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Parasite-associated phenotype modifications in threespine stickleback

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The threespine stickleback fish, *Gasterosteus aculeatus*, is parasitized by the tapeworm *Schistocephalus solidus* in freshwater habitats. The competency of the parasite to infect a definitive host, one of many piscivorous birds, is associated with behavioral changes in parasitized stickleback that are likely to increase the chance of transmission to a definitive host. Over a limited geographical range, large tapeworm size is also associated with demelanization, a phenotypic change in the stickleback host that involves a dramatic loss of melanin in the skin and, simultaneously, a darkening of the eye. We demonstrate that stickleback harboring a worm large enough to be infective to a definitive host exhibit behavioral shifts likely to enhance transmission, but that these changes are substantially magnified in demelanized individuals, all of which were infected by large tapeworms. The results were similar whether we used a model of a bird flown over an aquarium, or a preserved trout moved to simulate attack under field conditions. Because changes in the levels of response to both kinds of predators were similar, we infer that behavioral modifications that enhance susceptibility to visually hunting predators that are definitive hosts also enhance susceptibility to visually hunting predators (e.g. trout) that are not. In lakes where predatory fishes are common, the impact on *S. solidus* transmission could be substantial. Although other studies have suggested that positive buoyancy, forcing infected fish to remain near the surface, is one cause of these behavioral shifts, we detected no differences in water column position among unparasitized and parasitized classes of stickleback. Instead, infected stickleback appeared to move sluggishly and were less likely to respond to simulated attacks than were uninfected fish, and the behavioral shift was most dramatic in demelanized stickleback.

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The nature of parasitic relationships virtually insures that the host will be negatively affected in some way. Among the most dramatic effects of parasites are changes in the morphology, coloration, and behavior of their hosts (Oetinger and Nickol 1981, Moore 1983, 1995, Hurd 1990). These effects often are especially pronounced when the parasite requires transmission among hosts through the food chain in order to complete its life cycle (Webber et al. 1987). In these instances, transitions in host phenotype are typically associated with the attainment of infectivity to the next host in the life cycle of the parasite, and the shifts in host phenotype can be interpreted as enhancing the effectiveness of transmission (Bethel and Holmes 1974, Poulin et al. 1992, Tierney et al. 1993, Robb and Reid 1996, Bakker et al. 1997; but see Poulin 1995).

When transitions in phenotype appear to enhance transmission to successive hosts in the life cycle, host manipulation by the parasite is often inferred, although other interpretations may also be plausible (Holmes and Bethel 1972, Moore and Gotelli 1990). For example, decreases in stamina or ability to perform evasive maneuvers could result from stress or reduced energy reserves, and therefore be a direct, non-manipulative

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effect of parasitism. Although these alterations in host behavior can effect changes in vulnerability to predators, they need not be the products of selection favoring enhanced transmission efficiency. Instead, they may simply reflect pathological responses to the presence of the parasite – albeit responses of value to the parasite.

Nevertheless, the hypothesis that parasites modify host phenotypes in a way that enhances transmission is a reasonable starting point when the shifts in phenotype increase the probability of predation by the next host in the life history sequence. The inference is especially promising when the changes in phenotype are clearly associated with abrupt transitions to infectivity in the parasite. If transitions to infectivity do not coincide with increases in parasite size or physiology likely to require suddenly enhanced resource acquisition from the host, manipulation by the parasite is likely.

Host manipulation that increases vulnerability to predation by the next host in the parasite's life cycle may also enhance vulnerability to predators that are not suitable hosts (Brassard et al. 1982). As long as the probability of transmission to an appropriate host is greater than the mortality cost associated with transmission to a non-host species, the parasite can benefit from manipulation of host behavior. Enhanced vulnerability to predators which cannot serve as hosts may be a necessary cost of elevated transmission to an appropriate host when multiple predators use similar sensory modalities to detect prey. We suspect, as did Brassard et al. (1982), that these costs are routinely incurred by parasites that infect fish which must be eaten by an avian definitive host to complete their life cycles. The fishes these parasites infect are likely to fall prey to visually hunting piscine predators (non-host species) as well as to birds (definitive hosts) which also rely predominantly on vision to capture prey.

Here we take advantage of a fascinating host-parasite relationship in which the host is apparently able to change both the behavior and coloration of the host in such a way that it enhances transmission to a definitive host – but appears to do so in two general stages. The host is the threespine stickleback fish, *Gasterosteus aculeatus* L., and the parasite is the tapeworm *Schistocephalus solidus* (Muller) (Cestoda: Pseudophyllidea). Over most of the range of this association, parasitism is associated only with a shift in host behavior (reviewed in Lobue and Bell 1993). In certain geographically restricted populations, however, late stages of parasitism are associated with a dramatic demelanization of the body and darkening of the eye (Lobue and Bell 1993). We demonstrate that changes in behavior may be initiated prior to demelanization, but that they are far more dramatic once demelanization has occurred. Furthermore, we provide evidence that the behavioral transition is likely to increase vulnerability to piscine predators of the stickleback that cannot serve as definitive hosts, as well as to the avian predators that can. Finally, we explore relationships among total tapeworm load, size of the largest parasite, and shifts in host behavior and color.

Natural history and study populations

The threespine stickleback, *Gasterosteus aculeatus*, is distributed widely in coastal marine, freshwater, and estuarine habitats in the northern temperate zone (Bell and Foster 1994, for review). Throughout this range, threespine stickleback inhabiting freshwater are parasitized by the cestode *Schistocephalus solidus* (Arme and Owen 1967). Infection rates vary from approximately one in 4000 to nearly 95% of individuals in a population (Arme and Owen 1967, McPhail and Peacock 1983, Godin and Sproul 1988, Jakobsen et al. 1988, Lobue and Bell 1993). Individual fish may be infected with 50 or more of these tapeworms (Arme and Owen 1967) although smaller numbers are more common (Chappell 1969, Meakins 1974, Reimchen 1982, Lobue and Bell 1993).

Infection of the threespine stickleback by *S. solidus* occurs when the stickleback consumes an infected copepod, the first intermediate host (Wedekind and Milinski 1996). The procercoid stage of the cestode burrows through the stickleback's gut wall to the abdominal cavity where it transforms to the plerocercoid stage. Among cestodes, *S. solidus* is unusual in that the plerocercoid attains a large size, causing visible distention of the host's abdomen (Smyth 1962, Holmes and Bethel 1972). Completion of the cestode's life cycle requires that the stickleback be eaten by one of the more than forty species of avian predators within which it can attain sexual maturity in 36–40 h (Smyth 1962).

Stickleback infected with *S. solidus* plerocercoids exhibit differences in anti-predator and foraging behavior from that exhibited by uninfected conspecifics. The differences are consistent with manipulation of host behavior by the parasite, although the variance in behavior within classes is high. For example, infected stickleback "attacked" by a model heron recover more quickly, and forage more actively shortly after the attack than do uninfected fish (Giles 1983, 1987). They tend to forage closer to predatory fish (Milinski 1984, 1985, 1990), swim closer to or at the surface more frequently (e.g. Arme and Owen 1967, Meakins and Walkey 1975, Giles 1983, 1987, Lobue and Bell 1993) in comparison to uninfected fish. Infected fish exhibit increased prey handling times (Cunningham et al. 1994), alterations in diet during high competition periods (Jakobsen et al. 1988) and may accrue significantly increased costs during shoaling (Barber and Huntingford 1995). Suppression of the normal anti-predator, or fright response is apparent only in fishes harboring infective plerocercoids (Tierney et al. 1993). Indeed,

there exists some evidence that hosts harboring uninfective plerocercoids exhibit enhanced anti-predator behavior (Tierney et al. 1993).

Oddly, none of these studies have attempted to relate the sizes of individual infective parasites, or the total parasite load, to the magnitude of the difference in behavior from that of uninfected fish. The potential importance of these variables in parasitized stickleback is indicated by the larger values of each in demelanized fish than in those with normal coloration. Demelanization is a remarkable color change that increases the visual conspicuousness of the host dramatically. The body is demelanized, becoming uniformly white. Coincidently, the eye darkens, becoming nearly black (Lobue and Bell 1993). The contrast between the eye and skin, in combination with the strong edge effect, should enhance the conspicuousness of the demelanized stickleback to vertebrate predators (reviewed in Lythgoe 1979) and should cause it to stand out from normally colored conspecifics, making it more easily tracked by a predator when moving in a group (Landeau and Terborgh 1986, Barber and Huntingford 1995).

Demelanized stickleback are present in populations only in very low frequencies. They appear to turn over rapidly, suggesting that they are more susceptible to predation than are fish with normal coloration (Lobue and Bell 1993). Demelanization is a phenomenon that apparently occurs only over the portion of the geographic range of threespine stickleback that spans Japan (Mori pers. comm.), Alaska, and northern British Columbia (Lobue and Bell 1993). This range presumably includes the Pacific coast of Russia, but no data are available.

The apparent, dramatic increase in conspicuousness, combined with evidence of the rapid disappearance of parasitized, demelanized stickleback, suggested to us that demelanization might signal a change in the degree to which the parasite manipulates the behavior of its host. We wished to test this hypothesis under natural conditions to control for effects of confinement in the laboratory and to evaluate the relationships among behavior, color, and parasite size and load, an endeavor that required observations of known individuals in the laboratory. We selected Bruce Pond (61.6 N latitude, 149.6 W longitude), in the Cook Inlet region of Alaska, for in situ observations, and for all collections used in the laboratory research. This pond was chosen because demelanized stickleback are reasonably common, and the pond is clear enough to permit observations.

Methods

We evaluated the anti-predator responses of uninfected, normal coloration infected, and demelanized, infected fish in two ways. First, we observed the reaction to a bird silhouette under laboratory conditions, a technique that enabled us also to ascertain parasite loads in the focal hosts. Second, we observed responses to a preserved trout under field conditions. In this instance we were able to ascertain behavioral responses under natural conditions and to examine position in the water column for each fish to address the issue of whether parasitized fish in either class were, under natural conditions, closer to the surface than were uninfected fish. All of these experiments were conducted in late June and early July 1994, when demelanized stickleback were more common than is the case earlier in the breeding season.

Predatory bird experiment

We hand-netted stickleback from Bruce Pond in July 1994, and maintained all individuals in a single large indoor tank for 48 h before testing. During this time they were fed *Tubifex* worms ad libitum. At the beginning of each trial, a single fish was placed in a 1-m diameter circular pool, and allowed to acclimate for 10 min. At the end of this time the fish had resumed normal exploratory movements. Water depth in the pools was 10 cm and no protective cover was provided. A uniform level of light was provided by a halogen bulb mounted 1.3 m above the pool. A curtain separated the pool from the experimenter and the surrounding room.

Following the acclimation period, the focal fish was observed until it swam into the center of the pool. At that time, a life-size model of a tern was allowed to glide down an inclined rope above the pool. As it passed under the light, the model produced a distinctive shadow that passed directly over the fish. We chose the shape of an Arctic tern, *Sterna paradisaea*, because the species is a known predator on stickleback (Lemmetyinen 1973), and was routinely observed feeding on stickleback in the Bruce Pond area.

In each trial, we recorded whether the stickleback changed behavior as the shadow passed over it. If the fish maintained a constant swimming speed and trajectory, "no response" was scored. If it changed direction suddenly, changed the speed or pattern of swimming, or froze as the shadow passed over it, a response was scored. Recovery time, the time until normal swimming behavior was again observed, was also scored.

At the end of each trial, the fish was anesthetized in MS-222 until quiescent, and then was preserved in 10% formalin. Each fish was subsequently dissected and all *S. solidus* were removed, the fish was eviscerated, blotted dry, and individually weighed. The blotted, wet weight of each parasite was also recorded. Two parasite indices were calculated for each fish using the formula $PI = P/(P + H)$, in which P is the blotted wet weight of

the parasite and H, that of the host (Arme and Owen 1967). In the first instance the total weight of parasites was used, in the second only the weight of the heaviest parasite was used. These are termed combined PI and individual PI following Lobue and Bell (1993). A total of 59 fish were examined. Of these, 20 were demelanized and 15 were parasitized, but of normal color.

Predatory trout experiment

We used a 30-cm rainbow trout, coated in resin, to elicit evasive maneuvers from unparasitized, parasitized normal coloration, and demelanized stickleback under field conditions. The trout was caught with hook and line, anesthetized with MS-222, preserved in formalin, and coated in resin following the methods of Helfman (1983) and Ridgway (1988). The trout was mounted on a grey wooden pole 60 cm in length. Previous research has shown that the responses of stickleback to this type of trout model are very different, and much more pronounced, than are those to the pole alone (Rodewald and Foster 1998).

All presentations were made while snorkeling. The observer moved slowly and smoothly toward a solitary stickleback deemed to be in one of the three parasitization categories. Slender fish with normal color were classified as unparasitized. Parasitized fish were recognized on the basis of the distention in the region of the pelvic girdle or anterior to it that distinguishes them from gravid females. They were further categorized as demelanized or of normal color. Once the trout was positioned within 80 cm of the focal stickleback, the trout was moved rapidly toward the stickleback, simulating attack by the trout. Although the model sometimes arrived within 5 cm of the stickleback, physical contact was avoided. The trout was then removed from view (simulating normal post-attack behavior by trout that have lunged unsuccessfully at stickleback; Foster and Ploch 1990).

As in the avian silhouette experiment, a response was scored if the fish changed swimming direction, speed, or pattern, if it darted for the bottom or other cover, or if it ceased motion entirely, while orienting toward the model. If the stickleback continued moving at the same speed, along the same trajectory, "no response" was scored. The recovery time was recorded as in the predatory bird test, and the distance traveled in an apparent attempt to evade the trout was also scored in increments of approximately 1/3 of a meter.

Before each test, the color of the focal animal was noted as was any indication of swelling due to parasites. Only those showing no indication of swelling potentially due to either gravidity or *S. solidus* were included in the uninfected category. Infected fish with normal color were identified by pronounced swelling anterior to the region that enlarges during gravidity, and demelanized fish could be unambiguously identified. The effectiveness of our assessment is indicated by the results of the parasite load evaluation associated with the avian predator experiment (below). We were unable to assess parasite load directly in this experiment because of the difficulty of catching stickleback that had just been startled by the attack of a model. A total of 124 stickleback were tested, of which 36 were infected but of normal color, and 64 were demelanized.

Results

Predatory bird experiment

Clear differences in parasite load among the three classes of stickleback were apparent. No parasites were found in any of the 24 fish visually deemed to be free of parasites, and eight of the 15 parasitized fish with normal coloration contained more than one parasite. Eighteen of the demelanized fish were infected by single parasites, with only one harboring two. The demelanized fish were thus more likely to possess a single parasite only than were parasitized fish of normal color ($G_{(Yates)} = 8.009$, d.f. = 1, $P < 0.005$). The differences in parasite numbers were also reflected in differences in combined PI between normally colored, parasitized fish (= 0.37, SD = 0.12) and demelanized fish (= 0.27, SD = 0.11). Arcsine transformed values of these indices differed ($t = 2.44$, d.f. = 33, $P < 0.02$).

The mean size of the largest parasite was greater in demelanized stickleback (= 0.138 g) than in parasitized stickleback with normal coloration (= 0.100 g, $t = 3.10$, d.f. = 33, $P < 0.004$). However, the individual PI did not differ ($t = 0.29$, d.f. = 33, $P = 0.78$) between parasitized, normally colored fish (= 0.25, SD = 0.08) and demelanized fish (= 0.26, SD = 0.07).

The three classes of stickleback did not respond similarly to the sudden overhead appearance of the tern silhouette ($G = 9.19$, d.f. = 2, $P < 0.025$; Table 1). Nonparasitized and normally colored, parasitized fish displayed indistinguishable responses, as did the parasitized, normally colored, and the demelanized fish. However, the demelanized fish were less likely to respond to the shadow than were the unparasitized fish ($G > 8.609$, d.f. = 1, $k = 3$, $P < 0.05$, STP test, Sokal and Rohlf 1995). Among those that did respond, significant differences were observed in recovery time fol-

Table 1. The number of stickleback in each color/parasitism category that did or did not respond to the shadow of a model tern flown overhead.

Parasitism category	Response	No response
Unparasitized	20	4
Parasitized, normally colored	10	5
Parasitized, demelanized	8	12

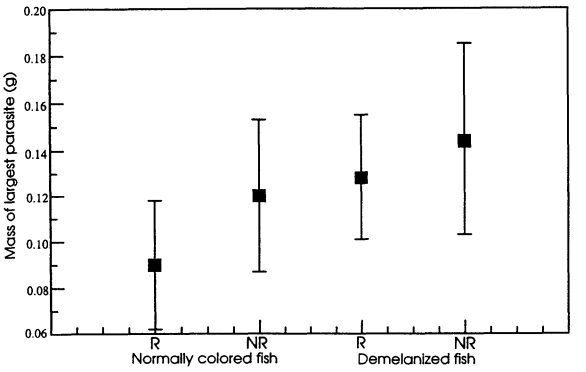

Fig. 1. Mean weights of the largest *Schistocephalus solidus* plerocercoid in demelanized and normally colored stickleback that responded to, or failed to respond to, a tern silhouette moving above. N indicates non-responders, R those that did respond. Plotted values are means ± SD.

lowing the response (Kruskal-Wallis test, $X^2 = 25.5$, d.f. = 2, $P = 0.0001$). Mean recovery time for unparasitized fish was 168 s (SD = 94), for parasitized fish with normal coloration, 42 s (SD = 48), and for demelanized stickleback, 4 s (SD = 4). When the parasitized stickleback were grouped according to color and response (Fig. 1) we found that the groups differed in size of the largest parasite (Kruskall-Wallis test, $X^2 = 10.8$, d.f. = 3, $P = 0.013$; Fig. 1). Post hoc comparisons among pairs of means (Mann-Whitney test, $P < 0.0083$ to adjust for making $n = 6$ non-independent contrasts) indicated that the primary difference in the overall significance among the four classes stemmed from the comparison of responsive, normally colored fish with non-responsive, demelanized fish.

Predatory trout experiment

The three classes of stickleback responded differently to simulated attacks by trout ($G = 35.85$, d.f. = 2, $P < 0.001$; Table 2). Demelanized fish responded less often than did those in the other two classes ($G > 8.609$, d.f. = 1, $k = 3$, $P < 0.05$, STP test), which fell into one non-significant subset. Fish in the different classes also fled differing distances ($G = 21.09$, d.f. = 2, $P < 0.001$; Table 3), such that demelanized fish fled shorter distances ($G > 8.609$, d.f. = 1, $k = 3$, $P < 0.05$, STP test) than did those in the other two groups, which did not differ (Table 3). The three classes also differed in the time between attack and resumption of normal swimming ($F = 20.58$, d.f. = 2,84, $P < 0.001$). Demelanized fish recovered most rapidly (= 11 s, SD = 8.3, $N = 34$), parasitized, normally colored fish recovered with intermediate speed (= 26 s, SD = 24.3, N = 31), and unparasitized fish recovered most slowly (= 48 s, SD = 35.3, $N = 22$; Tukey's studentized range test, d.f. = 84, $P < 0.05$).

There were no differences in the depth of water in which the classes of fish were found ($F = 2.93$, d.f. = 2,121, $P = 0.057$), although parasitized, normally colored (= 1.01 m, SD = 0.58, $N = 36$), and parasitized demelanized fish (= 1.01 m, SD = 0.56, $N = 64$), tended to be in shallower water than were non-parasitized fish (= 1.26 m, SD = 0.52; $N = 24$). None of the three types differed with respect to depth in the water column at which the fish were found ($F = 1.57$, d.f. = 2,121, $P = 0.21$).

Table 2. The number of stickleback in each color/parasitism category that did or did not respond to simulated attack by a trout.

Parasitism category	Response	No response
Unparasitized	24	0
Parasitized, normally colored	34	2
Parasitized, demelanized	35	29

Table 3. Distance fled by stickleback in the three parasitism/color categories following simulated attack by a trout.

	Distance fled (cm)			
	≤33	≤66	≤99	≥100
Unparasitized	8	8	3	5
Parasitized, normally-colored	19	10	0	5
Parasitized, demelanized	31	3	0	1

Discussion

Our data provide compelling evidence that infection with *Schistocephalus solidus* can result in pronounced changes in the behavior of the threespine stickleback host. The changes in behavior are consistent with the hypothesis of host manipulation in that they appear to involve the suppression of normal anti-predator behavior and therefore may increase the probability of transmission to the definitive avian host (Giles 1983, 1987, Tierney et al. 1993). The suppression of normal behavior appears to be greatest in demelanized fish, a finding that is not surprising given the difficulty of explaining the dramatic increase in conspicuousness other than as a consequence of parasite manipulation to increase the probability of transfer to a definitive host.

As we had anticipated, evasive maneuvers elicited from unparasitized fish by simulated attack with a preserved trout were less frequently observed in demelanized, parasitized fish. Not only did they respond less often, they fled shorter distances and recovered more rapidly than did unparasitized fish. These findings suggest that parasitized demelanized stickleback should be more vulnerable than unparasitized stickleback to inappropriate piscine predators as well as to definitive hosts. Because piscine predators are common in some lakes,

there is probably substantial parasite mortality associated with transmission to inappropriate hosts locally. This may be an unavoidable cost of host manipulation that enhances susceptibility to visually hunting vertebrate predators. The risk to the larger metapopulation will be substantially less however, as many lakes are devoid of piscine predators (e.g. Hagen and Gilbertson 1973, Bell et al. 1993, Bell and Foster 1994), and parasites can be readily transmitted among lakes by the definitive host. Consequently, the magnitude of selection imposed by transmission to piscine predators will be difficult to assess given the mosaic nature of the selective regime to which *S. solidus* is exposed. The problem will be exacerbated by directionality of gene flow that may be imposed by the migratory patterns of avian hosts.

An intriguing difference between our results and observations made previously involves the tendency of parasitized stickleback to exhibit buoyancy problems that cause them to remain closer to the surface than their uninfected counterparts. Earlier studies indicated that buoyancy problems could be severe and that they would hold stickleback close to the surface where they should be more vulnerable to avian predators (Arme and Owen 1967, Lester 1971, Meakins and Walkey 1975, Giles 1983, 1987, Lobue and Bell 1993). In contrast, our field observations failed to reveal differences in depth in the water column among the three classes of stickleback, and the depth of water in which parasitized and unparasitized fish were found did not differ.

Despite these findings, we were of the impression that demelanized fish experienced mild buoyancy problems in contrast to their uninfected counterparts. They appeared to move more slowly and to struggle more to remain at depth, although the effect was not of the magnitude described by Lobue and Bell (1993). As Lobue and Bell point out, the demelanized fish which they observed most closely had been held in aquaria for a long time, were larger than wild caught fish, and harbored much larger parasites than any they or we captured from natural populations. We suggest that although buoyancy problems may begin with the onset of demelanization, or even earlier, the problem does not ordinarily become as pronounced in this population as that described by Lobue and Bell. Instead, the behavioral changes we have described, coupled with changes in color, are apparently adequate to ensure rapid removal from the population before buoyancy disequilibrium forces the stickleback to remain near the surface.

As was the case in Lobue and Bell's (1993) study, all of our wild-caught demelanized stickleback harbored at least one parasite that weighed more than the 0.05 g that is thought to represent a threshold size for infectivity to the definitive host (Tierney and Crompton 1992). This result is consistent with parasite manipulation of host behavior. The absolute size of the largest plerocercoid seems to be better related to the probability of demelanization than is the individual PI, suggesting that effect of the parasite is not a simple consequence of the relative sizes of the host and parasite. This inference is further confirmed by the larger combined PI of normally colored than demelanized fish. However, there existed considerable overlap in the sizes of the largest plerocercoid between the two color phenotypes of infected hosts, suggesting that size of the largest tapeworm is not the sole determinant of demelanization.

The size of the largest parasite also seems to influence behavior as indicated by the difference in mean sizes of the largest plerocercoids between normally colored fish that responded to the tern shadow and demelanized fish that failed to respond. This difference, combined with the intermediacy of the size of the largest tapeworms in the other two behavior/color classes, suggests a gradual transition in control of the host by the maturing parasite. Because the behavioral transitions seem more closely linked to largest parasite size than to combined PI, dietary stress is a less likely cause of the behavioral transition than is a quantitative transition in host control with size and maturity of the largest tapeworm (Milinski 1984, 1985). Our results are equivocal, however, because of small sample sizes. They do suggest the value of further exploration of this relationship, especially if more response variables can be recorded and sample sizes can be increased.

The general pattern that emerges, then, is one in which there is a gradual loss of color and anti-predator behavior as a consequence of maturation of the largest tapeworm in the stickleback host. Other factors, including the number and size of parasites harbored by the host and the size and condition of the host at the time of infection are also likely to contribute to variation in the relationship between the size of the largest parasite and the resultant phenotype. An intriguing observation is that demelanized fish rarely host more than one tapeworm, suggesting that multiple infections or the presence of small individuals may suppress phenotype transitions that would otherwise be caused by larger parasites of the host.

Effects of *S. solidus* on host behavior are not restricted to a single trophic level. Copepods with infective procercoids were more active, demonstrated decreased swimming ability, were easier to catch, and tended to swim to the surface before and after simulated attacks in comparison to uninfected copepods. Consequently, they were consumed by stickleback disproportionately often (Wedekind and Milinski 1996). Clearly, behavioral modification by *S. solidus* has the potential to play an important role in the structuring of community interactions in populations with high levels of infection.

We suggest that demelanization signals a change in the relationship of *S. solidus* to its stickleback host. Behavioral changes in infected threespine stickleback

that increase susceptibility to the definitive host of the parasite begin prior to demelanization. However, there is a marked secondary increase in the suppression of normal anti-predator behavior associated with demelanization, which, in combination with the highly conspicuous coloration, likely increases susceptibility to visually hunting predators. Thus, although the parasite is large enough to be infective to a definitive host in many normally colored, parasitized fish, transfer is probably far more likely once demelanization and the associated secondary behavioral changes have occurred. The phenotypic transitions associated with demelanization are also likely to substantially increase predation upon the host and its parasite by inappropriate hosts, but not to the extent that would be measured within populations of stickleback exposed to piscine predators because so many populations are not exposed to these predators. Measurement of risk and its selective impact on *S. solidus* populations will certainly be extremely difficult – but could also provide substantial insight into our understanding of the evolution of host-parasite relationships across selective environments that vary geographically.

Given the probable dispersive capabilites of tapeworms that use birds as their difinitive hosts, the restricted geographic distribution of demelanization remains a puzzle. A possible, though untested, explanation lies in the existence of two morphologically cryptic clades of Pacific stickleback recently distinguished on the basis of allozyme and mtDNA sequence differences (Buth and Haglund 1994, Orti et al. 1994). The distribution of the "Japanese" stickleback clade matches the geographic distribution of demelanization, suggesting that the ability of the tapeworm to cause demelanization depends upon the host's clade. This intriguing possibility would suggest a change in host physiology while the Japanese clade was isolated from the more widespread "Euro-American" clade – a fascinating yet untested possibility.

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