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Human Allergy and Autoimmune Disorders: Parasitic Helminth Infections

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Introduction

The health profile of countries across the world reveal many stark contrasts, including a remarkable reciprocity between helminth parasite infections in most low-income tropical countries, and diseases of modernity such as allergy and autoimmunity in the more affluent, developed populations . In the latter, these syndromes are becoming increasingly prevalent, with asthma exceeding 10% of children in many European countries, while the incidence of autoimmune diseases such as type 1 diabetes as well as of inflammatory bowel disease (IBD) continues to surge. Allergic and autoimmune disorders represent exaggerated immunologic responses to harmless antigens such as those from innocuous environmental organisms or from our own body. The question has arisen of whether parasites dampen the immune system of their host to promote their own survival and while doing so also prevent untoward overreactions that generate immunopathology. Thus, in parasite-free environments the modulating effect of parasitic infections may be lost and the immune system more prone to causing disease—a scenario that Velasquez-Manoff has called an epidemic of absence [1].

Many significant disparities may be found in the health profiles of countries around the world, including a remarkable reciprocity between helminth parasite infections in most low-income tropical countries and contemporary disorders like allergies and autoimmune in more rich, developed societies [2]. These symptoms are growing more common in the latter, with asthma affecting more than 10% of children in many European nations, and the prevalence of autoimmune diseases such as type 1diabetes and inflammatory bowel disease continues to rise. Allergic and autoimmune illnesses are the result of excessive immune reactions to harmless antigens found in the environment or in our own bodies. The debate has emerged as to whether parasites depress their host's immune system to support their own survival while also preventing immunopathology-causing overreactions [3]. Thus, in parasite-free situations, parasitic infections may lose their regulating impact, making the immune system more susceptible to disease—a condition Velasquez-Manoff has dubbed "an epidemic of absence."

The disparity in illness profiles between different parts of the world must be attributed to a wide range of social, nutritional, environmental, and genetic factors [4]. As a result, the question isn't whether helminth parasites alone can explain these differences, but rather whether their effect on the host immune system plays a significant role in muting immunologic illnesses; if so, we'll ask if they can. We can learn from the helminth-driven pathways in order to develop novel medicines to treat the illnesses that are growing more widespread.

Helminths are a diverse group of parasitic and free-living worms with a lengthy evolutionary history; various lines have adopted the parasitic lifestyle and have grown exquisitely well adapted to the immune system of their chosen host over evolutionary time [5]. Their techniques have proven to be extraordinarily effective: over 2 billion people worldwide carry helminth infections now, and until the 19th century, it is possible that all humans would have been infected with one or more helminth species for the majority of their lives. Some of these parasites, such as the schistosomes that cause bilharzia, are extremely hazardous, causing visceral inflammation and liver fibrosis in children, as well as a variety of other negative consequences. In many other helminth infections, however, carriers are present. Indeed, it appears that an early, more robust immune response to infection is suppressed when chronic infection develops in residents who are exposed on a regular basis, and as developing parasites generate eggs or microfilarial larvae for further transmission. Immune responsiveness changes in helminth-infected patients are both quantitative and qualitative, indicating that the parasite is manipulating the host immune system or that the host has reached an agreement with the parasite to reduce collateral damage. In persistently infected filariasis and schistosomiasis patients, antigen-specific T-cell reactivity was found to be decreased, but reactivity may be restored after chemotherapeutic treatment of infection, demonstrating that the presence of helminths actively inhibits host immunity. Infection also changes the profile of immune reactivity, with a skewing of cytokine responses. A shift away from inflammatory mediators like interferon gamma and toward the regulating cytokine interleukin As a result, the pro-inflammatory Th1 and Th17 T-cell subsets are suppressed; more importantly [6], when the Th1/17 population breaks through and dominates the antiparasite response, patients develop more severe immune pathology, such as lymphadenitis and elephantiasis in lymphatic filariasis or granulomatous bladder pathology in Schistosomiasis haematobium.

Parallel to the anti-inflammatory lowering of Th1/17 responses, the host immune response's Th2 arm is also regulated, but more selectively. Part of the Th2 response is preserved, although the profile resembles that of allergic individuals who have undergone allergen desensitisation. The generation of high quantities of the immunoglobulin (Ig) G4 antibody isotype and relatively low levels of IgE, which is mechanistically linked to IL-10's ability to activate T cells, is most significant [7]. This fraction keeps the immune system in a stable state, limiting autoimmune and other potentially harmful reactions to harmless antigens from commensal bacteria. Helminth parasites, on the other hand, appear to have evolved techniques to utilise this pathway in order to avoid the host's immunological evacuation. The regulatory compartment suppresses effector T-cell responses, although they can be regained in vitro by removing the Treg population. Other suppressive populations, such as regulatory B cells, may be activated as well. The effects of reduced inflammatory immunity and increased regulatory activity can be demonstrated in a variety of circumstances;

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for example, children with helminth infections may be less sensitive to microbial vaccinations. The presence of helminths can actually help foreign tissue survive transplantation, and helminth infections can also make it difficult for the host to fight off other pathogens like tuberculosis.

Treg depletion rescues patients' in vitro T-cell responses to bacillus Calmette-Guérin and malaria antigens, which are suppressed relative to non-helminth-infected participants [8]. Each of these findings indicates that helminth parasites have a significant systemic impact on the immune system of the host.

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