THE EFFECT OF THE ECTOPARASITIC PYRAMIDELLID SNAIL, *BOONEA IMPRESSA*, ON THE GROWTH AND HEALTH OF OYSTERS, *CRASSOSTREA VIRGINICA*, UNDER FIELD CONDITIONS

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ABSTRACT

Boonea (= *Odostomia*) *impressa* are contagiously distributed on oyster reefs so that some oysters are parasitized more than others. The parasite's mobility and the ability of oysters to recover from snail parasitism may be important in assessing the impact of parasitism on oyster populations. During a 4-week exposure period in the field, *B. impressa* reduced American oyster, *Crassostrea virginica*, growth rate and increased the intensity of infection by the protozoan, *Perkinsus* (= *Dermocystidium*) *marinus*, but produced few changes in the oyster's biochemical composition because, although net productivity was reduced, the oysters retained a net positive energy balance (assimilation > respiration). During a 4-week recovery period, growth rate returned to normal (control) levels, but infection by *P. marinus* continued to intensify in previously parasitized oysters kept *B. impressa*-free. Most changes in biochemical composition during recovery, including increased lipid and glycogen contents, could be attributed to the continuing increase in infection intensity of *P. marinus*. Consequently, the temporal stability and size of snail patches, particularly as they regulate infection by *P. marinus*, may be the most important factors influencing the impact of *B. impressa* on oyster reefs.

Parasitism can be an important factor affecting the population dynamics (Wickham 1986; Brown and Brown 1986; Kabat 1986) and health (Brockelman 1978; Mohamed and Ishak 1981; Ford 1986) of host species. Three parasites are known to be especially important in oysters. *Perkinsus* (= *Dermocystidium*) *marinus*, *Haplosporidium nelsoni* (MSX), and *Boonea* (= *Odostomia*) *impressa* detrimentally affect oyster growth, health, and biochemical composition (Mengebier and Wood 1969; Feng et al. 1970; Soniat and Koenig 1982; White et al. 1984; Ford 1986; Ward and Langdon 1986; White et al. 1988, in press).

The pyramidellid gastropod, *Boonea impressa*, is one of a widely distributed group of parasitic, marine opisthobranchs (Fretter and Graham 1949; Fretter 1951; Allen 1958). *Boonea impressa* removes nutrients directly from its host by piercing the flesh with a hollow stylet and sucking the body fluids using a buccal pump (Fretter and Graham 1949; Fretter 1951; Allen 1958). The most common host of *B. impressa* is *Crassostrea virginica* (Hopkins 1956; Allen 1958; Wells 1959) but, like other odostomians, it is not entirely host specific (Wells 1959; Robertson 1978; Robertson and Mau-Lastovicka 1979). Found abundantly on oyster reefs from Massachusetts to the Gulf of Mexico, *B. impressa* has been reported in numbers as high as 100 per oyster (Hopkins 1956).

Under laboratory conditions, *B. impressa* reduced oyster growth rates as the result of both direct removal of assimilated carbon from the oyster and direct interference with the oyster's ability to feed (White et al. 1984, 1988; Ward and Langdon 1986). Not surprisingly, parasitism by *B. impressa* produced changes in the biochemical composition of oyster tissue. Parasitism by 15 snails, a relatively high field density found in dense snail patches decreased carbohydrate and free amino acid content and increased lipid content of mantle tissue. Reproduction was affected as well (White et al. in press).

Perkinsus marinus is an important cause of mortality of oysters in the Gulf of Mexico (Mackin 1962; Ray 1966a; Hofstetter 1977). Sublethal effects include reduced growth (Menzel and Hopkins 1955; Ray et al. 1953) and changes in biochemical composition (Soniat and Koenig 1982; White et al. in press). *Perkinsus marinus* can be transmitted from one oyster to another through the water (Ray 1954; Mackin 1962; Andrews 1965) or by *B. impressa* feeding (White et al. 1987). The intensity of infection by *P. marinus* is also increased in *B. impressa*-parasitized oysters (White et al. 1987).

Usually stress affects organisms by altering normal metabolic activity. Many organisms may be able

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to return to normal metabolic condition after the stress is removed (Pörtner et al. 1979; Pickering et al. 1982; Kendall et al. 1984). This ability, termed recovery, is an important adaptation to changes in the natural environment. Recovery is not always complete nor does it occur over relatively short time scales in all cases. The time required for recovery varies depending on the parameter being studied, the stress that was applied, and the species involved (Kendall et al. 1984; Haux et al. 1985; Neff et al. 1985). Apparently deleterious effects occurring after exposure, during the so-called recovery period, are well described (e.g. Kendall et al. 1984; Powell et al. 1984). White et al. (1984) showed that *C. virginica* could attain normal growth rates within one week after *B. impressa* were removed. However, growth rates frequently return to normal more rapidly than other metabolic parameters (Kendall et al. 1984). *Boonea impressa* are extremely mobile and are more contagiously distributed than their hosts, so that some oysters are highly parasitized while others remain parasite free (Powell et al. 1987). Individual *B. impressa* change hosts often but typically move between existing aggregates (Wilson et al. in press). Therefore, refugia from parasitism may exist and recovery may be important in assessing the overall impact of *B. impressa* on oysters.

Stress, produced by laboratory conditions, frequently accompanies laboratory experimentation (e.g., Koenig et al. 1981; Powell et al. 1984; Kukal and Kevan 1987). The effect of *B. impressa* on *C. virginica* has been assessed primarily through laboratory experimentation. Consequently, we examined the effect of *B. impressa* on *C. virginica* under field conditions and assessed the ability of oysters to recover normal growth rates and biochemical composition once snail parasitism ceased.

MATERIALS AND METHODS

Field Study

Oysters and snails used in this study were collected at Goose Island State Recreation Area near Rockport, TX. The oysters were weighed on a Mettler balance using the underwater method of Andrews (1961). Fifteen oysters (precontrols) were sacrificed to define the biochemical composition of the oysters and *Perkinsus marinus* levels that existed naturally at the collection site. The rest of the oysters were placed in semi-enclosed plexiglass domes (see figure 2 in Kendall et al. 1984 for description) in a tidal creek near the Aransas Pass Lighthouse on Lydia Ann Channel, near Port Aransas, TX. The domes allowed water to circulate over the oysters, while excluding such large predators as oyster drills and crabs. *Boonea impressa*, however, could readily move into or out of the domes.

Two of the four domes (20 oysters per dome) contained oysters exposed to *B. impressa* at a concentration of 10 snails per oyster, a level of parasitism commonly observed on reefs in the collection area (White et al. 1984). The domes were positioned so that the probability of snails moving from the exposure domes with parasitized oysters to the control domes was minimized (Fig. 1). Snails on each experimental oyster were counted twice weekly for 4 weeks. No *B. impressa* were ever found on control oysters. *Boonea impressa* did emigrate from the exposure domes, however, so snails were added as needed to maintain the 10:1, snail:oyster ratio. Control oysters were handled the same as experimentals each week in an effort to minimize differential effects caused by handling stress (see Pickering et al. 1982; Andrews and Hewatt [1957] were unable to find any effect of handling on *P. marinus* infection in oysters).

At the end of 4 weeks, the oysters were reweighed and half from each dome were sacrificed. The remaining oysters were replaced in the field for a 4-wk recovery period without *B. impressa*. All *B. impressa* were removed from the previously parasitized oysters by hand prior to replacement. The domes were visited twice weekly and each oyster handled as before. No *B. impressa* were found on the oysters during the recovery period.

Laboratory Analysis

Perkinsus marinus infection was measured in each oyster by incubating a small piece of mantle tissue in thioglycollate medium by the method of Ray (1966b). Intensity of infection was based on a semi-quantitative 0 to 5 rating assigned during microscopic inspection of the tissue after treatment with Lugol's solution (Mackin 1962). Small pieces of gonadal tissue were preserved in Bouin's fixative, sectioned and stained in 0.5% toluidine blue (Preece 1972) for gonadal analysis. Mantle cavity volume was determined by filling the shells with silicon caulking. Oyster growth, as expressed by shell deposition, was measured using the underwater weighing method. Condition index was derived by dividing the total lyophilized dry weight by the mantle cavity volume (Lawrence and Scott 1982).

The mantle and adductor muscle from each oyster

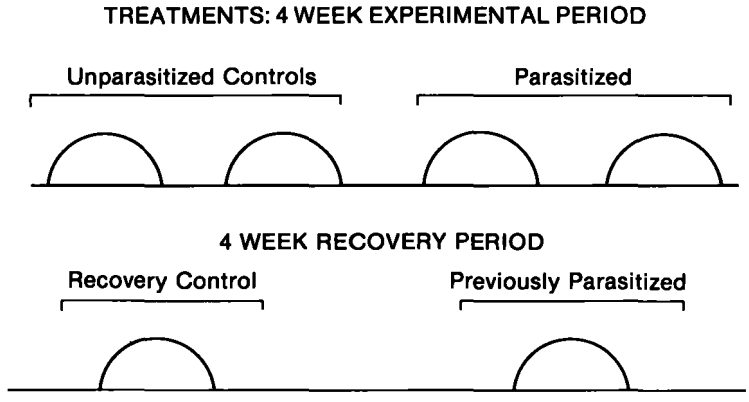

FIGURE 1.—Position of plexiglass domes during 4-wk experimental period and during 4-wk recovery period.

were frozen immediately on dry ice and kept at −40°C until lyophilized. Prior to biochemical analysis, lyophilized tissues were weighed and homogenized on ice. Total lipids were isolated from a portion of the homogenate using the water:methanol: chloroform method of Folch et al. (1957) with a modified ratio of 0.8:2.1. This isolation produced a two phase system and an insoluble pellet after centrifugation in the cold. The pellet was used for protein and glycogen assays. The water phase was used for amino acid analysis. The organic phase was divided and dried under N_2 gas at 40°C. One half was used to determine lipid phosphate concentration by the spectrophotometric method of White et al. (1979). The other half was dissolved in chloroform to which nonadecanoic acid was added as an internal standard. The redissolved lipids were fractionated on a silicic acid (Unisil,[3] 100–200 mesh) column. Neutral lipids were recovered by eluting with 10 times the column volume of chloroform. The chloroform was removed under N_2 at 40°C (Gehron and White 1982). The resulting chloroform fraction, containing fatty acid methyl esters, was analyzed by gas chromatography using a capillary, nonpolar, methyl silicone high performance column and flame ionization detector.

The amino acids were analyzed on a Dionex 3000 amino acid analyzer. Because residual chloroform interfered with the analysis, a second extraction was performed on the sample in the cold using a chloroform:water ratio of 1:3; α-amino-n-butyric acid (2.5 μmole · mL^{-1} sample) was used as an internal standard. The amino acids accounting for the bulk of the free amino acid (FAA) pool, taurine, hypotaurine, aspartic acid, serine, threonine, glutamine, glutamate, glycine, and alanine, were separated using a lithium citrate buffer and measured using *o*-phthalaldehyde as the detecting compound.

Total soluble protein was estimated by Peterson's (1977) modification of the Lowry method. Protein was precipitated using a final concentration of 10% trichloroacetic acid in the cold. After centrifugation at 4°C the resultant pellets were resuspended in 50:50, 10% SDS:0.8N NaOH for 30 minutes. Replicate samples were centrifuged and analyzed spectrophotometrically for protein concentration. Bovine serum albumin (Sigma) was used as the standard.

Glycogen was degraded to glucose enzymatically with amyloglucosidase (Carr and Neff 1984). After preincubation with amyloglucosidase, glucose was measured using the glucose oxidase-peroxidase procedure (Sigma glucose kit) (Roehrig and Allred 1974). Rat liver glycogen (Sigma) was used as a standard.

Statistical Analysis

To assess the effect of *Boonea impressa* on the oysters during the 4-wk exposure period, the level of snail parasitism for each oyster had to be determined. Because the number of snails on each oyster was counted only every third day, the total number of snails that parasitized each oyster was estimated by assuming that the same number of snails were present on days between counts as found on the previous visit. The total number of snail-days per oyster, the sum of the number of snails present on each day during the 4-wk exposure, will be referred to as the snail scale. For example, during the first week of the exposure period, one oyster had 15

[3]Reference to trade names does not imply endorsement by the National Marine Fisheries Service, NOAA.

snails on Monday, 10 on Wednesday, and 4 on Saturday; therefore Tuesday was assigned 15 snails, Thursday and Friday 10 snails, and the total for the week was 64 snail-days.

Except where noted in the text, results were analyzed by multiple analysis of covariance (MANCOVA) using ranked data followed by Duncan's multiple range tests ($\alpha = 0.05$) to locate significant differences within the MANCOVA. Mantle cavity volume was used as the covariate for comparing levels of *P. marinus* infection intensity, dry weight, and shell weight gain between treatment groups (e.g., parasitized and control oysters). Similar tests comparing biochemical parameters between treatment groups included the intensity of *P. marinus* infection and tissue dry weight as covariates. When the requirement of parallelism was not met, the MANCOVA was modified as described by Smith and Coull (1987). *Perkinsus marinus* infection intensity was used as both a dependent and independent variable because, although *B. impressa* can influence the intensity of *P. marinus* infection (White et al. 1987), *P. marinus* itself can affect the biochemical composition of oysters (Soniat and Koenig 1982; White et al. in press). Only parasitized oysters (or those recovering from parasitism) were used in analyses of the effect of snail scale (intensity of parasitism).

The FAA pool was analyzed three ways: 1) the entire pool, 2) the pool minus taurine and hypotaurine because these are the only amino acids not found in protein, and 3) after exclusion of the major components (taurine, hypotaurine, glycine, and alanine) so that changes in the lesser constituents of the pool could be examined.

All analyses for the 4-wk exposure period used MANCOVA analyses with nested variables, which took into account which of the 4 domes the oysters were in during the exposure period. Overall, significant differences in biochemical composition (i.e., amino acid content, glycogen, protein, etc.) between the 2 nonparasitized domes or between the 2 snail parasitized domes did not occur more frequently than expected by chance (Binomial Test, $\alpha = 0.05$). Therefore, the two equivalently treated domes of each treatment group (Fig. 1) were lumped together for comparison with precontrol and recovery oysters. Nevertheless, the experimental design represents a case of pseudoreplication; hence the caveats of Hurlbert (1984) should be considered when reviewing the statistical analysis.

RESULTS

Growth, *Perkinsus marinus* Intensity, and Reproduction

Boonea impressa affected oyster growth rate, the intensity of infection by *P. marinus*, and reproductive state. The mean initial shell weights of the precontrol, control, and parasitized groups were not significantly different from each other. By the end of the 4-wk exposure period, both parasitized and unparasitized oysters had gained weight, but oysters parasitized by *B. impressa* gained significantly less weight than unparasitized oysters (Table 1). Oysters with more snails typically gained less weight than oysters with fewer snails. The relationship between the intensity of *B. impressa* parasitism (snail scale) and weight gain among parasitized oysters was significant (Spearman's rank, $P = 0.03$; weight gain normalized to initial weight, $P = 0.001$, Fig. 2).

Mantle cavity volume did not vary among any of the treatment or recovery groups. Condition index was not significantly different between treatment groups or between recovery groups. Condition index was significantly higher in the two recovery

TABLE 1.—Mean and standard deviations for initial shell weight (g) and average shell weight gain per oyster during the 4-wk exposure period and 4-wk recovery period. Significance levels (sig.) from Duncan's multiple range test ($\alpha = 0.05$). A test was restricted to a single column. Groups having the same letter within a column are not significantly different.

	Initial weight		Weight gained during 4-wk exposure		Weight gained during 4-wk recovery	
Group	Mean ± SD	Sig.	Mean ± SD	Sig.	Mean ± SD	Sig.
Precontrol ($n = 15$)	22.82 ± 12.16	A				
Treatment						
Control ($n = 40$)	17.16 ± 8.48	A	3.49 ± 1.21	A		
Parasitized ($n = 40$)	16.84 ± 7.47	A	2.54 ± 1.31	B		
Recovery						
Control ($n = 6$)					2.16 ± 0.73	B
Previously parasitized ($n = 15$)					2.89 ± 1.05	A

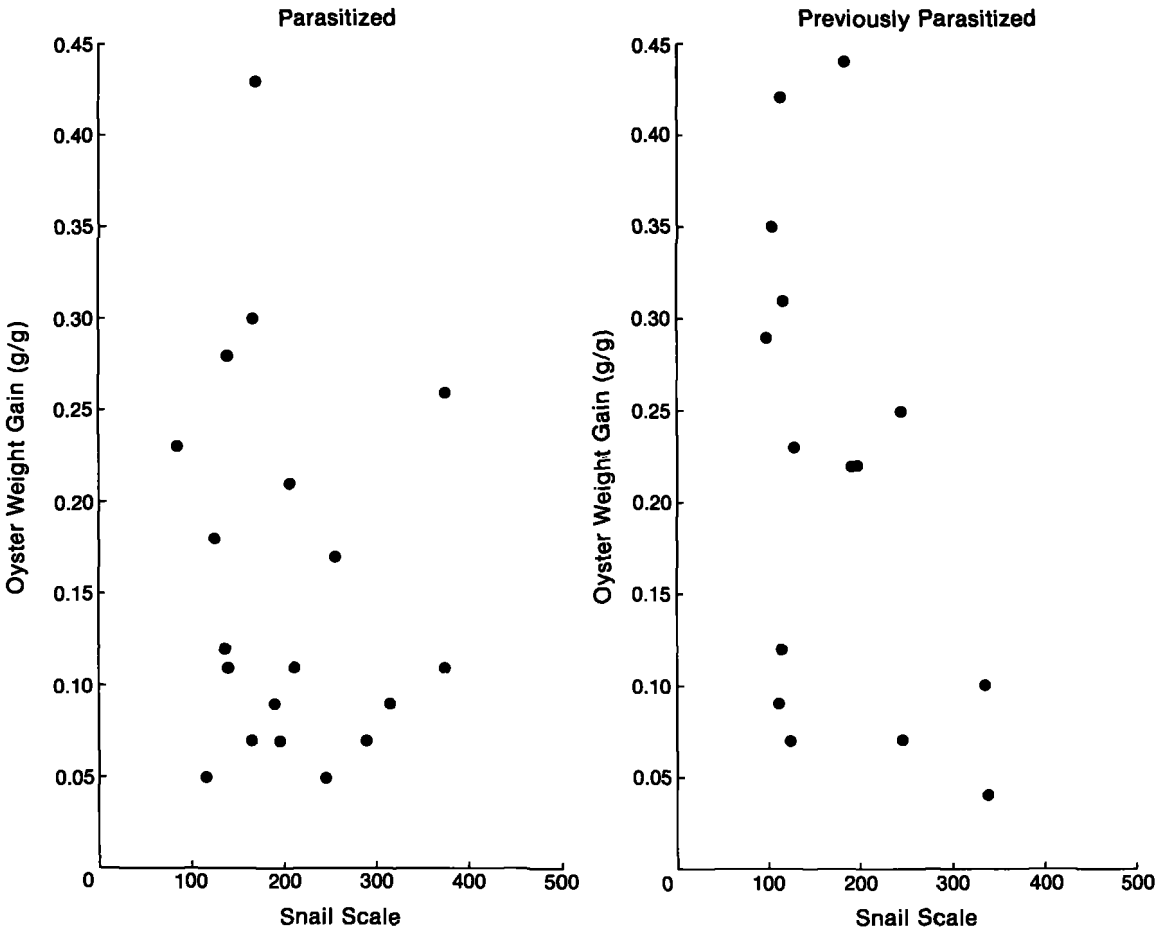

FIGURE 2.—Left: Weight gain (g) per g initial weight of individual snail-parasitized oysters after the 4-wk experimental period as a function of the level of *Boonea impressa* parasitism, or snail scale. Right: Weight gain (g) per g initial weight of individual oysters after the 4-wk recovery period as a function of the level of *B. impressa* (snail scale) during the 4-wk treatment period.

groups than in the treatment and precontrol groups (Table 2).

During the recovery period, when all oysters were parasite-free, the previously parasitized oysters (those with snails during the treatment period) gained significantly more weight than the recovery controls (Table 1). Oysters which were previously parasitized by fewer *B. impressa* during the treatment period gained more weight during the recovery period than those that were previously parasitized by more snails (Spearman's Rank correlation, $P = 0.07$, Fig. 2), but the relationship was considerably weaker than during the treatment period.

The intensity of infection by *P. marinus* increased throughout the experiment in parasitized oysters so that, after recovery, previously parasitized oysters had higher prevalences and intensities of infection than they did after 4 weeks of parasitism; these values in turn were higher than the precontrol values at the experiment's inception (Table 3). Exactly the opposite trend occurred in control oysters. Recovery controls had the lowest values of prevalence and intensity. Consequently, after the recovery period, previously parasitized oysters had significantly higher intensities of infection than recovery controls. The proportion of infected individuals (33% of the controls, 93% of the previously parasitized oysters) was significantly higher as well (χ^2, $P < 0.05$). Within the parasitized oysters, the intensity of parasitism (snail scale) did not correlate with the increase in intensity of *P. marinus* infection during either the treatment or recovery period (Fig. 3, Spearman's Rank, $P > 0.05$; the interaction

TABLE 2.—Means and standard deviations for volume (mL), condition index ($g \cdot mL^{-1}$) and dry weight (g). Significance levels (sig.) from Duncan's multiple range test ($\alpha = 0.05$). A test was restricted to a single column. Groups having the same letter within a column are not significantly different. *n*, see Table 1.

Group	Volume Mean ± SD	Sig.	Condition index Mean ± SD	Sig.	Dry weight Mean ± SD	Sig.
Precontrol	10.4 ± 4.1	A	3.4 ± 0.8	C	0.35 ± 0.14	AB
Treatment						
Control	11.2 ± 3.6	A	5.4 ± 1.0	B	0.34 ± 0.11	AB
Parasitized	9.2 ± 1.7	A	5.5 ± 1.0	B	0.28 ± 0.12	B
Recovery						
Control	9.5 ± 4.6	A	7.1 ± 1.5	A	0.32 ± 0.14	B
Previously parasitized	10.8 ± 3.8	A	7.7 ± 1.5	A	0.51 ± 0.24	A

TABLE 3.—Mean and standard deviations for *Perkinsus marinus* infection intensity among groups. Significance levels from Duncan's multiple range test ($\alpha = 0.05$) apply to the infection intensity data. Groups with the same letter are not significantly different. Percent incidence is the number of oysters infected divided by the number of oysters in the sample. Infection intensity was calculated using the 5-point scale of Mackin (1962). *n*, see Table 1.

Group	*Perkinsus marinus* Infection Intensity Mean ± SD	Significance	Prevalence
Precontrol	1.47 ± 1.45	AB	66.6
Treatment			
Control	1.10 ± 1.06	AB	65.0
Parasitized	1.63 ± 1.32	AB	79.0
Recovery			
Control	0.83 ± 1.60	B	33.3
Previously parasitized	2.21 ± 1.38	A	92.9

TABLE 4.—Results of histological examination of gonadal samples for each group. Significance levels (sig.) from Duncan's multiple range test ($\alpha = 0.05$) are for differences in the number of eggs present per female in a histological section. Percent of females spawning is the number of females with eggs observed in the gonoducts divided by the number of females in the sample.

Group	Sex Female	Male	Eggs present Mean ± SD	Sig.	Percent of females spawning
Precontrol	7	7	31.1 ± 4.4	A	86.0
Treatment					
Control	6	4	36.6 ± 11.0	A	100.0
Parasitized	4	6	21.6 ± 3.9	B	100.0
Recovery					
Control	5	1	37.6 ± 6.0	A	80.0
Previously parasitized	11	4	42.9 ± 9.8	A	81.0

term in the MANCOVA was also nonsignificant).

The number of eggs per microscopic field estimated from histological sections, was significantly lower in parasitized oysters after the 4-wk treatment period (Table 4). No differences between controls and previously parasitized oysters were present after the 4-wk recovery period. The proportion of oysters ready to spawn (female oysters which had eggs visible in the gonoducts) was not significantly different in parasitized and unparasitized oysters during treatment or recovery (χ^2, $\alpha = 0.05$).

Biochemical Composition

Mean levels of the various biochemical components measured in adductor muscle and mantle tissue are given in Tables 5 and 6. A comparison of precontrol, treatment control, and recovery control oysters documents the changes in biochemical composition produced by handling stress and natural environmental changes that occurred during the experiment. Glycogen increased significantly in the mantle tissue during the 4-wk treatment period, then decreased during the recovery period (Table 7). Glycogen in the adductor muscle also decreased during the recovery period (Table 7). Hypotaurine increased in both tissues during the treatment period (Table 8). Most other FAA and the total pool dropped in concentration in the mantle tissue in the first 4 weeks, but then stabilized. In contrast, in the adductor muscle, significant increases in glycine and hypotaurine during the treatment period were offset by a significant decrease in alanine, so the total pool changed little. Again, the FAA pool stabilized during the first 4 weeks. Hence, there was little difference in treatment and recovery controls in any measured biochemical component.

Snail parasitized and control oysters did not differ significantly in the concentration of any of the biochemical components in either the mantle or adductor muscle (Tables 7, 8). Only one biochemical parameter differed between the recovery control

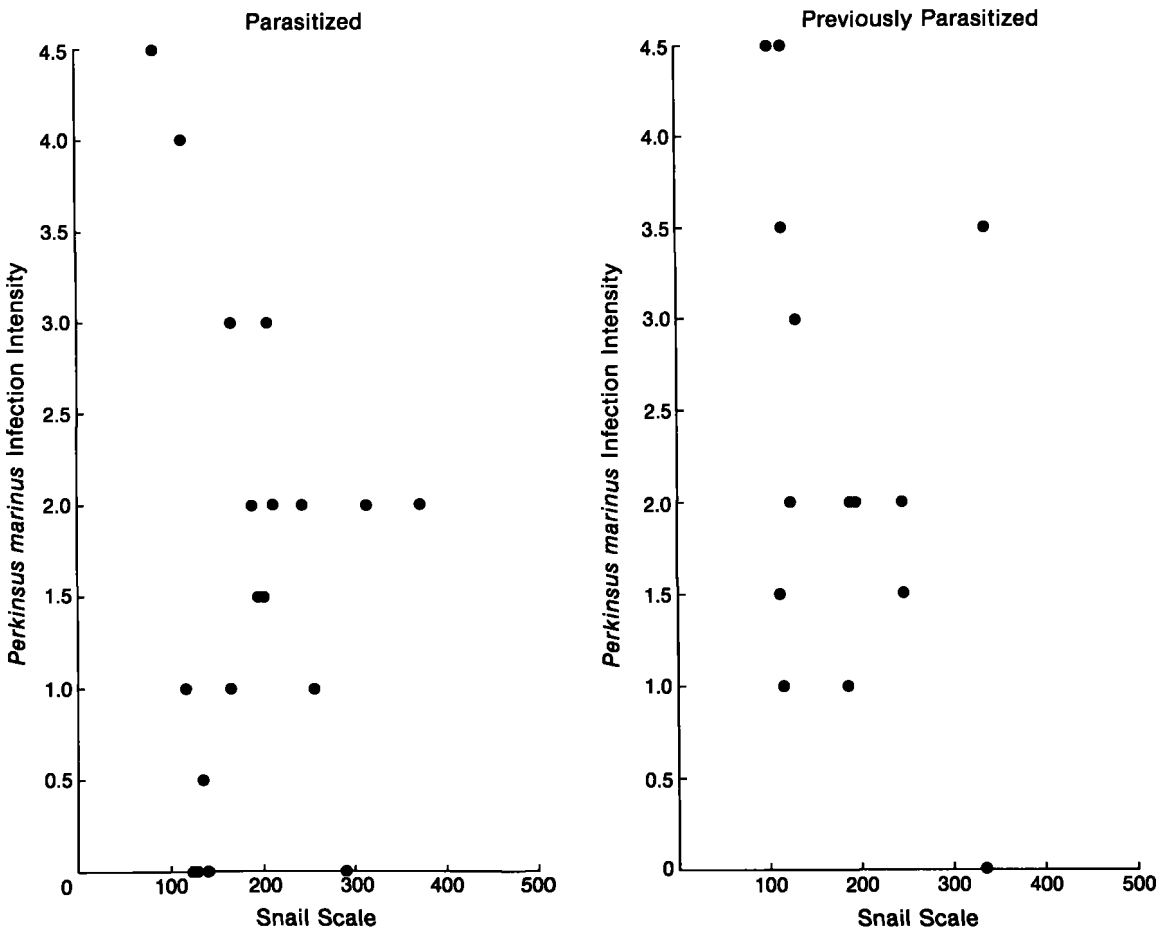

FIGURE 3.—Left: Intensity of *Perkinsus marinus* infection in snail-parasitized oysters as a function of the level of snail parasitism after the 4-wk treatment period. Right: Intensity of *P. marinus* infection in previously snail-parasitized oysters as a function of the level of snail parasitism after the 4-wk recovery period.

and previously parasitized oysters. Recovery controls had significantly less glycogen in the mantle tissue (Table 7). No biochemical component was significantly correlated with the intensity of snail parasitism (snail scale) in parasitized oysters during the 4-wk treatment period except taurine in the adductor muscle (Table 9). A similar comparison, using the previously parasitized oysters after the 4-wk recovery yielded only two significant correlations with snail scale; soluble protein and hypotaurine content. In addition, lipid phosphate in the mantle tissue and fatty acid content in the adductor muscle were significantly correlated with the intensity of *Perkinsus marinus* infection after the recovery period (all recovery oysters, controls and previously parasitized, were included in the analysis, Table 9).

DISCUSSION

Oysters averaged as few as about 3 and as many as 14 snails per day during the 4-wk treatment period; however, only 11% of the oysters had 10 or more snails per day and 76% had fewer than 9 per day. This range of parasitism is typical for many reefs in the experimental area (Copano Bay-Aransas Bay, TX—White et al. 1984; Powell et al. 1987) and corresponds to the lower parasite levels used by White et al. (1988, in press) in laboratory studies on these animals.

The effects of snail parasitism were minor on a biochemical level, but substantial on an organismal level. Growth, reproductive capacity, and health, as measured by *Perkinsus marinus* infection, were significantly affected. Other odostomians also re-

TABLE 5.—Means and standard deviations for all biochemical components in mantle tissue. FAA = total of the 9 free amino acids measured; FAA- = FAA less taurine and hypotaurine; FAA– = FAA less taurine, hypotaurine, glycine, and alanine; amino acids in μmoles · mL^{-1}; other tissue components in mg · g dry wt^{-1}.

		Treatment		Recovery	
	Precontrol (n = 15) $\bar{x}$ ± SD	Control (n = 10) $\bar{x}$ ± SD	Parasitized (n = 10) $\bar{x}$ ± SD	Control (n = 6) $\bar{x}$ ± SD	Previously parasitized (n = 15) $\bar{x}$ ± SD
FAA	351 ± 94	240 ± 39	280 ± 38	294 ± 64	270 ± 64
FAA-	220 ± 67	116 ± 14	121 ± 15	134 ± 50	117 ± 43
FAA–	57 ± 18	42 ± 6	46 ± 7	49 ± 26	42 ± 13
Taurine	102 ± 33	95 ± 27	119 ± 24	108 ± 15	99 ± 21
Hypotaurine	27 ± 10	39 ± 10	50 ± 23	49 ± 10	54 ± 20
Aspartic acid	22 ± 5	15 ± 2	19 ± 3	22 ± 13	18 ± 5
Serine	8 ± 5	5 ± 2	6 ± 2	5 ± 3	5 ± 3
Threonine	3 ± 1	1 ± 0.2	1 ± 0.7	1 ± 0.4	1 ± 0.5
Glutamine	8 ± 5	2 ± 1	3 ± 0.8	4 ± 3	5 ± 3
Glutamic acid	14 ± 5	16 ± 3	18 ± 5	14 ± 5	12 ± 4
Glycine	71 ± 23	33 ± 5	38 ± 10	44 ± 31	38 ± 17
Alanine	87 ± 33	36 ± 8	38 ± 10	31 ± 10	34 ± 16
Protein	62 ± 45	28 ± 39	60 ± 57	45 ± 40	23 ± 16
Glycogen	28 ± 22	111 ± 59	66 ± 28	37 ± 13	88 ± 48
Lipid phosphate (as PO_4^{-3})	0.62 ± 0.41	0.48 ± 0.27	0.82 ± 0.39	0.5 ± 0.2	0.87 ± 0.54
Fatty acids	20.7 ± 33.1	33.2 ± 49.1	48.7 ± 58.1	9.4 ± 10.5	6.3 ± 3.2

TABLE 6.—Means and standard deviations for biochemical components of adductor muscle tissue. See Table 5 for additional information.

		Treatment		Recovery	
	Precontrol (n = 11) $\bar{x}$ ± SD	Control (n = 10) $\bar{x}$ ± SD	Parasitized (n = 10) $\bar{x}$ ± SD	Control (n = 6) $\bar{x}$ ± SD	Previously parasitized (n = 15) $\bar{x}$ ± SD
FAA	328 ± 119	306 ± 56	289 ± 47	277 ± 110	347 ± 81
FAA-	272 ± 121	227 ± 54	207 ± 35	210 ± 95	266 ± 78
FAA–	57 ± 16	57 ± 8	44 ± 5	75 ± 25	84 ± 24
Taurine	52 ± 17	53 ± 25	66 ± 25	42 ± 16	47 ± 13
Hypotaurine	8 ± 4	22 ± 7	23 ± 3	24 ± 10	33 ± 6
Aspartic acid	17 ± 7	18 ± 4	14 ± 4	18 ± 6	21 ± 8
Serine	5 ± 4	7 ± 3	5 ± 2	27 ± 21	28 ± 21
Threonine	3 ± 1	2 ± 0.9	1 ± 0.5	2 ± 1	3 ± 1
Glutamine	6 ± 2	4 ± 2	2 ± 1	6 ± 4	10 ± 8
Glutamic acid	20 ± 6	18 ± 4	21 ± 6	21 ± 4	20 ± 5
Glycine	54 ± 15	124 ± 37	119 ± 42	85 ± 54	121 ± 44
Alanine	142 ± 63	49 ± 19	39 ± 10	49 ± 30	60 ± 26
Protein	41 ± 13	49 ± 40	45 ± 56	45 ± 35	36 ± 22
Glycogen	6 ± 3	7 ± 3	6.8 ± 0.3	4 ± 2	4 ± 2
Lipid phosphate (as PO_4^{-3})	0.1 ± 0	0.2 ± 0	0.34 ± 0.3	0.25 ± 0.28	0.2 ± 0.1
Fatty acids	15.0 ± 10.0	17.5 ± 16.7	15.3 ± 24.1	6.9 ± 11.5	8.6 ± 16.9

duce host growth rate (Nishino et al. 1983). Oysters parasitized by *Boonea impressa* gained significantly less weight than nonparasitized oysters. Parasitized female oysters had significantly fewer eggs than controls (the effect on males was not quantified).

Reduced growth and impaired reproduction in the host are commonly associated with marine parasites (e.g., Menzel and Hopkins 1955; Cheng et al. 1983; Hawkes et al. 1986). Starvation produces a similar phenomenon (Fair and Sick 1982; Pipe 1985; Wright and Hetzel 1985; Devi et al. 1985). Several lines of evidence suggest that a reduction in net productivity, but not a negative energy balance, produced the results observed here. White et al. (1988) developed an energy flow model for oysters and snails. Using that model, oysters of 4 to 7 cm long, the size we used, would not incur a negative energy balance un-

TABLE 7.—Results of Duncan's multiple range test for biochemical components of the mantle and adductor muscle. Each test considered data for one biochemical component (one vertical group of 5 means) for each tissue. Different letters within a vertical group indicate significant differences at $\alpha = 0.05$ within that group.

	Protein	Glycogen	Lipid phosphate	Fatty acids		Protein	Glycogen	Lipid phosphate	Fatty acids
Mantle					*Adductor muscle*				
Precontrol	A	B	A	A	Precontrol	A	AB	A	A
Treatment					Treatment				
Control	B	A	A	A	Control	A	A	A	A
parasitized	AB	A	A	A	Parasitized	A	AB	A	A
Recovery					Recovery				
Control	AB	B	A	A	Control	A	B	A	A
Previously parasitized	B	A	A	A	Previously parasitized	A	B	A	A

TABLE 8.—Results of Duncan's multiple range test for free amino acids in the mantle tissue and adductor muscle. Each test considered data for one biochemical component (one vertical group of 5 means) for each tissue. Different letters within a vertical group indicate significant differences at $\alpha = 0.05$ within that group. Tau = taurine; Hyp = hypotaurine; Asp = aspartic acid; Ser = serine; Glu = glutamic acid; Gln = glutamine; Gly = glycine; Ala = alanine; FAA = total for the amino acids measured; FAA- = FAA less taurine and hypotaurine; FAA– = FAA less taurine, hypotaurine, glycine, and alanine.

	Tau	Hyp	Asp	Ser	Thr	Gln	Glu	Gly	Ala	FAA	FAA-	FAA–
Mantle												
Precontrol	A	B	A	A	A	A	AB	A	A	A	A	A
Treatment												
Control	A	A	B	A	B	B	AB	B	B	B	B	A
Parasitized	A	A	AB	A	AB	B	A	B	B	AB	B	A
Recovery												
Control	A	A	AB	A	B	AB	B	B	B	AB	B	A
Previously parasitized	A	A	AB	A	B	AB	B	B	B	B	B	A
Adductor muscle												
Precontrol	A	C	AB	B	A	A	A	C	A	A	A	BC
Treatment												
Control	A	B	AB	B	AB	A	A	AB	B	A	A	CD
Parasitized	A	B	B	B	B	A	A	AB	B	A	A	D
Recovery												
Control	A	AB	AB	A	AB	A	A	BC	B	A	A	AB
Previously parasitized	A	A	A	A	A	A	A	AB	B	A	A	A

TABLE 9.—Significant *P*-values obtained by MANCOVA from comparison of snail scale and *Perkinsus marinus* infection intensity. Tests using snail scale considered only the parasitized oysters after the treatment period and the previously parasitized oysters after the recovery period. Tests using *P. marinus* considered all oysters, controls and snail parasitized. Abbreviations same as Table 8. M = mantle tissue; A = adductor muscle; — = not significant in either tissue at $\alpha = 0.05$.

	Glycogen	Lipid phosphate (as PO_4^{-3})	Fatty acids	Tau	Hyp	Asp	Thr	Ser	Glu	Gly	Ala	FAA	FAA-	FAA–	Protein
Treatment															
Snail scale	—	—	—	0.02(A)	—	—	—	—	—	—	—	—	—	—	—
Perkinsus marinus	—	—	—	—	—	—	—	0.004(A)	—	—	—	—	—	—	—
Recovery															
Snail scale	—	—	—	—	0.04(A)	—	—	—	—	—	—	—	—	—	0.004(M)
Perkinsus marinus	—	0.03(M)	0.002(A)	—	—	—	—	—	—	—	—	—	—	—	—

til fed upon by at least 25 average-sized (1.75 mm maximum width) snails. Seven snails, a typical value in our experiments, would reduce net productivity by only 5 to 30%, on a daily basis. In addition, condition index and mantle glycogen levels increased during the treatment period in both control and parasitized oysters and the effect of snail parasitism on all biochemical components was small (oysters can regulate some biochemical components even during starvation, Swift and Ahmed 1983; but see Riley 1976).

Consequently, both reduced growth and impaired reproductive capacity can be attributed to a reduction in assimilated carbon available to the host, as a result of either a reduction in filtration rate resulting in less energy being assimilated (Ward and Langdon 1986) or the direct removal of assimilated carbon by the snail. Neither effect was permanent. Growth rate resumed and reproductive state returned to control levels during recovery. In both cases compensatory adjustments occurred during the recovery period so that previously parasitized oysters gained weight and increased egg number faster than the controls. Loosanoff and Nomejko (1955) and Eagle and Chapman (1953) also noted compensatory shell growth in oysters.

Perkinsus marinus infection is an important cause of mortality in oysters (Mackin and Sparks 1962; Mackin 1962; Hofstetter 1977). *Boonea impressa* can transmit *P. marinus* from one oyster to another and can also increase the intensity of infection (White et al. 1987). Continued deterioration after a stress is removed occurs frequently in "recovery" experiments (Kendall et al. 1984; Powell et al. 1984) demonstrating the necessity of examining recovery capacity in acute (vs. chronic) stresses. Growth rate typically recovers more rapidly than most biochemical parameters. In snail-parasitized oysters, both prevalence and intensity of *P. marinus* infection increased during the recovery period. Hence, in contrast to growth rate, no recovery from *P. marinus* actually occurred. By the end of the 8-wk period, infection intensity had increased by about 1 unit on Mackin's (1962) 5 point scale. Growth still occurred and reproductive capacity returned to control levels, however, during this period. These results contradict those of Menzel and Hopkins (1955) who showed that *P. marinus* retarded growth in proportion to the intensity of infection and Mackin (1953) who observed decreased fecundity in heavily infected oysters. Haven (1962) obtained results analogous to ours. Possibly, the oysters in our study were not infected heavily enough to retard growth and reproduction. Mean infection intensity in previously parasitized oysters after the recovery period was only 2.2, a light to moderate infection.

Changes observed at the biochemical level among the previously parasitized oysters after the recovery period, particularly in fatty acid and lipid phosphate content, were related to increased infection intensity of *P. marinus* (Table 9). Stein and Mackin (1955), Mackin (1962), and White et al. (in press) also noted changing lipid levels related to infection intensity. Lipid phosphate is predominantly a structural component whereas fatty acids, usually as triglycerides, are storage materials in many marine invertebrates (Gabbott 1976; Trider and Castell 1980; Gehron and White 1982). The increased lipid phosphate content in mantle tissue, however, probably indicates an increase in structural material, noted histologically by Stein and Mackin (1955, 1957) to occur in conjunction with *P. marinus* infections.

Glycogen is the primary storage material in most bivalves (Beninger and Lucas 1984). Parasitism frequently affects carbohydrate metabolism (Cheng 1963; Mohamed and Ishak 1981; Thompson and Binder 1984; Thompson 1986). White et al. (in press) suggested that *P. marinus* alters oyster metabolism favoring gluconeogenesis. Our results support this hypothesis. Glycogen levels dropped only in recovery control oysters in which *P. marinus* infection intensity also declined. Changing fatty acid content might be similarly explained. In contrast, Stein and Mackin (1957) noted decreased glycogen levels in heavily infected oysters (3 to 5 on Mackin's scale). Few of our oysters were this heavily infected, however. An alternative explanation, that slower reproductive development in parasitized oysters was responsible for variation in glycogen and fatty acid content, cannot be completely excluded because *P. marinus* infection intensity did not correlate with glycogen levels in recovery oysters. Parasitized oysters had fewer eggs than control oysters, however. Additionally, neither the number of eggs present nor the number of oysters spawning differed significantly between control and previously parasitized oysters during the recovery period.

Results of previous workers suggest that significant decreases in storage compounds, whether caused by *B. impressa* or *P. marinus*, only occur in heavily infected animals (e.g., Stein and Mackin 1957; White et al. in press). This, too, is true for the amino acid pool where significantly decreased levels are usually associated with more severely stressed animals (e.g., Powell et al. 1982, 1984). The few significant effects on amino acids in this study, like all the other biochemical components measured,

were produced by increases in concentration. Soniat and Koenig (1982) observed significant changes in the free amino acid pool due to *P. marinus*, particularly in taurine concentration. We noted changes in taurine during the treatment period and hypotaurine during recovery in the adductor muscle but these were related to snail parasitism.

Biochemical components, though rarely significantly affected, were affected not just in the mantle tissue but also in the adductor muscle. One possibility, that the snail's effect is localized at the point of feeding, is not supported by the data. Snail parasitism produces systemic effects.

CONCLUSIONS

Complementary results of White et al. (1984; 1988, in press) and this study permit a general description of the impact of snail parasitism on oysters at normal field levels. Both growth rate and reproductive development slow significantly, but recover rapidly once the snails are removed. Hence, the temporal stability of snail patches must determine the cumulative effect on field populations of oysters. The prevalence and intensity of infection by *Perkinsus marinus* is significantly increased, but recovery does not occur. That is, *Boonea impressa* probably facilitates and encourages the normal spread and intensification of *P. marinus* from which oysters, if they recover, only do so the following winter when low temperatures typically reduce infection levels (Hewatt and Andrews 1956; Burrell et al. 1984; Soniat 1985). This effect, then, is long term. Most changes in biochemical components were due to infection by *P. marinus*. Snail feeding reduces net productivity but, at normal field levels, starvation is an unlikely result. Increased infection by *P. marinus* typically raises glycogen and lipid levels, at least in light to moderate infections. Of the free amino acids, taurine and hypotaurine have been shown to be affected by *P. marinus* and *B. impressa*. Little change in the remaining FAA or the total pool has been observed singly or in concert. Feng et al. (1970) noted increased taurine levels in oysters parasitized by *Bucephalus* sp. and *Minchinia nelsoni*. Hence, an increase in taurine and hypotaurine levels apparently is a general response to parasitism in oysters.

Yuill (1987) emphasized the importance of subtle effects produced by parasites on host populations. *Perkinsus marinus* is an important source of mortality in oyster populations (Mackin and Sparks 1962; Mackin 1962; Hofstetter 1977). Data suggest that one of the most important aspects of parasitism by *B. impressa* is to encourage this second parasitic organism. To this extent, over the year, *B. impressa* at normal field densities could be responsible for a substantial amount of mortality in oyster populations.

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