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Innate immunity against helminths: Pattern recognition revisited.

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Introduction

Helminths parasitic worms including nematodes, trematodes, and cestodes—have co-evolved with vertebrate hosts over millennia, developing complex strategies to evade immune detection. Innate immunity serves as the host's first line of defense, relying on pattern recognition receptors (PRRs) to detect conserved helminth-derived structures known as pathogen-associated molecular patterns (PAMPs). Despite their large size and multicellular nature, helminths possess molecular cues that trigger innate immune responses, albeit with outcomes that vary depending on the host environment and worm species. This article revisits the mechanisms of pattern recognition in innate immunity and how hosts navigate the immunological labyrinth presented by helminths [1, 2].

Unlike bacteria or viruses, helminths rarely trigger classical inflammatory PRRs like Toll-like receptor 4 (TLR4) due to their distinct biochemical makeup. However, several PRRs have emerged as central in helminth detection: CLRs such as Dectin-2 and SIGNR1 detect helminth glycoconjugates, including Lewis X structures on *Schistosoma mansoni* eggs, facilitating dendritic cell modulation and type 2 immunity [3, 4].

While helminths lack conventional ligands like lipopolysaccharide, TLR2 and TLR9 can be activated by helminth excretory/secretory products. Helminth-derived components may modulate NLRP3 inflammasome activity, influencing IL-1β and IL-18 production. Helminths strongly drive a type 2 immune response, characterized by the release of IL-4, IL-5, IL-13, and eosinophil recruitment. Pattern recognition alone does not fully explain this bias; instead, tissue damage and alarmins amplify the signal [5, 6].

Emerging data points to helminths inducing trained immunity, where innate cells undergo long-term functional reprogramming: Monocytes from helminth-infected individuals exhibit epigenetic changes that alter cytokine profiles upon re-stimulation. These cytokines are released from epithelial cells upon helminth invasion. IL-33 enhances group 2 innate lymphoid cells (ILC2s), promoting robust type 2 responses. Helminth penetration of mucosal barriers causes necrosis and release of ATP and uric acid, which further alert innate sensors [7, 8].

Activated by IL-5, they release cytotoxic granules such as major basic protein (MBP) to damage helminth cuticles. Recognize worm antigens via IgE-FcεRI engagement and release histamine and proteases, contributing to parasite expulsion. Helminths induce M2 polarization, which paradoxically supports tissue repair and parasite survival—a complex dance of defense and tolerance. One of the most intriguing aspects of helminth biology is their ability to reshape the host's innate immune landscape: Helminths display host-like glycans to engage CLRs without triggering inflammation [9, 10].

References

1. Klevens RM, Luman ET. US children living in and near poverty: risk of vaccine-preventable diseases. Am j prev med. 2001;20(4):41-6.
2. Bardenheier BH, Yusuf HR, Rosenthal J, et al. Factors associated with underimmunization at 3 months of age in four medically underserved areas. Pub heal rep. 2004;119(5):479-85.
3. Findley SE, Irigoyen M, Schulman A. Children on the move and vaccination

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coverage in a low-income, urban Latino population. Am j pub heal. 1999 Nov;89(11):1728-31.
4. Prislin R, Dyer JA, Blakely CH, et al. Immunization status and sociodemographic characteristics: the mediating role of beliefs, attitudes, and perceived control. Am J Pub Healt. 1998;88(12):1821-6.
5. Bataller, L, Kleopa, KA, Wu, GF. Autoimmune limbic encephalitis in 39 patients: immunophenotypes and outcomes. J Neurol, Neurosurg Psych. 2007;78: 381–385.

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