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MINI REVIEW

Success of Infection by Parasites

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Abstract

The success of infection by parasites during the host-parasite coevolution is related to increasing a parasite's ability to alter intermediate host behaviour, resulting in higher fitness of parasites. The "increased host abilities' hypothesis" posits that parasites manipulate the behavior of their intermediate hosts or improves its chances of intermediate host survival in order to enhance their transmission to the next host. Research shows that the transmission of the parasites to their definitive host is facilitated by non-host predator avoidance by the intermediate host, which would otherwise prevent completion of the parasite's life cycle.

Introduction

Parasites consist of approximately 50% of the total species in ecosystems [1]. In aquatic ecosystems their biomass, is greater than the biomass of their predators [2]. Parasites dominate the links in food webs. This means that food webs, which show the flow of energy throughout an ecosystem, include more parasite-host links, e.g., trophic transmission of the parasite from one intermediate host to the definitive host, than predator-prey links [3]. This is true, even though the probability of parasites finishing their life cycle is controlled by several factors, which include intermediate and definitive hosts, as well as non-host predators. Parasites start their development in the intermediate host and can manipulate the physiology and behaviour of their intermediate hosts [4]. Parasites complete their life cycle in the definitive host to which the parasites are transmitted by predation of the intermediate host [4]. At the end of the life cycle, adult parasites typically mate and reproduce inside the definitive host. The resulting eggs or larvae are transmitted via defecation of the definitive host and subsequent infection of the intermediate host [5].

Parasites could act as a digestive food source for non-host predator [6] or as a non-digestive food source for a paratenic host predator-a vertebrate host that comes before the definitive host and does not require a certain developmental stage of the parasite [7]. Digestion by non-host predators means the end of the parasite's life cycle and leads to no fitness for the parasite. The selection pressure on parasites to reach their definitive host predator is higher than reaching a non-host predator, because transmission to suitable definitive hosts is mandatory for the survival of the parasite (life-dinner principle) [8]. The extinction or low fitness of intermediate or definitive hosts or both could lead to alternative mechanisms exhibited by parasites to increase their survival and the completion of their life cycle. For example, parasites have changed their intermediate hosts within the same family over evolutionary history [9],

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expanded the diversity of definitive host predators [10], or increased intermediate hosts abilities upon non-host predators' avoidance behaviour [11] resulting in higher survival of parasites, completion of their life cycle and success in parasite colonization. Parasites have the ability to acquire both novel native and invasive intermediate hosts [12].

Increased Host Abilities' Hypothesis

In "increased host abilities' hypothesis" both the parasite and its intermediate host benefit from increased host ability to survive [11]. This hypothesis is in opposition of the "handicapped host' hypothesis" which emphasizes the handicapping effects of parasite on infected intermediated hosts. According to the "increased host abilities' hypothesis", it is the capability of the parasite to affect the host's phenotype which is the target of natural selection [13]. The multi-dimensionality in parasite-induced changes of the hosts' phenotype has attracted the interest of parasitologists [14]. Multi-dimensional host manipulation denotes the phenomenon when a single parasite alters multiple phenotypic traits of its intermediate host [14]. Changes in the micro-distribution of intermediate hosts are often viewed as a consequence of multi-dimensional host manipulation by the parasite to increase its probability of trophic transmission [15]. Multi-dimensional manipulation does not have to be specific to be adaptive e.g., carotenoid-based coloration of acanthocephalans has no adaptive value in terms of transmission [16]. Also, when predation risk by non-host predator is low, even highly nonspecific manipulation strategies can be adaptive. However, when initial predation risk is high, manipulation needs to be specific to increase parasite transmission success [17].

Conclusion

Previous studies have demonstrated that parasites decrease non-host predator exposure by manipulating the behaviour of their intermediate host[18]. To reach optimal fitness a parasite must keep their infected intermediate hosts in an optimal balance between survival - i.e., protection from non-host predator - and foraging - i.e., seeking food or mate[19,20]. The "increased host abilities" hypothesis posits that parasites have evolved in response to selection pressures on transmission to the next host, i.e., the ability to manipulate the behaviour of the intermediate hosts in such a way that it increases the chance of transmission [11,21]. The resent researches suggested that parasite ability to alter the host's behavior may have evolved to specifically target sympatric host species [22]. And also, same species of parasite comprises morphologically similar but genetically are divergent subspecies [23].

As emphasized at several occasions, natural selection in a host-parasite system may not necessarily target host traits directly, but instead on the ability of parasites to alter hosts traits in a manner enhancing the trophic transmission of the parasite [14,17]. This means that the host -parasite coevolution is directly related to the concept of the "extended phenotype" introduced by Richard Dawkins in 1982 [14, 17]. Evidence in support of new results suggested that modulation of intermediate host behaviour has evolved through 'adaptation' e.g., Acanthocephalans[24] and/or 'exaptation' by nematodes e.g., *Marshallagia marshalli* [25]. This trait can potentially be explained as an exaptation of a parasite manipulating its intermediate host behavior in a manner that places the intermediate host in the vicinity of the definitive host. Hence, parasite evolution may be explained by a shift in function, from regulation of survival in the intermediate host to reproduction in the definitive host.

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