International Journal of Health Systems and Medical Sciences
ISSN: 2833-7433
Volume 05 Number 01 (2026)
Impact Factor: 10.87
SJIF (2023): 3.656

www.inter-publishing.com

Article

Cellular and Molecular Mechanisms of Toxoplasma Gondii Infection and Its Role in Neuroinflammation

Mona Adel Ismail[*1]

1. Al-Furat Al-Awsat Technical University, Technical Institute / Kufa, Department of Medical Laboratory Techniques/Iraq

* Correspondence: mona.almontafaki@atu.edu.iq

Citation: Ismail M. A. Cellular and Molecular Mechanisms of Toxoplasma Gondii Infection and Its Role in Neuroinflammation. International Journal of Health Systems and Medical Sciences 2026, 5(1), 52-60.

Received: 12th Dec 2025
Revised: 17th Jan 2026
Accepted: 20th Feb 2026
Published: 06th Mar 2026

Abstract: Toxoplasma gondii is a protozoan parasite that is an obligate intracellular animal and can infect almost any type of nucleated cell, but it shows a specific predilection to neural and muscular tissue. Chronic infection is also described as development of tissue cysts within the brain in which the parasite exists in a latent form and can lead to the development of long term neuropathological changes. This review presents an overview of the most important cellular and molecular processes that are involved in the T. gondii infection and the contributions these would make to neuroinflammation. After host cell invasion, the parasite invades to form a special parasitophorous vacuole which eludes lysosomal degradation and the regulation of host signaling pathways by releasing effector proteins in rhoptries and dense granules. Such effectors regulate immune reactions by changing NF-κB, STAT and interferon-7-mediated pathways to favor parasite survival as well as to influence the inflammatory milieu. The infection within the central nervous system causes the activation of the microglia and astrocytes resulting in the release of pro-inflammatory cytokines including interleukin-1β, interleukin-6, tumor necrosis factor-alpha, and interferon-gamma. Although these reactions are necessary to regulate the replication of the parasites, the prolonged activation is involved in the formation of oxidative stress, synaptic dysfunction, and neuronal damage. The impairment of blood-brain barrier and changes in neurotransmitter systems, especially to glutamate and dopamine systems also contribute to the worsening of neuroinflammatory mechanisms. Parasite-derived protein molecular interactions with the immune mediators are key in the regulation of parasite survival and pathology mediated by the immune system. The knowledge of these mechanisms is vital in the understanding of the relationship between chronic toxoplasmosis and neuropsychiatric disorders and can help to develop specific immunomodulatory and antiparasitic strategies.

Keywords: Toxoplasma Gondii, Neuroinflammation, Microglia, Cytokines, Molecular Mechanisms

1. Introduction

Toxoplasma gondii, an obligate intracellular protozoan parasite belonging to the phylum Apicomplexa, is one of the most successful parasites in the world. With an estimated 30% of the world's population being infected, it is an important public health concern 1. Infection can occur through ingestion of oocysts, consumption of undercooked meat containing bradyzoite cysts, or vertical transmission from the mother to fetus [1], [2]. Tachyzoites and bradyzoites are the two stages of T. gondii. During the acute phase of the infection, multiplication occurs primarily in various parenchymal tissues, such as the liver, lungs, skeletal muscles, and heart. During the chronic phase of infection, T. gondii exists in the form of bradyzoite-containing cysts, particularly in the brain and muscle tissue. Because bradyzoites are able to survive for extended periods of time, T. gondii persists in

a latent form, unobserved by the host immune system. While oligodendrocytes, microgliac, neurons, and other cell types have been documented as potential target cells for T. gondii [3], the initial parasite entry routes and the parasitic stage involved remain under-investigated. Oocysts, present in contaminated water or food, are the main source of T. gondii. T. gondii can infect local cells in the intestine and spread throughout the body after initial immune escape, leading to widespread infection [4], [5].

2. Materials and Methods

This review article followed a methodology based on using previous published scientific literature concerning the cellular and molecular mechanisms of Toxoplasma gondii infection and the association of such an infection with neuroinflammation. To aggregate existing knowledge on parasite life history traits, host–parasite interaction and responses of the central nervous system, relevant peer-reviewed articles, reviews and experimental studies were systematically reviewed. Information integrating T. gondii staff, neuroinflammatory microglia, and cytokine signaling with blood-brain barrier disruption in various species was identified as conceptually primary from scientific databases including PubMed, Scopus, and Google Scholar. Studies outlining the life cycle of the parasite, immune responses of the host, changes to neurons and glia, and intracellular and extracellular diagnostic biomarkers were prioritized to maximize coverage of both cellular and molecular aspects. Collected literature was screened for relevance, scientific credibility and recency, with a focus on studies that explained mechanisms of parasite invasion, persistence and modulation of immunity in neural niche. Information collected from these logical sources was then organized thematically to describe the order of events following infection, including the entry and dissemination of parasites, immune activation, and chronic persistence within the brain. Furthermore, experimental evidence from animal models and in vitro studies were incorporated to decipher processes involved in neuroinflammatory responses induced by T. gondii infection. These included key pro-inflammatory cytokine signaling pathways, activation of microglia and astrocytes, and abnormal neurotransmitter systems that contribute to neuronal dysfunction and inflammatory pathology. This structured synthesis of the literature to build a conceptual framework to explain how parasite-derived factors and host immune responses interact to drive neuroinflammation and ultimately long-term neuropathological sequelae during chronic toxoplasmosis was made possible by the methodology.

3. Results and Discussion

Biology and Life Cycle of Toxoplasma gondii

Toxoplasma gondii (T. gondii) is an obligate parasitic protist belonging to the phylum Apicomplexa that is responsible for one of the widespread parasitic infections among warm blooded animals [6]. Following entry into the body, T. gondii infects a number of different tissues, organs and cell types but it is able to invade and persist in the nervous system, a key anatomical region in host–parasite interaction, including transmission and pathogenesis. Two stages of the life cycle are notably involved in the transmission of the parasite among warm blooded animals: the tachyzoite and the bradyzoite. Tachyzoites (active forms) are responsible for acute infections and are rapidly generated after host cell invasion; they disseminate rapidly throughout the body via blood circulation. Bradyzoites differentiate to form cysts (persistent forms) that are able to infect other hosts; they are responsible for chronic infections and are generated after some days of infection in the nervous system [7], [8]. T. gondii undergoes both sexual and asexual reproduction. The sexual cycle occurs in the intestinal epithelium of cats and feline excreta are the main sources of infection in warm-blooded animals. The asexual cycle takes place

during infection within the host and involves both tachyzoites and bradyzoites. The life cycles of the parasite and the role of different organ systems during the various steps of infection become particularly relevant when considering T. gondii infection in the nervous system, because the parasite is able to invade all the cell types found in the central nervous system including neurons and glial cells [9], [10].

When T. gondii enters the body, the parasite first disseminates through lymph, blood and different organs, and then reaches the nervous system where it can persist and reactivate later. During the acute phase of infection the parasite exposition is sufficiently high to produce detectable lesions in the central nervous system and consequently immune responses that, if exaggerated, may damage crucial anatomical areas. Throughout T. gondii life cycle bradyzoite-containing cysts are able to endure unfavorable conditions and persist inside the host lifetime [11], [12].

Entry, Dissemination, and Host Cell Interactions

Toxoplasma gondii is an intracellular parasite capable of infecting all warm-blooded vertebrates, including humans. Acute infection is usually asymptomatic in healthy individuals and is often detected incidentally through serological tests [13], [14]. However, the parasite can spread to the central nervous system (CNS) after hematogenous dissemination, where it can survive in a dormant State for a long time, causing neuroinflammation. T. gondii infection is the most prevalent zoonosis worldwide, yet relatively little is known about the cellular and molecular mechanisms underlying its propagation in the CNS. Even after decades of research, many aspects of the biology of the Cell and the immune response mediated by it in the CNS remain to be understood [15]. The purpose of this review is to present the cellular and molecular processes of the entry, dissemination, and spread of T. gondii in the CNS, the role of the parasite and the host immune response in the occurrence of neuroinflammation [16].

The acute phase of T. gondii is a time when the tachyzoite form of T. gondii proliferates at a rapid rate. The spread of the parasites within the organism is also determined by its ability to penetrate, reproduce, and then leave different types of cells. The T. gondii genome encodes over 60 invasion-related proteins, and additional virulence factors further enhance parasite dissemination [17], [18]. Most T. gondii strains enter through the oral route after the ingestion of contaminated food or water. Upon reaching the stomach, T. gondii oocysts are released and invade the intestinal epithelium [19], [20]. Most oocysts are cleared by the immune response, while the remaining ones differentiate into bradyzoite cysts that evade the immune system by residing inside cells. Alternatively, oocysts can be ingested by a variety of animals, including birds, pigs, and other mammals, establishing exogenous life cycles. In all these hosts, the parasite can enter the CNS in approximately 6 days. Nonetheless, dispersal remains limited, as encephalitic-type strains cannot cross the blood-brain barrier [21], [22].

Immunological Responses to Toxoplasma gondii

Toxoplasma gondii stimulates the innate immune system and its cytokine production through various pattern recognition receptors (PRRs), including Toll-like receptors (TLRs), nucleotide-binding oligomerization domain-like receptors (NLRs), and cyclic GMP-AMP synthase (cGAS) following its entry into the host. Tachyzoites and bradyzoites, the two infective stages of T. gondii, induce the production of several proinflammatory cytokines, including interleukins (IL) -1β, 6, 12, 18, 23, tumor necrosis factor (TNF)-α, interferon (IFN)-γ, and chemokines [23], [24]. Infected cells secrete these cytokines locally, which activate neighboring uninfected cells and, together with chemokines, signal the recruitment of antigen-presenting cells (APCs), such as dendritic cells (DCs) and macrophages, to the entry site. Infection of macrophages, dendritic cells, microglia, and astrocytes occurs in various peripheral organs at early stages, whereas in the mammalian central nervous system (CNS), microglia and astrocytes become infected. During the acute phase of T. gondii infection, the innate immune response generates a

protective and long-lasting adaptive immunity. T. gondii activates the type I cytokine cascade through the recognition of parasite nucleic acids by cytosolic cGAS that leads to cGAMP synthesis and transmission of signals via STING [25], [26].

Toxoplasma gondii–Mediated Neuroinflammation

Toxoplasma gondii is an obligate intracellular parasite that has the capability of infecting a wide range of warm-blooded animals, including humans. Infection in humans may subsequently lead to enormous socio-economic burden between increased medical cost and productivity loss. The infection is closely related to acquired immune deficiency syndrome (AIDS), and congenital transmission in a fetus may cause foetal malformations or even death in severe cases. When low-virulent strains of T. gondii infect certain rodent species, their innate aversion to odour cues left by infected cats (the final hosts of T. gondii) is abolished. The role of congenital infection in mental illness and behaviour abnormalities among children has drawn public attention [27].

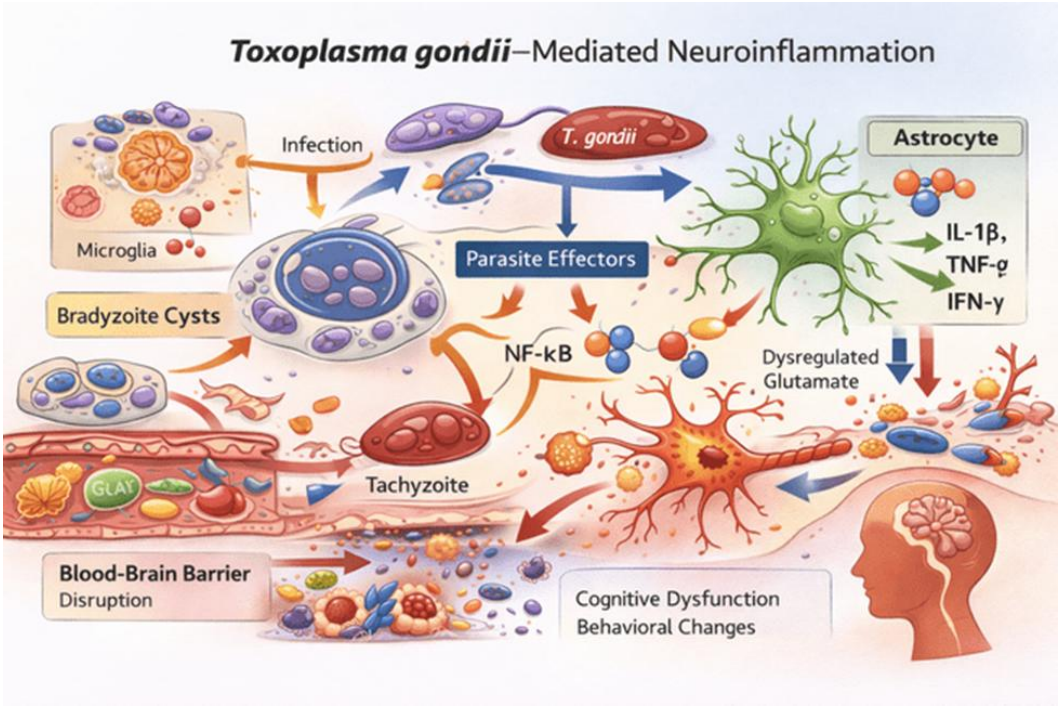

Figure 1. Pathogenic Mechanisms of Toxoplasma gondii–Mediated Neuroinflammation in the Central Nervous System.

Mechanisms of Blood–Brain Barrier Disruption and Parasite Persistence

Toxoplasma gondii efficiently reaches the mammalian central nervous system (CNS) from the circulation and persists in a chronic form. A major pathway of CNS access occurs before the full development of the immune response in acute systemic infection [28]. The parasite infects brain-resident immune cells, which migrate across the blood–brain barrier (BBB) carrying viable tachyzoites and establishing infection in neurons and astrocytes 29. There, these parasites undergo differentiation into cyst-forming bradyzoites and enter the dauer (retention) phase, which allows long-term persistence without replication. CNS access and maintenance of the dauer phase are important for T. gondii chronicity [29], [30].

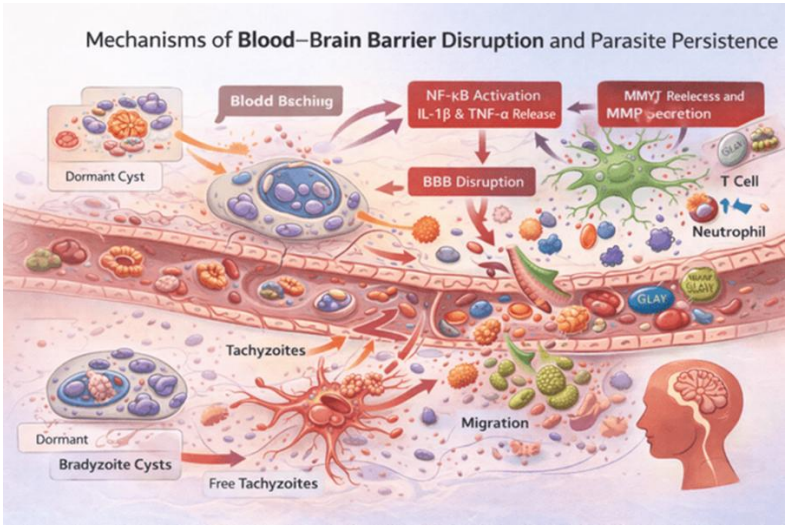

Figure 2. Cellular and Molecular Mechanisms of Blood–Brain Barrier Disruption and Toxoplasma gondii Persistence in the Central Nervous System.

Neuronal and Glial Pathophysiology in Toxoplasma gondii Infection

Toxoplasma gondii infection has significant effects on the biochemical machinery of neurons and glia. These effects are closely related to behavioral and psychiatric disorders that have been linked to infection. Analysis of neuronal and glial pathophysiology in experimental models provides insight into the manner in which this parasite mediates neural alterations. Infection results in the alteration of neurotransmitter levels such as glutamate and GABA, along with the formation of glial scars and excessive synthesis of complement factors such as C3. Such alterations are often accompanied by seizure activity in chronically infected models [31], [32].

Neuronal alterations include increased turnover of glutamate and GABA. Increased synaptic remodeling is evident by increased levels of phosphorylated Rps6 and Arc, together with a higher number of dendritic spines [33], [34]. The acquisition and release of excitatory signals is higher during the chronic phase of the infection. The excessive neurotransmitter turnover together with the induction of complement pathways leads to extensive excitotoxic damage, ultimately resulting in loss of neurons in the chronic phase. The induction of C3 also seems to enhance parasitic survival. Glial scars are frequently observed in many chronic diseases and are instead beneficial early during the infection, blocking the dissemination of the parasite to healthy cells [35], [36].

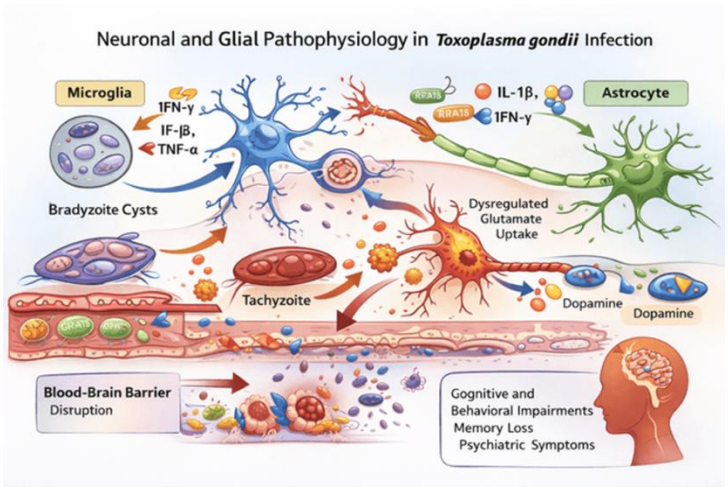

Figure 3. Neuronal and Glial Pathophysiological Alterations during Toxoplasma gondii–Induced Neuroinflammation.

Imaging, Biomarkers, and Diagnostic Considerations

The characteristics of CNS pathology in Toxoplasma gondii infection have led to the development of neurological and biological biomarkers for diagnostic purposes. Many cytokines, microRNA and parasite DNA have been analyzed as possible biomarkers in mouse models and other CNS imaging patterns reported in mice infected with T. gondii. Although these tools have shown various uses, a number of constraints have impact on their eventual usefulness [37].

A number of imaging methods give specific markers which, combined, are compatible with an infection of T. gondii. Widely used are positron emission tomography (PET) with 18F-fluorodeoxyglucose (18F-FDG), CT and MRI. Higher glucose consumption of the brain as monitored by 18F-FDG PET in a mouse model is an indication of active neuroinflammation. Increased 18F-FDG uptake has been also reported in cerebral toxoplasmosis. Other radiotracers including 11C-(R)-PK11195 and 18F-DPA-714 both bind to translocator protein (TSPO) present in the activation of microglia result in visible CNS lesions in the chronic model. During chronic infection, specific areas of interest such as the cortex, striatum and white matter are specifically affected. Both inflammation and parasite habitat can be represented in these types of examination [38].

At the biomarker level, the infection-related signals have been found to be proinflammatory cytokines including IL-1B, IL-6, TNF-alpha, and IFN- gamma in the serum and exosomes circulating microRNAs. The Cxcl10 mRNA can be observed in the brains of acutely and persistently infected murine models [39]. Specific brain origin signatures, including interferon (IFN) and mitochondrial stress response, are found in CD8+ T cells from T. gondii-infected mice. Detection of T. gondii DNA in serum has been proposed as a diagnostic criterion of this parasitic infection. However, the sensitivity and specificity of these cytokines, microRNAs, and DNA markers remain suboptimal for reliable diagnosis [40].

The above imaging signatures and biomarker candidates have been evaluated both in the early and advanced stages of infection, demonstrating their potential for longitudinal monitoring. Nevertheless, these approaches apply only to extrapolated animal models, precluding direct correlation with human acquisition and course of illness [41].

Therapeutic and Translational Implications

Toxoplasma gondii is an obligatory intracellular parasite that causes infection in a large variety of mammalian, avian and human hosts. Asymptomatic most infections; acute T. gondii infection can lead to the development of cysts in the central nervous system (CNS) and reactivation of these latent cysts is fatal in immunocompromised individuals. Although the CNS infection of T. gondii is controlled by the immune system, the presence of the infection causes chronic inflammatory changes [42], [43]. In line with the above, preclinical infection models of T. gondii infection have shown that acute or chronic T. gondii infection can induce neuroinflammation and result in neuronal, and glial physiological alterations, even in genetically resistant mice. These models have T. gondii-infected neurons with neurodegeneration, neurotransmitter disintegration, and unsuccessful electrical activity, and the role of these changes in causing behavioral changes has been examined 1. Simultaneously, astrocytes and microglia experience neuroinflammatory reactivity, and the expression of cytokines, chemokines, and neurotrophic factors change and the functional alteration is observed [44].

Gaps in Knowledge and Future Directions

Primary gaps include knowledge of multifaceted host factors influencing dissemination and persistence, the interplay between parasite stage and neural invasion, and the contribution of $CD8^+$ T cells to cyst maintenance. In vivo model trade-offs asynchronous and anti-inflammatory treatments in acute polyclonal models; differential

epitope determinants, infection routes, and parasite persistence in chronic models further hinder translational advances. Toward an integrated framework, research priorities involve elucidating key ili circular σ factors and ensP/Δ, upstream cholera-tetanus adjuvants and parasite-specific signals packaged in $CD11c^{+}$ exosomes, the role of parasite PI3P-acquisition effectors, temporal regulation of cytokine production by polymorphic effectors, the impact of cysts on central-rest state commandeering, strategies to mitigate immune tuning by circulating IL-6, and the relationship between tissue-cyst formation, immune states, and bead-trapping frequencies [44].

Methodological exploration should encompass maximal signal perturbation in T. gondii superinfection and transmissibility; metabolic network cooptation during cumulative T. gondii delivery; intracellular-microenvironment modulation, host-ribosome deviation, and potential drug synergy in T. gondii–Schistosoma coexposure; continuous in vivo monitoring of cyst maturation; and establishment of multiple-epitope dual-tracer tools for dissecting archival anti-T. gondii modules and mapping local parasitic-circuit interactions [45].

4. Conclusion

Toxoplasma gondii is an obligate intracellular parasite of the phylum Apicomplexa with drastic effects on host behavior and brain neurotransmitters. After acute infection it can reside in the brain for the life of the host. Parasite load is often low or undetectable during chronic infection, yet the accompanying inflammatory response can lead to neuroinflammation. Peripheral infections can trigger further inflammatory cascades in the central nervous system. The cellular and molecular architecture of the host response to T. gondii infection is complex and largely intact during chronic infection, which is uniquely advantageous for studying T. gondii–host interactions. Other viruses, bacteria, and parasites that target the brain tend to disrupt host signaling mechanisms by blocking cell cycle or apoptosis pathways in order to replicate unnoticed and remain latent. As far as is known, T. gondii does not compromise these fundamental pathways, making it easier to disentangle parasite and virus effects.

Chronic, subclinical infections may prevail in up to a third of the human population and 40% of the world's warm-blooded animals. The central nervous system (CNS), owing to the parasite's predilection for fleshy hosts, is the ultimate site of persistence in its definitive feline hosts and the primary site in aberrantly infected intermediate hosts. These reservoirs include humans, rodents, and other mammals. The natural history of T. gondii is closely linked to the evolution and sexual reproduction of its feline definitive hosts. Unless rodents are exposed to T. gondii while colonies are in their defaecation, the parasite is unlikely to disseminate and evolve within rodent populations. The selective pressure to manipulate the host's behavior and increase predation risk appears to be a driving force in sharpening the neurotropic features of T. gondii. Central infection enables the parasite to hijack behavioural host control to activate extra-cerebral transmission routes into its definitive hosts. Several viral, bacterial and parasitic pathogens that are highly neurotropic and induce prolonged central infections–such as Rabies, Lymphocytic Choriomeningitis, Japanese Encephalitis, Mycobacerium Tuberculosis, Schistosoma spp.–are capable of preventing host-directed responses and subtle metabolomic alterations. These properties provide a unique opportunity for disentangling pathogen–host interactions during cellular and transcriptional processes.

Declaration of Competing Interest

The authors say they don't have any known personal or financial relationships or financial interests that could have seemed to affect the work in this study.

REFERENCES

[1] French, T., et al. "Neuronal Impairment Following Chronic *Toxoplasma gondii* Infection Is Aggravated by Intestinal Nematode Challenge in an IFN-γ-Dependent Manner." 2019.

[2] Sang Hwang, Y., et al. "Characteristics of Infection Immunity Regulated by *Toxoplasma gondii* to Maintain Chronic Infection in the Brain." 2018.

[3] Adem, D., and M. Ame. "Toxoplasmosis and Its Significance in Public Health: A Review." *Journal of Biomedical and Biological Sciences*, 2023.

[4] Symeonidou, I., et al. "A Review of *Toxoplasma gondii* in Animals in Greece: A Food-Borne Pathogen of Public Health Importance." *Animals*, 2023.

[5] Hamad, Anas Abdullah, Hamssa Majid Mustafa, and Osama A. Mohsein. "Detection of the levels of immune cytokines (IL4, IL5, TNF-α) in school-age and preschoolers with an Ascaris lumbricoides infection." Journal of Parasitic Diseases 48.4 (2024): 782-787.

[6] Khairullah, A. R., et al. "A Comprehensive Review of Toxoplasmosis: Serious Threat to Human Health." 2024.

[7] Schlüter, D., and A. Barragan. "Advances and Challenges in Understanding Cerebral Toxoplasmosis." 2019.

[8] Umeda, K., et al. "Transcriptional Profiling of Toll-like Receptor 2-Deficient Primary Murine Brain Cells during *Toxoplasma gondii* Infection." 2017.

[9] Ramírez-Flores, C. J., et al. "Comprehensive Analysis of *Toxoplasma gondii* Migration Routes and Tissue Dissemination in the Host." *PLOS Neglected Tropical Diseases*, 2025.

[10] Ross, E. C., G. C. Olivera, and A. Barragan. "Early Passage of *Toxoplasma gondii* across the Blood–Brain Barrier." *Trends in Parasitology*, 2022.

[11] Cairney, P., and G. McConkey. "Pathophysiological Mechanisms of *Toxoplasma gondii* Infection in the Central Nervous System." *Neurobiology of Disease*, 2025.

[12] Abbas, Zainab Khamis, et al. "Immunological biomarkers and their role in the diagnosis and prognosis of leishmaniasis: A case–control study." Tropical Parasitology 15.1 (2025): 33-41.

[13] Miranda, T. A. V., et al. "Imaging of Central Nervous System Parasitic Infections." *Neuroimaging Clinics of North America*, 2023.

[14] Tawfik, O., et al. "Disseminated Opportunistic Infections Masquerading as Central Nervous System Malignancies." *Human Pathology*, 2022.

[15] Tunali, V., and M. Korkmaz. "Emerging and Re-emerging Parasitic Infections of the Central Nervous System in Europe." *Infectious Disease Reports*, 2023.

[16] Challa, S. "Granulomatous Diseases of the Central Nervous System: Approach to Diagnosis." *Indian Journal of Pathology and Microbiology*, 2022.

[17] Faqri, Ayoob Murtadha Alshaikh, et al. "The role of cytokines in autoimmune diseases: pathogenesis and therapeutic implications." Central Asian Journal of Medical and Natural Science 6.3 (2025): 852-873.

[18] Pan, M., et al. "The Determinants Regulating *Toxoplasma gondii* Bradyzoite Development." *Frontiers in Cellular and Infection Microbiology*, 2022.

[19] Delgado, I. L. S., et al. "The Apicomplexan Parasite *Toxoplasma gondii*." *Encyclopedia*, 2022.

[20] Yang, J., et al. "The Beta Subunit of AMP-Activated Protein Kinase Is Critical for Cell Cycle Progression and Parasite Development in *Toxoplasma gondii*." *Cellular and Molecular Life Sciences*, 2022.

[21] Vizcarra, E. A., et al. "An Ex Vivo Model of *Toxoplasma* Recrudescence Reveals Developmental Plasticity of the Bradyzoite Stage." *mBio*, 2023.

[22] Miyagaki, M., et al. "Ocular Toxoplasmosis: Advances in *Toxoplasma gondii* Biology, Clinical Manifestations, Diagnostics, and Therapy." *Pathogens*, 2024.

[23] Khan, I. A., and M. Moretto. "Immune Responses to *Toxoplasma gondii*." *Current Opinion in Immunology*, 2022.

[24] Sana, M., et al. "Immune Response against Toxoplasmosis—Recent Updates." 2022.

[25] Malik, A. W., et al. "Bacterial strains isolated from sinusitis infections and their drug resistance profiles in Nasiriyah (Iraq)." Regulatory Mechanisms in Biosystems 16.2 (2025): e25049-e25049.

[26] Hu, Z., et al. "Inflammasome Activation Dampens Type I IFN Signaling to Strengthen Anti-*Toxoplasma* Immunity." *mBio*, 2022.

[27] Bakhraibah, A. O. "Warm-Blooded Animal Toxoplasmosis and Its Connection to COVID-19: A Review." *Microbial Biosystems*, 2022.

[28] Olivera, G. C., et al. "Blood-Brain Barrier-Restricted Translocation of *Toxoplasma gondii* from Cortical Capillaries." 2021.

[29] Masocha, W., and K. Kristensson. “Passage of Parasites across the Blood–Brain Barrier.” 2012.
[30] Nayeri, T., S. Sarvi, and A. Daryani. “Toxoplasmosis: Targeting Neurotransmitter Systems in Psychiatric Disorders.” *Metabolic Brain Disease*, 2022.
[31] Shokor, Z. “Human Primary Cell-Based In Vitro Blood-Brain Barrier to Study *Toxoplasma gondii* Brain Invasion and Drug Screening.” 2025.
[32] Cavalari, V. C., et al. “*Toxoplasma gondii*, Endothelial Cells and Schizophrenia: Is It Just a Barrier Matter?” 2025.
[33] Pandya, K., P. N. Tripathi, and D. Kumar. “Neuronal Loss and Synaptic Dysfunction.” *Neurobiology of Aging*, 2026.
[34] Khan, S., et al. “Excitotoxicity, Oxytosis/Ferroptosis, and Neurodegeneration: Emerging Insights into Mitochondrial Mechanisms.” 2024.
[35] Sakban, G. H., et al. "Exploring biomarkers, adipokines, and cytokines in patients with colon cancer: A comparative study with healthy controls." Regulatory Mechanisms in Biosystems 16.3 (2025): e25150-e25150.
[36] Sayehmiri, F., et al. “Mitochondrial Plasticity and Synaptic Plasticity Crosstalk in Health and Alzheimer’s Disease.” 2024.
[37] Boyle, A. J., et al. “PET Imaging of Fructose Metabolism in a Rodent Model of Neuroinflammation with 6-Fluoro-6-Deoxy-D-Fructose.” *Molecules*, 2022.
[38] Soltani, S., et al. “Relationships between Brain Glucose Metabolism Patterns and Impaired Glycemic Status: A Systematic Review of FDG-PET Studies with a Focus on Alzheimer’s Disease.” 2025.
[39] Stormezand, G. N., et al. “Current Developments on FDG PET/CT in Inflammatory Disorders of the Central Nervous System.” 2025.
[40] Palandira, S. P., et al. “A Dual Tracer PBR28 and FDG microPET Evaluation of Neuroinflammation and Brain Energy Metabolism in Murine Endotoxemia.” 2022.
[41] Salvadó, G., et al. “Reactive Astrogliosis Is Associated with Higher Cerebral Glucose Consumption in the Early Alzheimer’s Continuum.” 2022.
[42] Xiao, J. “Behavioral Changes Induced by Latent Toxoplasmosis Could Arise from CNS Inflammation and Neuropathogenesis.” 2022.
[43] Moghaddami, R., M. Mahdipour, and E. Ahmadpour. “Inflammatory Pathways of *Toxoplasma gondii* Infection in Pregnancy.” 2024.
[44] Everett, A., and H. M. Elsheikha. “Neuroinflammation and Schizophrenia: The Role of *Toxoplasma gondii* Infection and Astrocytic Dysfunction.” *Journal of Neuroimmunology*, 2025.
[45] Pires, L. B., et al. “Infection of Mouse Neural Progenitor Cells by *Toxoplasma gondii* Reduces Proliferation, Migration, and Neuronal Differentiation In Vitro.” 2023.