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Trained immunity and parasitic infections: Rethinking adaptive boundaries.

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Introduction

These observations highlight the broader health impacts of parasitic exposures and their modulation of innate immunity. The discovery of trained immunity forces a reevaluation of the rigid innate-adaptive dichotomy: Monocytes and macrophages retain functional imprints that affect future responses. The traditional view of the immune system separates innate and adaptive immunity into distinct compartments. While adaptive immunity is antigen-specific and retains memory, innate immunity has long been considered non-specific and short-lived. However, the discovery of trained immunity—the capacity of innate immune cells to exhibit memory-like behavior—has challenged these assumptions. In the context of parasitic infections, trained immunity plays a pivotal role, reshaping the host response beyond classical paradigms and opening new therapeutic possibilities [1, 2].

Mouse models have been pivotal in demonstrating trained immunity: which lack adaptive immunity, show enhanced innate responses after parasitic challenge, supporting the concept of trained innate immunity. Use of chromatin immunoprecipitation sequencing (ChIP-seq) has revealed persistent changes in histone marks following parasite exposure. Trained immunity refers to a functional reprogramming of innate immune cells, such as monocytes, macrophages, and natural killer (NK) cells, after an initial challenge. This epigenetic and metabolic rewiring enables these cells to respond more robustly upon secondary encounters, even to unrelated pathogens. Unlike adaptive immune memory, trained immunity is non-specific but persistent, lasting weeks to months [3, 4].

These findings reinforce the biological relevance of trained immunity and provide tools to study its dynamics. Harnessing trained immunity offers novel avenues: Immunomodulators derived from parasites (e.g., helminth glycans) can be used to train innate cells in autoimmune or inflammatory diseases. Trained monocyte therapies are being explored for cancer, viral infections, and vaccine adjuvant strategies. Parasitic organisms—especially helminths and protozoa—engage the host immune system in chronic and complex interactions. Their life cycles, antigenic variability, and immune evasion strategies make them potent modulators of immune function. Helminths, such as *Schistosoma mansoni*, induce long-term shifts in myeloid cell function, promoting anti-inflammatory states but also training innate cells to respond more efficiently to secondary infections [5, 6].

They actively rewire host immunity in ways that may benefit or harm the host. Incorporating innate training mechanisms could enhance efficacy and coverage. This paradigm shift underscores the complexity of host-parasite interactions and suggests a more integrated model of immunity. Protozoan parasites, including *Leishmania donovani*, prime macrophages via epigenetic changes that enhance cytokine production upon subsequent microbial challenges.These interactions question the traditional boundary that only adaptive immunity can generate long-term protection and memory. Several molecular mechanisms underlie trained immunity in parasitic infections: Changes in histone methylation and acetylation at promoters of genes like IL-6 and TNF-α have been observed after exposure to parasitic antigens [7, 8].

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Despite its promise, trained immunity faces hurdles: Overactivation can lead to chronic inflammation or immunopathology. Understanding how long trained states last and how they're terminated is essential.Parasite species, host genetics, and microbiome composition all influence outcomes. A shift from oxidative phosphorylation to glycolysis known as the Warburg effect is critical for the trained phenotype. Parasites can manipulate macrophage metabolism to sustain this shift. Recognition of parasite-derived molecules through Toll-like receptors (TLRs) and C-type lectin receptors (CLRs) initiates signaling cascades essential for trained immunity. One of the most exciting implications of trained immunity is cross-protection: Exposure to certain parasitic antigens can enhance resistance to unrelated pathogens, including bacteria and viruses [9, 10].

Conclusion

This raises the possibility of designing vaccines or adjuvants that harness trained immunity to provide broader protection, especially in resource-limited settings. Regions endemic with parasitic diseases show interesting correlations: Individuals with helminth infections often exhibit reduced severity of autoimmune and allergic diseases, possibly due to reprogrammed innate responses. BCG vaccination, known to induce trained immunity, also offers protection against malaria and leishmaniasis in some studies.

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