

Latent toxoplasmosis and neuropsychobehavioral

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ABSTRACT

Latent *Toxoplasma gondii* infection affects about one-third of the global population and, although long considered asymptomatic in immunocompetent people, is increasingly linked to psychological, behavioral, cognitive, and psychiatric outcomes. Evidence across reviews, meta-analyses, cohort studies, and cross-sectional studies indicates that seropositivity is associated most consistently with schizophrenia, and also with obsessive-compulsive disorder, anxiety, bipolar disorder, and some other psychiatric presentations, although effect sizes and consistency vary by disorder and study design. An overview of the relationship between latent toxoplasmosis and psychology is the goal of this article.

Keywords: toxoplasmosis, neuropsychobehavioral

To cite this article: Ismael SS, IF Yousif & LD Mohammad. Latent toxoplasmosis and neuropsychobehavioral. Biological Times. 2026. August 5(8): 33-34.

Introduction

Toxoplasma gondii infects roughly one-third of the global human population, forming lifelong dormant cysts in the brain that were traditionally considered clinically silent in immunocompetent individuals [1,2]. Over the past thirty years, extensive research has linked this latent infection to noticeable changes in personality, cognition, and mental health. However, the evidence consists of both significant connections and findings that have been questioned [3, 4].

In immunocompetent people, toxoplasmosis is typically mild or asymptomatic, but latent toxoplasmosis (LT) is a condition in which dormant tissue cysts, particularly in the brain, persist throughout the host's life. According to recent research, LT may have an impact on human neurological, immunological, psychiatric, and behavioral conditions such as schizophrenia, bipolar disorder, Alzheimer's disease, depression, suicidal thoughts, anxiety, and sleep disturbances. LT effects are debatable, and it's still unclear exactly how they work [1,5,6]

Psychiatric Disorder Associations

With more than 40 studies demonstrating increased *Toxoplasma* seroprevalence in patients with schizophrenia, the most often reported psychiatric association is with schizophrenia [7, 8]. *T. gondii* seropositivity was linked to schizophrenia in a sizable Danish case-control study involving 81,912 participants [9, 10]. There is still disagreement over the evidence supporting the depression connection. Toxoplasmosis is not a risk factor for major depressive disorder, according to a comprehensive review and meta-analysis of 30 publications that reported a pooled OR of 1.15 (95% CI 0.95–1.39) for anti-*Toxoplasma gondii* IgG in depressed patients [11, 12]. However, substantial depression relationships were seen in both the Taiwan cohort and Chinese inpatient investigations, indicating that the link may vary depending on the population, study design, or kind of depression [13, 14].

Proposed Mechanisms

Research on latent toxoplasmosis's neuropsychiatric effects is guided by two general theories. According to the manipulation theory, the parasite actively modifies host behavior in order to maximize the likelihood that it will spread from intermediate hosts to the final feline host [3, 7]. According to the second idea, negative consequences are caused by the host's physiological reaction, which includes changes in testosterone levels and neurotransmitter systems, specifically dopamine, serotonin, norepinephrine, and glutamate [3].

- As demonstrated in a chronic mouse infection model exhibiting anxiety, depressive-like behavior, and hyperactivity, persistent parasite cysts in the brain cause persistent neuroinflammation and blood-brain barrier breakdown, allowing circulating cytokines to seep into brain tissue [8].
- Because dopaminergic dysregulation is a hallmark of schizophrenia, *T. gondii* increases dopamine metabolism in infected brain tissue, suggesting a possible mechanistic connection [9].
- Immune activation: Cyst presence and periodic reactivation produce inflammatory and immune responses that, combined with genetic and environmental factors, may predispose individuals to psychiatric pathology [8,9].

The association between latent *Toxoplasma gondii* infection and schizophrenia has been documented across decades of epidemiological research, with multiple meta-analyses confirming significantly elevated seroprevalence among schizophrenia patients [15, 16, 17]. However, most evidence is observational, temporality has been addressed in only a minority of studies, and at least one well-designed case-control study found no positive association.

Limitations and Conflicting Evidence

The evidence base carries significant methodological constraints. Most studies are cross-sectional, precluding causal inference, and rely on seropositivity as an indirect marker of infection without capturing temporal dynamics [10]. A population-representative birth cohort of 837 individuals found little evidence that *T. gondii* was related to increased psychiatric disorder risk, poor impulse control, personality aberrations, or neurocognitive impairment, with only marginal associations for suicide attempt ($p = .06$) and one of 14 neuropsychological measures. Effect sizes for many associations are small, comparable to those of cigarette smoking or alcohol consumption in explaining variation in quantitative traits [10]. Inconsistencies across studies may reflect strain variability, diagnostic heterogeneity, and regional differences in infection prevalence. The strongest support comes from longitudinal and registry-based studies that account for infection timing and confounders, but definitive causality remains unproven.

Current evidence indicates that latent *T. gondii* infection is associated with a broad spectrum of psychological and psychiatric effects, with the schizophrenia link being the most robust and consistent finding across decades of research. Whether these associations reflect causal manipulation by the parasite, secondary neuroinflammatory consequences of chronic infection, or confounding by shared risk factors remains an open question that prospective, mechanistic, and interventional studies will need to resolve [18, 19].

Conclusion

Overall, the literature supports latent toxoplasmosis as a plausible contributor to psychological variation and neuropsychiatric risk, with the strongest evidence for schizophrenia-related associations and subtler cognitive effects, while definitive causal pathways and disorder-specific specificity remain unresolved.

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