BRAIN. Broad Research in Artificial Intelligence and Neuroscience
e-ISSN: 2067-3957 | p-ISSN: 2068-0473
Covered in: Web of Science (ESCI); EBSCO; JERIH PLUS (hkdir.no); IndexCopernicus; Google Scholar; SHERPA/RoMEO; ArticleReach Direct; WorldCat; CrossRef; Peeref; Bridge of Knowledge (mostwiedzy.pl); abcdindex.com; Editage; Ingenta Connect Publication; OALib; scite.ai; Scholar9; Scientific and Technical Information Portal; FID Move; ADVANCED SCIENCES INDEX (European Science Evaluation Centre, neredataltics.org); ivySCI; exaly.com; Journal Selector Tool (letpub.com); Citefactor.org; fatcat!; ZDB catalogue; Catalogue SUDOC (abes.fr); OpenAlex; Wikidata; The ISSN Portal; Socolar; KVK-Volltitel (kit.edu)
2025, Volume 16, Issue 4, pages: 295-308
Submitted: April 24th, 2025 | *Accepted for publication:* August 23rd, 2025

Neurological Implications of T.gondii and the Role of Artificial Intelligence in Parasitic Psychiatry

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Abstract: *Toxoplasma gondii (T.gondii), a protozoan parasite with global prevalence, shows a unique neurotropism that allows it to persist chronically within the central nervous system (CNS). While often clinically silent, chronic toxoplasmosis has been increasingly associated with a range of psychiatric conditions, including schizophrenia, bipolar disorder, suicidal behaviour, and potentially Alzheimer's disease. The underlying mechanisms are multifactorial,involving direct cyst localization, neuroinflammation, altered neurotransmission, and immunomodulation. In parallel, artificial intelligence (AI) and machine learning (ML) tools are transforming the landscape of biomedical research. In the context of T. gondii, these tools are being employed in neuroimaging analysis, behavioural prediction models, and systems-level simulation of brain-parasite interactions. AI has the potential to enhance early detection of neuroanatomical changes, predict psychiatric outcomes based on infection markers, and uncover latent patterns in large-scale datasets. This descriptive review aims to synthesize the current knowledge on the neurological effects of T. gondii infection while highlighting the emerging integration of AI methodologies in parasitic neuroscience. We discuss pathophysiological mechanisms, psychiatric implications, and how computational tools may bridge gaps in understanding this enigmatic pathogen's impact on the human brain.*

Keywords: *toxoplasma gondii; artificial intelligence; psychiatry; schizophrenia; neuroinflammation; machine learning; neuroimaging.*

How to cite: Dumitrescu, A.-M., Buşilă, I. F., Misailoaie, A., Rădulescu, A.-M., Rîpă, C. V., Iancu, L. S., Costea, C. F., Paraschiv, C.-M., Dima-Cozma, L. C., Barbu, R. M., Chirica, V. A., & Cobzaru, R.-G. (2023). Neurological implications of T. gondii and the role of artificial intelligence in parasitic psychiatry. *BRAIN. Broad Research in Artificial Intelligence and Neuroscience*, *16*(4), 295-308.
https://doi.org/10.70594/brain/16.4/18

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1. Introduction

Toxoplasma gondii (*T. gondii*) is a widespread intracellular protozoan parasite with a remarkable ability to invade and persist within the central nervous system (CNS). It infects nearly one-third of the global human population and has traditionally been considered a significant threat mainly to immunocompromised individuals and developing fetuses (Montoya & Liesenfeld, 2004). However, emerging research over the past two decades has increasingly implicated *T. gondii* in a wide spectrum of neurological and psychiatric disorders, including schizophrenia, bipolar disorder, epilepsy, and even Alzheimer's disease. The parasite has a complex life cycle and an impressive capacity to evade immune surveillance, establishing chronic infections by forming latent tissue cysts in the brain and muscles. Within the brain, *T. gondii* preferentially encysts in neurons and astrocytes, where it can persist for the host's lifetime. Its presence in the CN is not merely passive; it has been shown to influence host behaviour, modulate neurotransmitter systems (especially dopamine and GABA), alter synaptic plasticity, and induce low-grade chronic inflammation (Parlog et al., 2015; David et al., 2016; Boillat et al., 2020). These mechanisms provide a plausible biological basis for the association between chronic toxoplasmosis and psychiatric outcomes.

Recent studies using advanced neuroimaging techniques such as diffusion tensor imaging (DTI) and functional magnetic resonance imaging (fMRI) have revealed structural and connectivity changes in the brains of seropositive individuals. These include alterations in the prefrontal cortex, amygdala, and hippocampus—regions heavily implicated in emotional regulation, memory, and cognition (Henriquez et al., 2009). Moreover, data from genome-wide association studies (GWAS) and transcriptomic profiling suggest that Toxoplasma gondii may influence host gene expression patterns associated with synaptic signaling and immune modulation (Wang et al., 2019).

The integration of artificial intelligence (AI) into parasitology and neuroscience has further expanded our capacity to detect, classify, and predict the impact of latent *T. gondii* infections. Machine learning algorithms are now being trained to identify infection-related biomarkers from neuroimaging datasets, gene expression profiles, and clinical metadata (Diab, 2023). AI systems in psychiatry may also serve as tools for supporting team wellbeing by monitoring burnout risk and workload distribution (Popa et al., 2024). Training AI systems to identify parasitic neuroinflammation may benefit from multimodal inputs—clinical signs, imaging, and serological data—structured similarly to interdisciplinary diagnostic protocols in other fields (Mitrea et al., 2022).

This review synthesises current findings on the neurological implications of *T. gondii*, with particular emphasis on its involvement in psychiatric pathology and the emerging role of artificial intelligence in advancing this field.

2. Material and method

We aimed to synthesise interdisciplinary findings on the neurological consequences of *T.gondii* infection and the emerging use of artificial intelligence in parasitic psychiatry.

A systematic search of the literature was conducted in PubMed, Scopus, and Web of Science for peer-reviewed articles published between 2000 and 2025. Search terms included combinations of: "*Toxoplasma gondii*" AND ("psychiatric" OR "schizophrenia" OR "bipolar disorder" OR "dementia" OR "suicide" OR "neuroinflammation" OR "neuroimaging" OR "dopamine" OR "glutamate") AND ("artificial intelligence" OR "machine learning" OR "deep learning"). We included original articles, reviews, and meta-analyses involving: human or animal studies of *T. gondii* with CNS involvement, neuroimaging findings in *T. gondii* infection, AI applications in neuroscience, psychiatry, or infectious disease related to *T. gondii*. We excluded articles not available in English, case reports without broader inference, and studies not involving the central nervous system or AI. The articles were analysed in two stages: title/abstract review and full-text review. Data extracted included sample size, study design, and relevant key findings for the descriptive review. Themes were synthesisl'ed narratively and grouped into the following domains:

neurobiology of *T. gondii*, psychiatric associations, neuroimaging evidence, the role of artificial intelligence in parasitic psychiatry.

3. Results and Discussion

3.1. T.gondii: Life Cycle and Neurotropism

T. gondii is a coccidian protozoan with a complex life cycle involving both sexual and asexual stages. The definitive hosts are members of the Felidae family, where sexual reproduction occurs within enteric epithelium, leading to the shedding of oocysts in faeces (Dubey, 2010; Tenter, Heckeroth, & Weiss, 2000). Intermediate hosts, including humans, acquire infection by ingesting sporulated oocysts from contaminated environments or tissue cysts from undercooked meat (Montoya & Liesenfeld, 2004). Following ingestion, the parasite differentiates into rapidly replicating tachyzoites, which disseminate via the bloodstream and lymphatics (Montoya & Liesenfeld, 2004).

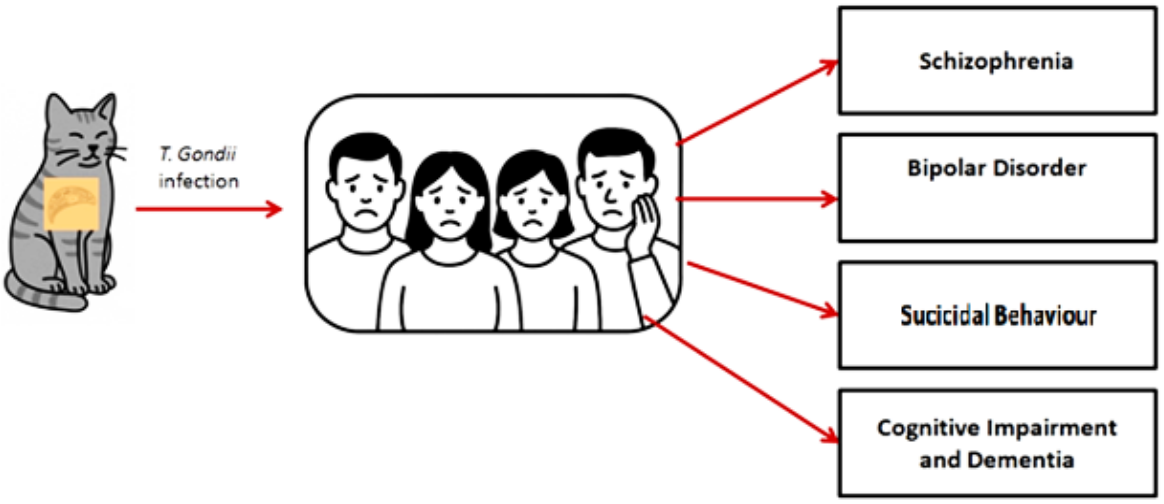

Figure 1. Diagram of the correlation between T. gondii infection transmission from cat to humans and the development of psychiatric disorders in the infected population. Conceptual representation by authors.

The parasite's ability to cross the blood-brain barrier (BBB) is a crucial aspect of its neuropathogenicity. Tachyzoites can invade brain endothelial cells via active penetration or by hijacking leukocytes.

Once in the CNS, *T. gondii* converts into the bradyzoite stage and forms intracellular tissue cysts, predominantly within neurons and astrocytes (Parlog et al., 2014). These cysts may persist lifelong in the brain, particularly in the amygdala, hippocampus, basal ganglia, and cortex—regions involved in emotion, memory, and motor control (Gaskell et al., 2009).

Neurotropism is not passive; studies suggest that the parasite exhibits a non-random distribution in the brain. Evidence from rodent models indicates preferential encystment in regions modulating fear, reward, and executive function (Vyas et al., 2007). This tropism underpins theories of behavioural manipulation and supports the hypothesis that *T. gondii* may subtly influence host neurobiology to favour its transmission cycle (Berdoy et al., 2000).

Recent transcriptomic and proteomic studies have revealed that bradyzoite cysts are metabolically active and capable of secreting effector proteins that interfere with host cell signaling. These include rhoptry proteins (ROPs) and dense granule proteins (GRAs), which modulate host immunity, neurotransmitter metabolism, and synaptic function (Hakimi, Olias, & Sibley, 2017; Fentress et al., 2010). Collectively, the parasite's neurotropism and molecular interactions lay the foundation for its potential to contribute to CNS dysfunction.

3.2. Pathophysiological Mechanisms in the CNS

The neurological impact of *T. gondii* stems from a confluence of direct parasitic invasion, immune-mediated inflammation, and neurochemical alterations. The key mechanisms through which *T. gondii* affects the CNS are discussed below:

a. Neuroinflammation

Chronic toxoplasmosis induces low-grade normalities in chronically infected individuals, particularly in fronto-temporal and limbic tracts (Müller et al., 2018). Such changes may mediate cognitive and affective dysfunction observed in *T. gondii*-seropositive individuals (Müller et al., 2018).

b. Epigenetic and Synaptic Modulation

Emerging evidence suggests that *T. gondii* may alter host gene expression through epigenetic mechanisms, including DNA methylation and histone modification (Kannan et al., 2019). These changes may be driven by host immune responses or direct parasite-secreted effectors. Such modifications can persist beyond the acute phase of infection and may predispose to long-term psychiatric sequelae (Prandovszky et al., 2011).

Synaptic proteins such as synapsin and PSD-95 are downregulated in animal models of chronic toxoplasmosis, implicating disrupted synaptogenesis and plasticity (Parlog et al., 2014). These deficits may underlie cognitive dysfunction and altered behaviour in both animals and humans (Parlog et al., 2014).

3.3. Parasitic Infections - Associated Psychiatric Disorders

One of the key mechanisms linking *T. gondii* infection to psychiatric neuroinflammation, characterised by microglial activation and the release of pro-inflammatory cytokines such as IL-1β, IL-6, TNF-α, and IFN-γ. While these immune responses are critical for controlling parasite proliferation, they may also contribute to neurotoxicity, synaptic pruning, and disruption of neural plasticity (Kannan et al., 2019). Similar oxidative and redox mechanisms have been observed in interdisciplinary studies evaluating UV-induced cellular responses and antimicrobial surface interactions, highlighting the role of oxidative stress modulation in biological systems (Negrila CC et al., 2021). Persistent microglial activation has been linked to altered dopaminergic signalling and white matter abnormalities—features commonly observed in schizophrenia (Miller et al., 2011; Najjar & Pearlman, 2015).

Given the role of neuroinflammation in *T. gondii*-induced brain impairment, investigating the inflammatory pathways of the renin–angiotensin system (RAS) may offer fundamental insights into host–pathogen interactions at the neuroimmune level. As demonstrated by Gurzu et al. (2015), RAS blockades reduce localised inflammation, providing a conceptual framework that could inform AI-driven models for predicting and characterising parasitic neuroinflammatory responses.

a. Alteration of Neurotransmitter Systems

One of the most studied effects of *T. gondii* infection is its influence on the dopaminergic system. The parasite encodes a gene homologous to tyrosine hydroxylase, a rate-limiting enzyme in dopamine synthesis (Gaskell et al., 2009; Prandovszky et al., 2011). This suggests a direct contribution to elevated dopamine levels in infected neural tissue. Hyperdopaminergia is a well-established feature in the pathophysiology of schizophrenia and may also underlie behavioural changes seen in infected rodents and humans (Flegr et al., 2014).

In addition to dopamine, *T. gondii* affects the GABAergic and glutamatergic systems. Animal studies indicate altered GABA release and disrupted glutamate uptake by astrocytes, contributing to excitotoxicity and seizure susceptibility (David et al., 2016; Parlog et al., 2014). These neurotransmitter imbalances can affect cognition, mood, and executive function.

b. Disruption of Neural Connectivity

Chronic toxoplasmosis has been associated with subtle but widespread changes in brain connectivity. Functional MRI (fMRI) studies in infected individuals have revealed altered resting-state networks, especially in the default mode and salience networks (Mendy et al., 2015). These changes may reflect both structural damage and compensatory mechanisms due to chronic low-grade inflammation and neurotransmitter imbalance.

Diffusion tensor imaging (DTI) studies suggest white matter microstructural ab

3.3.1. Schizophrenia and Suicidal Behaviour

Perhaps the most well-documented association is between *T. gondii* and schizophrenia. Numerous meta-analyses conducted over multiple decades have reported significantly higher seropositivity rates for *Toxoplasma gondii* IgG antibodies in individuals diagnosed with schizophrenia compared to healthy controls. A 2012 meta-analysis of over 40 studies from 17 countries concluded that infected individuals had a 2.7-fold increased risk of developing schizophrenia (Torrey et al., 2012).

The mechanisms underpinning this association likely involve a combination of parasite-induced hyperdopaminergia, chronic neuroinflammation, and altered glutamate signalling—pathways that converge with established neurobiological models of schizophrenia (Miller et al., 2011; Prandovszky et al., 2011). Additionally, certain *T. gondii* strains (e.g., type I or atypical strains) may be more neurovirulent, contributing to greater behavioural and cognitive impairment (Akins, Furtado, & Smith, 2024).

AI-driven analyses of neuroimaging data have further elucidated this relationship, revealing structural brain abnormalities in *T. gondii*-seropositive individuals that overlap with regions implicated in schizophrenia, such as the prefrontal cortex and hippocampus.

Suicidal behaviour has also been linked to *T. gondii* infection. A systematic review found that *T. gondii* seropositivity was associated with an increased risk of suicide, particularly among individuals with pre-existing psychiatric conditions.

Animal studies support this behavioural association, showing increased risk-taking and reduced fear responses in infected rodents. In humans, these changes may translate into impulsivity, altered decision-making, and affective dysregulation (Flegr et al., 2014).

A compelling body of research links *T. gondii* infection to an increased risk of suicidal ideation and completed suicide. In one large retrospective cohort study of U.S. military personnel, T. gondii seropositivity was associated with a 53% increased risk of suicide attempts, independent of other known psychiatric risk factors (Ling et al., 2011).

Animal models further support this behavioural association, demonstrating increased risk-taking behaviour and decreased aversion to predator odour in rodents infected with *T. gondii*, likely due to cyst-induced neural changes in the amygdala and limbic system (Berdoy et al., 2000; Vyas et al., 2007). In humans, such neurobiological alterations may translate into impulsivity, emotional dysregulation, and impaired decision-making—key contributors to suicidal behaviour (Sutterland et al., 2015).

Furthermore, studies have observed a higher prevalence of *T. gondii* antibodies in individuals with a history of self-harm and suicide attempts, particularly among those with co-occurring depression and schizophrenia (Pedersen et al., 2012). These findings raise important public health concerns and underscore the need for neuroimmunology assessment in populations at risk.

AI methodologies, including predictive modelling and natural language processing, have been employed to identify patterns in neuroimaging and clinical data that distinguish individuals at risk of suicidal ideation and attempts, potentially facilitating early intervention strategies.

3.3.2. Cognitive Impairment and Dementia

Chronic *T. gondii* infection has been associated with subtle cognitive deficits, particularly in attention, memory, and executive function. These impairments may reflect long-term neural remodelling, neuroinflammation, and disruption of glutamatergic signalling pathways due to persistent latent infection (Mendy et al., 2015). Some studies have reported a higher prevalence of *T. gondii* antibodies in patients with Alzheimer's disease and mild cognitive impairment (MCI), suggesting a possible role in accelerating neurodegenerative processes (Bennett et al., 2012).

However, longitudinal studies are still needed to clarify whether *T. gondii* plays a causative role in cognitive decline or acts as a comorbid risk factor in vulnerable populations (Kavakebian et al., 2025).

Several studies have documented elevated levels of anti-*T. gondii* IgG in individuals with Alzheimer's disease (AD) and mild cognitive impairment (MCI), suggesting a potential modulatory role in the pathophysiology of neurodegeneration (Mahami-Oskouei et al., 2016; Kusbeci et al., 2011). The parasite's ability to induce chronic low-grade inflammation and alter neurotransmitter metabolism, particularly dopamine and serotonin pathways, may further contribute to neurocognitive decline (Fekadu et al., 2010).

Despite these associations, causality remains unproven. Longitudinal studies and neuroimaging correlations are necessary to clarify whether *T. gondii* acts as a precipitating factor, accelerates neurodegeneration, or simply coexists as an epiphenomenon in vulnerable aging brains (Torrey, Bartko, & Yolken, 2007). Recent literature has highlighted that psychiatric symptoms, such as cognitive disturbances or behavioural changes, may precede the diagnosis of brain tumours and could be easily misattributed to primary psychiatric conditions (Moroșan et al., 2024). Interestingly, the neuropathological patterns seen in chronic toxoplasmosis—such as neuroinflammation, synaptic loss, and progressive cognitive decline— share similarities with those described in chronic traumatic encephalopathy (CTE). The updated criteria outlined by Mavroudis et al. (2023) for CTE may offer a comparative framework for studying parasitic neurodegeneration.

3.4. Translational Perspective: Integrating AI in T. gondii-Related Psychiatry

Artificial intelligence (AI) and machine learning (ML) are increasingly applied in biomedical research to analyse complex datasets, detect subtle patterns, and generate predictive models. In the context of *T. gondii* infection, AI offers novel opportunities to study its neuropsychiatric impact and optimise diagnostic and research strategies. AI applications can be broadly categorised into data integration, neuroimaging analysis, predictive modelling, natural language processing, and experimental research (Huang et al., 2025, Hinze-Selch, 2015).

AI algorithms can integrate diverse types of data, including serological markers, neuroimaging results (MRI, PET), electrophysiological data (EEG), and multi-omics datasets (genomic, transcriptomic, proteomic, metabolomic). This integration facilitates the construction of comprehensive models of host-parasite interactions and enables the identification of subtle associations between chronic *T. gondii* infection and psychiatric outcomes (Halonen, 2023).

ML has been employed to detect structural and functional changes in the brains of seropositive individuals. AI-assisted analysis of MRI and DTI data can identify volumetric reductions in the hippocampus, amygdala, and prefrontal cortex, as well as microstructural white matter abnormalities in fronto-limbic tracts, which are associated with cognitive and affective dysfunction (Faiyaz et al., 2023). Functional MRI data can also be analysed using AI to detect alterations in resting-state connectivity, particularly in the default mode and salience networks, providing insight into compensatory neural mechanisms or pathology-induced hyperactivation (Faiyaz et al., 2023).

AI algorithms can be trained to predict the risk of neuropsychiatric manifestations in *T. gondii*-positive populations. By integrating clinical, serological, and neuroimaging data, predictive

models can identify individuals at higher risk of psychiatric conditions such as schizophrenia, suicidal behaviour, or cognitive impairment (Latifi & Flegr, 2025).

Moreover, Natural Language Processing (NLP) techniques enable the extraction of meaningful patterns from unstructured data, including electronic health records, clinical notes, and the scientific literature. By automating literature mining and clinical data extraction, AI can reveal latent associations between *T. gondii* infection and neuropsychiatric outcomes, generating novel hypotheses for mechanistic studies (Omar et al., 2024).

AI-assisted image recognition and data analysis in animal and in vitro models allow detailed study of parasite-host interactions at cellular and molecular levels. For instance, AI can identify morphological changes in neurons or astrocytes due to parasitic cysts and integrate these findings with omics data to explore neuroimmune pathways and neurotransmitter dysregulation (Parija & Poddar, 2024).

Despite these promising applications, AI approaches face limitations. The quality and heterogeneity of input data, lack of standardised analysis pipelines, and potential algorithmic biases can compromise the validity and reproducibility of results. Moreover, many studies remain preliminary and require external validation across larger, more diverse cohorts.

In conclusion, AI represents a powerful tool in parasitic psychiatry. By integrating complex datasets and supporting predictive modelling, neuroimaging analysis, and literature mining, AI has the potential to refine risk stratification, improve diagnostic accuracy, and accelerate mechanistic discovery in *T. gondii*-associated psychiatric disorders.

When applied rigorously, AI can complement traditional research methodologies and advance precision psychiatry in the context of parasitic infection.

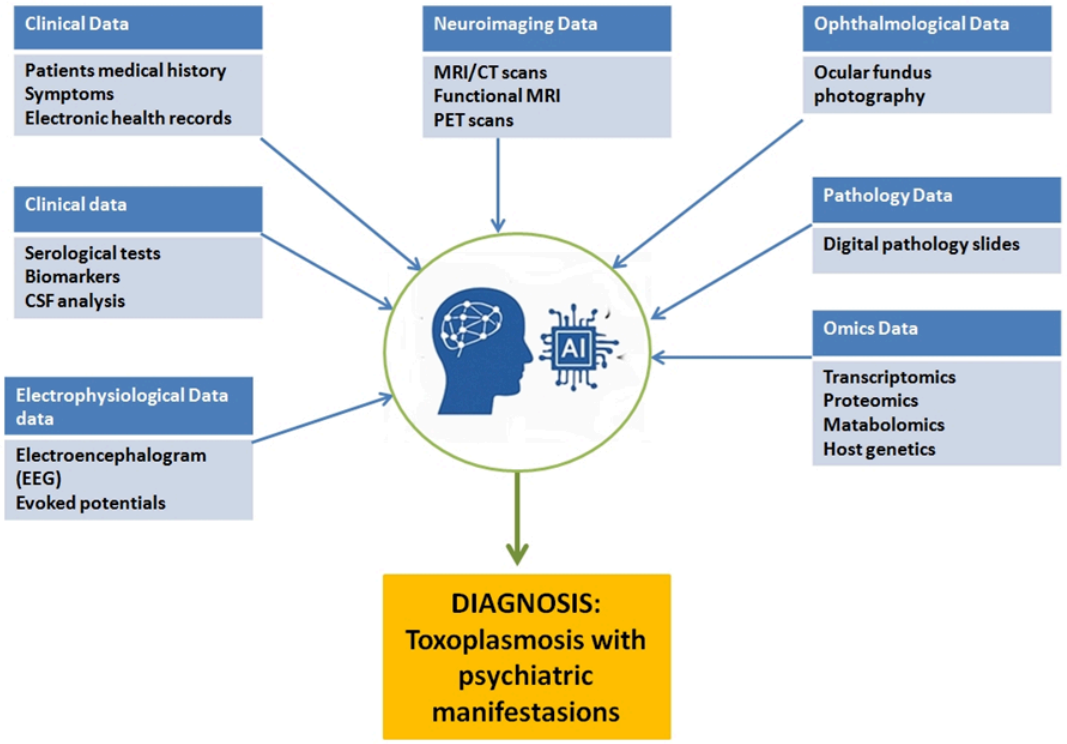

Figure 2. Diagram illustrating the role of AI role in T. gondii – related psychiatric disorders

3.5. Neuroimaging Evidence of the CNS Involvement in infections with T.gondii

While histopathological studies clearly demonstrate *T. gondii* cysts within human brain tissue, in vivo imaging evidence remains limited but is growing. Neuroimaging modalities—particularly MRI and PET—have been instrumental in identifying structural and functional abnormalities potentially attributable to chronic toxoplasmosis (Ningsih et al., 2025).

Notably, the regions most frequently affected—such as the hippocampus, amygdala, and prefrontal cortex—correspond to key domains of memory, emotional regulation, and executive function, respectively, supporting the neuropsychiatric correlations described throughout this review. Moreover, neuroimaging advances have also elucidated the neural underpinnings of rare neurological syndromes, emphasising the importance of differentiating organic from psychiatric etiologies (Manea MC et al., 2024). MRI studies in patients with schizophrenia have consistently reported volumetric reductions in the hippocampus, amygdala, and prefrontal cortex, along with white matter abnormalities in the corpus callosum and cingulate regions, supporting the hypothesis of disrupted neural connectivity (Iliuta FP et al., 2021). These findings overlap with the neuroanatomical alterations observed in *T. gondii*-infected individuals, further reinforcing the potential link between chronic infection and schizophrenia-related neurocircuitry changes.

3.5.1. Structural MRI Findings

Studies using high-resolution MRI have identified volume reductions in several brain regions among *T. gondii*–seropositive individuals. These include the hippocampus, amygdala, and prefrontal cortex—areas implicated in memory, emotion regulation, and psychiatric disorders (Erickson et al., 2025).

In a structural MRI study comparing seropositive and seronegative individuals, *the T. gondii*–positive group showed reduced grey matter volume in bilateral medial temporal lobes and the dorsolateral prefrontal cortex, correlating with poorer performance on memory tasks (Flegr et al., 2014).

3.5.2. Diffusion Tensor Imaging (DTI)

DTI has been used to assess white matter integrity. Some studies report reduced fractional anisotropy in fronto-limbic tracts and the corpus callosum of seropositive individuals, suggesting subtle microstructural disruption. These abnormalities may contribute to impaired inter-hemispheric communication and cognitive dysfunction (Jindal et al., 2025). The differential diagnosis in these cases should consider frontal lobe tumours that can severely affect both cognitive and behavioural functions of the patient (Moroșan et al., 2024).

3.5.3. Functional MRI (fMRI)

Resting-state fMRI studies have revealed altered connectivity in default mode and salience networks in *T. gondii*–infected individuals. These findings align with the neuroanatomical distribution of cysts and suggest a systems-level impact on brain network architecture (Tabaie et al., 2025).

One notable study found decreased functional connectivity in the anterior cingulate cortex and increased connectivity in the amygdala and orbitofrontal cortex among infected individuals—patterns that may reflect compensatory reorganisation or pathology-induced hyperactivation (Alvarado-Esquivel et al., 2016). Given the nonspecific nature of some neuroimaging alterations, it is important to consider the diagnostic overlap with functional neurological disorders, where similar connectivity patterns may occur without identifiable structural lesions. Mavroudis et al. (2024) highlight these challenges and emphasise the need for refined imaging-based criteria and AI-supported analysis to differentiate functional from infectious or structural aetiologies.

3.5.4. PET and Metabolic Imaging

Although limited, positron emission tomography (PET) studies have reported altered glucose metabolism and increased binding of neuroinflammation tracers (e.g., translocator protein ligands) in seropositive individuals. These findings support the hypothesis that chronic infection promotes low-grade, widespread neuroinflammation (Largeau et al., 2017).

3.6. Advancements in AI and Neuroimaging Methodologies

Recent developments in AI and neuroimaging have significantly enhanced the study of the impact of *T. gondii* infection on the brain. Multimodal fusion techniques, which integrate structural, functional, and molecular imaging data, allow for a more comprehensive understanding of how *T. gondii* infection may alter brain architecture and function. For example, the Cross-Attentive Multimodal Fusion framework (CAMF) employs self-attention and cross-attention mechanisms to capture complex interactions between different imaging modalities, improving classification accuracy in schizophrenia diagnosis (Carruthers & Suzuki, 2007).

Explainable AI approaches are also gaining traction in neuroimaging research. These methods aim to make AI models more interpretable, facilitating the identification of specific brain regions and networks affected by *T. gondii* infection. Techniques such as Gradient-weighted Class Activation Mapping (Grad-CAM) have been used to visualise areas of the brain most influential in AI-driven predictions, providing insights into the neurobiological underpinnings of psychiatric disorders (Farahani et al., 2022;

AI and ML are powerful tools in advancing our understanding of the neuropsychiatric implications of *T. gondii* infection. While the associations with schizophrenia and suicidal behaviour are well-documented, further research is needed to explore other potential links cautiously. The integration of multimodal neuroimaging data and the application of explainable AI methodologies hold great promise for uncovering the complex interactions between *T. gondii* infection and brain function, paving the way for more precise diagnostic and therapeutic strategies in the field of infectious psychiatry (Zhu et al., 2024; Chen et al., 2022).

The hypothesis that *T. gondii* infection may contribute to psychiatric pathology is supported by a growing body of epidemiological, serological, and mechanistic evidence. Though causality remains under debate, multiple studies suggest a strong correlation between chronic *T. gondii* infection and several psychiatric disorders. These include schizophrenia, bipolar disorder, suicidal behaviour, and cognitive decline (Torrey et al., 2012; Sugden et al., 2016; Sutterland et al., 2015).

4. Limitations

This review has several methodological limitations. Many primary studies relied on serological testing, most commonly enzyme-linked immunosorbent assays (ELISA), to determine *T. gondii* infection status, which may be influenced by variability in test sensitivity and specificity, potentially affecting the reported associations with neuropsychiatric outcomes. Most studies did not stratify participants by *T. gondii* strain, despite known differences in virulence and neurotropism, limiting the generalisability of findings. The predominance of cross-sectional designs in the literature further constrains interpretation, as causal relationships between infection and psychiatric manifestations cannot be established. AI-based studies are also limited by the heterogeneity and quality of input data, potential algorithmic biases, and the lack of standardised methods for integrating multimodal datasets. Finally, variations in neuroimaging protocols, clinical assessment tools, and population demographics contribute additional uncertainty. Collectively, these limitations demonstrate the need for longitudinal, standardised, and multidisciplinary approaches to clarify the neuropsychiatric implications of *T. gondii* infection and optimise the application of AI in this field.

5. Conclusions

T. gondii remains a significant global health concern due to its widespread prevalence and underestimated neurological impact. While traditionally associated with immunocompromised individuals and congenital infections, growing evidence supports its role in modulating neural function in immunocompetent hosts. The parasite's ability to establish chronic infections in the brain, alter neurotransmitter systems such as dopamine and GABA, and influence

neuroinflammation suggests a potential contribution to the pathophysiology of several psychiatric disorders, including schizophrenia, bipolar disorder, depression, and epilepsy.

Despite compelling associations, causality remains difficult to establish, and many mechanisms are yet to be fully elucidated. Advances in neuroimaging, molecular biology, and animal models have improved understanding of how latent toxoplasmosis may subtly alter brain function and behaviour, yet discrepancies between studies and the multifactorial nature of psychiatric conditions necessitate cautious interpretation.

AI offers a promising avenue to accelerate discovery by integrating clinical, laboratory, imaging, electrophysiological, pathological, and omics data. When properly validated, these tools can enhance diagnostic precision, refine risk stratification, and ultimately support precision psychiatry in parasite-linked mental illness. Future research should focus on longitudinal studies, standardised methodologies, and multidisciplinary approaches combining neurology, psychiatry, immunology, parasitology, and computational modelling to clarify the molecular mechanisms of *T. gondii*-induced neuroplastic changes and to explore potential interventions aimed at mitigating neurological and psychiatric sequelae.

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