediate hosts increased their susceptibility to predation by definitive host

species. Similarly, effects of parasites on host behavior have also been reported. Carpenter ants infected with *Brachylecithum mosquensis* metacercariae exhibit atypical sluggish behavior and are no longer photophobic (Carney 1969), and amphipods infected with acanthocephalan larvae have altered evasive behavior and responses to light (Bethel and Holmes 1977). In both cases, the altered behavior was thought to increase the intermediate hosts' vulnerability to predation.

Here I present data on a digenetic trematode, *Plagioporus* sp., whose presence results in the alteration of both the behavior and appearance of its intermediate host, a scleractinian coral. The parasites' metacercarial stage encysts in two species of coral, *Porites compressa* and *Porites lobata*, from Kaneohe Bay, Oahu, Hawaii (Cheng and Wong 1974). The infected polyps appear as irregularly shaped nodules on the coral, ranging in color from bright pink to white. In addition, infected polyps have lost the ability to retract into their calices, which may increase their vulnerability to predation. Preliminary

[2] Department of Zoology, University of Hawaii at Manoa, Honolulu, Hawaii 96822.

studies suggested that the alteration of the infected polyps resulted in corallivorous fish being attracted to and feeding on the infected polyps (unpublished data). The current study investigated the effect of parasite encystment on coral growth as well as the effect of fish predation on both coral growth and on the parasites' rate of transmission.

## Typical Life Cycle of the Parasite

The life cycle of a typical digenetic trematode is well known (Dogiel et al. 1961, Olsen 1962, Eramus 1972, Smyth and Halton 1983) and usually includes the adult worm living in the gall bladder of a fish. Eggs pass out with the host feces and hatch into a swimming miracidium stage, which enters a molluscan first intermediate host. The parasite leaves the mollusk in the cercaria stage, which penetrates its second intermediate host, *Porites* sp. coral. It enters the tentacles of the coral polyps and encysts as a metacercaria (Cheng and Wong 1974). Completion of the life cycle occurs when a coral-feeding fish ingests the encysted metacercaria.

### MATERIALS AND METHODS

The effect of parasitism and fish predation on the growth of host corals was examined in an experiment of factorial design: parasitized, unparasitized, protected from fish predation, and exposed to fish predation. Ten colonies of parasitized *Porites compressa* and 10 colonies of unparasitized *P. compressa* were haphazardly collected from patch reefs in Kaneohe Bay, Oahu, Hawaii. Each coral colony was cut in half with a rock saw to produce two genetically identical specimens, with the two halves of each parasitized coral colony having relatively similar levels of parasitism. All corals were stained with Alizarin red stain to permit measurement of linear growth (Barnes 1972), from the stained skeleton to the outer edge of the corallum. The buoyant weight (Jokiel et al. 1976) of each coral colony half was measured at the beginning and end of the 60-day experimental period to determine colony growth rate.

To assess the effect of fish predation, one-half of each coral colony was randomly selected for placement in a wire-mesh predator-exclusion cage. All corals were wired onto metal screens, with wire cages protecting those designated for the fish exclusion treatment. The screens were transported to the windward side of a fringing reef off Coconut Island in Kaneohe Bay and secured to the edge of the reef crest with iron stakes. The corals were placed in an area where coral-feeding butterflyfish (*Chaetodon trifasciatus*, *C. auriga*, *C. lunula*, *C. lineolatus*, and *C. ephippium*) were known to reside. The experimental site was checked weekly to ensure that cages were free of sediment and algae. Corals were removed after 60 days and reweighed. Linear growth was determined by cutting five branches from the center of each coral and measuring the skeletal growth in the axial plane beyond the Alizarin red stain. To assess the parasite lode for each of the parasitized *P. compressa* colonies, a 3 cm × 5 cm quadrat was placed on the side of a coral midway between the base and top of the coral, and the number of cysts visible within that quadrat was recorded. A reading was taken on two opposite sides of the coral. The unparasitized *P. compressa* colonies were thoroughly examined for signs of parasite infection, and the number of cysts found on the colony was recorded.

All statistical analyses were performed with the SAS system (SAS Institute, Inc. 1985). One coral was lost in the caged unparasitized treatment, resulting in a sample size of nine for that group.

### RESULTS

Parasitized *P. compressa* showed a significantly smaller percentage gain in weight than unparasitized *P. compressa* (two-way ANOVA, $P = .0007$, $n = 39$; Table 1). Parasitized *P. compressa* had a mean weight gain of 11.7% (SE ± 0.88) while that of unparasitized *P. compressa* was 23.3% (SE ± 3.2). This reflects a 50% reduction in the growth of parasitized *P. compressa* (Figure 1). A comparison of the mean linear growth also

TABLE 1

Two-way ANOVA for Mean Weight Gain (%) for Parasitized and Unparasitized *P. compressa*

| SOURCE | df | MS* | F | |
|---|---|---|---|---|
| Parasites | 1 | 0.2101 | 13.78 | $P = .0007$ |
| Caging | 1 | 0.0000 | 0.00 | $P = .9668$ |
| Paras × Cage | 1 | 0.0127 | 0.84 | $P = .3662$ |
| Error | 35 | 0.0152 | | |

* Indicates mean square.

TABLE 2

One-way ANOVA for Mean Linear Growth of Parasitized and Unparasitized *P. compressa*

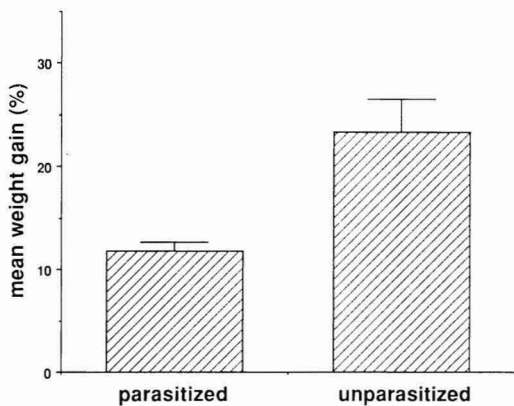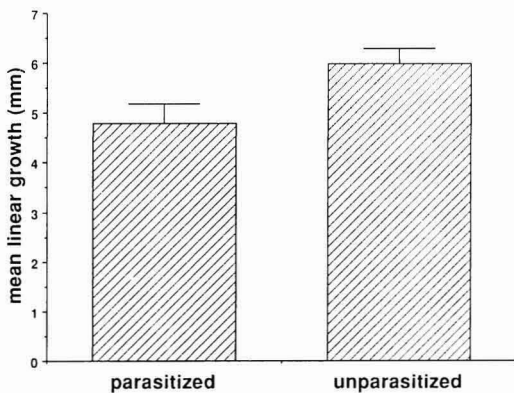| SOURCE | df | MS* | F | |
|---|---|---|---|---|
| Parasites | 1 | 14.7622 | 5.85 | $P = .0204$ |
| Error | 38 | 2.5218 | | |

* Indicates mean square.



FIGURE 1. Comparison of mean weight gain (%) for parasitized and unparasitized *P. compressa*. Bars indicate standard error. (Two-way ANOVA, $P = .0007$, $n = 39$.)



FIGURE 2. Comparison of mean linear growth for parasitized and unparasitized *P. compressa*. Bars indicate standard error. (One-way ANOVA, $P = .0204$, $n = 40$.)
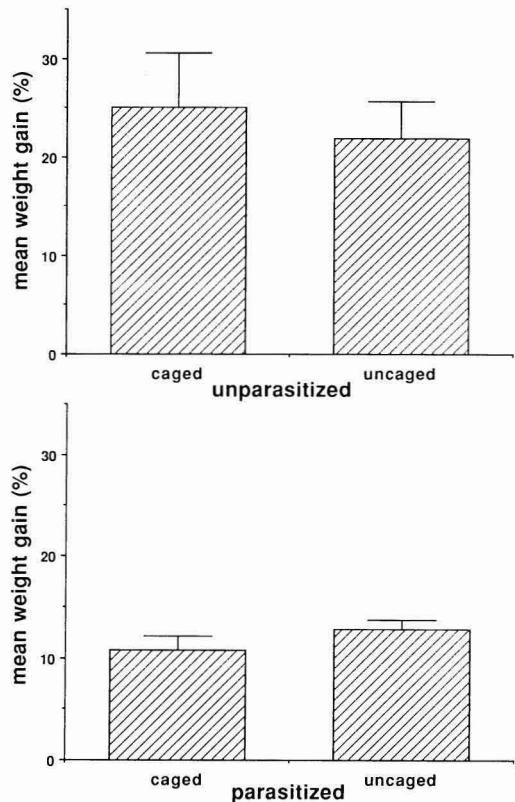


FIGURE 3. Comparison of mean weight gain (%) for caged and uncaged treatments of parasitized *P. compressa* (Wilcoxon signed-rank test, $P = .2867$, n.s., $n = 19$) and unparasitized *P. compressa* (Wilcoxon signed-rank test, $P = .8405$, n.s., $n = 20$). Bars indicate standard error.

showed similar significant differences between the parasitized and unparasitized coral colonies (one-way ANOVA, $P = .0204$; Figure 2, Table 2).

A Wilcoxon signed-rank test showed no significant differences in growth between
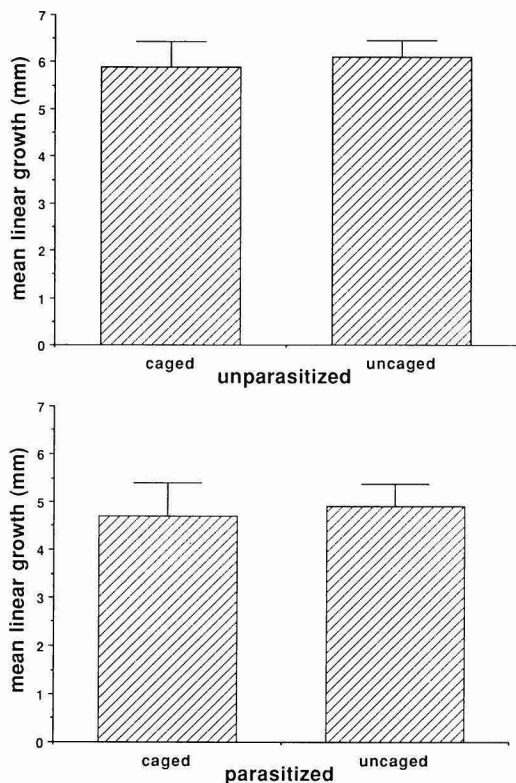
FIGURE 4. Mean linear growth of caged and uncaged treatments for parasitized and unparasitized *P. compressa*. Bars indicate standard error.
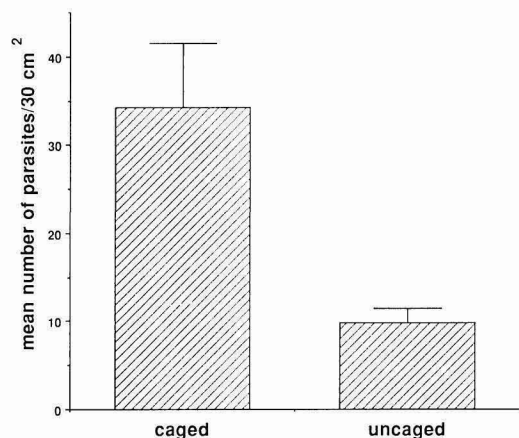


FIGURE 5. Mean number of parasites for caged and uncaged treatments of parasitized *P. compressa*. Bars indicate standard error. (Wilcoxon signed-rank test, $P = .0075$, $n = 20$.)

caged and uncaged *P. compressa* in either group, parasitized or unparasitized (Figure 3). Parasitized *P. compressa* within predator-exclusion cages had a mean weight gain of 10.7% (SE ± 1.5) compared with uncaged parasitized *P. compressa*, which had a mean weight gain of 12.8% (SE ± 0.92) ($T = 0.2867$, n.s.). Unparasitized *P. compressa* within predator-exclusion cages had a mean weight gain of 25.0% (SE ± 5.5) compared with uncaged unparasitized *P. compressa*, which had a mean weight gain of 21.9% (SE ± 3.7) ($T = 0.8405$, n.s.). A comparison of the mean linear growth also showed no significant differences between caged and uncaged *P. compressa* (Figure 4).

At the end of the 60-day experimental period, uncaged parasitized *P. compressa* had significantly fewer parasitic cysts than the *P. compressa* protected from fish predation (Wilcoxon signed rank test, $T = 0.0075$). Caged parasitized *P. compressa* had a mean number of parasites of $34.3 ± 22.9$ cysts/30 cm$^2$ as compared with the uncaged parasitized *P. compressa*, which had a mean number of parasites of $9.8 ± 5.2$ cysts/30 cm$^2$ (Figure 5). The unparasitized *P. compressa* showed signs of becoming newly infected in both caged and uncaged treatments ($x = 2.1$ parasites per coral colony, SD ± 2.3).

## DISCUSSION

A parasite, by definition, is an organism that lives in intimate association with and has an unfavorable impact on another organism, its host (Minchella 1985). *Porites compressa* infected with *Plagioporus* sp. had an overall reduction in growth rate consistent with this definition of a parasitic infection. Some stages of parasites derive their food directly from the host, but after encystment the metacercariae of *Plagioporus* sp. place no metabolic demands on the coral host. However, *Plagioporus* affects *P. compressa* in a number of other ways, which suggests that there is some cost to the polyps and that their contribution to the growth of the coral colony may be reduced. First, infected polyps appear swollen, and they are unable to retract adequately into

their calices. Second, infected polyps have a reduced number of zooxanthellae; because coral growth is enhanced by the presence of their symbiotic zooxanthellae (Muscatine 1973), the lack of zooxanthellae in infected polyps may have contributed to the reduced growth. Last, infected polyps produce small, abnormal skeletal protrusions; Cheng and Wong (1974), who found significantly less ionic calcium in the polyps of parasitized *P. compressa* as compared with unparasitized *P. compressa*, suggested that the presence of *Plagioporus* metacercariae in a polyp results in the disruption of normal calcium deposition by polyp epidermal cells. It should also be noted that the corals used in this experiment had high levels of infection (Figure 5), which may have resulted in the large reduction in growth exhibited by parasitized *P. compressa*. It is hypothesized that a correlation exists between level of parasite infection and degree of reduced coral growth, but further investigation is needed to verify this.

Different genotypes of *P. compressa* may have various susceptibilities to parasitism. However, the unparasitized corals in this study showed signs of infection after 2 months on a reef where *Plagioporus* occurs, suggesting that the lack of previous infection of unparasitized *P. compressa* was due to lack of exposure to the parasite rather than an inherent immunity.

Presence of a larval parasite often increases the rate of predation on the intermediate host by the definitive host (Hoogenboom and Dijkstra 1987, Quinn et al. 1987, Godin and Sproul 1988), thereby increasing the opportunities for the parasite to complete its life cycle. It follows that selection would favor the parasite whose encystment results in an increase in the vulnerability of the intermediate host to predation by the definitive host. This would be particularly important where the parasites' encysted metacercarial stage has a limited period of viability, with senescence occurring if it is not ingested by the definitive host (Eramus 1972). The parasite would then have only a certain amount of time in which to complete its life cycle. This seems to be consistent with *Plagioporus*, which does have a finite metacercarial stage that usually

seneces within a few months (unpublished data). Coral-feeding butterflyfish are attracted to and readily feed on infected corals. In addition, parasitized *P. compressa* exposed to fish predation did experience a dramatic reduction in the number of cysts, suggesting an enhancement of the parasites' rate of transmission.

There were no significant differences in growth found between *P. compressa* exposed to fish predation and those protected from fish predation. This was true for both parasitized *P. compressa* and unparasitized *P. compressa*. This is in contrast with other studies that have shown that corallivorous fish can limit the growth and distribution of corals (Neudecker 1979, Cox 1986). However, the previous studies used species of coral other than *Porites*. *Porites* has a lower energy content than other species of coral found on the experimental reef (Tricas 1985), and it is not the preferred food of most butterflyfishes (Reese 1977, Tricas 1985). Therefore, the level of fish predation on *P. compressa* may not have been large enough to significantly affect its growth. This is a reasonable explanation for unparasitized *P. compressa*; however, the reduction in number of parasitic cysts on parasitized *P. compressa* indicates that predation was probably occurring. Why then was the growth rate of parasitized *P. compressa* not reduced? The effects of corallivores are probably similar to those of herbivorous grazers, where only partial predation occurs without complete loss of the coral polyp (Cox 1983). I observed that corals regenerated healthy polyps after removal of the encysted parasite. The negative effect of fish predation may have been offset by the positive effect of the parasite removal.

One might question why fish would evolve to feed on infected polyps, thereby becoming parasitized themselves, instead of avoiding the cysts. I can only speculate about this, but there are several hypothetical explanations:

1. The parasite residing in the fish may have adopted the "prudent parasite" strategy (Holmes 1983) in which the parasite produces minimal damage in the host. There would then be a lack of selective pressure on the fish to avoid the infected polyps.

2. There may be a positive selective pressure on the fish to maximize feeding efficiency by eating the infected polyps that are no longer able to retract into their calices.

3. It may also be a combination of the above two hypotheses in which there is a positive selection to maximize feeding efficiency by feeding on the infected polyps combined with minimal parasitic damage to the host, creating a lack of pressure to avoid the infected polyps.

In summary, the presence of the parasite *Plagioporus* has produced an alteration in both the appearance and the behavior of its intermediate host, *P. compressa*. These alterations have resulted in increased predation on *P. compressa*, facilitating the transmission of the parasite to its probable final host. The growth of *P. compressa*, however, was not significantly affected by exposure to fish predation. Encystment of *Plagioporus* metacercariae was found to have a negative impact on the growth of *P. compressa*.

## LITERATURE CITED

BARNES, D. J. 1972. The structure and formation of growth ridges in scleractinian coral skeletons. Proc. R. Soc. London 182:331–350.

BETHEL, W. M., and J. C. HOLMES. 1977. Increased vulnerability of amphipods to predation owing to altered behavior induced by larval acanthocephalans. Can. J. Zool. 55:110–115.

CARNEY, W. P. 1969. Behavioral and morphological changes in carpenter ants harboring dicrocoeliid metacercariae. Am. Midl. Nat. 82(2): 605–611.

CHENG, T. C. 1986. General parasitology. Academic Press, Orlando, Florida.

CHENG, T. C., and A. K. WONG. 1974. Chemical, histochemical and histopathological studies on corals, *Porites* spp., parasitized by trematode metacercariae. J. Invertebr. Pathol. 23:303–317.

COX, E. F. 1983. Aspects of corallivory by *Chaetodon unimaculatus* in Kaneohe Bay, Oahu. M.S. thesis, University of Hawaii.

———. 1986. The effects of a selective corallivore on growth rates and competition for space between two species of Hawaiian corals. J. Exp. Mar. Biol. Ecol. 101:161–174.

DOGIEL, V. A., G. K. PETRUSHEVSKI, and Y. I. POLYANSKI. 1961. Parisitology of fishes. Oliver and Boyd, Edinburgh and London.

ERAMUS, D. A. 1972. The biology of trematodes. Crane, Russak and Co., New York.

GODIN, J., and C. SPROUL. 1988. Risk taking in parasitized stickle-backs under threat of predation: Effects of energetic need and food availability. Can. J. Zool. 66:2360–2367.

HOLMES, J. C. 1983. Evolutionary relationships between parasitic helminths and their hosts. Pages 161–185 *in* D. F. Futuyma and M. Slatkin, eds. Coevolution. Sinauer Press, Sunderland, Massachusetts.

HOLMES, J. C., and W. M. BETHEL. 1972. Modifications of intermediate host behavior by parasites. Pages 123–149 *in* E. V. Canning and C. A. Wright, eds. Behavioral aspects of parasite transmission. Academic Press, New York.

HOOGENBOOM, I., and C. DIJKSTRA. 1987. *Sarcocystis cernae*: A parasite increasing the risk of predation of its intermediate host, *Microtus arvalis*. Oecologia (Berlin) 74:86–92.

JOKIEL, P. L., J. E. MARAGOS, and L. FRANZISKET. 1976. Measurement of skeletal growth in scleractinian corals by buoyant weight technique. *In* D. R. Stoddart and R. E. Johannes. Handbook of coral reef research methods. UNESCO Monogr. Oceanogr. Methodol.

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

MUSCATINE, L. 1973. Nutrition of corals. Pages 98–115 *in* O. A. Jones and R. Endean, eds. Biology and geology of coral reefs, Vol. II, Biology I. Academic Press, New York.

NEUDECKER, S. 1979. Effects of grazing and browsing fishes on the zonation of corals in Guam. Ecology 60:666–672.

OLSEN, O. W. 1962. Animal parasites: Their

biology and life cycles. Burgess Publ., Minneapolis, Minnesota.

QUINN, S., R. BROOKS, and R. CAWTHORN. 1987. Effects of the protozoan parasite *Sarcocystis rauschorum* on open-field behavior in its intermediate vertebrate host, *Dicrostonyx richardsoni*. J. Parasitol. 73(2): 265–271.

REESE, E. S. 1977. Coevolution of corals and coral feeding fishes of family Chaetodontidae. Proc. 3rd Int. Coral Reef Symp. 1:267–274.

ROTHSCHILD, M. 1962. Changes in behavior in the intermediate hosts of trematodes. Nature (London) 193:1312–1313.

SAS INSTITUTE, INC. 1985. User's guide: Statistics. Version 5. SAS Institute, Inc., Cary, North Carolina.

SMYTH, J. D., and D. W. HALTON. 1983. The physiology of trematodes. 2d ed. Cambridge University Press, Cambridge.

TRICAS, T. C. 1985. The economics of foraging in coral-feeding butterflyfishes of Hawaii. Proc. 5th Int. Coral Reef Congr., Tahiti 5:409–414.