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EFFECTS OF PARASITES ON FISH BEHAVIOR: INTERACTIONS WITH HOST PHYSIOLOGY

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I. INTRODUCTION

A. Parasites, Physiology, and Behavior

Being infected with parasites is the normal state for the vast majority of animals in natural, as well as in most managed populations. Consequently, developing an understanding of how infections impact on the biology of host organisms has considerable fundamental and applied value. Given the intimate associations between parasites and their hosts, infections are expected to impact on their hosts at a range of organizational levels, from gene expression to population dynamics. Parasites that cause mortality through direct pathogenic effects on their hosts have obvious importance because they impose a direct selection pressure on host populations. However, in recent years there has been recognition that sublethal physiological effects on hosts—which may lead to alterations in the behavior of infected animals—may also play an important role in regulating populations through demographic effects (Dobson, 1988; Hudson and Dobson, 1997; Finley and Forrester, 2003). The rise of behavioral ecology as a discipline devoted to the study of the adaptive function of behavior (Krebs and Davies, 1997) has facilitated our understanding of the role of sublethal parasite infections as selective agents in evolution. Elegant experimental tests of key hypotheses (e.g., Moore, 1983; Milinski and Bakker, 1990; Lafferty and Morris, 1996) strongly suggest that parasite infections can impact significantly on the natural and sexual selection of host populations through their effects on behavior.

Although behavioral and evolutionary ecologists have focused on examining the function and evolutionary consequences of behavioral changes associated with infection, there has also been interest in the proximate (physiological) mechanisms of host behavioral modification. A significant body of research has focused on the mechanisms of behavioral change in parasitized invertebrates (reviewed by Hurd, 1990; Adamo, 1997; Moore, 2002), which offer scientists an opportunity to study the mechanisms of manipulation in biological systems with relatively simple nervous systems (Adamo, 1997). However, research into the mechanisms by which parasites alter the behavior of vertebrate hosts has been limited by a lack of general knowledge regarding the physiological control of behavior in vertebrates. Writing over a decade ago, primarily from a mammal research perspective, Thompson and Kavaliers (1994) pointed out that:

> ''While it is clear that parasitism often brings about dramatic changes in host behaviour, little is understood of the physiological bases for these

changes and this in large measure reflects our primitive knowledge of the interaction of behaviour and physiology.''

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In the intervening years, considerable research effort by those authors and others has begun to shed light on the physiological control of behavior in mammals, and the mechanisms by which parasites can exert their influence.

Coordinated studies examining the physiological mechanisms of infection-associated behavioral change in fish have been similarly slow to emerge, and there are very few studies available that unambiguously link parasite-induced behavioral change to a physiological mechanism in fish. However, both physiologists and behaviorists have realized the value of fish as models with which to study the effects of parasites, and there is a wealth of information on the (separate) behavioral and the physiological effects of a wide range of parasitic infections. As a consequence, this Chapter often uses evidence from studies demonstrating a link between behavior and physiology in noninfected fish to identify potential mechanisms by which parasites may induce their observed behavioral effects on hosts. Clearly, more investigations are required that examine contemporal physiological and behavioral effects in the same host–parasite systems (Figure 4.1). As more of the mechanistic bases of behavior are being demystified by fish physiologists, there are an increasing number of opportunities for coordinated investigations into how parasites exert these influences on host behavior.

B. Why Study the Physiological Basis of Behavioral Changes Associated with Infections?

There are two fundamental reasons for studying the physiological bases of infection-associated behavioral change in fish. First, there is still very little data in support of a key hypothesis in evolutionary biology, which states that behavioral changes associated with infection in hosts have arisen as parasite adaptations to maximize transmission success (the Manipulation Hypothesis; Moore and Gotelli, 1990; see reviews by Barnard and Behnke, 1990; Barber *et al.*, 2000; Moore, 2002). Studies that demonstrate evolutionarily relevant changes in host ecology that are mediated through behavioral changes associated with infection have proven difficult to undertake and interpret (but for notable exceptions see Moore, 1983; Lafferty and Morris, 1996). Given the difficulty of acquiring data on the fitness effects of behavioral changes, studies that uncover the complexity of the physiological mechanism of behavioral change potentially provide alternative indirect evidence on the likely evolution of infection-associated traits (Poulin, 1998). Altered host behaviors that result from ''simple'' mechanisms, such

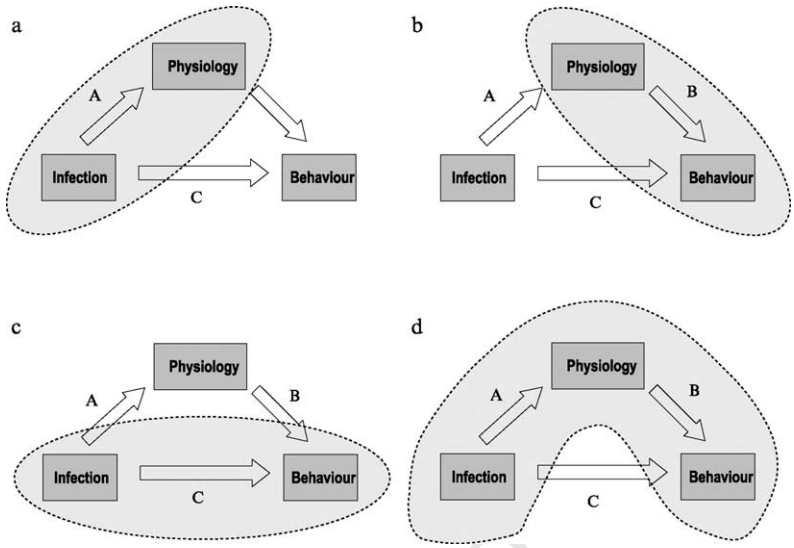

Fig. 4.1. Schematic illustrating the types of studies that have examined interactions between parasite infection, behavior and physiology in fish. (a) Differences in the physiology of parasitized fish, compared with uninfected individuals, are often recorded by physiologists or parasitologists studying either naturally infected or experimentally exposed individuals. (b) Physiological parameters are also commonly associated with behavioral traits, for example, reduced swimming performance associated with anemia. (c) In many cases, parasite infections may be demonstrated to influence host behavior. (d) More studies are needed to provide causal links between infection, host physiology, and behavior in the same host–parasite system.

as occupying a specific tissue to be in the required place for development or egg production, may arise as unavoidable side effects of infection that have neutral, or even detrimental, consequences for parasite transmission. On the other hand, behavioral effects of parasites that are mediated through complex mechanisms, such as parasite secretions that interfere with the neurochemical control of host behavior, are more likely to be true adaptations because they require investment on the behalf of the parasite (Poulin, 1998).

Second, because behavioral effects of parasites ultimately arise from physiological changes in the host, understanding the mechanisms by which parasites manipulate host behavior can yield important general information on the physiological processes responsible for control and modulation of behavior in the healthy organism (Helluy and Holmes, 1990; Thompson and Kavaliers, 1994).

There may also be applied value in examining the physiological basis of infection-associated behavioral change, because some of the traits exhibited

by infected fish may ultimately prove to be exploitable. For example, fish infected with fast-growing plerocercoids of pseudophyllidean cestodes feed more readily and as a consequence grow more quickly under particular feeding regimes than nonparasitized conspecifics (see Section 4.III.A.1). Such traits may be desirable in aquaculture or commercial fishery species, and understanding the physiological mechanisms by which parasites alter fish behavior (for example, by increasing the fish's appetite) may provide future opportunities to maximize productivity.

C. Biological Diversity of Fish Parasites

If we define parasites as organisms that live in or on another organism (termed the *host*) for at least some of their lifecycle and as a result cause harm to the host, then parasitic organisms include all viruses, many bacteria, and some fungi. However, the majority of parasitologists focus on protozoan and metazoan parasites (Bush *et al.*, 2001), as will this Chapter. Fish parasites are a diverse assemblage of organisms, both in terms of their taxonomy and in the ways in which they exploit fish as hosts. Lifecycle variation between parasite groups is substantial and important in understanding the ecological consequences of—and therefore the evolutionary pressures acting on—infection-associated changes in host behavior. From this perspective, perhaps the most important consideration is the type of lifecycle exhibited by the parasite. Some taxa (directly-transmitted parasites) are capable of completing their lifecycle on one individual fish, whereas others (indirectly-transmitted parasites) utilize the fish as one of a number of essential, sequential hosts. If more than one host is involved in the parasite's lifecycle, then fish may harbor the sexually active adult stage—in which case the fish is termed the *definitive host*—or they may serve as an intermediate host for a nonreproductive, developmental, or dormant stage of parasites. Because of their abundance and their typically intermediate position in aquatic food webs, fish are utilized as intermediate hosts by many indirectly transmitted parasites, with transmission to definitive hosts (which are often birds, mammals, or predatory fish) occurring through the food chain (*trophic transmission*). However, it is important to recognize that in many cases—particularly for marine parasites, and many of the myxozoans and microsporidians—lifecycles are not yet fully resolved, and so inferring likely ecological or transmission outcomes of infection or their evolutionary significance is not always possible.

This biological diversity of fish parasites—in terms of their taxonomy, life cycle details, the variety of ways in which they acquire their nutrients from host fish, their sites of infection and the extent to which they are nutritionally demanding—means that there are both mechanistic and

evolutionary reasons why we should not expect all parasites to have the same kinds of effects on the physiology or behavior of hosts.

D. Parasites and Fish Behavior

Parasites can impact on normal patterns of fish behavior in three main ways. First, because animals are expected to have evolved behavioral mechanisms to limit contact with infective stages to reduce the demand placed on the immune system (Hart, 1990), the threat posed by the presence of parasites in an environment may influence the behavior of fish even before they become infected. For example, fish have been shown to avoid particular types of habitat associated with infection risk (e.g., Poulin and FitzGerald, 1989), select against joining shoals containing parasitized individuals (e.g., Krause and Godin, 1996; Barber *et al.*, 1998), and reject parasitized sexual partners (e.g., Kennedy *et al.*, 1987; Milinski and Bakker, 1990; Rosenqvist and Johansson, 1995).

Second, parasitized fish may perform behaviors that reduce levels of infection with already-acquired parasites. Such behaviors range from simple ''flashing'' against the substratum or rubbing against other structural components of their environments to dislodge ectoparasites (e.g., Urawa, 1992) to complex interspecific social behaviors such as visiting ''cleaning stations'' on coral reefs (see Losey, 1987; Poulin and Grutter, 1996 for reviews). These first two types of behavioral effects of parasites constitute the ''behavioral resistance'' repertoire of a host (Hart, 1990), and these behaviors of hosts or potential hosts are generally regarded as host adaptations (e.g., Grutter, 1999). An in-depth coverage of behaviors that limit contact with parasites or reduce infection levels is outside the scope of this review, but further discussion can be found in Barber *et al.* (2000).

Third, parasite infections may cause host behaviors to alter in ways that serve to mediate the detrimental effects of infection. For example, the altered prey preferences and foraging behavior of sticklebacks infected with *Schistocephalus solidus* plerocercoids (e.g., Milinski, 1990; Ranta, 1995) increase food intake rates, and as such compensate to some extent for the nutritional demands of the parasites. Such behaviors are therefore readily explained as host adaptations to infection.

However, not all infection-induced behavioral changes are likely to be host adaptations, because parasite infections often cause behavioral change in infected hosts that have no obvious direct benefit to their hosts. In some cases, these behavioral changes may reflect parasite adaptations, increasing the probability of successful transmission; in other cases, the behavioral changes may be inevitable side effects of infection that benefit neither parasite nor host (Poulin, 1998). However, phenotypic correlations between

infection status and behavior can also be generated if behavior influences infection status rather than vice versa, because fish that exhibit pre-existing atypical behavior may be more exposed, or less resistant, to infections. For this reason, studies that demonstrate naturally infected hosts to display atypical behaviors cannot definitively identify causality; conversely, studies that demonstrate behavioral changes in experimentally infected fish may provide convincing evidence that behavioral changes are caused by infections. Fortunately, there are an increasing number of fish parasite systems for which experimental infections are possible.

E. How do Parasites Alter the Behavior of Fish Hosts?

Parasite infections that alter the behavior of their hosts have traditionally been proposed to do so through direct or indirect physiological mechanisms (Milinski, 1990). Direct mechanisms of behavioral manipulation are those in which the parasites themselves, or their biochemical secretions, act directly on the host's system of behavioral control. Examples of these kinds of mechanisms would include parasites that alter host behavior by releasing substances with neurotransmitter or neuromodulation capabilities, or which locate in and damage specific lobes of the brain. Alternatively, host behavior may be altered indirectly if parasites impose a constraint on some other aspect of the host's physiology. An example of indirect behavioral modification would be the increased foraging behavior of fish infected with nutritionally demanding parasites such as larval cestodes.

However, although separating indirect from direct mechanisms of behavioral manipulation may have heuristic value, it requires a level of detail regarding the host–parasite interaction that is generally not available (Milinski, 1990; Barber *et al.*, 2000). For instance, it is not known whether the altered swimming behavior of cyprinid fishes harboring heavy infections of brain-dwelling diplostomatid trematodes (see Section 4.IV.1) results from physical damage to the brain itself, a build-up of metabolic compounds from the parasites affecting neural or muscle function, or the secretion of behavior-modifying chemicals. In this Chapter, a different approach to understanding the physiological basis of behavioral change in parasitized fish is used.

Behavior can be broadly described as an animal's motor responses to multiple perceived external stimuli, detected and transduced into afferent nerve signals by a number of different sensory systems that are integrated by the central nervous system (CNS) and modulated by the physiological status of the animal. This Chapter highlights the impact of parasitic infections on normal patterns of host behavior by interference at each of four different physiological levels (Figure 4.2). First, parasites may influence behavior by

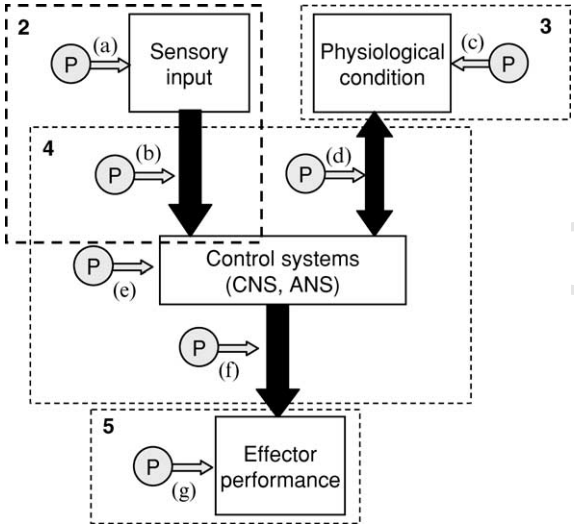

Fig. 4.2. Schematic illustrating the different ways in which parasite infections potentially influence host behavior. Parasites (P) can directly influence the quality of information received by a host by interfering with sensory function (a) or with the transmission of input signals by affecting neural performance (b). Parasites can also impact directly on host physiological condition (c) and on how host physiology and control systems (such as the CNS and ANS) interact (d). Furthermore, parasites can affect control systems if they cause either physical or neurochemical disruption (e) and they may also affect efferent neural function (f). Finally, parasites can impact directly on the functioning of effectors, such as muscles (g). Numbers relate to sections of this review dealing each type of effect.

affecting the quality and/or quantity of information obtained by hosts as they sample their environments, by affecting the functioning of peripheral sense organs (Section II). Second, parasites may change the internal nutritional status of fish hosts—and thus the motivational basis to respond to external stimuli—if infection has significant energetic consequences (Section III). Third, parasite infections may interfere directly with the control of host behaviors by physically damaging the CNS by their site selection, by manipulating levels of hormones or neurotransmitters, or by having neuromodulatory effects (Section IV). Finally, many of the effects that parasites have on host respiration, circulation, locomotion, or stamina can impact on the host's capacity to perform normal patterns of behavior in response to perceived stimuli (Section V).

The primary intention of this Chapter is to stimulate others to provide much-needed studies linking behavior and physiology in parasitized fish. With this in mind, the conclusion presents major gaps in knowledge and highlights new opportunities to exploit postgenomic technologies.

II. EFFECTS OF PARASITES ON THE SENSORY PERFORMANCE OF FISH HOSTS

Because parasites cause local pathology to host tissues by their attachment, movements, growth, or development, the specific sites they occupy may have important consequences for the type and extent of host behavioral change (Holmes and Zohar, 1990). Many endoparasites have a predilection for occupying sensory organs within their hosts, and damage to sensory tissues or occlusion of sensory organs may be sufficient to bring about changes to host behavior. It is thought that the evolution of parasite preferences for such sites has arisen primarily as a mechanism for evading the host's immune system (Szidat, 1969; Ratanarat-Brockelman, 1974; Cox, 1994), with any associated behavioral changes arising as (potentially fortuitous) side effects of immune avoidance (O'Connor, 1976). In fish, the eyes, nares, inner ear, and lateral line are frequently used as sites of infection by parasites (Williams and Jones, 1994). Here we examine the sensory systems of fish that are used as infection sites by parasites and review studies that have investigated their effects on host behavior and/or physiology. The sensory systems of fish are described in detail in Chapter II.

A. Behavioral Effects of Visual Impairment

A number of parasites utilize the fish eye as an infection site. Particularly common are the metacercariae of diplostomatid trematodes, including *Diplostomum* and *Tylodelphys* spp., which locate in the lens, retina, or vitreous humour. A range of freshwater species become infected with these and other "eyeflukes" when free-swimming cercariae, released from aquatic snails, penetrate the skin and travel to the eye in the host's circulatory system (Erasmus, 1959). In the case of *D. spathaceum*, the parasites invade the lens tissue, causing parasitic cataract disease in heavy infections (Chappell *et al.*, 1994). Intact fish lenses are optically complex tissues with remarkable resolving capacity (Fernald, 1993), largely as a consequence of variable refractive index throughout their depth, which counteracts spherical aberration (Sivak, 1990). Infected fish display behavioral changes that suggest the physical presence of parasites in the eye, and the damage caused during tissue migration is sufficient to impair vision. Infection with *D. spathaceum* is associated with reduced visual acuity in the three-spined stickleback *Gasterosteus aculeatus*, with infected fish initiating approaches toward motile prey at significantly reduced distances compared to noninfected fish (Owen *et al.*, 1993; Figure 4.3). Similar effects suggestive of reduced visual acuity have been demonstrated in infected dace (*Leuciscus leuciscus*), which feed less

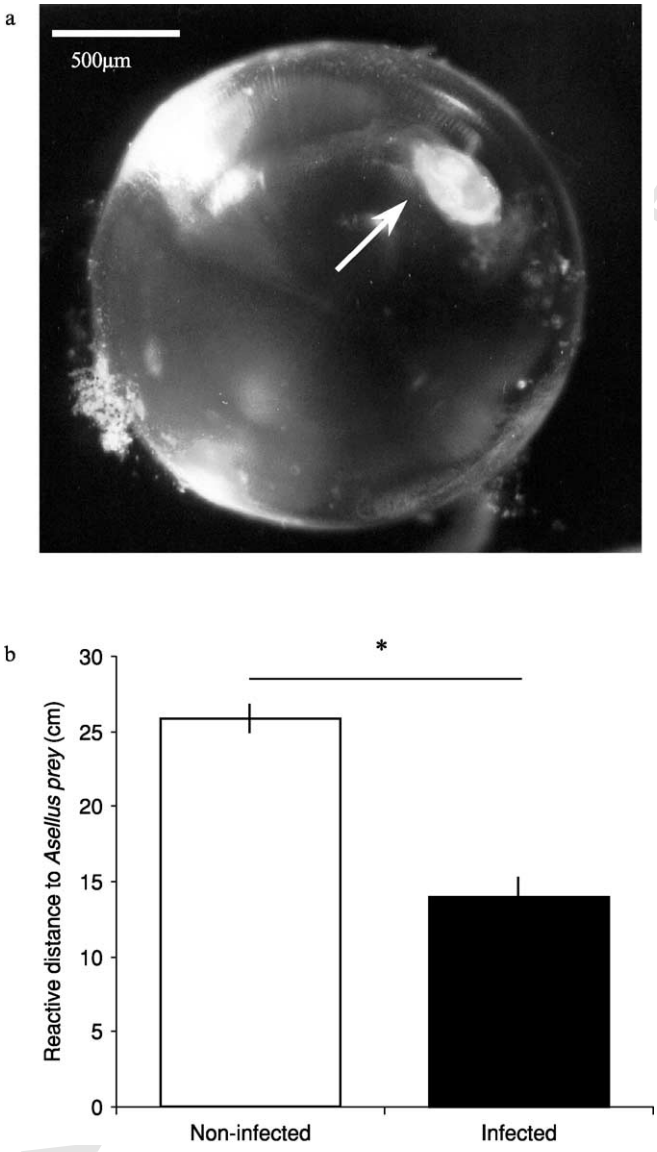

Fig. 4.3. (a) A single *Diplostomum spathaceum* metacercaria infecting the lens of a three-spined stickleback *Gasterosteus aculeatus*. (b) The effect of *D. spathaceum* infection on the reactive distance of three-spined sticklebacks to aquatic prey (*Asellus* sp.). Bar heights are means; error bars represent 1 standard deviation. $*P < 0.05$. (Data from Owen *et al.,* 1993.)

successfully on surface prey items and as a consequence spend a greater proportion of time feeding than noninfected fish (Crowden, 1976; Crowden and Broom, 1980), and in trout, which are caught less frequently by anglers in rod-and-line fisheries than in gill nets when infected with eyeflukes (Moody and Gaten, 1982). The reduced visual performance of infected fish, and the altered time budgets and spatial distributions that result, apparently increase their predation risk (Brassard *et al.*, 1982), with consequences for parasite transmission.

Other parasites, which are not restricted to infecting optical tissues, may nonetheless impair vision if they locate in the cornea or elsewhere in the head region. For example, xenomae of the microsporidian *Glugea anomala* often form in the head, causing the eye to swell (I. Barber, personal observation) and metacercariae of the digenean trematodes *Bucephaloides gracilescens* and *Cryptocotyle lingua* may encyst in the cornea (Karlsbakk, 1995), with likely but untested consequences for vision and behavior. Parasites that locate in lobes of the brain associated with vision or in the optical nerves may additionally impact on vision and behavior (see Section 4.IV.A).

B. Behavioral Effects of Impaired Chemoreception

The chemosensory systems of fish are extremely well developed and mediate many behaviors of fundamental importance (Hara, 1992; Sorenson and Caprio, 1998) including the location of food (Jones, 1992) and predators (Chivers and Smith, 1993). Fish in the order Ostariophysi have the additional ability to detect alarm substance ('Schreckstoff') released from the damaged skin of conspecifics and heterospecifics, and thereby can acquire immediate information on predator activity (Chapter 3.II.D; von Frisch, 1938; Wisenden *et al.*, 1994). Chemoreception is also known to play a major role in the return migrations of anadromous fish returning to spawn in natal rivers (Chapter 7.XII; Hara, 1993; Lucas and Barras, 2001) and in chemical communication (Brønmark and Hansson, 2000), with recent studies demonstrating that individuals can identify kin (Winberg and Olsén, 1992) and even nonrelated familiar individuals (Brown and Smith, 1994; Griffiths, 2003) by their odors. Moreover, recent research on sticklebacks suggests that mate choice decisions may be mediated by odors that relate to major histocompatibility complex allelic diversity (Reusch *et al.*, 2001; Milinski, 2003).

Chemosensation is achieved via at least two different channels in fish: olfaction (smell) and gustation (taste), but because both involve the detection of waterborne molecules, the distinction is not always clear (Hara, 1993). The primary sites of chemosensation in fishes are the olfactory epithelium situated within the narial canals (nares), taste buds located in the

mouth, and taste cells on the skin (Sorenson and Caprio, 1998), all of which may be invaded by parasites (Williams and Jones, 1994). Parasites locating in these organs may affect the sensitivity of their hosts to waterborne odors if they either restrict the passage of water through olfactory organs or damage the olfactory epithelium. Because fish use chemosensory detection to detect predators, there are reasons to expect that the chemosensory abilities of hosts may be a target of behavioral manipulation by trophically transmitted parasites. Given the current level of interest in the role of chemoreception in fish behavior, it is perhaps surprising that as yet there have been no studies that we are aware of examining the influence of parasite infections on the chemosensory abilities of fish and subsequent consequences for their behavior, and we highlight this as an area of significant research potential (Section 4.VI.A).

As well as impacting on the chemosensory abilities of their fish hosts, parasite infections may also alter host odors, allowing them to be discriminated by conspecifics in social or sexual contexts. In laboratory trials, mice are capable of detecting conspecifics infected with the nematode *Heligmosomoides polygyrus* based on odor cues alone (Kavaliers and Colwell, 1995; Kavaliers *et al.*, 1998a). Parasitized fish are frequently demonstrated to be unattractive companions or mates to individuals making shoaling or mate choice decisions (e.g., Rosenqvist and Johansson, 1995; Krause and Godin, 1996; Barber *et al.*, 1998) and although visual cues can be used to identify some infections (e.g., Krause and Godin, 1996), infection-associated odor variation may be a potential, though untested, mechanism allowing determination of the infection status of individuals by conspecifics. Given the importance of chemosensation to fish, it is not unrealistic to expect fish to be capable of identifying parasitized individuals by their odors, particularly if there are fitness costs of associating with them.

C. Behavioral Effects of Impaired Octavolateral Functioning

The octavolateral system of fish comprises three separate mechanosensory systems—the auditory, the vestibular (or equilibrium), and the lateral-line systems—that are traditionally studied together because they have a common sensory cell type as their major receptor (Schellart and Wubbels, 1998). The lateral-line and auditory systems detect mechanical vibrations, whereas the major roles of the vestibular apparatus of fishes are in the control of balance and posture and in visual stabilization (Popper and Platt, 1993; Schellart and Wubbels, 1998).

The organization of the auditory systems of fish has been reviewed in detail by Schellart and Wubbels (1998), and the importance of the production and detection of underwater sound in fish behavior was reviewed by Hawkins (1993) and in Chapter 2.VI of this volume. The auditory system

comprises three semicircular canals and three calcareous otolith organs and is located in the inner ear. Underwater sound waves are detected when movements of the fish's body, caused by molecular vibration of the water, cause the hair cells to oscillate with respect to the otoliths. The orthogonally arranged semicircular canals of the inner ear form the equilibrium system, providing information on the three-dimensional orientation of the fish (Bone *et al.*, 1995). The inner-ear labyrinth, containing both systems, is calcified to form a single massive structure in teleosts and is filled with a fluid (endolymph). Parasites that inhabit the inner ear, or cause a change in the structure of the semicircular canals or the endolymph contained within, have the potential to impact significantly on the ability to achieve balance and postural control and to detect underwater sounds.

The most common parasite infections of the inner ear of teleost fish are various species of myxosporean, including *Myxobolus cerebralis*, which destroy the cartilage of the inner ear of salmonids (Markiw, 1992). The main behavioral symptom of infection is uncontrolled erratic circular swimming at the water surface termed *whirling* (Uspenskaya, 1957; Kreirer and Baker, 1987; Markiw, 1992), as would be expected for a parasite that damaged the major balance organs. However, other pathomechanisms generating the observed whirling behaviors have been suggested (see Section 4.IV.A) and it is possible that a range of physiological effects may generate phenotypically similar behavior effects in parasitized fish.

An additional component of the auditory system of fishes is the swimbladder, which functions as a pressure-to-displacement transducer and lowers the auditory threshold (Schellart and Wubbels, 1998). Parasites that inhabit the swimbladder (see also Section 4.V.C) therefore potentially interfere with sound detection, but we are aware of no studies examining this.

The lateral-line system of fishes serves primarily as a hydrodynamic receiver, detecting pressure changes in the water immediately surrounding the fish (Popper and Platt, 1993; Schellart and Wubbels, 1998), and is utilized by a range of parasites as an infection site. Metacercariae of the digenean *Ribeiroia marini* are found in the lateral-line scale canals of goldfish *Carassius auratus* (Huizinga and Nadakavukaren, 1997) and philichthyid copepods inhabit the lateral-line sensory canals of tropical and temperate marine fishes (Kabata, 1979; Grabda, 1991; Hayward, 1996). The latter may be common in fish populations: 31% of corkwing wrasse (*Crenilabrus melops*: Labridae) off the coast of Ireland were infected with *Leposphilus labrei* (Donnelly and Reynolds, 1994), with heavy infections being associated with tumor formation. The behavioral functions of the lateral line include the localization of moving prey, the detection of approaching conspecifics and predators, obstacle detection (particularly in low-visibility conditions), and schooling behavior (Bleckmann, 1993).

Studies in which lateral-line function is experimentally impaired demonstrate the potential for such parasites to impact on host behavior. For example, mechanical blocking of lateral-line canal pores eliminates the feeding response of mottled sculpin *Cottus bairdi* (Hoekstra and Janssen, 1985), and cauterization of the posterior lateral line nerves is sufficient to impair the ability of saithe *Pollachius virens* to maintain accurate spatial positions within dynamic swimming schools (Partridge and Pitcher, 1980). Infections with parasites such as those detailed above are likely to impact on water flow in the lateral-line canals and reduce sensitivity to pressure changes; however, despite the potential for infection-associated behavioral change, the authors are aware of no studies that have examined behavioral effects of lateral-line–dwelling parasites.

Endoparasites such as *Ligula intestinalis* or *Schistocephalus solidus* that grow to a large size and cause gross swelling of their hosts' bodies also have the potential to impact on lateral-line function. Although there are as yet no physiological data available, the schooling behavior of European minnows infected with *Ligula* is strikingly similar to that of saithe deprived of lateral-line function. In mixed infection schools, infected minnows exhibit larger nearest-neighbor distances (NNDs) and a greater propensity to occupy peripheral positions within schools than uninfected conspecifics; although they respond to a simulated avian strike by reducing their NND, they continue to take up spatially inappropriate positions after attack (Barber and Huntingford, 1996). Sticklebacks infected with *Schistocephalus* plerocercoids also exhibit impaired escape responses, including a reduced propensity to react to a striking model heron (Giles, 1983; Godin and Sproul, 1988; Tierney *et al.*,1993; Ness and Foster, 1999; Barber *et al.*, 2005), as do roach infected with *Ligula intestinalis* (Loot *et al.*, 2002a). Because the model predators may strike the water surface in these trials, one explanation for the absence of a response from infected fish is that infected fish fail to sense water pressure changes via their lateral line. Future studies of escape response using model predators should be designed more carefully to allow a detailed interpretation of the results and ascertain which sensory modalities are impaired by infection.

D. Behavioral Effects of Impaired Electrosensation

Some fish have the capacity to produce electrical pulses that are used actively in foraging, object detection, or communication (Bleckmann, 1993). However, the ability to detect electric fields (*electroreception*) is not just limited to these electric fish; the majority of nonteleost fish, as well as some teleosts including the Ostariophysans and Osteoglossiformes, also have low-frequency electroreceptors and use these passively in prey detection,

long-distance orientation, and migration (Bleckmann, 1993). Despite considerable research interest in electrosensation and electrobiology of fishes (comprehensively reviewed by Moller, 1995), the authors are not aware of any studies that have examined the impact of parasites on active or passive electrosensory function or behavior.

III. BEHAVIORAL CONSEQUENCES OF PARASITE-IMPOSED CONSTRAINTS ON HOST PHYSIOLOGY

The internal physiological state of animals may have a considerable effect on their behavior. For example, following a period of food deprivation, fish will take more risks while foraging (Damsgård and Dill, 1998) and ingest more food in a single meal than fish in better nutritional condition (Ali *et al.*, 2003), whereas fish that are under osmoregulatory stress often exhibit altered locomotory and antipredator behavior (e.g., Handeland *et al.*, 1996). Hence, parasites that impose constraints on the physiological status of their hosts potentially impact indirectly on the behavior of their hosts. This Section focuses on the observed and potential behavioral consequences of infections resulting from their effects on host energetic status and capacity for osmoregulation.

A. Behavioral Consequences of Nutritionally Demanding Parasites

1. Effects on Foraging Behavior

All parasites utilize host-derived resources for their growth and development, and as such impose an energetic drain on host organisms. As a consequence, energetic effects of infection are commonly reported, particularly when the parasites involved are large, rapidly-growing, numerous, or highly pathogenic. In addition, if infections reduce the competitive ability or foraging success of hosts (for example, as a consequence of infection-induced damage to sense organs; see Section II), then parasites may limit the nutrient intake of their hosts. Fish infected with parasites that impose a significant energetic cost may therefore be forced to increase their foraging effort or alter their foraging strategies to compensate.

The most detailed studies of behavioral changes induced by energetically demanding parasites have examined fish harboring the large plerocercoid larvae of the closely related pseudophyllidean cestodes *Schistocephalus solidus* and *Ligula intestinalis*, which infect sticklebacks and cyprinids respectively. Infections are acquired when fish hosts feed on parasitised copepods, and the plerocercoids grow rapidly to a large size in the peritoneal cavity of

host fish. Evidence from laboratory and field studies shows that, during the parasite growth phase, these parasites impose a considerable energetic drain on their hosts. Infected wild caught fish typically have lower somatic body condition and/or liver energy reserves (Arme and Owen, 1967; Pennycuick, 1971; Tierney, 1994; Tierney *et al.*, 1996) as do experimentally infected fish held under a fixed ration of 8% body weight per day over the parasite growth phase (Barber and Svensson, 2003). Furthermore, data on the amino acid composition of *Ligula*-infected roach are consistent with those of starved fish (Soutter *et al.*, 1980). The energetic problems faced by infected fish are compounded by two further factors: (1) the growing plerocercoid reduces the space available for stomach expansion, limiting meal size (Cunningham *et al.*, 1994; Wright *et al.*, manuscript in preparation); and (2) infected fish are poor competitors for food (Milinski, 1990; Tierney, 1994; Barber and Ruxton, 1998) as a result of impaired foraging performance (Cunningham *et al.*, 1994).

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Fish under nutritional stress, such as after a period of food deprivation, typically exhibit hyperphagia on refeeding to maximize their food intake (Ali *et al.*, 2003). Although increasing meal size is not an option for fish infected with cestode plerocercoids, they are able to partially alleviate the combined effects of infection by altering their foraging behavior in other ways (Milinski, 1990). First, infected sticklebacks switch foraging strategies, taking advantage of ''risky'' food sources such as those available close to potential predators (Giles, 1983; Milinski, 1985; Godin and Sproul, 1988; Barber *et al.*, 2005) and altering food selection strategies to focus on prey types for which there is less competition (Milinski, 1984; Tierney, 1994; Ranta, 1995; see discussion in Barber and Huntingford, 1995). Second, infected fish may spend a greater proportion of their time foraging (e.g., Giles, 1987), possibly at the expense of shoal membership (Barber *et al.*, 1995).

Intriguingly, these strategies employed by infected sticklebacks to counter the energetic costs of infection are remarkably similar to those documented for salmonids injected with a growth hormone (GH) supplement (e.g., Jönsson *et al.*, 1998). By stimulating growth, GH increases metabolic demands, which must largely be met by increased nutrient intake, and one possibility is that energetically demanding parasites may exert their behavioral effects on hosts through the same physiological mechanisms. The control of food intake in fish is under the influence of a complex suite of factors including stomach distension, the level of circulating hormones (including insulin), and blood nutrients (including glucose) (see reviews by Le Bail and Boeuf, 1997; Donald, 1998). Indirect evidence supporting the hypothesis that parasites can exploit the growth hormone pathway to induce behavioral change in their hosts comes from two separate studies. First, mice infected with plerocercoids of *Spirometra mansonoides* (a close relative of

Schistocephalus and *Ligula*) show almost identical changes in growth and physiology to those given GH supplementation (see review by Phares, 1997). Subsequent investigations have revealed that plerocercoids secrete a 27.5-kD protein known as plerocercoid growth factor (PGF), which has growth hormone activity (Phares and Kubik, 1996). Second, Arnott *et al.* (2000) demonstrated that three-spined sticklebacks experimentally infected with *Schistocephalus solidus*, when allowed access to unlimited food resources, outgrew sham-infected controls, suggesting that infections increased feeding motivation and total food intake. As yet there is no direct evidence that a parasite-produced growth factor is involved in mediating foraging behavior in sticklebacks infected with *Schistocephalus solidus*, but one explanation for their enhanced growth in laboratory studies is that they are more willing than uninfected fish to feed under unfamiliar conditions. Future studies should focus on whether this readiness to feed is also observed under natural conditions, and whether it may be mediated through infection-induced changes to GH pathways.

Although we have focused primarily on infections that are associated with an increase in host foraging behavior, fish infected with highly pathogenic infections may more typically show a reduction in food intake. Reductions in voluntary meal size have been demonstrated in rainbow trout (*Oncorhynchus mykiss*) infected with the kinetoplast *Cryptobia salmositica* (Lowe-Jinde and Zimmerman, 1991) and in Atlantic salmon (*Salmo salar*) infected with the sea lice *Lepeophtheirus salmonis* (Dawson *et al.*, 1999). The physiological mechanisms reducing appetite in these groups have not been explicitly investigated.

2. Effects on Reproductive Behavior

Gonadogenesis is typically delayed, impaired, or reversed in fish that are under nutritional stress, so it is not unexpected that parasite infections are often associated with reduced gonadogenesis or fecundity (e.g., Chen and Power, 1972; Wiklund *et al.*, 1996). Female common gobies (*Pomatoschistus microps*) harboring the adult stage of the trematode *Aphalloïdes coelomicola* in their body cavities exhibit reduced gonad weight as a consequence of reduced mass and energy content (but not diameter or number) of individual ova (Pampoulie *et al.*, 1999). The myxozoan *Kudoa paniformis*, which infects fibers of the skeletal muscle system of Pacific hake *Merluccius productus*, is associated with reduced fecundity of female hosts, with increased infection intensity having more severe effects (Adlerstein and Dorn, 1998). Furthermore, because reproductive behavior in fish is initiated by the release of hormones from mature gonads (see Chapter 9) then parasite infections that impair gonadogenesis through their nutritional effects are likely to have consequences for the sexual behavior of hosts.

However, it is difficult to attribute parasite-associated changes in sexual development solely to the energetic consequences of infection, as gonadogenesis in fishes is under the control of pituitary gonadotropins (GTH-I and GTH-II) as well as a multitude of hormones and growth factors (see Van Der Kraak *et al.*, 1998 for a detailed review). Yet the fact that *Schistocephalus solidus* infections impair gonadogenesis in female three-spined sticklebacks in natural populations—with infected individuals having smaller ovaries (McPhail and Peacock, 1983; Heins *et al.*, 1999) that contain fewer and smaller eggs (Heins and Baker, 2003) despite pituitary function being apparently unaffected in this system (Arme and Owen, 1967)—suggests that the energetic effects of infection per se may be important. In male sticklebacks, the finding that *S. solidus* infection does not impair nest building and courtship behavior in lab trials (with freely-available food), despite the fact that few infected breeding males are ever located in surveys of natural populations (Candolin and Voigt, 2001), also suggests that infection reduces sexual behavior solely through its nutritional effects.

Parasites that localize inside the host gonads may also considerably reduce the reproductive capacity of host fish through direct nutrient depletion. *Proteocephalus ambloplitis* plerocercoids penetrate the ovaries of host bass *Micropterus salmoides*, destroying individual oocytes and possibly utilizing nutrients in the yolk for their own growth (McCormick and Stokes, 1982). Not all parasite infections reduce gonad size, though. Infections with *Kudoa ovivora*—a recently discovered myxozoan that lives inside the ova of Caribbean labroid fishes—are associated with increased egg mass as the parasite channels nutrient resources from the host into gonad development. This benefits the parasites developing within individual ova, but as infected ova are nonviable, infections impact negatively on host fecundity and sexual activity (Swearer and Robertson, 1999). Roach infected with *Ligula* do not engage in species-typical spawning behavior (Dogiel *et al.*, 1961), but it is not clear whether this is a consequence of direct manipulation (via pituitary action of the worm; see Section 4.IV.D) or an indirect effect of the failure of gonadogenesis.

B. Behavioral Consequences of Parasites with Homeostatic Effects

Although the intact teleost epidermis is relatively impervious to water, all fishes have large areas of permeable epithelia (including the gill lamellae, oral and narial mucosae) in contact with their aquatic medium (Bone *et al.*, 1995). As very few fish are isosmotic with the water they live in, they must cope with diffusion gradients that build up across these permeable surfaces by investing in active osmoregulation (Karnaky, 1998).

Fish ectoparasites that damage water-resistant epidermal tissues or permeable epithelia of their hosts, or which impair the functioning of osmoregulatory systems or tissues such as the gills, thus have the potential to impose osmoregulatory stress on their hosts. The effects of salmon lice (*Lepeophtheirus salmonis*) on the osmoregulatory capacity of host fish have been particularly well studied. Heavy infections with these parasites, which feed by sloughing off skin cells and damage the epidermis, result in osmoregulatory failure (Birkeland, 1996; Birkeland and Jakobsen, 1997), which leads to a reduction in swimming and cardiovascular performance (Wagner *et al.*, 2003) and ultimately results in the cessation of swimming activity (Grimnes and Jakobsen, 1996). As active osmoregulation is energetically expensive, locating ideal (isosmotic) aquatic environments may have significant benefits even to healthy fish, because emancipation from osmoregulation means that more energy can be made available for growth (e.g., Woo and Kelly, 1995; Riley *et al.*, 2003). Although this option is unavailable to marine or landlocked freshwater species, infected diadromous or estuarine fishes may be able to locate to osmotically favorable environments to facilitate survival and recuperation from infections. Infection-induced osmotic intervention may therefore be expected to impact on the migratory strategies of diadromous fish, and data exists to suggest this may be the case (see Chapter 7 for more details on fish migration).

Bjørn *et al.* (2001) found that anadromous sea trout *Salmo trutta* and Arctic char *Salvelinus alpinus* returning earliest to freshwaters were those most heavily infected with *L. salmonis*, suggesting that early return migrations are triggered to reduce the physiological (osmoregulatory) consequences of infection (see also Birkeland and Jakobsen, 1997). Boyce and Clarke (1983) demonstrated that experimentally induced *Eubothrium salvelini* infections reduce the capacity of migratory sockeye salmon to adapt physiologically to seawater, and suggest that as this inability would be likely to reduce ocean survival, and so infection of fish may delay or prevent seaward migrations. Sproston and Hartley (1941) recorded disproportionately high levels of infection with the ectoparasitic copepod *Lernaeocerca branchialis* among whiting (*Merlangius merlangus*, Gadidae) and pollack (*Pollachius pollachius*, Gadidae) delaying annual seaward migrations in an English estuary at the end of the winter. The authors suggest that the delay in migration could result from chronic anemia or a physiological response to ionic imbalance and subsequent avoidance of higher salinity water to which migrations normally are directed. However, as it has also been suggested that heavily infected individuals may also alter the timing of migrations to maximize foraging success (e.g., Bean and Winfield, 1992), experimental studies examining the salinity preferences of experimentally infected fish would be valuable in separating these factors.

IV. EFFECTS OF PARASITES ON HOST CONTROL SYSTEMS

A. Behavioral Effects of Parasites that Invade the Central Nervous System

Like the peripheral sense organs, the CNS offers a refuge from the host's immune system and therefore is attractive to parasites. Locating in the CNS also provides parasites with the potential to directly affect behavior and there is evidence that, in some cases, fine-scale site selection may have evolved to influence host behavior in ways that benefit the parasites. Some of the most commonly recorded parasites of the CNS are the metacercariae of diplostomatid trematodes, including *Diplostomum phoxini* (Figure 4.4) and *Ornithodiplostomum ptychocheilus*, which infect old and new world minnows respectively. Histological studies have demonstrated that metacercariae are not randomly dispersed throughout the brains of infected fish, but are aggregated in lobes of brain concerned with vision and motor control (Barber and Crompton, 1997; Shirakashi and Goater, 2002). Heavy infections are associated with impaired optomotor responses (Shirakashi and Goater, 2002), altered shoaling behavior (Radabaugh, 1980), and altered swimming behavior of host fish (Ashworth and Bannerman, 1927; Rees, 1955; Lafferty and Morris, 1996), suggesting that damage caused to specific

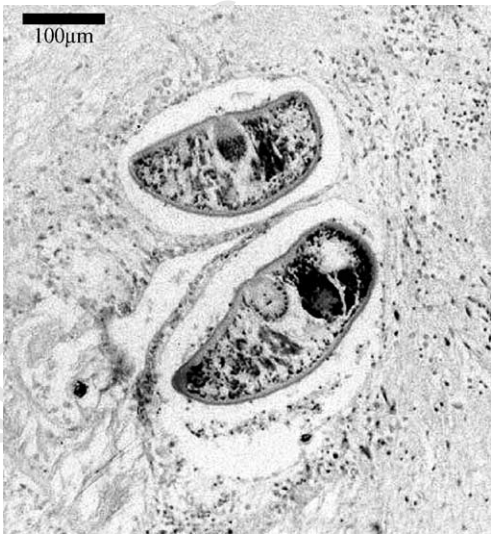

Fig. 4.4. Light micrograph of a sagittal section of two metacercariae of the digenean trematode *Diplostomum phoxini* in the medulla oblongata of the brain of a European minnow *Phoxinus phoxinus*.

brain regions by large aggregations of parasites is likely to be responsible. Such infections may have a considerable ecological impact through their effects on host behavior. In a landmark study, Lafferty and Morris (1996) found that California killifish (*Fundulus parvipinnis*) infected with the brain-encysting metacercariae of the trematode *Euhaplorchis californiensis* performed more "conspicuous" swimming movements than noninfected conspecifics, and in field enclosure experiments were 30 times more likely than noninfected conspecifics to be eaten by piscivorous birds—the definitive hosts of the parasites.

Other parasites that do not live in the brain tissues may nonetheless damage the CNS and impact directly on host behavior if they occupy the cranial cavity and/or vertebral column and exert pressure on the CNS tissues. The myxozoan *Myxobolus arcticus* inhabits the cranial cavity of sockeye salmon, and infections are associated with a significant reduction in swimming speed (Moles and Heifetz, 1998). Salmonids infected with *Myxobolus cerebralis* display a behavioral syndrome consisting of sequences of tight turns (whirling), interspersed with periods at rest with the tail elevated above the head, and episodes of postural collapse and immobility. Although the behavioral syndrome has previously been thought to result from damage to the vestibular apparatus (see Section 4.II.C), evidence from recent studies suggests a neurophysiological basis of behavioral changes associated with infection. Rose *et al.* (2000) argue that the behavioral syndrome is inconsistent with impaired vestibular function, alternatively proposing that behavioral changes result from granulomatous inflammation (associated with parasite invasion of the skull and vertebral column), which constricts the upper spinal cord, compressing and deforming the lower brainstem. Supportive evidence comes from studies of other myxozoans, for which infection is associated with both swimming disorders and similar host pathology (Grossel *et al.*, 2003; Longshaw *et al.*, 2003), and further studies are required to determine the precise physiological basis of whirling behavior in infected fish.

B. Behavioral Effects of Parasites Impacting on the Autonomic Nervous System

The autonomic nervous system (ANS) of fish regulates the cardiovascular system, gastrointestinal system, swimbladder function, spleen and urogenital system, as well as controlling the expansion and contraction of melanophores and iris colouration (Donald, 1998). The ANS therefore plays an important role in regulating aspects of behavior, and studies on nonfish taxa have demonstrated that parasite infections can impact on host ANS function. For example, in the rat (*Rattus rattus*), L3 larvae of the parasitic

nematode Anisakis simplex (a fish parasite) induce cholinergic hyperactivity and adrenergic blockade in the whole of the small intestine, causing gastrointestinal symptoms that influence foraging behavior and appetite (Sanchez-Monsalvez *et al.*, 2003). In humans, although the mechanism is not well understood, some trypanosome infections are associated with autonomic dysfunction, including neurogenic cardiomyopathy and digestive damage (Sterin-Borda and Borda, 2000; Pinto *et al.*, 2002). Recent studies on brown trout Salmo trutta have shown that infections with the intestinal acanthocephalan Pomphorhynchus laevis and the cestode Cyathocephalus truncates are associated with changes in the numbers of endocrine cells in the gastrointestinal tract showing immunoreactivity to a range of neurotransmitters (including bombesin, cholecystokinin-8 [CCK-8], leu-enkephalin and 5-hydroxytryptamine [5-HT], serotonin; Dezfuli *et al.*, 2000, 2002) known to control gut motility and digestive/absorptive processes in fish (Donald, 1998). Behavioral studies allied to these investigations of ANS and gut function infection would be invaluable in generating a more complete understanding of how parasite infections impact on the foraging ecology and growth of infected fish.

C. Neurochemical Interference with Host Behavior

Brain monoamine neurotransmitters are involved in the control of feeding behavior (De Pedro *et al.*, 1998), social behavior and aggression in fish (Winberg and Nilsson, 1993), and are sensitive to a range of stressors (Chapter 5.III.; Winberg and Nilsson, 1993), immunological factors (Lacosta *et al.*, 2000) and nutritional status (Levin and Routh, 1996). Monoamine neurotransmitters are also essential in pathways associated with sensory and motor performance, in addition to higher brain function (at least in mammals). Moreover, monoamines such as serotonin are also now known to act as neuromodulators, playing an active role in the recruitment of neurones to neuronal assemblies, resculpting neural circuits and giving animals the flexibility to shape their behavior in response to the changing demands of their internal state and the external environment (Thompson and Kavaliers, 1994; Adamo, 2002). This feature of monoamines potentially provides parasites with a ready-made mechanism for manipulating host behavior.

A number of studies have examined the role of neurotransmitters in the control of behavior and the influence of parasites in invertebrate host models (see reviews by Adamo, 1997; Moore, 2002). Helluy and Holmes (1990) demonstrated that the peculiar impaired escape responses of gammarids infected with *Polymorphus paradoxus* (Bethel and Holmes, 1973) could be precisely replicated in healthy specimens by the injection of serotonin;

moreover, the altered behaviors could be reversed by injecting with octopamine, a serotonin antagonist. More recently, Helluy and Thomas (2003) used immunocytochemical methods to examine changes in the serotonin-immunoreactivity of brain regions in gammarids infected with another behavior-changing parasite, *Microphallus papillorobustus*. Infection was found to be associated with altered serotonergic sensitivity in specific brain regions and altered architecture of the serotonergic tracts and neurons, suggesting that parasites may have exerted their influence through neuromodulatory mechanisms.

In mammals, the most detailed studies of the neurochemical basis of parasite-induced behavioral change have been carried out by Kavaliers and coworkers (reviewed by Kavaliers *et al.*, 1998b; Moore, 2002) and have suggested a role for serotonin. In noninfected mice, brief exposure to predatory threat typically induces nonopioid-mediated, serotonin-sensitive analgesia (reduced pain sensitivity). However, Kavaliers and Colwell (1994) found that mice infected with the coccidian *Eimeria vermiformis* failed to exhibit serotonin-sensitive analgesia when presented with a cat stimulus. In addition, although general olfactory functioning appeared unaffected, infected mice failed to avoid cat odor. Although the parasite would not benefit from increased predation because transmission occurs directly between mice via fecal contamination, the observed "fearlessness" of infected mice may also increase mouse–mouse social contact, which would have consequences for transmission. As with infected gammarids, one physiological explanation for this fearlessness of infected mice is that the parasite influences host behavior by interfering with serotonergic activity.

Although there is considerable research interest in the neurochemical regulation of behavior in fish, there is very little information on parasite modulation. In the only study of parasite-associated neuromodulation in fish, Øverli *et al.* (2001) investigated the effects of *Schistocephalus solidus* infection status on the concentrations of neuromodulators, including norepinephrine (NE) and serotonin, in the brains of three-spined sticklebacks. Infected fish had elevated concentrations of NE and serotonin in the telencephalons, but the clearest effect of infection was on the serotonin metabolite, 5-hydroxy-indoleacetic acid (5-HIAA), which increased in the brainstem of infected sticklebacks. As a consequence, the ratio of 5-HIAA to serotonin (an indicator of stress in fish) was elevated in both the hypothalamus and brainstem regions of infected sticklebacks (Figure 4.5). One explanation for these patterns is that the parasite induces chronic stress, but it is also possible that the parasite manipulates the neuroendocrine status of its host directly. Behavioral changes in sticklebacks infected with the plerocercoid larvae of *Schistocephalus solidus* have been extremely well documented, and infected fish are known to show altered risk-taking and antipredator behavior (see

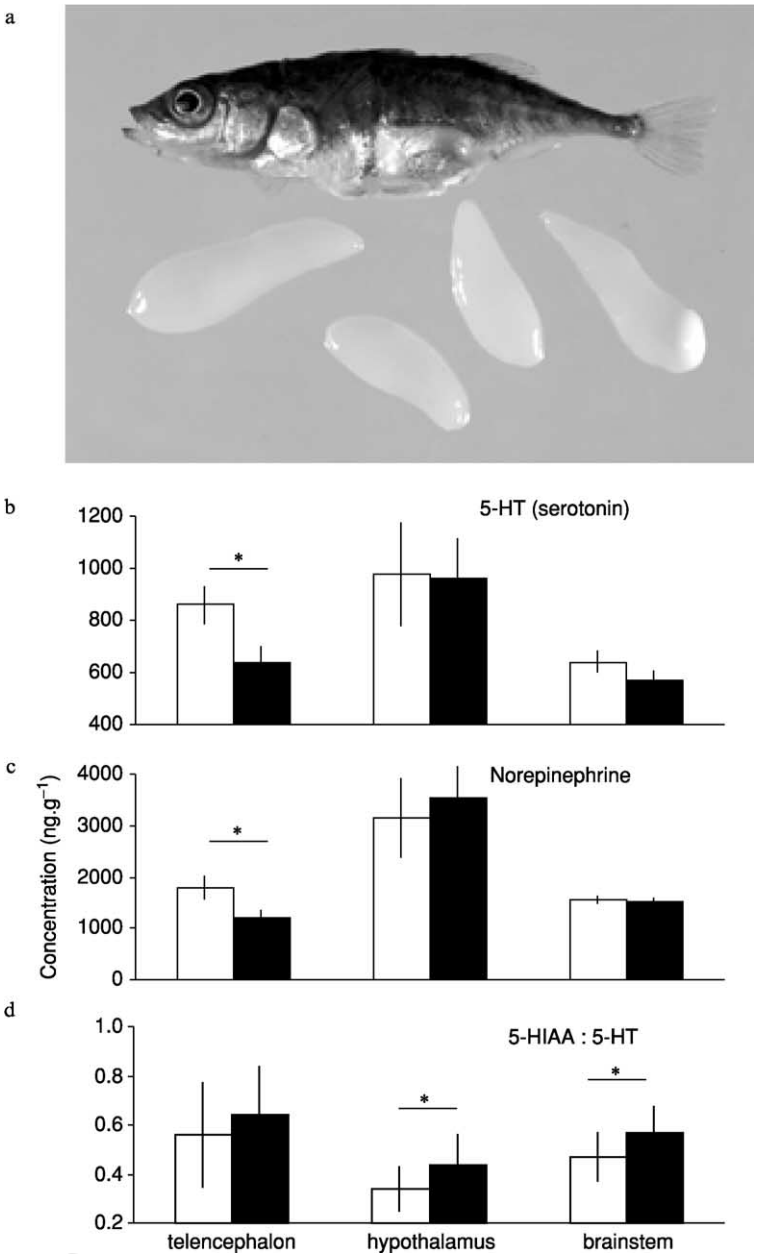

Fig. 4.5. (a) A dissected three-spined stickleback *Gasterosteus aculeatus*, and four plerocercoids of the pseudophyllidean cestode *Schistocephalus solidus* recovered from its body cavity. (b-d) Effects of *S. solidus* infection on the concentration of 5-hydroxytryptamine (5-HT, serotonin),

Section 4.III.A.1; Milinski, 1990; Barber *et al.*, 2000 for reviews). These behaviors closely match the fearlessness shown by parasitized mice and gammarids, and because *Schistocephalus solidus* is transmitted when birds ingest host sticklebacks (Smyth, 1985), it is possible that adaptive host behavioral change in this system is mediated through neurochemical manipulation. Further research is required to separate the potential direct and indirect mechanisms of infection-induced behavioral change in sticklebacks infected with *Schistocephalus solidus*.

D. Endocrinologic Interference with Reproductive Behavior

As well as impacting on host reproduction by reducing the amount of energy available for gonadogenesis (see Section 4.III.A.2), parasites may also interfere directly with the reproductive endocrinology of their fish hosts, with consequences for the reproductive physiology and behavior of infected fish. The tapeworm *Ligula intestinalis* achieves host castration by interfering with the pituitary-gonadal axis of fish hosts (Arme, 1968). Although the precise details of the mechanism are still to be discovered (Arme, 1997), the parasite appears to act at the level of the host's hypothalamus, restricting gonadotropin-releasing hormone (GnRH) secretion. This results in poor gonad development (Williams *et al.*, 1998), which in turn is likely to be responsible for the impaired spawning behavior (Dogiel *et al.*, 1961). With increasing interest in the mechanisms causing the increased levels of endocrine disruption in freshwater fish, particularly resulting from anthropogenic pollution events, the value of further research into the mechanistic basis by which *Ligula* alters host sexual development and behavior is clear (Jobling and Tyler, 2003).

V. PHYSIOLOGICAL EFFECTS OF INFECTION THAT IMPAIR THE HOST'S BEHAVIORAL CAPACITY

A. Cardiovascular Effects of Parasites on Host Swimming Performance

Parasites that inhabit the heart muscle, live in the lumen of blood vessels, or reduce the oxygen-carrying capacity of the blood may reduce the efficiency of the cardiovascular system, with likely consequences for behavior

norepinephrine (NE), and the ratio of 5-hydroxy-indoleacetic acid (5-HIAA) to 5-HT in various brain regions of infected fish. Open bars represent values for control (noninfected) fish and filled bars represent values for infected fish. Bar heights are means; error bars represent 1 standard deviation. $*P < 0.05$. (Data from Øverli *et al.,* 2001).

and stamina of infected fish. Sanguilicolid and heterophyid digenean flukes are understood to be the most pathogenic of the helminths that inhabit the heart and vascular system of fishes (Williams and Jones, 1994). Infections reduce the capacity of the blood to carry and exchange gases by causing mechanical obstruction, by altering the number and type of blood cells, and by causing hemorrhage (Smith, 1972). *Ascocotyle pachycystis*, a heterophyid trematode, locates in and occludes the bulbus arteriosus of sheepshead minnows (*Cyprinodon variegatus*, Cyprinodontidae), physically obstructing blood flow and reducing the time infected fish are able to swim at their maximum sustainable velocity before becoming exhausted (Coleman, 1993). A number of species of philometrid nematodes are also known to occupy the cardiovascular system of the fish hosts, with similar pathological effects.

Anemia is a commonly reported symptom of infection, particularly for fish that are infected with parasites that feed on the blood of their hosts, such as ectoparasitic lice and copepods. Mann (1952, 1953; cited in Grabda, 1991) reports that the haemoglobin levels of whiting infected with the copepod *Lernaeocera* were reduced from 30–40% (normal) to 20–22%. Anemia typically decreases the swimming performance of fish (Jones, 1971; Gallaugher *et al.*, 1995). Wagner *et al.* (2003) demonstrated that sublethal *Lepeophtheirus salmonis* infections caused a reduction in critical swimming speed (U_{crit}) of rainbow trout. Although the behaviorally significant sublethal infections were not associated with anemia in that study, a subsequent study showed that controlled blood loss from noninfected fish could generate similar negative effects on U_{crit} (Wagner and McKinley, 2004), suggesting that as infection levels increase a change in swimming performance would accompany other morbidity effects of infection. Parasitic blood-dwelling haematozoans, including leech-vectored kinetoplastids such as *Trypanoplasma borreli*, may also impact on host swimming behavior as a consequence of their effects on renal function, osmoregulation and hematocrit (Bunnajirakul *et al.*, 2000).

B. Metabolic and Respiratory Effects of Parasites that Affect Habitat Selection

Fish infected with metabolically demanding parasites, such as rapidly growing cestode plerocercoids, may have elevated oxygen requirements with consequences for habitat selection and swimming performance. Lester (1971) demonstrated that *Schistocephalus*-infected three-spined sticklebacks had increased oxygen consumption and as a result selected shallower water habitats, with potential consequences for predation risk. Nine-spined

sticklebacks (*Pungitius pungitius*) infected with the same parasite exhibited an increased frequency of aquatic surface respiration (gasping at the surface), and when exposed to constantly decreasing oxygen tensions in experimental studies were found to have a higher lethal oxygen level (Smith and Kramer, 1987).

Infections may also reduce the level of oxygen available for host respiration if they interfere with ventilation or gill function. Parasites that are ectoparasitic on the gills damage filaments and epithelia of the secondary lamellae as well as causing an increase in host mucous secretion (e.g., Ishimatsu *et al.*, 1996; Dezfuli *et al.*, 2003) and potentially restrict the flow of water over these tissues. Eggs of *Sanguinicola* spp. flukes, described in Section 4.V.A above, block gill capillaries causing thrombosis and necrosis of gill tissue (Smith, 1972). Such changes are likely to impair gas exchange efficiency, with the consequence that infected fish may be unable to withstand low oxygen levels and be forced into seeking oxygen-rich waters.

C. Buoyancy Regulation Effects of Parasites

Teleost fish rely on the swimbladder, which acts as a hydrostatic organ by replacing parts of the fish body with gas to maintain vertical position in the water column, so parasites that impact on swimbladder function and/or inflation might be expected to impair the ability of fish to achieve buoyancy regulation. Largemouth bass (*Micropterus salminoides*) infected with the swimbladder-dwelling nematode *Eustrongylides ignotus* exhibit buoyancy abnormalities (Coyner *et al.*, 2001), as do *Eimeria*-infected haddock (Odense and Logan, 1976). *Anguillicola crassus* is an introduced and economically important swimbladder parasite of European eels *Anguilla anguilla*, which has significant impact on commercial and natural populations (Kirk, 2003). Histological studies show that *Anguillicola* infections cause swimbladder pathology (Nimeth *et al.*, 2000), and infection is associated with a change in the composition of swimbladder gases (Wurtz *et al.*, 1996). Although the consequences of infection for the migratory performance of eels is not yet known, the potential for an effect would appear to be substantial, because *Anguillicola* infection in eels is associated with a reduction in maximum swimming speed (Sprengel and Lüchtenberg, 1991). However, whether this is due to buoyancy effects or to general morbidity is as yet unknown.

Buoyancy control may also be impaired if parasite infections alter the specific gravity of fish hosts. This is most likely for fish infected with the large plerocercoid larvae of pseudophyllidean cestodes, which have a different specific gravity than their fish hosts and can thus potentially interfere with swimbladder inflation (Ness and Foster, 1990; LoBue and Bell, 1993).

D. Effects of Parasites on Swimming Performance

Techniques for quantifying fish swimming performance are well established, and a number of studies have documented quantitative effects of parasite infection. In some cases, it is likely that the effects of the parasites are related to energetic consequences of infection. Smith and Margolis (1970) demonstrated that sockeye salmon smolts infected with adult *Eubothrium salvelini* cestodes, which inhabit the intestine and reduce growth rates (Saksvik *et al.*, 2001), fatigued more quickly than uninfected smolts. Such effects may have consequences for the migratory performance of anadromous fish. Boyce (1979) demonstrated significant impacts of experimentally-induced *Eubothrium salvelini* infections on the swimming performance of sockeye and Smith (1973) found that numbers of infected fish were concentrated towards the end of the run, probably as a result of their impaired swimming ability.

In most cases, there is little information available on the physiological basis of impaired swimming performance, but the location of the parasite may often be informative. Parasites encysting in the musculature, or otherwise impacting on muscle development or function, clearly have the potential to interfere directly with host swimming performance. Metacercariae of the trematode *Nanophyetus salmonicola* encyst throughout the musculature and organ systems of salmonid fish, with effects on burst swimming performance of coho salmon (*Oncorhynchus kisutch*) and steelhead rainbow trout (*Oncorhynchus mykiss*) smolts (Butler and Millemann, 1971). Anisakid nematodes encysted in the musculature of host gadoids secrete metabolic products (alcohol and ketones) which anesthetize host musculature (Ackman and Gjelstad, 1975), and McClelland (1995) speculates that this mechanism is likely to be responsible for the reported slow swimming speed of smelt *Osmerus eperlanus* infected with *Pseudoterranova decipiens* (Sprengel and Lüchtenberg, 1991). Because the definitive hosts of these indirectly transmitted parasites are phocid seals, which acquire the worms after eating infected fish, reduced swimming performance could conceivably enhance transmission.

Parasites that have significant nutritional effects also potentially reduce muscle mass of fish: *Ligula* infections in roach are associated with atrophy of the body wall musculature (Sweeting, 1977), and in common bream *Abramis brama* the same parasite reduces the mass of the body wall muscle (Richards and Arme, 1981).

Changes in the body shape of fish infected with cestodes that grow to a large size in the body cavity or intestine (e.g., Smith, 1973; Barber, 1997; Loot *et al.*, 2002b) are likely to increase the flow resistance and frictional drag of hosts (Rodewald and Foster, 1998) and reduce the body flexibility

necessary for fast starts, as well as being costly in terms of locomotive speed and efficiency (Blake, 1983; Videler, 1993). The ''jerky'' swimming movements characterized by increased lateral amplitude relative to swimming speed reported from bleak (*Alburnus alburnus*, Cyprinidae) infected with *Ligula intestinalis* (Harris and Wheeler, 1974), and the ''sluggish'' movements of common shiners (*Notropis cornutus*; Dence, 1958) probably result from such mechanisms. However, although infection-associated modifications in the swimming movements (kinematics) of host fish are likely to be important mechanisms in determining detection and selection by predators, this area has so far attracted little attention from researchers, and few quantitative studies have been undertaken.

E. Effects of Parasites on Sound Production

The production and detection of underwater sounds is an important component of fish ecology (Chapter 2.VI; Myrberg and Spires, 1980; Hawkins, 1993) and plays a significant role in many aspects of fish behavior including aggressive interaction, courtship, and intraspecific communication. Fish are capable of using three types of mechanisms to produce sound: by stridulation (the grinding or rubbing together of body parts such as fins or pharyngeal teeth), by percussive action (typically involving the swimbladder), and by performing rapid turns (which result in hydrodynamic sounds) (Hawkins, 1993). During all types of sound production, the swimbladder plays a key role in amplifying the noises produced, and so parasites that inhabit the swimbladder have the potential to interfere with the sound production capabilities of host fish.

As discussed in Section 4.V.C, the swimbladder is a common site for parasites and pathological infections may have consequences for sound production. The coccidian protozoan *Eimeria gadi* infects the swimbladders of a number of gadoid species. Odense and Logan (1976) reported that among haddock *Melanogrammus aeglefinus* from Nova Scotia fishing banks, prevalence of *Eimeria* ranged between 4% and 58% and the swimbladders of infected fish were filled with ''a creamy viscous to yellow semisolid material, [consisting] of various parasite stages, fibrous and cellular debris and lipid material.'' The authors speculate that infections would impact sound production. As courtship in haddock and other gadoids is a complex process, requiring subtle posturing and the production of drumming sounds made by the vibrations of specially adapted muscles surrounding the swimbladder (Hawkins and Amorim, 2000; Bremner *et al.*, 2002), it seems likely that they have the potential to impact significantly on spawning success, with potential consequences for the conservation of these threatened species (Rowe and Hutchings, 2003).

VI. FUTURE DIRECTIONS

A. Parasites and the Sensory Ecology of Hosts

With significant developments in our understanding of the physiology of the nonvisual senses of fish including chemosensation, sound perception, and electrosensation, and an improved recognition of the pivotal role such senses play in behavior, the scarcity of studies focused on examining the physiological and behavioral effects of parasites on these systems is surprising. It seems apparent to us that further research on the effects of parasites on both the odor profiles and chemosensation ability of fishes would be highly valuable, and we highlight this as a potentially fruitful area for collaborations between behavioral ecologists, sensory ecologists, and parasitologists.

B. Extending the Range of Systems under Study

As is evident from this review, there has been a bias towards studying the behavioral and physiological consequences of parasite infections in freshwater and anadromous hosts. This bias is understandable given the commercial value of many of these species and the relative ease with which they are kept in the laboratory, compared to marine species. Furthermore, our most detailed knowledge of the physiological basis of behavioral change associated with infection stems from studies into a small number of conveniently studied host–parasite systems, such as the stickleback–*Schistocephalus* system. Although there is considerable value in developing model systems such as this, the fact that parasite infections have such diverse effects on hosts means that the number of model systems developed should be increased. Studies examining behavioral and physiological impacts of parasites of marine species are relatively scarce, and yet ecologists are beginning to realize that parasites can have a major effect on the demography and population dynamics of marine host fish species (Finley and Forrester, 2003). The development of studies investigating behavioral consequences of infection in marine species, and their physiological bases, are therefore to be encouraged.

C. Studying Host Behavioral Change in the Postgenomic Era

Biology is moving into an exciting era, with tools developed as part of large-scale genome sequencing projects, such as transcriptomics, proteomics, and metabolomics, now being routinely used in a large number of molecular biology laboratories. Fish physiologists are beginning to utilize these

technologies to investigate the environmental and ontogenetic control of gene expression and metabolism in their study organisms (Parrington and Coward, 2002). These analytical tools, which allow infection-associated changes in gene regulation, protein expression, and metabolic activity to be qualified and in some cases quantified (Barrett *et al.*, 2000), can be used to investigate physiological aspects of host–parasite interactions in systems for which experimental infection protocols are well-established. By combining the various omic technologies with carefully designed behavioral examination of experimentally-infected fish hosts, it is now possible to track changes in the behavior of parasitized fish and relate these to concurrent changes in the expression of host genes and proteins. These approaches clearly offer a significant opportunity to further our understanding of how parasites, through their physiological effects on hosts, impact on patterns of host behavior.

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VII. SUMMARY

There is an increased interest from fisheries biologists, aquaculturists, evolutionary, and behavioral ecologists in the behavioral changes in fish hosts that are associated with parasite infections. This Chapter introduced the various ways in which parasites may influence the behavior of teleost fishes, focusing particularly on behavioral changes that are induced by parasites following infection. We systematically reviewed each of the major physiological systems of fish (e.g., ionic balance, neurochemistry, endocrine function, and nutritional status), the effects of parasites on them, and examined how infection-associated changes in functioning may impact on normal patterns of host behavior. There are few host–parasite systems for which physiological and behavioral effects of infection have been quantified experimentally, but where possible those studies for which both types of data are available were reviewed. Major gaps in knowledge were also highlighted for further research. This Chapter ended by emphasizing the value of a mechanistic approach for understanding the evolution and likely fitness consequences of infection-associated host behavior modification, and highlighting opportunities to exploit postgenomic technologies to further elucidate the physiological basis of infection-associated changes in host behavior.

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