Review Article

The Interleukin-12 (IL-12) Family: Pivotal Immunoregulatory Cytokines in Malarial Infection

Khalid Abosalif

ABSTRACT

Objective: To review the Interleukin-12 (IL-12) cytokine family in malaria, focusing on its immunoregulatory roles, contribution to disease severity, and potential clinical relevance.

Place and Duration of Study: The review was conducted at the Department of Clinical Laboratory Sciences, College of Applied Medical Sciences, Jouf University, Sakaka, Saudi Arabia from 1st Nov. 2025 to 30th June 2026.

Methodology: Relevant literature was reviewed using PubMed, Scopus, Web of Science, and Google Scholar, with emphasis on malaria-specific evidence and key studies describing IL-12 family biology.

Results: Malaria induces a tightly regulated inflammatory response in which IL-12 family cytokines help determine parasite control and immunopathology. IL-12 promotes Th1 differentiation and IFN-γ production and is particularly important in early protection. IL-23 supports Th17-associated inflammation, whereas IL-27 has stage-dependent pro- and anti-inflammatory effects. IL-35 is predominantly immunosuppressive and may limit tissue injury while also permitting parasite persistence. The magnitude and timing of these responses vary with parasite burden, host factors, and disease stage.

Conclusion: An extensive, evidence-based synthesis of the structure, immune role, and importance of the IL-12 family in malaria and considers new research on both humans and animals. The understanding of these cytokines may lead to the development of novel immunotherapeutic strategies and improved malaria control.

Key Words: Plasmodium infection, Immunopathology, Immunoregulation, Cytokines

doi:http://doi.org/10.60110/medforum.37.10.31

Citation of Review article: Abosalif K. The Interleukin-12 (IL-12) Family: Pivotal Immunoregulatory Cytokines in Malarial Infection. Med Forum 2026;37(10):160-165. doi:http://doi.org/10.60110/medforum.37.10.31.

This is an Open Access article distributed under the terms of the Creative Commons Attribution License (CC BY 4.0) (https://creativecommons.org/licenses/by/4.0/), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

INTRODUCTION

Malaria remains a major infectious disease caused principally by Plasmodium falciparum and Plasmodium vivax.[1] Protective immunity requires coordinated innate and adaptive responses, yet excessive inflammation contributes to severe manifestations.[2] Cytokines are central to this balance, and the Interleukin-12 (IL-12) family, IL-12, IL-23, IL-27 and IL-35 has emerged as an important regulatory network.4 IL-12 promotes Th1 differentiation and IFN-γ production, whereas IL-23 supports Th17 responses. IL-27 can enhance early Th1 immunity but later restrain inflammation, while IL-35 is largely suppressive.[3]

Malaria therefore provides a setting in which the protective and pathological effects of these cytokines are closely intertwined. Recent studies also emphasize host genetics, parasite-driven immunoregulation, and the need to distinguish findings from experimental models from those in human disease.[4]

Assistant Professor, Department of Clinical Laboratory Sciences, College of Applied Medical Sciences, Jouf University, Sakaka 72388, Saudi Arabia

Correspondence: Khalid Abosalif, Assistant Professor, Department of Clinical Laboratory Sciences, College of Applied Medical Sciences, Jouf University, Sakaka 72388, Saudi Arabia.
Contact No: +966509290730
Email: koabosalif@ju.edu.sa

Received: June, 2026
Reviewed: July-August, 2026
Accepted: September, 2026

METHODOLOGY

IL-12, IL-23, IL-27, and IL-35 are heterodimeric cytokines that share subunits and signaling components but have distinct functions. IL-12 is composed of p35 and p40 and signals through IL-12Rβ1/IL-12Rβ2, activating STAT4 and promoting Th1 differentiation and IFN-γ production.[5] IL-23 shares p40 with IL-12 but combines it with p19 and signals through IL-12Rβ1/IL-23R, mainly activating STAT3 and supporting Th17 responses.[6] IL-27 contains p28 and EBI3 and signals through IL-27Rα/gp130, with STAT1/STAT3 activation underlying both stimulatory and regulatory effects.[7] IL-35 consists of p35 and EBI3 and is produced mainly by regulatory lymphocytes, where it suppresses effector T-cell activity.[8] These shared

structural elements help explain why IL-12 family members can produce overlapping yet context-dependent effects during infection. Their relevance to malaria depends on the stage of infection, cellular source, local inflammatory environment, and balance between parasite elimination and host tissue injury. A further feature of this family is subunit sharing, which creates biological cross-talk between pathways. The shared p40 chain links IL-12 and IL-23 biology, whereas EBI3 contributes to both IL-27 and IL-35.[9] Receptor expression also varies between immune-cell subsets, so the same circulating cytokine concentration may produce different effects depending on which cells are present and activated. This structural organization is important when considering therapeutic blockade because targeting a shared subunit may alter more than one pathway (Fig. 1).

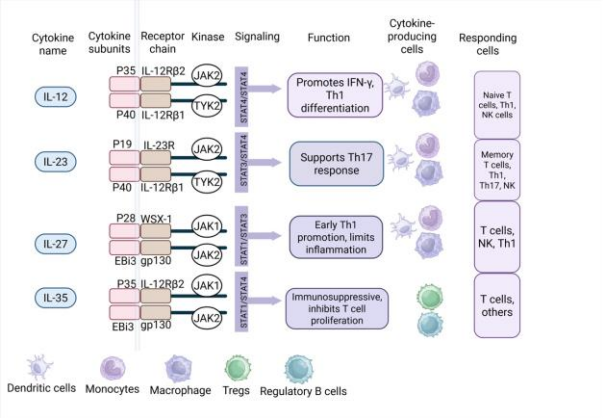


Figure No. 1: Overview of the IL-12 cytokine family, including major subunits, receptors, signaling pathways, cellular sources, and principal immunoregulatory effects

Expression of IL-12 family cytokines is strongly influenced by innate sensing of Plasmodium. Dendritic cells and macrophages respond to parasite-derived signals through pattern-recognition pathways and become important sources of IL-12 and IL-27 during early infection.[10] Plasmodium DNA and other parasite products can activate Toll-like receptor pathways, including TLR9, promoting dendritic-cell activation, NF-κB signaling, and downstream IFN-γ responses.[11] IL-23 is generally linked to maintenance of IL-17-associated inflammation, whereas IL-27 may shift from early immune activation toward later regulation.[12] IL-35 is associated mainly with regulatory T- and B-cell responses and becomes more relevant when immune suppression is established.[8] Parasite-driven IL-10 and other inhibitory mechanisms may reduce effective antigen-presenting-cell function and blunt protective cytokine responses.[5] Thus, the timing and magnitude of cytokine production are as important as absolute concentrations: insufficient early activation may permit parasite expansion, while prolonged inflammatory signaling can amplify tissue damage. Regulation also occurs beyond transcription. Cellular activation state, receptor availability, and negative-feedback pathways can modify the strength and duration of signaling. In malaria, repeated parasite exposure may therefore reshape cytokine responsiveness rather than simply increasing or decreasing cytokine production. This provides a plausible explanation for differences between primary infection, repeated exposure in endemic regions, and severe disease, and highlights why single time-point measurements can be difficult to interpret (Fig. 2).

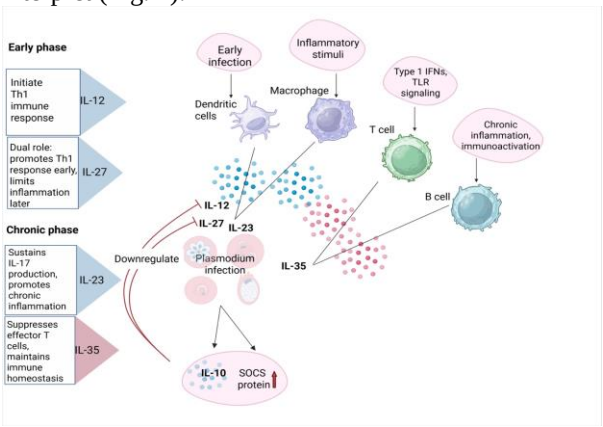


Figure No. 2: Stage-dependent roles of IL-12 family cytokines in malaria: IL-12 and IL-27 support early Th1 responses, IL-23 promotes IL-17-associated inflammation, and IL-35 contributes to immune regulation

During the early liver and blood stages, dendritic cells, macrophages, natural killer cells, and other innate populations rapidly respond to Plasmodium.[13] IL-12 is a key bridge between parasite recognition and cellular immunity because it activates natural killer cells and promotes IFN-γ production, which enhances macrophage function and supports parasite control.[14] Experimental and human studies consistently place the IL-12/IFN-γ axis among the major determinants of an effective early response.[15] IL-23 contributes indirectly by supporting IL-17-producing cells and neutrophil recruitment, although malaria-specific evidence is less extensive and excessive activation may worsen inflammation.[16] IL-27 also has a dual role, it can support early Th1 activity yet induce regulatory pathways that limit damaging inflammation.[10] These opposing effects illustrate a central feature of malaria immunity - strong early activation is desirable only if it is subsequently controlled. Failure to establish this balance may lead either to high parasitemia or to immune-mediated pathology. Innate responses also influence the quality of subsequent T-cell priming.[16]Activated dendritic cells provide cytokine and co-stimulatory signals that determine whether developing responses are predominantly inflammatory, regulatory, or functionally exhausted. In endemic settings, repeated stimulation may alter this balance and

contribute to partial clinical immunity without sterile parasite elimination. Understanding these early events is therefore relevant not only to acute disease but also to the development of naturally acquired immunity (Fig. 3).

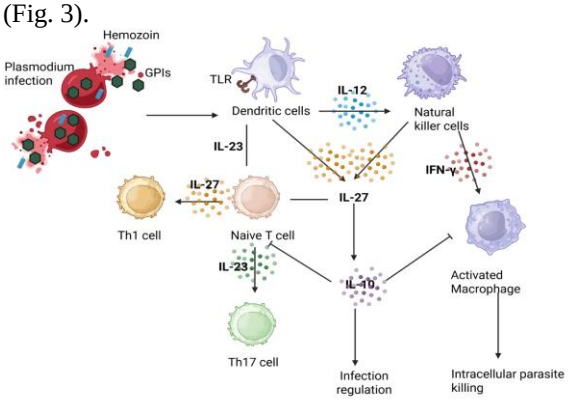


Figure No. 3: Innate sensing of Plasmodium activates dendritic-cell cytokine production. IL-12 promotes NK-cell IFN-γ responses, while IL-23 and IL-27 modulate inflammatory and regulatory pathways

Adaptive immunity is required for parasite clearance, clinical protection, and the development of immunological memory.[17] IL-12 promotes STAT4/T-bet-driven Th1 differentiation and stimulates IFN-γ production by CD4+ and CD8+ T cells, supporting control of liver-stage and blood-stage infection.[18] IL-23 maintains Th17 responses and can influence neutrophil recruitment; however, its contribution appears to vary with the inflammatory environment, and elevated IL-17-related activity has been associated with malaria in human cohorts.[16] IL-27 is again context-dependent, enhancing early Th1 responses but subsequently promoting IL-10-producing regulatory T-cell activity that limits tissue injury.[19]

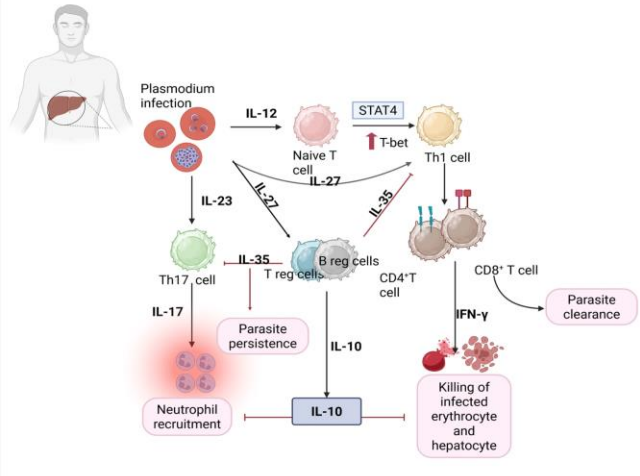


Figure No. 4: IL-12 family cytokines shape adaptive immunity in malaria by regulating Th1, Th17, CD8+ T-cell, and regulatory responses that influence parasite clearance and inflammation

IL-35, produced by regulatory lymphocytes, can suppress Th1 and Th17 responses and may therefore protect against immunopathology at the cost of weaker parasite elimination.[10] The IL-12 family consequently shapes both the strength and durability of adaptive immunity. This balance is also relevant to vaccination, because effective malaria vaccines must generate robust cellular and antibody responses without provoking persistent inflammatory or suppressive pathways.[13] CD8+ T cells are particularly relevant to the liver stage, where recognition of infected hepatocytes can limit establishment of blood-stage infection. At the blood stage, CD4+ T cells provide both inflammatory effector functions and help for B-cell responses. Regulatory pathways become increasingly important as parasite exposure continues, preventing uncontrolled inflammation but potentially reducing the efficiency of clearance. These interacting effects explain why acquired immunity to malaria develops gradually and is often incomplete (Fig. 4).

Clinical malaria ranges from asymptomatic or uncomplicated infection to severe anemia, cerebral malaria, multiorgan dysfunction, and death.[20] Disease outcome reflects both parasite burden and the host inflammatory response. IL-12 is generally associated with protective Th1 immunity and IFN-γ-mediated parasite control, although excessive or prolonged IFN-γ can contribute to pathology.[21] In contrast, heightened IL-23/IL-17 activity may amplify inflammatory injury, and increased IL-17A has been reported in malaria cohorts.[12] IL-27 can be protective when it limits excessive inflammation but may also suppress antiparasitic responses if regulatory signaling is sustained.[19] IL-35 is likely to act in a similar regulatory direction by suppressing effector lymphocytes, although direct malaria-specific evidence remains limited.[8] These observations argue against interpreting any single IL-12 family member as uniformly beneficial or harmful. Rather, cytokine kinetics, ratios, and the phase of infection are likely to be more informative than isolated measurements, particularly when considering biomarkers or therapeutic manipulation. Severe disease also reflects organ-specific consequences of inflammation. Endothelial activation, altered vascular permeability, anemia, metabolic disturbance, and tissue sequestration interact with systemic cytokine responses. Consequently, associations between a cytokine and severity do not necessarily establish causality. Longitudinal measurements before, during, and after treatment are needed to distinguish cytokines that drive pathology from those that rise as compensatory responses to tissue injury or high parasite biomass.

Cerebral malaria is among the most severe complications of P. falciparum infection and results from parasite sequestration together with dysregulated host inflammation in the cerebral microvasculature.[22] IFN-γ is required for effective antiparasitic immunity, but sustained Th1 activation can promote endothelial activation, leukocyte recruitment, and blood-brain barrier dysfunction.[23] IL-12 may therefore contribute to both protection and pathology through its ability to drive IFN-γ responses. IL-23/Th17 signaling is also

biologically plausible as a contributor to neuroinflammation, although direct human evidence is less complete than for the broader inflammatory response.[24] IL-27 may counterbalance these pathways by promoting IL-10 and restraining excessive T-cell activity.[25] Experimental studies additionally demonstrate that modifying inflammatory signaling can reduce neurological pathology without necessarily eliminating parasitemia.[26] Evidence for IL-35 in cerebral malaria is still mainly inferential, based on its established suppressive effects on effector T cells.[8]

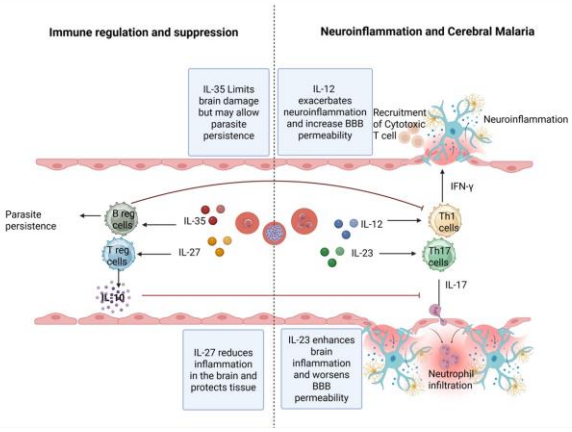


Figure No. 5: Balance between neuroinflammation and immune regulation in cerebral malaria. IL-12/IL-23-associated pathways may enhance inflammation, whereas IL-27 and IL-35 can restrain effector responses

Overall, cerebral malaria illustrates why therapeutic targeting of the IL-12 family would require careful attention to timing: early suppression could impair parasite control, whereas later regulation might reduce tissue injury.[24] The cerebral microenvironment adds another layer of complexity because systemic cytokines interact with endothelial cells, sequestered infected erythrocytes, platelets, monocytes, and brain-resident immune cells. Blood-brain barrier dysfunction is therefore unlikely to reflect a single mediator. A clinically useful model should integrate parasite sequestration with inflammatory and regulatory signals and should recognize that mechanisms identified in experimental cerebral malaria may not reproduce all features of human disease (Fig. 5).

RESULT

Antibody-mediated immunity is essential for control of blood-stage malaria and protection against reinfection,[27] IL-12 can support humoral responses indirectly through Th1-derived IFN-γ and may influence B-cell differentiation and class switching. Human vaccination studies also demonstrate that malaria-specific B-cell responses contribute to protective immunity.[28] IL-23 and IL-27 may modify follicular helper T-cell and B-cell activity, although their malaria-specific roles are less well defined than their effects on T-cell inflammation. IL-35, by contrast, is produced by regulatory B cells and can suppress effector immunity. The key implication is that the IL-12 family can influence antibody quality as well as cellular responses, but strong claims about individual family members should be made cautiously because direct clinical evidence remains limited.[8] Naturally acquired antibody protection depends on repeated exposure and the generation of durable memory B-cell and plasma-cell responses. Cytokines from helper and regulatory T-cell populations can influence germinal-center reactions, antibody affinity, and the persistence of memory (Table 1).

Table No. 1: IL-12 family of cytokines: immunological functions and malaria-related implications

Cytokine	IL-12	IL-23	IL-27	IL-35
Structure/subunits	Heterodimer (p35 + p40)	Heterodimer (p19 + p40)	Heterodimer (EBI3 + p28)	Heterodimer (p35 + EBI3)
Receptor components	IL-12Rβ1 + IL-12Rβ2	IL-12Rβ1 + IL-23R	IL-27Rα + gp130	Unclear; likely IL-12Rβ2/gp130
Primary producers	Induced by Plasmodium-infected antigen-presenting cells	Induced via TLR2/Dectin-1 (e.g, zymosan)	Expressed during the acute and resolution phases	Produced by Tregs upon stimulation
Main targets	CD4+, natural killer, and CD8+ T cells	Th17 cells	Multiple T cell subsets	Th17 and Th1 cells
Signaling pathways	STAT4	STAT3	STAT1/3	STAT1/4
Key immune functions	Stimulates cytotoxicity, Th1 differentiation, and IFNγ production	Maintains Th17 responses	Immunoregulatory stimulates IL-10; inhibits Th17 and Th1	Inhibits effector T cells; induces Tr35
Malaria-related role	Critical to early resistance to Plasmodium; low IL-12 is associated with severe/cerebral malaria	Role of the IL-23/IL-17 axis in malaria-associated anemia and inflammation	IL-27 controls a strong immune balance and memory in malaria; genetic variants affect susceptibility	Putatively implicated in function: negative regulation of the immune system process in cerebral malaria (T cells and B cells regulate inhibitory effects)
References	2,5	6,12,16	7,19,25	3,8

DISCUSSION

Since cytokine profiles vary according to the level of parasite burden and the degree of inflammation, members of the IL-12 family have been suggested as potential adjunct biomarkers. A recent umbrella review has found an association between cytokine levels and the severity of falciparum malaria.[21] IL-12 might indicate protective Th1 activity, while signals associated with IL-23/IL-17 could suggest increased inflammation. Variability in the assays is another obstacle, especially when dealing with low-abundance cytokines and when making comparisons between different laboratories.[29] The measurements of IL-12 family members should be seen as research biomarkers rather than as standalone diagnostic tests. The development of biomarkers could be improved by combining cytokine data with information on parasite density, age, previous exposure, treatment status, and markers of organ dysfunction.[8] Panels that include multiple variables are more likely to reflect the balance between inflammation and immune regulation than a single cytokine.

The IL-12 family does provide a number of possible immunotherapeutic approaches, but none of these is currently suitable for routine use in the treatment of malaria. Increasing IL-12 activity could boost Th1 immunity and promote parasite clearance, while inhibiting excessive IL-23/IL-17 signalling might help to reduce inflammatory damage.[5,6] IL-27 is more difficult to deal with since the same pathway can both enhance early immunity and later suppress inflammation.[7] Blocking IL-35 might in theory help to reinstate effector responses in cases of chronic immune suppression, although this idea is mainly based on evidence obtained outside the context of malaria.[8] The cerebral malaria indicate that careful regulation of inflammatory pathways can lead to an improvement in neuropathology, thus supporting the general idea of host-directed therapy.[23] Nevertheless, there is a considerable risk associated with cytokine manipulation - that it could either impair parasite control or exacerbate immunopathology. Systemic cytokine administration or broad neutralization could adversely affect many tissues, whereas more targeted or short-term approaches could theoretically offer a safer therapeutic window.

Cytokine modulation may also improve malaria vaccines. IL-12 is attractive as an adjuvant because it promotes Th1-biased cellular immunity and can enhance antigen-specific IFN-γ responses.[30] Effective vaccination nevertheless requires balanced cellular and humoral immunity, and excessive inflammatory signaling may be counterproductive.[13] IL-23 could support inflammatory recruitment but has a narrower therapeutic window because of its association with Th17-mediated pathology.[6] IL-27 may enhance early T-cell activation while later regulatory effects could attenuate vaccine responses, and IL-35 may suppress both T- and B-cell function.[8] These possibilities remain largely preclinical. The most realistic strategy is likely to involve transient and precisely timed cytokine modulation rather than sustained activation or blockade. Continued vaccine development must also account for parasite immune evasion and antigenic diversity, which remain major obstacles to durable protection.[31] Vaccine should therefore measure not only peak immunogenicity but also durability, functional antibody activity, T-cell quality, and evidence of excessive regulation. The optimal cytokine profile may differ between pre-erythrocytic vaccines, which rely heavily on cellular responses, and blood-stage vaccines, where antibody-mediated mechanisms are particularly important.

CONCLUSION

The IL-12 family plays a significant role in malaria by linking parasite detection to innate immune activation, T-cell differentiation, inflammation, and immune suppression. While IL-12 typically promotes protective Th1 and IFN-γ responses, IL-23 is more associated with Th17-related inflammation. IL-27 has a clearly context-dependent function, since it helps to initiate early immune activation but also restricts subsequent inflammation, and IL-35 is mainly suppressive. It is clear in severe and cerebral malaria, since the immune responses that are necessary for controlling the parasite can also cause tissue damage. The members of the IL-12 family should continue to be investigated as part of biomarker panels and as possible targets for carefully timed immunomodulation, direct clinical application is still too early to justify.

Conflict of Interest: The study has no conflict of interest to declare by any author.

Source of Funding: None

REFERENCES

1. Weiss DJ, Dzianach PA, Saddler A, et al. Mapping the global prevalence, incidence, and mortality of Plasmodium falciparum and Plasmodium vivax malaria, 2000–22: a spatial and temporal modelling study. Lancet 2025; 405(10483):979-90.
2. Antwi-Baffour S, Mensah BT, Annison L. Overview of the host immune response to P. falciparum malaria. J Parasit Dis 2025;49(3): 548-64.
3. Yi P, Yu W, Xiong Y, Dong Y, et al. IL-35: new target for immunotherapy targeting the tumor microenvironment. Mol Cancer Ther 2024;23(2): 148-58.

4. Segbefia SP, Asandem DA, Amoah LE, et al. Cytokine gene polymorphisms implicated in the pathogenesis of Plasmodium falciparum infection outcome. Front Immunol 2024;15:1285411.
5. Ullrich KA, Schulze LL, Paap EM, et al. Immunology of IL-12: An update on functional activities and implications for disease. Excli J 2020; 19:1563-89.
6. Krueger JG, Eyerich K, Kuchroo VK, et al. IL-23 past, present, and future: a roadmap to advancing IL-23 science and therapy. Front Immunol 2024; 15:1331217.
7. Xu WD, Wang DC, Zhao M, Huang AF. An updated advancement of bifunctional IL-27 in inflammatory autoimmune diseases. Front Immunol 2024;15:1366377.
8. Choi JK, Egwuagu CE. Interleukin 35 regulatory B cells. J Mol Biol 2021;433(1):166607.
9. Prenen F, Van den Steen PE. Malaria-associated liver dysfunction: a forgotten challenge. Trends Parasitology 2025;41(7):547-59.
10. Abosalif K, Atif M, Farooq H, et al. Toll-Like Receptors in Malaria: Gatekeepers of Innate Immunity and Pathogenesis. J Pure Appl Microbiol 2025;19(2).
11. Yao S, Zhao Y, Yao C, et al. TLR9/NF-κB-mediated dendritic cell activation by neutrophil extracellular traps drives pathogenesis in experimental cerebral malaria. J Neuroinflam 2025;22(1):206.
12. Thiam F, Diop G, Coulonges C, et al. An elevated level of interleukin-17A in a Senegalese malaria cohort is associated with rs8193038 IL-17A genetic variant. BMC Infect Dis 2024;24(1):275.
13. Su X, Xu F, Stadler RV, et al. Malaria: Factors affecting disease severity, immune evasion mechanisms, and reversal of immune inhibition to enhance vaccine efficacy. PLoS Pathog 2025;21(1):e1012853.
14. Gbedande K, Ibitokou SA, Endrino MJ, et al. Heightened innate immune state induced by viral vector leads to enhanced response to challenge and prolongs malaria vaccine protection. iScience 2024;27(12):111468.
15. Ibraheem Y, Bayarsaikhan G, Macalinao ML, et al. γδ T cell-mediated activation of cDC1 orchestrates CD4+ Th1 cell priming in malaria. Front Immunol 2024;15(1426316.
16. García-Domínguez M. The role of IL-23 in the development of inflammatory diseases. Biology 2025;14(4):347.
17. Thawornpan P, Kochayoo P, Salsabila ZZ, et al. Development and longevity of naturally acquired antibody and memory B cell responses against Plasmodium vivax infection. PLoS Negl Trop Dis 2024;18(10):e0012600.
18. Zhu C, Jiao S, Xu W. CD8+ Trms against malaria liver-stage: prospects and challenges. Front Immunol 2024;15(1344941.
19. Iwasaki Y, Fujio K, Okamura T, et al, Interleukin-27 in T cell immunity. Int J Mol Sci 2015;16(2): 2851-2863.
20. Li Q, Liu T, Lv K, Liao F, et al. Malaria: past, present, and future. Signal Transduct Target Ther 2025;10(1):188.
21. Naing C, Ni H, Basavaraj AK, et al. Cytokine levels in the severity of falciparum malaria: An umbrella review. Acta Trop 2024;260(107447.
22. Sharma I, Kataria P, Das J. Cerebral malaria pathogenesis: Dissecting the role of CD4+ and CD8+ T-cells as major effectors in disease pathology. Int Rev Immunol 2024;43(5):309-25.
23. Jin Z, Pang W, Zhao Y, et al. Oral administration of IPI549 protects mice from neuropathology and an overwhelming inflammatory response during experimental cerebral malaria. Int J Parasitol Drugs Drug Resist 2024;25(100539.
24. Stins MF, Mtaja A, Mulendele E, et al. Inflammation and elevated osteopontin in plasma and CSF in cerebral malaria compared to plasmodium-negative neurological infections. Int J Mol Sci 2024;25(17):9620.
25. Yazdanpanah E, Pazoki A, Dadfar S, et al. Interleukin-27 and Autoimmune Disorders: A Compressive Review of Immunological Functions. Biomolecules 2024;14(12):1489.
26. Sadler R, Possemiers H, Prenen F, et al. The differential effect of Interferon-gamma on acute kidney injury and parasitemia in experimental malaria. Sci Rep 2025;15(1):6402.
27. Lu F, Xu J, Liu Y, et al. Plasmodium vivax serological exposure markers: PvMSP1-42-induced humoral and memory B-cell response generates long-lived antibodies. PLoS Pathog 2024;20(6):e1012334.
28. Elias SC, Choudhary P, de Cassan SC, et al. Analysis of human B-cell responses following ChAd63-MVA MSP1 and AMA1 immunization and controlled malaria infection. Immunology 2014;141(4):628-644.
29. Knight V, Long T, Meng QH, et al. Variability in the Laboratory Measurement of Cytokines. Arch Pathol Lab Med 2020;144(10):1230-33.
30. Pirahmadi S, Zargar M, Pourhashem Z, et al. Selection of combination adjuvants for enhanced immunogenicity of a recombinant CelTOS vaccine against Plasmodium falciparum. Biochem Biophys Res Commun 2025;748(151310.
31. Stanisic DI, Barry AE, Good MF. Escaping the immune system: How the malaria parasite makes vaccine development a challenge. Trends Parasitol 2013;29(12):612-22.