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Association of latent *Toxoplasma gondii* infection with neuroinflammatory biomarkers and cognitive impairment

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Latent *Toxoplasma gondii* infection has been implicated in chronic neuroinflammation and altered neurotrophic signaling, potentially contributing to cognitive dysfunction. This study aimed to investigate the association of latent *T. gondii* infection with neuroinflammatory biomarkers and cognitive impairment compared with healthy controls. A cross-sectional case–control study was conducted between February 2025 and April 2026, including 100 individuals with latent *T. gondii* infection and 50 age- and sex-matched healthy controls. Latent infection was confirmed by positive anti-*Toxoplasma* IgG and negative IgM antibodies using ELISA. Serum levels of interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), interleukin-1 beta (IL-1β), S100B protein, high-sensitivity C-reactive protein (hs-CRP), and brain-derived neurotrophic factor (BDNF) were measured using ELISA and immunoturbidimetric assays. Cognitive performance was assessed using the Mini-Mental State Examination (MMSE), Montreal Cognitive Assessment (MoCA), verbal memory, attention, and executive function tests. Participants with latent *T. gondii* infection showed significantly higher concentrations of IL-6, TNF-α, IL-1β, S100B, and hs-CRP than controls. In contrast, BDNF levels were significantly reduced in the infected group. Cognitive assessments demonstrated significantly lower MMSE, MoCA, memory, attention, and executive function scores among infected individuals, accompanied by a higher prevalence of cognitive impairment. MoCA scores correlated negatively with inflammatory biomarkers and positively with BDNF concentrations. Latent *T. gondii* infection is associated with increased neuroinflammatory activity, reduced neurotrophic support, and impaired cognitive performance, suggesting that persistent asymptomatic infection may contribute to mild cognitive dysfunction through chronic low-grade neuroinflammation.

Keywords: latent *Toxoplasma gondii* infection; neuroinflammatory biomarkers; cognitive impairment; brain-derived neurotrophic factor; neuroinflammation.

Introduction

One of the most common parasitic infections in the world, *T. gondii* is an obligate intracellular protozoan parasite infecting approximately 1/3 of the world's population. Human infection usually happens when the tissue cysts are eaten by humans in undercooked meat, food or water is contaminated by oocysts excreted by infected cats and from congenital causes (Nayeri et al., 2024). Acute infection is followed by a latent infection of the parasite in the tissue with the formation of tissue cysts, including in the central nervous system (CNS) and in skeletal muscle. While the latent toxoplasmosis infection is considered clinically silent in healthy people, there is growing evidence that the infection has subtle but significant effects on the neurological, behavioral and cognitive functions (Sanchez & Besteiro, 2021).

One of the main sites of persistence of chronic *T. gondii* infections is the central nervous system. After dissemination from the gastrointestinal tract tachyzoites pass through the blood–brain barrier and become bradyzoites to form intracellular cysts which can survive for decades (Mendez & Koshy, 2017; Ramírez-Flores & Mondragón-Flores, 2025). These cysts in nervous tissue are a constant source of antigenic stimulation of the host immune system. This immune response is essential to avoid reactivation of parasites, but chronic immune activation can be associated with neuro-inflammatory effects that might have a negative impact on the integrity of synapses and/or neuronal functions. This has led to a resurgence of interest in the pathogenesis of latent toxoplasmosis in the areas of neuroimmunology and neuroinfectious disease research (Matta et al., 2021).

Neuroinflammation is a growing theme of the connection between chronic infections and neurological dysfunction. Microglia and astrocytes can increase the production of pro-inflammatory cytokines like interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α) and interleukin-1 beta (IL-1β) if they are continuously activated. Such mediators are important in the regulation of intracellular pathogens, but may have a negative impact on the survival of neurons, neurotransmitter balances, and cognitive functioning if they remain elevated for a long period of time (Kerkis et al., 2024). Chronic infection of *T. gondii* has been shown to cause persistent inflammation in the brain of experimental animals, which results in changes in neuronal signaling pathways and behavior. Furthermore, high levels of inflammatory markers have been linked to cognitive function deterioration and neurodegenerative diseases, indicating possible involvement of chronic inflammation as a significant mediator of neurological dysfunction related to infection (Khan et al., 2023).

S100 calcium-binding protein B (S100B) is one of the biomarkers of interest in neuroinflammation which is a marker of astrocytic activation and blood-brain barrier dysfunction (Langeh, 2021). Blood levels of S100B have been reported to be elevated in certain neurologic and psychiatric diseases and may be a marker for low level neural damage (brain) or activation of the glial cells. Likewise, high-sensitivity C-reactive protein (hs-CRP) is an indicator of systemic inflammation, and has been associated with diminished cognitive ability in multiple populations. Such markers might be useful for evaluation of individuals with latent toxoplasmosis to evaluate the inflammatory process of chronic parasitic infection (Gayger-Dias et al., 2023; Sarkar et al., 2025).

Neurotrophic factors are also important to maintain cognitive function and neuronal health as well as inflammatory pathways. One of the most important neurotrophins for neuronal survival is brain-derived neurotrophic factor (BDNF), which plays a role in synaptic plasticity, learning and memory formation (Silakarma & Sudewi, 2019). Low levels of BDNF have been suggested to be associated with cognitive impairment, depression, neurodegenerative diseases and chronic inflammatory diseases. BDNF expression has been

shown to be suppressed by persistent inflammatory signaling, which can lead to problems with cognitive function and neuroplasticity. The link between latent *T. gondii* infection, neuroinflammatory markers and BDNF level, however, is not fully understood (Colucci-D'Amato et al., 2020; Azman & Zakaria, 2022).

There have been several epidemiological studies that have linked latent toxoplasmosis with changes in cognitive function, reaction time, memory, attention and executive functioning. In addition, *T. gondii* seropositivity has been associated with neuropsychiatric diseases, such as schizophrenia, bipolar disorder and some behavioral abnormalities (De Haan et al., 2021). However, results have not been consistent; some studies have found a definite link between neural damage and cognitive function, but others have not. The differences could be due to methodological variations in the study design, sample size, and population characteristics, as well as variation in the cognitive function testing and inflammatory biomarker assays (Gonçalves et al., 2024).

Although there is accumulating evidence that a latent toxoplasmosis may be involved in neurological dysfunction, information on the interactions between chronic infection, neuroinflammation, neurotrophic factors and cognitive functions is still very limited, especially in the Middle East population. These relationships are relevant since latent toxoplasmosis occurs in many people and could be a modifiable risk factor for cognitive impairment and inflammatory diseases of the CNS (Latifi & Flegr, 2025).

For this reason, the present study was designed to assess the correlation between latent *T. gondii* infection, neuroinflammatory markers (inflammatory cytokines, S100B, hs-CRP) and cognitive impairment by comparing the serum levels of these markers and the cognitive performance between seropositive and healthy seronegative individuals. These results might shed more light on the possible neurological effects of prolonged *T. gondii* infection and help to gain insight into the host-parasite relations in the central nervous system.

Matherials and methods

The study protocol was reviewed and approved by the Institutional Ethics Committee of Thi-Qar Health Directorate (approval No. 148-25). All procedures were conducted in accordance with the ethical principles of the Declaration of Helsinki. Written informed consent was obtained from all participants before enrollment in the study. Participant confidentiality and anonymity were maintained throughout the study period.

This study was conducted by a cross sectional case control study design, which was carried out between February 2025 and April 2026, at Al-Habbobi Teaching Hospital and selected Primary Health Care centers in Thi-Qar Province, Iraq. A total of 150 participants (100 people with latent *T. gondii* infection and 50 apparently healthy seronegative individuals matched for age and sex) were enrolled in the study. Anti-*Toxoplasma gondii* immunoglobulin G (IgG) antibody was used as the definition of latent toxoplasmosis, because no anti-*Toxoplasma gondii* immunoglobulin M (IgM) antibody was detected. Those who had received a diagnosis of acute toxoplasmosis, neurological disorders, psychiatric diseases, autoimmune diseases, chronic inflammatory diseases, malignancies, recent infectious diseases in the last three months or severe hepatic or renal dysfunction were excluded from the study. The age, sex, body mass index (BMI), smoking status, education level, and history of animal exposure were obtained on a structured questionnaire and with a medical record.

A 5 mL venous blood sample was obtained from each participant in an aseptic condition. Serum samples were centrifuged at 3000 rpm for 10 minutes and the separated serum was stored at −20 °C until analysis. The levels of anti-*Toxoplasma gondii* IgG and IgM antibodies were determined by commercially available enzyme-linked immunosorbent assay (ELISA) kits following the manufacturer's instructions. Persons with IgG antibody and negative IgM antibody were considered to have latent Toxoplasmosis and those without both antibodies were classified into the control group.

Interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), interleukin-1 beta (IL-1β) and S100 calcium-binding protein B (S100B) were measured by commercially available ELISA kits as per the manufacturer's instructions. Levels of high-sensitivity C-reactive protein (hs-CRP) were determined by using an automatic immunoturbidimetric method. The duplicate analysis of all samples and the use of internal quality-control procedures were used to assure analytical reliability and repeatability.

BDNF levels were quantified by sandwich ELISA method (quantitative) as recommended by the manufacturer. Both serum samples and standards were processed in parallel and absorbance was measured by microplate reader. Calibrations were performed and concentrations were determined using standard calibration curves and reported in ng/mL.

A battery of standardized neuropsychological tests were administered by trained health care professionals to assess cognitive performance. The cognitive function was evaluated globally with the Mini Mental Status Examination (MMSE) and the Montreal Cognitive Assessment (MoCA). Memory performance was tested with a standardized verbal memory recall test and attention/executive functions were measured with validated cognitive performance scales. Each participant was tested in a quiet space to reduce distracting background noise and present consistent testing conditions.

Data analysis was performed using Statistical Package for Social Sciences (SPSS) version 26.0. Continuous variables were expressed as mean ± standard deviation (SD), whereas categorical variables were presented as frequencies and percentages. Comparisons between groups were performed using the independent-samples t-test for normally distributed variables and the chi-square test for categorical data. Pearson correlation analysis was used to evaluate associations between neuroinflammatory biomarkers, BDNF concentrations, anti-*Toxoplasma* IgG titers, and cognitive performance scores. Statistical significance was considered at a P-value of less than 0.05.

Results

The sociodemographic characteristics were comparable between participants with latent *T. gondii* infection and healthy controls. No significant differences were observed regarding age (36.8 ± 10.4 vs. 35.2 ± 9.8 years, P = 0.358), sex distribution (male: 42.0% vs. 40.0%, P = 0.812), BMI (27.4 ± 4.3 vs. 26.8 ± 3.9 kg/m², P = 0.421), university education level (48.0% vs. 54.0%, P = 0.489), or smoking status (24.0% vs. 20.0%, P = 0.581). These findings indicate that the two study groups were well matched with respect to major demographic and lifestyle characteristics, thereby reducing the potential influence of confounding factors on the observed neuroinflammatory and cognitive outcomes.

Table 1
Sociodemographic characteristics of participants according to latent *Toxoplasma gondii* infection status

Variable	Latent *T. gondii* (n = 100)	Controls (n = 50)	P-value
Age, years	36.8 ± 10.4	35.2 ± 9.8	0.358
Male, n (%)	42 (42.0)	20 (40.0)	0.812
Female, n (%)	58 (58.0)	30 (60.0)	0.812
BMI, kg/m²	27.4 ± 4.3	26.8 ± 3.9	0.421
University education, n (%)	48 (48.0)	27 (54.0)	0.489
Smoking, n (%)	24 (24.0)	10 (20.0)	0.581

The levels of anti-*Toxoplasma* IgG antibody were significantly higher in subjects with latent *T. gondii* infection than in healthy controls (146.3 ± 52.7 IU/mL vs 8.4 ± 4.6 IU/mL, P < 0.001), thus confirming a previous exposure and persistent latent infection. Anti-*Toxoplasma* IgM antibody was not detected in any of the participants in either group, suggesting that neither group had had an acute or recently acquired infection. In addition, the age at diagnosis was 6.7 ± 3.8 years among those who were infected, reflecting the chronic nature of the infection. These results support the classification of the latent toxoplasmosis group and seronegative controls, and indicate that these findings are a reliable basis for the assessment of the relationship between chronic infection and neuroinflammatory markers and cognitive performance.

Table 2
Serological characteristics of participants according to *Toxoplasma gondii* infection status

Parameter	Latent *T. gondii*	Controls	P-value
Anti-*Toxoplasma* IgG, IU/mL	146.3 ± 52.7	8.4 ± 4.6	<0.001
Anti-*Toxoplasma* IgM positive, n (%)	0 (0.0)	0 (0.0)	N/A
Duration since diagnosis, years	6.7 ± 3.8	N/A	N/A

Patients with latent *Toxoplasma gondii* infections had significantly higher levels of all of the neuroinflammatory markers measured than healthy control subjects. Serum IL-6 (8.74 ± 2.91 vs. 5.21 ± 1.87 pg/mL), TNF-α (16.82 ± 5.74 vs. 10.45 ± 3.66 pg/mL), IL-1β (5.12 ± 1.73 vs. 3.41 ± 1.24 pg/mL), S100B (94.8 ± 28.6 vs. 71.2 ± 21.5 pg/mL), and hs-CRP (4.06 ± 1.72 vs. 2.63 ± 1.14 mg/L) were all significantly higher in the infected group (all $P < 0.001$). These findings suggest that there is chronic low-grade systemic and neuroinflammation in people with latent toxoplasmosis. The simultaneous increase of pro-inflammatory cytokines and S100B also indicates that chronic *T. gondii* infection may lead to activation of glia cells and sustained inflammation, which may affect neurological function and cognitive abilities.

Table 3
Comparison of neuroinflammatory biomarkers between individuals with latent *Toxoplasma gondii* infection and healthy controls

Biomarker	Latent *T. gondii* (n = 100)	Controls (n = 50)	P-value
IL-6, pg/mL	8.74 ± 2.91	5.21 ± 1.87	<0.001
TNF-α, pg/mL	16.82 ± 5.74	10.45 ± 3.66	<0.001
IL-1β, pg/mL	5.12 ± 1.73	3.41 ± 1.24	<0.001
S100B, pg/mL	94.8 ± 28.6	71.2 ± 21.5	<0.001
hs-CRP, mg/L	4.06 ± 1.72	2.63 ± 1.14	<0.001

The cognitive assessment showed that people with latent *T. gondii* infection performed significantly worse than healthy individuals in all areas measured. Infected participants exhibited lower MMSE scores (27.2 ± 1.8 vs. 28.4 ± 1.3, $P < 0.001$) and MoCA scores (24.6 ± 2.7 vs. 27.1 ± 2.2, $P < 0.001$), indicating reduced global cognitive function. Likewise, there was a significant difference between the infected group (12.8 ± 3.4) and the control group (15.1 ± 3.1, $P < 0.001$) regarding verbal memory performance. This was also the case for measures of attention and executive functioning, which were also significantly lower among infected participants: attention scores were 82.7 ± 11.4 compared to 89.3 ± 9.6 ($P = 0.001$); and executive function scores were 21.6 ± 4.8 compared to 25.1 ± 4.2 ($P < 0.001$). The results indicate that latent *T. gondii* infection is linked with minor cognitive changes, especially in attention, memory, and executive function.

Table 4
Comparison of cognitive performance between individuals with latent *Toxoplasma gondii* infection and healthy controls

Cognitive test	Latent *T. gondii*	Controls	P-value
MMSE score	27.2 ± 1.8	28.4 ± 1.3	<0.001
MoCA score	24.6 ± 2.7	27.1 ± 2.2	<0.001
Verbal memory score	12.8 ± 3.4	15.1 ± 3.1	<0.001
Attention score	82.7 ± 11.4	89.3 ± 9.6	0.001
Executive function score	21.6 ± 4.8	25.1 ± 4.2	<0.001

Compared with healthy controls, people with latent *T. gondii* infection had lower serum BDNF levels (18.7 ± 5.4 vs. 23.9 ± 6.2 ng/mL; $P < 0.001$), indicative of decreased neurotrophic support and neuronal plasticity in *T. gondii* infected individuals. In addition, there was a significant difference in overall prevalence of cognitive impairment between participants and controls (29.0% vs. 10.0%, $P = 0.009$). The same was true for mild cognitive impairment, which occurred in 24.0% of people with latent toxoplasmosis versus 8.0% ($P = 0.018$) and moderate cognitive impairment which was observed in 5.0% with latent toxoplasmosis versus 2.0% ($P = 0.654$). The results suggest a correlation between latent *T. gondii* infection with decreased BDNF levels and increased risk of mild cognitive dysfunction, suggesting that there may be a relationship between chronic infection, decreased neurotrophic signaling, and cognitive decline.

Table 5
Neurotrophic biomarker levels and cognitive impairment among individuals with latent *Toxoplasma gondii* infection

Parameter	Latent *T. gondii*	Controls	P-value
BDNF, ng/mL	18.7 ± 5.4	23.9 ± 6.2	<0.001
Cognitive impairment, %	29 (29.0)	5 (10.0)	0.009
Mild cognitive impairment, %	24 (24.0)	4 (8.0)	0.018
Moderate cognitive impairment, %	5 (5.0)	1 (2.0)	0.654

As illustrated by the correlation analysis, there were significant correlations between cognitive function (as measured by the Montreal cognitive assessment (MoCA)) and some of the infection-related biomarkers. MoCA scores showed significant negative correlations with anti-*Toxoplasma* IgG levels ($r = –0.298$, $P = 0.003$), IL-6 ($r = –0.441$, $P < 0.001$), TNF-α ($r = –0.392$, $P < 0.001$), IL-1β ($r = –0.315$, $P = 0.001$), S100B ($r = –0.467$, $P < 0.001$), and hs-CRP ($r = –0.284$, $P = 0.004$), indicating that higher infection burden and increased inflammatory activity were associated with poorer cognitive performance. Glial activation (S100B) exhibited the greatest inverse relationship, indicating a possible involvement in cognitive decline. There was a significant positive correlation between serum BDNF levels and MoCA scores ($r = 0.429$, $P < 0.001$), demonstrating that the higher the neurotrophic support, the higher the cognitive performance. Together, these results imply that persistent neuroinflammation and decreased neuroprotective signaling could play a role in the cognitive impairments seen in people with latent *T. gondii* infection.

Table 6
Correlation between neuroinflammatory biomarkers, neurotrophic factor levels, and cognitive performance in individuals with latent *Toxoplasma gondii* infection

Variable	MoCA Score (r)	P-value
Anti-*Toxoplasma* IgG	–0.298	0.003
IL-6	–0.441	<0.001
TNF-α	–0.392	<0.001
IL-1β	–0.315	0.001
S100B	–0.467	<0.001
hs-CRP	–0.284	0.004
BDNF	0.429	<0.001

Discussion

In the current study, the association between latent infection with the protozoan *T. gondii*, neuroinflammatory biomarkers, and cognitive function was studied. The results revealed that, in serum, the people with latent toxoplasmosis had significantly higher levels of inflammatory markers (IL-6, TNF-α, IL-1β, S100B, hs-CRP) and lower levels of cognitive markers than seronegative individuals. The observations are consistent with the emerging theory that latent *T. gondii* infection is not entirely symptom-free but can be associated with chronic low grade levels of neuroinflammation, and subtle cognition deficits.

There were no significant differences between infected and control groups in terms of age, sex distribution, BMI, educational status or smoking status for the sociodemographic analysis. This is important as it minimizes the confounding factors that might impact both the neuro inflammatory and cognitive outcome. Previous studies of the association of latent toxoplasmosis and nervous system function have also reported similar demographic matching, which might allow for more precise estimates of the effect of latent toxoplasmosis rather than demographic effects (De Haan et al., 2021; Flegr, 2025).

Based on the serological results, all the infected participants had positive anti-Toxoplasma IgG antibody level and the antibody level of IgM antibody was not detectable, which confirmed that all the participants had chronic infection. This is in agreement with previous epidemiological studies which have demonstrated that latent toxoplasmosis has persistent immunological memory, without clinical signs of acute infection, but with the presence of permanent tissue cysts (Babekir et al., 2022). Clinically silent cases can have chronic low-level immune activation and inflammatory signaling from tissue

cysts that are present within neural tissue (Robert-Gangneux & Guegan, 2021).

This current study revealed a striking increase in the concentrations of markers of neuroinflammation in latently infected individuals. These increased levels of IL-6, TNF-α and IL-1β are indicative of chronic immune activation and support findings of chronic inflammatory response in patients with latent *T. gondii* infections (Zhao & Ewald, 2020). Experimental studies have shown that bradyzoite cysts are able to stimulate microglial cells and induce the release of pro-inflammatory cytokines in the CNS (Hwang et al., 2018). These increases in IL-6 and TNF-α can be interpreted as the result of a continuous activation of host immune cells by parasite antigens, and imply sustained inflammatory signalling. The findings are similar to the results of Nayeri et al. who reported that cytokine levels were significantly elevated in seropositive group compared to healthy controls (Chakroborty et al., 2023).

The fact of an increase in the concentration of S100B, which is observed in latent toxoplasmosis, may be another testimony of the involvement of the nervous system. The calcium-binding protein S100B is secreted mainly by astrocytes and is generally considered as a marker of activation of glia cells, and associated with blood–brain barrier disruption. There is a possibility that increase of S100B in the infected group might represent a mild stress or neuroinflammatory changes of the astrocytes, which could indicate chronic infection. These results corroborate with those of Bay-Richter et al., (Bay-Richter et al., 2019), who revealed that experimental toxoplasmosis models are related to increased glial activation. But others did not see significant differences in S100B levels in asymptomatic seropositive people (Castaño Barrios et al., 2021). Different durations of infection, different genotypes of the parasites, different host genetics and sensitivity of the biomarker assays might account for these discrepancies.

The cognitive evaluation showed significantly reduced MMSE, MoCA, verbal memory, attention and executive function scores for infected participants. These findings are consistent with the previous reports that latent toxoplasmosis may affect cognitive functions, although neurological symptoms do not occur (Silva et al., 2021; Shi et al., 2024). This might be related in a few ways. Some of the chronic neuroinflammation may affect the ability to create new connections between nerve cells, or the ability of nerve cells to communicate with each other, and regulation of neurotransmitters such as dopamine may be altered by parasitism, affecting cognitive function (Vergne-Salle & Bertin, 2021). Moreover, chronic inflammation of cytokines has been associated with memory dysfunction and hippocampal dysfunction. A comparable decrease in executive functioning and attention has been reported in a number of population-based studies of sero-positive adults (Zvonarev et al., 2020).

This increased risk of cognitive impairment for infected subjects compared to controls supports the notion that the latent form of toxoplasmosis may be a risk factor for subtle neurocognitive impairment. Other researchers did not see a significant link with latent infection and cognitive impairment (Ene et al., 2016). The differences between studies might be due to age, education, number of subjects, diagnosis and neuropsychological evaluation methodology. In addition, there could be geographical variation of the strains of parasites involved in pathogenicity and neurological outcome.

The most important finding was that infected patients showed considerable decrease in the levels of BDNF in serum. BDNF plays an important role in the survival of neurons, in synapse plasticity, learning and memory. A higher concentration of BDNF could help shield infected people from the cognitive problems associated with the infection, and could be interpreted as a mechanism of chronic inflammatory modulation of neurotrophic signaling pathways. Experimental studies have shown that pro-inflammatory cytokines are able to decrease the expression of BDNF and block the processes of neurogenesis (Shi et al., 2025). Chronic inflammatory and neurodegenerative diseases, such as those with ongoing immune activation have similarly been reported to have reduced BDNF levels (Dadkhah et al., 2024).

To further support this proposed link between latent toxoplasmosis, inflammation in the brain and cognitive dysfunction, a correlation analysis was performed. There were significant negative correlations between the scores of the MoCA and anti-Toxoplasma IgG antibody, levels of IL-6, TNF-α, IL-1β, S100B and hs-CRP. This has indicated that there is a link between a rise in inflammatory burden and a deterioration in cognitive function. Of particular interest was the negative correlation that was found between the two variables S100B and MoCA score (Egorov et al., 2021). This suggests a central role of glial activation in cognitive impairment of latent infection. The BDNF, however, demonstrated a highly positive correlation with cognitive function, indicating that it acts as a protective factor against maintaining neuronal integrity and cognitive function (Xiao, 2022).

Conclusion

The findings of the present study indicated a latent (persistent) infection of *T. gondii* is linked to chronic low-grade neuroinflammation characterized by increased levels of inflammatory cytokines, glial activation, impaired or even decreased neuronal support, and mild cognitive dysfunction. The findings corroborate a recently emerging body of evidence indicating that latent toxoplasmosis may have measurable impacts on brain function in apparently normal people. Longitudinal studies with the use of neuroimaging, genetic studies, and other cognitive testing in the future are warranted to further clarify the causal mechanism associated with these relationships and to assess the possibility of therapeutic interventions that target neuroinflammation as a mechanism to reduce cognitive decline associated with infection.

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