Ecology, 77(5), 1996, pp. 1390–1397

ALTERED BEHAVIOR OF PARASITIZED KILLIFISH INCREASES SUSCEPTIBILITY TO PREDATION BY BIRD FINAL HOSTS[1]

KEVIN D. LAFFERTY AND A. KIMO MORRIS[2]
Marine Science Institute and Department of Biological Sciences, University of California, Santa Barbara, California 93106 USA

Abstract. Parasites that are transmitted from prey to predator are often associated with altered prey behavior. Although many concur that behavior modification is a parasite strategy that facilitates transmission by making parasitized prey easier for predators to capture, there is little evidence from field experiments. We observed that conspicuous behaviors exhibited by killifish (*Fundulus parvipinnis*) were associated with parasitism by larval trematodes. A field experiment indicated that parasitized fish were substantially more susceptible to predation by final host birds. These results support the behavior-modification hypothesis and emphasize the importance of parasites for predator–prey interactions.

Key words: behavior-modification hypothesis; bird predation; differential predation; effects of parasites on host behavior; Euhaplorchis californiensis; *field experiment;* Fundulus; *killifish; metacercariae; parasites; trematode.*

INTRODUCTION

Parasites are frequently associated with odd host behaviors such as unusual levels of activity, increased conspicuousness, disorientation, and altered responses to stimuli (Holmes and Bethel 1972). For the many life cycles where transmission depends on predation, it is often suggested that parasites alter host behavior and increase the susceptibility of intermediate hosts to predation by final hosts (e.g., Rothschild 1962, Holmes and Bethel 1972). Three main lines of evidence currently support the hypothesis that behavior modification is a parasite strategy evolved to increase transmission: hosts infected by transmissible stages of parasites often behave differently (Holmes and Bethel 1972, Dobson 1988, Curio 1988, Moore and Gotelli 1990 and Poulin 1994*a* discuss several examples); are eaten more readily by predators in the laboratory than are unparasitized hosts (Holmes and Bethel 1972, Kennedy et al. 1978, Camp and Huizinga 1979, Brassard et al. 1982, Moore 1983, Helluy 1984, Webber et al. 1987, Poulin et al. 1992); and are taken more frequently by predators than expected in the wild (VanDobben 1952, Feare 1971, Rau and Caron 1979, Moore 1983, Hoogenboom and Dijkstra 1987). As a whole, this evidence is quite convincing and, because the ingestion of larval parasites during predation is a frequent occurrence, helps us to better understand foraging dynamics and food webs.

Several studies have used a combination of approaches, helping to expand the base of evidence used to support the behavior modification hypothesis. For example, Moore (1983) found that terrestrial isopods infected with a larval acanthocephalan were more active and spent more time in dry areas, on contrasting backgrounds, and away from shelter than did unparasitized isopods. In aviary predation trials, 59% of isopods eaten by Starlings were parasitized, compared with an initial 47% prevalence of infection among the isopods available in the cage (Margolis et al. [1982] define "prevalence" as the proportion of hosts in a sample that are parasitized). There was indirect evidence that transmission was not random in nature because the prevalence of adult acanthocephalans in wild Starling nestlings (13%) was higher than expected, given the rates at which parents fed isopods to their young multiplied over the age of nestlings and the very low prevalence of parasitized isopods nearby (0.2%).

Although the link between conspicuous behaviors induced by parasites and increased parasite transmission is logical and well supported, Moore and Gotelli (1990) discuss alternative explanations. Pathology can affect host behavior in ways that do not necessarily increase transmission. For example, hosts may alter their behaviors to help rid themselves of parasites (Hart 1990) or compensate for metabolic drains of parasitism (Milinski 1985). Thus, it is important to also assess how predation risk varies with parasitism. From studies of predator gut contents, it might appear that parasites make prey more susceptible to predation if predators prefer larger, older prey that have had a longer time to accumulate parasites. Also, if the dispersal of hosts and parasites is limited, areas where predators abound will have higher rates of parasite transmission to nearby prey, leading to more parasitized prey in the predator's diet compared with the prevalence of parasitism seen in the prey population on a broader spatial scale. Another potential limitation of gut-content studies is the difficulty of accurately determining the prevalence of the parasite in the prey population. As an example, the relatively high proportion of *Sarcocystis*-infected voles

[1] Manuscript received 8 May 1995; revised 23 August 1995; accepted 19 October 1995.

[2] Present address: Department of Entomology, Oregon State University, Corvallis, Oregon 97331-2907 USA.

in the diet of Kestrels could reflect either increased predation or decreased trapping success for parasitized voles (Hoogenboom and Dijkstra 1987).

In combination with evaluations of host behavior, predation experiments can best test the link between behavior and increased transmission (Bethel and Holmes 1977). Unfortunately, results from laboratory predation experiments may only allow limited inference about events in nature. Although field experiments with natural final hosts can effectively determine whether behavior modification increases parasite transmission in the wild, few have been conducted. A notable exception is the work by Aeby (1991, 1992). Aeby found coral polyps (*Porites* spp.) become distended following infection with metacercariae (*Plagioporous* sp.), causing colonies to suffer reduced growth. The metacercariae appear as bright pink nodules and hinder the ability of parasitized polyps to retract into the calyx. Using manipulative field and laboratory experiments, Aeby demonstrated that butterfly fish, the appropriate definitive host, forage more frequently on parasitized coral polyps. Ironically, due to the regenerative capabilities of the colony, parasitized corals did better in treatments that allowed butterfly fish to feed on them, suggesting that the parasite-induced alteration of the parasitized polyp is beneficial to the coral, the trematode, and, perhaps, the fish.

To test whether a parasite can alter the behavior of its intermediate host and increase transmission to its final host, we studied *Euhaplorchis californiensis,* the most common trematode in many southern California salt marshes (Martin 1955, Kuris 1990, Lafferty 1993, Lafferty et al. 1994). *E. californiensis,* like most digenetic trematodes, has a three-host life cycle. Worms mature sexually in a number of bird species (Martin [1971] notes experimental infections in chicks, gulls, cats, monkeys, etc.), mate, and lay eggs that pass with the birds' feces. The horn snail, *Cerithidea californica,* then ingests these eggs while foraging on mudflats. The trematode castrates the snail and produces cercariae that leave to infect the second intermediate host, the killifish *Fundulus parvipinnis* (Plate 1), where they encyst in the brain case in high intensities (Margolis et al. [1982] define "intensity" as the number of parasites per parasitized host) (Martin 1950, Yoshino 1971). Predation on a parasitized fish by a bird completes the life cycle. Although parasitizing the fish's brain could simply reflect an attempt to escape the host immune system (Szidat 1969), this site of infection should allow a parasite to manipulate its host with little effort (Poulin 1994*b*). Relatively few studies have looked at the effect of trematode metacercariae on host behavior (Poulin 1994*a*).

We compared parasitized and unparasitized fish with respect to their behaviors in the laboratory and susceptibility to predation in the field. We suspected that the trematode could best increase its rate of transmission to visually oriented piscivorous birds by making killifish more conspicuous. Consistent with this prediction, parasitized fish frequently swam to the surface and were eaten strikingly more than unparasitized fish.

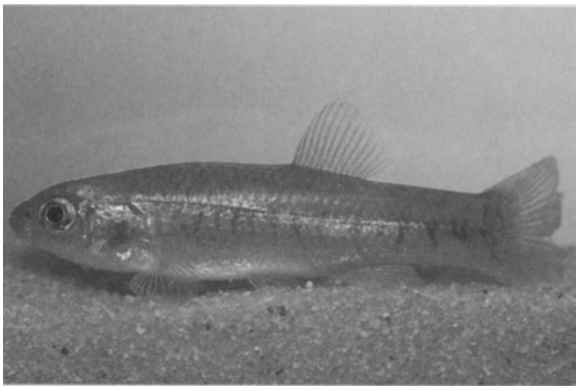

PLATE 1. The Pacific killifish, *Fundulus parvipinnis,* which acts as second intermediate host for the trematode *Euhaplorchis californiensis.* Photo: T. C. Huspeni.

METHODS

Altered behavior

To assess whether the trematode altered host behavior, we observed killifish in the laboratory (summer 1993). Although our attempts at experimentally infecting fish were unsuccessful, we were able to compare killifish from parasitized and unparasitized populations because they occur in habitats with and without the first intermediate host snail. We trapped 12 killifish from Devereux Slough (Coal Oil Point Natural Reserve [near Santa Barbara, California, USA]), an area without the snail or parasite. We also collected 30 parasitized fish from Carpinteria Salt Marsh Reserve. Here, the snail is abundant and trematodes infect all except the smallest killifish (Martin 1950, Yoshino 1971). These sites are ≈30 km apart (both are in Santa Barbara County, California, USA), and are similar hydrogeomorphic habitats. We combined the fish from both populations into a 150-L glass aquarium (lined on three sides with black plastic) with flow-through sea water and left them to acclimate for several days.

By observing fish over a few days, we identified and defined several discrete behaviors that made fish more conspicuous to us. Admittedly, our evaluation of behaviors may have been different from the perception of a foraging bird. Because this imperfection seemed more likely to obscure than enhance the pattern predicted by the behavior-modification hypothesis, we considered that our estimation of conspicuousness to birds was conservative (i.e., we were more likely to commit Type II than Type I error). The behaviors that we characterized as conspicuous were surfacing, flashing, contorting, shimmying, and jerking. We did not assess escape behavior in the lab. Surfacing fish made abrupt dashes up to the tank's surface, flashing fish

turned laterally so that one side of the body faced upward (often associated with chafing on the tank's bottom), contorting fish performed a slow, acute, dorsal–ventral bending, usually bending the head and tail in opposite directions, shimmying fish vibrated for a few seconds, and jerking fish moved suddenly forward 3–5 cm. We noticed many of these behaviors in the field as well.

Having unparasitized and parasitized fish together in the same observational aquarium allowed for a blind assessment of behavior (we did not know if, or to what extent, a fish was parasitized when we scored its behavior). After the fish had acclimated, we recorded conspicuous behaviors (during daylight hours) over a 30-min period for each fish. Once we scored a fish's behavior, we captured (with a hand net), euthanized, and dissected it to determine the intensity of larval trematodes (metacercarial cysts) in the brain case. Because we had previously observed that fish behaved differently at low densities, we halted our observations before the number of fish remaining in the aquarium was too low for "natural" schooling behavior. In total, we observed and dissected 18 parasitized and 6 unparasitized fish over a period of 2 wk. In the process of dissecting the fish, we found a second species of trematode, *Renicola buchanani,* in the liver of all the fish parasitized with *Euhaplorchis californiensis.* Because it also completes its life cycle in piscivorous birds (where it lives in the bird's kidneys, Martin 1971) and would also benefit from increased predation, we included it in our analyses. We quantified the intensity of this species by squashing the fish's liver under a glass slide and counting the large metacercariae. In our investigation of the association between conspicuous behaviors and parasitism, we analyzed each behavior separately and pooled (conspicuousness could be a composite of several behaviors), and considered the effect of each parasite species individually and combined (parasites could have an additive effect on behavior).

Differential predation

To test for differential predation on parasitized fish, on 8 November 1994 we stocked a mixture of parasitized and unparasitized fish into two fish pens placed in the University of California, Santa Barbara, Campus Lagoon (Santa Barbara County). We collected parasitized fish from Carpinteria Salt Marsh Reserve and unparasitized fish from the Ventura River Estuary (Ventura County, California, USA). To keep track of the two different populations throughout the experiment, we clipped the distal 25% of the left pectoral fin (parasitized fish from Carpinteria) or right pectoral fin (unparasitized fish from Ventura). We apportioned the fish by length into two groups, each with 95 parasitized fish and 53 unparasitized fish (total length ranged from 5 cm to 7 cm, with an average of 6 cm for each group). We then combined each unparasitized group with a parasitized group, resulting in two identical mixed groups. We made a substantial effort to build pens large enough to allow the fish to behave normally, constructing each fish pen out of a 15 m long, 3-mm mesh seine. To reduce escape, we folded the leaded end of each net and sewed it together to form a purse. We positioned the top edge of the net into a 20-m^2 U-shape with the open end made flush against the shore. The resulting density (7.4 fish/m^2) was well within the natural variation exhibited by local killifish populations. We propped the top edges of the seines 25 cm above the water's surface with stakes driven into the soft sediment. The depth of the pens sloped from the shore to 1.3 m in depth (there was no halocline between this depth and the surface). We left the surface of the experimental pen accessible to piscivorous birds in hopes that they would forage in it. To estimate escape and mortality not associated with predation, we covered the surface of a second, otherwise-identical pen, with bird netting (made of black plastic 5-cm mesh that was wide enough for killifish to easily jump through, but prevented birds from effectively foraging) and installed it next to the experimental pen. To eliminate the possibility that differences in cage placement or construction might affect the results, we switched the fish populations and the bird netting between cages midway through the experiment (so that fish remained in the same treatment as before).

During the experiment, we observed Great Egrets (*Casmerodius albus*), Great Blue Herons (*Ardea herodias*), and Snowy Egrets (*Egretta thula*) foraging in the pens. Other potential predatory birds common at the lagoon during the experiment included: Greater Yellowlegs (*Tringa melanoleuca*), Lesser Scaup (*Aythya affinis*), Red-breasted Merganser (*Mergus serrator*), Bonaparte's Gull (*Larus philadelphia*), Ring-billed Gull (*Larus delawarensis*), Western Gull (*Larus occidentalis*), Forster's Tern (*Sterna forsteri*), Eared Grebe (*Podiceps nigricollis*), Pied-billed Grebe (*Podilymbus podiceps*), Horned Grebe (*Podiceps auritius*), Brown Pelican (*Pelecanus occidentalis*), Double crested Cormorant (*Phalocrocorax auritus*), Belted Kingfisher (*Ceryle alcyon*), Black-crowned Night Heron (*Nycticorax nycticorax*), and Green-backed Heron (*Butorides striatus*). In these small estuaries, there are typically no predatory fish large enough to eat killifish of the size we used in the experiment.

We recaptured fish from both pens after 20 d and brought them back to the laboratory. Here, we euthanized and dissected a subsample of the parasitized fish ($N = 99$) from which we removed and counted all *E. californiensis* cysts from the brain case and *R. buchanani* from the liver. We used this subsample to compare parasite intensities between the two treatments.

Because the relative abundance of parasitized and unparasitized fish changed over 20 d (the birds were, in effect, sampling without replacement), we could not directly quantify the relative susceptibility of parasitized and unparasitized fish. To derive a value for the

TABLE 1. Means (and range) of conspicuous behaviors observed over a 30-min period for parasitized and unparasitized killifish.

	N†	Behavior					
		Flashing	Surfacing	Contorting	Shimmying	Jerking	Sum‡
Parasitized	18	3.8 (0–14)	11.2 (2–22)	0.9 (0–4)	2.2 (0–7)	2.9 (0–11)	21.0 (11–29)
Unparasitized	16	0.8 (0–3)	4.5 (2–6)	0 (0)	0 (0)	0 (0)	5.3 (2–9)
Mann–Whitney *U*		85	96	78	93	90	108
P (two-tailed)		0.033	0.005	0.056	0.006	0.010	<0.001

† Number of fish observed.
‡ Total number of odd behaviors observed.

difference in susceptibility to predation between parasitized and unparasitized fish, we created a simple computer simulation that iterated sequential predation events for the estimated number of fish eaten. For each simulation, we used a different value for the parameter representing differential susceptibility. We solved for the value that produced a simulated number of parasitized and unparasitized fish eaten identical to that observed in the predation experiment.

RESULTS

Altered behavior

Consistent with our prediction, parasitized fish exhibited conspicuous behaviors more frequently than did the unparasitized fish (parasitized fish had a mean of 21 conspicuous behaviors per 30 min, while unparasitized fish had a mean of 5.3 conspicuous behaviors per 30 min, $P < 0.001$, Table 1). Only parasitized fish contorted, shimmied, or jerked. We found all behaviors to be more frequent in parasitized fish than unparasitized fish with the conditional exception that the difference in contortions was not statistically significant based on a two-tailed hypothesis ($P = 0.056$, Table 1).

A multiple regression of the intensity of both parasite species and the sum of conspicuous behaviors indicated that parasites explained a substantial proportion of the variation in the behavior of parasitized fish (multiple $R^2 = 0.795$). This analysis detected a significant effect of *Euhaplorchis californiensis* (Fig. 1, $P = 0.002$) but not of *Renicola buchanani* ($P = 0.137$, Table 2).

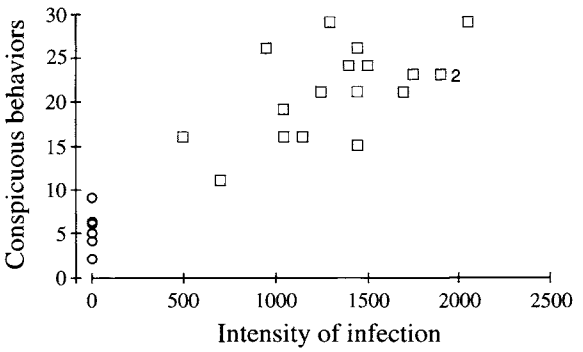

FIG. 1. The frequency of conspicuous behaviors each fish displayed in a 30-min observational period in relation to the intensity of parasitism of *Euhaplorchis californiensis* (number of metacercarial cysts per fish brain). In the parasitized population (squares), the number of conspicuous behaviors increased with parasite intensity. All unparasitized fish (circles) had a smaller number of conspicuous behaviors than parasitized fish. The square directly left of the "2" represents two fish.

There was a positive relationship between conspicuous behavior and parasite intensity so that heavily parasitized fish were more conspicuous than lightly parasitized fish (Table 3). The strongest association seen was between the sum of behaviors and the weighted sum of the intensities of the two parasite species (see Table 3 for a description of how we calculated the weighted sum). Surfacing was the behavior most strongly associated with *E. californiensis*, yet surfacing was not associated with *R. buchanani* intensity. In comparison, jerking and shimmying were the behaviors most associated with *R. buchanani*. The two parasite species were positively correlated with one another ($R = 0.553$, df = 15, $P < 0.05$).

Differential predation

In support of the hypothesis that behavior modification results in increased parasite transmission, predation rates on parasitized fish were substantially higher than predation rates on unparasitized fish. This was not confounded with size-selective foraging by birds (fish from all treatments started and ended with a mean total length of 6 cm). In the exclosure pen, unparasitized fish declined in number from 53 to 50, while parasitized fish declined from 95 to 91—a negligible rate of escape and non-predation mortality. In the experimental (open) pen, the number of unparasitized fish declined from 53 to 49 while parasitized fish declined

TABLE 2. Multiple-regression statistics for an examination of the intensity of *Euhaplorchis californiensis* and *Renicola buchanani* on the number of conspicuous behaviors of killifish. $N = 24$ killifish.

	Coefficient		Std. Coef.	*t*	*P*†
	Mean	1 SE			
Constant	6.386	1.429	0.000	4.468	<0.001
Euhaplorchis	0.007	0.002	0.637	3.446	0.002
Renicola	0.028	0.018	0.286	1.547	0.137

Notes: $R = 0.892$, $R^2 = 0.795$, adjusted $R^2 = 0.775$; standard error estimate = 3.891.
† *P* values reflect a two-tailed test.

TABLE 3. Coefficients of Pearson correlations between parasite intensity and the number of conspicuous behaviors of fish. The weighted sum† of parasite intensities was derived to give equal weight to each species of parasite because the smaller *Euhapchloris californiensis* occurs at intensities 7 times higher than does *Renicola buchanani*. For 16 df, the critical value of R at $P = 0.05$ is 0.468.

	Behavior					
	Flashing	Surfacing	Contorting	Shimmying	Jerking	Sum‡
Euhaplorchis	−0.020	0.428	−0.232	0.134	0.208	0.576
Renicola	0.180	0.004	0.086	0.331	0.342	0.516
Weighted sum†	0.108	0.208	−0.055	0.281	0.323	0.613

† For a single fish, the weighted sum was equal to $R/(\Sigma\ R/N) + E/(\Sigma\ E/N)$, where E = no. of *E. californiensis*, N = no. of fish, and, for example, R equals the number of *R. buchanani* cysts in a fish, and $\Sigma\ R/N$ equals the mean number of *R. buchanani* cysts in the sample of fish.

‡ The total no. of odd behaviors.

from 95 to 44 (Fig. 2). Subtracting the incidental loss observed in the control from the total loss in the experimental pen yielded an estimate that birds ate a much higher proportion of parasitized fish (47/91) than unparasitized fish (1/50) (Fig. 3, $\chi^2 = 35.4$, 1 df, $P < 0.001$). Our computer simulation found that parasitized fish were, on average, 31 times more susceptible to predation than unparasitized fish (we estimated a more conservative 10-fold increase in susceptibility of parasitized fish if we assumed no background mortality).

There was an effect of parasite intensity on predation. The mean intensity of cysts of each parasite species was lower in fish from the experimental pen than in fish from the bird-exclosure pen (Fig. 2, Table 4). Heavily parasitized fish (those with more parasites than the median intensity) were more susceptible to predation than lightly parasitized fish; these results were the same whether we assessed the intensity of each parasite species separately or pooled (Fig. 3).

DISCUSSION

Our study supports the hypothesis that parasites modify the behavior of their intermediate hosts and make them more susceptible to predation. A recent meta-analysis found that the degree of alteration of host behavior by parasites was moderate (Poulin 1994*a*). In contrast, our results suggest a 30-fold effect of parasitism on predation that stems from a four-fold effect of parasitism on behavior. For this reason, we would have underestimated the effects of parasites on predation risk if we had simply quantified behavior. The magnitude of the effect surprised us. For example, a previous mathematical model of behavior modification set the upper limit of differential predation as only 10-fold (Lafferty 1992). The lesson here is that a parasite can parlay a small behavioral modification into a large increase in predation. An association between parasite intensity and behavior modification is expected in systems like ours where intensities are high because one parasite is unlikely to need to modify the host's behavior by itself; it can rely on the actions of the group (Poulin 1994*b*).

It is difficult to separate the effects of the two parasite species without making use of experimental infections. Their effects on behavior could be additive or complementary. Alternatively, one of the parasites might be disproportionately responsible for modifying the behavior of the killifish. If so, our observations are most consistent with the hypothesis that *Renicola buchanani* is the less potent modifier and it may benefit from its

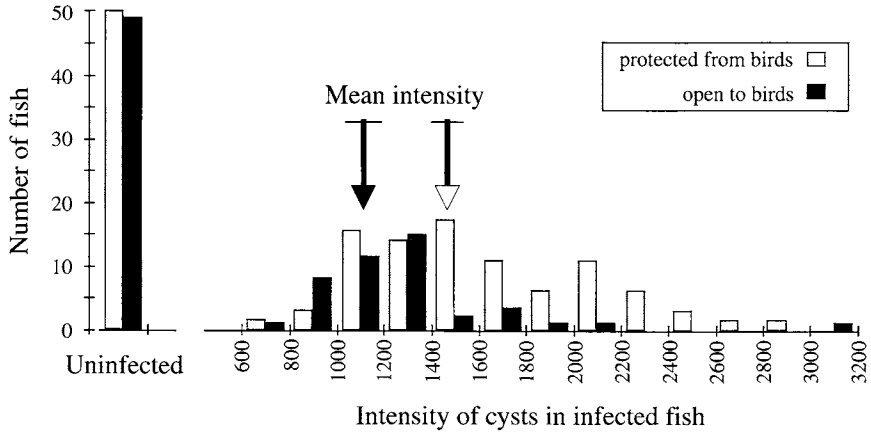

FIG. 2. The effect of bird predation on parasitized fish. The histogram shows the estimated frequency of infection intensities of the control (protected) and treatment (open) pens at the end of 20 d, indicating that parasitized fish were more likely to be eaten than unparasitized fish and that highly parasitized fish were more likely to be eaten than lightly parasitized fish.

TABLE 4. Comparison of intensities of each parasite species between parasitized killifish from the netted and open pens.

		Intensity of parasitism			
		E. californiensis		*R. buchanani*	
	N†	Mean	Range	Mean	Range
Netted pen	60	1454	550–2650	208.5	20–510
Open pen	39	1102	550–3000	163.6	32–400
Mann–Whitney *U*		1759		1449	
P‡		>0.001		0.046	

† *N* = no. of parasitized fish.
‡ *P* values reflect a two-tailed test.

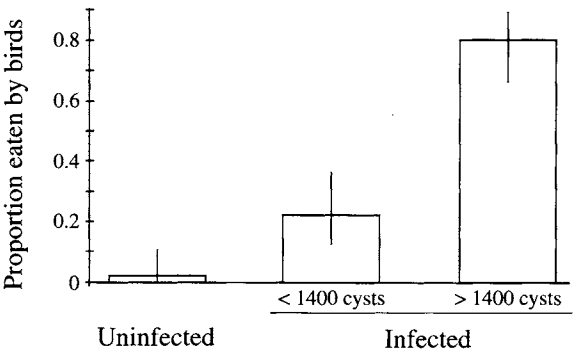

FIG. 3. A comparison of the proportion of fish estimated to have been eaten by birds after 20 d (total number missing in the open pen minus the number that escaped or died in the netted pen) showing that heavily parasitized fish were preyed on more frequently than lightly parasitized fish, which were preyed on more frequently than unparasitized fish. From left to right, percentages = 1/50, 10/45, and 37/46, respectively. Error bars are 95% confidence intervals indicating that all three groups were significantly different.

association with *Euhaplorchis californiensis*. We plan experiments to address these issues.

We have considered other explanations for our results but, as we argue below, they do not appear to be better alternatives to the behavior-modification hypothesis. First, it is possible that, independent of parasitism, fish from Carpinteria behave more oddly and are more susceptible to predation than fish from Devereux and Ventura. This explanation, however, does not explain the associations of parasite intensity with behavior and predation seen with the fish from Carpinteria only. Second, it could be that odd behavior in hosts leads to increased susceptibility to infection by parasites (Moore and Gotelli 1990), not the reverse as we have assumed. If this was the case, all else being equal, each population (parasitized and unparasitized) should have had individuals with highly conspicuous behaviors—but only fish from the parasitized population did. Third, increased time at the surface might be a host adaptation if the host is attempting to produce a behavioral fever to kill the parasite (Horton and Moore 1993). In our study, surfacing was not the only behavior associated with parasitism. Also, although surface temperatures were slightly warmer in our field experiment (16°C at the surface vs. 14°C at the bottom), surface waters were not warmer in the flow-through aquaria where we conducted our laboratory observations on behavior. Fourth, parasitized fish might be going to the surface to feed more, to help meet increased metabolic demands caused by parasitism (Milinski [1985] observed this effect with larval tapeworms that place a high metabolic demand on their hosts). Again, surfacing was not the only behavior associated with parasitism. Also, the metacercariae in our study probably do not extract much, if any, energy from the killifish and we fed fish ad libitum in the laboratory and mixed their food into the water column. Still, in spite of our arguments against these four alternatives, it is always possible that there are other factors associated with parasitism and behavior that we have not considered.

Other studies have shown that larval trematodes affect the feeding behavior and time at the surface (Crowden and Broom 1980), schooling behavior (Radabaugh 1980), swimming performance (Coleman 1993), predator avoidance (Poulin 1993), vulnerability to non-host predators (Brassard et al. 1982), vision (Dubois 1970, Owen et al. 1993), survival over winter (Lemly and Esch 1984), and mate choice (Rosenqvist and Johansson 1995) of their fish hosts. Some of these effects likely also act to increase transmission to final hosts.

We know little about the mechanisms parasites use to alter host behavior, but some evidence exists for sophisticated manipulation of hormones and neurochemicals (Kavaliers and Podesta 1988, Helluy and Holmes 1990). For larval tapeworms, increased oxygen demand partially explains why parasitized fish frequently surface (Smith and Kramer 1987). In our study, the physical presence of hundreds of metacercarial cysts in the brain case might be sufficient to alter killifish behavior. Alternatively, the odd behaviors we observed are consistent with an interference with glutamate or dopamine (V. L. Trudeau, *personal communication*).

The behavior-modification hypothesis, in conjunction with the ubiquity of trophically transmitted parasites, greatly alters how we understand both host–parasite and predator–prey interactions. Clearly, larval parasites are not simply inert cysts waiting for transmission (see reviews by Holmes and Bethel 1972, Curio 1988, Dobson 1988, Moore and Gotelli 1990, and Poulin 1994*a*). Instead, parasites may, without extracting any significant host energy, greatly increase predation rates on intermediate hosts (e.g., Cram 1931, VanDobben 1952, Rothschild, 1962, Szidat 1969, Holmes and Bethel 1972). Intermediate hosts suffer the brunt of this parasite strategy. If parasitism is common, as it is for killifish, behavior modification might substantially reduce intermediate host densities. The implications for predators are also interesting. Because adult *E. californiensis* worms are small (Martin 1950) and probably short lived, it is unlikely that they impart a significant energy cost upon the bird host. *Renicola*

buchanani might be more pathogenic but is less abundant. If the cost of parasitism is less than the energy gained from capturing more fish, parasites might benefit birds by acting as a delivery service that enables birds to eat fish that are otherwise difficult to capture (Lafferty 1992). In some cases, parasites might allow the persistence of a predator in areas where one could not previously exist (Freedman et al. 1987). If so, trophically transmitted parasites could be more important for food web dynamics than has been acknowledged by most ecologists.

ACKNOWLEDGMENTS

Thanks to A. Brooks, A. Kuris, M. Martinez, J. Moore, A. Mruthyunjaya, G. Rosenqvist, C. Sandoval, and M. Torchin for assistance and intellectual input. Access to sites was provided, in part, by the UCSB Natural Reserve System. Funding was provided to A. K. Morris from the University of California Natural Reserve System's Robert M. Norris Fellowship and the Museum of Systematics and Ecology at the Department of Biological Sciences, UCSB.

LITERATURE CITED

Aeby, G. S. 1991. Behavioral and ecological relationships of a parasite and its hosts within a coral reef system. Pacific Science **45**:263–269.

———. 1992. The potential effect the ability of a coral intermediate host to regenerate has had on the evolution of its association with a marine parasite. Proceedings of the 7th International Coral Reef Symposium, Guam **2**:809–815.

Bethel, W. M., and J. C. Holmes. 1977. Increased vulnerability of amphipods to predation owing to altered behaviour induced by larval acanthocephalans. Canadian Journal of Zoology **55**:110–116.

Brassard, P., M. E. Rau, and M. A. Curtis. 1982. Parasite-induced susceptibility to predation in diplostomiasis. Parasitology **85**:495–501.

Camp, J. W., and H. W. Huizinga. 1979. Altered color, behavior and predation susceptibility of the isopod *Asellus intermedius* infected with *Acanthocephalus dirus*. Journal of Parasitology **65**:667–669.

Coleman, F. C. 1993. Morphological and physiological consequences of parasites encysted in the bulbus-arteriosus of an estuarine fish, the sheepshead minnow, *Cyprinodon variegatus*. Journal of Parasitology **79**:247–254.

Cram, E. B. 1931. Developmental stages of some nematodes of the Spiruroidea parasitic in poultry and game birds. United States Department of Agriculture Technical Bulletin **227**.

Crowden, A. E., and D. M. Broom. 1980. Effects of the eyefluke, *Diplostomum spathaceum*, on the behaviour of dace (*Leuciscus leuciscus*). Animal Behaviour **28**:287–294.

Curio, E. 1988. Behavior and parasitism. Pages 149–160 *in* H. Mehlhorn, editor. Parasitology in focus; facts and trends. Springer Verlag, Berlin, Germany.

Dobson, A. P. 1988. The population biology of parasite-induced changes in host behavior. Quarterly Review of Biology **63**:139–165.

Dubois, G. 1970. Synopsis de Strigeidae et des Diplostomatidae (Trematoda). Volume 2. Mémoires de la Société Neuchâteloise Des Sciences Naturelles **10**:257–727.

Feare, C. J. 1971. Predation of limpets and dogwhelks by oystercatchers. Bird Study **18**:121–129.

Freedman, H. I., J. F. Addicott, and B. Rai. 1987. Obligate mutualism with a predator: persistence of three-species models. Theoretical Population Biology **32**:157–175.

Hart, B. L. 1990. Behavioral adaptations to pathogens and parasites—5 strategies. Neuroscience and Biobehavioral Reviews **14**:273–294.

Helluy, S. 1984. Relations hôtes-parasite du trématode *Microphallus papillorobustus* (Rankin, 1940). III. Facteurs impliqués dans les modifications du comportement des *Gammarus* hôtes intermédiares et tests de prédation. Annales de Parasitologie Humaine et Comparée **59**:41–56.

Helluy, S. A., and J. C. Holmes. 1990. Serotonin, octopamine, and the clinging behavior induced by the parasite *Polymorphus paradoxus* (Acanthocephala) in *Gammarus lacrustis* (Crustacea). Canadian Journal of Zoology **68**: 1214–1220.

Holmes, J. C., and W. M. Bethel. 1972. Modification of intermediate host behaviour by parasites. Zoological Journal of the Linnean Society **51**, Supplement **1**:123–149.

Hoogenboom, I., and C. Dijkstra. 1987. *Sarcocystis cernae*: a parasite increasing the risk of predation of its intermediate host, *Microtus arvalis*. Oecologia **74**:86–92.

Horton, D. R., and J. Moore. 1993. Behavioral effects of parasites and pathogens in insect hosts. Pages 107–124 *in* N. E. Beckage, S. N. Thompson, and B. A. Federici, editors. Parasites and pathogens of insects. Volume 1. Academic Press, New York, New York, USA.

Kavaliers, M., and R. Podesta. 1988. Opioid involvement in parasite-induced behavioural modifications: evidence from hamsters infected with *Schistosoma mansoni*. Canadian Journal of Zoology **66**:2653–2657.

Kennedy, C. R., P. F. Broughton, and P. M. Hine. 1978. The status of brown and rainbow trout, *Salmo trutta* and *S. gairdneri*, as hosts of the acanthocephalan, *Pomphorynchus laevis*. Journal of Fish Biology **13**:265–275.

Kuris, A. M. 1990. Guild structure of larval trematodes in molluscan hosts: prevalence, dominance and significance of competition. Pages 69–100 *in* G. W. Esch, A. O. Bush, and J. M. Aho, editors. Parasite communities: patterns and processes. Chapman & Hall, London, England.

Lafferty, K. D. 1992. Foraging on prey that are modified by parasites. American Naturalist **140**:854–867.

———. 1993. Effects of parasitic castration on growth, reproduction and population dynamics of *Cerithidea californica*. Marine Ecology Progress Series **96**:229–237.

Lafferty, K. D., D. T. Sammond, and A. M. Kuris. 1994. Analysis of larval trematode community structure: separating the roles of competition and spatial heterogeneity. Ecology **75**:2275–2285.

Lemly, A. D., and G. W. Esch. 1984. Effects of the trematode *Uvulifer ambloplitis* on juvenile bluegill sunfish, *Lepomis macrochirus*: ecological implications. Journal of Parasitology **70**:475–492.

Margolis, L., G. W. Esch, J. C. Holmes, A. M. Kuris, and G. M. Schad. 1982. The use of ecological terms in parasitology (report of an ad hoc committee of the American Society of Parasitologists). Journal of Parasitology **68**:131–133.

Martin, W. E. 1950. *Euhaplorchis californiensis* n.g., n.sp., Heterophyidae, Trematoda, with notes on its life cycle. Transactions of the American Microscopical Society **69**: 194–209.

———. 1955. Seasonal infections of the snail, *Cerithidea californica* Haldeman, with larval trematodes. Pages 203–210 *in* Essays in natural science in honor of Captain Alan Hancock on the occasion of his birthday, July 26 1955. University of Southern California Press, Los Angeles, California, USA.

———. 1971. Larval stages of renicolid trematodes. Transactions of the American Microscopical Society **90**:188–194.

Milinski, M. 1985. Risk of predation of parasitized sticklebacks (*Gasterosteus aculeatus* L.) under competition for food. Behaviour **93**:203–216.

Moore, J. 1983. Responses of an avian predator and its isopod prey to an acanthocephalan parasite. Ecology **64**:1000–1015.

Moore, J., and N. J. Gotelli. 1990. A phylogenetic perspective on the evolution of altered host behaviours: a critical look at the manipulation hypothesis. Pages 193–233 *in* C. J. Barnard and J. M. Behnke, editors. Parasitism and host behaviour. Taylor and Francis, London, England.

Owen, S. F., I. Barber, and P. J. B. Hart. 1993. Low level infection by eye fluke, *Diplostomum spp,* affects the vision of 3-spined sticklebacks, *Gasterosteus aculeatus.* Journal of Fish Biology **42**:803–806.

Poulin, R. 1993. Age-dependent effects of parasites on antipredator responses in two New Zealand freshwater fish. Oecologia **96**:431–438.

———. 1994*a*. Meta-analysis of parasite-induced behavioural changes. Animal Behaviour **48**:137–146.

———. 1994*b*. The evolution of parasite manipulation of host behaviour: a theoretical analysis. Parasitology **109S**: S109–S118.

Poulin, R., M. A. Curtis, and M. E. Rau. 1992. Effects of *Eubothrium salvelini* (Cestoda) on the behavior of *Cyclops vernalis* (Copepoda) and its susceptibility to fish predators. Parasitology **105**:265–271.

Radabaugh, D. C. 1980. Changes in minnow, *Pimephales promelas* Rafinesque, schooling behavior associated with infections of brain-encysted larvae of the fluke, *Ornithodiplostomum ptychocheilus.* Journal of Fish Biology **16**: 621–628.

Rau, M. E., and F. R. Caron. 1979. Parasite-induced susceptibility of moose to hunting. Canadian Journal of Zoology **57**:2466–2468.

Rosenqvist, G., and K. Johansson. 1995. Male avoidance of parasitized females explained by direct benefits in a pipefish *Sygnathus typhle.* Animal Behaviour **49**:1039–1045.

Rothschild, M. 1962. Changes in the behaviour of intermediate hosts of trematodes. Nature **193**:1312–1313.

Smith, R. S., and D. L. Kramer. 1987. Effects of a cestode (*Schistocephalus sp.*) on the response of ninespine sticklebacks (*Pungitius pungitius*) to aquatic hypoxia. Canadian Journal of Zoology **65**:1862–1865.

Szidat, L. 1969. Structure, development and behaviour of new Strigeatoid metacercariae from subtropical fishes of South America. Journal of the Fisheries Research Board of Canada **26**:753–786.

VanDobben, W. H. 1952. The food of the cormorant in the Netherlands. Ardea **40**:1–63.

Webber, R. A., M. E. Rau, and D. J. Lewis. 1987. The effects of *Plagiorchis noblei* (Trematoda: Plagiorchiidae) metacercariae on the susceptibility of *Aedes aegypti* larvae to predation by guppies (*Poecilia reticulata*) and meadow voles (*Microtus pennsylvanicus*). Canadian Journal of Zoology **65**:2346–2348.

Yoshino, T. P. 1971. Helminth parasitism in the Pacific killifish, *Fundulus parvipinnis,* from Goleta Slough, California. Dissertation. University of California, Santa Barbara, California, USA.