

# [Editorial: regulation of immunity to parasitic infections endemic to africa](https://assignbuster.com/editorial-regulation-of-immunity-to-parasitic-infections-endemic-to-africa/)

[](https://assignbuster.com/)[Health & Medicine](https://assignbuster.com/essay-subjects/health-n-medicine/)

Editorial on the Research Topic   
Regulation of Immunity to Parasitic Infections Endemic to Africa

Parasitic diseases that affect both humans and animals are major causes of morbidity and mortality across the world, and particularly in Africa where they are endemic ( [1](#B1) ). They are often closely related to poverty and with the exception of malaria, are considered neglected because they receive relatively lower treatment and research funding in comparison to HIV/AIDS and tuberculosis ( [2](#B2) ). However, their combined socioeconomic impact in sub-Saharan Africa is comparable to that of tuberculosis and HIV/AIDS ( [3](#B3) ). Parasitic infections can be transmitted across geographical barriers and warrant global attention ( [4](#B4) ). Environmental factors such as humidity and warm temperature promote year-round development of parasites and insect vectors, thereby sustaining transmission. In addition, poor sanitary living conditions and overcrowding promote disease transmission.

A better understanding of parasite biology, pathology, immunology, and parasite-host interactions has resulted in better therapeutics and management strategies that have significantly improved patient outcomes. Unfortunately, many parasites develop resistance ( [5](#B5) ) thereby thwarting the effectiveness of therapeutic strategies. Parasite genomes encode diverse proteins that interact with the host immune system in a dynamic and complex fashion that leads to evasion of protective anti-parasitic mechanisms ( [6](#B6) ). The articles published in this Research Topic provide insights on current advances in research on parasite biology, host immune responses to parasites and novel therapeutic strategies for parasitic infections such as malaria, trypanosomiasis, leishmaniasis, toxoplasmosis, and fascioliasis.

Several original articles in this Research Topic focus on antigen-specific immune responses to the malaria parasite. Aniweh et al. demonstrated that antibodies against the newly characterized *Plasmodium falciparum* merozoite associated protein (PfMAAP) potently prevented the infection of red blood cells by *P. falciparum* merozoites and were associated with a reduced risk of malaria in humans. Kivisi et al. showed that maternal-derived antibodies against variant surface antigens of *P. falciparum* imposed a selection pressure on the parasites and was associated with reduced parasitemia in infants. This latter study supports a potentially new mechanism for maternal-induced immunity against malaria in the early stages of infancy. The transmission of *P. falciparum* from mosquitoes to humans can be significantly reduced by targeting the transmission stage (gametocyte) of the parasite and this has energized the search for transmission blocking vaccine (TBV) candidates. Although significant strides have been made against *P. falciparum* infection, TBV development against *P. vivax* has not been adequately explored.

*P. vivax* is the leading cause of malaria in Latin America. To investigate the potential of TBVs against *P. vivax* , Tentokam et al. analyzed the seroprevalence of antibodies against Pvs230D1M in naturally infected individuals in Brazil and Cambodia. This antigen is located in gametes of *P. vivax* and few polymorphisms have been reported worldwide. They detected similar levels of antibody responses to Pvs230 in these distinct populations from regions with differing transmission intensities, highlighting its potential as a TBV. A separate study focused on the significant role of T-cells in immunity against malaria. Frimpong et al. compared the expression of markers of T-cell inhibition and senescence in healthy children to those with symptomatic or asymptomatic malaria, and have made striking differences observations. Children with symptomatic malaria expressed higher levels of inhibitory and senescent markers on their T cells compared to asymptomatic patients and healthy controls. This suggests that effector T cell function may be impaired in patients with symptomatic malaria and could result in elevated parasitemia.

Parasites belonging to the genus *Trypanosoma* are transmitted by the tsetse fly and cause “ sleeping sickness” in humans and wasting disease in livestock. Although the disease currently affects thousands of people and millions more are at risk, trypanosomiasis in animals is more prevalent and poses serious agricultural and economic problems in affected regions. The production of proinflammatory cytokines by macrophages is essential for resistance. However, trypanosome-induced intracellular signaling pathways that lead to macrophage activation and production of proinflammatory cytokines remain poorly defined.

2. Hotez PJ, Aksoy S, Brindley PJ, Kamhawi S. What constitutes a neglected tropical disease? *PLoS Negl Trop Dis.* (2020) 14: e0008001. doi: 10. 1371/journal. pntd. 0008001

3. Hotez PJ. NTDs V. 2. 0: “ Blue Marble Health”—Neglected Tropical Disease Control and Elimination in a Shifting Health Policy Landscape. *PLoS Negl Trop Dis.* (2013) 7: e2570. doi: 10. 1371/journal. pntd. 0002570

4. Mischlinger J, Rönnberg C, Álvarez-Martínez MJ, Bühler S, Paul M, Schlagenhauf P, et al. Imported malaria in countries where malaria is not endemic: a comparison of semi-immune and nonimmune travelers. *Clin Microbiol Rev.* (2020) : 33: e00104-19. doi: 10. 1128/CMR. 00104-19

5. Vanaerschot M, Huijben S, Van den Broeck F, Dujardin J-C. Drug resistance in vectorborne parasites: multiple actors and scenarios for an evolutionary arms race. *FEMS Microbiol Rev.* (2014) 38: 41–55. doi: 10. 1111/1574-6976. 12032

6. Geiger A, Bossard G, Sereno D, Pissarra J, Lemesre J-L, Vincendeau P, et al. Escaping deleterious immune response in their hosts: lessons from trypanosomatids. *Front Immunol.* (2016) 7: 212. doi: 10. 3389/fimmu. 2016. 00212

7. McFarlane E, Carter KC, McKenzie AN, Kaye PM, Brombacher F, Alexander J. Endogenous IL-13 plays a crucial role in liver granuloma maturation during Leishmania donovani infection, independent of IL-4Rα-responsive macrophages and neutrophils. *J Infect Dis.* (2011) 204: 36–43. doi: 10. 1093/infdis/jir080

8. Stäger S, Alexander J, Carter KC, Brombacher F, Kaye PM. Both interleukin-4 (IL-4) and IL-4 receptor alpha signaling contribute to the development of hepatic granulomas with optimal antileishmanial activity. *Infect Immun.* (2003) 71: 4804–7. doi: 10. 1128/iai. 71. 8. 4804-4807. 2003