Overview On The Effects Of Parasites On Fish Health

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Abstract

It is believed by many that parasites are only as important as the fish they infect. Parasites are ubiquitous, primarily surviving in a dynamic equilibrium with their host(s) and they are often overlooked in fish health assessments. Changes in the environment, both anthropogenic and environmental, can alter the parasite/host equilibrium and cause disease or mortality in fish. Therefore it is imperative that we have knowledge of both parasites and parasitic communities within a given population. When fish kills occur, it can often be associated with changes in parasite density and community composition. Often the damage associated with these fish is relative to the rate of infestation with the parasite; a fish that is lightly infected will show few signs of the parasite, while a heavily infected fish may become physiologically impaired and even die. Parasites can cause mechanical damage (fusion of gill lamellae, tissue replacement), physiological damage (cell proliferation, immunomodulation, detrimental behavioral responses, altered growth) and reproductive damage. As parasitism is the most common lifestyle on the planet, understanding its role in the environment may help researchers understand changes in a given fish population or stream ecosystem.

Key words: Fish Health, Parasites, Environment, Community

Introduction

Researching environmental stressors on fish health generally combines investigating emergent health issues associated with genetics, histopathology and pathogens. Many times, the pathogens of interest are those commonly associated with disease (i.e.: viruses, bacteria, nutrition, and pollution). As such, in many fish health assessments, the role of parasites on fish health can be overlooked. Their presence is usually only a concern when they affect a fish species of interest, or cause detrimental effects to the economy or a recreational activity, or a commercial fishery.

Parasitic species can be found everywhere, and on every living organism. Their presence in their host is generally at equilibrium in aquatic organisms and the most common lifestyle on the planet (Marcogliese 2005). Consequently, it is difficult to find any environment or organism that can be labeled as 'pristine' or parasite-free. When researchers describe control sites as being pristine, pathogen or disease-free, they are merely describing the lack of vi-ruses, bacteria and xenobiotics, and are not generally referring to parasites.

There are times when changes in the environment (natural or anthropogenic) can change the state of balance of the parasite between host and nature, thus resulting in disease. These changes can be environmental such as temperature, climate, or anthropogenic such as pollution and urbanization (Lafferty and Kuris 1999). When the dynamic equilibrium between host and parasite is lost, some changes can occur within the host. These changes can cause mechanical damage (fusion of gill lamellae, tissue replacement), physiological damage (cell proliferation, immunomodulation, altered growth, detrimental behavioral re-

sponses,) and/or reproductive damage (Buchman and Lindstrøm 2002, Knudsen et al. 2009, Al-Jahdali and Hassanine 2010).

The roles, functions, and life-styles of parasites help to characterize an ecosystem. Knowledge of parasites and parasitic communities, allows researchers to recognize the role of the fish host in the food web or ecosystem (Marcogliese and Cone 1997, Overstreet 1997, Marcogliese 2005), determine changes in host diet (Campbell et al. 1980, Huxham et al. 1995, Pascual et al. 1996, Knudsen et al. 2004), relationships of host with other organisms (Marcogliese 2005), describe niche changes (Marcogliese 2005), determine the presence of predators or seasonal migrants (George-Nascimento 1987), and determine changes from pollution and climatic stressors (Khan and Thulin 1991, Overstreet 1993, Mackenzie et al. 1995, Marcogleise 2004). This overview is meant to provide a deeper appreciation for the role of parasites in fish health assessments.

Mechanical Damage

Fusion of gill lamellae. Many species of parasites invade the gills of fish. They can range from microscopic tubulinea or monogenea, to macroscopic annelida and arthropoda, and all can be viewed on the gill arches or nestled between the gill filaments. Grossly visible reactions to these parasites on the fish may be noncritical and include a mild discoloration of the gill filaments or one or two white spots. In more critical cases, the fish may display heavy eroding, massive discolorations (often paler), numerous white spots, and increased mucus secretion (Toksen 2007).

Colonization of the gills by parasites often causes proliferative cell changes, including severe epithelial hyperplasia (lamellar gill fusion), hypertrophy, edema, and interlamellar vesicle formation (Adams and Nowalk 2001, Bennett and Bennett 2001, Adams and Nowak 2004, Taylor et al. 2009, Arafa et al. 2009). In the field, this is often recorded as visible white spots, or white mucoid lesions on the gill surface (Taylor et al. 2009). A confirmatory diagnosis cannot generally occur in the field because the presence of microscopic parasites must be confirmed by histological and/or molecular techniques. A method to score gill condition is often used to describe the extent of visible white patches on a scale from negative to heavy infestation (Taylor et al. 2009). These methods are often accurate when there are heavy infestations, but are not accurate with moderate to low infestations when compared to histopathology (Taylor et al. 2009). The degree of lesion development is directly proportional to the infective parasite concentration and progression of the infestation (Morrison et al. 2004, Taylor et al. 2009).

When individual parasites can be seen with the naked eye, an intensity range is often used to describe the infectivity level within the fish host (Siquier and Ostrowski de Núňez 2009). This number is determined by the quantity of parasites that are found per fish. An intensity range is most useful to compare different species from the same site or the same species from multiple sites (Siquier and Ostrowski de Núňez 2009). Positive correlations between fish length or age and parasite intensity have also been reported (Heupel and Bennett 1998). Therefore sampling fish of similar size is important; otherwise results may be skewed with larger fish having more parasites.

Tissue Replacement. Parasite loads in individual fish can often rise to such high numbers that they occupy the majority of the total area of a specific organ. Although

percent tissue replacement is easy to determine, it is one that is frequently ignored in many assessments. Host tissue replacement was determined by Ciordia (1956), when he examined gonad tissue replacement among unidentified species of bivalves by the trematode *Catatropis johnstoni*. Host tissue replacement by trematode sporocysts of *C. johnstoni* located in the gonad of bivalves ranged from 40 to 80% and was believed to be positively correlated with age of infection (Ciordia 1956).

Determination of host tissue replacement by a parasite can aid researchers evaluate the causes of fish stock depletions or decreases in immune responses. Morrison (1984) studied the coccidians *Goussia clupearum* from liver and *Eimeria sardinae* from testis of herring (*Clupea harengus*) caught in waters off Nova Scotia. He found that *G. clupearum* elicited an intense host reaction, infested 85% of mature fish and in heavy infestations, up to 14% of a host's liver tissue was replaced by oocysts. When he looked closely at *E. sardinae*, 90-100% of all fish studied were infested, and in heavy infestations as much as 19% of the testis was replaced by the oocyst. In those fish, spermatic cords were distorted but still distinguishable. Replacement of hepatic tissue by *G. clupearum* stressed fish and replacement of testicular tissue by *E. sardinae* reduced sperm production, thus detrimentally affected herring stocks (Morrison 1984).

Similarly, infection of the coccidian *Goussia lusca* found in the liver of pouting (*Trisopterus luscus*) also caused significant replacement of the host liver parenchyma (Gestal and Azevedo 2006). Known as hepatic coccidiosis, observations of large agglomerates of oocysts with developed granulomas from connective tissue or fibrotic capsules partially replaced the host liver parenchyma. The observations clearly supported previous reports that infection by *Goussia* sp. contributed to a deterioration in host condition due to the loss of functional activity of the liver, which leads to a reduction in total body weight and thus in the market value of infected fish (MacKenzie 1981, Kent and Hedrick 1985, Jendrysek et al. 1994, Abollo et al. 2001, Gestal and Azevedo 2005, Gestel and Azevedo 2006). Knowledge of a parasite's host response may help researchers more quickly identify problems within a fish population, emphasizing that parasites should be considered significant pathogens to fish (Gestel and Azevedo 2006).

Physiological Damage

Cell Proliferation. Proliferation of a sin-gle type of cell can cause detrimental effects in the fish host. This same proliferation of cell types is found in human diseases such as cancer. For example, carcinogene-sis, especially during the initiation and promotion stages, may include interactions between a variety of agents (infectious and chemical). Generalized cell hyperplasia or cellular proliferation, observed in carcinogenesis, is recognized as a causative factor in human liver cancer. Cell proliferation is often caused by the presence of parasites; for example, epithelial cell proliferation is commonly found in Atlantic salmon (Kania et al. 2010) and mucous cell hyperplasia has been found in Atlantic halibut (Otessen et al. 2010). In our lab, parasites are often seen in association with bile duct proliferation in the liver of brown bullheads. Although the relationship between parasites and cancer is rarely studied, these parasites may act as causative agents for carcinogenesis observed in fish species. Although the above statement is speculative, this remains a relatively unresearched topic in fish health.

Amoebic gill disease caused by *Neoparamoeba sp.* in farmed salmon represents

another example of parasite-induced cell proliferaion. This parasite induces the proliferation of epithelial cells and initiates a hyperplastic response that reduces the surface area available for gaseous exchange (Villavedra et al. 2005). The gills of bream (*Abramis brama*) infected with *Ergasilus sieboldi* exhibited hyperplasia and mucus cell proliferation of the respiratory epithelium (Dezfuli et al. 2003). Dezfuli et al. (2003) also found that parasitized primary and secondary lamellae in bream had a higher number of eosinophilic granular cells and rodlet cells. He determined that the increase of inflammatory cells at the site of *E. sieboldi* attachment may be related to an intense host cellular reaction. Rodlet cells represent an inflammatory cell type closely linked to other piscine inflammatory cells (eosinophile granule cells, epithelioid cells, mesothelial cells) (Dezfuli et al. 2003).

Immunomodulation. All parasites have evolved ways to evade the host's immune response and host immune systems have evolved numerous ways to counter these evasive strategies (Sitja-Bobadilla 2008). A trade-off is established that is essential to the survival of the parasite and provokes a state of illness in the host, which is not necessarily lethal (Sitja-Bobadilla 2008). However, when a parasite efficiently evades the host immune system, it may damage the host, but actually reduce damage to the parasite (Sitja-Bobadilla 2008).

Some parasites have evolved strategies that use the host immune system to aid their attachment to the fish host. For example, *Pseudocatylogyrus bini* improves its attachment to the gill structures due to the embedding host reaction, while the mechanisms responsible for this are unknown (Buchmann 2000, Sitja-Bobadilla 2008). *Cyathocephalus trucatus* attaches firmly to the trout pyloric caeca because of the inflammatory reaction that encapsulates its scolex in a spherical structure (Buchmann 2000, Sitja-Bobadilla 2008). Inflammation by the host around the proboscis and bulb of the acanthocephalan *Pomporhynchus laevis* secures a firm attachment, and finally one of the host reactions to the ciliate *Ichthyophthirius multifiliis* is the increased production of mucus which reduces the immune damage to the parasite (Matthews 2005).

Other research on immunomodulation has focused on the deleterious effects of secondary infections that occur in fish hosts already infected with parasitic species. Densmore et al. (2004) studied rainbow trout (*Oncorhynchus mykiss*) co-infected with the bacterial pathogen *Yersinia ruckeri* and the protozoan, *Myxobolus cerebralis*. Densmore et al. (2004) determined that *M. cerebralis* infected fish had lower proliferative lymphocyte responses to four mitogens, displayed greater bactericidal activity of anterior kidney macrophages, and had slightly lower survival and a more rapid onset of mortality. These results can often lead to complicated conclusions that cannot always be inferred to different parasites or pathogens. This is because immunomodulatory changes in parasite infected fish involve both functional enhancement and suppression of different leukocyte populations, disease resistance, secondary pathogens, and the nature of the immune response that the pathogen evokes (Densmore et al. 2004).

Detrimental Behavioral Responses. Although parasites generally do not cause negative impacts to their host, occasionally parasites can develop in such a way as to alter their host's behavior. This usually occurs with parasites that have complex life-cycles, as it may be more difficult for them to go from one host to the next. For example, the behavior of arthropods, the intermediate hosts of acanthocephalan parasites,

may show various changes when infested, including changes in activity, photoreaction, escape behavior, substrate color choice, and vertical distribution (Moore 1984, Poulin 1994, Bakker et al. 1997).

For example, the acanthocephalan parasite *Pomphorhynchus laevis*, has a complex life-cycle (Bakker et al. 1997) where it is transmitted by the crustacean *Gammarus pulex* to the final fish host, the three-spined stickleback (*Gaterosteus aculeatus*). In this case, the orange color of the parasite *P. laevis*, is visible through the transparent cuticle of the crustacean host (Bakker 1997). These more visibly infested orange crustaceans were eaten significantly more often than the uninfested crustaceans (Bakker et al. 1997). Other species of parasites, such as the trematode *Euhaplorchis californiensis*, are known to infest California killifish (*Fundulus parvipinnis*) brains, which alters their behavior and causes the fish to swim slowly and in circles at the water surface (Lafferty 2008). When infected, the fish were 10–30 times more susceptible to predation by the birds that serve as definitive hosts (Lafferty 2008). When a parasite elicits such a behavioral response in the intermediate host (such as the crustacean or fish), it provides a better prospect of completing its life-cycle.

Altered Growth. Altered growth is perhaps the most difficult mechanism to validate effects due to parasitism. In many studies, researchers have determined that altered growth (delayed growth, stunting) only occurs in extreme laboratory conditions, and would not be observed in the wild (Karvonen and Sepälä 2008, Tops et al. 2009). This may be because parasite infested "stunted" fish may not survive in the wild, and they be taken more readily by predation. For the most part, parasites depend on host-derived energy for growth and development, and so they are potentially affected by the host's ability to acquire nutrients under competitive foraging scenarios (Barber 2005). Research by Barber (2005) found that the fastest growing fish developed the largest parasites; therefore, faster growing hosts apparently provide ideal environments for growing parasites.

Reproductive Damage

Parasites often influence their hosts through the diversion of resources either directly by using up energy and nutrients or indirectly by increasing the activity of the immune system (Wedekind 1992, Deerenberg et al. 1997, Candolin and Voigt 2001). Parasites may also change the behavior and food intake of the host (Milinski 1990, Candolin and Voigt 2001). This causes a trade-off between the allocations of limited resources used in reproduction, parasitic infestations, and parasite resistance (Candolin and Voigt 2001). With unlimited resources, there may be no deleterious effect of the parasite on the fish host, whereas the effect of the parasite may increase with a decrease in resource availability (Candolin and Voigt 2001). Deaton (2009) examined the effects of parasitic larval nematodes, *Eustrongylides ignotus*, on male mate choice in the western mosquitofish (*Gambusia affinis*). She found that male mosquitofish preferred to mate with non-parasitized rather than parasitized females, but showed no differences in association time between females (Deaton 2009). The nematode also decreased female body mass and fecundity via reduction in embryos (Deaton 2009). However, when Candolin and Voigt (2001) studied wild courting three-spined stickleback males infected with the tapeworm, *Schistocephalus solidus,* they found that courting stickleback males were less infected than shoaling males. However, in the laboratory both uninfected and in-

fected males built nests and courted females. They also determined that in the field, infected males that did court females expressed less red nuptial coloration than uninfected courting males, but in the laboratory color differences were not detected (Candolin and Voigt 2001). These differences can be explained by the fact that in laboratory conditions, there are no resource limitations to the infected fish and reproduction could occur without negative effects on the parasite (Candolin and Voigt 2001, Deaton 2009).

Conclusion

Researchers need to constantly consider the effects that parasites have on fish health. Parasites affect fish health through mechanical, physical and reproductive damage. These changes can reduce growth, fecundity and survival, change behavior and sexual characteristics, and result in many other maladaptive alterations of the infected host. These changes could have significant consequences at not only the individual level, but population, community and ecosystem levels as well (Bush et al. 2001, Thomas et al. 2005, Shirakashi and El-Matbouli 2009). The use of parasites to discriminate among fish populations inhabiting sites of different environmental quality is conceptually possible (Marcogliese 2005). Research options are not limited, as there are existing criteria for both the selection of appropriate parasites for analysis of fish stocks (Williams et al. 1992, MacKenzie and Abaunza, 1998, Marcogliese 2005), and the selection of hosts and parasites as indicators of pollution and other stressors (Mackenzie et al 1995, Overstreet 1997, Marcogliese 2005).

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