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Behavioural modification in host by helminths

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Abstract
Behaviour-altering parasites are parasites with two or more hosts, capable of causing changes in the behavior of one of their hosts to enhance their transmission, sometimes directly affecting the hosts' decision-making and behavior control mechanisms. Among the behavioral changes caused by parasites is carelessness, making their hosts easier prey. Parasites may alter the host's behavior by infecting the host's central nervous system, or by altering its neurochemical communication. Parasites may alter hosts' behaviors in ways that increase their likelihood of transmission.

Keywords: Behaviour-altering parasites, host, central nervous system, neuro chemicals

Introduction
Over the last 500 million years, parasites across phyla have evolved mechanisms to elude, inhibit and subvert host defence mechanisms (Klein, 2004) [8], suggesting that evolution has selected for parasites capable of manipulating host nervous systems, just as parasites have been selected to manipulate host immune systems (Webster, 2007) [13].

Host Modification
Host modification by a parasite can be defined as any alteration in host phenotype, induced by a parasite, that has fitness benefits for the parasite. Infected host behaves in ways that facilitate the transmission or dispersal of the parasite and therefore the completion of its life cycle (Barnard and Behnke, 1990) [2]

Modification Hypothesis
The idea that a parasite can modify the phenotype of its host, by either taking control of host behaviour or changing the host's appearance may have first appeared in science fiction stories, but is now a well established concept in study of animal behaviour (Poulin, 2010) [11].
From the first empirical demonstrations, amphipods harboring larval acanthocephalan parasites displayed both aberrant behaviour and abnormal coloration making them more susceptible to predation by parasite's next host (Adamec *et al* 1999) [1].

Modification of Behaviour in Flour Beetle
The parasite *Hymenolepis diminuta*, a rat tapeworm, requires the use of an intermediate host, *Tribolium confusum*, the flour beetle as a vector to get the parasite to its definitive host, the rat, *Rattus norvegicus*. The parasite's eggs are released in the feces of the infected rat and eaten by the flour beetle. During eight days from initial ingestion, the parasite will mature to an infective cysticercoid (immature parasite) within the beetles' hemocoel. Once a rat ingests an infected beetle, the cysticercoid will then develop into a mature parasite, thus completing its life cycle. The goal of rat tapeworm is to gain access into its definitive host by way of an intermediate
host. This is accomplished by means of the behavioral modifications of the flour beetle (Hart, 2003) [6].
The rat tapeworm changes the normal behavior of the flour beetle in two ways: by decreasing its activity and by causing it to exhibit behaviors in order for it to avoid concealment. Both of the above behaviors will modify the behavior of flour beetle in order to increase the chances of it to be preyed upon by the definitive host, the rat. This relationship will fulfill two of the parasite's needs; not only does the parasite have a readily available source of food, but it also has a way of reaching its definitive host. Both behaviors are extremely beneficial to the parasite and detrimental to the flour beetle. The hypothesis surrounding this parasite's life cycle alludes to the fact that the parasite needs to increase the probability of its transmission to

the definitive host in order to evolve and propagate its species. (Berdoy, 1994) [3].

Behavioural Modifications in Snails

1. Behavioural modification in *Lymnaea* snail

The schistosome parasite *Tricholbilharzia ocellata* secretes compounds that interfere with the immune function of its snail host, *Lymnaea stagnalis*, allowing the survival of the parasite within the host. Infection also leads to a reduction in egg-laying in the snail, and the energy the host would have invested in reproduction is redirected to support parasitic growth (Gerard, 2001) [5].

Infection by *T. ocellata* results in the host secreting schistosomin, a molluscan cytokine-like molecule. Egg-laying is suppressed in part by the effects of schistosomin on the physiology of the snail's neuroendocrine cells (caudodorsal cells) that prevents them from triggering egg-laying. Although the function of schistosomin in the uninfected snail is unclear, it is thought to depress reproduction during
unfavourable conditions. It appears that the parasite secretes a compound that induces the host to activate this immune–neural connection, leading to benefits for the parasite (Barnard and Behnke, 1990) [2]

2. Behavioural modification in *Succinea* snail

Leucochloridium paradoxum is a trematode of birds. It alters the size, shape and colouration of the tentacles of its snail intermediate host (*Succinea putris)* and causes them to pulsate violently in response to light. These attract the attention of birds to which the parasite must be transmitted, presumably fooloing them into seeing the colourful and pulsating tentacles as potential caterpillar prey (Moore, 2002) [10].

3. Behavioural modification in *Biomphlaria* snail

In *Schistosoma mansoni*, the snail *Biomphalaria glabrata* is the intermediate host. Parasite larvae emerge from snail and actively infect humans. Many studies have been conducted on the behaviour of *B. glabrata* most likely because of the serious pathogenicity of the parasite in the human host. These behavioral studies have been conducted to bring schistosomiosis under control. They concern either snail food preferences or snail–snail attractions (Etges, 2002) [4].

The individual and choice behaviors of the snail *B. glabrata* are altered when it is infected with *S. mansoni*. These alterations are not dependent on the sex of the parasite. When infected with the schistosome parasite, snails that were receptive to stimuli from other snails had longer latency periods before they began their exploratory movements and required longer times to make choices toward their stimulators. Uninfected snails were able to complete the choice trials more frequently than the infected snails. The reduction in the ability of infected snails to complete their trials could be explained by the reduction of their rates of travel found in the individual activity experiments. Decreases in rates of travel in snails have been reported to be related to either morphological and biochemical alterations of the foot or the consequence of energy constraints induced by the parasite (Gerard, 2001) [5].

However, the longer latency periods and the lower choice frequencies in the infected snails cannot be explained by their reduced activity because these parameters are not related to the rate of travel. Indeed, on the basis of their rates of travel, their rest phase, and size of the Y-maze, the infected snails had enough time to complete a choice. An alternative explanation could be a change in chemoreception related to infection that was not tested in our experiments. Etges (2002) [4] has shown that chemosensitivity can be altered i*n B. glabrata* infected with *S. mansoni*. How these snails perceive chemoattractants is in question because the organ responsible for chemoreception remains enigmatic. Berdoy (1994) [3] proposed that the osphradium plays an important role in chemoreception in *B. glabrata*, but Etges (2002) [4] contested this proposal because cauterizing the osphradium did not impair the food finding ability of the snails. However, the orientation mechanisms of the snails can be affected by amputation of both tentacles. Parasitic castration occurs in which the reproductive capacity of snails get lost.

Behavioural modification in Crab

Sacculina carcini is a parasite that starts its simple lifecycle as a free swimming nauplius, which after a few molts will morph into a cypris larva. It is this larva that will find and infect a crab. The female larva will be first to colonize the crab. She is drawn to the crab by scent organs located on her legs and will swim through the water until she lands on the crab's
body. She will then walk up the arm/leg, anchor herself on exposed hairs around a body joint where she will begin to insert herself into the crab's body. She then extends a long root-like filament into the crab and injects a 'blob' made up of a few cells. This process is just another way of molting for the crustaceans, enabling them to grow (Vyas, 2007) [12]. This behavior produces a hard exoskeleton, or husk, that is left behind. In this case, most of parasite is left behind as a husk and the part that lives on looks nothing like a barnacle but more like a slug. The crab parasite will then burrow into the body of the crab, finally occupying the brood pouch of the crab. From this position, the parasite is now able to shoot out rhizoids (root-like filaments) throughout the crab's body, including areas like the eye stalks. The purpose of this behavior is to gain nourishment at the expense of the host. Shockingly, the parasite does not trigger an immune or physical response from the crab. (Adamec *et al* 1999) [1].

Behavioural Modification in Ants

Dicrocoelium dentriticum must be transmitted from an ant to a sheep by accidental ingestion. Most of the cercariae encyst in the haemocoel of the ant and mature into metacercariae, but one moves to the sub- esophageal ganglion (a cluster of nerve cells underneath the esophagus). There, the fluke takes control of the ant's actions by manipulating these nerves. As evening approaches and the air cools, the infected ant is drawn away from other members of the colony and upward to the top of a blade of grass. Once there, it clamps its mandibles onto the top of the blade and stays there until dawn. Afterward, it goes back to its normal activity at the ant colony. If the host ant were to be subjected to the heat of the direct sun, it would die along with the parasite. Night after night, the ant goes back to the top of a blade of grass until a grazing animal comes along and eats the blade, ingesting the ant along with it, thus putting lancet flukes back inside their host (Webster, 2007) [13].

Conclusion

To summarise, the behaviour of the host is altered by parasites to facilitate the transmission or dispersal of the parasite, thereby to complete its life cycle. Apart from the behavioural modification of intermediate hosts, the definitive

hosts are also manipulated in behaviour. Globally, research are being carried out in laboratory animals to study the further behavioural changes/modifications in hosts due to parasites. Behavioural modification may or may not harm the host, but the motto is to facilitate transmission to complete the life cycle of the parasite.

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