STM JOURNALS

Recent Trends in
Infectious Diseases

ISSN: 3048-9857
Volume 3, Issue 1, 2026
January—June
DOI (Journals): 10.37591/RTID

https://journals.stmjournals.com/rtid

Review RTID

The Parasitic Fingerprint: Tracing Infection to Host Illness

Shubham Sah[1,*], Deepa Kumari[2]

Abstract

Parasitic infections remain a major global health burden, causing significant morbidity and mortality, particularly in tropical and subtropical regions. A critical aspect of disease pathogenesis is the unique molecular and cellular imprint left by parasites in their hosts, termed the "parasitic fingerprint." These fingerprints encompass parasite-derived proteins, nucleic acids, metabolites, extracellular vesicles, and host responses, including gene expression changes, cytokine profiles, and metabolic adaptations. Advances in multi-omics technologies, which integrate genomics, transcriptomics, proteomics, metabolomics, and epigenomics, have enabled high-resolution mapping of host–parasite interactions, revealing strategies parasites employ to invade, survive, and manipulate host systems. Molecular fingerprints offer clinical relevance by providing sensitive diagnostic markers, informing disease prognosis, guiding vaccine design, and enabling targeted therapeutic interventions. Case studies in malaria, leishmaniasis, trypanosomiasis, and helminth infections demonstrate the utility of fingerprints in linking parasite activity to tissue-specific pathology and host immune modulation. Integration of these insights into emerging technologies, such as single-cell multi-omics, spatial transcriptomics, and artificial intelligence, holds promise for personalized medicine approaches, improving disease management and reducing the global burden of parasitic diseases.

Keywords: Parasitic fingerprint, host-parasite interactions, multi-omics, diagnostics, therapeutics, malaria, leishmaniasis, trypanosomiasis, helminths, precision medicine

INTRODUCTION

Parasitic diseases remain a major global health challenge, affecting millions of people and causing substantial morbidity and mortality, particularly in tropical and subtropical regions. Parasites, including protozoa, helminths, and ectoparasites, have evolved complex life cycles and sophisticated strategies to invade, survive, and propagate within their hosts [1]. These infections are often chronic, leading to long-term complications that affect organs, tissues, and the host immune system. Despite advances in therapeutics and control measures, parasitic diseases continue to impose a heavy clinical and socioeconomic burden, particularly in resource-limited settings. A fundamental aspect of parasitic disease pathogenesis is the molecular and cellular imprint that parasites leave on their hosts, which can be described as the "parasitic fingerprint." This concept encompasses the unique biochemical, proteomic, genomic, and immunological signatures generated during infection, which not only facilitate parasite survival but also influence disease severity and clinical outcomes. Understanding these fingerprints is crucial for elucidating the mechanisms of disease progression, identifying potential biomarkers, and developing targeted therapeutic strategies [2]. Host–parasite interactions are dynamic and multifaceted. Upon infection, parasites encounter the host immune system and other cellular defences, triggering a cascade of

***Author for Correspondence**
Shubham Sah
E-mail: shubham24@iisertvm.ac.in

[1]Student, Department of Biological Science, Indian Institute of Science Education and Research, Thiruvananthapuram, Kerala, India
[2]Student, Department of Biochemistry, Central University of Haryana, Mahendergarh, Haryana, India

Received Date: February 04, 2026
Accepted Date: February 05, 2026
Published Date: February 20, 2026

Citation: Shubham Sah, Deepa Kumari. The Parasitic Fingerprint: Tracing Infection to Host Illness. Recent Trends in Infectious Diseases. 2026; 3(1): 29–50p.

molecular events. While hosts mount innate and adaptive immune responses to eliminate or control the invading parasite, parasites simultaneously deploy evasion strategies, such as antigenic variation, molecular mimicry, and immune modulation, to avoid clearance [3]. These interactions leave discernible molecular traces in host tissues, blood, and other biological fluids, forming the basis of the parasitic fingerprint. Recent advances in multi-omics technologies, including genomics, transcriptomics, proteomics, and metabolomics, have enabled high-resolution mapping of host-parasite interactions, providing unprecedented insight into disease mechanisms. Furthermore, the integration of molecular fingerprints with clinical phenotypes offers new opportunities for developing sensitive diagnostics, predictive biomarkers, and precision therapeutics. This review comprehensively explores the concept of the parasitic fingerprint, tracing the journey from infection to host illness [4]. The molecular signatures generated during parasitic infections, the mechanisms underlying host pathology, and the implications for diagnostics, therapeutics, and disease control are examined. By bridging the gap between parasite biology and clinical outcomes, this synthesis provides a framework for understanding the complex interplay between parasites and their hosts and highlights future directions for research and translational applications [5].

HOST-PARASITE INTERACTIONS

Understanding host–parasite interactions is central to tracing the molecular and pathological fingerprints that define parasitic diseases. These interactions are dynamic, multifaceted, and occur across molecular, cellular, and systemic levels. Parasites must overcome numerous host defences to establish infection, while hosts employ innate and adaptive mechanisms to control or eliminate the invader. The outcome of this evolutionary arms race shapes the clinical manifestations, tissue pathology, and long-term consequences of parasitic infections [6].

Entry and Invasion

Parasites utilize diverse strategies to enter the host, dictated by their life cycle and ecological niche. Protozoan parasites, such as *Plasmodium spp.*, *Leishmania spp.*, and *Trypanosoma spp.*, often enter through vectors or contaminated fluids, whereas helminths may penetrate the skin or gastrointestinal tract. Successful invasion depends on the parasite's ability to recognize host receptors and adhere to specific cell types. For example, *Plasmodium falciparum* merozoites exploit erythrocyte surface receptors, including glycophorin A, to invade red blood cells, while *Leishmania* promastigotes interact with macrophage receptors, such as complement receptor 3 (CR3) and mannose receptor, to gain intracellular entry. Parasites secrete specialized effector proteins and enzymes that facilitate penetration of host barriers. Proteases, lipases, and adhesion molecules remodel host membranes and extracellular matrices, promoting parasite entry and dissemination. Some parasites induce endocytosis or phagocytosis, while others actively traverse cell junctions. This precise orchestration of molecular mechanisms leaves identifiable footprints in host tissues, forming the basis of early parasitic fingerprints (Figure 1) [7–10].

Host Immune Recognition

The host immune system is the first line of defence against parasitic invasion. Innate immunity involves physical barriers, complement activation, phagocytic cells, and pattern recognition receptors (PRRs) that detect pathogen-associated molecular patterns (PAMPs). Toll-like receptors (TLRs), C-type lectin receptors, and nucleotide-binding oligomerization domain (NOD)-like receptors play key roles in sensing parasites and initiating inflammatory responses [11, 12]. For instance, *Trypanosoma cruzi* glycosylphosphatidylinositol anchors activate TLR2 and TLR4 pathways, triggering cytokine release and oxidative responses. Adaptive immunity provides specificity and memory through T and B lymphocytes. CD4+ T helper cells orchestrate immune responses by differentiating into Th1, Th2, or Th17 subsets, each promoting distinct effector mechanisms [13]. Th1-mediated interferon–gamma production is crucial for controlling intracellular parasites, such as *Leishmania*, while Th2 responses drive eosinophilia and antibody-mediated immunity against helminths. B cells produce parasite-specific antibodies that neutralize antigens, opsonize pathogens, or activate complement pathways. These immune interactions generate detectable molecular patterns that contribute to the parasitic fingerprint.

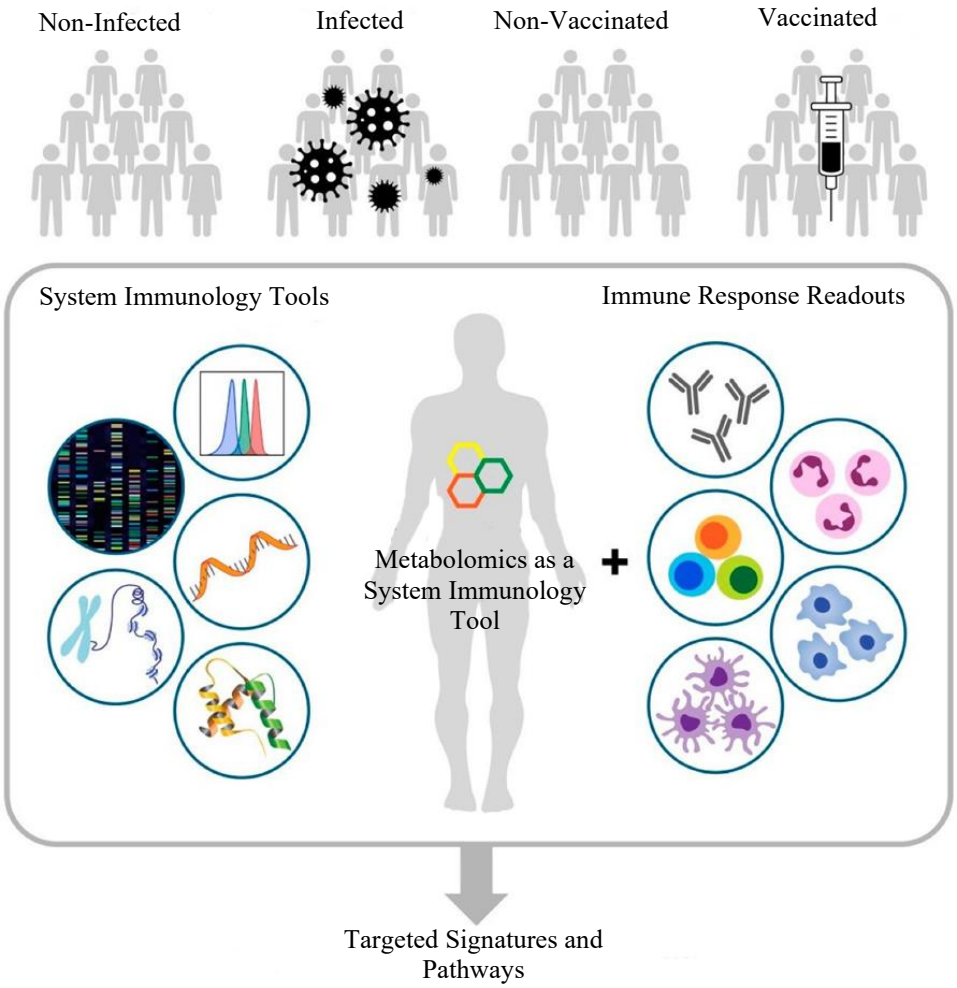

Figure 1. Metabolomics as a systems immunology tool for profiling host responses during parasitic infection. This figure illustrates how metabolomic approaches integrate with systems immunology to distinguish between infected and non-infected as well as vaccinated and non-vaccinated states. It highlights the identification of targeted metabolic signatures and pathways alongside immune response readouts, enabling comprehensive analysis of host–parasite interactions.
Source: https://www.mdpi.com/metabolites/metabolites-10 00492/article_deploy/html/images/metabolites-10-00492-g001.png

Immune Evasion by Parasites

To persist within hosts, parasites employ sophisticated evasion strategies that modulate or escape immune responses. Antigenic variation, a hallmark of *Trypanosoma brucei*, allows parasites to periodically change surface glycoproteins, evading antibody recognition [14, 15]. Molecular mimicry enables parasites to express host-like molecules, reducing immune detection. Other parasites secrete immunomodulatory proteins that inhibit antigen presentation, block complement activation, or induce regulatory T cell expansion, thereby dampening host immunity. Helminths are particularly adept at immune modulation, releasing excretory–secretory products that skew host immunity toward anti-inflammatory Th2 responses. This not only facilitates chronic infection but also leaves a lasting immunological imprint that can be traced as part of the parasitic fingerprint. These evasion mechanisms highlight the dynamic interplay between host defenses and parasite survival strategies [16–20].

Cellular and Tissue Tropism

Different parasites exhibit tropism for specific host tissues, shaping the clinical manifestations and molecular fingerprints of infection. *Plasmodium* species target erythrocytes and hepatocytes, leading to anemia, hepatosplenomegaly, and systemic inflammation. *Leishmania* species infect macrophages in

the skin, liver, and spleen, causing cutaneous or visceral lesions. Tissue tropism determines localized cytokine profiles, cellular damage, and host metabolic alterations, all of which contribute to disease-specific molecular signatures. Within target tissues, parasites can trigger apoptosis, necrosis, or altered cellular signaling. For example, *Toxoplasma gondii* modulates host gene expression to suppress apoptosis in infected cells, facilitating intracellular replication. Helminths induce fibrosis, angiogenesis, and immune cell infiltration in the gut or liver, generating distinct histopathological and molecular markers. Understanding these tissue-specific footprints is critical for linking parasite invasion to host illness [21–25].

Host–Parasite Molecular Interplay

The interaction between host and parasite is not merely antagonistic; it is a dynamic molecular dialogue. Parasites manipulate host signaling pathways, metabolic networks, and gene expression to create a permissive environment for survival. For instance, *Plasmodium falciparum* remodels erythrocyte membranes by exporting proteins, such as PfEMP1, altering adhesion and immune recognition. Similarly, *Leishmania* secretes GP63 protease to degrade host signaling proteins, impairing macrophage activation. Host cells respond by activating stress pathways, inflammatory mediators, and cell death mechanisms, leaving detectable molecular traces. These interactions form the core of the parasitic fingerprint, which can be identified through proteomic, transcriptomic, or metabolomic profiling. High-resolution mapping of these interactions allows researchers to trace the molecular trajectory from infection to tissue pathology and disease outcomes [25, 26].

MOLECULAR SIGNATURES OF INFECTION

Parasitic infections leave discernible molecular imprints in their hosts, collectively referred to as the "parasitic fingerprint." These signatures encompass alterations in host proteins, gene expression, metabolites, and extracellular factors induced by the invading parasite. Understanding these molecular changes provides insights into parasite survival strategies, disease pathogenesis, and potential diagnostic and therapeutic avenues (Figure 2) [27–30].

Proteomic Fingerprints

Proteomic profiling has revealed that parasites introduce unique proteins into host tissues and fluids, which can serve as direct indicators of infection. For example, *Plasmodium falciparum* exports numerous erythrocyte membrane proteins, such as PfEMP1, RIFINs, and STEVORs, that remodel the host cell surface and mediate cytoadherence, immune evasion, and pathogenicity [31–35]. These exported proteins generate measurable alterations in host blood composition and cell membrane architecture, forming identifiable proteomic fingerprints [36]. Similarly, *Leishmania* species secrete proteins, like GP63 and lipophosphoglycan (LPG) into macrophages, modulating immune signaling and enabling parasite survival. Proteomic analysis of infected tissues reveals differential expressions of host proteins involved in antigen processing, signal transduction, and apoptosis [37]. Helminth infections also induce protein expression changes in the host, often linked to tissue remodeling, fibrosis, and immune regulation. Identifying these protein-level changes is crucial for tracing infections and predicting disease outcomes.

Genomic and Epigenomic Changes

Parasites influence host gene expression through direct and indirect mechanisms. Intracellular parasites, such as *Toxoplasma gondii* and *Plasmodium spp.*, manipulate host transcriptional machinery to suppress apoptosis, modulate inflammatory responses, and enhance nutrient availability. Host transcriptomic analyses of infected cells often reveal upregulation of stress response genes, pro-inflammatory cytokines, and interferon-stimulated genes [38–40]. Conversely, genes involved in antigen presentation, cell cycle regulation, or metabolic homeostasis may be downregulated, reflecting parasite-driven modulation. Epigenetic modifications, including DNA methylation and histone modifications, further contribute to the molecular fingerprint. For instance, *Toxoplasma* infection can induce host histone modifications that alter chromatin accessibility and gene expression patterns, promoting long-term changes in immune responsiveness. Such genomic and epigenomic alterations

provide a molecular map connecting parasite presence to host pathology and are increasingly explored as biomarkers for infection severity and chronicity [41].

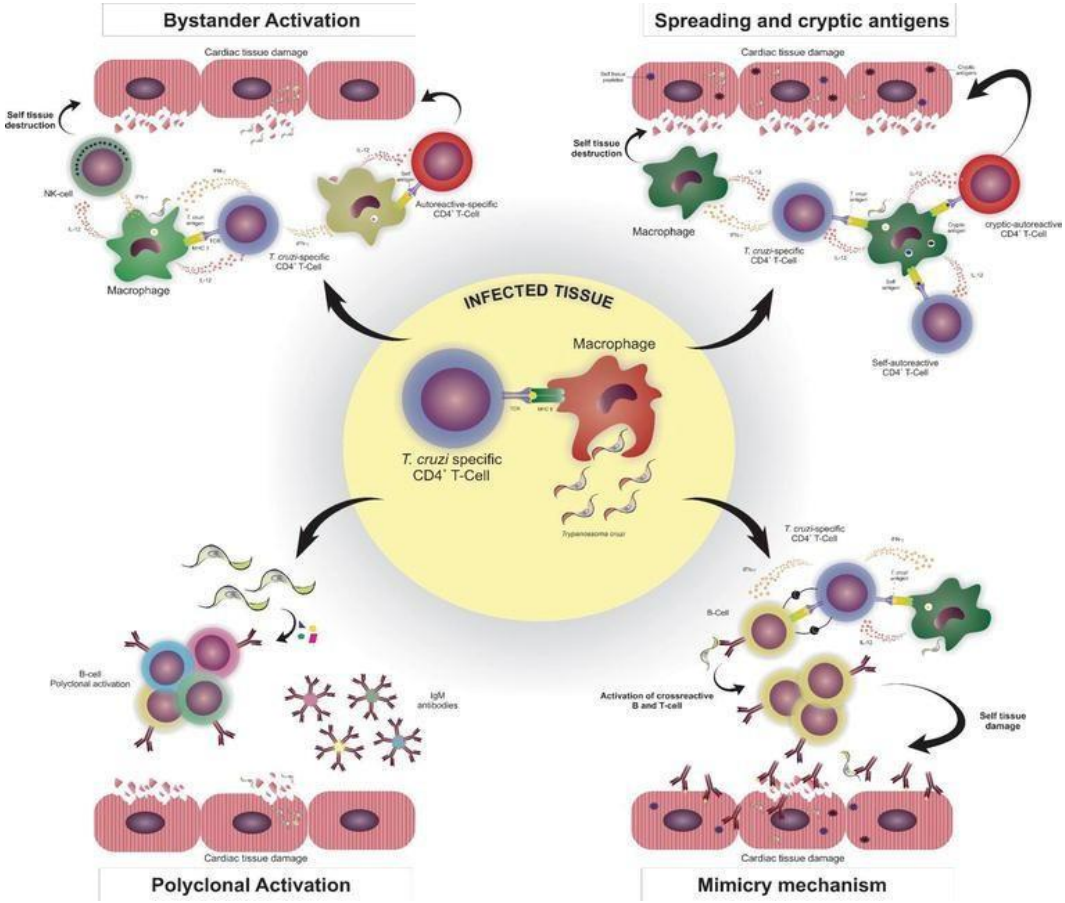

Figure 2. Molecular signatures of parasitic infection across multi-omics platforms. This figure represents the diverse molecular fingerprints generated during parasitic infections, including proteomic, genomic, transcriptomic, and metabolomic alterations. It emphasizes how these interconnected layers contribute to understanding parasite survival strategies, host immune modulation, and disease progression.
Source: https://www.intechopen.com/media/chapter/56631/media/F2.png

Metabolomic and Biochemical Markers

Parasitic infections also perturb host metabolism, creating distinct metabolomic signatures. In malaria, *Plasmodium*-infected erythrocytes consume glucose at high rates, leading to hypoglycaemia and altered plasma metabolite profiles. Amino acid depletion, lipid remodeling, and accumulation of parasite-derived metabolites, such as hemozoin, contribute to both host pathology and molecular fingerprints detectable through metabolomics [42–45]. Helminth infections, particularly chronic gastrointestinal infestations, modulate host lipid, carbohydrate, and vitamin metabolism, reflecting both parasite nutrient acquisition and host immune adaptation. Alterations in metabolites, like short-chain fatty acids, polyamines, and nitric oxide derivatives, can serve as indicators of infection and disease progression. Integrating metabolomic data with proteomic and genomic information provides a multi-dimensional view of the parasitic fingerprint, enabling a holistic understanding of host–parasite interactions [46–48].

Extracellular Vesicles and Secreted Factors

Parasites actively communicate with host cells via extracellular vesicles (EVs) and secreted molecules. EVs carry proteins, RNAs, microRNAs, and lipids that modulate host immune responses,

cellular signaling, and gene expression. For example, *Leishmania* exosomes deliver GP63 and small RNAs to host macrophages, suppressing pro-inflammatory cytokines and altering transcriptional programs. *Plasmodium*-derived microvesicles influence endothelial cell activation and vascular inflammation, contributing to cerebral malaria pathogenesis [49–52]. Secreted factors, such as proteases, lipids, and glycoproteins, similarly manipulate host signaling pathways, promoting immune evasion and tissue remodeling. These vesicles and secreted molecules leave measurable molecular footprints that can be detected in blood, urine, or tissue biopsies, making them valuable biomarkers for infection and disease monitoring [53].

Integration of Molecular Signatures

The parasitic fingerprint is best understood by integrating multiple molecular layers. Proteomic, transcriptomic, metabolomic, and secretomic data collectively provide a detailed map of host responses and parasite activity [54–59]. Multi-omics approaches enable researchers to identify critical nodes in host-parasite interactions, predict disease severity, and discover novel diagnostic markers. For instance, combining proteomic data of exported parasite proteins with host transcriptomic changes can reveal pathways essential for immune evasion and tissue damage, highlighting targets for therapeutic intervention [60]. Recent advances in computational biology, machine learning, and systems biology facilitate the integration of large-scale molecular data. Predictive models can correlate specific molecular signatures with clinical outcomes such as organ damage, anemia, or neurological complications in malaria, or tissue fibrosis in helminth infections [61, 62]. These approaches not only enhance our understanding of disease mechanisms but also allow for personalized diagnostics and targeted treatment strategies based on the molecular fingerprint of infection.

DISEASE MECHANISMS AND PATHOLOGY

The molecular fingerprints left by parasites in the host are intricately linked to the mechanisms of disease and pathology observed during infection [63–65]. Parasites induce host tissue damage through a combination of direct cellular injury, immunopathological responses, and long-term organ remodeling. Understanding these processes is crucial for connecting infection to the manifestation of illness and interpreting the clinical consequences of parasitic diseases [66–70].

Direct Tissue Damage

Parasites cause direct damage to host tissues through physical disruption, cytolysis, and nutrient depletion [71–73]. Protozoan parasites, like *Plasmodium falciparum*, invade red blood cells (RBCs) and undergo repeated cycles of replication and lysis, leading to hemolysis, anemia, and microvascular obstruction. Similarly, *Trypanosoma cruzi* invades cardiac myocytes, replicates intracellularly, and triggers cell rupture, resulting in myocarditis and chronic cardiomyopathy [74, 75]. Helminths, by contrast, inflict mechanical damage during migration through tissues. Schistosome larvae penetrate the skin, travel through the bloodstream, and lodge in the liver or intestines, causing granulomatous inflammation and fibrosis. Larval migration by *Ascaris lumbricoides* through the lungs can result in tissue hemorrhage and eosinophilic inflammation [76–80]. These forms of direct tissue injury leave molecular footprints, including cellular debris, altered extracellular matrix components, and the presence of parasite-derived antigens, which can be detected through histopathological and molecular assays.

Immunopathology

Host immune responses, while protective, can contribute to disease through immunopathological mechanisms. The inflammatory response to parasitic infection often generates tissue damage in addition to parasite clearance [81, 82]. Cytokines, such as interferon–gamma (IFN–γ), tumor necrosis factor–alpha (TNF–α), and interleukins (IL-1β, IL-6), are released in response to infection, activating macrophages, neutrophils, and other immune cells. While these mediators limit parasite proliferation, excessive or dysregulated responses can damage host tissues. For instance, cerebral malaria is associated with overactivation of endothelial cells and sequestration of infected RBCs, mediated by parasite proteins like PfEMP1 and host inflammatory cytokines [83–85]. The resulting blood–brain

barrier disruption, edema, and microvascular obstruction are hallmarks of severe immunopathology. Similarly, visceral leishmaniasis induces splenomegaly and hepatomegaly through excessive macrophage activation and chronic inflammatory signaling. Helminth-induced Th2 responses, while controlling worm burden, can lead to tissue fibrosis, airway hyperreactivity, or gut hypertrophy. These immunopathological events are reflected in molecular and cellular fingerprints that can serve as biomarkers for disease severity [86–90].

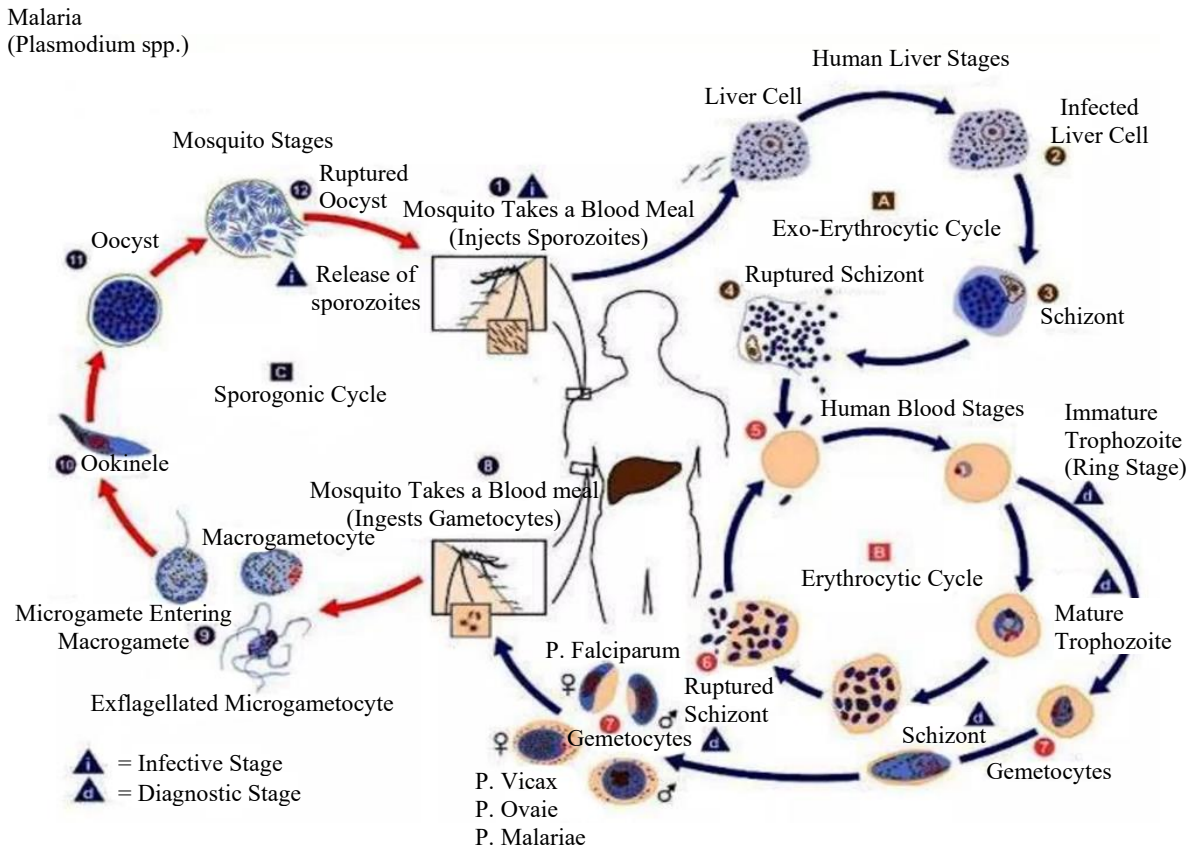

Figure 3. Life cycle of Plasmodium species and stages of malaria infection in human and mosquito hosts. This diagram depicts the complete life cycle of Plasmodium spp., including the mosquito (sporogonic) and human (exo-erythrocytic and erythrocytic) stages. It highlights key infective and diagnostic stages, illustrating parasite development in the liver and red blood cells, as well as transmission between mosquito and human hosts.

Source: https://image.slidesharecdn.com/jahnavi-200123122645/75/Malarial-parasite-morphology-and-lifecycle-mosquito-phase-15-2048.jpg

Chronic Infection and Host Remodeling

Chronic parasitic infections often result in long-term alterations of host tissues and physiology. Persistent infection by helminths, for example, induces remodeling of the liver, intestines, and lymphatic system. Fibrosis in schistosomiasis is driven by prolonged inflammation and deposition of extracellular matrix proteins, leading to portal hypertension and hepatosplenic disease [91, 92]. Chronic *Trypanosoma cruzi* infection promotes cardiac remodeling, conduction abnormalities, and progressive cardiomyopathy through combined direct parasitic damage and sustained inflammatory responses. Parasite-induced metabolic and cellular remodeling also contributes to long-term host pathology [93–96]. *Plasmodium* infection alters erythrocyte deformability, oxidative stress balance, and nitric oxide bioavailability, while *Toxoplasma gondii* can manipulate neuronal signaling and immune surveillance, leading to latent infections with subtle but persistent consequences. Mapping these molecular changes provides critical insight into how chronic infection transitions into observable disease phenotypes [97].

Linking Molecular Fingerprints to Clinical Outcomes

The molecular fingerprints of parasites are intimately connected to clinical manifestations of disease. Proteins exported by parasites, host transcriptional changes, secreted metabolites, and immune response patterns all contribute to observable pathology. For example, PfEMP1 variants correlate with cerebral malaria severity, while GP63 expression in *Leishmania* correlates with tissue destruction and lesion progression. Helminth-secreted molecules can predict fibrosis severity and immune skewing in chronic infections [98–100]. High-throughput omics approaches have enabled the correlation of molecular signatures with disease outcomes. Proteomic and metabolomic profiling can identify host and parasite biomarkers predictive of severe disease, relapse, or treatment response. Transcriptomic analyses reveal immune activation patterns associated with specific clinical phenotypes, allowing researchers to link molecular fingerprints directly to tissue pathology and systemic illness [101–105].

Host Systems and Organ-Specific Pathology

Parasites exhibit tropism for particular organs, shaping both molecular fingerprints and disease manifestations. In malaria, sequestration of infected RBCs in the brain microvasculature leads to cerebral malaria, while liver-stage development contributes to hepatomegaly and metabolic alterations. In visceral leishmaniasis, infection of macrophages in the spleen and liver results in organ enlargement and cytopenias [106–110]. Helminth infections, such as *Schistosoma* species, target the liver and intestines, promoting fibrosis and portal hypertension. Each organ-specific infection leaves distinct molecular and cellular footprints, enabling the identification of biomarkers and diagnostic signatures.

Immunomodulation and Parasite Survival

Disease mechanisms are often shaped by parasite-induced immunomodulation. By manipulating host immune responses, parasites balance survival and pathology [111–114]. *Trypanosoma brucei* periodically changes its surface glycoproteins to evade antibodies, prolonging infection and allowing gradual organ damage. Helminths secrete immunomodulatory proteins that suppress inflammatory responses, promoting chronic infection while limiting overt tissue destruction [115–117]. This immunomodulatory fingerprint is detectable through cytokine profiling, transcriptional analysis, and metabolomics, linking host immune status to disease progression.

DIAGNOSTIC AND THERAPEUTIC IMPLICATIONS

The concept of parasitic fingerprint has profound implications for both diagnosis and treatment of parasitic diseases. By identifying molecular, cellular, and immunological signatures left by parasites in host systems, researchers, and clinicians can develop sensitive diagnostic tools, targeted therapies, and personalized intervention strategies. The integration of multi-omics data with clinical phenotypes enhances precision medicine approaches, improving outcomes for patients suffering from parasitic infections [118–120].

Diagnostics Based on Parasitic Fingerprints

Traditional diagnostic methods, such as microscopy, serology, and culture, often lack sensitivity, specificity, or speed. The identification of molecular fingerprints provides a new paradigm for accurate and early detection [121–125]. Proteomic signatures, including parasite-derived proteins, such as PfEMP1 in malaria or GP63 in *Leishmania* infections, can be detected in blood or tissue samples, providing highly specific diagnostic markers. Genomic approaches, including polymerase chain reaction (PCR) and next-generation sequencing (NGS), enable detection of parasite DNA or RNA even at low parasitemia levels [126, 127]. For instance, quantitative PCR targeting *Plasmodium* 18S rRNA genes can diagnose subclinical malaria infections, while NGS-based metagenomics can identify co-infections and novel parasites. Transcriptomic profiling of host responses can also indicate infection, as specific gene expression patterns are triggered by parasite-host interactions. Metabolomic and biochemical markers offer additional diagnostic opportunities. Changes in metabolites, such as hemozoin, polyamines, nitric oxide derivatives, and lipid profiles, can indicate parasite presence and disease stage [128–130]. Helminth infections, for example, alter amino acid and lipid metabolism, which can be detected in urine or serum as non-invasive biomarkers. Extracellular vesicles released by

parasites carry proteins, microRNAs, and lipids that provide a rich source of diagnostic information. Combining these multi-layered molecular signatures with computational models can enhance diagnostic accuracy and enable early detection before clinical symptoms manifest [131–133].

Targeted Therapies

Understanding the molecular fingerprints of parasites allows the development of highly specific therapies aimed at critical parasite survival pathways. For protozoan infections, drugs targeting exported proteins, enzymes, or metabolic pathways can inhibit parasite replication and reduce pathogenicity. Artemisinin derivatives for malaria, for example, target parasite hemoglobin metabolism and oxidative stress pathways, directly interfering with molecular processes reflected in the parasitic fingerprint. Helminth-targeted therapies exploit vulnerabilities in neuromuscular function, metabolism, or tegumental structures [134–136]. Drugs, such as praziquantel, induce calcium influx and tegumental disruption in schistosomes, while albendazole inhibits microtubule polymerization in nematodes. Understanding molecular footprints also enables drug repositioning and combination therapies that enhance efficacy while minimizing toxicity. Immunomodulatory therapies are particularly promising, as they can correct parasite-induced immune dysregulation without compromising protective responses. Cytokine modulators, immune checkpoint inhibitors, or small molecules targeting regulatory T cell pathways can counteract parasite-driven immunosuppression [137–140]. By leveraging molecular fingerprints, these interventions can be tailored to individual host responses, reducing disease severity and improving therapeutic outcomes.

Vaccine Development

Parasitic fingerprints also guide vaccine design by identifying immunogenic molecules critical for parasite survival or host-pathogen interactions. Surface proteins, secreted enzymes, and extracellular vesicle components serve as potential vaccine targets [141, 142]. For example, PfEMP1, and circumsporozoite protein (CSP) in *Plasmodium* are being investigated as vaccine candidates, as they are central to parasite invasion and immune evasion. *Leishmania* GP63, a protease involved in macrophage manipulation, represents another promising target. Vaccines designed to elicit strong humoral and cellular immune responses against these molecules can prevent infection or reduce disease severity [143–145]. Multi-epitope vaccines and recombinant protein formulations benefit from knowledge of molecular fingerprints, as they enable the selection of conserved, immunodominant antigens while avoiding cross-reactivity.

Personalized Medicine Approaches

The integration of parasitic fingerprints with host genetic and immunological data facilitates personalized medicine strategies. Host-specific molecular signatures, including transcriptomic, proteomic, and metabolomic profiles, can predict susceptibility, disease severity, and response to therapy [146–150]. For example, host cytokine profiles can indicate the risk of severe malaria or visceral leishmaniasis, guiding prophylactic or therapeutic interventions. High-resolution omics analyses allow stratification of patients based on molecular risk factors, enabling individualized treatment regimens. Patients with high-risk molecular signatures may benefit from early aggressive therapy, while low-risk individuals may require supportive care and monitoring. This approach maximizes efficacy, minimizes adverse effects, and optimizes resource allocation in endemic regions.

Challenges and Future Directions in Diagnostics and Therapy

Despite the promise of molecular fingerprints, several challenges remain. The heterogeneity of parasite strains, co-infections, and host genetic diversity complicates the identification of universal biomarkers. Additionally, high-throughput omics technologies can be resource-intensive and require sophisticated computational analysis, limiting accessibility in low-resource settings where parasitic diseases are most prevalent [151, 152]. Future research should focus on standardizing molecular fingerprint assays, integrating multi-omics data, and developing portable diagnostic platforms. Point-of-care devices leveraging proteomic or metabolomic signatures could enable rapid, accurate detection in endemic areas. On the therapeutic side, combining molecular fingerprint information with drug

discovery pipelines, immunotherapy, and vaccine development will enhance precision and efficacy [153–155].

CASE STUDIES OF PARASITIC FINGERPRINTS

Examining specific parasitic infections illustrates how molecular fingerprints link infection to host illness. Here, malaria, leishmaniasis, trypanosomiasis, and helminth infections are reviewed as representative cases, highlighting how parasite-induced molecular and immunological signatures correspond to disease pathogenesis and clinical outcomes.

Malaria (Plasmodium spp.)

Malaria, caused primarily by *Plasmodium falciparum* and *Plasmodium vivax*, remains a major global health challenge. The parasitic fingerprint in malaria is characterized by both parasite-derived molecules and host responses [156, 157]. Parasite proteins are exported to erythrocytes, such as PfEMP1, RIFINs, and STEVORs, alter red blood cell deformability, adhesion properties, and immune recognition. These changes contribute to microvascular obstruction, anemia, and cerebral malaria. Host molecular responses include upregulation of pro-inflammatory cytokines (TNF-α, IFN-γ, IL-6) and oxidative stress markers, which contribute to fever, tissue damage, and systemic inflammation. Metabolomic studies reveal depletion of glucose and amino acids in plasma, alongside accumulation of parasite-specific metabolites like hemozoin. The combined profiling of parasite proteins, host cytokines, and metabolites constitutes a molecular fingerprint that correlates with disease severity and informs prognosis.

Leishmaniasis

Leishmaniasis, caused by *Leishmania* species, demonstrates how intracellular parasites manipulate host macrophages to survive. The GP63 metalloprotease and lipophosphoglycan (LPG) are secreted by the parasite modulate host signaling pathways, suppressing pro-inflammatory cytokine production and inhibiting antigen presentation. This molecular interference allows the parasite to persist in tissues such as skin, spleen, and liver. Host transcriptomic and proteomic analyses reveal altered expression of immune regulatory genes, including IL-10 and TGF-β, which promote immune tolerance and chronic infection. Metabolomic profiling shows changes in amino acid and lipid metabolism, reflecting both parasite consumption and host immune adaptation [158–160]. These molecular signatures provide diagnostic markers for visceral versus cutaneous forms of the disease and predict clinical outcomes, including organ enlargement and cytopenias.

Trypanosomiasis

Trypanosomiasis, caused by *Trypanosoma brucei* (African sleeping sickness) and *Trypanosoma cruzi* (Chagas disease), illustrates the role of antigenic variation and host immune modulation in disease progression. *T. brucei* periodically changes its variant surface glycoproteins (VSGs), evading antibody detection and sustaining chronic infection. These VSG switching events constitute a dynamic molecular fingerprint associated with parasitemia waves and disease severity. In *T. cruzi* infection, the parasite invades cardiac and smooth muscle cells, causing direct cytolysis and chronic inflammation. Host transcriptomic and proteomic profiling shows upregulation of inflammatory cytokines and oxidative stress pathways, while metabolomic analysis indicates mitochondrial dysfunction and altered energy metabolism in cardiac tissues [161, 162]. The molecular fingerprints of trypanosomiasis thus reflect both parasite survival strategies and host tissue remodeling, linking infection to cardiomyopathy, neurological symptoms, and systemic illness.

Helminth Infections

Helminths, such as *Schistosoma*, *Ascaris*, and *Fasciola* species, cause chronic infections that reshape host tissues and immune responses. Parasite excretory–secretory products, including proteases, glycoproteins, and immunomodulatory molecules, induce fibrosis, angiogenesis, and Th2-skewed immune responses. These molecules form identifiable molecular fingerprints in host serum and tissue, reflecting both parasite activity and host adaptation. Host molecular signatures include elevated levels

of IL-4, IL-5, and IL-13 cytokines, eosinophilia, and changes in collagen and extracellular matrix components in affected tissues. Metabolomic studies reveal altered lipid metabolism, oxidative stress markers, and vitamin deficiencies [143]. Chronic helminth infections also induce epigenetic modifications, including DNA methylation changes that affect immune gene expression. Together, these molecular and cellular fingerprints explain the clinical manifestations of organomegaly, anaemia, growth retardation, and fibrosis.

Integration of Case Studies

Across these case studies, a common theme emerges: the parasitic fingerprint integrates parasite-derived molecules, host immune responses, metabolic alterations, and tissue-specific changes to link infection with disease outcomes. In malaria, fingerprints correlate with acute severity and organ dysfunction; in leishmaniasis, they predict chronicity and organ involvement; in trypanosomiasis, they reflect cycles of immune evasion and tissue damage; and in helminth infections, they explain long-term pathology and immune modulation. High-throughput multi-omics approaches—combining proteomics, transcriptomics, metabolomics, and epigenomics—allow researchers to map these fingerprints comprehensively. Integrating molecular data with clinical phenotypes enhances the ability to predict disease progression, identify early biomarkers, and design targeted interventions [133–135]. The case studies illustrate the practical value of the parasitic fingerprint concept for understanding disease mechanisms, improving diagnostics, and guiding therapeutics.

FUTURE DIRECTIONS AND RESEARCH GAPS

The concept of the parasitic fingerprint offers a promising framework for understanding the progression from infection to host illness [1, 2]. Despite advances in molecular biology, omics technologies, and immunology, several critical knowledge gaps remain. Addressing these gaps will improve disease prediction, diagnostic accuracy, therapeutic strategies, and the overall understanding of host–parasite interactions.

High-Resolution Molecular Mapping

While current studies have identified numerous parasite-derived molecules and host response markers, comprehensive, high-resolution mapping of the parasitic fingerprint remains incomplete. Multi-omics approaches integrating genomics, transcriptomics, proteomics, metabolomics, and epigenomics are essential to capture the full complexity of host–parasite interactions. For instance, single-cell RNA sequencing can reveal cell-type-specific host responses, while spatial proteomics can identify tissue-specific parasite protein localization [55–57]. Future research should focus on constructing detailed molecular atlases of infection stages, tissue tropism, and host immune modulation. High-resolution mapping will enable researchers to pinpoint critical nodes in host–parasite interactions that drive pathology, guiding the development of targeted diagnostics and therapies.

Integration with Systems Biology and Computational Modelling

Systems biology approaches offer opportunities to integrate large-scale molecular data with host physiology, clinical outcomes, and epidemiological trends. Computational models can simulate host–parasite interactions, predict disease progression, and identify key molecular drivers of pathology. Machine learning algorithms can analyze multi-omics datasets to discover novel biomarkers, classify infection severity, and predict therapeutic responses. Integrating parasitic fingerprints with systems biology will allow a more holistic understanding of infection dynamics, bridging molecular signatures with clinical phenotypes [11, 12]. Predictive models could facilitate personalized medicine approaches, enabling interventions tailored to individual molecular and immunological profiles.

Translational Opportunities in Diagnostics

The molecular signatures identified in parasitic fingerprints hold great promise for translational applications. Development of rapid, point-of-care diagnostics based on proteomic, metabolomic, or transcriptomic markers could revolutionize disease detection, particularly in resource-limited endemic regions. Portable platforms, such as microfluidic devices, biosensors, and lateral-flow assays, could

detect parasite-specific proteins, nucleic acids, or metabolites with high sensitivity and specificity. Further research is needed to validate these biomarkers across diverse parasite strains, host populations, and disease stages. Standardizing molecular fingerprint assays and ensuring reproducibility will be critical for clinical implementation. Integration of molecular diagnostics with traditional methods could enhance accuracy, speed, and patient outcomes.

Therapeutic and Vaccine Development

Understanding the molecular and immunological fingerprints of parasites informs targeted therapeutic strategies. Future research should explore drugs that disrupt parasite-specific pathways, secreted factors, or immune evasion mechanisms identified in the fingerprint. Combination therapies guided by molecular signatures may enhance efficacy and reduce resistance development. Vaccine development can also benefit from fingerprint-guided approaches. Identifying conserved, immunogenic parasite molecules that contribute to survival or pathogenesis enables rational vaccine design. Multi-epitope, recombinant, or nanoparticle-based vaccines targeting critical fingerprint molecules could induce protective immunity while minimizing off-target effects. Additionally, immunomodulatory therapies that restore host immune balance disrupted by parasite fingerprints could improve outcomes in chronic infections.

Host-Parasite Co-Evolution and Molecular Diversity

Parasites exhibit remarkable genetic and phenotypic diversity, resulting in variation in molecular fingerprints across strains, species, and geographic regions. Understanding how host genetics and immune status interact with parasite variability is crucial for interpreting molecular signatures and predicting disease outcomes. Future studies should investigate co-evolutionary dynamics, including how host immune pressures shape parasite molecular strategies and vice versa. Comparative studies across populations and species can identify conserved molecular fingerprints, informing universal diagnostics and therapeutic targets.

Knowledge Gaps and Research Challenges

Despite advances, several challenges persist:

- *Heterogeneity of Fingerprints:* Molecular signatures vary by parasite species, strain, infection stage, and host genetics. Identifying robust, reproducible markers remains a challenge.
- *Limited Access in Endemic Regions:* High-throughput omics technologies often require resources unavailable in areas most affected by parasitic diseases. Developing cost-effective, field-adapted assays is critical.
- *Integration of Multi-Layered Data:* Combining proteomic, transcriptomic, metabolomic, and clinical data is computationally complex. Standardized frameworks for data integration are needed.
- *Longitudinal Studies:* Understanding how fingerprints evolve over time during acute and chronic infections require longitudinal sampling and analysis, which is currently limited.

Addressing these challenges will improve the reliability, applicability, and translational impact of parasitic fingerprint research.

Emerging Technologies and Future Directions

Several emerging technologies offer new avenues for fingerprint research:

- *Single-Cell Multi-Omics:* Captures host and parasite signatures at the cellular level, revealing heterogeneity in infection and immune responses.
- *Spatial Transcriptomics and Proteomics:* Maps molecular fingerprints within tissue architecture, linking local parasite activity to organ-specific pathology.
- *Artificial Intelligence and Machine Learning:* Facilitates pattern recognition, biomarker discovery, and predictive modelling of disease outcomes based on molecular fingerprints.
- *CRISPR-Cas9 and Gene Editing:* Enables functional validation of fingerprint molecules and exploration of parasite survival strategies.

- *Point-of-Care Biosensors:* Translates fingerprint-based diagnostics to field-deployable, real-time detection platforms.

By leveraging these technologies, future research can enhance our understanding of parasitic fingerprints, improve early detection, optimize therapeutic strategies, and ultimately reduce the global burden of parasitic diseases.

CONCLUSION

Parasitic infections remain a significant global health challenge, causing extensive morbidity and mortality across diverse populations. Understanding the pathogenesis of these diseases requires a comprehensive view of the interactions between parasites and their hosts. The concept of the "parasitic fingerprint" provides such a framework, linking the molecular, cellular, and immunological signatures left by parasites to the clinical manifestations of disease. By tracing these fingerprints, researchers can connect the initial infection to tissue-specific pathology, immune dysregulation, and long-term illness. Molecular fingerprints encompass parasite-derived proteins, secreted factors, nucleic acids, metabolites, and extracellular vesicles, alongside host responses including gene expression changes, cytokine profiles, and metabolic adaptations. Multi-omics approaches integrating proteomics, genomics, transcriptomics, metabolomics, and epigenomics have greatly enhanced the ability to characterize these signatures at high resolution. Such analyses reveal the strategies parasites employ to invade, survive, and manipulate host systems, while also identifying host pathways critical for immunity and disease progression. The clinical relevance of parasitic fingerprints is substantial. They provide precise biomarkers for early and accurate diagnosis, enable stratification of disease severity, and inform prognosis. Furthermore, fingerprint-guided research supports the development of targeted therapeutics, vaccines, and immunomodulatory interventions. Personalized medicine approaches, informed by host and parasite molecular signatures, promise optimized treatment strategies and improved patient outcomes, particularly in regions heavily burdened by parasitic diseases. Despite these advances, challenges remain. Parasite diversity, host heterogeneity, resource limitations, and the complexity of multi-omics integration hinder the translation of molecular fingerprints into practical applications. Addressing these gaps through high-resolution mapping, longitudinal studies, and emerging technologies, such as single-cell multi-omics, spatial transcriptomics, and artificial intelligence, will enhance the understanding of host–parasite interactions and accelerate translational research.

Declarations

Ethics and Consent to Participate

Not Applicable.

Consent for Publication

Not Applicable.

Availability of Data and Materials

Not Applicable.

Competing Interests

The authors declare no competing interests.

Funding

There was no funding for this review article.

Author's Information

- *Shubham Sah:* School of Biology, Indian Institute of Science Education and Research (IISER), Thiruvananthapuram, Kerala, India.
- *Deepa Kumari:* Department of Biochemistry, Central University of Haryana (CUH), Mahendergarh, Haryana, India.

Authors' Contributions

Shubham Sah (SS) and Deepa Kumari (DK) contributed to the conception, drafting, literature review, data interpretation, and critical revision of the manuscript. Both authors read and approved the final version of the manuscript.

Acknowledgments

Sincere gratitude is extended to the Indian Institute of Science Education and Research, Thiruvananthapuram, and the Central University of Haryana, Mahendergarh, for their continuous academic support. Special thanks are extended by Shubham Sah and Deepa Kumari to their mentors, colleagues, and peers for creating an encouraging environment that inspired this review and for the constructive discussions that shaped its final form.

REFERENCES

1. Ward PK, Mohammed MA, Ayana Heda M, Broadfield LA, Dahlberg P, Dana D, Leta GT, Mekonnen Z, Nabatte B, Kabatereine N, Orrling KM. An artificial intelligence-powered digital pathology platform to support large-scale deworming programs against soil-transmitted helminthiasis and intestinal schistosomiasis in resource-limited settings. PLOS Neglected Tropical Diseases. 2026 Mar 18;20(3):e0013432.
2. Edoa JR, Adégbitè BR, Honkpéhèdji YJ, Zinsou JF, Boussougou-Sambe ST, Woldearegai TG, Mordmüller B, Adegnika AA, Dejon-Agobé JC. Epidemiology of soil-transmitted helminth infections and the differential effect of treatment on the distribution of helminth species in rural areas of Gabon. Tropical medicine and health. 2024 Jan 2;52(1):3.
3. Kaminsky R, Mäser P. Global impact of parasitic infections and the importance of parasite control. Frontiers in Parasitology. 2025 Mar 10;4:1546195.
4. Maizels RM, Yazdanbakhsh M. Immune regulation by helminth parasites: cellular and molecular mechanisms. Nature Reviews Immunology. 2003 Sep 1;3(9):733–44.
5. Allen JE, Maizels RM. Diversity and dialogue in immunity to helminths. Nature Reviews Immunology. 2011 Jun;11(6):375–88.
6. Torgerson PR, Macpherson CN. The socioeconomic burden of parasitic zoonoses: global trends. Veterinary parasitology. 2011 Nov 24;182(1):79–95.
7. Mishra R, Panda SK, Sahoo PK, Bal MS, Satapathy AK. Increased Fas ligand expression of peripheral B-1 cells correlated with CD 4+ T-cell apoptosis in filarial-infected patients. Parasite Immunology. 2017 Apr;39(4):e12421.
8. Dobson A, et al. Epidemiology of parasitic infections and molecular signatures of host response. Parasit Vectors. 2020;13:450.
9. Loke P, Allen JE. The parasitic worm host–parasite interface. Nat Rev Immunol. 2004;4(1):1–12. doi:10.1038/nri1316
10. Shanmugam S, Nichols AK, Saravanabalaji D, Welsch C, Yi M. HCV NS5A dimer interface residues regulate HCV replication by controlling its self-interaction, hyperphosphorylation, subcellular localization and interaction with cyclophilin A. PLoS pathogens. 2018 Jul 23;14(7):e1007177.
11. Velleman Y, Blair L, Fleming F, Fenwick A. Water-, sanitation-, and hygiene-related diseases. InInfectious Diseases 2023 Feb 8 (pp. 189-219). New York, NY: Springer US.
12. Aksoy S, et al. Host–parasite molecular fingerprinting: implications for diagnostics and therapeutics. Front Cell Infect Microbiol. 2021;11:703412. doi:10.3389/fcimb.2021.703412
13. Walker MJ, et al. Immune modulation by parasites: molecular mechanisms and clinical implications. Clin Exp Immunol. 2019;196(3):315–330.
14. Figueiredo C, et al. Proteomic insights into host–parasite interactions. Mol Biochem Parasitol. 2022;248:111525.
15. Antony B, et al. Parasitic infection-induced transcriptomic signatures in humans. Nat Commun. 2021;12:1547. doi:10.1038/s41467-021-21842-2
16. Maizels RM, McSorley HJ. Regulation of the host immune system by helminth parasites. Journal of Allergy and Clinical Immunology. 2016 Sep 1;138(3):666–75.

17. Ward PK, Mohammed MA, Ayana Heda M, Broadfield LA, Dahlberg P, Dana D, Leta GT, Mekonnen Z, Nabatte B, Kabatereine N, Orrling KM. An artificial intelligence-powered digital pathology platform to support large-scale deworming programs against soil-transmitted helminthiasis and intestinal schistosomiasis in resource-limited settings. PLOS Neglected Tropical Diseases. 2026 Mar 18;20(3):e0013432.
18. Desowitz RS. The malaria capers: more tales of parasites and people, research and reality. WW Norton & Company; 1993.
19. Maizels RM. Parasitic fingerprints: molecular signatures and the immunological footprint of helminth infections. Curr Opin Immunol. 2020;66:1–8.
20. Kaminsky R, Mäser P. Global impact of parasitic infections and the importance of parasite control. Frontiers in Parasitology. 2025 Mar 10;4:1546195.
21. Maizels RM, Yazdanbakhsh M. Immune regulation by helminth parasites: Cellular and molecular mechanisms. Nat Rev Immunol. 2003;3:733–744. Available from: https://www.nature.com/articles/nri1183
22. Mishra R, Panda SK, Sahoo PK, Bal MS, Satapathy AK. Increased Fas ligand expression of peripheral B-1 cells correlated with CD 4+ T-cell apoptosis in filarial-infected patients. Parasite Immunology. 2017 Apr;39(4):e12421.
23. Olano JP, Weller PF, Guerrant RL, Walker DH. Principles of parasitism: host–parasite interactions. Tropical infectious diseases: Principles, pathogens and practice. 2011 Apr 29:1
24. Desowitz RS. The malaria capers: more tales of parasites and people, research and reality. WW Norton & Company; 1993.
25. Petri WA Jr, Haque R. Host–parasite interactions in protozoan infections. Annu Rev Pathol. 2019;14:353–379. doi:10.1146/annurev-pathmechdis-012418-012754
26. Allen JE, Maizels RM. Diversity and dialogue in immunity to helminths. Nature Reviews Immunology. 2011 Jun;11(6):375–88.
27. Shanmugam S, Nichols AK, Saravanabalaji D, Welsch C, Yi M. HCV NS5A dimer interface residues regulate HCV replication by controlling its self-interaction, hyperphosphorylation, subcellular localization and interaction with cyclophilin A. PLoS pathogens. 2018 Jul 23;14(7):e1007177.
28. Chulanetra M, Chaicumpa W. Revisiting the mechanisms of immune evasion employed by human parasites. Frontiers in cellular and infection microbiology. 2021 Jul 29;11:702125.
29. Babunovic GH. Improving and understanding macrophage restriction of Mycobacterium tuberculosis infection. Harvard University; 2022.
30. Berriman M, Haas BJ, LoVerde PT, Wilson RA, Dillon GP, Cerqueira GC, Mashiyama ST, Al-Lazikani B, Andrade LF, Ashton PD, Aslett MA. The genome of the blood fluke Schistosoma mansoni. Nature. 2009 Jul 16;460(7253):352-8.
31. Mu Y, Zumuk CP, Jones MK, Cai P. Leveraging single-cell RNA-seq in helminthology. Trends in Parasitology. 2025 Dec 4.
32. Aksoy S. Control of tsetse flies and trypanosomes using molecular genetics. Veterinary parasitology. 2003 Jul 25;115(2):125-45.
33. Figueiredo J, Santos RB, Figueiredo A. Exploring Plant-Pathogen Interactions through Subcellular Proteomics: Insights and Challenges. InPlant Pathogen Interaction 2024 Jan 6 (pp. 287-310). Singapore: Springer Nature Singapore.
34. Casadevall A, Pirofski LA. Host-pathogen interactions: basic concepts of microbial commensalism, colonization, infection, and disease. Infection and immunity. 2000 Dec 1;68(12):6511-8.
35. Hotez PJ. Reducing the global burden of human parasitic diseases. Comparative Parasitology. 2002 Jul;69(2):140-5.
36. Maizels RM, Smits HH, McSorley HJ. Modulation of host immunity by helminths: the expanding repertoire of parasite effector molecules. Immunity. 2018 Nov 20;49(5):801-18.
37. Blackwell JM. Role of macrophage complement and lectin-like receptors in binding Leishmania parasites to host macrophages. Immunology letters. 1985 Jan 1;11(3-4):227-32.
38. Dubey JP, Jones JL. Toxoplasma gondii infection in humans and animals in the United States. International journal for parasitology. 2008 Sep 1;38(11):1257-78.

39. Maizels RM. Parasitic helminth infections and the control of human allergic and autoimmune disorders. Clinical Microbiology and Infection. 2016 Jun 1;22(6):481-6.
40. Vasoo S, Pritt BS. Molecular diagnostics and parasitic disease. Clinics in Laboratory Medicine. 2013 Sep 1;33(3):461-503
41. Smith JD. The role of PfEMP1 adhesion domain classification in Plasmodium falciparum pathogenesis research. Molecular and biochemical parasitology. 2014 Jul 1;195(2):82-7.
42. Menezes JP, Almeida TF, Petersen AL, Guedes CE, Mota MS, Lima JG, Palma LC, Buck GA, Krieger MA, Probst CM, Veras PS. Proteomic analysis reveals differentially expressed proteins in macrophages infected with Leishmania amazonensis or Leishmania major. Microbes and Infection. 2013 Jul 1;15(8-9):579-91
43. Florens L, Washburn MP, Raine JD, Anthony RM, Grainger M, Haynes JD, Moch JK, Muster N, Sacci JB, Tabb DL, Witney AA. A proteomic view of the Plasmodium falciparum life cycle. Nature. 2002 Oct 3;419(6906):520-6.
44. Gomez MA, Contreras I, Hallé M, Tremblay ML, McMaster RW, Olivier M. Leishmania GP63 alters host signaling through cleavage-activated protein tyrosine phosphatases. Science signaling. 2009 Sep 29;2(90):ra58-.
45. Sahoo MP. Experimenatal studies on murine host inflammatory responses to malarial parasite-derived HMGB1.
46. Hunter CA, Sibley LD. Modulation of innate immunity by Toxoplasma gondii virulence effectors. Nature Reviews Microbiology. 2012 Nov;10(11):766-78.
47. Minarovits J. Microbe-induced epigenetic alterations in host cells: the coming era of patho-epigenetics of microbial infections: a review. Acta microbiologica et immunologica Hungarica. 2009 Mar 1;56(1):1-9.
48. Diray-Arce J, Conti MG, Petrova B, Kanarek N, Angelidou A, Levy O. Integrative metabolomics to identify molecular signatures of responses to vaccines and infections. Metabolites. 2020 Nov 30;10(12):492.
49. Sikder S, Pierce D, Sarkar ER, McHugh C, Quinlan KG, Giacomin P, Loukas A. Regulation of host metabolic health by parasitic helminths. Trends in Parasitology. 2024 May 1;40(5):386-400.
50. Regan JC, Brandão AS, Leitão AB, Mantas Dias AR, Sucena É, Jacinto A, Zaidman-Rémy A. Steroid hormone signaling is essential to regulate innate immune cells and fight bacterial infection in Drosophila. PLoS pathogens. 2013 Oct 24;9(10):e1003720.
51. Silverman JM, Clos J, Horakova E, Wang AY, Wiesgigl M, Kelly I, Lynn MA, McMaster WR, Foster LJ, Levings MK, Reiner NE. Leishmania exosomes modulate innate and adaptive immune responses through effects on monocytes and dendritic cells. The Journal of Immunology. 2010 Nov;185(9):5011-22.
52. Ortega-Pajares A, Rogerson SJ. The rough guide to monocytes in malaria infection. Frontiers in immunology. 2018 Dec 7;9:2888.
53. Rezende LG, Barros MD, Moreira OB, de Oliveira MA, Chellini PR, Jaeger LH. Analytical Approaches for Parasitic Biomarkers Diseases Discovery: Trends and Perspectives of Metabolomics in the Clinical Laboratory. Critical Reviews in Analytical Chemistry. 2025 Oct 31:1-7.
54. Tonkin-Hill GQ, Trianty L, Noviyanti R, Nguyen HH, Sebayang BF, Lampah DA, Marfurt J, Cobbold SA, Rambhatla JS, McConville MJ, Rogerson SJ. The Plasmodium falciparum transcriptome in severe malaria reveals altered expression of genes involved in important processes including surface antigen–encoding var genes. PLoS Biology. 2018 Mar 12;16(3):e2004328.
55. Cross GA. Antigenic variation in trypanosomes. Proceedings of the Royal Society of London. Series B. Biological Sciences. 1978 Jun 5;202(1146):55-72.
56. Silvie O, Goetz K, Matuschewski K. A sporozoite asparagine-rich protein controls initiation of Plasmodium liver stage development. PLoS pathogens. 2008 Jun 13;4(6):e1000086.
57. Sarfraz RA, Rizwan HM, Nazish N, Taseer MS, Hassan SS. Integrative Omics in Parasitology. InOmics Approaches in Veterinary Parasitology 2024 Nov 28 (pp. 109-123). CRC Press.
58. Cobbold SA, V Tutor M, Frasse P, McHugh E, Karnthaler M, Creek DJ, Odom John A, Tilley L, Ralph SA, McConville MJ. Non-canonical metabolic pathways in the malaria parasite detected by isotope-tracing metabolomics. Molecular Systems Biology. 2021 Apr 1;17(4):MSB202010023.

59. Maizels RM, Smits HH, McSorley HJ. Modulation of host immunity by helminths: the expanding repertoire of parasite effector molecules. Immunity. 2018 Nov 20;49(5):801-18.
60. White NJ. Malaria parasite clearance. Malaria journal. 2017 Feb 23;16(1):88.
61. Cowman AF, Tonkin CJ, Tham WH, Duraisingh MT. The molecular basis of erythrocyte invasion by malaria parasites. Cell host & microbe. 2017 Aug 9;22(2):232-45.
62. Menezes TP, Machado BA, Toledo DN, Dos Santos PV, Ribeiro L, Talvani A. Insights into CX3CL1/Fractalkine during experimental Trypanosoma cruzi infection. Parasitology international. 2022 Apr 1;87:102530.
63. Burke ML, Jones MK, Gobert GN, Li YS, Ellis MK, McManus DP. Immunopathogenesis of human schistosomiasis. Parasite immunology. 2009 Apr;31(4):163-76.
64. Hotez PJ. Human parasitology and parasitic diseases: heading towards 2050. Advances in parasitology. 2018 Jan 1;100:29-38.
65. Idro R, Jenkins NE, Newton CR. Pathogenesis, clinical features, and neurological outcome of cerebral malaria. The Lancet Neurology. 2005 Dec 1;4(12):827-40.
66. Olivier M, Gregory DJ, Forget G. Subversion mechanisms by which Leishmania parasites can escape the host immune response: a signaling point of view. Clinical microbiology reviews. 2005 Apr;18(2):293-305.
67. Gazzinelli RT, et al. Host immune responses and pathology in parasitic infections. Nat Rev Immunol. 2014;14(4):219–231. Available from: https://www.nature.com/articles/nri3630
68. Fallon PG, Mangan NE. Suppression of TH2-type allergic reactions by helminth infection. Nature Reviews Immunology. 2007 Mar;7(3):220-30.
69. Clark IA, Schofield L. Pathogenesis of malaria. Parasitology Today. 2000 Oct 1;16(10):451-4.
70. Afrin F, Khan I, Hemeg HA. Leishmania-host interactions—an epigenetic paradigm. Frontiers in immunology. 2019 Mar 22;10:492.
71. Andrade LO, Andrews NW. The Trypanosoma cruzi–host-cell interplay: location, invasion, retention. Nature Reviews Microbiology. 2005 Oct 1;3(10):819-23.
72. Fabre V, Beiting DP, Bliss SK, Gebreselassie NG, Gagliardo LF, Lee NA, Lee JJ, Appleton JA. Eosinophil deficiency compromises parasite survival in chronic nematode infection. The Journal of Immunology. 2009 Feb;182(3):1577-83.
73. Jang JC, Chen G, Wang SH, Barnes MA, Chung JI, Camberis M, Le Gros G, Cooper PJ, Steel C, Nutman TB, Lazar MA. Macrophage-derived human resistin is induced in multiple helminth infections and promotes inflammatory monocytes and increased parasite burden. PLoS pathogens. 2015 Jan 8;11(1):e1004579.
74. Pearce EJ, MacDonald AS. The immunobiology of schistosomiasis. Nature Reviews Immunology. 2002 Jul 1;2(7):499-511.
75. Baer M, Sawa T, Flynn P, Luehrsen K, Martinez D, Wiener-Kronish JP, Yarranton G, Bebbington C. An engineered human antibody fab fragment specific for Pseudomonas aeruginosa PcrV antigen has potent antibacterial activity. Infection and immunity. 2009 Mar;77(3):1083-90.
76. Parody N, Soto M, Requena JM, Alonso C. Adjuvant guided polarization of the immune humoral response against a protective multicomponent antigenic protein (Q) from Leishmania infantum. A CpG+ Q mix protects Balb/c mice from infection. Parasite immunology. 2004 Jun;26(6-7):283-93.
77. Kaye PM, Cruz I, Picado A, Van Bocxlaer K, Croft SL. Leishmaniasis immunopathology—impact on design and use of vaccines, diagnostics and drugs. InSeminars in immunopathology 2020 Jun (Vol. 42, No. 3, pp. 247-264). Berlin/Heidelberg: Springer Berlin Heidelberg.
78. Rassi Jr A, Rassi A, Rassi SG. Predictors of mortality in chronic Chagas disease: a systematic review of observational studies. Circulation. 2007 Mar 6;115(9):1101-8.
79. Maizels RM, Yazdanbakhsh M. Immune regulation by helminth parasites: cellular and molecular mechanisms. Nature Reviews Immunology. 2003 Sep 1;3(9):733-44.
80. World Health Organization. World malaria report 2022. World Health Organization; 2022 Dec 8.
81. Cowman AF, Healer J, Marapana D, Marsh K. Malaria: biology and disease. Cell. 2016 Oct 20;167(3):610-24.
82. White NJ. Malaria parasite clearance. Malaria journal. 2017 Feb 23;16(1):88.
83. SenGupta SB, Delhanty JD. Preimplantation genetic diagnosis: recent triumphs and remaining challenges. Expert Review of Molecular Diagnostics. 2012 Jul 1;12(6):585-92.

84. Chen L, Chen Y, Zhang D, Hou M, Yang B, Zhang F, Zhang W, Luo X, Ji M, Wu G. Protection and immunological study on two tetraspanin-derived vaccine candidates against schistosomiasis japonicum. Parasite immunology. 2016 Oct;38(10):589-98.
85. Nikulkova M, Abdrabou W, Carlton JM, Idaghdour Y. Exploiting integrative metabolomics to study host–parasite interactions in Plasmodium infections. Trends in parasitology. 2024 Apr 1;40(4):313-23.
86. Fairhurst RM, Dondorp AM. Artemisinin-resistant Plasmodium falciparum malaria. Microbiology spectrum. 2016 Jun 30;4(3):10-128.
87. Simner PJ, Miller S, Carroll KC. Understanding the promises and hurdles of metagenomic next-generation sequencing as a diagnostic tool for infectious diseases. Clinical Infectious Diseases. 2018 Feb 15;66(5):778-88.
88. Doolan DL, Hoffman SL. Multi-gene vaccination against malaria: a multistage, multi-immune response approach. Parasitology Today. 1997 May 1;13(5):171-8.
89. Gomez MA, Contreras I, Hallé M, Tremblay ML, McMaster RW, Olivier M. Leishmania GP63 alters host signaling through cleavage-activated protein tyrosine phosphatases. Science signaling. 2009 Sep 29;2(90):ra58-.
90. Rezende LG, Barros MD, Moreira OB, de Oliveira MA, Chellini PR, Jaeger LH. Analytical Approaches for Parasitic Biomarkers Diseases Discovery: Trends and Perspectives of Metabolomics in the Clinical Laboratory. Critical Reviews in Analytical Chemistry. 2025 Oct 31:1-7.
91. Tuteja R. Malaria− an overview. The FEBS journal. 2007 Sep;274(18):4670-9.
92. Hotez PJ, Brindley PJ, Bethony JM, King CH, Pearce EJ, Jacobson J. Helminth infections: the great neglected tropical diseases. The Journal of clinical investigation. 2008 Apr 1;118(4):1311-21.
93. Moorthy VS, Good MF, Hill AV. Malaria vaccine developments. The lancet. 2004 Jan 10;363(9403):150-6.
94. Mu Y, McManus DP, Gordon CA, Cai P. Parasitic helminth-derived microRNAs and extracellular vesicle cargos as biomarkers for helminthic infections. Frontiers in Cellular and Infection Microbiology. 2021 Jun 25;11:708952.
95. Trager W. Living together: the biology of animal parasitism. Springer Science & Business Media; 2012 Dec 6.
96. Jyotisha, Qureshi R, Qureshi IA. Development of a multi-epitope vaccine candidate for leishmanial parasites applying immunoinformatics and in vitro approaches. Frontiers in Immunology. 2023 Nov 15;14:1269774.
97. Sajid MS, Ghazanfer S, Rizwan HM, Bajwa MH, Anwar N, Saqib M. Introduction to Omics Technologies. InOmics Approaches in Veterinary Parasitology 2024 Nov 28 (pp. 22-46). CRC Press.
98. Freitas-Junior LH, Hernandez-Rivas R, Ralph SA, Montiel-Condado D, Ruvalcaba-Salazar OK, Rojas-Meza AP, Mâncio-Silva L, Leal-Silvestre RJ, Gontijo AM, Shorte S, Scherf A. Telomeric heterochromatin propagation and histone acetylation control mutually exclusive expression of antigenic variation genes in malaria parasites. Cell. 2005 Apr 8;121(1):25-36.
99. Maizels RM, Yazdanbakhsh M. Immune regulation by helminth parasites: cellular and molecular mechanisms. Nature Reviews Immunology. 2003 Sep 1;3(9):733-44.
100. Cowman AF, Healer J, Marapana D, Marsh K. Malaria: biology and disease. Cell. 2016 Oct 20;167(3):610-24.
101. White NJ. Severe malaria. Malaria journal. 2022 Oct 6;21(1):284.
102. Miller LH, Good MF, Milon G. Malaria pathogenesis. Science. 1994 Jun 24;264(5167):1878-83.
103. Colvin HN, Joice Cordy R. Insights into malaria pathogenesis gained from host metabolomics. PLoS Pathogens. 2020 Nov 12;16(11):e1008930.
104. Gomez MA, Contreras I, Hallé M, Tremblay ML, McMaster RW, Olivier M. Leishmania GP63 alters host signaling through cleavage-activated protein tyrosine phosphatases. Science signaling. 2009 Sep 29;2(90):ra58-.

105. Kaye P, Scott P. Leishmaniasis: complexity at the host–pathogen interface. Nature reviews microbiology. 2011 Aug;9(8):604-15.
106. Handman E, Bullen DV. Interaction of Leishmania with the host macrophage. Trends in parasitology. 2002 Aug 1;18(8):332-4.
107. Kennedy PG, Rodgers J. Clinical and neuropathogenetic aspects of human African trypanosomiasis. Frontiers in immunology. 2019 Jan 25;10:39.
108. Gupta S, Braun M, Tischler ND, Stoltz M, Sundström KB, Björkström NK, Ljunggren HG, Klingström J. Hantavirus-infection confers resistance to cytotoxic lymphocyte-mediated apoptosis. PLoS pathogens. 2013 Mar 28;9(3):e1003272.
109. Rassi Jr A, Rassi A, Marin-Neto JA. Chagas disease. InNeglected Tropical Diseases-Latin America and the Caribbean 2015 May 8 (pp. 45-71). Vienna: Springer Vienna.
110. Hotez PJ, Bethony JM, Diemert DJ, Pearson M, Loukas A. Developing vaccines to combat hookworm infection and intestinal schistosomiasis. Nature Reviews Microbiology. 2010 Nov;8(11):814-26.
111. McSorley HJ, Maizels RM. Helminth infections and host immune regulation. Clinical microbiology reviews. 2012 Oct;25(4):585-608.
112. MacDonald AS, Araujo MI, Pearce EJ. Immunology of parasitic helminth infections. Infection and immunity. 2002 Feb;70(2):427-33.
113. Nikulkova M, Abdrabou W, Carlton JM, Idaghdour Y. Exploiting integrative metabolomics to study host–parasite interactions in Plasmodium infections. Trends in parasitology. 2024 Apr 1;40(4):313-23.
114. Felipin KP, Paloschi MV, Silva MD, Ikenohuchi YJ, Santana HM, Setubal SD, Rego CM, Lopes JA, Boeno CN, Serrath SN, De Medeiros EH. Transcriptomics analysis highlights potential ways in human pathogenesis in Leishmania braziliensis infected with the viral endosymbiont LRV1. PLOS Neglected Tropical Diseases. 2024 May 14;18(5):e0012126.
115. Agbo EE, Majiwa PA, Claassen HJ, te Pas MF. Molecular variation of Trypanosoma brucei subspecies as revealed by AFLP fingerprinting. Parasitology. 2002 Apr;124(4):349-58.
116. Marcilla A, Sánchez-López CM, González-Arce A, Cortés A, Bernal D. Understanding the extracellular vesicles in helminths. InRecent Advances in Parasitomics: Implications for Parasite and Vector Research 2025 Jan 26 (pp. 247-272). Cham: Springer Nature Switzerland.
117. Simon S, Wagner MA, Rothmeier E, Müller-Taubenberger A, Hilbi H. Icm/Dot-dependent inhibition of phagocyte migration by L egionella is antagonized by a translocated Ran GTPase activator. Cellular microbiology. 2014 Jul;16(7):977-92.
118. O'Brien XM, Reichner JS. Neutrophil integrins and matrix ligands and NET release. Frontiers in immunology. 2016 Sep 19;7:363.
119. Oliveira CS, Negut I. Precision Medicine and Omics Approaches for Managing Infectious Diseases: Technologies, Applications, and Current Achievements. InAI and Precision Medicine in Infectious Disease Management 2025 Dec 2 (pp. 71-97). CRC Press.
120. Hildebrandt F, Iturritza MU, Zwicker C, Vanneste B, Van Hul N, Semle E, Quin J, Pascini T, Saarenpää S, He M, Andersson ER. Host-pathogen interactions in the Plasmodium-infected mouse liver at spatial and single-cell resolution. Nature Communications. 2024 Aug 19;15(1):7105.
121. Reid AJ, Talman AM, Bennett HM, Gomes AR, Sanders MJ, Illingworth CJ, Billker O, Berriman M, Lawniczak MK. Single-cell RNA-seq reveals hidden transcriptional variation in malaria parasites. elife. 2018 Mar 27;7:e33105.
122. Chahine Z, Le Roch KG. Decrypting the complexity of the human malaria parasite biology through systems biology approaches. Frontiers in systems biology. 2022 Sep 16;2:940321.
123. Bamarni SS, Said MB. Genomic approaches in parasitic diagnosis. InOmics Approaches in Veterinary Parasitology 2024 Nov 28 (pp. 47-61). CRC Press.
124. Gramberg S, Puckelwaldt O, Schmitt T, Lu Z, Haeberlein S. Spatial transcriptomics of a parasitic flatworm provides a molecular map of drug targets and drug resistance genes. Nature Communications. 2024 Oct 16;15(1):8918.

125. Hollin T, Chahine Z, Le Roch KG. Epigenetic regulation and chromatin remodeling in malaria parasites. Annual review of microbiology. 2023 Sep 15;77(1):255-76.
126. Ishibashi K, Mawatari N, Miyashita S, Kishino H, Meshi T, Ishikawa M. Coevolution and hierarchical interactions of Tomato mosaic virus and the resistance gene Tm-1.
127. Krishnan A, Kloehn J, Lunghi M, Chiappino-Pepe A, Waldman BS, Nicolas D, Varesio E, Hehl A, Lourido S, Hatzimanikatis V, Soldati-Favre D. Functional and computational genomics reveal unprecedented flexibility in stage-specific Toxoplasma metabolism. Cell host & microbe. 2020 Feb 12;27(2):290-306.
128. Stelzle D, Makasi CE, Schmidt V, Van Damme I, Trevisan C, Ruether C, Fleury A, Noh J, Handali S, Dorny P, Magnussen P. Evaluation of a point-of-care test for the diagnosis of Taenia solium neurocysticercosis in rural southern Tanzania: a diagnostic accuracy study. The Lancet Infectious Diseases. 2024 Jan 1;24(1):98-106.
129. Moses HL, Phillips JK, Graig LA, editors. Biomarker tests for molecularly targeted therapies: key to unlocking precision medicine.
130. Kafle A, Ojha SC. Advancing vaccine development against Opisthorchis viverrini: A synergistic integration of omics technologies and advanced computational tools. Frontiers in Pharmacology. 2024 Jul 15;15:1410453.
131. Stutzer C, Richards SA, Ferreira M, Baron S, Maritz-Olivier C. Metazoan parasite vaccines: present status and future prospects. Frontiers in cellular and infection microbiology. 2018 Mar 13;8:67.
132. Cui Y, Yu L. Application of the CRISPR/Cas9 gene editing technique to research on functional genomes of parasites. Parasitology international. 2016 Dec 1;65(6):641-4.
133. Yan YH. Multi-Omic and multi-scale data integration for the characterization of malaria infection in non-human primates.
134. Kang HJ, Chu KB, Lee SH, Kim MJ, Park H, Jin H, Moon EK, Quan FS. Toxoplasma gondii virus-like particle vaccination alleviates inflammatory response in the brain upon T gondii infection. Parasite Immunology. 2020 Jun;42(6):e12716.
135. Rao SP, Manjunatha UH, Mikolajczak S, Ashigbie PG, Diagana TT. Drug discovery for parasitic diseases: powered by technology, enabled by pharmacology, informed by clinical science. Trends in Parasitology. 2023 Apr 1;39(4):260-71.
136. Bartelt LA, Bolick DT, Mayneris-Perxachs J, Kolling GL, Medlock GL, Zaenker EI, Donowitz J, Thomas-Beckett RV, Rogala A, Carroll IM, Singer SM. Cross-modulation of pathogen-specific pathways enhances malnutrition during enteric co-infection with Giardia lamblia and enteroaggregative Escherichia coli. PLoS pathogens. 2017 Jul 27;13(7):e1006471.
137. Steiner S, Becker SC, Hartwig J, Sotzny F, Lorenz S, Bauer S, Löbel M, Stittrich AB, Grabowski P, Scheibenbogen C. Autoimmunity-related risk variants in PTPN22 and CTLA4 are associated with ME/CFS with infectious onset. Frontiers in immunology. 2020 Apr 9;11:578.
138. Ashraf MA, Mustafa BE, Wahaab A, Batool H, Ashraf M, Said MB, Sevinc F, Stevenson NJ. Conventional and molecular diagnosis of parasites. InParasitism and parasitic control in animals: strategies for the developing world 2023 Jul 10 (pp. 56-72). GB: CABI.
139. Panagiotou A. Single-cell transcriptomics of host-parasite interactions (Doctoral dissertation, Université Paris Cité).
140. Zhang Z, Guo D, Huber ME, Park SW, Sternad D. Exploiting the geometry of the solution space to reduce sensitivity to neuromotor noise. PLoS Computational Biology. 2018 Feb 20;14(2):e1006013.
141. Kang HJ, Chu KB, Lee SH, Kim MJ, Park H, Jin H, Moon EK, Quan FS. Toxoplasma gondii virus-like particle vaccination alleviates inflammatory response in the brain upon T gondii infection. Parasite Immunology. 2020 Jun;42(6):e12716.
142. Paredes-Santos T, Wang Y, Waldman B, Lourido S, Saeij JP. The GRA17 parasitophorous vacuole membrane permeability pore contributes to bradyzoite viability. Frontiers in cellular and infection microbiology. 2019 Sep 12;9:321.
143. Nikulkova M, Abdrabou W, Carlton JM, Idaghdour Y. Exploiting integrative metabolomics to study host–parasite interactions in Plasmodium infections. Trends in parasitology. 2024 Apr 1;40(4):313-23.

144. de Souza W, Barrias ES. Membrane-bound extracellular vesicles secreted by parasitic protozoa: cellular structures involved in the communication between cells. Parasitology research. 2020 Jul;119(7):2005-23.
145. Valigurová A, Kolářová I. Unrevealing the mystery of latent leishmaniasis: what cells can host leishmania?. Pathogens. 2023 Feb 3;12(2):246.
146. Yu X, Feng G, Zhang Q, Cao J. From metabolite to metabolome: Metabolomics applications in plasmodium research. Frontiers in Microbiology. 2021 Jan 11;11:626183.
147. MacDonald AS, Araujo MI, Pearce EJ. Immunology of parasitic helminth infections. Infection and immunity. 2002 Feb;70(2):427-33.
148. Lee HJ, Georgiadou A, Otto TD, Levin M, Coin LJ, Conway DJ, Cunnington AJ. Transcriptomic studies of malaria: a paradigm for investigation of systemic host-pathogen interactions. Microbiology and Molecular Biology Reviews. 2018 Jun;82(2):10-128.
149. Machado FS, Dutra WO, Esper L, Gollob KJ, Teixeira MM, Factor SM, Weiss LM, Nagajyothi F, Tanowitz HB, Garg NJ. Current understanding of immunity to Trypanosoma cruzi infection and pathogenesis of Chagas disease. InSeminars in immunopathology 2012 Nov (Vol. 34, No. 6, pp. 753-770). Berlin/Heidelberg: Springer-Verlag.
150. World Health Organization. World malaria report 2021. World Health Organization; 2021 Dec 6.
151. Alvar J, Vélez ID, Bern C, Herrero M, Desjeux P, Cano J, Jannin J, Boer MD, WHO Leishmaniasis Control Team. Leishmaniasis worldwide and global estimates of its incidence. PloS one. 2012 May 31;7(5):e35671.
152. Ospina-Villa JD, Cisneros-Sarabia A, Sánchez-Jiménez MM, Marchat LA. Current advances in the development of diagnostic tests based on aptamers in parasitology: a systematic review. Pharmaceutics. 2020 Oct 31;12(11):1046.
153. Minghui Z, Kunhua H, Yunwen B, Hongmei L, Jing L, Shaowen W, Longqiaozi S, Chaohui D. Analysis of differentially expressed proteins involved in autoimmune cirrhosis and normal serum by iTRAQ Proteomics. PROTEOMICS–Clinical Applications. 2019 May;13(3):1700153.
154. Kioko M, Pance A, Mwangi S, Goulding D, Kemp A, Rono M, Ochola-Oyier LI, Bull PC, Bejon P, Rayner JC, Abdi AI. Extracellular vesicles could be a putative posttranscriptional regulatory mechanism that shapes intracellular RNA levels in Plasmodium falciparum. Nature Communications. 2023 Oct 13;14(1):6447.
155. Lustigman S, Geldhof P, Grant WN, Osei-Atweneboana MY, Sripa B, Basanez MG. A research agenda for helminth diseases of humans: basic research and enabling technologies to support control and elimination of helminthiases. PLoS neglected tropical diseases. 2012 Apr 24;6(4):e1445.
156. Cortopassi WA, Celmar Costa Franca T, Krettli AU. A systems biology approach to antimalarial drug discovery. Expert Opinion on Drug Discovery. 2018 Jul 3;13(7):617-26.
157. Coakley G, McCaskill JL, Borger JG, Simbari F, Robertson E, Millar M, Harcus Y, McSorley HJ, Maizels RM, Buck AH. Extracellular vesicles from a helminth parasite suppress macrophage activation and constitute an effective vaccine for protective immunity. Cell reports. 2017 May 23;19(8):1545-57.
158. Howick VM, Russell AJ, Andrews T, Heaton H, Reid AJ, Natarajan K, Butungi H, Metcalf T, Verzier LH, Rayner JC, Berriman M. The Malaria Cell Atlas: Single parasite transcriptomes across the complete Plasmodium life cycle. Science. 2019 Aug 23;365(6455):eaaw2619.
159. Oliveira TG, Venturini G, Alvim JM, Feijó LL, Dinardo CL, Sabino EC, Seidman JG, Seidman CE, Krieger JE, Pereira AC. Different transcriptomic response to T. cruzi infection in hiPSC-derived cardiomyocytes from chagas disease patients with and without chronic cardiomyopathy. Frontiers in cellular and infection microbiology. 2022 Jul 7;12:904747.
160. Rodriguez-Cruz A, Vesin D, Ramon-Luing L, Zuñiga J, Quesniaux VF, Ryffel B, Lascurain R, Garcia I, Chávez-Galán L. CD3+ macrophages deliver proinflammatory cytokines by a CD3-and transmembrane TNF-dependent pathway and are increased at the BCG-infection site. Frontiers in immunology. 2019 Nov 7;10:2550.

161. Diray-Arce J, Conti MG, Petrova B, Kanarek N, Angelidou A, Levy O. Integrative metabolomics to identify molecular signatures of responses to vaccines and infections. Metabolites. 2020 Nov 30;10(12):492.
162. Mattoso-Barbosa AM, Sathler-Avelar R, Coelho-dos-Reis JG, Martins-Filho OA, Teixeira-Carvalho A, Vitelli-Avelar DM. Physiology and Pathology of Infectious Diseases: The Autoimmune Hypothesis of Chagas Disease. InPhysiology and Pathology of Immunology 2017 Dec 20. IntechOpen.

