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Beneficial Effects of Parasites on Their Hosts

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Abstract. It is widely known that parasites can compromise a host's health, survival, and reproductive success. However, recent research indicates that parasites can also confer benefits upon their hosts. Centered on the theory of the parasitism-theory continuum, this paper examines three pathways through which parasites affect hosts: modulating the immune system, enhancing disease resistance, and sequestering unwanted nutrients. Furthermore, the paper examines the experimental dependencies inherent in parasitological research and the dynamic nature of host–parasite interactions, providing an objective assessment of these potential benefits and their implications for future research. By systematically discussing the beneficial impacts of parasites across three dimensions - immune system function, disease defense, and resource allocation - and analyzing the mechanisms by which they alleviate inflammatory diseases, detoxify the host, and regulate resource distribution, this paper aims to foster a dialectical understanding of mutually beneficial parasitic relationships and provide a theoretical basis for the development and utilization of parasitic biological resources.

Keywords: parasites, beneficial impact, immune system, ward off disease, allocate resources

1. Introduction

Current academic research largely focuses on the pathogenic damage parasites inflict upon their hosts. While recent discoveries have provided new insights into how parasites shape ecological communities and interact with host immune systems, most studies remain focused on the negative effects of individual parasite species. There is a lack of understanding regarding the positive mechanisms underlying parasite-host symbiotic interactions, and significant gaps remain in integrated research spanning the fields of ecology and medicine. Focusing on the beneficial effects of parasites on their hosts, this paper integrates perspectives from eco-immunology and evolutionary biology to systematically examine the continuum of host–parasite relationships—ranging from detrimental to potentially beneficial—and the mechanisms underlying these transitions. Specifically, it investigates how parasites modulate host immune responses, enhance disease resistance, and influence resource allocation within hosts. Using bibliometric analysis to review recent cutting-edge findings, the study employs multi-factor environmental gradient experiments - controlling for infection by single parasite species - to dynamically monitor changes in host growth, reproduction, and immune investment. This research aims to challenge the prevailing view of parasites as solely detrimental and to offer new insights for ecological conservation and the development of novel

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immunomodulatory agents, ultimately facilitating the establishment of safety standards for low-risk parasitic interventions and the secure practical application of these findings.

2. Re-evaluating the parasite-host relationship

As parasites and their hosts have undergone a long process of co-evolution, some parasites have adapted to evade recognition and elimination by the host immune system, while hosts have developed varying degrees of tolerance to chronic parasitic infections. With rapid technological advancements, an increasing number of studies indicate that parasites can modulate the host immune system.

2.1. Conventional understanding: pathogenic effects of parasites

As a major global public health issue, parasitic diseases pose a serious threat to human health, particularly in impoverished regions [1]. Parasites can harm their hosts through three primary mechanisms [2]: nutrient deprivation, where parasites obtain necessary nutrients from the host and intestinal parasites further impede nutrient absorption; mechanical damage, caused by the migration and colonization of parasites within the host's tissues and organs; and immunopathological damage, resulting from the host's immune response to the parasite's secretions, excretions, shed tegument, and decomposition products - all of which contain abundant antigens capable of triggering immunopathological injury at the site of infection or throughout the host's body. Through these mechanisms, parasitic diseases can severely compromise human health and hinder socioeconomic development; consequently, effective prevention and control efforts can save countless lives [1].

2.2. Interactions along the "parasitism-symbiosis continuum"

Through a long-term process of co-evolutionary selection, certain parasites have adapted to evade recognition and elimination by the host immune system, while hosts have developed varying degrees of tolerance to chronic parasitic infections [3]. With the rapid advancement of second-generation sequencing technologies, increasing research has revealed that the gut microbiota plays a pivotal role in maintaining the balance between host health and disease [4]. Multi-omics data indicate that gut microbiota and intestinal parasites interact to modulate host immune function; acting as "immunological drivers", these interactions offer novel insights for the diagnosis and treatment of diseases associated with gut microbiota dysbiosis [5]. Close and complex interactions exist among parasites, gut microbiota, and the host, with the three establishing a stable survival dynamic as they evolve and shift along the "parasitism-symbiosis continuum" [6]. This article examines the relationships and interactions among the host, parasites, and gut microbiota; analyzes the impact of parasitic infection on gut microbiota diversity; and explores the interplay between parasites and gut microbiota, as well as the consequences of these interactions for host immune-mediated diseases and immune homeostasis.

Interactions between parasites and the gut microbiota can modulate host inflammatory responses and nutrient metabolism, thereby influencing the onset and progression of immune-related diseases such as asthma, colitis, multiple sclerosis, and type 1 diabetes [7]. Co-infection with *Giardia lamblia* and enteroaggregative Escherichia coli (EAEC) can induce protein metabolic disorders in mice; specifically, *G. lamblia* infection disrupts the microbiota-host co-metabolism of protein hydrolysis, and thus, concurrent infection exacerbates host malnutrition [8]. A study using a rabbit model infected with *Trichostrongylus retortaeformis* to examine the diversity and abundance of the

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duodenal microbiota revealed that parasitic infection significantly affects the species composition of the duodenal microbiota and host's immune response [9].

The interaction between parasites and gut microbiota not only contributes to host malnutrition and inflammatory responses but may also exacerbate the progression of certain immune-related diseases [10]. Infection with *Necator americanus* in patients with multiple sclerosis can suppress gut microbiota alpha diversity and modulate immune function by maintaining the stability of the gut microbiota [11]. Excretory-secretory products (ESPs) derived from *Echinococcus granulosus* larvae can ameliorate host gut microbiota dysbiosis induced by a high-fat diet and further alleviate cognitive impairment by reducing inflammatory responses [12].

3. Host immune regulation mediated by parasitic infections

The most notable benefit of parasites lies in their ability to suppress excessive host immune responses, thereby protecting against the onset and progression of autoimmune and allergic diseases. Patients with autoimmune diseases typically exhibit Th1/Th2 polarization imbalance (often skewed toward Th1), while those with allergies show Th2-skewed polarization; as potent immunomodulators, parasites can alter these polarization states to establish an environment of immune homeostasis within the host [13, 14]. Furthermore, parasites—or their excretory–secretory products (ESPs)—can modulate the host's innate and adaptive immunoregulatory networks. Innate regulatory cells involved in this process include alternatively activated macrophages, myeloid-derived suppressor cells, regulatory dendritic cells, and regulatory NK cells, while adaptive regulatory cells comprise regulatory T cells (Tregs) and regulatory B cells [1].

3.1. Therapeutic potential of health therapy for inflammatory bowel disease

Inflammatory bowel disease (IBD) is a group of disorders characterized primarily by chronic, nonspecific intestinal inflammation, comprising ulcerative colitis (UC), Crohn's disease (CD), and unclassified IBD [15]. Certain parasitic infections can activate relevant immune responses and trigger the release of anti-inflammatory factors in the host, thereby helping the intestine resist bacterial infection and alleviate inflammatory reactions [16-18].

Recent research has revealed that helminths and their derivatives can intervene in and treat inflammatory diseases, leading to the proposal of the concept of "heminth therapy" [19]. Although parasite-based therapy remains controversial regarding safety and ethics, a limited number of recent clinical trials have demonstrated that it is relatively safe and well-tolerated. In one study conducted abroad, seven patients with inflammatory bowel disease (IBD) received an initial oral dose of 2500 *Trichuris suis* (pig whipworm) ova every two weeks for 12 weeks, followed by a maintenance regimen of the same dose every three weeks; all patients showed clinical improvement, with no adverse events or abnormal laboratory findings reported [20]. Another trial involving 29 patients with Crohn's disease (CD) administered 2,500 *T. suis* ova orally every three weeks for 24 weeks; nearly 80% of patients experienced clinical improvement, with 72.4% achieving a Crohn's Disease Activity Index (CDAI) score below 150, and no adverse events occurred [21]. These clinical trials demonstrate that the use of helminths and their derivatives holds promise as a safe and effective new therapeutic option for IBD.

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3.2. Regulatory effects of parasite-derived molecules on immune-mediated diseases

Given that parasitic infections modulate immune-mediated diseases primarily through their immunoregulatory effects on the host-mediated by parasite-derived molecules or excretory-secretory products (such as excretory-secretory antigens) - these molecules or products hold promise as novel therapeutic agents to replace live parasitic infections [22-24]. *Schistosoma japonicum* eggs are among the most potent parasite-derived components known to exert immunoregulatory effects on the host immune system during infection. Studies on experimental colitis in mice have demonstrated that *S.japonicum* eggs effectively alleviate intestinal pathological damage; this protective effect is associated with the upregulation of regulatory T cells within the host [25]. *Trichuris suis* (pig whipworm) eggs represent the most successful example to date of using parasitic agents to threat immune-mediated diseases; clinical trials (ranging from Phase I to Phase III) have been conducted for their use in treating food allergies, inflammatory bowel disease, psoriasis, and autism [26].

4. Parasite-mediated remodeling of host metabolism and resource allocation

From a metabolic perspective, parasitic infection compels the host to reallocate limited resources - a shift that my confer a survival advantage under certain conditions. When exposed to parasitic infection, hosts balance avoidance, resistance, and tolerance, with the effectiveness of these strategies heavily dependent on the host's nutritional status and resource availability [27]. In environments where resources are abundant but parasitic pressure is high, hosts may achieve infection tolerance by adjusting metabolic pathways to prioritize the maintenance of critical physiological functions. For instance, a significant correlation exists between the nutrient composition of specific tissues in large fish and their parasite load; while nutrient-rich tissues attract more parasites, they may also drive the evolution of more efficient local immune defenses or tissue repair mechanisms within the host [28].

Further research has revealed that parasites can precisely regulate host amino acid metabolism to meet their own developmental needs. In aphid-parasitoid systems, the parasitoid ensures sufficient nutritional reserves by modulating the host's amino acid metabolic pathways [29]. This tripartite interaction (among the host, parasite, and microbiota) suggests that parasites can act as biological regulators by modulating the host's microbial ecosystem and potentially influencing overall host health.

5. Conclusion

As organisms that have co-evolved with humans, parasites have both beneficial and detrimental effects on human health; while they can act as pathogens that cause disease, the human immune system may have been shaped in part by their immunomodulatory effects during the long process of host-parasite co-evolution. Consequently, parasites are both adversaries and allies to humans. With future technological advancements, a primary objective in parasite research is to systematically identify highly specific immunomodulatory molecules secreted by parasites and to elucidate their molecular targets. A deeper understanding of these biological mechanisms not only reveals the complexities of host-parasite symbiosis shaped by evolution but also offers critical insights for developing novel therapies for autoimmune disorders, metabolic syndrome, and inflammatory diseases.

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