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A Review about The Relationship Between *Toxoplasmosis* and Some Immunological Indicators

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ABSTRACT

Background: *Toxoplasma gondii is an intracellular pathogen that elicits evolution of compound immune responses involving the innate and adaptive immune systems. Toll-like receptors (TLRs) stimulate innate cells (dendritic cells and macrophages) at the early stages in the infection cascade, resulting in the production of IL-12, which is required for IFN-gamma production by T cells and natural killer (NK) cells for the control of the parasites. Interleukin-6 (IL-6) is also secreted during the early stages of the infection process; its overexpression is linked to tissue destruction. While an anti-inflammatory cytokine (IL-10) produced by regulatory T cells (Tregs) and some macrophages suppresses excessive immune response and enables the parasite to evade immune responses during chronic infection, IL-17 induces an inflammatory response and upregulation of this cytokine during acute infection is associated with tissue inflammation. TNF- a is one of the most remarkable necrosis factors found in the infection condition. Although it contributes to the enhancement of the immune cell response, it is responsible for an increase in chronic inflammation, which explains why the parasite is able to evade more complex immunodefense mechanisms through the first line of defense against of T. gondii.*

Keywords: Toxoplasmosis, Immunological indicators, Toxoplasma gondii.

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INTRODUCTION

Toxoplasmosis is an infection that is common in animals and humans and is caused by a parasite known as *Toxoplasma gondii* which infects various mammals and birds. It is a rigid intracellular parasite (Hotea & Daruabas G., 2023). The definitive host of the parasite is cats and the parasite sexual stage entails the incorporation into the intestinal cell epithelium of the definitive host (Sena *et al*,2023). While humans, some mammals, *Toxoplasma gondii* is ubiquitous in nations but the rate of infection differs based on food and social customs and the character of the housing, ambient and age of the host (David *et al*,2023). Adults who are infected with this parasite usually show no sign of the infection. Nevertheless, the clinical symptoms are inadequate, so the definitive diagnosis of *Toxoplasma gondii* infection is based on histological, immunological and molecular methods in women, especially pregnant women, who can contact the infection and can cause health problems, in particular, mental retardation, microcephaly, blindness, hydrops and the death of the fetus (Jeske *et al*,2023). *Toxoplasma gondii* is contracted in two ways: firstly, by ingesting food and water contaminated with cat feces Or ingestion of unpasteurized milk and its products (Almashhadany, *et al*, 2024), and secondly,

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through blood and organ transfusion (Assoni, *et al*, 2024). The second transmission route of the Toxoplasma parasite is the congenital route between the infected mother and the fetus through the placenta (Caceres *et al*, 2024).

Adaptive immune response by way of the humoral immune response which consists of IgG, M, A, and E has the ability to clear the free parasite in body fluid with the assistance of complement or by the cellular one in B cells, monocytes, macrophages, killer cells and others (Dals & Muhammad,2024). Given the importance of the topic and its relationship to reproduction and women's health, especially, the current review aimed to collect information on the relationship between infection with the Toxoplasma gondii parasite and some immune indicators.

Immunity

The adaptive immune response and the innate immune mechanism are significant in the control of toxoplasmosis infection as toxoplasmosis infection causes the activation of two types of immune response, that is, the humoral immune response and the cell-mediated immune response (Al-Hadraawy,2024).

Innate immunity against the parasite *Toxoplasma gondii*

The capability to invade the numerous types of the cells and tissues can be utilized in defining the danger of the *Toxoplasma gondii* parasite and those cells attached to the central nervous system that coincidentally happens to be the eye. The initial response that is occurring when the parasite is introduced into the body is the natural immune system i.e. the immune cells are aware that there is something which belongs to the parasite, through the pattern recognition receptor i.e. TLR. TLR 11 and TLR 12 are justified because they justify the presence of the parasite surface protein profilin in the mice that initiates the cascade of cellular signals that in turn initiate the inflammatory response that involves the release of interleukin-12 (IL-12) which is one of the most relevant regulators of the cellular immunity (Saeij *et al*, 2023). It is applied in the signalment of the natural killer cells (NK cells) cells, T-cells of the type CD 4+ and CD 8+ to produce the interferon-gamma (IFN-gamma). The end product, or the product of the right immune response to *T. gondii* will be the IFN- 7 that will activate the antigenic response of the nitric oxide (NO) and the antiparasitic enzyme of the macrophages. It also helps to express GTPases that break down the vacuoles that the parasite uses to conceal itself in a cell (Mahmoudzadeh *et al*, 2021).

The TNF- α plays an important role in immune response to the parasite *Toxoplasma gondii*. It also assists in the stocking of the immunological cells and structuring of various innate and acquired factors. It is also one of the biggest players of the battle against infection and the reduction of its effects. These are the macrophages and the T helper cells (Th1) which produce TNF- α as a response to acute infection. This cytokine is the key in the production of inflammatory processes especially when it is combined with interferon-gamma (IFN- γ). It strongly stimulates the production of nitric oxide (NO) and ant parasitic enzymes in the macrophages and it assists in the slowering down of growth rate of the intracellular parasites. It has also been shown in recent researches that MIC 3 protein of the parasite has the potential to trigger TNF- α production though the TLR 11/ MyD 88 pathway that activates NF- κB and enhances the iNOS expression further, especially in mice as TLR 11 is absent in human since the first outbreak of the infection is prevented because of the ability of the parasite in inducing the development of tissue cyst (zhou *et al*,2023) . TNF- α has also been found to be implicated in infection control in the central nervous system. Its deficiency in animal models has been shown to increase the rate of parasite reproduction in the brain and severe neurological symptoms. However, overproduction of TNF- α can have severe

inflammatory consequences in particular in sensitive tissues, such as the eye and brain. In the case of ocular toxoplasmosis, the higher the amount of TNF- α, the higher the amount of inflammation and damage to the retina. Studies have also shown that TNF- α may stimulate the parasite to escape the infected cells in favour of its dissemination and make this difficult for the immune response.(Seo&Lee,2024). *T. gondii* has evolved immune mechanisms to block the TNF- α production, including the activation of the signal pathway. This is accomplished, in part, by activating the signal pathway independently of IL-10, which is associated with reduced expression of inflammatory cytokines. The parasite also influences the chromatin organisation of the TNF- α gene, which inhibits its expression and diminishes the efficiency of the host immune system (Lüder,2024).

Interleukin-6 (IL-6) is a pleiotropic cytokine, the primary source of which is innate cells, such as macrophages and dendritic cells, in response to inflammatory stimuli or microbe infection. Interleukin-6 (IL-6) belongs to a large group of cytokines involved in the immunological response to *T. gondii* and optimal cellular immune response is required for successful containment of the parasite and control of its spread(Chauhan,2024). Innate immune system receptors (toll-like receptors) of cells are activated in the acute phase of the infection, as a result of recognition of components of the parasite and lead to the production of IL-6. In addition to its important role in the local inflammatory reaction, and also its function in activation of macrophages, this cytokine is also implicated in the recruitment of other immune cells to the location of infection (Wang 2024). Furthermore, IL-6 has been shown to be implicated in the differentiation of T cells to the Th17 cell type that is important for resistance against many intracellular parasite including *T. gondii*.

In addition, it indirectly increases the secretion of interferon-gamma (IFN- γ) by Th1 cells and natural killer (NK) cells which is most important cytokine in the resistance against *T. gondii* (Chen,2024).. Recent studies have revealed that IL-6 deficiency results in better mortality in murine models of *T. gondii* infection which is associated with high parasite load and control of parasite dissemination through the tissues. On the other hand, an excess production of IL-6 could be implicated in an excessive inflammation and damage of tissues, neural or intestinal tissue in particular, that reflects the importance of a proper regulation of the amount of this cytokine produced during infection. Therefore, IL-6 plays a dual role in Toxoplasma infection, as it plays a role in host protection by activating effective immune mechanisms, but due to its excess, it could lead to pathological consequences due to *hyperinflammation* (da Silva,2025).

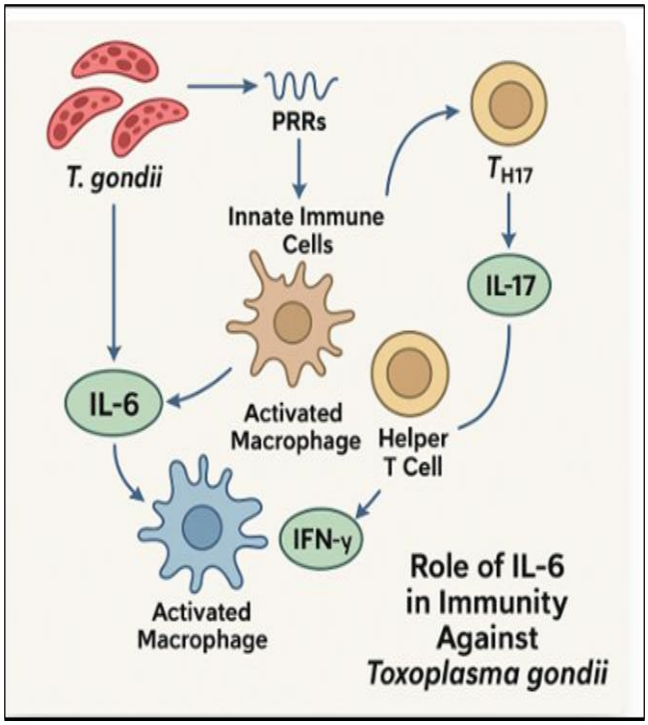
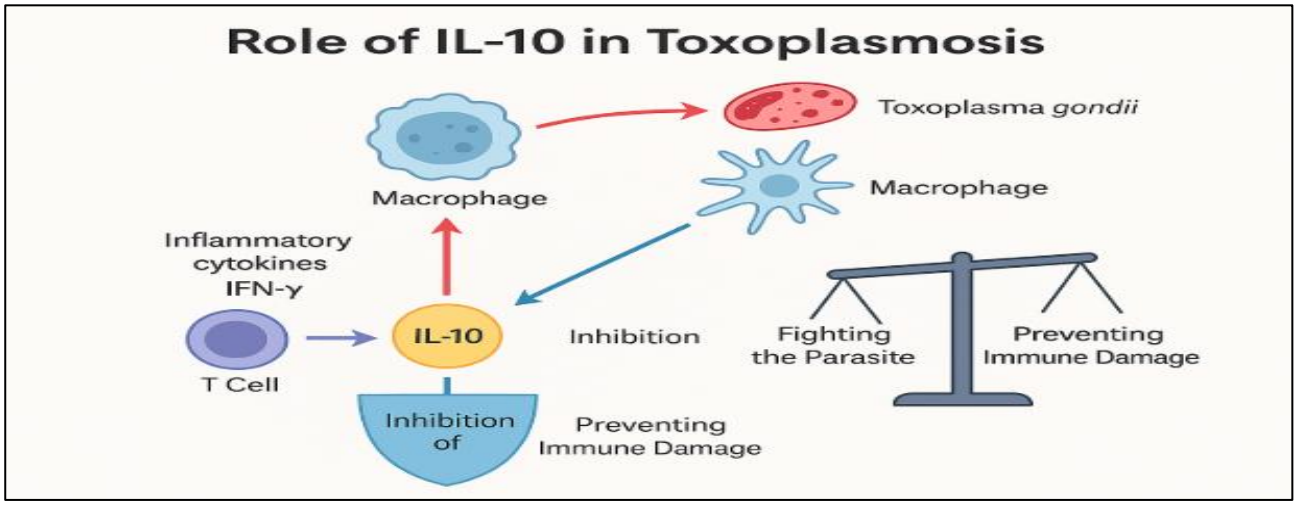


Figure 1: Role of IL-6 in Immunity against *Toxoplasma gondii.*

Interleukin-10 or IL-10 is a potent immunosuppressive (anti-inflammatory) cytokine. It has been shown to be an important regulator of the immune response in infection with a parasite called Toxoplasma gondii.

Immune activation is a delicate balance between infection control and the destruction of tissues due to hyperinflammation (Mustafa,2025) and recent evidence shows that IL-10 is mostly secreted by macrophages and regulatory T cells during infection and suppresses pro-inflammatory cytokine such as IFN- γ and TNF-α thereby reducing immune-mediated tissue damage. However, excessive IL-10 secretion may result in abnormal regulation of parasites (by immune system) and allow its survival and growth in the host (Bhol *et al*., 2024); in pregnancy, it has been demonstrated that T. gondii causes the alteration of molecular expression in decidual macrophages in the endometrium leading to cytokine imbalance, in particular IL-10. A study conducted by Wang *et al* (2024) demonstrated that these macrophages release IL-10 when infected, which suppresses the activity of natural killer cells (NK cells) and weakened the fetus immune protection, leading to the defect in innate immunity of pregnant women. A recent study by Guo *et al*. (2025) demonstrated that the ROP18, a parasite-secreted protein, inhibits CD73 expression, thus resulting in the reduction of IL-10 production by decidual macrophages and the dysfunction of immune functions of the pregnant women which would result in more risks of fetal complications. (omer salman *et al*, 2023) demonstrated the association between single-nucleotide polymorphism (SNP), that is gene IL-10 gene with the risk of toxoplasmosis in patients with type 2 diabetes. This finding is suggestive that genetic mutations in IL-10 gene may modulate the severity infection and body response to infection, suggesting IL-10 as an important factor in chronic diseases associated with parasitic infections. IL-10 has a dual regulatory function during Toxoplasma gondii infection, where it down-regulates excessive inflammation and tissue damage, but is able to compromise a successful immune response to the parasite. As a result of this dual role IL-10 is also a promising target for clinical and immunological studies in order to understand and to design a worthwhile therapeutic approach, in particular in sensitive conditions like pregnancy or chronic diseases.(Jafari, *et al*, 2023).



The Role of IL-12 in *Toxoplasma gondii* Infection.

Interleukin-12 (IL-12): This is one of the key cytokines in the activation of the mobilization of the cellular immune response to the protozoan parasite Toxoplasma gondii. Interleukin-12 (IL-12) is an early secretory cytokine expressed by dendritic cells and macrophages in response to stimulation of toll-like receptors (TLRs 11 and TLR-12) by pathogen-associated molecular patterns (PAMPs), including a multiple repeating unit protein (MRP) from parasites and Nod-like receptor (NLR) family member receptor-interacting protein 2 (RIP 2) kinase.

This cytokine causes the secretion of interferon-alpha (IFN- α) from natural killer cells (NK cells) and T cells for the containment of the infection (Mahmoudzadeh et al .2021) IFN-γ stimulates the macrophages to kill intracellular parasites by producing NO (nitric oxide) via stimulation of the intracellular resistance factors (IRGs) and parasite-killing factors (GBPs). IL-12 is also responsible for Th1 differentiation of T cells, the best T cell type capable of killing internal parasites. Previous studies have shown that IL-12 deficiency prevented the production of IFN-γ and increased tissue infection and parasite replication. Immune homeostasis is important to prevent the adverse consequences of an overreaction of the inflammatory system. However, IL-12 overproduction is responsible for tissue damage in chronic TBI. The conclusion from this process is that inflammatory cytokine IL-10 will be produced to inhibit the IL-12and IFN-γ immune response and inhibit anti-inflammatory and autodestructive responses. The role of IL-12 is crucial in mice for resisting infection (Ihara et al., 2024). It was also found that IL-12 KO mice were prone to dying, either because they were unable to mount a cellular immune response. Therefore, it appears that IL-12 is not only a product of early immunity of the host to T. gondii, but it also has regulatory influence in the relationship between effective host immunity and protective anti-inflammatory response, preventing pathological inflammation (Yoon et al, 2024).

Most infectious intracellular diseases, such as Toxoplasma gondii have been shown to demonstrate the importance of the Th17 cells (interleukin-17A). The studies also support that IL-17 is a positive response from the initiation of the infection process due to its facilitation of the induction of the innate type of immunity - recruitment of neutrophils to limit the spread of the parasites within the host. in addition to its protective effect, the pathologic role of IL-17A in chronic infection of a murine model is also evident since it does not target the neutrophils at the infection site. In the instance of the example, it is also known that the occurrence of the IL-17A expression contributes to the worsening of the inflammation that primarily occurs in the intestine, as well as the nervous model that also contributes to the pathogenesis of the chronic inflammation and the course (Taha et al, 2025, and Mills, 2023). However, intestinal inflammation associated with an oral infection of T. gondii can be controlled by IL-17A, indicating that this cytokine is not necessarily pathologic. Cytokine provoked by IL-6 release, IL-23 release, and TGF-b release are then provoked by IL-17A. There are other immune cells (natural killer (NK)) cells and ILC3 which produces IL-17A that may be present in some cases immediately following the initial infection of parasites. It is further indicated in the article that such a type of cells coordination reacts to being infected when the event of adequately responding mucosal tissues immunity and the central nervous system cannot be addressed by Th17 response (Sardinha-Silva, et al ,2022),(Navarro-Compan *et al,* 2023),(Zheng & Luo,2025).

Studies such as Munoz *et al* (2009) have shown that there is little balance between IL-17A and other cytokines such as IL-22 during infection, where IL-22 play a protective role in the intestine while IL-17A may promote inflammation which demonstrates the importance of regulation of the immune system between cytokines to prevent tissue damage from an excessive immune response. While it is considered a protective response by induction of innate responses, it is also considered to be one of the mechanisms by which chronic inflammatory responses develop if there is not a tight regulation of its response. Understanding the mechanisms that regulate IL-17A secretion but also the way IL-17A is balanced with other cytokines could provide a new opportunity for the development of a better therapeutic strategy for the toxicity of toxoplasmosis.

The role of adaptive immunity in the resistance of Toxoplasma gondii

Adaptive immunity is one of the important factors for resistance to infection with Toxoplasma gondii parasite. This host immune response is distinguished by a highly specialized cellular and molecular response, which is essential for containing acute phase infection and preventing establishment of latent phase infection or reactivation by immunocompromised persons. Role of adaptive immunity is demonstrated by the co-ordination of helper T cell (Th1), cytotoxic T cell (CD 8+) and B cell, along with a suite of regulatory cytokines and receptors, resulting in an effective and balanced immune response.(Khan & Moretto, 2022) Th1 helper T cells are important for the induction of anti-parasitic immunity through the secretion of interferon-gamma (IFN-γ), which is required for the activation of macrophages and cytotoxic T cells by promoting them to become more efficient at killing intracellular parasites. IFN-γ is responsible for the expression of immune GTPases (IRG, GBP), which demonstrate increased parasite elimination. These Th1 cells also produce TNF-alpha which has inflammatory effects that are essential for the ability to control infection. However, if left uncontrolled, it could result in the overproduction of an inflammatory reaction called a "cytokine storm."Conversely, regulatory T cells (Tregs) produce IL-10 that suppress excessive inflammatory response leading to immune damage: this is a delicate balance between the need to protect against infection, and the need to protect tissue from destruction(Ahmadpour,2023), (Ihara &Yamamoto,2024). CD8+ cytotoxic T lymphocytes are thought to contribute to killing of parasitically infected cells via direct mechanisms involving secretion of perforin and granzymes and their interaction with MHC-I surface-presented antigen. These cells are also crucial for restricting infection to the chronic phase as they are implicated in surveillance of parasitologically dormant tissue cysts in the nervous and muscular tissues. Recent research has shown that the parasite exports proteins (such as ROP18) to inhibit its presentation to antibodies and thus prevent activation of CD8+, in other words, to confirm their ability to evade the immune system.(Ferragut,,2021) and (Wang et al, 2024)

In addition to these, B cells under the guidance of the Th cells produce a special antibody called IgG which destroys the tachyzoites or stops them from attaching to the uninfected cells. Aside from the role these antibodies play in restricting the spread of the parasite during acute infection, they also have an important protective role during pregnancy in preventing the parasite from being transmitted to the fetus. But the parasite has proteins that may prevent the B cell differentiation (such as GRA15).Jafari et al , 2023). In addition, dendritic cells are important for the bridging of the infection from the innate to the adaptive immune system by presenting the parasite antigens to T cells and induction of differentiation of T h1 and CD 8+ T cells. However, the studies have been conducted using this type of cells as a therapeutic platform by loading with antigen from Toxoplasma gondii to enhance against antiparasitic immunity or as an adjuvant therapy against cancer. It has been demonstrated that the interaction between innate and adaptive immunity is necessary for the efficient activation of these cells by the TLR-like receptors, especially TLR11 and TLR -12. (Khan & Moretto,2022).

From a clinical perspective, the adaptive immune response is particularly relevant in immunocompromised states such as HIV infection; where a decline in CD 4+ cell number impairs the Th1 response allowing the latent parasite to become active and cause toxoplasmic encephalitis (a fatal neurological complication in AIDS patients). This immunity is also important during pregnancy where this

physiological shift in a host to an immunosuppressive state increases the risk of maternal-fetal transmission of the disease especially if the infection is primary during the first trimester of gestation. In such cases, regulation of cytokines, and a balance of IFN-gamma and IL-10 is important for the outcome of the pregnancy and for the safety of the fetus(Wesolowski et al,2023) and (Santos et al , 2023). Despite the potency of this immunity, repeated infection elicits a 'fatigue' of T-cells that gradually lose their potency and exhibit signs of functional inhibition such as up-regulation of PD-1 and LAG-3 receptors that allow the parasite to survive for life in vivo. This exhaustion can be recovered by the use of immune checkpoint inhibitors, which have been proposed as a potential new candidate for therapeutic strategies in the treatment of chronic infections.Therefore, adaptive immunity is the most effective defense mechanism against T. gondii and it is also the most specific defense mechanism. However, success of this immunity depends on a balance between induction of the defense mechanism and containment of the damage inflicted by the inflammation and/or capability of the host to overcome the intricate mechanisms that were developed by the parasite to subvert the host immune system." "this interplay provides evidence for the need to broaden the view of the proper immunological pathways that need to be incorporated into the design of effective vaccines and future therapeutic strategies(Li,*et al*,2025).

CONCLUSION

Toxoplasmosis is another infectious disease prevalent not only among humans, but also animals and spread in various countries at various levels. It can be asymptomatic. the danger of this parasite is that the number of cells and tissues it can infect can be extremely large, as the immune system recognizes the components of the parasite with special receptors which cause a cascade of cell signaling which leads to the appearance of inflammatory responses. And also Adaptive immunity is also involved in infection by the parasite Toxoplasma gondii, which is highly regulated cellular and molecular response and involves a combination of T helper cells(Th1), T cytotoxic cells(+CD 8), and B cells, and a cascade of cytokines. the efficacy of this immunity depends on the balance between the response of defensive immunity, containment of inflammatory damage, and host resistance to the advanced immune evasion strategies expressed by the parasite. this would help in designing an effective one.

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