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Impact of environmental change on parasite-induced immune responses.

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Introduction

Environmental change—whether through climate shifts, urbanization, biodiversity loss, or pollution—has profound implications for host–parasite dynamics. Beyond altering geographic distributions and transmission cycles of parasitic diseases, such changes also modulate host immunity. Parasite-induced immune modulation is heavily dependent on host nutritional status—an increasingly unstable factor due to global environmental shifts. The immune responses elicited by parasites are particularly sensitive to environmental variables, making this an urgent frontier in both immunology and global health [1, 2].

Chemical pollutants, including heavy metals, pesticides, and microplastics, can influence the immune system in complex ways: Mercury and lead impair macrophage activity and cytokine profiles. Air pollution is linked to increased susceptibility to respiratory parasites and altered mucosal immunity. Parasitic infections invoke complex immune responses that vary by parasite type and life cycle stage: Helminths (e.g., *Schistosoma*, *Ascaris*) typically induce a Th2-dominant response, leading to eosinophil activation and antibody production. Protozoa (e.g., *Plasmodium*, *Leishmania*) often require Th1-mediated immunity for clearance, characterized by IFN-γ and macrophage activation. Parasite-induced immune modulation can result in either protective immunity or immune evasion and chronic infection. Environmental factors heavily influence this delicate balance [3, 4].

Rising global temperatures, shifting precipitation patterns, and increased humidity can extend the geographic range and seasonal activity of many vectors: Mosquito-borne parasites such as *Plasmodium* and *Wuchereria bancrofti* are now found in regions previously too cool for transmission. Temperature affects parasite development rates and vector immunity, influencing the intensity and frequency of infections. Climate-driven expansion may lead to naïve populations encountering new parasitic pathogens, resulting in exaggerated or ineffective immune responses due to lack of prior exposure or adaptation [5, 6].

Environmental change can drive food insecurity, altering immune readiness: Malnutrition impairs T-cell responses, antibody production, and barrier immunity, intensifying parasitic disease burden. Conversely, diets rich in processed foods may increase systemic inflammation and impair immune homeostasis. Rapid urban expansion brings sanitation challenges, altered ecosystems, and closer human–animal contact: Increased sewage contamination promotes transmission of waterborne parasites like *Entamoeba histolytica*. Crowded living conditions can foster fecal–oral transmission, impacting mucosal immunity. Urbanization also correlates with shifts in diet, microbiome diversity, and pollution exposure, all of which affect immune competence and resilience to parasitic infections [7, 8].

Endocrine-disrupting compounds may interfere with immune cell signaling, compromising parasite clearance. Such exposures not only increase susceptibility to infection but also exacerbate inflammatory responses, leading to more severe clinical manifestations. Environmental degradation and deforestation reduce host and parasite biodiversity: Dilution effect hypothesis suggests

that increased biodiversity can buffer against parasite transmission by disrupting host–parasite specificity. Loss of parasite diversity may reduce immune conditioning, particularly through early-life exposures to benign helminths, which are known to regulate allergic and autoimmune responses. This disruption in "old friends" exposure can skew immune systems toward hyperreactivity, potentially increasing risk of allergies and autoimmune disorders [9, 10].

Conclusion

Despite mounting evidence, gaps remain: Most immune studies focus on short-term laboratory exposures, not long-term environmental stressors. DNA methylation and histone modifications influence cytokine production and lymphocyte proliferation. These changes can be passed transgenerationally, potentially leading to population-level shifts in susceptibility and resistance. Many endemic populations are understudied, making it hard to generalize findings. Integrating ecological immunology, remote sensing, and public health data is essential to forecasting parasite-related immune risks in a changing world.

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