NIU Journal of Health Sciences Copyright©2026
Nexus International University ISSN: 3136-6449; 12(3) 171-180

Systemic Physiological Consequences of Human Parasitic Diseases: Mechanisms of Hematological, Immunological, Oxidative, Endocrine, and Multiorgan Dysfunction: A Comprehensive Review

SUHAD SHAHEED ALI
Department of Pathological Analysis, College of Science, Wasit University, Iraq
Suhadshaheed@uowasit.edu.iq

DONIA FADHEL ABASS
Department of Biology, College of Science, University of Wasit, Iraq
Donia.fadhel@uowasit.edu.iq

Abstract. Human parasitic infections damage various physiological systems by direct invasion of tissue, damage to cells, immune-mediated inflammation, loss of nutrients and dysregulation of metabolism. Clinical manifestations vary depending on the protozoa, helminths, and ectoparasites, which have different life cycles, tissue tropism, and pathogenic mechanisms. This review aims to bring together the knowledge about the hematological, immunological, gastrointestinal, nutritional, hepatic, renal, cardiovascular, pulmonary, neurological, endocrine and metabolic effects of human parasitic diseases. Malaria may cause anemia by any of the following mechanisms: destruction of erythrocytes, splenic clearance and impaired erythropoiesis; hookworm can cause anemia due to chronic loss of blood from the gut; and visceral leishmaniasis may cause anemia through reticuloendothelial involvement and hypersplenism. Helminths typically induce type 2 immune responses and regulatory pathways, while intracellular protozoa require good cell-mediated immunity. Intestinal protozoa may cause disruption of epithelial barrier, nutrient absorption and micro biome interactions. Specific complications are schistosomal hepatic fibrosis, parasitic renal injury, Chagas cardiomyopathy, cerebral malaria and neurocysticercosis. There are other mechanisms of infection-tissue damage interaction, such as oxidative stress and changes in antioxidant defenses. The review highlights the importance of considering physiological biomarkers in the context of the species, burden, host, inflammation, nutrition and comorbidities. The integrated approach to diagnosis, applying both parasite-specific testing and haematological and biochemical assessment, can complement the clinical interpretation and patient management.

Keywords: Human parasitic infections; pathophysiology; hematology; immunology; iron metabolism; oxidative stress; endocrine function; organ dysfunction; host–parasite interactions.

1. Introduction

Parasitic diseases are still a significant cause of morbidity globally and are caused by a variety of different organisms, such as protozoa, helminths and ectoparasites. The clinical effects of their interaction with the host are not limited to the site of infection: the survival of blood cells, immune regulation, function of epithelial cells, and absorption of nutrients, metabolism and physiology of organs can be affected by the interaction between host and parasite. The impact and clinical manifestation is dependent on the species of the parasite, the level of infection, its duration, the age of the host, nutrition, immune competence, and availability of diagnosis and treatment (Bethony et al., 2006; Burza et al., 2018; Colley et al., 2014; Jourdan et al., 2018).

Hematological effects include hemolytic and iron-deficiency anemia, eosinophilia, leukopenia and thrombocytopenia. The two pathogens, malaria and hookworm, stimulate the destruction of infected erythrocytes, and in the case of

malaria, the clearance of uninfected erythrocytes, and also cause chronic blood loss in the intestine. Pancytopenia can be seen in visceral leishmaniasis, due to splenic and bone marrow involvement (Camaschella, 2015; White et al., 2014).

Immune responses vary from protective cell mediated responses against helminths and type 2 responses to regulatory responses found with helminths and intracellular pathogens. Intestinal parasites can disrupt the integrity of the intestinal epithelium and nutrient absorption, some species can lead to hepatic, renal, cardiovascular, pulmonary or neurological disease (Allain & Buret, 2020; Daher et al., 2022; Loke et al., 2022; Matta et al., 2021).

The systemic physiological consequences of human parasitic infection are discussed and the mechanism of relationships between parasite biology and clinical and laboratory abnormalities.

2. Review Methodology

The article has been organized as a narrative review. Relevant topics of literature covered were parasite classification, host–parasite interactions, hematological and immune effects, gastrointestinal and nutritional physiology, hepatic and renal complications, cardiovascular and respiratory manifestations, neurological disease, endocrine and metabolic responses, oxidative stress, and laboratory assessment. Peer-reviewed reviews, clinical investigations, large disease reviews, and authoritative World Health Organization guidance were the primary sources that were given priority in the source framework. Publications which had laid the ground work were included if they had created mechanisms that are still utilized in contemporary interpretation. This is not a systematic review or meta-analysis registered in the PROSPERO database; hence, there are no cumulative or synthesized effect estimates or formal risk-of-bias analysis. Concepts addressed in the search were human parasitic infection, pathophysiology, anemia, iron metabolism, immune response, intestinal epithelial dysfunction, organ complications, oxidative stress and metabolic regulation.

3. Classification and Biological Characteristics of Human Parasites

3.1 Protozoa

Protozoa are unicellular eukaryotes, which invade the intestinal tract, blood, tissues and central nervous system. Plasmodium spp., Toxoplasma gondii, Leishmania spp., Giardia duodenalis, Cryptosporidium spp., Entamoeba histolytica and Trypanosoma spp. are important human pathogens. They have been shown to have several pathogenic mechanisms, including intracellular replication, erythrocyte invasion, damage to the epithelial barrier, modulation of the immune system, and tissue destruction (Matta et al., 2021; White et al., 2014; Burza et al., 2018).

3.2 Helminths

Helminths are multicellular parasites which are categorised in 3 groups: nematodes, cestodes and trematodes. Ascaris lumbricoides, Trichuris trichiura, hookworms, Strongyloides stercoralis, Taenia solium, Echinococcus spp. and Schistosoma spp. are examples. Exposure to antigens over the long-term and tissue migration can result in type 2 immune responses, eosinophilia, tissue remodeling, blood loss or organ fibrosis, in some infections (Bethony et al., 2006; Colley et al., 2014; Jourdan et al., 2018).

3.3 Ectoparasites

Lice, mites and ticks live on the outside of the body. They can cause blood loss, secondary infection, inflammatory and allergic reactions, and skin irritation, and in certain situations, the spread of other pathogens to the host.

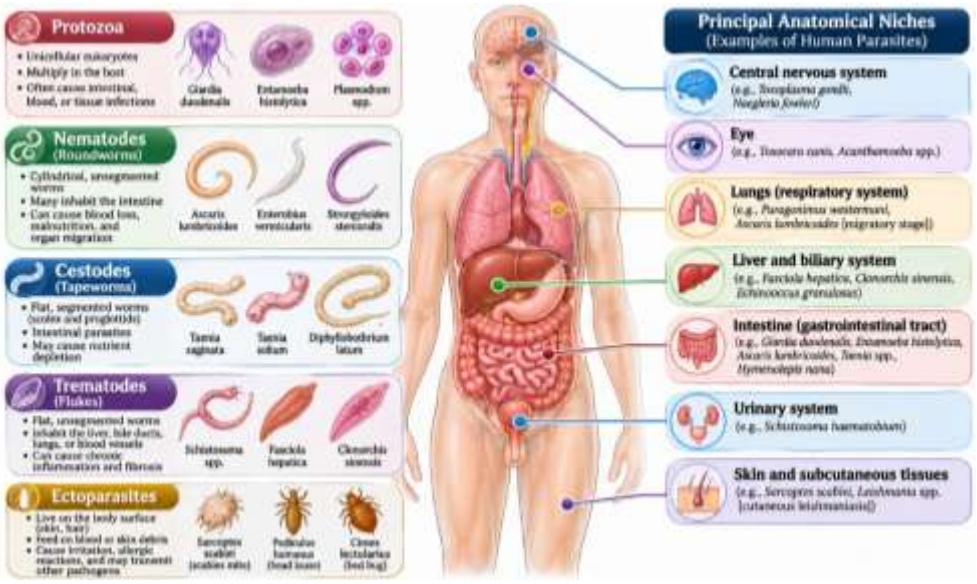


Figure 1: Major groups of human parasites and their principal anatomical niches. Schematic illustration prepared for this review.

4. Host–Parasite Interactions and Physiological Mechanisms

Disease is a result of interaction between Virulence factors of parasite and host defence mechanism. The parasites: attach or invade tissues; obtain nutrition; evade recognition by immune system; cause acute or chronic infection. Host responses consist of epithelial barriers, activation of complement, phagocytosis, antigen presentation, release of cytokines and adaptive immunity. These responses can help to control infection, but can also lead to tissue injury if there is too much or too long inflammation (Douglas et al. 2021; Loke et al. 2022; Maizels 2020).
Physiological pathways that are major include cellular injury, inflammatory signaling, oxidative stress, alteration in hematopoiesis, nutrient depletion, epithelial dysfunction, immune regulation, and/or organ-specific pathology.

5. Hematological and Hematopoietic Alterations

5.1 Anemia and Erythrocyte Dysfunction

Many of the parasitic diseases are associated with anemia. Anemia in malaria occurs due to the destruction of infected erythrocytes, clearance of uninfected erythrocytes, and sequestration of erythrocytes in the spleen and the disruption of erythropoiesis by inflammation. In chronic intestinal blood loss due to Hookworm infection, iron is lost from the body. Schistosomiasis has the potential to add to this impact, because of chronic blood loss, inflammation and nutritional impairment (Bethony et al., 2006; Colley et al., 2014; Smith & Brooker, 2010; White et al., 2014).

5.2 Red-Cell Indices and Iron Status

The hemoglobin, mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC) and red-cell distribution width (RDW) all help to characterise anemia. MCV and MCH are typically decreased by iron deficiency and can be elevated by it; RDW is typically elevated by iron deficiency though this finding is not specific to parasites. Ferritin should be considered in the context of inflammatory markers, since it is an acute-phase reactant, with serum iron, transferrin saturation and total iron-binding capacity, which yield complementary information (Camaschella 2015; WHO, 2020).

5.3 Leukocytes and Platelets

Eosinophilia is linked to helminths with tissue invasion as well as type 2 activation of the immune system. In both malaria and visceral leishmaniasis, thrombocytopenia is due to a variety of mechanisms such as sequestration in the

spleen, immune-mediated destruction, inflammation and altered production. Abnormalities of leukocytes vary with the different parasites and different stages of the disease.

Table 1: Major Hematological Mechanisms and Laboratory Abnormalities Associated with Parasitic Diseases

Parasite/disease	Principal hematological mechanism	Common laboratory pattern
Malaria	Erythrocyte destruction, splenic clearance, impaired erythropoiesis	Anemia; thrombocytopenia may occur
Hookworm	Chronic intestinal blood loss	Iron-deficiency anemia
Visceral leishmaniasis	Splenic and marrow involvement	Anemia, leukopenia, thrombocytopenia
Tissue-invasive helminths	Type 2 immune activation	Eosinophilia
Schistosomiasis	Chronic blood loss/inflammation in selected disease forms	Anemia may occur

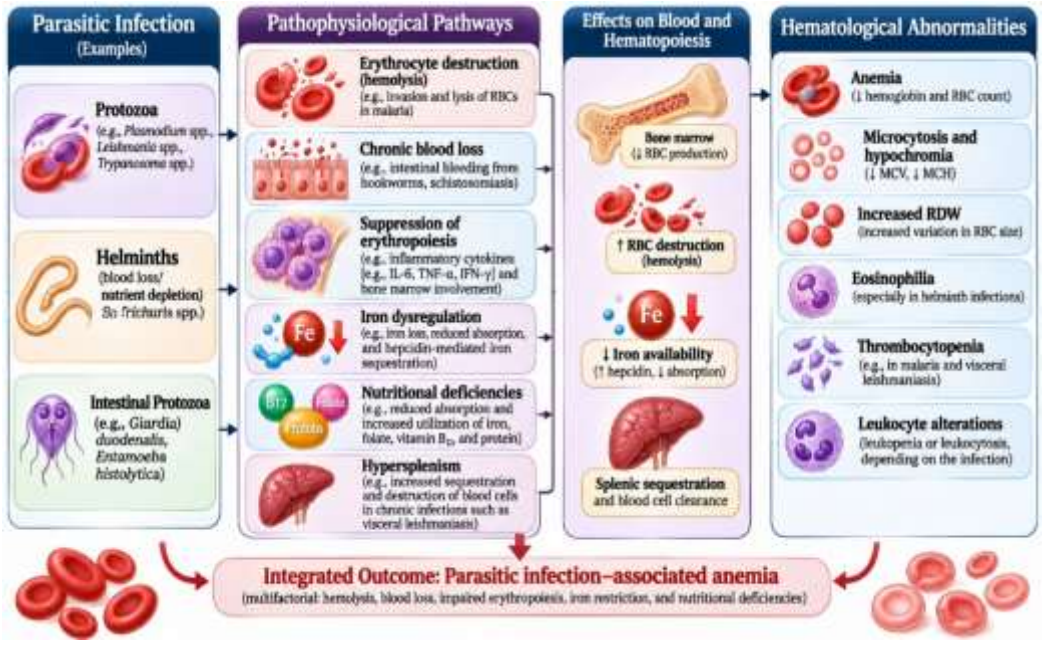


***Figure* 2:** *Major pathways linking parasitic infection with hematological abnormalities.*

6. Immunological and Inflammatory Responses

6.1 Innate Immunity

Early-stage parasite recognition by epithelial cells, macrophages, dendritic cells, natural killer cells and complement components. Derived molecules from parasites activate inflammatory signaling and the production of cytokines that depend on the location of the tissue and the biology of the parasite.

6.2 Adaptive Immunity

CMI, including interferon-gamma-associated pathways is generally required for intracellular protozoa. Type 2 responses, mediated by interleukins (IL) 4, 5, 13, eosinophils, mast cells and immunoglobulin E (IgE) are often associated with helminth infections. The pathways are involved in parasite control, production of mucus, repair of tissues and regulation of immune responses (Loke et al., 2022; Maizels, 2020).

6.3 Immune Regulation and Chronic Infection

Regulatory T cells, regulatory cytokines and alternatively activated macrophages may help to regulate tissue damaging inflammation, and facilitate the persistence of parasites. A key aspect of chronic helminth infections is the balance between protective immunity and immunopathology.

7. Gastrointestinal and Nutritional Physiology

7.1 Epithelial Dysfunction

Giardia duodenalis and Cryptosporidium spp. affect the barrier function, the structure of the microvilli and the signalling in the mucosa. Diarrhea and post-infectious gastrointestinal complications (Allain & Buret, 2020; Buret, 2007) have been associated with giardiasis. The invasion of the epithelium and mucosal immune responses are involved in Cryptosporidium infection (Pardy et al., 2024).

7.2 Nutrient Absorption and Growth

Infection of the intestine may lead to decreased intake, diarrhea, malabsorption, loss of blood and nutritional deficiencies. Hookworm is especially significant with regard to iron loss and giardiasis could affect intestinal function. Other factors that affect nutritional outcomes include nutrition, sanitation, co-infection and socioeconomic status (Bethony et al., 2006; Jourdan et al., 2018).

7.3 Microbiome Interactions

The intestinal microbiome is involved in mucosal immunity, intestinal metabolism, and intestinal homeostasis. The impact of helminths on microbe communities and immune responses may vary depending on host diet, geography, infection status and the microbial community (Loke et al., 2022).

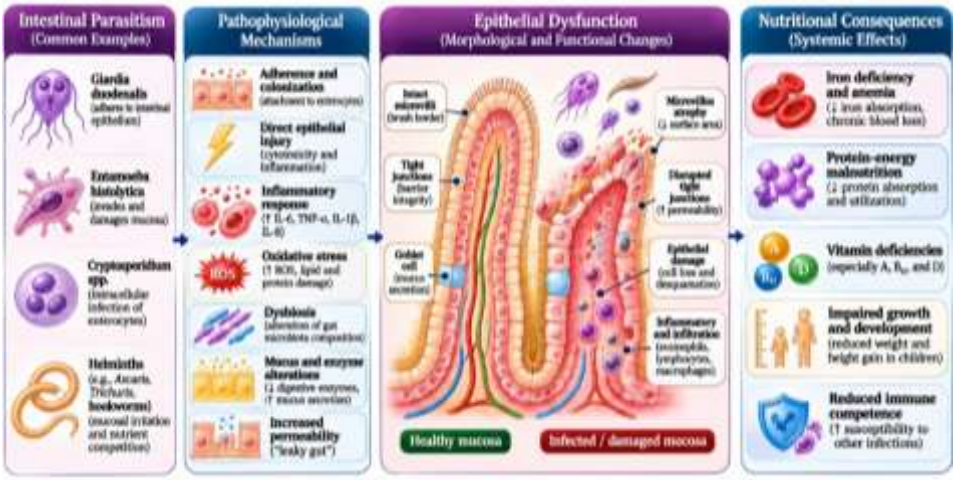


***Figure* 3:** *Potential pathways from intestinal parasitism to epithelial dysfunction and nutritional consequences.*

8. Hepatic and Renal Physiological Alterations

8.1 Liver and Spleen

Schistosomiasis may cause granulomatous inflammation and periportal fibrosis in the liver around eggs, which can then cause portal hypertension and hepatosplenic disease. Visceral leishmaniasis is a disease of the reticuloendothelial system and can lead to hepatosplenomegaly and cytopenias. Interpretation of liver enzymes, bilirubin and albumin is dependent on disease stage and disease organ involvement (Colley et al., 2014; Burza et al., 2018).

8.2 Kidney

Immune-complex glomerular injury, vascular damage, tubular damage and altered renal perfusion are the parasitic renal complications. Acute or chronic renal manifestations may occur with malaria, schistosomiasis and certain systemic parasitic infections. Routine assessments include urinalysis, creatinine and estimated glomerular filtration rate (GFR), and measurements of urinary protein (Daher et al., 2022).

9. Cardiovascular and Respiratory Effects

9.1 Cardiovascular System

The protozoan T. cruzi may develop into chronic inflammatory cardiomyopathy in the heart of a Chagas patient. Clinical disease is caused by myocardial inflammation, injury of cardiomyocytes, fibrosis, conduction abnormalities and dysfunction of the ventricles. The pathogenesis involves multiple factors such as parasite persistence, immune responses of the host and microvascular injury (Bonney et al., 2019; Rassi et al., 2010).

9.2 Respiratory System

In some helminth infections (during larval migration), and in heavy strongyloidiasis, the lungs are involved. In certain patients, schistosomiasis can result in pulmonary vascular disease and pulmonary hypertension. Parasite burden and life cycle, as well as the immune status of the host, are important factors to determine respiratory involvement (Colley et al., 2014; Jourdan et al., 2018).

10. Neurological and Neurophysiological Effects

Neurological parasitic disease may be caused by direct invasion, vascular occlusion, inflammation, edema and immunological damage to the tissues. Cerebral malaria is characterized by sequestration of infected erythrocytes in microvessels of the brain, activation of the endothelial cells and disruption of microvascular function. Variations in location and inflammatory reaction of the cysts may lead to neurocysticercosis, which may result in seizures, hydrocephalus and focal deficits. Encephalitis is one of the clinical manifestations of toxoplasmosis (Garcia, 2021; Matta et al., 2021; White et al., 2014), especially in immunocompromised individuals.

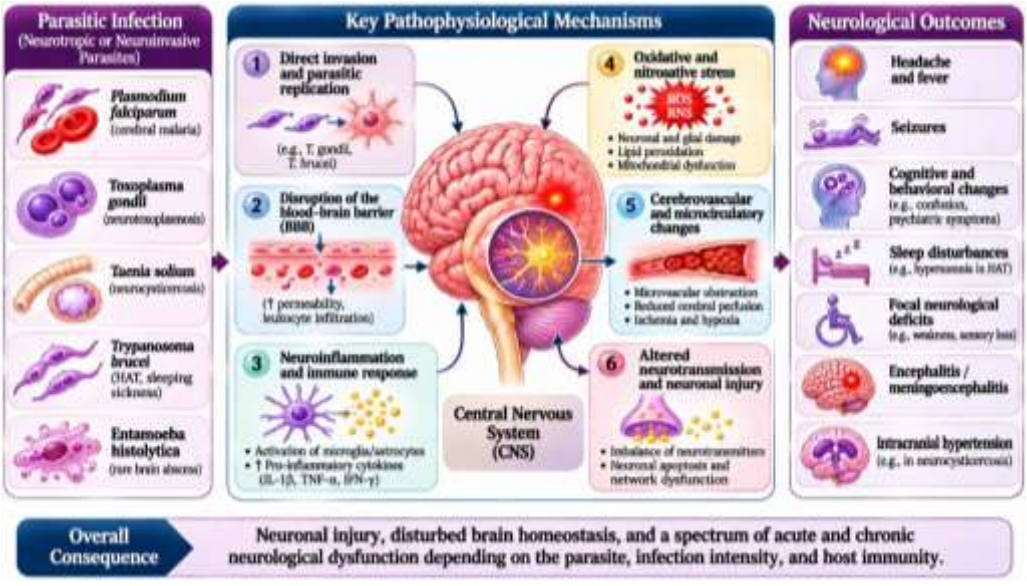


***Figure* 4:** *Simplified mechanisms connecting parasitic infection with neurological dysfunction.*

11. Endocrine and Metabolic Alterations

Systemic infection and inflammation can affect the regulation of the endocrine system and metabolism through the effects of cytokines, stress, nutritional shifts, and organ involvement. Cortisol is responsible for the physiological adaptation by regulating glucose metabolism and immune activity, which is mediated by the hypothalamic–pituitary–adrenal axis. But endocrine abnormalities in this context are not as well established as in other diseases and cautions should be used in interpreting the evidence.

The research that takes place in helminth-associated immune regulation has found its way into the field of metabolism, such as insulin sensitivity and inflammatory pathways. These observations do not prove any causal relationship between helminth infection and diabetes nor suggest that helminth infection can be a therapeutic target. In addition to modifying the metabolic pathways of the host cells to enhance their ability to replicate, parasites also modify the metabolism of the host cell; this, however, may not always lead to an overall metabolic disease in the host.

12. Oxidative Stress and Antioxidant Defense

Reactive oxygen and nitrogen species (ROS and RNS) play a role in antimicrobial defence and cell signalling. Overproduction of oxidants can cause damage to lipids, proteins, DNA and membranes. Immune-cell activation, mitochondrial effects, parasite metabolism, and tissue injury can alter the redox balance due to parasite infection. The research biomarkers include malondialdehyde, superoxide dismutase, catalase, glutathione peroxidase, reduced glutathione and total antioxidant capacity. They are direction and magnitude dependent on the infection stage and the disease and diagnostic oxidative markers are not present.

13. Clinical Biomarkers and Laboratory Assessment

Table 2: Laboratory Biomarkers and Diagnostic Tests Used in the Multidimensional Assessment of Parasitic Infections

Domain	Representative biomarkers/tests	Interpretive relevance
Parasitology	Microscopy, antigen tests, serology, PCR	Species-specific detection and infection assessment
Hematology	Hb, RBC, WBC, platelets, MCV, RDW	Anemia, cytopenias, eosinophilia
Iron metabolism	Ferritin, serum iron, TIBC, transferrin saturation	Iron stores and availability; interpret ferritin with inflammation
Inflammation	CRP, ESR; selected cytokines in research	Systemic inflammatory activity
Hepatic	ALT, AST, ALP, bilirubin, albumin	Hepatocellular/cholestatic patterns and synthetic function
Renal	Creatinine, eGFR, urea, urinalysis, urine protein	Filtration and renal injury
Oxidative stress	MDA, SOD, CAT, GPx, GSH, TAC	Research assessment of redox balance
Endocrine/metabolic	Glucose, insulin, cortisol, TSH, FT4	Context-dependent metabolic and endocrine assessment

No single biomarker indicates the presence, severity or physiological impact of any of the parasitic infections. Interpretation should be based on reference intervals appropriate to each species, age and clinical context, and also take into account the possibility of inflammation, nutritional and co-morbidity factors. Ferritin and hemoglobin levels are recommended to be interpreted using WHO guidance which offers standard ways of interpreting these parameters among relevant populations (WHO, 2020, 2024).

14. Therapeutic Implications and Future Perspectives

The parasite is, of course, an important part of the management because the disease is determined by the type of parasite involved, its severity and whether or not the organs are involved and the characteristics of the host. Treatment with an antiparasitic medicine may require treatment of nutritional deficiencies, blood counts and biochemical abnormalities, and renal/hepatic/cardiac/neurological complications be monitored. The sanitation element and safe water, food hygiene, vector control and health education are components of prevention. A better understanding of host–parasite mechanisms through molecular diagnostics, metabolomics and proteomics, and longitudinal clinical studies may help to better understand and interpret biomarker messages.

15. Research Gaps and Limitations

The physiological reactions vary widely between species of parasites, the amount of infection, the stage of disease and the host. A few mechanistic pathways are only supported by models of experiments and not large clinical studies of human subjects. The challenge of attribution of the physiological changes to a specific parasite is made more difficult by the co-existence of malnutrition, inflammation and multiple infections. The evidence for endocrine and/or long-term metabolic effects is not as consistent as the evidence for malaria associated anemia, hookworm associated iron loss, schistosomal organ pathology, and Chagas cardiomyopathy. This is not a registered systematic search, quantitative risk of bias assessment or meta-analysis.

16. Conclusion

Human parasitic infections have an effect on physiology by directly injuring cells, triggering immune-mediated inflammation, causing the depletion of nutrients, metabolic dysfunction and organ specific pathogenesis. The hematological abnormalities, immune responses, gastrointestinal dysfunction, hepatic and renal complications, cardiovascular and neurological manifestations and oxidative stress are interrelated aspects of the host–parasite interactions. The integrated parasitological diagnosis and physiological assessment proves to be useful to understand the clinical consequences and to provide patient care. More human research is needed with standardized protocols and longitudinal assessment of biomarkers to better understand the impact of individual parasites and longer-term consequences.

References

Allain, T., & Buret, A. G. (2020). Pathogenesis and post-infectious complications in giardiasis. Advances in Parasitology, 107, 173–199. https://doi.org/10.1016/bs.apar.2019.12.001

Allen, J. E., & Maizels, R. M. (2011). Diversity and dialogue in immunity to helminths. Nature Reviews Immunology, 11, 375–388. https://doi.org/10.1038/nri2992

Bethony, J., Brooker, S., Albonico, M., Geiger, S. M., Loukas, A., Diemert, D., & Hotez, P. J. (2006). Soil-transmitted helminth infections: Ascariasis, trichuriasis, and hookworm. The Lancet, 367(9521), 1521–1532. https://doi.org/10.1016/S0140-6736(06)68653-4

Black, R. E., Victora, C. G., Walker, S. P., Bhutta, Z. A., Christian, P., de Onis, M., Ezzati, M., Grantham-McGregor, S., Katz, J., Martorell, R., & Uauy, R. (2013). Maternal and child undernutrition and overweight in low-income and middle-income countries. The Lancet, 382(9890), 427–451. https://doi.org/10.1016/S0140-6736(13)60937-X

Bonney, K. M., Luthringer, D. J., Kim, S. A., Garg, N. J., & Engman, D. M. (2019). Pathology and pathogenesis of Chagas heart disease. Annual Review of Pathology, 14, 421–447. https://doi.org/10.1146/annurev-pathol-020117-043711

Brooker, S., Clements, A. C. A., & Bundy, D. A. P. (2006). Global epidemiology, ecology and control of soil-transmitted helminth infections. Advances in Parasitology, 62, 221–261. https://doi.org/10.1016/S0065-308X(05)62007-6

Burza, S., Croft, S. L., & Boelaert, M. (2018). Leishmaniasis. The Lancet, 392(10151), 951–970. https://doi.org/10.1016/S0140-6736(18)31204-2

Camaschella, C. (2015). Iron-deficiency anemia. The New England Journal of Medicine, 372, 1832–1843. https://doi.org/10.1056/NEJMra1401038

Colley, D. G., Bustinduy, A. L., Secor, W. E., & King, C. H. (2014). Human schistosomiasis. The Lancet, 383(9936), 2253–2264. https://doi.org/10.1016/S0140-6736(13)61949-2

Daher, E. D. F., da Silva Junior, G. B., Trivedi, M., et al. (2022). Kidney complications of parasitic diseases. Nature Reviews Nephrology, 18, 396–406. https://doi.org/10.1038/s41581-022-00558-z

Dewey, K. G., & Mayers, D. R. (2011). Early child growth: How do nutrition and infection interact? Maternal & Child Nutrition, 7(Suppl. 3), 129–142. https://doi.org/10.1111/j.1740-8709.2011.00357.x

Douglas, B., Oyesola, O., Cooper, M. M., Posey, A., & Wojno, E. T. (2021). Immune system investigation using parasitic helminths. Annual Review of Immunology, 39, 639–665. https://doi.org/10.1146/annurev-immunol-093019-122827

Garcia, H. H. (2021). Parasitic infections of the nervous system. Continuum, 27(4), 943–962.

Ganz, T., & Nemeth, E. (2012). Hepcidin and iron homeostasis. Biochimica et Biophysica Acta, 1823(9), 1434–1443. https://doi.org/10.1016/j.bbamcr.2012.01.014

Holmes, P. H. (1987). Pathophysiology of parasitic infections. Parasitology, 94(Suppl.), S29–S51. https://doi.org/10.1017/S0031182000085814

Jourdan, P. M., Lamberton, P. H. L., Fenwick, A., & Addiss, D. G. (2018). Soil-transmitted helminth infections. The Lancet, 391(10117), 252–265. https://doi.org/10.1016/S0140-6736(17)31930-X
Kennedy, P. G. E. (2013). Clinical features, diagnosis, and treatment of human African trypanosomiasis (sleeping sickness). The Lancet Neurology, 12(2), 186–194. https://doi.org/10.1016/S1474-4422(12)70296-X
Kotloff, K. L., et al. (2013). Burden and aetiology of diarrhoeal disease in infants and young children in developing countries (GEMS): A prospective, case-control study. The Lancet, 382(9888), 209–222. https://doi.org/10.1016/S0140-6736(13)60844-2
Loke, P., Lee, S. C., & Oyesola, O. O. (2022). Effects of helminths on the human immune response and the microbiome. Mucosal Immunology, 15, 1224–1233. https://doi.org/10.1038/s41385-022-00532-9
Maizels, R. M. (2020). Regulation of immunity and allergy by helminth parasites. Allergy, 75(3), 524–534.
Matta, S. K., Rinkenberger, N., Dunay, I. R., & Sibley, L. D. (2021). Toxoplasma gondii infection and its implications within the central nervous system. Nature Reviews Microbiology, 19, 467–480.
Miller, L. H., Baruch, D. I., Marsh, K., & Doumbo, O. K. (2002). The pathogenic basis of malaria. Nature, 415, 673–679. https://doi.org/10.1038/415673a
Montoya, J. G., & Liesenfeld, O. (2004). Toxoplasmosis. The Lancet, 363(9425), 1965–1976. https://doi.org/10.1016/S0140-6736(04)16412-X
Muhsen, K., & Levine, M. M. (2012). A systematic review and meta-analysis of the association between Giardia lamblia and endemic pediatric diarrhea in developing countries. Clinical Infectious Diseases, 55(Suppl. 4), S271–S293. https://doi.org/10.1093/cid/cis762
Pardy, R. D., Wallbank, B. A., Striepen, B., & Hunter, C. A. (2024). Immunity to Cryptosporidium: Insights into principles of enteric responses to infection. Nature Reviews Immunology, 24, 142–155.
Platts-Mills, J. A., et al. (2015). Pathogen-specific burdens of community diarrhoea in developing countries: A multisite birth cohort study (MAL-ED). The Lancet Global Health, 3(9), e564–e575.
Pullan, R. L., Smith, J. L., Jasrasaria, R., & Brooker, S. J. (2014). Global numbers of infection and disease burden of soil-transmitted helminth infections in 2010. Parasites & Vectors, 7, 37. https://doi.org/10.1186/1756-3305-7-37
Rassi, A., Rassi, A., & Marin-Neto, J. A. (2010). Chagas disease. The Lancet, 375(9723), 1388–1402. https://doi.org/10.1016/S0140-6736(10)60061-X
Rogawski, E. T., et al. (2018). Use of quantitative molecular diagnostic methods to investigate the effect of enteropathogen infections on linear growth in children in low-resource settings: Longitudinal analysis of results from the MAL-ED cohort study. The Lancet Global Health, 6(12), e1319–e1328.
Smith, J. L., & Brooker, S. (2010). Impact of hookworm infection and deworming on anaemia in non-pregnant populations: A systematic review. Tropical Medicine & International Health, 15(7), 776–795. https://doi.org/10.1111/j.1365-3156.2010.02542.x
Torgerson, P. R., et al. (2015). World Health Organization estimates of the global and regional disease burden of 11 foodborne parasitic diseases, 2010: A data synthesis. PLOS Medicine, 12(12), e1001920.
Weiss, G., & Goodnough, L. T. (2005). Anemia of chronic disease. The New England Journal of Medicine, 352, 1011–1023. https://doi.org/10.1056/NEJMra041809
White, N. J., Pukrittayakamee, S., Hien, T. T., Faiz, M. A., Mokuolu, O. A., & Dondorp, A. M. (2014). Malaria. The Lancet, 383(9918), 723–735. https://doi.org/10.1016/S0140-6736(13)60024-0
World Health Organization. (2020). WHO guideline on use of ferritin concentrations to assess iron status in individuals and populations. https://www.who.int/publications/i/item/9789240000124
World Health Organization. (2024). Guideline on haemoglobin cutoffs to define anaemia in individuals and populations. https://www.who.int/publications/i/item/9789240088542
World Health Organization. (2024). World malaria report 2024. World Health Organization.
World Health Organization. (2023). Soil-transmitted helminth infections. https://www.who.int/news-room/fact-sheets/detail/soil-transmitted-helminth-infections
World Health Organization. (2023). Schistosomiasis. World Health Organization.
World Health Organization. (2023). Leishmaniasis. World Health Organization.
World Health Organization. (2024). Chagas disease (American trypanosomiasis). World Health Organization.
Allain, T., & Buret, A. G. (2017). Giardia duodenalis: Current understanding of the host–parasite interaction. Current Tropical Medicine Reports, 4, 1–8.
Albonico, M., Allen, H., Chitsulo, L., Engels, D., Gabrielli, A. F., & Savioli, L. (2008). Controlling soil-transmitted helminthiasis in pre-school-age children through preventive chemotherapy. PLoS Neglected Tropical Diseases, 2(3), e126.
Anthony, R. M., Rutitzky, L. I., Urban, J. F., Stadecker, M. J., & Gause, W. C. (2007). Protective immune mechanisms in helminth infection. Nature Reviews Immunology, 7, 975–987.

Artis, D., & Grencis, R. K. (2008). The intestinal epithelium: Sensors to effectors in nematode infection. Mucosal Immunology, 1, 252–264.
Barrett, J. (2009). Forty years of helminth biochemistry. Parasitology, 136, 1633–1642.
Blackwell, J. M., Gosset, K., & Wyllie, S. (2020). The host response to Leishmania infection. Current Opinion in Immunology, 66, 1–8.
Burke, M. L., Jones, M. K., Gobert, G. N., Li, Y. S., Ellis, M. K., & McManus, D. P. (2009). Immunopathogenesis of human schistosomiasis. Parasite Immunology, 31, 163–176.
Chitsulo, L., Engels, D., Montresor, A., & Savioli, L. (2000). The global status of schistosomiasis and its control. Acta Tropica, 77, 41–51.
Coakley, G., Maizels, R. M., & Buck, A. H. (2016). Exosomes and other extracellular vesicles: The new communicators in parasite infections. Trends in Parasitology, 32, 688–697.
Fried, B., & Reddy, A. (2011). The role of parasites in human disease. Clinical Microbiology Reviews, 24, 1–24.
Geiger, S. M., & Fujiwara, R. T. (2016). Immunology of human helminth infections. Current Opinion in Immunology, 42, 1–7.
Maizels, R. M., & McSorley, H. J. (2016). Regulation of the host immune system by helminth parasites. Journal of Allergy and Clinical Immunology, 138, 666–675.
McSorley, H. J., & Maizels, R. M. (2012). Helminth infections and host immune regulation. Clinical Microbiology Reviews, 25(4), 585–608.
Nutman, T. B. (2015). Human infection with Strongyloides stercoralis and other related Strongyloides species. Parasitology, 142, 1–14.
Okhuysen, P. C., & Chappell, C. L. (2002). Cryptosporidium and Giardia: Practical approaches to diagnosis and treatment. Infectious Disease Clinics of North America, 16, 1–17.
Pearce, E. J., & MacDonald, A. S. (2002). The immunobiology of schistosomiasis. Nature Reviews Immunology, 2, 499–511.
Roberts, L. S., Janovy, J., & Nadler, S. (2013). Foundations of parasitology (9th ed.). McGraw-Hill.
Ryan, U., & Hijjawi, N. (2015). New developments in Cryptosporidium research. International Journal for Parasitology, 45, 367–373.
Stensvold, C. R., & van der Giezen, M. (2018). Associations between gut microbiota and common luminal intestinal parasites. Trends in Parasitology, 34, 369–377.
World Health Organization. (2022). Ending the neglect to attain the Sustainable Development Goals: A road map for neglected tropical diseases 2021–2030. World Health Organization.