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Immunological arms race: Co-evolution of hosts and parasites.

We Jeang*

Department of Oncology and Immunology, University of Hopkins, US

Correspondence to: We Jeang, Department of Oncology and Immunology, University of Hopkins, US, E-mail: wejeang09@zzu.edu.cn

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Introduction

The relationship between hosts and parasites represents one of the most dynamic examples of co-evolution in nature. Over millions of years, parasites have developed intricate strategies to invade, survive, and replicate within their hosts, while hosts have concurrently evolved robust immune systems to detect and eliminate them. This constant evolutionary back-and-forth—a molecular tug of war—has shaped immunological landscapes across species, with implications for disease susceptibility, vaccine development, and ecological stability. Co-evolution refers to reciprocal evolutionary changes that occur between interacting species [1].

Parasites exert pressure on hosts to enhance immunity; hosts, in turn, push parasites toward immune evasion mechanisms. Host immune genes (e.g., MHC genes) and parasite surface antigens often display high polymorphism as a result of this evolutionary arms race. While hosts aim to eliminate invaders, some parasites settle for coexistence, manipulating immune responses for survival without killing the host. Hosts have evolved diverse mechanisms to detect and respond to parasitic threats: TLRs, NOD-like receptors, and RIG-I-like receptors sense conserved pathogen molecules, leading to innate immune activation. High polymorphism allows presentation of a broad array of parasite peptides to T cells, enhancing adaptive responses [2].

Evolution of cytokine networks enables finely tuned immune responses suited to different parasitic strategies, such as Th1 against intracellular protozoa and Th2 against helminths. To survive immune onslaught, parasites have evolved sophisticated tactics: Parasites like *Trypanosoma brucei* and *Plasmodium falciparum* cyclically change their surface antigens, evading antibody responses. Helminths release molecules that mimic host cytokines, suppress inflammation, and promote regulatory T-cell expansion [3].

Parasites produce proteins resembling host molecules to avoid detection and dampen immune activation. These strategies allow chronic infections, long-term survival, and transmission to new hosts. Advances in genomics have illuminated molecular footprints of co-evolution: Genes involved in immunity show signs of rapid evolution in host genomes, particularly PRRs and MHC. Gene duplication, epigenetic changes, and recombination aid parasites in developing new virulence traits [4].

Genes directly involved in cell entry (e.g., DARC for *Plasmodium vivax*) evolve under parasite pressure.This dynamic shapes populations, affecting susceptibility and disease outcomes. High parasite pressure can maintain genetic diversity within host populations, especially for immune loci. Parasite adaptation to new hosts may drive speciation or population divergence. Avoidance behaviors (e.g., grooming, habitat selection) evolve under parasitic stress. Targeting conserved parasite antigens can bypass antigenic variation and improve efficacy [5].

Conclusion

Historical parasitic pressures may have shaped immune regulation, influencing autoimmune risks in modern, low-parasite settings. Identifying host alleles under selection can predict disease susceptibility and inform public health strategies. These insights bridge evolutionary biology with clinical immunology. Confers resistance to *P. falciparum*, reflecting host adaptation. Common in West Africa, prevents *P. vivax* invasion. Promotes

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Treg expansion and The dominance, illustrating parasite-driven immune remodeling. As tools like single-cell sequencing and epigenomics advance, new questions arise:

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