



NEGLECTED ASPECTS ABOUT TOXOPLASMOSIS IN IMMUNE DEFICIENCIES: TIME TO ATTENTION!

ASPECTOS NEGLIGENCIADOS DA TOXOPLASMOSE EM IMUNODEFICIÊNCIAS: HORA DE PRESTAR ATENÇÃO!

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ABSTRACT

Toxoplasmosis is a zoonotic disease with a wide global distribution, caused by the obligate intracellular protozoan *Toxoplasma gondii*. It is recognized as an important foodborne illness, although it is not yet formally included in the global list of neglected diseases. Its occurrence is frequently associated with contexts of social vulnerability, low sanitation coverage, immunosuppression, and lack of knowledge about its life cycle. The parasite has three infective forms—tachyzoites, bradyzoites, and sporozoites—and a heteroxenic cycle involving felines as definitive hosts. In immunocompromised individuals, the infection can progress to severe forms, often resulting from the reactivation of latent infections. In this context, the tissue microenvironment and the parasite-host interaction play a central role in disease progression, influencing immunological, inflammatory, and metabolic mechanisms that determine the clinical outcome. The objectives of this article were: To identify mortality related to toxoplasmosis in different regions of Brazil. Identifying gaps and proposing priorities for health education. This is a descriptive epidemiological study on toxoplasmosis mortality in different regions of Brazil, based on secondary data obtained from the Ministry of Health's Toxoplasmosis Panel. Additionally, an integrative review focusing on primary health care was conducted, encompassing clinical, immunological, epidemiological, and therapeutic aspects of toxoplasmosis, especially in immunocompromised populations. The findings show that toxoplasmosis mortality in Brazil has a heterogeneous distribution, with predominantly high rates in the North Region throughout the years analyzed (2019–2024), followed by variations in the Central-West, Northeast, and Southeast regions. States such as Acre, Tocantins, and Amapá stand out with persistently high rates, while states like São Paulo have lower rates, possibly due to greater surveillance in at-risk populations, such as transplant recipients. These data reflect regional inequalities in access to diagnosis, epidemiological surveillance, and health care. Toxoplasmosis, although highly prevalent, remains underdiagnosed, especially in immunosuppressed individuals, in whom reactivation of the infection can result in severe manifestations such as encephalitis and disseminated forms, often fatal. The literature highlights a relevant association with conditions such as AIDS, neoplasms, and the use of immunosuppressive therapies, including anti-TNF- α , which can trigger reactivation of the parasite. Furthermore, kidney transplant recipients constitute a high-risk group, with high mortality when diagnosis is delayed. From a pathophysiological point of view, the tissue microenvironment plays a fundamental role in the dynamics of the infection, being characterized by immunometabolic alterations that favor the persistence of the parasite. Cytokine modulation, the action of immune system cells, and the subversion of cellular mechanisms, such as lysosomal function, contribute to the maintenance of chronic infection. The possible association between toxoplasmosis and neoplasms is discussed,

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although there is no established causal evidence, highlighting the need for more robust studies in the area of immunoparasitology. The use of natural products with potential antiparasitic activity does not replace conventional treatment, as they are aimed at the parasite's life cycle. It is concluded that toxoplasmosis remains a significant public health problem, especially in immunocompromised populations, where it can present high mortality rates. The unequal distribution of mortality in Brazil highlights weaknesses in surveillance, diagnosis, and healthcare access systems, reinforcing the need to strengthen primary care and health education strategies. A deeper understanding of the infection microenvironment and the immunological mechanisms involved contributes to the advancement of scientific knowledge and the development of new therapeutic approaches. The urgency of investments in research, epidemiological surveillance, public policies, and health education actions that prioritize vulnerable populations, aiming to reduce morbidity and mortality associated with toxoplasmosis, is emphasized.

Keywords: Toxoplasmosis; *Toxoplasma gondii*; Immunosuppression; Mortality; Health Education.

RESUMO

A toxoplasmose é uma zoonose de ampla distribuição mundial, causada pelo protozoário intracelular obrigatório *Toxoplasma gondii*, sendo reconhecida como uma importante doença transmitida por alimentos, embora ainda não seja formalmente incluída na lista global de doenças negligenciadas. Sua ocorrência está frequentemente associada a contextos de vulnerabilidade social, baixa cobertura de saneamento básico, imunossupressão e desconhecimento sobre seu ciclo de vida. O parasito apresenta três formas infectantes — taquizoítos, bradizoítos e esporozoítos — e um ciclo heteroxênico envolvendo felinos como hospedeiros definitivos. Em indivíduos imunocomprometidos, a infecção pode evoluir para formas graves, frequentemente decorrentes da reativação de infecções latentes. Nesse contexto, o microambiente tecidual e a interação parasita-hospedeiro assumem papel central na progressão da doença, influenciando mecanismos imunológicos, inflamatórios e metabólicos que determinam o desfecho clínico. O presente artigo teve como objetivos: Identificar a mortalidade relacionada à toxoplasmose em diferentes regiões do Brasil. Identificar lacunas e propor prioridades para a educação em saúde. Trata-se de estudo epidemiológico descritivo sobre a mortalidade por toxoplasmose nas diferentes regiões do Brasil, com base em dados secundários obtidos no Painel de Toxoplasmose do Ministério da Saúde. Adicionalmente, foi realizada revisão integrativa com enfoque na atenção primária à saúde, contemplando aspectos clínicos, imunológicos, epidemiológicos e terapêuticos da toxoplasmose, especialmente em populações imunocomprometidas. Os achados evidenciam que a mortalidade por toxoplasmose no Brasil apresenta distribuição heterogênea, com predomínio de taxas elevadas na Região Norte ao longo dos anos analisados (2019–2024), seguida por variações nas regiões Centro-Oeste, Nordeste e Sudeste. Estados como Acre, Tocantins e Amapá destacam-se com índices persistentemente elevados, enquanto unidades como São Paulo apresentam taxas mais baixas, possivelmente em decorrência de maior vigilância em populações de risco, como os transplantados. Esses dados refletem desigualdades regionais em acesso ao diagnóstico, vigilância epidemiológica e assistência à saúde. A toxoplasmose, embora altamente prevalente, permanece subdiagnosticada, sobretudo em indivíduos imunossuprimidos, nos quais a reativação da infecção pode resultar em manifestações graves, como encefalite e formas disseminadas, frequentemente fatais. A literatura destaca associação relevante com condições como SIDA, neoplasias e uso de terapias imunossupressoras, incluindo anti-TNF- α , que podem desencadear reativação do parasito. Além disso,





receptores de transplante renal constituem um grupo de alto risco, com elevada mortalidade quando o diagnóstico é tardio. Do ponto de vista fisiopatológico, o microambiente tecidual exerce papel fundamental na dinâmica da infecção, sendo caracterizado por alterações imunometabólicas que favorecem a persistência do parasito. A modulação de citocinas, a atuação de células do sistema imune e a subversão de mecanismos celulares, como a função lisossomal, contribuem para a manutenção da infecção crônica. Discute-se a possível associação entre toxoplasmose e neoplasias, embora não haja evidência causal estabelecida, destacando-se a necessidade de estudos mais robustos na área de imunoparasitologia. O uso de produtos naturais com potencial atividade antiparasitária não substituem o tratamento convencional, pois são para o ciclo do parasita. Conclui-se que a toxoplasmose permanece como um agravamento de significativa relevância em saúde pública, especialmente em populações imunocomprometidas, nas quais pode apresentar elevada letalidade. A distribuição desigual da mortalidade no Brasil evidencia fragilidades nos sistemas de vigilância, diagnóstico e acesso à saúde, reforçando a necessidade de fortalecimento da atenção primária e de estratégias de educação em saúde. O entendimento aprofundado do microambiente da infecção e dos mecanismos imunológicos envolvidos contribui para o avanço do conhecimento científico e para o desenvolvimento de novas abordagens terapêuticas. Destaca-se a urgência de investimentos em pesquisa, vigilância epidemiológica, políticas públicas e ações de educação em saúde que priorizem populações vulneráveis, visando reduzir a morbimortalidade associada à toxoplasmose.

Palavras-chave: Toxoplasmose; *Toxoplasma gondii*; Imunossupressão; Mortalidade; Educação em Saúde.

BACKGROUND

World Health Organization recognizes toxoplasmosis as a major foodborne disease worldwide, but does not include it in the global list of neglected diseases (Centers for Disease Control and Prevention USA - CDC, 2024, a; WHO, 2025).

Toxoplasmosis is a zoonosis with a worldwide prevalence, occurring in areas of greater social vulnerability due to a lack of knowledge about the life cycle of the agent, the protozoan *Toxoplasma gondii*, when it contaminates soil, water, and consequently, food (Attias et al., 2020).

Regarding the life cycle, *T. gondii* has three main infective forms: tachyzoite (acute multiplication phase), bradyzoite (tissue cysts, latent form), and sporozoite within oocysts excreted in feline feces and capable of contaminating soil/water/food (Attias et al., 2020a).

Humans become infected primarily by ingesting meat containing bradyzoites or food/water/soil contaminated with oocysts; after ingestion, bradyzoites/sporozoites convert into tachyzoites (Attias et al., 2020; CDC, 2024b).





The life cycle of *Toxoplasma gondii* is heteroxenic, with felines, particularly domestic cats, as definitive hosts and virtually any bird or mammal as intermediate hosts. Humans can become infected in several ways: ingestion of sporulated oocysts eliminated by felines; ingestion of undercooked poultry or mammalian meat; congenital transmission if the pregnant woman is in the acute phase of toxoplasmosis; and, more rarely, through whole blood transfusion or organ transplantation (CDC, 2024b).

Many articles discuss about the high prevalence of Toxoplasmosis. Nevertheless, indeed, several immunocompromised people pass through activation of this high prevalent illness and die from serious manifestations of Toxoplasmosis as complication, around the world, among them, kidney post-transplanted patients (CDC, 2024b).

The tissue microenvironment is defined as the dynamic three-dimensional space composed of resident cells, extracellular matrix (ECM), soluble factors (cytokines, chemokines, metabolites), and local physicochemical conditions (pH, oxygen tension, nutrient availability). Under physiological conditions, this niche regulates tissue homeostasis, intercellular communication, and adaptive responses to external stimuli. In the context of infectious diseases, the microenvironment is profoundly remodeled by pathogen-host interaction, becoming a crucial determinant for the progression, control, or chronicity of the infection (Gabriel; Ross; Kim, 2022; Varricchio, Da Silva, Pyrrho, 2026).

Toxoplasmosis, caused by the obligate intracellular protozoan *Toxoplasma gondii*, represents a paradigmatic model of microenvironmental modulation. After cellular invasion, the parasite resides in a non-fusogenic parasitophorous vacuole (PV), manipulating host signaling pathways to subvert destruction mechanisms, promote its replication, and subsequently establish bradyzoites in tissue cysts, especially in the central nervous system (CNS) and striated muscle tissue (Blader; Saeij, 2021). This transition between acute and chronic phases is sustained by a constantly remodeling immunometabolic microenvironment, in which lysosomes, organelles traditionally associated with degradation, emerge as central regulators of the immune response, antigen presentation, and autophagy.





What is the distribution of Toxoplasmosis's mortality in Brazil regions? Is it well diagnosed?

AIMS

To identify the related mortality by toxoplasmosis in different regions of Brazil .

To identify gaps, and propose priorities for health education.

METHODS

Epidemiological study on mortality from Toxoplasmosis by regions of Brazil by access to the Toxoplasmosis Panel (<https://www.gov.br/saude/pt-br/composicao/svsa/cnie/painel-toxoplasmose>). Moreover, a bibliographic review study was performed based on primary health care.

RESULTS

Natural products do not replace antiparasitic therapy prescribed by a physician (e.g., pyrimethamine + sulfadiazine + leucovorin) (CDC, 2024). However, the control of this zoonosis may be achieved through chemical classes of natural products capable of acting on different stages of the *T. gondii* cycle.

The epidemiology of the risk and harm of Toxoplasmosis in Brazil can be understood from the Mortality Rates shown in Table 1:

Table 1: Mortality rate due to toxoplasmosis in Brazil: prevalence and distribution by region from 2019 to 2024.
<https://www.gov.br/saude/pt-br/composicao/svsa/cnie/painel-toxoplasmose>

Year		2019		2020		2021		2022		2023		2024		2025	
Region	UF	Deaths	Mortality Rate	Deaths	Mortality Rate	Deaths	Mortality Rate	Deaths	Mortality Rate	Deaths	Mortality Rate	Deaths	Mortality Rate	Deaths	Mortality Rate
South		9	2.33	9	2.4	7	1.93	5	1.39	6	1.68	4	1.12	0	0
	Santa Catarina	1	1.02	2	2.05	2	2.08	0	0	3	3.11	1	1.04	0	0
	Rio Grande do Sul	6	4.46	2	1.53	2	1.61	2	1.65	0	0	1	0.83	0	0





	Paraná	2	1.3	5	3.41	3	2.11	3	2.13	3	2.14	2	1.43	0	0
South east		27	2.45	24	2.28	18	1.78	20	2.04	21	2.17	18	1.86	0	0
	São Paulo	8	1.37	12	2.17	6	1.14	12	2.34	9	1.78	4	0.79	0	0
	Rio de Janeiro	9	4.33	7	3.52	7	3.69	5	2.77	9	5.11	9	5.11	0	0
	Minas Gerais	5	1.95	5	2.02	5	2.07	2	0.85	3	1.29	4	1.72	0	0
	Holy Spirit	5	9.18	0	0	0	0	1	1.94	0	0	1	1.93	0	0
North		9	2.88	10	3.32	16	5.19	12	4.16	7	2.47	5	1.76	0	0
	Tocantins	0	0	3	12.78	2	8.59	0	0	1	4.36	1	4.36	0	0
	Roraima	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	Rondônia	2	7.42	1	3.9	1	3.95	1	4.03	0	0	1	4.2	0	0
	To	4	2.94	5	3.82	8	5.91	5	3.97	2	1.61	1	0.81	0	0
	Amazon	0	0	1	1.33	3	3.85	4	5.55	2	2.86	0	0	0	0
	Amapá	2	11.83	0	0	0	0	1	6.64	0	0	0	0	0	0
	Acre	1	5.87	0	0	2	12.08	1	6.58	2	13.09	2	13.09	0	0
North East		11	1.37	17	2.2	15	1.96	9	1.27	17	2.42	7	0.99	0	0
	Sergipe	0	0	0	0	0	0	2	6.78	0	0	0	0	0	0
	Rio Grande do Norte	0	0	0	0	0	0	1	2.46	0	0	0	0	0	0
	Piauí	3	5.94	0	0	3	6.16	3	6.73	3	6.74	0	0	0	0
	Pernambuco	2	1.49	2	1.54	1	0.78	0	0	3	2.56	1	0.85	0	0
	Paraíba	1	1.74	2	3.56	0	0	0	0	0	0	0	0	0	0
	Maranhão	2	1.8	1	0.96	2	1.88	1	1.04	3	3.15	1	1.05	0	0
	Ceará	0	0	5	4.1	4	3.33	2	1.78	2	1.79	0	0	0	0
	Bahia	2	1.03	7	3.76	3	1.64	0	0	3	1.8	3	1.8	0	0
	Alagoas	1	2.01	0	0	2	4.11	0	0	3	6.44	2	4.29	0	0
Centr al- West		8	3.32	4	1.73	9	3.93	9	4.01	11	4.86	1	0.44	0	0
	Mato Grosso do Sul	1	2.31	0	0	0	0	0	0	1	2.5	0	0	0	0
	Mato Grosso	3	5.1	0	0	2	3.45	2	3.44	6	10.27	0	0	0	0
	Goiás	3	3.59	4	4.98	5	6.38	6	7.74	3	3.75	1	1.25	0	0
	Federal District	1		0	0	2	3.9	1	2.05	1	2.08	0	0	0	0

Source: <https://www.gov.br/saude/pt-br/composicao/svsa/cnie/painel-toxoplasmose>.

Considering a mortality rate of 4.0 or higher as quite high, it was identified that in 2019, the highest mortality rates due to Toxoplasmosis has occurred in the North Region of Brazil, highlighting three states of the federation (Amapá 11.83; Rondônia 7.42; Acre 5.87), followed by Southeast Region (Espírito Santo 9.18; Rio de Janeiro 4.33), after Northeast



Region (Piauí 5.94), besides Central-West Region (Mato Grosso 5.1) and, at last, South Region (Rio Grande do Sul 4.46).

In 2020, the highest mortality rates were in the North Region (Tocantins 12.78) and Central-West Region (Goiás 4.98). In 2021, the highest mortality rates were in the North Region (Acre 12.08; Tocantins 8.59; Pará 5.91), followed by the Central-West Region (Goiás 6.38) and the Northeast Region (Piauí 6.16; Alagoas 4.11).

In 2022, although the highest mortality rates showed a decrease, the prevalence remained high in the Central-West Region (Goiás 7.74), Northeast Region (Sergipe 6.78; Piauí 6.73), and North Region (Amapá 6.64; Acre 6.58; Amazonas 5.55; Rondonia 4.03).

In 2023, the highest mortality rates increased again in the North Region (Acre 13.09; Tocantins 4.36), Central-West Region (Mato Grosso 10.27), Northeast Region (Piauí 6.74; Alagoas 6.44), and Southeast Region (Rio de Janeiro 5.11).

In 2024, the highest mortality rates remained in the North Region (Acre 13.09; Tocantins 4.36; Rondonia 4.2), Southeast Region (Rio de Janeiro 5.11) and Northeast Region (Alagoas 4.29).

Indeed, the state of São Paulo, with several hospitals that perform transplants and employ immunosuppressants for patients to ensure their success, has nevertheless maintained relatively low mortality rates, even showing a downward trend (2019: 1.37; 2020: 2.17; 2021: 1.14; 2022: 2.34; 2023: 1.78; 2024: 0.79), possibly due to increased surveillance of these patients.

DISCUSSION

This review study investigates the prevalence of mortality from Toxoplasmosis in Brazil from 2019 to 2024, based on official data. The distribution of mortality throughout the years is noteworthy, remaining consistent across all regions, for a disease that primarily affects immunocompromised individuals with chronic or acute diseases (Varricchio, Da Silva, Pyrrho, 2026).





Considering the Brazilian epidemiological reality and the factors emphasized by Fiocruz, the main points that justify this classification are:

- **Context of Poverty:** Like most neglected diseases, toxoplasmosis is often endemic in low-income populations.
- **Focus on Pregnancy and Congenital Transmission:** Gestational and congenital toxoplasmosis is a specific public health concern, as it can cause severe fetal malformations, outbreaks of ocular toxoplasmosis, miscarriages, and fetal death, requiring increased prenatal monitoring.
- **Environmental Contamination:** The lack of adequate basic sanitation contributes to environmental contamination by oocysts (feline feces), facilitating the infection of humans through food and water, which characterizes the need for comprehensive preventive actions.
- **Importance in Primary Care:** Early detection during prenatal care is fundamental, as immediate treatment reduces the risk of congenital transmission.

Reflecting differences in the criteria adopted by each health institution, according to the Brazilian Fiocruz News Agency, neglected diseases are those caused by infectious agents or parasites and are considered endemic in low-income populations. These diseases also present unacceptable indicators and reduced investments in research, drug production, and control. In this context, the Fiocruz classifies toxoplasmosis as a neglected disease in the Brazilian context due to its disproportionate impact on vulnerable populations and the limited prioritization it receives in public policies, surveillance, and research (Valverde, 2022).

Reactivation of a latent infection by the protozoan *Toxoplasma gondii* can result in serious neurological consequences and even death. Cases of *T. gondii* reactivation have been frequently associated with acquired immunodeficiency syndrome (AIDS), however other immunosuppressive conditions may also be related. Anti-TNF- α therapy consistently triggers the reactivation of latent *T. gondii* cysts in murine models (De Abreu Guimarães et al., 2022).

Reactivation of *T. gondii* by blocking TNF- α is rare in humans but possible, despite the widespread use of these medications. Serological evidence of possible latent *T. gondii*





infection in humans is common worldwide, therefore, the reason why anti-TNF- α -induced reactivation is not more frequent remains unknown (Montoro et al., 2025).

Montoro and colleagues reported a clinical case of a human with rheumatoid arthritis who developed fatal cerebral toxoplasmosis after treatment with anti-TNF- α . The patient was seen in a local emergency room with mental confusion. Computed tomography raised suspicion of metastatic disease, leading to treatment with corticosteroids. Lumbar puncture ruled out bacterial or viral meningitis. With persistent cognitive decline, magnetic resonance imaging of the head revealed an increased number of ring-enhancing lesions associated with *T. gondii*. Brain biopsy confirmed the presence of this parasite (Montoro et al., 2025).

Despite standard treatment for toxoplasmosis, the patient died. Possibly, two factors may have contributed to this fatal outcome. First, in rheumatoid arthritis, there is evidence of loss of immune resilience and abnormal subsets of T cells. Second, some strains of *T. gondii* are more virulent than others. Post-mortem mass spectrometry and proteomic analysis of cerebrospinal fluid revealed several *T. gondii* peptides (Montoro et al., 2025).

A literature review suggests that the risks associated with anti-TNF- α therapy in patients seropositive for *T. gondii* have not been adequately recognized. Therefore, it is recommended that testing for *T. gondii* seropositivity be considered before and after the start of anti-TNF- α therapy, as is done for other infections such as tuberculosis (Montoro et al., 2025).

It is observed that outbreaks of severe toxoplasmosis apparently occur, which may be related to acute infections from the ingestion of contaminated food (contaminated vegetables and raw or undercooked meats) as well as exposure during work in contaminated soils and groundwater. However, other causes of severe immunosuppression capable of leading to death from toxoplasmosis in unusual circumstances (Renoult et al., 1997; Hébraud et al., 2008), neurotoxoplasmosis and disseminated toxoplasmosis involve underlying diseases such as malignant neoplasms, acquired immunodeficiency syndrome, chemical dependency, in addition to treatment with immunosuppressants (Gay et al., 2019; Ka; Salibindla; Kendler, 2024).



In-depth epidemiological studies urgently need to be developed to complement this reasoning.

However, although it is rare, there is a group of immunosuppressed patients who are closely monitored due to the high risk of developing severe forms of toxoplasmosis (either through reactivation or dissemination): Kidney transplant recipients.

- Toxoplasmosis in kidney transplant recipients: reports and frequency

Due to prolonged use of immunosuppressive medications, transplant recipients are typically classified as immunocompromised individuals, making them very susceptible to acute infection or latent infection caused by various microorganisms (Zheng, 2025).

Toxoplasmosis is a recognized, yet relatively rare, complication in kidney transplant patients, but it can be serious and even fatal in these immunocompromised individuals, as shown in Table 2.

Table 2: Summary of the main reports and case series on the frequency of toxoplasmosis in kidney transplant recipients.

Toxoplasmosis in kidney transplant recipients: Type of study/report	Number of cases	Mortality	Post-transplant period	Citations
Literature review + series	31	64.5%	Up to 3 months	(Renoult et al., 1997)
Multicenter study in Spain	6/15.800	13.6%	Median: 92 days	(Fernández-Sabé et al., 2012)
European study (2010-2014)	29/87 SOT	High	Variable	(Robert-Gangneux et al., 2018)
Recent case reports	1-2 each	Variable	2 weeks to years	(Ka et al., 2024; Gay et al., 2019; Francí et al., 2023; Abbas et al., 2020; Filloy et al., 2013; Hébraud et al., 2008)

Source: Consensus AI.

Most studies indicate that clinical toxoplasmosis after kidney transplantation is uncommon, but there are several case reports and small series documenting its occurrence, mainly in the first weeks or months after transplantation (Renoult et al., 1997; Martina et al.,





2011; Robert-Gangneux et al., 2018; Adekunle et al., 2021; Adekunle et al., 2019; Fernández-Sabé et al., 2012; Hébraud et al., 2008).

Reviews and multicenter studies show that the incidence is low: for example, in a Spanish study, only six cases were identified among 15.800 solid organ transplants (0.14%), all 6 in kidney transplant patients (Fernández-Sabé et al., 2012). The European study identified 29 cases of toxoplasmosis in solid organ transplants over 5 years, including kidneys, in 87 transplant recipients (Robert-Gangneux et al., 2018).

Recent case reports continue to be published, indicating that the disease, although rare, remains a relevant risk (Ka et al., 2024; Gay et al., 2019; Francí et al., 2023; Abbas et al., 2020; Filloy et al., 2013; Hébraud et al., 2008).

Most cases occur due to reactivation of latent infection or transmission via donor organ (Renoult et al., 1997; Martina et al., 2011; Robert-Gangneux et al., 2018; Fernández-Sabé et al., 2012). The clinical presentation is varied, including encephalitis, pneumonia, septic shock, and multiple organ failure (Renoult et al., 1997; Martina et al., 2011; Fernández-Sabé et al., 2012; Robert-Gangneux et al., 2018; Adekunle et al., 2019; Adekunle et al., 2021).

Mortality is high, especially in disseminated cases and those diagnosed late (Renoult et al., 1997; Martina et al., 2011; Robert-Gangneux et al., 2018; Adekunle et al., 2019; Fernández-Sabé et al., 2012). Therefore, although toxoplasmosis is rare in kidney transplant recipients, there are consistent reports in the literature highlighting the potential for high mortality. Early recognition and prophylaxis are essential to reduce the risk and improve the prognosis of these patients.

Immunosuppression may lead to the reactivation of a latent infection, as patients with uremia are prone to infection by *T. gondii*. In kidney transplant recipients, if not treated promptly, it can be deadly. It typically happens within the first three months, either because of the reactivation of a latent infection or transmission from the donor. The highest risk is seen in seronegative recipients receiving organs from seropositive donors; however, it is less common





for seropositive patients to experience reactivation of a latent infection (Fendandez, 2012; Derouin, Pelloux, Escmid, 2008).

Although the primary independent risk factor found is negative pre-transplant serological status, other related factors should be taken into account. (Zheng, 2025) To identify high-risk patients, pre-transplant serological screening of donors and recipients is crucial. Studies have shown that sulfamethoxazole-trimethoprim prophylaxis is very efficient in treating clinical toxoplasmosis; no cases of *T. gondii* DNA were found in patients receiving this treatment.(Galván-Ramírez et al., 2018).

The degree of immunosuppression, which can reactivate latent infection, is also linked to the risk of toxoplasmosis (Galvan, 2018). Additionally, research has shown that in patients with uremia and kidney transplant recipients, a higher prevalence of anti-Toxoplasma IgG is linked to a lower percentage of CD3+ and CD8+ T cells (ROBERT-GANGNEUX, 2018).

In terms of environmental exposure, interaction with cats has been linked to greater seroprevalence in uremic patients and kidney transplant recipients (ROBERT-GANGNEUX, 2018). Additionally, toxoplasmosis raises the possibility of transplant failure and perhaps mortality.

Due to the difficulty in clinically monitoring these severely ill patients undergoing immunosuppression to prevent rejection, they require careful and highly complex management. In this sense, health educational solutions must be thought to be able of modifying outcomes not only in immunosuppressed individuals but also in others, reducing exposure.

Therefore, there's a positive relationship between post-transplant treatment (with immunosuppressors) and the associated eclosion of cancerous cellular lines in tissues, condition that, which in turns, also compromises the immune system, so it becomes a vicious cycle with high potential of reactivation or dissemination of Toxoplasmosis.





Features about Toxoplasmosis

Toxoplasmosis is widely prevalent and can cause serious complications in immune compromised individuals, but it is not officially recognized as an oncoparasitosis in medical or parasitological classifications (Xiao; Yolken, 2015; Sanchez; Besteiro, 2021; Liu et al., 2015; Blume; Seeber, 2018).

Another aspect of concern regarding the prevalence and severity of this parasitic infection is the growing perception of Toxoplasmosis as an oncoparasitic disease. Toxoplasmosis is not formally classified as an oncoparasitic disease, although there is research suggesting possible associations between *Toxoplasma gondii* infection and cancer.

Although Toxoplasmosis is not recognized as an oncoparasitosis (Liu et al., 2015; Xiao; Yolken, 2015; Sanchez; Besteiro, 2021), because there is no proven causal relationship (Hodge et al., 2021; Dupont et al., 2023), nevertheless, an epidemiological association with cancer has been suggested (Cong et al., 2015; Anvari et al., 2019; Liu et al., 2019; Kalantari et al., 2020; Hodge et al., 2021). Meanwhile, the probable biological mechanisms have been investigated, suggested and discussed (Chen et al., 2022; Dupont et al., 2023; Yang et al., 2022; Chen et al., 2023; Song et al., 2024).

However, there is evidence of its association with cancer. Epidemiological studies and meta-analyses show a higher prevalence of *T. gondii* infection in cancer patients, especially brain, lung, hematological, and prostate tumors, suggesting a possible association, but not proven causality (Cong et al., 2015; Kalantari et al., 2020; Hodge et al., 2021; Anvari et al., 2019; Shihab et al., 2025; Liu et al., 2019). The association may be explained through the mechanisms of immune suppression during Cancer development which may reactivate the latent illness.

Indeed, there are no scientific evidences related Toxoplasmosis to Cancer. What is the definition of Oncoparasitosis? Oncoparasitosis are parasitic diseases known to be directly associated with the induction of cancer, such as Schistosomiasis (*Schistosoma haematobium*)





and cancer bladder besides Cysticercosis (*Taenia solium*) and neurologic cancer (Dupont et al., 2023).

Recent research discusses the hypothesis that *T. gondii* may interfere with cellular pathways related to apoptosis and the cell cycle, raising the possibility of an oncogenic role, especially in brain tumors. However, robust clinical studies confirming this causal relationship, still lack (Hodge et al., 2021; Dupont et al., 2023).

Animal models show that acute infection can inhibit tumor growth, while chronic infection of *T. gondii* can favor tumor development, indicating a complex and context-dependent relationship within the immune system (Filloy et al., 2012; Song et al., 2024; Yang et al., 2022; Chen et al., 2023; Chen et al., 2022).

So, although some evidence suggests a possible link between Toxoplasmosis and certain types of cancer, toxoplasmosis is not considered an oncoparasitic disease. The causal relationship remains uncertain, and further studies are needed to clarify the oncogenic potential of *T. gondii* through immune parasitological studies.

According to Roitt and Delves (2012), Immune parasitology is the study of the relationships between parasites and the host's immune system, including the immune responses triggered besides the parasite's evasion mechanisms. It is the branch of science that studies the interaction between the host's immune system and parasites, including the organism's defense mechanisms. This area involves understanding the innate and adaptative immune responses triggered by parasitic infections, as well as the impact of these interactions on the pathogenesis and control of parasitic diseases (Khan et al., 2021).

So, Immune Parasitology is the branch of science that studies these complex interactions and its main focus includes:

- Defense mechanisms: Investigate how the host organism (human or animal) reacts to defend itself against protozoa and helminths (worms), which are the main parasites studied.
- Parasite evasion: Analyze the strategies that parasites use to escape or modulate the host's immune response and thus survive within the organism.





- Immunopathogenesis: Study the mechanisms by which the immune response, in some cases, can contribute to the lesions and diseases caused by parasitic infection.
- Vaccine and therapy development: Use this knowledge to seek new ways to prevent and treat parasitic diseases, such as Chagas disease, Leishmaniasis, and Malaria.

Characteristics of the Microenvironment in Toxoplasmosis

The microenvironment in *T. gondii* infection is characterized by marked spatial and temporal heterogeneity. In the acute phase, a pro-inflammatory environment predominates, with increased vascular permeability, leukocyte influx, local production of reactive oxygen and nitrogen species (ROS/RNS), and extracellular acidification resulting from the accelerated glycolytic metabolism of activated cells (Silva et al., 2023). In the chronic phase, the microenvironment becomes immunosuppressive and metabolically restricted, with decreased essential nutrients (such as tryptophan and glucose), increased TGF- β and IL-10, and the formation of a pericystic fibrosis network that isolates bradyzoites from immune surveillance (Gazzinelli et al., 2023).

From a physicochemical point of view, the microenvironmental pH tends to oscillate between 6.8 and 7.2 in infected tissue, influencing lysosomal enzymatic activity, cytokine diffusion, and the expression of cell surface receptors. Relative hypoxia, induced by accelerated oxygen consumption by activated macrophages and microvasculature dysregulation, activates factors such as HIF-1 α , which modulate macrophage polarization and parasite survival (Montiel et al., 2022).

The cellular architecture of the microenvironment in toxoplasmosis is composed of innate and adaptive populations that interact synergistically or antagonistically:

- Antigen-presenting cells (APCs): Macrophages and dendritic cells (DCs) are the first to recognize tachyzoites via pattern recognition receptors (TLRs, NLRs, cGAS-STING), triggering the production of IL-12 and activation of NK and T lymphocytes (Blader; Saeij, 2021).





- Resident cells of the CNS: Microglia and astrocytes are fundamental in containing parasitic dissemination in the brain. Microglia phagocytize cellular debris and release neuroinflammatory mediators, while astrocytes contribute to the blood-brain barrier and cyst formation (Silva et al., 2023).

- CD4⁺ and CD8⁺ T lymphocytes: CD4⁺ cells assist in Th1 polarization and maintenance of immune memory; cytotoxic CD8⁺ cells recognize parasitic peptides presented by MHC-I and eliminate infected cells (Gazzinelli et al., 2023).

- Non-immune cells: Fibroblasts, endothelial cells, and neurons serve as permissive hosts for parasite replication and persistence, adapting their metabolism to withstand oxidative stress and nutritional depletion (Montiel et al., 2022).

While biochemical communication in the microenvironment is mediated by a complex network of signaling molecules:

- Pro-inflammatory cytokines: IFN- γ , TNF- α , and IL-12 are central to activating the Th1 response. IFN- γ induces the expression of enzymes such as IDO (indoleamine 2,3-dioxygenase), which depletes tryptophan, an essential amino acid for the parasite, and stimulates nitric oxide production via iNOS (Gazzinelli et al., 2023).

- Regulatory cytokines: IL-10 and TGF- β modulate the response to avoid excessive tissue damage, but favor chronic persistence by suppressing the activity of macrophages and effector lymphocytes (MONTIEL et al., 2022).

- Chemokines: CXCL9, CXCL10, and CCL2 recruit T cells and monocytes to the infectious focus, establishing gradients that guide leukocyte migration (Silva et al., 2023).

- Reactive species and lipid mediators: ROS and NSRs damage parasitic structures, but in excess cause tissue necrosis. Prostaglandins and leukotrienes derived from arachidonic acid regulate vascular permeability and neutrophil activation (Blader; Saeij, 2021).

Physiologically, these mediators coordinate phagocytosis, antigen presentation, clonal lymphocyte proliferation, and the resolution of inflammation. Pathophysiologically, dysregulated production or parasite evasion results in chronic low-grade inflammation,



neuronal damage, focal demyelination, and a risk of reactivation in immunosuppressed states (WHO, 2023).

Regarding pathophysiological changes in the microenvironment, toxoplasmosis promotes profound structural and functional alterations in the infected tissue. In the acute phase, edema, mixed inflammatory infiltrate, and focal necrosis are observed, especially in lymph nodes, retina, and cerebral cortex. In the chronic phase, cysts containing bradyzoites establish a modified commensal relationship with the host, surrounded by a capsule rich in collagen and ECM glycoproteins, which limits the diffusion of drugs and immune cells (Montiel et al., 2022).

Persistent neuroinflammation is associated with alterations in neurotransmission (dopamine, glutamate), blood-brain barrier dysfunction, and chronic microglial activation, with phenomena correlated to neuropsychiatric manifestations in susceptible individuals (SILVA et al., 2023). Furthermore, the metabolic reprogramming of host cells, with increased aerobic glycolysis and reduced oxidative phosphorylation, creates a microenvironment favorable to parasite survival and cyst formation (Gazzinelli et al., 2023).

The Role of Lysosomes in the Microenvironment of Toxoplasmosis

Lysosomes are dynamic organelles responsible for macromolecular degradation, recycling of cellular components, antigen presentation via MHC-II, and regulation of metabolic and immune signaling pathways. In the context of toxoplasmosis, their role is dual: they act as effectors of host defense and, simultaneously, are targets of subversion by the parasite (Khaminets et al., 2022).

Physiologically, lysosome-phagosomal fusion degrades internalized pathogens, while lysosomal autophagy removes damaged organelles and controls intracellular replication. Activation of the TFEB transcription factor, in response to cellular stress, induces lysosomal biogenesis and exocytosis of hydrolytic enzymes, strengthening the antimicrobial response (Khaminets et al., 2022).



However, *T. gondii* has evolved sophisticated mechanisms to avoid lysosomal fusion with its parasitophorous vacuole (PV). Proteins secreted by density granules (GRAs) and rhoptries (ROPs) recruit host proteins that coat the PV, preventing acidification and the delivery of hydrolases (SAEIJ et al., 2021). In addition, the parasite hijacks lysosomal calcium signaling pathways and modulates the mTORC1 pathway, suppressing autophagy and redirecting vesicular trafficking for nutrient acquisition (Khaminets et al., 2022).

In the chronic phase, lysosomal dysfunction contributes to cyst persistence. Reduced proteolytic capacity, associated with lipofuscin accumulation and cellular senescence, limits bradyzoite elimination and favors an immunosuppressive microenvironment (Yamamoto et al., 2022). On the other hand, pharmacological or genetic induction of lysosomal autophagy has shown promise in reducing parasite load and reversing immune tolerance, highlighting the lysosome-autophagy axis as an emerging therapeutic target (Khaminets et al., 2022).

In summary, immune parasitology combines knowledge from immunology and parasitology (the study of parasites and parasitism) in microenvironment to understand the dynamics of the parasite-host relationship from the perspective of the immune system response. Thus, Natural Products may help the both way of study: parasitic charge effects as showed, besides immune response (Gomes et al., 2023; 2023a).

De Souza and colleagues (2021) demonstrated that cyclooxygenase 2 modulates *T. gondii* infection, immune response, and the formation of lipid droplets in human trophoblastic cells and villous explants.

Punica granatum extract could also have an effect on host cells by inhibiting NF- κ B activation, as it has been demonstrated in chondrocytes and microglial cells. The plant extract could therefore inhibit the transcription of NF- κ B-dependent anti-apoptotic genes and the subsequent extension of host cell lifespan, as described in fibroblasts infected with *T. gondii* tachyzoites (Elkoraichi et al., 2025).

These results indicate that the activity of the plant is not only dependent on the type of extract, but also on the target species (Ebong; Owu; Isong, 1999; Annan et al., 2012; Soundararajan; Sreenivasan, 2012; Abubacker; Deepalakshmi, 2013; De Carvalho; Caramujo,





2018; Tepongning et al., 2018; Bisht; Bankoti; Bharti, 2021; Tow et al., 2021; Ouattara et al., 2025; Varricchio et al., 2026; 2026a).

Therefore, the microenvironment in toxoplasmosis constitutes an integrated and dynamic system, in which cellular interactions, biochemical mediators, and physicochemical conditions determine the outcome of the infection. The transition from the acute to the chronic phase is sustained by an immunometabolic reprogramming that favors the persistence of *T. gondii* in tissue cysts, especially in the CNS. In this scenario, lysosomes play a paradoxical role: they are essential components of the innate immune response, antigen presentation, and autophagy, but are actively subverted by the parasite to ensure its intracellular survival.

Understanding microenvironmental dynamics and lysosomal biology in toxoplasmosis not only elucidates fundamental mechanisms of pathogenesis but also opens perspectives for targeted therapeutic interventions. Strategies that restore lysosomal function, modulate autophagy, or interrupt VP evasion may represent viable alternatives for controlling chronic infection and preventing reactivation. Future research should integrate omics approaches, three-dimensional human tissue models, and high spatial resolution in vivo assays to map microenvironmental evolution in real time, consolidating the basis for precision medicine applied to chronic parasitic infections.

Some natural products are capable of interfering with the *T. gondii* cycle. Once again, the actions of plant species originating from the African and Asian continents are observed, which may not only be due to the active substances produced but also to investment in research with natural products by various countries on these continents (Nagamune et al., 2007).

Finally, it is known that natural products are not indicated for adjuvant treatment of Toxoplasmosis, as they may exhibit drug interactions: Resveratrol may interact with anticoagulants, p450; Curcumin in high doses has gastrointestinal effects and potential drug interactions. They should never be used as substitutes for conventional prescribed treatment, especially in pregnant women, immunosuppressed individuals (post-transplant patients), and immunosuppressed patients with toxoplasmic encephalitis (CDC, 2024b).





Natural products are a promising source of "lead" compounds, but they should not be used in isolation to treat patients with proven toxoplasmosis. Prioritize robust translational research (standardization, toxicology, PK/PD, assays in chronicity models). The ethical evaluation of translational use should involve pharmacologists, parasitologists, and toxicologists in the design of the assays.

As already discussed by Elkoraichi et al. (2025), common plants offer diverse therapeutic options for personalized medicine and make treatments more affordable and easier to obtain. It preserves traditional knowledge and promotes sustainable practices and reduces reliance on synthetic chemicals. The discovery of plant-based treatments of toxoplasmosis can enrich traditional medicine by validating cultural practices, enhancing treatment efficacy, promoting sustainability, and creating economic opportunities.

As discussed by Varricchio and colleagues (2026; 2026a), this approach not only benefits individual health but also leads to a more comprehensive and effective healthcare system and contributes to broader societal and environmental goals aiming public policies to population (César et al., 2026), specially those people under vulnerability and risk.

LIMITS

Although a comprehensive approach was adopted, several clinical, immunological, and epidemiological aspects were undoubtedly not emphasized.

CONTRIBUTIONS

The authors sought to present the importance of toxoplasmosis from a broad perspective, addressing aspects such as mortality, the impact of host immunity, and the significance of refined studies evaluating natural products that may reduce the harmful effects of this important parasitic infection.





PRODUCT

SAPB-LIPAT-FF/UFRJ Project: **TOXOPLASMOSIS, REFUGÉES, ENVIRONMENTAL AND SPIRITUAL HEALTH EVENT**, 2018. Link available at: https://sites.google.com/view/lipat/sapb-eventos_sapb?authuser=0#h.67d9h2wossw0. Accessed in 2026, March 24.

CONCLUSION

This study aimed to analyze the concept of microenvironment applied to toxoplasmosis, describing its local characteristics, the main cells involved, the biochemical mediators, the associated pathophysiological alterations, and, with emphasis, the functional role of lysosomes in these contexts. The approach integrates knowledge from immunology, cell biology, and parasitology, based on recent scientific literature and updated guidelines.

Here is a long-standing clinical diagnosis, specially in adults immune compromised that still deserves contemporary consideration: Toxoplasmosis.

Authors declare there's no conflict of interest.

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