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Human Immune Responses to Parasitic Infections: A Review

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Abstract

Parasitic infections remain a major public health challenge worldwide, particularly in tropical and subtropical regions where poor sanitation, inadequate healthcare facilities and unfavourable socioeconomic conditions facilitate parasite transmission. This review examines human immune responses to parasitic infections, with emphasis on the major components of the immune system, cellular and humoral immunity, cytokines, and immune responses to specific parasitic diseases. The review highlights the roles of physical and chemical barriers, macrophages, neutrophils, eosinophils, natural killer cells and dendritic cells in innate immunity. It further discusses adaptive immune responses mediated by T-helper 1 (Th1), T-helper 2 (Th2), T-helper 17 (Th17), regulatory T cells, $CD8^+$ cytotoxic T cells and B lymphocytes. Particular attention is given to the roles of cytokines, including interleukin-4 (IL-4), IL-5, IL-10, IL-12, IL-13, tumour necrosis factor-alpha (TNF-α) and interferon-gamma (IFN-γ), in regulating parasite clearance, inflammation and immune protection. Furthermore, the review examines immune responses associated with malaria, leishmaniasis, schistosomiasis, intestinal helminthiasis and toxoplasmosis. Findings from the reviewed literature indicate that intracellular protozoan parasites generally stimulate Th1-mediated immune responses involving IFN-γ and activated macrophages, whereas helminth infections predominantly induce Th2-mediated responses involving eosinophils, mast cells, immunoglobulin E (IgE) and alternatively activated macrophages. However, parasites have developed sophisticated immune-evasion and immunoregulatory mechanisms that enable their persistence within human hosts. The review concludes that effective immunity against parasitic infections depends on the coordinated interaction between innate and adaptive immune responses and the regulation of inflammatory processes.

Keywords: Adaptive immunity, innate immunity, cytokines, immune responses, parasitic infections

1.0 Introduction

Parasitic infections remain a major public health challenge worldwide, particularly in tropical and subtropical regions where environmental conditions, poor sanitation, inadequate access to healthcare and socioeconomic factors facilitate the transmission of parasitic organisms. Parasites are eukaryotic organisms that depend on their hosts for survival,

nutrition and reproduction. They include protozoa, helminths and ectoparasites, all of which possess different biological characteristics and mechanisms of interaction with the human immune system (Alghanmi et al., 2024).

The human immune system has evolved a complex network of cellular and molecular mechanisms to recognize, control and eliminate parasitic organisms. These mechanisms involve both innate and adaptive immunity, which work together to provide immediate defence, develop antigen-specific responses and establish immunological memory. However, unlike many bacterial and viral infections, parasitic infections frequently involve large organisms, complex developmental stages, prolonged tissue residence and sophisticated immune-evasion strategies (Lekki-Jóźwiak and Bąska, 2024).

The nature of the immune response depends largely on the type of parasite, its location within the host, the stage of infection and the immune status of the individual. For instance, intracellular protozoa such as Leishmania and Toxoplasma gondii generally stimulate cell-mediated immune responses involving T-helper 1 (Th1) cells and activated macrophages. In contrast, helminths such as Ascaris lumbricoides, Schistosoma species and hookworms typically induce T-helper 2 (Th2)-mediated responses involving eosinophils, mast cells, immunoglobulin E (IgE) and alternatively activated macrophages (Loke et al., 2022).

Nevertheless, immune responses to parasites are not always completely protective. Some parasites can alter antigen expression, interfere with antigen presentation, inhibit immune-cell functions and induce regulatory pathways that suppress host immunity. These mechanisms enable parasite survival and may contribute to chronic infection, recurrent disease and tissue damage. Understanding human immune responses to parasitic infections is therefore essential in immunology, parasitology, infectious disease management, vaccine development and the design of novel immunotherapeutic interventions (Maizels and Gause, 2023).

2.0 Classification of Parasites in Relation to Host Immune Responses

Parasites can be classified according to their biological characteristics, morphology, life cycles and interactions with the human host. Their classification is important because different parasite groups activate distinct immune pathways (Zaiss et al., 2024).

2.1 Protozoan parasites

Protozoa are unicellular eukaryotic organisms capable of infecting different tissues and organ systems. They may exist extracellularly, intracellularly or in both forms during their life cycles. Important human protozoan parasites include Plasmodium (species, which cause malaria), Entamoeba histolytica, (which causes amoebiasis), Giardia duodenalis, (which causes giardiasis), Leishmania (species, which cause leishmaniasis), Trypanosoma (species, which cause trypanosomiasis), Toxoplasma gondii, (which causes toxoplasmosis) and Cryptosporidium (species, which cause cryptosporidiosis) (Zaiss et al., 2024)

Protozoan infections stimulate diverse immune responses. Intracellular protozoa usually activate macrophages, dendritic cells, natural killer cells and T lymphocytes. These immune cells produce cytokines, including interferon-gamma (IFN-γ), tumour necrosis factor-alpha (TNF-α) and interleukin-12 (IL-12), which contribute to parasite control. Extracellular protozoa, particularly those inhabiting the intestinal lumen, are frequently controlled through mucosal immunity, antibodies, epithelial defence mechanisms and innate immune responses (Serrano-Coll, 2026).

2.2 Helminth parasites

Helminths are multicellular parasitic worms that include nematodes, cestodes and trematodes. They are responsible for several neglected tropical diseases and may cause chronic infections associated with malnutrition, anaemia, organ damage and impaired development. Major helminth groups include Nematodes (roundworms) such as Ascaris lumbricoides, Trichuris trichiura, Ancylostoma duodenale, Necator americanus Strongyloides stercoralis and Wuchereria bancrofti. Cestodes (tapeworms) including Taenia solium, Taenia saginata and Echinococcus granulosus

and Trematodes (flukes) such as Schistosoma haematobium, Schistosoma mansoni and Fasciola hepatica (Murphy and Weaver, 2022). Helminth infections are particularly associated with type 2 immune responses. These responses involve Th2 lymphocytes; group 2 innate lymphoid cells, eosinophils, mast cells, basophils, IgE and alternatively activated macrophages. Helminths can also induce regulatory T cells and anti-inflammatory cytokines, such as IL-10 and transforming growth factor-beta (TGF-β), which limit excessive inflammation but may facilitate parasite persistence (Punt et al., 2022).

2.3 Ectoparasites

Ectoparasites are organisms that live on the external surfaces of their hosts or temporarily feed on them. Examples include lice, ticks, mites and fleas. The immune responses to ectoparasites involve the skin's physical barrier, keratinocytes, dendritic cells, mast cells, eosinophils, antibodies and inflammatory cytokines. In some infestations, such as scabies caused by Sarcoptes scabiei, immune-mediated inflammation contributes substantially to itching, skin lesions and secondary bacterial infections (Droghini et al., 2026).

3.0 Components of the Human Immune System Involved in Parasitic Infections

The immune system consists of interconnected cells, tissues, organs and soluble molecules that protect the host against infectious agents. Its major components are innate and adaptive immunity.

3.1 Innate immune responses

Innate immunity represents the first line of defence against parasitic organisms. It provides rapid, relatively nonspecific responses and influences the development of subsequent adaptive immunity. The innate immune system recognizes parasite-derived molecules through pattern-recognition receptors (PRRs), including Toll-like receptors (TLRs), C-type lectin receptors and NOD-like receptors. Activation of these receptors initiates intracellular signalling pathways that stimulate cytokine production, inflammatory responses and immune-cell recruitment (Maizels et al., 2018). Major components of innate immunity against parasites include:

3.1.1 Physical and chemical barriers

The skin and mucosal surfaces constitute important protective barriers against parasite invasion. The epidermis provides a physical barrier against ectoparasites and infective larval stages, while mucus and epithelial secretions help prevent parasite attachment and facilitate the removal of organisms from mucosal surfaces. Gastrointestinal epithelial cells produce antimicrobial peptides, mucus and other protective molecules. Intestinal peristalsis and epithelial turnover also contribute to parasite elimination. During helminth infections, epithelial cells release cytokines such as IL-25, IL-33 and thymic stromal lymphopoietin (TSLP). These molecules activate type 2 immune pathways and promote the recruitment and activation of immune cells involved in worm expulsion (Maizels et al., 2018).

3.1.2 Macrophages

Macrophages are important phagocytic cells that participate in the recognition, ingestion and destruction of parasites. They originate from circulating monocytes or tissue-resident macrophage populations. During protozoan infections, macrophages may become activated by IFN-γ and microbial signals. Classical macrophage activation enhances the production of reactive oxygen species, nitric oxide and inflammatory mediators that contribute to intracellular parasite destruction (Zaiss et al., 2024).

For example, during Leishmania infection, activated macrophages can restrict parasite multiplication through nitric oxide-dependent mechanisms. However, Leishmania species have developed mechanisms that interfere with phagolysosomal function and macrophage activation, enabling their intracellular survival. During helminth infections, macrophages can undergo alternative activation under the influence of IL-4 and IL-13. Alternatively activated macrophages participate in tissue repair, regulation of inflammation and responses to parasite-associated tissue injury (Serrano-Coll, 2026).

3.1.3 Neutrophils

Neutrophils are among the earliest leukocytes recruited to sites of infection. They migrate toward inflammatory signals and can destroy pathogens through phagocytosis, degranulation, reactive oxygen species production and the release of neutrophil extracellular traps. Neutrophils participate in immune responses against several protozoan parasites, including Leishmania and Toxoplasma gondii. Their functions may be protective or harmful, depending on the parasite and the stage of infection. In some infections, neutrophils can also provide an intracellular environment that supports parasite survival or contributes to excessive inflammation (Serrano-Coll, 2026).

3.1.4 Eosinophils

Eosinophils are granulocytes strongly associated with immune responses to helminth infections. Their recruitment and development are promoted primarily by IL-5 and chemotactic factors such as eotaxins. Eosinophils express receptors for antibodies and can recognize antibody-coated parasites. Upon activation, they release granule proteins, including major basic protein, eosinophil cationic protein and eosinophil peroxidase. These molecules may damage the surface structures of parasites, particularly larval stages. However, eosinophils can also contribute to inflammatory tissue injury, especially during prolonged or intense infections (Zaiss et al., 2024).

3.1.5 Natural killer cells

Natural killer (NK) cells are innate lymphocytes involved in the early defence against intracellular pathogens. They recognize changes in the expression of host-cell surface molecules and respond to cytokines released during infection. During infections such as toxoplasmosis and malaria, NK cells can produce IFN-γ and TNF-α, which help activate macrophages and influence adaptive immune responses. NK-cell activity is particularly important during early infection, before fully developed parasite-specific T-cell responses become established (Serrano-Coll, 2026).

3.1.6 Dendritic cells

Dendritic cells are professional antigen-presenting cells that connect innate and adaptive immunity. They capture parasite antigens, process them and present antigenic peptides to T lymphocytes through major histocompatibility complex (MHC) molecules. Dendritic cells also produce cytokines that influence T-cell differentiation. For example, IL-12 production promotes Th1 responses, whereas the cytokine environment and tissue-derived signals can support the development of type 2 immunity. Some parasites interfere with dendritic-cell maturation, antigen presentation and cytokine production. These changes can impair the development of effective adaptive immunity (Droghini et al., 2026).

4.0 Adaptive Immune Responses to Parasitic Infections

Adaptive immunity provides antigen-specific protection against parasites through the activities of T lymphocytes, B lymphocytes and antibodies. Unlike innate immunity, adaptive immune responses are characterized by antigen specificity, clonal expansion and the potential to develop immunological memory. The major adaptive immune mechanisms involved in parasitic infections include cell-mediated immunity, humoral immunity and regulatory immune responses (Zaiss et al., 2024).

4.1 T-lymphocyte-mediated immunity

T lymphocytes play essential roles in the recognition and elimination of parasitic organisms. They are broadly classified into $CD4^+$ helper T cells and $CD8^+$ cytotoxic T cells. $CD4^+$ T cells differentiate into distinct functional subsets depending on the cytokine environment and the signals received during antigen presentation. These subsets include Th1, Th2, Th17, follicular helper T cells (Tfh) and regulatory T cells (Tregs) (Droghini et al., 2026).

4.1.1 T-helper 1 (Th1) responses

Th1 cells are particularly important in the control of intracellular protozoan parasites. They develop under the influence of IL-12 and other differentiation signals and produce IFN-γ, TNF-α and IL-2. IFN-γ activates macrophages and enhances their antimicrobial activities, including the production of reactive oxygen intermediates and nitric oxide. These

mechanisms contribute to the destruction or restriction of intracellular parasites. Th1 responses are particularly important in infections caused by Leishmania species and Toxoplasma gondii. In leishmaniasis, effective Th1 responses are generally associated with improved macrophage-mediated parasite control, although the relationship between immune response and disease outcome varies with parasite species and clinical form. Excessive Th1 activity, however, may cause tissue damage through sustained inflammatory cytokine production. Therefore, effective protection requires a balance between parasite elimination and the regulation of inflammatory responses (Serrano-Coll, 2026).

4.1.2 T-helper 2 (Th2) responses

Th2 cells are central to immunity against helminth parasites. They differentiate in response to an appropriate cytokine environment, particularly IL-4, and produce IL-4, IL-5, IL-9 and IL-13.

These cytokines coordinate several protective mechanisms:

- IL-4 promotes Th2 differentiation, B-cell class switching to IgE and alternative macrophage activation.
- IL-5 stimulates eosinophil development, maturation, recruitment and activation.
- IL-9 supports mast-cell responses and contributes to mucosal defence.
- IL-13 stimulates mucus production, epithelial changes, intestinal motility and tissue repair.

The combined activities of these cytokines facilitate the expulsion of intestinal worms and contribute to the containment of tissue-dwelling helminths. Th2-mediated responses are not uniformly protective against every helminth. Some parasites persist despite strong type 2 immunity, and prolonged responses may cause allergic inflammation, fibrosis or other forms of tissue pathology. Recent research emphasizes the coordinated functions of innate and adaptive type 2 immune cells in parasite expulsion and tissue repair (Maizels & Gause, 2023).

4.1.3 T-helper 17 (Th17) responses

Th17 cells are characterized by the production of IL-17A, IL-17F and IL-22. These cytokines stimulate epithelial defence mechanisms, recruit neutrophils and promote inflammation at mucosal surfaces. Th17 responses can contribute to host defence against certain intestinal protozoan parasites and influence inflammatory responses during other parasitic infections. However, their functions vary considerably depending on the parasite species and infection site. In some infections, excessive IL-17-mediated inflammation may contribute to tissue injury rather than parasite clearance (Serrano-Coll, 2026).

4.1.4 Follicular helper T cells

Follicular helper T cells are specialized $CD4^+$ T lymphocytes that regulate B-cell responses within germinal centres of secondary lymphoid organs. They express molecules such as CXCR5, PD-1 and ICOS and produce IL-21, which supports B-cell proliferation, antibody affinity maturation, class switching and the generation of memory B cells. During parasitic infections, Tfh cells help coordinate the production of parasite-specific antibodies. Their activity is important in malaria, where antibodies directed against different parasite stages contribute to protection against infection and disease (Serrano-Coll, 2026).

4.1.5 Regulatory T cells

Regulatory T cells are essential for maintaining immune tolerance and preventing excessive inflammation. Most classical Tregs express CD4, CD25 and the transcription factor FOXP3. Tregs suppress immune responses through several mechanisms, including the production of IL-10 and TGF-β, consumption of IL-2 and interactions with antigen-presenting cells. During chronic parasitic infections, Tregs can limit immunopathology and preserve tissue integrity. However, excessive regulatory activity may suppress parasite-specific effector responses and facilitate persistent infection (Droghini et al., 2026).

4.2 $CD8^+$ cytotoxic T-cell responses

$CD8^+$ T lymphocytes recognize parasite-derived peptides presented by MHC class I molecules on infected host cells. Following activation, they differentiate into cytotoxic effector cells capable of releasing perforin and granzymes. These molecules can induce apoptosis in infected cells and help control intracellular pathogens. $CD8^+$ T cells are particularly important in certain stages of malaria infection. During the liver stage, Plasmodium parasites develop inside hepatocytes. Parasite-specific $CD8^+$ T cells can recognize infected hepatocytes and contribute to the elimination of liver-stage infection. $CD8^+$ T cells also contribute to immune responses against Toxoplasma gondii and other intracellular protozoa. Their protective effectiveness depends on antigen recognition, tissue localization, cytokine production and the ability to maintain functional activity during persistent infection (Droghini et al., 2026).

4.3 Humoral immune responses

Humoral immunity involves B lymphocytes and the antibodies they produce. It is particularly important in the control of extracellular parasites, the neutralization of parasite-derived molecules and the prevention of reinfection. Following antigen recognition and appropriate T-cell help, B lymphocytes undergo clonal expansion and differentiate into antibody-secreting plasma cells or memory B cells. The major antibody classes involved in parasitic infections are as follows.

Immunoglobulin M (IgM): IgM is usually among the earliest antibodies produced during primary immune responses. Its pentameric structure enables effective antigen binding and complement activation.

Immunoglobulin G (IgG): IgG is important in systemic immunity against parasites. It can neutralize parasite antigens, promote opsonization and mediate antibody-dependent cellular cytotoxicity.

Immunoglobulin A (IgA): IgA plays a major role in mucosal immunity. Secretory IgA can prevent parasite attachment to epithelial surfaces and interfere with parasite invasion.

Immunoglobulin E (IgE): IgE is particularly associated with helminth immunity and allergic responses. It binds to high-affinity FcεRI receptors on mast cells and basophils, facilitating their activation following antigen exposure (Serrano-Coll, 2026).

Although parasite-specific antibodies are important in protective immunity, their effectiveness differs considerably between infections. Some parasites modify surface antigens, hide within host tissues or produce molecules that interfere with antibody-mediated destruction (Droghini et al., 2026).

5.0 Major Cytokines Involved in Immune Responses to Parasites

Cytokines are small signalling proteins that regulate communication among immune cells. They control immune-cell differentiation, activation, recruitment and effector functions during parasitic infections.

Cytokine	Major source	Role in parasitic immunity
IL-1β	Macrophages and other innate cells	Promotes inflammation and immune-cell recruitment
IL-4	Th2 cells, ILC2s and other cells	Promotes Th2 differentiation, IgE production and alternative macrophage activation
IL-5	Th2 cells and ILC2s	Stimulates eosinophil production and activation
IL-6	Macrophages and other cells	Promotes inflammatory responses and influences T-cell differentiation

IL-10	Tregs, macrophages and other cells	Suppresses excessive inflammation and regulates effector responses
IL-12	Dendritic cells and macrophages	Promotes Th1 differentiation and IFN-γ production
IL-13	Th2 cells and ILC2s	Enhances mucus production, parasite expulsion and tissue repair
IL-17	Th17 cells and other lymphocytes	Promotes neutrophil recruitment and mucosal inflammation
IL-21	Tfh cells and other activated T cells	Supports B-cell responses and antibody production
IL-25	Epithelial cells and other sources	Initiates and amplifies type 2 immunity
IL-33	Epithelial and stromal cells	Activates ILC2s and promotes type 2 inflammation
IFN-γ	Th1 cells, NK c ells and $CD8^+$ T cells	Activates macrophages and supports control of intracellular parasites
TNF-α	Macrophages and activated lymphocytes	Promotes inflammation and contributes to parasite control
TGF-β	Tregs, macrophages and other cells	Regulates immune responses and supports tissue repair

The balance between pro-inflammatory and anti-inflammatory cytokines influences whether an infection is controlled, persists chronically or produces severe tissue damage (Douglas et al., 2021).

6.0 Immune Responses to Specific Parasitic Infections

6.1 Immune response to malaria

Malaria is caused by protozoan parasites of the genus Plasmodium, which are transmitted through the bites of infected female Anopheles mosquitoes. Important human malaria parasites include Plasmodium falciparum, P. vivax, P. malariae, P. ovale and P. knowlesi. The immune response to malaria is complex because the parasite undergoes different developmental stages in the mosquito vector and human host. Following inoculation, sporozoites enter the bloodstream and migrate to the liver, where they invade hepatocytes and develop into liver-stage parasites. Merozoites subsequently enter the bloodstream and invade red blood cells.

Innate immune cells recognize parasite-derived molecules and initiate inflammatory responses. Monocytes, macrophages, dendritic cells and NK cells participate in the early recognition and control of infection. Parasite nucleic acids and other components activate innate signalling pathways, leading to the production of cytokines and chemokines. IFN-γ and TNF-α contribute to parasite control but may also participate in the development of severe inflammatory complications (Boyle et al., 2024).

Antibody-mediated immunity is particularly important during the blood stage of malaria. Antibodies directed against merozoite surface proteins and other parasite antigens can interfere with red blood cell invasion and promote the removal of infected erythrocytes. $CD4^+$ T cells support antibody production and coordinate cellular responses. $CD8^+$ T cells are especially important during the liver stage, where they can recognize and destroy infected hepatocytes. Repeated exposure to malaria may result in partial clinical immunity, particularly in individuals living in endemic areas. This immunity can reduce the risk of severe disease without necessarily preventing infection (Daily and Parikh, 2025).

Malaria induces regulatory mechanisms involving IL-10, regulatory T cells and other immune pathways. These mechanisms may limit excessive inflammation but can also weaken parasite-clearing responses. In severe P. falciparum malaria, sequestration of infected erythrocytes in small blood vessels and dysregulated inflammatory responses contribute to organ dysfunction. The clinical consequences depend on parasite burden, host immunity, age and other risk factors (Duffy et al., 2024).

6.2 Immune response to leishmaniasis

Leishmaniasis is caused by protozoan parasites of the genus Leishmania, which are transmitted through the bites of infected phlebotomine sand flies. Following transmission, parasites are taken up by macrophages and other phagocytic cells, where they develop into intracellular amastigotes. The development of effective Th1 responses is an important component of protective immunity against many forms of leishmaniasis. IL-12 stimulates Th1 differentiation, while IFN-γ activates macrophages to increase their intracellular parasite-killing activity. TNF-α may act synergistically with IFN-γ to enhance macrophage activation and inflammatory defence. Antibodies are produced during leishmaniasis, but their protective contribution varies according to the parasite species and clinical form. In some settings, high antibody levels do not correspond to effective parasite clearance. IL-10-producing regulatory cells and alternatively activated macrophages may contribute to parasite persistence by suppressing macrophage microbicidal activity. The outcome of infection reflects the balance between parasite replication, cellular immunity, inflammatory injury and immune regulation (Li et al., 2025).

6.3 Immune response to schistosomiasis

Schistosomiasis is a chronic parasitic disease caused by blood flukes of the genus Schistosoma. Human infection occurs when cercariae penetrate the skin during contact with contaminated freshwater. The parasites mature into adult worms within the vascular system. Female worms release eggs that become trapped in tissues, particularly the intestinal wall and liver in intestinal schistosomiasis. Schistosome eggs stimulate strong type 2 immune responses involving IL-4, IL-5 and IL-13. Eosinophils, macrophages, mast cells and other immune cells accumulate around deposited eggs. Granuloma formation represents an important host response to parasite eggs. Granulomas help contain parasite-derived antigens and limit their immediate effects on surrounding tissues. Although granuloma formation can protect surrounding tissues, prolonged inflammation may contribute to fibrosis and organ dysfunction. In hepatosplenic schistosomiasis, chronic egg-induced inflammation can result in periportal fibrosis, portal hypertension and splenomegaly. Regulatory mechanisms involving IL-10, Tregs and other immune populations influence granuloma size and the severity of tissue injury (Moroz et al., 2026).

6.4 Immune response to intestinal helminths

Intestinal helminths such as Ascaris lumbricoides, Trichuris trichiura and hookworms stimulate immune responses involving epithelial cells, innate lymphoid cells, Th2 lymphocytes, eosinophils, mast cells and antibodies. When helminths invade or migrate through tissues, epithelial damage and parasite-derived molecules stimulate the release of IL-25, IL-33 and TSLP. These cytokines activate ILC2s, which produce IL-5 and IL-13. These mediators promote eosinophil recruitment, mucus secretion, and epithelial changes and enhanced intestinal motility. Goblet cells increase mucus production, while smooth-muscle contraction and changes in epithelial physiology facilitate the expulsion of intestinal worms. Antibodies, especially IgE and parasite-specific IgG, can also contribute to immune recognition and effector mechanisms. However, the protective response differs among intestinal helminths. Some species establish long-term infections despite sustained Th2 responses. In addition, parasite-induced changes in intestinal immunity and the gut microbiome may influence susceptibility to reinfection and the severity of disease (Kasal et al., 2024).

6.5 Immune response to toxoplasmosis

Toxoplasmosis is caused by Toxoplasma gondii, an intracellular protozoan parasite capable of infecting humans and many warm-blooded animals. Infection may occur through ingestion of contaminated food or water, exposure to tissue cysts in undercooked meat or congenital transmission. The immune response to T. gondii is predominantly cell-mediated, with Th1 responses playing a central role in parasite control. Dendritic cells and macrophages produce IL-12 following recognition of parasite components. IL-12 stimulates NK cells and T lymphocytes to produce IFN-γ, which

activates intracellular antimicrobial mechanisms and restricts parasite replication. $CD8^+$ T cells contribute through cytotoxic activity and IFN-γ production, while $CD4^+$ T cells coordinate and sustain protective immunity. However, T. gondii can establish persistent infection by forming tissue cysts, particularly in the brain and skeletal muscles. Regulatory immune mechanisms are essential for limiting inflammatory tissue damage while maintaining sufficient parasite control (Hunter and Sibley, 2012).

7.0 Conclusion

Human immune responses to parasitic infections involve complex interactions between innate immunity, adaptive immunity, inflammatory mediators and regulatory mechanisms. The type and effectiveness of the response depend on parasite species, developmental stage, tissue location, infection intensity and host characteristics. Innate immunity provides the initial defence through epithelial barriers, phagocytic cells, natural killer cells, dendritic cells and inflammatory signalling. Adaptive immunity provides antigen-specific protection through Th1, Th2 and other T-cell subsets, antibody production and immunological memory. Intracellular protozoan parasites generally stimulate Th1-mediated responses involving IFN-γ and activated macrophages, whereas helminths characteristically induce Th2-mediated immunity involving IL-4, IL-5, IL-13, eosinophils, mast cells and IgE. Nevertheless, these response patterns are not absolute, and many parasitic infections involve multiple overlapping immune pathways. Parasites have evolved sophisticated mechanisms of immune evasion and immunoregulation that enable prolonged survival within human hosts. Although regulatory immune responses help prevent excessive inflammation and tissue injury, they may also permit parasite persistence.

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