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THE DUALITY OF THE PARASITE-HOST NEXUS

BIOLOGY 498WI:
CRITICAL ANALYSIS OF BIOLOGICAL ISSUES WITH DR. AARON REED

ABSTRACT:

The large majority of biotic interactions stem from the concept of the parasite-host relationship. This relationship is immensely diverse and convoluted, ranging from one host and one parasite to multiple hosts and one parasite. The immensity of this interaction can be seen in the fact that parasites inhabit both terrestrial and aquatic organisms. Through the course of many years, parasites have evolved in order to adapt to their host. The evolution of genetics has allowed the parasite to gain the ability to manipulate its environment to increase its fitness. There are several methods that the parasite can employ to attain maximum survival. One such method is behavioral modification in either the intermediate or definitive host. The parasite can also induce forced castration in its host in order to enhance parasite survival and propagation. As result of parasitic load, the host is able to modify its own behavior and sexual reproduction as a means for evasion. The study of these diverse interactions will shed more light on the complexity of parasite-host interactions.

Many different types of biological interactions exist in the natural world. Some of the most thoroughly studied interactions include predation, commensalism, and symbiosis. However, possibly the most derived interaction is that of a parasite and its host. Over the course of millions of years, the parasite-host relationship has led to some amazing and unique behaviors. Some behaviors are controlled by the parasite while others are controlled by the host. The resulting behaviors are as diverse as there are species of hosts and parasites.

Parasite-host interactions can result in host behavioral changes that range from incredibly simple to extremely complex. The simplest of these behavior changes is seen in Bombus impatiens (the common bumble bee), which is infected with the parasite Crithidia bombi. This parasite interferes

with the bee's vision, making it hard to arrive at the correct flower. A far more derived behavior change is seen in cockroaches infested with wasp larvae. As the larva grow up they devour the cockroach from the inside out. In the subsequent paragraphs these behaviors will be extrapolated to shed light on the parasite-host duality.

EVOLUTION OF DUALITY

Chloroplasts and mitochondria can shed light on the evolution of parasites. These cellular organelles have arisen by way of symbiotic relationships between viruses, microbes and higher-order organisms. Chloroplasts and mitochondria share a similarity with parasites in the fact that all of these organisms started their existence as free living and mobile organisms. However, their uniqueness lies in the fact that there must have been other organisms that were exploited by the parasites, which ultimately led to parasite-host interactions.[1] To become a successful species of parasite, the parasite must first find a suitable host. This is naturally accomplished by trial and error; if the host's immune system was too strong, the invader would be killed, however, if the host's immune system was suitable, the invader was able to colonize and create a new species of parasite. Furthermore, genetic components must have preceded this initial relationship to arrive at a successful parasite-host relationship.[2]

Two bits of information have now been identified; parasites were free living before host colonization and genetics plays a role in parasite-host relationships. To fully understand parasitism in an evolutionary context, the evolution of the host must also be taken into account. "The entire life cycle of a parasite with all closely associated organisms, including the host, is a unit in evolutionary development. This evolution of a parasitological system is as important as evolution of individual parasites."[2] With this understanding, the next few paragraphs shed light on the intense interplay that is exhibited between the host and the parasite. These behaviors are possible because both parties have evolved side by side for millions of years. The few behaviors that will be discussed in this paper are only a handful of the actual number of behaviors that are seen in parasite-host communities.

Parasite-host interactions are very complicated in nature but one way to explain the foundation of the interaction is to consider a ball in water. The ball is sitting in a body of water, free from weight, and able to float freely. Now, if a light weight is placed on the ball, it will still be able to remain buoyant. However, as heavier and heavier weight is placed on the

ball, it will cause the ball and subsequently the weight to sink. This situation is analogous to a virulent parasitic infection. As the parasite kills off its host, the parasite will also die but if the parasite is not too virulent the host stays alive and the evolution of parasite-host relations can persist in the population.[3] The parasite-host relationship must remain in equilibrium for the parasite to propagate its own species.

PARASITE-INDUCED BEHAVIORAL MODIFICATIONS AS SEEN IN THE INTERMEDIATE HOST

A little background information must be presented to clarify the proceeding paragraphs; an intermediate host is considered a vector that the parasite uses to arrive at the definitive host. This host is where the parasites are able to sexually reproduce and propagate the species. A common behavioral modification is seen in intermediate hosts that are used as vectors to infect their definitive hosts; these behaviors primarily serve to increase the chances of the intermediate hosts to be preyed upon by the definitive hosts.[4] Behavioral changes are also seen in paratenic (accidental) hosts, where immature parasites wreak havoc on the internal organs instead of maturing in a suitable location. [5] The parasite can elicit behavioral modifications in the host via chemical signals that make the host a better place to raise the parasite's offspring. However, the host can fight back by increasing its opportunity for sexual reproduction.[6] Sexual reproduction can increase fitness and the population's adaption efficiency to different environmental conditions by facilitating genetic diversification of its lineages.[7] Consequently, the parasite responds by decreasing its host's chances of sexual reproduction by forced castration of its host. A castrated host has the ability to provide the parasite with more resources and will be less aggressive.[8 9] As a result of parasite-induced castration, the host will inevitably increase in body size so as to fulfill the needs of the parasite.

Parasites can have a profound effect on the behavior of their chosen host. Three theories persist to explain why parasites modify their hosts' behavior: enhanced transmission effectiveness, accidental or pathological side effects of parasitic infection, and host behavior based on the act of trying to rid itself of the parasite.[10] The concept of enhanced transmission is exemplified in adaptive behavioral modifications of the host by its coevolving parasite.[7] This is likely to be seen in parasites that require different hosts to complete their life cycles. The parasite Hyme-

nolepis 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.[11] 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.

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.[12] 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.[13]

Parasites are generally successful at finding and infecting their target hosts. However, when the parasite is unable to find its correct host, the parasite will alter its behavior in very drastic and irreversible ways. A good example of this infection pattern is seen in the canine roundworm, *Toxocara canis*. This parasite's life cycle is direct in nature and consists of increased tissue migration with the end goal of maturation in the i ntestines.[14] The first phase of the parasite's life cycle consists of unembryonated eggs that are shed in the feces of the definitive host, the canine, which are then embryonated in the soil.[15] Once in the soil, young canines, less than three months, ingest these embryonated eggs, which hatch and penetrate the intestinal mucosa. Eventually these eggs get to the canine's lungs by the way of the bloodstream and liver. The parasite is then coughed up, swallowed, and matured in the small intestine. Conversely, when embryonated eggs are ingested by older canines greater than three months of age, the larvae hatch in the

intestines, penetrate the intestinal mucosa and find their way to the liver, lungs, muscles, connective tissues, kidneys, and many other tissues. These are the sites in the host in which the larvae's development is arrested to a point where the parasite does not harm the host.[16] If the parasitic roundworm is ingested by a paratenic host in the environment as an embryonated organism, it will undergo a somatic migration through bodily organs but fail to reach maturity in the intestines.[17] [18]

A behavioral hallmark of the canine roundworm is that it is able to infect many paratenic hosts, all of which behave in very different ways. One example of a paratenic host for the parasite is the common house mouse, *Mus musculul*. The parasite is unable to complete a full lifecycle in the body cavity of the mouse; this results in the larval forms of the parasite being encysted in the brain of mouse. Heavily infected mice will have impaired brain activity to the extent of extremely retarded levels of activity, exploratory behavior, and aggressiveness. Consequently, it is known that the canine roundworm can infect various types of hosts, which make it unlikely that it could select the same behavior that would have the same repercussions for all of the different types of hosts.[17] Canine roundworm infection does induce behavioral changes in its paratenic host but the observed changes are likely to be a consequence of parasite-induced pathology rather than an adaptive mechanism of parasite-altered host behavior.[17]

PARASITE-INDUCED BEHAVIOR MODIFICATIONS AS SEEN IN THE DEFINITIVE HOST

The above examples solidify the notion that parasites do in fact change the behavior of the intermediate and paratenic hosts, however, they also modify the behavior of the definitive hosts in ways unlike what is seen elsewhere. Parasite modification of definitive host behavior is usually seen in direct systems that involve one host. A few examples that better explain this characteristic is that of *Sacculina carcini*, a crab parasite, *Crithidia beombi*, a bee parasite and the parasitoid Jewel Wasp, *Ampulex compressa*.

Sacculina carcini is of the cirripedia family or barnacle phylogeny and when mature, looks nothing like a classic barnacle. The parasite 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, and an-

chor herself on exposed hairs around a body joint where she will begin to insert herself into the crab's body.[19] 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. 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.[19] 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.[19] 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. In fact, the crab continues to eat and walk as if nothing is out of the ordinary. In other words, the barnacle produces its own 'zombie' so to speak.

Once large enough, the crab parasite will form a brood chamber, equipped with an entrance hole to the outside. If this female is lucky enough, a male will find his way into her. Once the male has found her, he will then molt, and inject himself into her. The male, now able to produce sperm, will venture down a long canal, ending up at the female's visceral sac. The female parasite has two of these canals, allowing her to carry two males for her whole life span. The male will ceaselessly fertilize her eggs, creating thousands of offspring every few weeks. Consequently, the female parasite has created a living food source that will take her wherever she needs to go. In other words the "crab" has been changed into a servant of the parasite, providing the parasite with an endless supply of food, protection, and mobility; the crab even stops molting and growing, funneling all of its energy to the parasite. The crabs are able to escape from predation by severing a claw that regrows later. However, crabs that are infected with *Sacculina* are able to sever a claw but it will not be regrown. Parasitized crabs will also forgo mating to care for the parasite.[20]

As a result of parasitism, *S. carcini*, has castrated the host crab. The reason for this is that the parasite has chosen to colonize the brood pouch of the crab. In doing so, the crab has not lost the ability to nurture. This is exemplified when parasite cypris larvae are ready to come out; the male or female crab finds a rock or hill to which it lifts up its abdomen and begins to spray the parasites into the surf. This behavior has been seen in both males and females and is another great example of the ways in which

parasites can control their hosts. Another striking example of this can be seen in parasitized bumble bees.

Crithidia bombi is a flagellated trypanosome who is the primary parasite of the common bumble bee, *Bombus impatiens.* The lifecycle of this parasite is remarkably simple in relation to the damage it can cause on a single bumble bee and its colony. A host is infected when it comes into contact with feces laden with the parasite cells and once ingested, the cells will attach to the gut wall where they proliferate.[21] This cycle of infection and cell growth will continue until the entire hive has been infected. "Because *B. impatiens* and other bumblebees lack any ability to transfer food among members of the hive, trophallaxis, infectious pathogens like *C. bombi* cannot be transmitted directly but are picked up from surfaces of the nest." [21]

Parasites of many different families are able to alter their host's behavior in drastically different ways and the *B. impatiens* and *C. bombi* system is no different. The parasite is able to induce central nervous system changes that are seen when compared to wild, un-parasitized bumblebees. Parasites like *C. bombi* are able to accomplish this task in two ways; directly, where the parasite is able to physically and chemically destroy central nervous system functioning and indirectly through the action of the host's immune system reaction to infection. The parasite is an example of indirect control, where the host's responses to the parasite are produced by its own immune system. This is accomplished in the fact that parasitized bumblebees show a drastically declined ability to utilize flower assets, which in turn stresses the hive and decreases the abundance of the available floral assets. The parasite is able to stress the body of the bumblebee enough that it is unable to correctly distinguish good flowers from bad flowers. Infected bees are able to acquire floral cues but lose the ability to retain this information.[22] The parasitized bees, in turn, would be responsible for total colony decline to the point of decreased survivorship. In social bees, the reproductive success of the colony is directly related to the acquisition of floral resources by foragers. Given that plant species vary tremendously in the quality of floral rewards offered, bees that are better able to recognize and discriminate profitable species will acquire more resources and increase colony success.[23] Not only would the health of the colony and the individual bees be at risk but the health of the surrounding plants will also be at risk.

Another parasite-host interaction involves parasitoid wasps, more

specifically the Jewel Wasp, *Ampulex compressa,* which hunts *Periplaneta americana*, American cockroaches, which serve as a food source and housing structure for its growing offspring. This behavior modification in the host is: once the parasite finds a suitable host, it will sting the cockroach twice. The first sting is in the thorax and will induce transient paralysis of the legs. The second sting is aimed at the head, which is responsible for certain cognitive behavioral changes. The first notable behavior that the cockroach exhibits is excessive grooming for thirty minutes. After the grooming behavior ceases the cockroach will lose the ability of self-initiated motility. Rendered completely docile, the wasp will guide its host by the antenna and lead it back into the nest that the wasp created. The wasp will then lay one egg on the exoskeleton of the cockroach and then seal the nest off. After two days, the larvae will hatch inside the host and begin to feed for another three days. The larvae will then pupate and eat the cockroach from the inside out, ultimately killing it. After a month, adults emerge ready to reproduce.[24]

The wasp controls its victim by the use of neurotoxins that interfere with the functioning of the central nervous system.[24] The chemistry of the neurotoxin is able work in such a way as to decrease the cockroach's fitness and increase the wasp's fitness. The way in which the wasp has adapted to brood offspring will allow more mating attempts in the natural world due to the fact that immediately after mating has occurred, female wasps can find a suitable host in which to deposit eggs allowing her to go out and breed again. This behavior will increase the females' and males' fitness, while giving the offspring a better chance at life. The parasitic wasp has evolved a pattern of parasitic infection that benefits its own fitness by allowing an optimal place to rear its offspring.

The above examples show that a parasite is not exclusionary, meaning it is not affected by what type of host it uses; rather its focus is on growing and propagating. The notion that only parasites modify the behavior of the intermediate or paratenic hosts has been destroyed. The crab and bee systems were highlighted to shed light on this fact, but there are many more examples that are around us every day. We looked at what the parasite does to the host, but there are also ways in which the host is able to fight back.

RESPONSES TO PARASITE VIRULENCE AS SEEN IN THE HOST

Parasite-host interactions are able to shed light on the hypothesis that certain host behaviors are a result of parasitism; one such behavior is the result of fighting off the parasite. *Chaetodon capistratus*, the four-eye butterfly fish, is the host for an isopod parasite, *Anilocra chaetodontis*, which is a sexual reproducing ectoparasite of marine fishes. The females have a pouch where embryonated eggs hatch and undergo two or more molts to form the manca or pullus II stage. After a short free-swimming life stage, the juvenile larvae are parasitic and need to find a suitable host for each of the separate stages of juvenile molts.[25] Once infected with the parasite the four-eye butterfly fish will begin to move away from the pack and stay motionless in areas of low light or underneath rocks.[25] The energy that butterfly fish would normally expend actively moving about its environment is now shifted into energy to fight off the parasite. This type of behavior is a way in which the host can decrease the spread of the parasite.[26]

SEXUAL REPRODUCTION AS A MEANS TO ELUDE PARASITES

Behavior modifications are just one of the examples of the parasite-host interaction. The examples stated above explain how the parasite controls the host to increase the parasite's fitness and how the behavior of the host tries to thwart the virulence of the parasite. The way in which *C. capistratus* can negatively impact the spread of *A. chaetodontis* is a good behavior in this particular situation to try and ward off the parasite; however, a better behavior is seen where the host can use the act of sexual reproduction to elude parasitic genetic fixation.

The modest explanation is that sexual reproduction, in host and non-host animals, evolved alongside virulent parasites. Sexual reproduction allows for new combinations of genes that are better adapted for life in a parasitic world. Consequently, it has been hypothesized that parasites could be the driving agents in the long-term preservation of sexual reproduction in host populations.[27] The Red Queen hypothesis, proposed in 1973 by evolutionary biologist Leigh Van Valen, attempts to understand the 'give and take' that is seen in parasitic relationships. The Red Queen hypothesis proposes that what one species gains from the interaction the other will lose.[28]

Red Queen dynamics can provide a structural framework for the comprehension of sex, why sex exists, and what benefits it offers. The first question is, why is there sexual reproduction in the first place? While

asexual reproduction is in fact less resource dependent, it does not allow for genetic variation. The Red Queen conundrum, some researchers have argued, may give an evolutionary edge to sex. Asexual strains can never outcompete sexual strains because whenever they get too successful, parasites build up and devastate the strain. Sexual organisms, meanwhile, can avoid these dramatic booms and busts because they can shuffle their genes into new combinations that are harder for parasites to adapt to.[29] A nematode by the name of *Caenorhabditius elegans* provides a practical explanation of the Red Queen and the benefits of sexual reproduction.

The nematode can either be male or hermaphrodite. The hermaphrodites are able to reproduce asexually or seek out a male to reproduce sexually. "Although usually low, outcrossing rates can be genetically manipulated to produce either obligate selfing (asexual reproduction) or obligate outcrossing (sexual reproduction) individuals."[30] Astonishingly, the nematode can be home to many pathogens. The soil pathogen, *Serratia marcescens* "is highly virulent and capable of exerting strong selection on *C. elegans*." [30] Once ingested, the parasite can kill the nematode; however if exposed over a long period of time, the host is able to evolve resistance against the parasite while *S. marcescens* is able to evolve increase virulence. This system is also an example of Red Queen dynamics. In one study, *C. elegans* and *S. marcescens* revealed some astonishing results. The sexual worms that faced co-evolving germs were annihilated in just twenty generations. If the germs could not evolve, however, the asexual worms did fine. The *S. marcescens* that was allowed to co-evolve with the asexual *C. elegans* became much deadlier. The co-evolving sexual *C. elegans*, on the other hand, suffered far lower mortality rates from their germs."[31]

This study shows that the interplay between parasites and hosts is far deeper than behavior modification, but that "the ability of antagonistic co-evolution to continually generate novel environmental conditions under which outcrossing is favored and populations persist when interacting with a virulent pathogen."[32] The overall theme is that sexual reproduction can expedite adaptation to the surrounding environment, but long term sexual reproduction must persist in the population to hold to the Red Queen dynamics.

The life cycle of *Toxoplasma gondii* consists of both sexual and asexual stages. The definitive hosts are member of the cat family, *Felidae*. In this case, *Ratus Norvegicus*, the brown rat, serves as the intermediate host. Members of the cat family will shed unsporulated oocytes in their feces,

where they will sporulate in the environment leading to their infectivity. The brown rat will then ingest infected soil or plant material. Shortly after the oocysts will transform into tachyzoites, which will then encyst in nervous or skeletal muscular tissue forming bradyzoites. Members of *Felidae* will then ingest infected rats, thereby, allowing the parasite to complete its life cycle.[33]

Female brown rats are able to sense that a male has been infected and avoid them in most situations. Aversion behavior seen in females is likely driven by the need to seek out healthy males avoiding sexually transmitted infections.[34] Female aversion behavior to parasitic infection is seen as a negative to the parasite's fitness when transmitted by sexual contact.[34] The parasite is able to manipulate the male brown rat to be more appealing to the female brown rat. Since the parasite has evolved so closely with the brown rat, it is able to manipulate its host to increase its chances of sexual transmission.

RESULTS OF PARASITE-HOST RELATIONSHIP

Another consequence of this interaction is the forced castration and sterilization of the host as achieved by the parasite. Since the growth of the parasite and its reproduction efforts are severely limited by the host resources, these limited resources decrease the fertility of the host and the transmission of the parasite. Reproduction draws precious energy away from the parasite and into the host's offspring; however, if the parasite can keep this from happening, it will gain increased fitness while the host will be at a detriment.[35]

Another hypothesis for parasite castration involves the host's reproductive tissue as a readily available source of high quality nutrients.[35] To better examine parasitic castration the interactions of a plant-ant community is examined. *Crematogaster nigriceps*, an ant, resides in the Acacia tree, *Acacia greggii,* and manipulates the trees to better suit its needs. The Acacia tree serves as shelter for the colony of parasitic ants, providing nutrition and protection from predators. On the other hand, the parasite protects the Acacia tree from consumption and overgrowth by the surrounding vegetation. This relationship is not symbiotic but more closely related to parasitism because the action of the ant sterilizing the Acacia tree is a way of protecting their colony. While the parasite prunes the tree, it inadvertently kills most growing apical meristems. This pruning behavior creates unique canopy architecture and causes the sterilization of the tree.[36] The pruning behavior seen in the parasite increases its fitness while

decreasing the fitness of the Acacia tree. As a result of castration, the host will experience two unique phenomena: gigantism and early-infection fertility compensation occurrence.[37] Gigantism is the physical increase of body mass driven by the parasite by increasing parasitic lifetime reproductive success and it may allow for more resources to be available to the parasite.[38] A gigantic host allows for more room for the parasites to grow to maturity, which is why gigantism is seen in parasitized Acacia trees.

The early-infection fertility compensation occurrence allows for a reproduction race between the host and the parasite. The infected host has only a finite amount of time to reproduce until the parasite castrates the host and uses the remaining host energy to complete its lifecycle. The early-infection fertility compensation occurrence principle could be applied to the *C. nigriceps-A. greggii* relationship. It is advantageous for the parasitic ants to sterilize the Acacia trees as soon as possible to stop new tree branches from growing, which will decrease the amount of territoriality disputes between colonies of ants.

CONCLUSION

The above parasite-host interactions are just a representative sampling of the unique relationships that exist between the parasite and the host. If nothing more, these interactions should shed light on the fact that parasites have a complex interaction with their host species. The examples presented here allude to the fact that the parasite-host relationship is a very dynamic and specialized relationship that formed from a lifetime of interactions between parasites and their hosts. Specialization is the way in which parasites and host have evolved to be members of this interaction, however, it is important for a balance be achieved to allow for successful parasites and successful host defenses.

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