Did helminths guard against autoimmune conditions?

Mohsin Abdulhussein Hasan[1],	[1] College of Medicine, University of Al-Ameed, Karbala, PO No: 198, Iraq. Mohsen@alameed.edu.iq
Hujran Abdulraheem Abed[2]	2Department of Medical Laboratory Techniques College of Health and Medical Techniques University of AlKafeel, Najaf, Iraq
Hawraa Hamid Hussein[1],	[1] College of Medicine, University of Al-Ameed, Karbala, PO No: 198, Iraq.
Anas E Almousawi[1],	[1] College of Medicine, University of Al-Ameed, Karbala, PO No: 198, Iraq.
Roaa Noori[1]	[1] College of Medicine, University of Al-Ameed, Karbala, PO No: 198, Iraq.
AliHayder Talib Mahdi[1],	[1] College of Medicine, University of Al-Ameed, Karbala, PO No: 198, Iraq.
Zainab Khaleel Ibrahim1[2]	2Department of Medical Laboratory Techniques College of Health and Medical Techniques University of AlKafeel, Najaf, Iraq

ABSTRACT

Long-lived multicellular parasites known as helminths cause their hosts' immune systems to respond strongly and distinctly. The buildup of parasite loads over time, the infection's chronicity recurrence, and the dominant function of immunity in pathogenesis are typical characteristics of helminth infections

Keywords: Helminths, immune systems

Introduction: Helmint

Long-lived multicellular parasites known as helminths cause their hosts' immune systems to respond strongly and distinctly. The buildup of parasite loads over time, the infection's chronicity recurrence, and the dominant function of immunity in pathogenesis are typical characteristics of helminth infections. It is well known that Th2-polarization predominates in the immune response specific to parasites. (1–3), also showing more and more signs of a T regulatory factor (4–6). The immune system situation produced by parasitic infection significantly influences parallel responses to antigens from other infections, allergens, or auto-antigens, even though the forces that produce Th2 and regulatory T cells (Tregs) have not yet been fully understood. Although the Th2 response is frequently cited as defensive versus helminth parasites, the data is only conclusive for the instance of gastrointestinal worms. (7,8). Protection internal to the tissues could need a further intricate array of type 2 and type 1 cell-mediated defense, comprising granulocytes and activated macrophages. (9,10), because the conventional T helper 2 reaction is crucial for limiting immunopathology (11,12). There may be an inherent recognition mechanism that can

identify these parasites and start the type-2 pathway because Th2 responses to helminths are so common, Nevertheless, no molecular patterns unique to helminths have yet been identified. Alternately, It's possible that some helminth parasites, particularly in tissues, benefit from Th2 responses, and certain organisms have developed the ability to improve this element of the immune response.

Hygiene hypothesis

The hygiene hypothesis, which was developed in 1989, argued that lower infection intensities in infancy might account for the increase of allergic respiratory illnesses and hay fever in adulthood (13). The hypothesis claimed that smaller families, better amenities for the home, and an increase in personal hygiene lowered the likelihood of infections among young families, leading to a more common medical presentation of atopic disorders. With time, this hypothesis has expanded to encompass several chronic inflammatory disorders. In fact, urban migration, better sanitation, and increased availability to clean water have decreased contact with a variety of infectious micro organisms, such as helminths (14). Many epidemiological studies have demonstrated the opposite direction between contact with microorganisms and the emergence of autoimmune disorders (15–16). According to the concept of "old friends," a range of organisms, such as helminths and microbes, along with their mammalian hosts for a very long time. and serve as inducers of immune-regulatory agent (17, 18). This idea makes sense in that pathological agents, particularly helminths, are well-known to be powerful T-cell modulators action and that defect of T-cells subsets (Th17 and Th1) is central to auto-immune disease processes. (19–20) including Multiple Sclerosis (21), Rheumatoid arthritis (22), and psoriasis (23). It is worth noting that there is an opposite relationship between the occurrence of some helminths and the prevalence of autoimmune disorders (24). Among probable environmental causes, the drop in prevalent infectious illnesses has been most striking, as realized by the opposite link between the incidence of infectious microbes and of atopic illnesses. (25–26). Since many years ago, when Strachan brought interest to the impact of size of the family on the prevalence of allergic rhinitis, As a result, the "hygiene hypothesis " has emerged (27). Later, within an immunological framework, this idea was extended, indicating that fewer bacterial and viral infections throughout childhood would hinder the establishment of a Th1 reaction, allowing an excessive Th2 reaction to be generated against aeroallergens. (28).

Autoimmune Disease

Damage to healthy tissue results from autoimmunity, which occurs when the immunity is incapable to discriminate amid infection and self-antigen (29). Today, more than 80 autoimmune diseases have been identified, including inflammatory bowel disease (IBD) (T1D), multiple sclerosis, rheumatoid arthritis, and insulin-dependent diabetes (30). According to current estimates, autoimmune illnesses impact over 10% of the worldwide population, posing enormous health and financial costs (31). The majority of autoimmune diseases are incurable and have no known cures. Uncomfortably, the incidence of autoimmune disease has been steadily increasing in the industrialized world for several decades (32). While it is well established that hereditary predisposition is a major contributor to susceptibility (33), Genetics cannot explain the dramatic increase in these illnesses over such a short period of time; environmental and/or lifestyle factors must also be at play. (34). The "hygiene theory" and the "old friends' hypothesis" are two key hypotheses put out to explain this epidemiology (35).

Immunity and the role of Helminthes

The effects of inhibited inflammatory reactions and increased regulatory activity can be demonstrated in a variation of environments; for example, helminthes infected child may be less sensitive to bacterial and viral vaccinations. [36]; the occurrence of helminths can essentially decrease the chance of transplanted foreign tissue rejection. [37]; and helminth infection weakens the host's capacity

to battle a multiplicity of other diseases, including TB [38]. At least some of this impact is attributable to Tregs: Tregs ablation restores patients' in vitro T-cell responses to malaria and bacillus Calmette-Guérin antigens to those of non-helminth-infected individuals. [39Each of these data points to helminth parasites having a strong systemic influence on the activities of the immune system of the entire infected body. [40]. However, one of the most notable adverse epidemiological effects of helminth infection has been their apparent immunity to immunologic disorders common to inhabitants of affluent environments. [41] The relatively low frequency of rheumatoid arthritis and other auto-immune diseases in African areas with high parasite prevalence suggests that Recently, it was shown that schistosome-infected Gabonese kids had less incidence of atopic skin allergy sensitivity than healthy classmates [42-44] So, the Helminth parasites have a harmful impact on allergic sensitivity [45], However, the magnitude of the defensive consequence is probable to be dependent on the severity and time of infections [46]. Furthermore, in these conditions, the observed reduction is mostly at the level of allergic sensitization as opposed to overt clinical allergy [47].

Does Helminth Burden Regulate Human Autoimmunity?

There is currently sufficient evidence, both during monitoring and following treatment, to suggest a conflicting relationship between chronic helminth infection and overt allergic reactivity in humans (reference [29]). Furthermore, the existing results point to an impact mediated by effector-phase mechanism control. Infected individuals retain their proto-allergic IgE response. Some of the most important discoveries from recent studies are covered here. Van den Biggelaar's seminal work *(38)* Skin-prick test reactions to a conventional allergen in 520 Gabonese kids (5-14 years) were compared to parasitological facts for *S. haematobium* and filarial worm infection. Children with urinary schistosomiasis exhibited less skin sensitivity to home dust mites than those who were not infected. The fact that infected youngsters were substantially sensitive to home dust mite allergens (as measured by typical IgE) yet had decreased overt skin response is important. The negative connection between Skin Prick Test scores and the parasites-specific IL-10 reaction in infected youngsters supports the hypothesis that helminths downregulate the effector phase. Continuing to follow this research group (59), Groups of older people had higher rates of schistosome and filarial infection, as well as rising mite sensitization, and had the least skin-test reaction, according to Van den Biggelaar. Once more, the evidence is in favor of an IL-10–mediated immunomodulatory mechanism that targets the effector phases of an allergic response (60). The presence of Der p 1-specific IgE was unaffected by infection status, as in previous studies, demonstrating that helminth infection does not alter the origin of atopy but rather its translation into asthma.

Conclusion

In light of the growing global burden of autoimmune disease, helminths have piqued scientists' interest due to their capacity to activate immunoregulatory circuits and modify immunity. There is solid proof that helminthic therapy, ES components, and synthetic chemicals derived from helminths can treat and/or prevent inflammatory diseases in rodent models.

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