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Toxoplasma gondii-Driven Cytokine Dysregulation as a Differentiating Biomarker Between Schizophrenia and Depression

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Abstract
Schizophrenia and depression are among the most prevalent psychiatric disorders, and both have been mechanistically linked to systemic and neuroinflammatory processes. Infection with the protozoan parasite Toxoplasma gondii has been proposed as a potential trigger of these inflammatory pathways, yet direct comparisons of proinflammatory cytokine profiles between the two disorders in the context of T. gondii infection remain scarce. Consequently, it is still unclear whether schizophrenia and depression share a common toxoplasmosis-associated inflammatory signature or instead display distinct immunological fingerprints. This systematic review aimed to characterize the proinflammatory cytokine biomarker patterns—particularly IL-6, IFN-γ, and TNF-α—among T. gondii-infected patients with schizophrenia and depression. Conducted in accordance with the PRISMA 2020 guidelines and the PICO framework, the review searched PubMed Central, PubMed, ScienceDirect, and Scopus for original English-language studies published between 2021 and 2025. Of 454 records initially retrieved and screened for eligibility using Mendeley Reference Manager, five studies satisfied all inclusion criteria. The synthesis revealed that toxoplasmosis-associated schizophrenia was characterized by elevated MCP-1, RANTES, GRO-α, IP-10, and IL-8, along with reduced MIP-1α, MIP-1β, and IL-8, whereas toxoplasmosis-associated depression was primarily characterized by increased IL-6 and TNF-α. These divergent cytokine patterns suggest that chronic T. gondii infection induces disorder-specific immune dysregulation rather than a uniform inflammatory response. Such distinct profiles may hold promise as biomarkers for differentiating infection-related psychiatric subtypes and could inform the development of more targeted, immunomodulatory therapeutic strategies for affected patients.
Keywords: Depression, Immune Biomarkers, Neuroinflammation, Schizophrenia, *Toxoplasma Gondii*

INTRODUCTION

Mental disorders arise from disruption of the brain regions that govern emotion, mood, consciousness, and cognition, giving rise to conditions such as schizophrenia, depression, bipolar disorder, anxiety disorders, neurodevelopmental disorders, substance-use disorders, and stress-related disorders (Kip & Parr-Brownlie, 2023). Among these, schizophrenia and depression stand out for their global burden: the World Health Organization estimates that schizophrenia affects roughly 0,7 – 1% of the population, while depression affects an estimated 3,8%, or approximately 280 million people, worldwide (Li et al., 2022; Maulana et al., 2022). Beyond their individual burden, schizophrenia and depression also frequently co-occur or share overlapping symptom domains, such as anhedonia, cognitive impairment, and social withdrawal, which has prompted increasing interest in transdiagnostic biological mechanisms that might explain this clinical overlap. Despite their distinct clinical presentations, the two disorders appear to converge on shared genetic and clinical pathways, with systemic

inflammation and immune dysregulation among the most consistently implicated (Perry et al., 2021).

When the immune response becomes excessive, it can injure host tissue, an effect that is particularly consequential in the brain given its restricted capacity for neuronal regeneration. Microglia and astrocytes, the brain's resident phagocytic cells, can amplify neuroinflammation and neuronal injury once activated, and neuroinflammation itself is now recognized as a shared mechanism underlying neurodegenerative, psychiatric, traumatic, demyelinating, ischemic, and epileptic conditions alike (Gorji, 2022; Kip & Parr-Brownlie, 2023). This glial activation cascade can become self-reinforcing, as activated microglia and astrocytes release additional cytokines and chemokines that recruit and activate further immune cells within the central nervous system, prolonging the inflammatory state. Some of these peripheral cytokines can also cross a compromised blood-brain barrier, or signal indirectly via vagal and humoral pathways, providing additional routes by which systemic inflammation can influence central nervous system function. Consistent with this, multiple studies link mental disorders to elevated concentrations of C-Reactive Protein (CRP), IL-1β, TNF-α, and IL-6 in blood, cerebrospinal fluid, and brain tissue; when these proinflammatory cytokines rise, they can disturb neurotransmitter signaling and, combined with genetic susceptibility and glutamatergic dysregulation, hasten the onset of mental disorders (Chang et al., 2024; Lv et al., 2024).

Parasitic infection is a further, less widely recognized driver of neuroinflammation and mental-disorder risk (Kip & Parr-Brownlie, 2023). *Toxoplasma gondii*, an apicomplexan protozoan with a complex, multi-stage life cycle, infects an estimated one-third of the world's population; seroprevalence varies widely, from 10% to 90%, depending on age, climate, dietary habits, and exposure to domestic cats (Bisetegn et al., 2023; Poursalar et al., 2025; Wang et al., 2023). Infection is usually asymptomatic and remains latent in immunocompetent hosts, but in immunocompromised individuals it can become life-threatening and provoke marked behavioral change (Bisetegn et al., 2023; Poursalar et al., 2025; Virus et al., 2021). Even in its latent form, however, the parasite is not necessarily immunologically silent, and a growing body of evidence suggests it can continue to shape host immune and neurological function over the long term (Eberhard et al., 2025; Seizova et al., 2022). This pattern of chronic, subclinical persistence has made toxoplasmosis a focus of growing interest in psychiatric epidemiology, since a substantial proportion of the general population may carry a long-term parasitic burden without overt clinical symptoms, potentially confounding studies of psychiatric risk factors that do not account for infection status (Flegr & Horáček, 2020).

Felines serve as the definitive host in the *T. gondii* life cycle. Cats shed oocysts in their feces, which become infectious within one to five days and can then persist for years in soil or water (Jańczak et al., 2025), contaminating shellfish, vegetables, and water supplies (Thebault et al., 2021; Ureña et al., 2022). Humans and other warm-blooded animals typically acquire infection by ingesting these mature oocysts from contaminated sources, through direct contact with cat feces, or by consuming raw or undercooked meat containing tissue cysts; vertical transmission during pregnancy and, less commonly, transmission via blood transfusion or organ transplantation represent additional routes (Friesema et al., 2023). Once established, *T. gondii* infection–toxoplasmosis–can reshape host behavior by acting on the forebrain and amygdala. The parasite raises glutamate production, colonizes an estimated 30% of brain microglia and 10% of astrocytes, and drives neuroinflammation (Rantala et al., 2022). These behavioral and neurochemical effects are thought to arise, at least in part, from the parasite's tendency to encyst preferentially within neural tissue, where bradyzoite-containing cysts can persist for the lifetime of the host and continue to provoke a low-grade local immune response. Poursalar et al. (2025). Reported that elevated proinflammatory cytokine concentrations were

linked to behavioral change and suicidal ideation among individuals with depression, with IL-6, IFN-γ, and TNF-α being the cytokines most characteristic of toxoplasmosis in this context. Similarly, Everett & Elsheikha (2025) found that the proinflammatory response triggered by *T. gondii* infection raises IL-1β, IL-6, and chemokine expression, contributing to greater schizophrenia severity when toxoplasmosis is present.

Taken together, a growing body of literature links schizophrenia and depression, considered separately, to elevated proinflammatory cytokines in *T. gondii*-seropositive individuals (Everett & Elsheikha, 2025; Harsanyi et al., 2023), and other work has compared proinflammatory biomarker profiles between the two disorders directly (Yu et al., 2024). What remains scarce, however, is research examining *T. gondii* infection in relation to both psychiatric disorders within a single comparative frame. Clarifying whether schizophrenia and depression share a common toxoplasmosis-related inflammatory signature, or instead display distinct immune fingerprints, could help clinicians better interpret elevated inflammatory markers observed in psychiatric patients with confirmed or suspected *T. gondii* exposure. To our knowledge, this is among the first reviews to systematically juxtapose proinflammatory cytokine data from schizophrenia and depression within the same methodological frame, rather than treating each disorder's association with toxoplasmosis in isolation, which constitutes the novel contribution of the present work. To help close this gap, the present review examines the patterns of proinflammatory cytokine biomarkers–specifically IL-6, IFN-γ, and TNF-α–in patients with schizophrenia and depression who are infected with *T. gondii*, with the aim of characterizing disorder-specific immune signatures and clarifying the contribution of parasitic infection to neuroinflammatory processes.

RESEARCH METHODS

This review was conducted in accordance with the PRISMA 2020 guidelines (Preferred Reporting Items for Systematic Reviews and Meta-Analyses), and the review question was structured using the PICO framework. The population (P) comprised individuals diagnosed with schizophrenia or depression; the exposure of interest (I) was infection with *T. gondii*, established through seropositivity or another confirmed diagnostic method; the comparison (C) consisted of *T. gondii*-seronegative individuals, uninfected controls, or comparison groups defined by diagnostic status within the same study; and the outcome (O) was the concentration or expression level of proinflammatory cytokines and chemokines. Because cytokine and chemokine nomenclature can vary across laboratory platforms and research groups, outcome measures were mapped to standard nomenclature (for example, treating CCL2 as a synonym of MCP-1) during data extraction to enable consistent comparison across the five included studies. This framework guided both the search strategy and the eligibility criteria described below.

We searched four databases–PubMed Central (PMC), PubMed, ScienceDirect, and Scopus–using the search string "cytokine" AND "toxoplasmosis" AND "schizophrenia" AND "depression". The search was restricted to original research articles published in English between 2021 and 2025 that reported cytokine changes in patients with schizophrenia or depression who had confirmed *T. gondii* infection. The five-year publication window was selected to capture the most recent and methodologically current evidence on this emerging research question, while the language restriction to English helped ensure consistent data extraction and interpretation. Reference management and duplicate removal were performed using Mendeley Reference Manager. Studies were included if they were original research reporting quantitative measurements of proinflammatory cytokines or chemokines in patients with schizophrenia or depression in relation to *T. gondii* infection status. Studies were excluded if they were duplicate records; lacked an accessible full-text PDF; were not published in English;

were literature reviews, editorials, meta-analyses, commentaries, case reports, or theses rather than original research; addressed topics irrelevant to the review question; reported outcomes other than cytokine measures; or were conducted in animal models.

The search across the four databases returned 454 records. Before screening, 321 records were removed–103 as duplicates, 3 flagged as ineligible by automation tools, and 215 for other reasons–leaving 133 records for title and abstract screening, of which 1 was excluded. Full-text reports were then sought for the remaining 132 records; 2 could not retrieved, leaving 130 reports assessed for eligibility. Of these, 125 were excluded for the following reasons: no accessible full-text PDF (n = 22), literature reviews, editorials, meta-analyses, commentaries, case reports, or these (n = 53), irrelevant topic (n = 24), outcomes other than cytokine measures (n = 21), and animal experiments (n = 5). Five articles ultimately met all inclusion criteria and were included in this review (Figure 1).

For each included study, we extracted the authors and year of publication, country, mental disorder diagnosis, participant characteristics and group definitions, and reported proinflammatory cytokine findings; these details are summarized in Table 1. Given the heterogeneity in study populations, cytokine panels measured, and outcome reporting across the five included studies, a narrative synthesis approach was used to compare cytokine profiles between schizophrenia and depression, rather than a pooled statistical meta-analysis. This narrative approach also allowed the review to attend to differences in how each study defined and diagnosed schizophrenia and depression, since diagnostic criteria and disease-severity measures were not uniformly reported across the five included studies. The implicit hypothesis guiding this review was that *T. gondii* infection is associated with detectable, and potentially disorder-specific, alterations in proinflammatory cytokine profiles among patients with schizophrenia and depression.

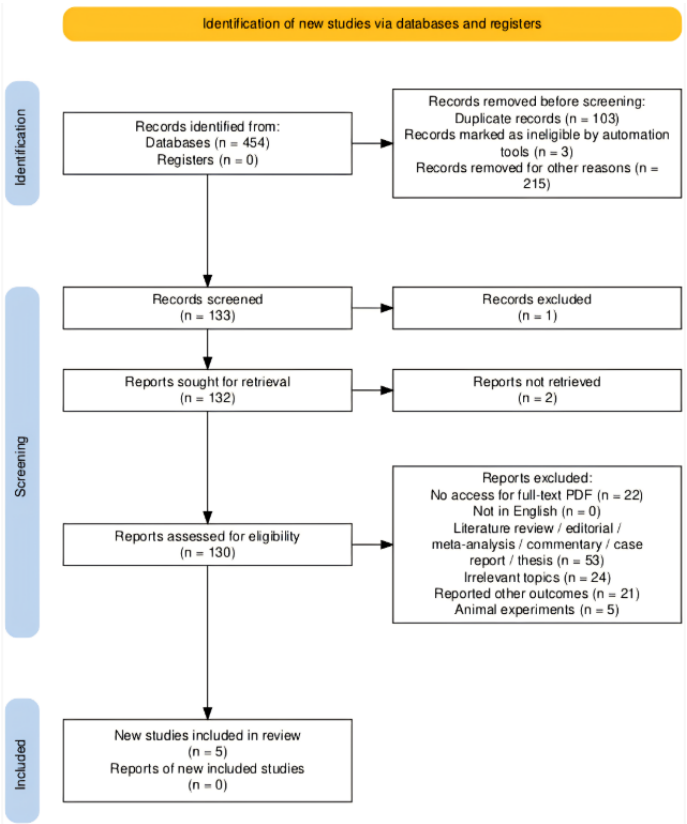

Figure 1. PRISMA Flow Diagram of the Study Selection Process
Source: Adapted from (Haddaway et al., 2022)

RESEARCH RESULTS AND DISCUSSION

Research Result

The final sample comprised five original studies – cross-sectional and case-control in design – examining mental disorders in relation to *T. gondii* infection, identified through the PRISMA 2020 selection process described above. These studies were conducted in Brazil, Iran, Norway, Rusia, and published between 2021 and 2025; their characteristics are summarized in Table 1. Across the five included studies, sample sizes ranged from 45 to 1.253 participants, reflecting substantial variation in study scale. Study populations also differed considerably: two studies enrolled pregnant women undergoing prenatal care (Lopes et al., 2025; Silva et al., 2024), one enrolled patients with a documented suicide attempt (Poursalar et al., 2025), and two enrolled broader psychiatric or community populations, including individuals with schizophrenia-spectrum or bipolar-spectrum disorders (Abramova et al., 2025; Andreou et al., 2024). Despite this heterogeneity, all five studies shared a common analytic logic, comparing proinflammatory cytokine concentrations between *T. gondii*-infected and uninfected groups, between groups defined by psychiatric diagnosis, or both.

Table 1. Characteristics of Included Studies

Authors, Year	Mental Disorder	Participants	Study Groups	Proinflammatory Cytokine Findings
(Abramova et al., 2025)	Schizophrenia	n = 160; Patients with schizophrenia and mentally healthy volunteers	• Group I Mentally healthy volunteers without *T. gondii* infection • Group II Mentally healthy volunteers with *T. gondii* infection • Group III Schizophrenia without *T. gondii* infection • Group IV Schizophrenia with *T. gondii* infection	• Increased MCP-1, RANTES, GRO-α, and IP-10 • Decreased MIP-1α, MIP-1β, and IL-8 • No difference in IL-17A
(Andreou et al., 2024)	Schizophrenia and bipolar disorder	n = 1.253; Patients with schizophrenia-spectrum disorders, bipolar-spectrum disorders, and healthy controls	• Group I SMI patients (schizophrenia or bipolar spectrum) • Group II Healthy controls Further grouped by *T. gondii* serostatus (seropositive vs. seronegative)	• Increased IL-18
(Lopes et al., 2025)	Major depressive disorder	n = 79; Pregnant women (18 – 40 years) in the third trimester of an uncomplicated pregnancy	• Group I Depressive disorder, *T. gondii* seropositive • Group II No depressive disorder, *T. gondii* seropositive • Group III Depressive disorder, *T. gondii* seronegative • Group IV No depressive disorder, *T. gondii* seronegative	• Increased TNF and IL-6 • Decreased MIF and IL-8 • No difference in IFN-γ
(Poursalar et al., 2025)	Suicide attempt	n = 500;	• Group I	• Increased IL-6 and IFN-γ

Authors, Year	Mental Disorder	Participants	Study Groups	Proinflammatory Cytokine Findings
	associated with depressive disorder	Patients diagnosed with attempted suicide (AS) and healthy individuals without attempted suicide (non-AS)	Patients diagnosed with attempted suicide (AS) • Group II Healthy individuals without attempted suicide (non-AS)	• TNF-α increased, but not significantly
(Silva et al., 2024)	Depressive disorder	n = 45; Pregnant women (18 – 39 years) receiving prenatal care	• Group I Pregnant women without *T. gondii* infection • Group II Pregnant women with *T. gondii* infection Further grouped by depression severity (minimal/mild vs. moderate/severe)	• Increased IL-33 and IL-17A • No difference in CCL2

Two of the five studies characterized cytokine profiles in schizophrenia associated with chronic *T. gondii* infection. Abramova et al. (2025) documented a mixed pattern of change: MCP-1, RANTES, GRO-α, and IP-10 were elevated, whereas MIP-1α, MIP-1β, and IL-8 were lower than in *T. gondii*-infected controls without schizophrenia. Andreou et al. (2024) Studying a cohort with severe mental illness (schizophrenia or bipolar spectrum), reported higher IL-18 concentrations in *T. gondii*-seropositive than in seronegative participants. The remaining three studies addressed depressive disorders. Among *T. gondii*-seropositive pregnant women with depressive disorder, Lopes et al. (2025) observed elevated IL-6 and TNF alongside reduced IL-8 and MIF. Poursalar et al. (2025) reported a broadly similar pattern–raised IL-6 and IFN-γ–among toxoplasmosis patients who had attempted suicide, along with a rise in TNF-α that did not reach statistical significance in multivariate analysis. Silva et al. (2024) Found elevated IL-33 and IL-17A in *T. gondii*-infected pregnant women diagnosed with mild depression. Table 2 consolidates these individual findings into a direct comparison across the two disorders.

Table 2. Comparison of Proinflammatory Cytokine Profiles

Cytokine	Schizophrenia + Toxoplasmosis	Depression + Toxoplasmosis	Summary
MCP-1	Increased (Abramova et al., 2025)	No difference (Silva et al., 2024)	Opposite pattern: unchanged in depression, increased in schizophrenia.
RANTES	Increased (Abramova et al., 2025)	Not measured	Specific to schizophrenia; correlates with anti-*T. gondii* IgA/IgM.
GRO-α	Increased (Abramova et al., 2025)	Not measured	Specific to schizophrenia; correlates with impaired social functioning.
IP-10	Increased (Abramova et al., 2025)	Not measured	Specific to schizophrenia; correlates with impaired social functioning.
MIP-1α	Decreased (Abramova et al., 2025)	Not measured	Decrease appears linked to the schizophrenia diagnosis rather than *T. gondii* infection alone.
MIP-1β	Decreased (Abramova et al., 2025)	Not measured	Decreased in schizophrenia with *T. gondii* infection.

Cytokine	Schizophrenia + Toxoplasmosis	Depression + Toxoplasmosis	Summary
IL-6	Not measured	Increased (Lopes et al., 2025; Poursalar et al., 2025)	Consistently elevated in depression with *T. gondii* infection; no schizophrenia data available.
TNF-α	Not measured	Increased (Lopes et al., 2025; Poursalar et al., 2025)	Elevated in depression; no schizophrenia data available.
IFN-γ	Not measured	Increased (Poursalar et al., 2025) No difference (Lopes et al., 2025)	Inconsistent findings in depression, possibly reflecting severity or pregnancy-related factors.
IL-8	Decreased (Abramova et al., 2025)	Decreased (Lopes et al., 2025)	Consistently decreased in both disorders with *T. gondii* infection.
IL-18	Increased (Andreou et al., 2024)	Not measured	Specific to schizophrenia/severe mental illness; correlates with neuronal damage.
MIF	Not measured	Decreased (Lopes et al., 2025)	Depression-specific; potential discriminatory marker.
IL-33	Not measured	Increased (Silva et al., 2024)	Specific to mild depression with *T. gondii* infection.
IL-17A	No difference (Abramova et al., 2025)	Increased (Silva et al., 2024)	Elevated in depression, not in schizophrenia.

Table 2 shows that the proinflammatory cytokine profiles of *T. gondii*-infected patients with schizophrenia and depression are largely non-overlapping. MCP-1 was elevated in schizophrenia (Abramova et al., 2025), but showed no difference in depression (Silva et al., 2024), indicating that its response depends on the underlying psychiatric diagnosis. RANTES, GRO-α, IP-10, MIP-1α, and MIP-1β were assessed only in schizophrenia (Abramova et al., 2025): RANTES, GRO-α, and IP-10 were elevated, with GRO-α and IP-10 increases correlating with altered social functioning, while MIP-1α and MIP-1β were reduced–a pattern that appears more closely tied to the schizophrenia diagnosis itself than to *T. gondii* infection per se. IL-18 measured only in schizophrenia (Andreou et al., 2024), was likewise elevated and has been linked to neuronal injury in severe mental illness.

Within the depression group, IL-6 and TNF-α were elevated (Lopes et al., 2025; Poursalar et al., 2025), with no comparable data available for schizophrenia. Findings for IFN-γ were inconsistent–elevated in Poursalar et al. (2025) but unchanged in Lopes et al. (2025)–possibly reflecting differences in depression severity or pregnancy status across the two samples. IL-8 was reduced in both schizophrenia and depression (Abramova et al., 2025; Lopes et al., 2025), suggesting this decrease may be a shared feature of *T. gondii* infection regardless of psychiatric comorbidity. MIF was reduced, and both IL-33 and IL-17A were elevated in depression (Lopes et al., 2025; Silva et al., 2024)whereas IL-17A was unchanged in schizophrenia (Abramova et al., 2025), pointing to IL-17A dysregulation as comparatively specific to depression. Collectively, these patterns suggest that *T. gondii* infection provokes disorder-specific inflammatory responses and that proinflammatory cytokine profiles may help differentiate schizophrenia from depression in infected individuals (Sadek & Mahmoud, 2026).

Not all cytokine findings in this review carry equal evidentiary weight. MCP-1 and IL-8 were each assessed in at least two independent samples spanning both schizophrenia and depression, and their direction of change was broadly consistent across studies, making them the most consistently replicated observations in this review. By contrast, cytokines measured in only a single included study–such as IL-18, IL-33, IL-17A, MIF, RANTES, GRO-α, and IP-10–represent preliminary findings that have not yet been externally replicated and should

therefore be interpreted with corresponding caution pending confirmation in independent samples. This distinction between replicated and single-study findings is particularly relevant given that none of the five included studies were designed explicitly to compare schizophrenia and depression within the same sample; the disorder-specific patterns described here therefore emerge from an indirect, cross-study comparison rather than a head-to-head trial.

Discussion

T. gondii cysts that from within the brain can alter neuroanatomy, neurochemistry, and behavior through direct neuroinflammatory action. Beyond schizophrenia and depression, *T. gondii* infection has been linked to a wider range of psychiatric conditions, including bipolar disorder, suicide attempts, personality disorders, self-harming behavior, autism, anxiety, and obsessive-compulsive disorder (Grada et al., 2022; Tunç & Erbaş, 2022). Mechanistically, the parasite activates innate immune cells, which recognize it through Pattern-Recognition Receptors (PRRs) such as Toll-Like Receptors (TLRs) and, in turn, initiate proinflammatory cytokine production (Moghaddami et al., 2024). These parasite-specific findings sit within a broader body of evidence linking systemic inflammation to psychiatric illness independent of infectious triggers (Chang et al., 2024; Lv et al., 2024; Perry et al., 2021). Viewed in this light, *T. gondii* infection may represent one of several possible upstream drivers that converge on a shared downstream neuroinflammatory pathway, alongside other recognized contributors such as chronic stress, metabolic dysfunction, and autoimmune processes (Hassamal, 2023; Spezani & Mandarim-de-Lacerda, 2026). This framing suggests that the cytokine signatures identified in this review should be interpreted as one contributing pathway among several, rather than as evidence that toxoplasmosis is either necessary or sufficient to cause schizophrenia or depression.

Interpreting these biomarker findings in a clinical context requires attention to how toxoplasmosis is currently diagnosed. In patients with schizophrenia or depression, diagnosis relies mainly on blood serology–specifically, detection of IgG and IgM antibodies by Enzyme-Linked Immunosorbent Assay (ELISA) (Fernandes et al., 2021; Pino & Zanón-Moreno, 2024). A positive IgG result indicates longstanding latent infection, whereas a positive IgM result typically requires confirmation with an avidity test to distinguish acute from chronic infection. Molecular assays such as PCR can detect *T. gondii* directly, but in patients with chronic mental disorders the parasite is rarely detectable in blood because it persists predominantly as bradyzoites within brain cysts; blood PCR therefore has low sensitivity in this population and is not recommended for routine screening (Pino & Zanón-Moreno, 2024). This diagnostic complexity matters for interpreting the present findings, since none of the five included studies used brain-tissue PCR or biopsy confirmation; all relied on peripheral serological or clinical criteria, meaning that some individuals classified as uninfected could theoretically harbor undetected latent infection confined to neural tissue, as has been demonstrated in other clinical population (Pawełczyk et al., 2022).

Diagnostic limitations aside, clarifying the biological mechanisms that connect toxoplasmosis to psychiatric illness depends heavily on proinflammatory cytokine measurement. In schizophrenia with toxoplasmosis, elevated TNF-α, IL-6, CCL2, CCL3, CCL4, and CCL5 point to state of chronic neuroinflammation (Barrios et al., 2021). Depression, by contrast, tends to show a different pattern of immune dysregulation–one centered on activation of the indoleamine 2,3-dioxygenase (IDO) pathway rather than large increases in antibody titers–which may explain why the association between *T. gondii* seropositivity and depression is often weaker and less consistent than in schizophrenia (Fernandes et al., 2021).

Cytokines and chemokines, as signaling proteins that govern immune-cell proliferation and activation, are frequently dysregulated in neuroinflammatory conditions, schizophrenia among them (Abramova et al., 2025). As shown in Table 1, Abramova et al. (2025) reported elevated MCP-1, RANTES, GRO-α, and IP-10 alongside reduce MIP-1α, MIP-1β, and IL-8 in schizophrenia patients with *T. gondii* infection. This pattern aligns with Ali & Al-Hadraawy (2024), who likewise observed elevated MCP-1 following *T. gondii* infection, and with Guo et al. (2024), who found MCP-1 elevated in chronic schizophrenia more broadly–together suggesting a persistent, low-grade inflammatory state in this population. Elevated RANTES and MCP-1 are thought to coordinate immune-cell recruitment and deployment, helping the host contain the parasite and limit tissue damage (Ullmann et al., 2025), while elevated IP-10 and GRO-α may reflect additional inflammatory pathways that stay active alongside clinical symptoms and the severity of social dysfunction (Abramova et al., 2025).

IL-8 illustrates this complexity well. As a proinflammatory chemokine, it recruits neutrophils, T lymphocytes, and monocytes to sites inflammation; these cells subsequently release free radicals capable of damaging surrounding tissue, including neurons (Shnayder et al., 2022). IL-8 is typically elevated in acute schizophrenia, where it can also disturb serotonergic, dopaminergic, and glutamatergic signaling and thereby contribute to depressive symptoms (Xu et al., 2025). Against this backdrop, the decrease in IL-8 that Abramova et al. (2025) observed in schizophrenia patients with toxoplasmosis–mirrored by a similar decrease in the depression group studies by Lopes et al. (2025)–is notable. Xu et al. (2025) suggest this may reflect individual variation in immune response or the influence of antipsychotic treatment, a possibility supported by (Zhao et al., 2024), who found that olanzapine treatment lowered IL-8 levels in schizophrenia patients. IL-18 adds a further layer to the schizophrenia picture. It participates in antimicrobial defense and in regulating neuronal differentiation and microglial activation, functions that can be neuroprotective when tightly controlled but neurotoxic when excessive (Gajewski et al., 2025; Xu et al., 2025). The elevated IL-18 that Andreou et al. (2024) reported in *T. gondii*-infected patients with severe mental illness (schizophrenia or bipolar spectrum) suggest heightened innate inflammatory activation, potentially disrupting inflammasome function – a process implicated in schizophrenia pathology, and ultimately, in neuronal damage (Andreou et al., 2024; Szabo et al., 2022).

The cytokine profile in depression associated with *T. gondii* infection differs markedly from that of schizophrenia, instead following a more classical pattern of inflammatory-pathway activation. Lopes et al. (2025) found that pregnant women with depressive disorder and chronic toxoplasmosis had higher TNF and IL-6 alongside lower MIF and IL-8, and Poursalar et al. (2025) reported a comparable elevation in serum IFN-γ and IL-6 among individuals who had attempted suicide, with TNF-α also rained, though not significantly. These elevated levels of TNF- α, IFN-γ, and IL-6 are mechanistically important to depression: they can activate IDO-mediated tryptophan metabolism, and the resulting neuroinflammation reduces tryptophan availability, lowers brain serotonin synthesis, and thereby contributes to depressive symptoms (Lopes et al., 2025; Moradi et al., 2023; Shi et al., 2025).

Silva et al. (2024) add a further dimension by reporting elevated IL-33 and IL-17A in *T. gondii*-infected pregnant women with mild depression. IL-33 acts as an alarmin, signaling cellular or tissue damage (Hasegawa et al., 2022; Silva et al., 2024), and its differential brain expression appears important for regulating emotional function and depression risk (Liu et al., 2023). IL-17A secreted by Th17 cells, stimulates microglia and macrophages to release further proinflammatory cytokines (Shnayder et al., 2022); at excessive concentrations it can damage tissue directly and has been shown to compromise blood-brain barrier integrity, a mechanism implicated in the onset of major depression (Tsuboi et al., 2024).

Considered together, these findings indicate that *T. gondii* infection provokes a diagnosis-specific pattern of inflammation. In schizophrenia, IL-18–a marker of innate inflammatory activation–correlates consistently with neuronal damage, while a more variable chemokine signature (decrease IL-8, MIP-1α, and MIP-1β; increased IP-10, MCP-1, RANTES, and GRO-α) points toward neurodegenerative processes and broader systemic immune dysregulation (Abramova et al., 2025; Andreou et al., 2024). In depression, the profile instead centers on Th1 (TNF-α, IFN-γ) and Th17 (IL-17A) activation, more consistent with neurotransmitter modulation than with structural brain injury (Lopes et al., 2025; Poursalar et al., 2025; Silva et al., 2024). These disorder-specific signatures carry clinical significance: they point to a need for immunomodulatory therapies tailored to each condition (Comai et al., 2025; Miller et al., 2025) and for inflammatory biomarkers capable of distinguishing inflammatory subtypes more broadly, including those potentially linked to infection (Bishop et al., 2022), ultimately supporting more individualized patient management.

From a clinical perspective, these disorder-specific cytokine signatures suggest that inflammatory biomarker panels could eventually support differential diagnosis or risk stratification in psychiatric patients with confirmed or suspected *T. gondii* exposure, particularly in settings where toxoplasmosis is endemic. For example, a chemokine-dominant profile (elevated MCP-1, RANTES, GRO- α, and IP-10) alongside elevated IL-18 may prompt closer monitoring for neurodegenerative features in patients with schizophrenia (Ma et al., 2024; Vida et al., 2025), whereas a Th1/Th17-dominant profile (elevated IL-6, TNF- α, and IL-17A) may be more informative in patients presenting with depressive symptoms. More broadly, incorporating *T. gondii* serological status into psychiatric research, and where clinically indicated into patient assessment, may help clarify whether a subset of schizophrenia and depression cases represent a distinct, infection-associated neuroinflammatory phenotype rather than a purely idiopathic presentation. Such biomarker-informed approaches remain hypothetical at this stage and would need prospective validation before informing clinical decision-making.

These conclusions should nonetheless be weighed against the heterogeneity of the included evidence. The five studies drew on markedly different populations – pregnant women, the general adult population, and hospitalized patients – and used varying cytokine measurement methods, which together limit how far the findings can be generalized despite our efforts to account for these differences (Benkarim et al., 2022). Several of the included studies also had comparatively small sample sizes for immunological biomarker research relative to the number of cytokines assessed, raising the possibility of false-positive or false-negative associations that would require confirmation by larger, adequately powered studies. Every included study was also cross-sectional, meaning none can establish whether *T. gondii* infection drives cytokine changes that lead to mental disorders, or whether the reverse sequence – or some bidirectional relationship – is at play. Longitudinal designs would be better suited to resolving this temporal question and to building stronger evidence for causality (Chirico, 2023). Future research would also benefit from prospective cohorts that track proinflammatory cytokine trajectories before and after documented *T. gondii* seroconversion, together with more standardized cytokine panels and diagnostic criteria across studies, to enable more direct cross-study comparison and, eventually, quantitative meta-analysis.

CONCLUSION

This review indicates that chronic *T. gondii* infection is associated with distinct proinflammatory cytokine profiles in schizophrenia and depression: schizophrenia is characterized by predominant elevations in MCP-1, RANTES, GRO-α, IP-10, and IL-18, a pattern linked to systemic inflammation, neuronal injury, and impaired social functioning, whereas

depression is marked chiefly by elevated IL-6 and TNF- α, with an immune response more closely tied to Th1/Th17 cytokine activity affecting neurochemical signaling. These disorder-specific cytokine signatures underscore the potential value of proinflammatory biomarkers in differentiating infection-related psychiatric phenotypes and in guiding more targeted, immunomodulatory approaches to patient care, addressing the review's aim of characterizing disorder-specific immune signatures linked to *T. gondii* infection; given the cross-sectional nature of the available evidence, however, future longitudinal studies are needed to clarify the causal direction between *T. gondii* infection and cytokine dysregulation in these disorders.

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