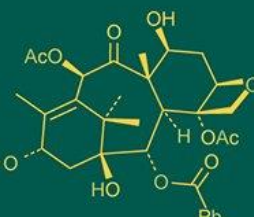
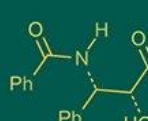


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From fur to faeces: Cat-associated biohazards, toxoplasmosis, and human health integrating artificial intelligence and translational medicine: A comprehensive health review

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Abstract

The longstanding cohabitation of humans with domestic cats (*Felis catus*) yields considerable psychological and social benefits, yet sustains an underappreciated spectrum of zoonotic hazards at the human-animal interface. Among the pathogens transmissible from cats to humans, *Toxoplasma gondii* occupies a position of singular epidemiological importance, owing to its extraordinary global prevalence, obligate dependence on felids for sexual reproduction, and capacity to cause life-threatening disease in immunocompromised hosts and the developing fetus. This review comprehensively characterizes cat-associated biohazards with primary emphasis on toxoplasmosis, examining parasite biology, transmission dynamics, immunopathology, and the full clinical spectrum from asymptomatic latency to severe congenital disease and cerebral reactivation. Epidemiological evidence documenting latent infection in roughly one-third of the global population is presented alongside emerging neuropsychiatric associations. Conventional diagnostic modalities serological, molecular, histopathological, and neuroimaging are appraised alongside advanced computational tools that augment diagnostic precision and risk stratification. The translational pipeline targeting bradyzoite-stage parasites is critically evaluated, and vaccine development platforms, including subunit, DNA, and messenger RNA approaches, are assessed. Prevention strategies within a One Health framework complete the synthesis. This review provides clinicians, researchers, and public health professionals with an integrated, evidence-based framework for understanding and managing cat-associated infectious disease.

Keywords: *Toxoplasma gondii*, toxoplasmosis, cat-associated biohazards, zoonotic disease, translational medicine, one health, drug discovery, vaccine development, neuropsychiatric associations

Introduction

The Human-Cat Interface

Domestic cats number more than 600 million globally, with surveys indicating that 25-30% of households in developed nations include at least one feline companion (Chomel and Sun) ^[1]. This extensive proximity creates recurring and varied opportunities for pathogen transfer at the human-animal interface, situating domestic cats as meaningful reservoirs and amplifying hosts within the broader landscape of emerging and re-emerging infectious disease. The conceptual framework most suited to understanding these risks is One Health, which recognizes the functional inseparability of human, animal, and environmental health and advocates integrated surveillance and management across all three domains (Mackenzie and Jeggo) ^[2].

Cats serve as definitive or intermediate hosts for a taxonomically diverse array of pathogens. Bacterial hazards include *Bartonella henselae* and *Pasteurella multocida*; fungal exposure is most frequently caused by *Microsporum canis*; parasitic threats encompass *Toxoplasma gondii* and *Toxocara cati*; and viral risks, though geographically restricted, include rabies (Chomel and Sun) ^[1]. Of these, *T. gondii* commands the greatest sustained public health attention on account of its globally distributed prevalence, its obligate intracellular biology, and the severity of disease it causes in vulnerable populations.

Globally, approximately one-third of the human population exceeding two billion individuals harbours serological evidence of past *T. gondii* infection (Pappas, Roussos, and Falagas) ^[4].

In immunocompetent adults, this infection is overwhelmingly subclinical; however, primary infection during pregnancy, reactivation in immunocompromised hosts, and direct congenital transmission collectively generate a burden of morbidity and mortality warranting serious clinical attention. Emerging epidemiological data further associate chronic latent infection with a range of neuropsychiatric outcomes, substantially broadening the public health relevance of this parasite (Sutherland *et al.*)^[5]. This review aims to: (i) characterize the principal cat-associated biohazards; (ii) elucidate *T. gondii* biology, transmission, and pathophysiology; (iii) appraise conventional and advanced diagnostic modalities; (iv) evaluate the translational drug-discovery and vaccine development pipeline; and (v) propose integrative prevention strategies consistent with One Health principles.

Taxonomy of cat-associated biohazards

Physical interaction with cats carries several well-characterized risks. Cat scratch disease, caused by *B. henselae*, affects an estimated 12,000 individuals annually in the United States; immunocompromised patients may develop severe systemic manifestations including bacillary angiomatosis and peliosis hepatis (Nelson, Saha, and Mead)^[6]. Bite wounds inoculate *P. multocida* into subcutaneous and deeper tissues, potentially causing rapidly progressive cellulitis, osteomyelitis, or septic arthritis. Dermatophytosis attributable to *M. canis* is sustained by asymptomatically infected feline carriers who serve as persistent environmental reservoirs. Separately, the secretoglobin Fel d 1-elaborated principally by feline sebaceous glands sensitizes approximately 10% of the general population, contributing substantially to allergic rhinitis and asthma exacerbations (Bonnet *et al.*)^[7].

The faecal-oral route underpins transmission of the maximum globally consequential cat-associated pathogens. A single acutely infected cat may shed hundreds of millions of *T. gondii* oocysts during primary infection, with environmental oocyst viability extending beyond twelve months under suitable soil conditions (Dubey)^[3]. Additional pathogens transmissible via this route include *Cryptosporidium* species, *Giardia duodenalis*, and *Toxocara cati*-the latter responsible for visceral and ocular larva migrans syndromes in humans, disproportionately affecting young children through close contact with contaminated soil and sandpits.

The cat flea (*Ctenocephalides felis*) aids as a competent biological vector for *B. henselae*, *Rickettsia felis*, and the cestode *Dipylidium caninum*. Domestic flea infestations generate sustained human exposure opportunities, with

young children at elevated risk owing to close contact with infested pets and floor surfaces.

Toxoplasma gondii: Biology and Life Cycle

Toxoplasma gondii is an obligate intracellular apicomplexan protozoan exhibiting remarkable biological versatility. Three principal clonal lineages-designated Types I, II, and III-display distinct geographic distributions and virulence profiles. Type II strains predominate in Europe and North America, whereas atypical and recombinant genotypes show comparatively greater prevalence in South America, where they are associated with more severe ocular and systemic manifestations (Shwab *et al.*)^[8].

The parasite exists in three morphologically and functionally distinct infectious forms, each adapted to specific phases of the life cycle:

- Tachyzoites-rapidly dividing forms responsible for acute infection and haematogenous dissemination to tissues throughout the host.
- Bradyzoites-slowly replicating forms encysted within tissue cysts, establishing chronic latent infection with particular tropism for neural and muscular tissues.
- Sporozoites-contained within environmentally resistant oocysts shed exclusively by felid definitive hosts following sexual reproduction.

Cats occupy an ecologically irreplaceable position in *T. gondii* biology as the sole hosts permitting sexual reproduction of the parasite. Following ingestion of infected prey or viable tissue cysts, parasites undergo gametogenesis within the intestinal epithelium of the definitive host, culminating in unsporulated oocyst formation and faecal excretion (Dubey)^[3]. Primary infection in cats typically provokes oocyst shedding over 7-21 days, after which most individuals develop protective immunity limiting subsequent shedding episodes. Young, immunologically naive cats, particularly those that hunt rodents therefore represent the highest-risk source of environmental contamination.

Virtually all warm-blooded vertebrates may serve as intermediate hosts, harboring tissue cysts indefinitely. Human infection is acquired principally through three pathways: (i) ingestion of oocysts from contaminated soil, water, or unwashed produce; (ii) consumption of undercooked meat particularly pork, lamb, and game containing viable tissue cysts; and (iii) transplacental transmission to the fetus during acute maternal infection (Pappas, Roussos, and Falagas)^[4]. Blood transfusion and solid-organ transplantation represent uncommon but clinically significant transmission routes.

Table 1: Cat-Associated Biohazards and Human Health Implications

Biohazard Category	Causative Agent	Mode of Transmission	Major Clinical Manifestations	Public Health Significance
Parasitic	<i>Toxoplasma gondii</i>	Fecal-oral, contaminated food, congenital	Toxoplasmosis, encephalitis, congenital disease	Global zoonotic burden
Bacterial	<i>Bartonella henselae</i>	Cat scratch, flea vectors	Cat-scratch disease, lymphadenopathy	Common zoonosis
Bacterial	<i>Pasteurella multocida</i>	Cat bites and scratches	Cellulitis, septic arthritis	Frequent wound infection
Helminthic	<i>Toxocara cati</i>	Ingestion of contaminated soil	Visceral and ocular larva migrans	High pediatric risk
Protozoal	<i>Giardia duodenalis</i>	Fecal contamination	Gastroenteritis	Waterborne outbreaks
Protozoal	<i>Cryptosporidium</i> spp.	Contaminated water and faeces	Diarrheal disease	Immunocompromised host risk
Fungal	<i>Microsporum canis</i>	Direct contact with infected cats	Dermatophytosis (ringworm)	Community transmission
Allergic	Fel d 1 allergen	Airborne exposure	Allergic rhinitis, asthma	Affects ~10% of population
Viral	Rabies virus	Animal bites	Fatal encephalitis	Preventable but lethal

Table 1. Overview of major cat-associated biohazards, transmission routes, and clinical implications in humans.

Epidemiology of Toxoplasmosis

Seroprevalence estimates for *T. gondii* infection range from approximately 10% to above 80% across different regions, reflecting the combined influence of dietary practices, climate, cat population density, and sanitary infrastructure (Pappas, Roussos, and Falagas) [4]. Seroprevalence rises progressively with age, consistent with cumulative lifetime environmental exposure. France historically records seroprevalences exceeding 50%, linked in part to culinary traditions favouring lightly cooked or raw meat, while rates in the United Kingdom and the United States approximate 10-20% (Jones *et al.*) [9].

Three populations bear disproportionately severe manifestations of *T. gondii* infection. First, primary maternal infection during gestation may produce congenital toxoplasmosis affecting 1-10 per 10,000 live births globally, with disease severity inversely correlated with gestational age at the time of infection (Wallon *et al.*) [10]. Second, immunocompromised individuals including those with advanced HIV infection, solid-organ transplant recipients, and patients receiving biological immunosuppressants are at substantial risk of cerebral reactivation disease. Third, congenitally infected children may suffer long-term sequelae encompassing chorioretinitis, sensorineural hearing loss, and neurodevelopmental impairment even when neonatal screening reveals subclinical disease.

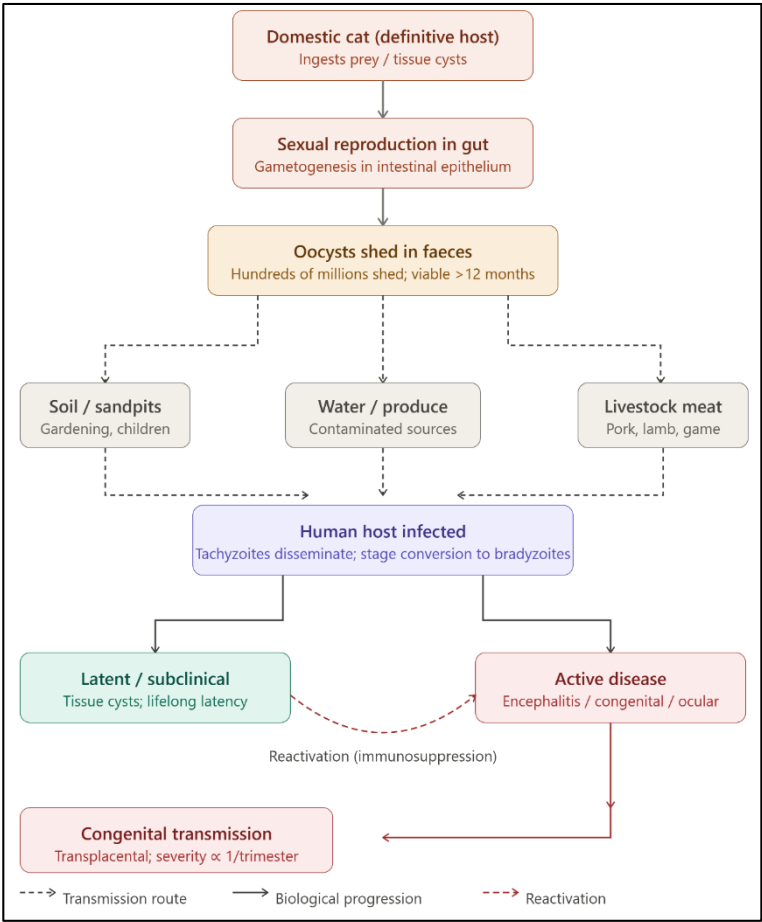
Anthropogenic climate change is projected to alter the geographic envelope of *T. gondii* transmission by extending the range of latitudes within which environmental conditions favour oocyst sporulation and persistence (Yan *et*

al.) [11]. Simultaneously, progressive expansion of immunocompromised populations driven by the global HIV epidemic, broadened transplantation programmes, and escalating use of biologic immunosuppressants amplifies the cohort at risk for severe and reactivated disease.

Pathophysiology and clinical manifestations

Following ingestion, *T. gondii* actively traverses the intestinal epithelium through a highly coordinated invasion process involving secretory products of apical organelles. The moving junction complex assembled from rhoptry neck proteins and the micronemal ligand AMA1 mediates host cell penetration without triggering lysosomal fusion pathways (Dubremetz and Lebrun) [12]. Intracellular tachyzoites reside within a parasitophorous vacuole that resists destruction, establishing a protected replicative niche. Immunological pressure subsequently drives stage conversion from tachyzoites to bradyzoites, with tissue cysts persisting in neural and muscular compartments for the lifetime of the host.

Innate immune control is initiated predominantly through IL-12-driven interferon- γ (IFN- γ) production by natural killer cells and macrophages. Adaptive responses-particularly IFN- γ -secreting CD8 + cytotoxic T lymphocytes and CD4 + Th1 helper cells-are essential for long-term parasite containment (Yarovinsky) [13]. Regulatory immunological circuits limit collateral tissue damage while permitting cyst persistence in latency. The critical dependence on T-lymphocyte-mediated immunity explains why HIV-induced CD4 + T-cell depletion precipitates life-threatening toxoplasmic encephalitis through cyst reactivation.



Flowchart 1: *T. gondii* life cycle and transmission pathways

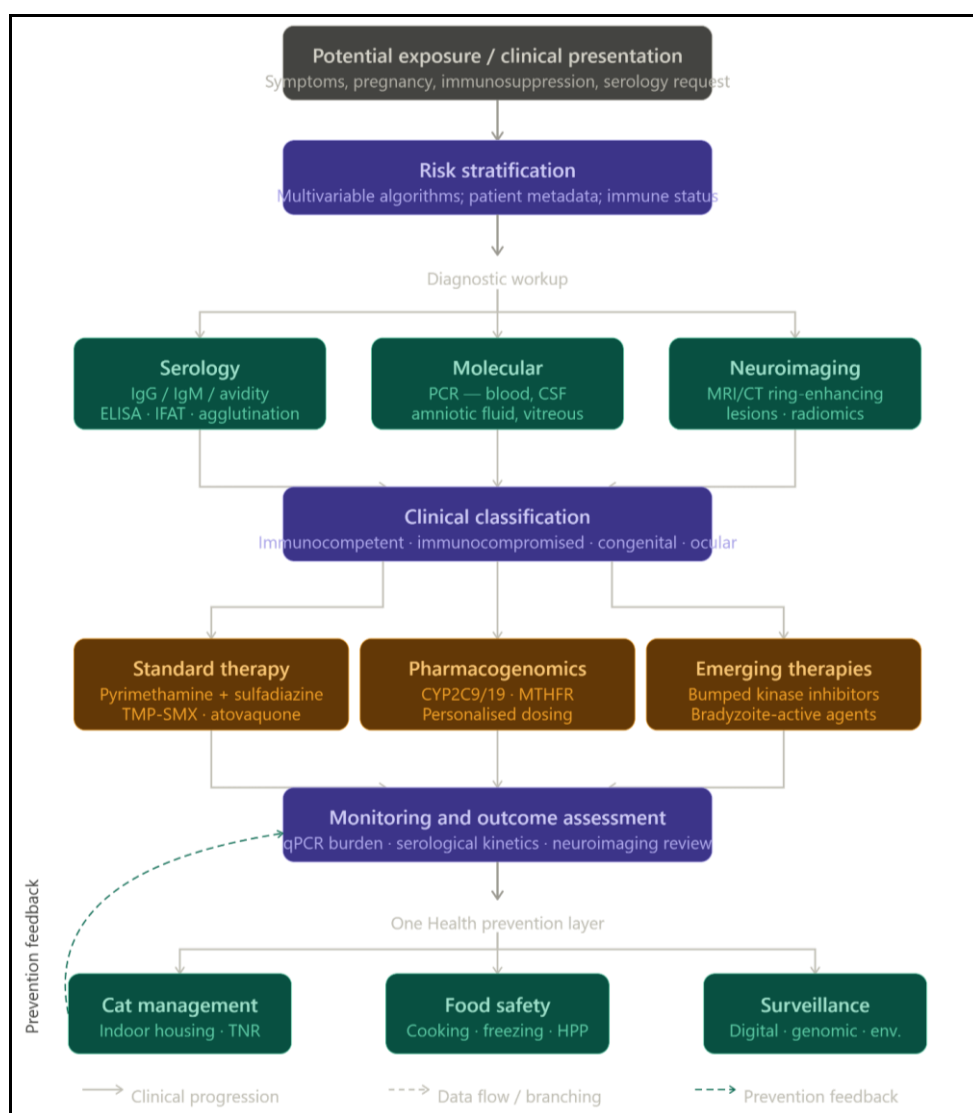
In immunocompetent adults, the overwhelming majority of primary infections are asymptomatic. A minority present with self-limiting cervical lymphadenopathy occasionally mimicking lymphoma and prompting biopsy. Ocular toxoplasmosis, manifesting as necrotizing retinochoroiditis, is the most frequent cause of infectious posterior uveitis and may result from both congenital and postnatally acquired infection; recurrent episodes pose a cumulative threat to vision (Holland) [14]. Congenital toxoplasmosis spans a broad severity spectrum: first-trimester infection carries the greatest risk of severe outcomes—including hydrocephalus, intracranial calcifications, and bilateral chorioretinitis or fetal demise, while third-trimester infection more commonly produces subclinical neonatal disease with later sequelae risk. In immunocompromised hosts, cerebral reactivation presents with focal neurological deficits, altered consciousness, and characteristic multiple ring-enhancing lesions on magnetic resonance imaging (MRI), requiring urgent differentiation from primary central nervous system (CNS) lymphoma.

An accumulating body of epidemiological evidence links chronic *T. gondii* seropositivity with behavioral alterations and psychiatric diagnoses. Pooled meta-analyses report elevated schizophrenia risk among seropositive individuals, with odds ratios in the range of 1.8-2.7; associations with bipolar disorder, obsessive-compulsive disorder, and suicide

attempts have also been documented across multiple independent cohorts (Sutherland *et al.*) [5]. Proposed biological mechanisms include parasite-mediated modulation of dopaminergic neurotransmission, focal neuroinflammation, and production of tyrosine hydroxylase by the parasite an enzyme capable of augmenting catecholamine biosynthesis in infected neural tissue. Whether these associations reflect true causality or confounded correlation remains an active area of scientific investigation.

Conventional diagnostic approaches

Serological detection of *T. gondii*-specific immunoglobulins underpins most diagnostic algorithms in clinical practice. IgG antibodies indicate prior exposure without distinguishing infection chronicity, while IgM positivity suggests recent primary infection though IgM may persist for many months, complicating gestational dating (Montoya and Liesenfeld) [15]. IgG avidity testing exploits progressive maturation of antibody-antigen binding affinity to discriminate recent from remotely acquired infection, and is of particular diagnostic utility in the pregnant patient. Available platforms encompass enzyme-linked immunosorbent assay (ELISA), immunofluorescent antibody tests (IFAT), and various agglutination formats, each characterised by distinct sensitivity-specificity profiles.



Flowchart 2: Integrated toxoplasmosis management pipeline

Polymerase chain reaction (PCR) amplification of *T. gondii* genomic sequences permits direct pathogen detection across a wide range of clinical specimens, including blood, amniotic fluid, cerebrospinal fluid, and vitreous (Robert-Gangneux and Dardé) ^[16]. Real-time quantitative PCR additionally provides parasite burden estimates that may inform treatment monitoring in transplant recipients under active surveillance. In the prenatal setting, amniotic fluid PCR has largely superseded invasive cordocentesis for diagnosis of congenital toxoplasmosis, offering acceptable sensitivity while substantially reducing procedural risk to the fetus.

Tissue biopsy with immunohistochemical staining provides definitive identification of *T. gondii* in tissue-invasive disease; however, the focal distribution of cerebral lesions reduces diagnostic sensitivity. Computed tomography (CT) and MRI of the brain remain the neuroimaging modalities of choice. MRI demonstrates the characteristic profile of toxoplasmic encephalitis: multiple ring-enhancing lesions with surrounding edema, predilection for basal ganglia, and the "eccentric target sign" a finding that, while not entirely pathognomonic, supports the diagnosis in an appropriate clinical and immunological context.

Advanced Diagnostic Approaches

Traditional serological interpretation is constrained by fixed threshold values that generate diagnostic uncertainty in equivocal cases, particularly regarding infection timing during pregnancy a determination with direct clinical management implications. Contemporary multivariable algorithms integrating IgG titers, IgM reactivity, avidity indices, and antigen-specific serological profiles substantially improve infection-timing accuracy (Dard *et al.*) ^[17]. Ensemble statistical methods combining these serological parameters with patient-level metadata including gestational trimester and immunosuppression status generate probability scores that support more nuanced clinical decision-making than is achievable with conventional dichotomous cut-offs, reducing both unnecessary treatment and missed diagnoses.

Deep learning algorithms trained on large brain MRI datasets have demonstrated expert-level performance in detection and characterization of toxoplasmic encephalitis lesions. Convolutional neural network architectures enable automated lesion segmentation, volumetric quantification, and differentiation of toxoplasmosis from mimicking conditions most critically, primary CNS lymphoma, which may present with indistinguishable conventional imaging findings yet demands fundamentally different management (Defined Health) ^[18]. Radiomics pipelines extract high-dimensional quantitative features textural, morphological, and spatial imperceptible to human observers, enabling machine learning classifiers to achieve area-under-the-curve values exceeding 0.90 for this diagnostically challenging differential, with potential for direct integration into clinical reporting workflows.

Risk stratification for vertical *T. gondii* transmission has benefited from multivariable predictive approaches integrating maternal serological kinetics, gestational age at seroconversion, timing of spiramycin prophylaxis initiation, and prenatal ultrasound findings. Such approaches have outperformed traditional logistic regression models in predicting both fetal infection probability and likelihood of clinically significant sequelae (Wallon *et al.*) ^[19]. Natural

language processing algorithms applied to electronic health records can identify at-risk pregnancies by recognizing documented symptom patterns, relevant exposure histories, and flagged laboratory results potentially capturing cases that evade conventional symptom-based screening.

Internet search query analytics, social media activity, and participatory mobile health platforms generate near-real-time signals of disease activity that complement traditional passive surveillance (Charles-Smith *et al.*) ^[20]. Geographic clustering of symptom-related search activity may indicate community-level foodborne exposure events, prompting early targeted public health investigation. Integration of whole-genome sequencing with phylogenetic analysis further enables outbreak source attribution and transmission network reconstruction, with genotyping algorithms distinguishing endemic from imported *T. gondii* strains and informing tailored control responses.

Translational Medicine: From Bench To Bedside

The standard treatment regimen for toxoplasmosis the antifolate combination of pyrimethamine and sulfadiazine supplemented with leucovorin is encumbered by significant toxicity including bone marrow suppression, cutaneous hypersensitivity reactions, and gastrointestinal intolerance (Dunay *et al.*) ^[21]. Alternative regimens incorporating trimethoprim-sulfamethoxazole, atovaquone, or clindamycin offer improved tolerability in selected patients but variable and often inferior efficacy. The most fundamental limitation shared by all currently available agents is their failure to eradicate bradyzoite-stage parasites residing within tissue cysts. This inability to achieve parasitological cure leaves hosts with a persistent viable parasite reservoir and lifelong reactivation risk a therapeutic gap that represents the central scientific impetus for ongoing translational research efforts. Systems biology approaches integrating *T. gondii* genomic, transcriptomic, proteomic, and metabolomic datasets have identified numerous essential parasite pathways amenable to pharmacological disruption. Candidate targets are evaluated according to their essentiality for parasite viability, structural drug ability, selectivity relative to host orthologs to minimize toxicity, and critically activity against the bradyzoite stage enabling potential cure. The apicoplast, a vestigial non-photosynthetic plastid unique to apicomplexan parasites, offers particularly attractive targets because its prokaryote-derived metabolic pathways encompassing isoprenoid biosynthesis, fatty acid synthesis, and heme biosynthesis lack functional mammalian equivalents (Winzer *et al.*) ^[22]. Calcium-dependent protein kinases (CDPKs), absent from mammalian cells but essential to *T. gondii*, represent another selectivity-rich target class, with bumped kinase inhibitors demonstrating encouraging preclinical activity against both tachyzoite and bradyzoite stages.

Computational approaches have substantially compressed the early phases of the drug-discovery timeline. Structure-based virtual screening, now facilitated by high-accuracy protein structure predictions generated by tools such as AlphaFold, enables systematic interrogation of large chemical libraries for compounds with predicted binding affinity to defined target sites (Jumper *et al.*) ^[23]. Generative molecular design algorithms propose novel chemical scaffolds simultaneously optimized for potency, selectivity, aqueous solubility, and metabolic stability, reducing dependence on large-scale empirical compound screening

(Schneider *et al.*)^[24]. Toxicity prediction classifiers trained on historical preclinical datasets enable prospective identification of compounds likely to fail safety evaluation, supporting informed early deprioritization and more efficient resource allocation across the discovery pipeline. No human toxoplasmosis vaccine has achieved regulatory licensure. The live-attenuated Toxovax® preparation, approved for use in ovines, demonstrates proof-of-principle for immunoprophylaxis but is entirely unsuitable for human application. Current translational efforts pursue multiple platform strategies. Subunit vaccines based on recombinant surface antigens-most prominently SAG1 and SAG2-alongside rhoptry proteins (ROP2, ROP18) and dense granule proteins (GRA1-GRA7) elicit protective humoral and cellular immune responses in rodent models (Ong *et al.*)^[25]. DNA vaccines deliver plasmid-encoded antigens and are particularly effective at priming the CD8 + cytotoxic T-cell responses critical for intracellular parasite control. Messenger RNA platforms, whose rapid development was catalysed by the SARS-CoV-2 pandemic, offer the prospect of iterative antigen optimization and robust immunogenicity. Nanoparticulate delivery systems-including lipid nanoparticles and polymer-based carriers enhance antigen stability, facilitate cellular uptake, and augment innate immune activation. Computational epitope prediction tools that identify immunodominant peptide sequences and optimize multivalent antigen combinations are increasingly integrated into the vaccinology design workflow (Ong *et al.*)^[25].

Personalized Medicine Approaches

Interindividual variation in drug-metabolising enzyme activity-particularly polymorphisms in CYP2C9 and CYP2C19-as well as in folate pathway enzymes (MTHFR, DHFR) and drug transporters, substantially influences the pharmacokinetics of pyrimethamine and sulfadiazine and modulates the likelihood of adverse drug reactions (Montazeri *et al.*)^[26]. Pharmacogenomic-guided dosing, integrated within clinical decision-support frameworks, offers a mechanism to personalize therapeutic regimens in a manner that minimizes toxicity whilst preserving anti-parasitic efficacy, particularly in patients requiring long-term maintenance therapy.

Multivariable modelling integrating host immune parameters-including T-lymphocyte subsets, IFN- γ production capacity, and regulatory T-cell frequency-with *T. gondii* genotype data and clinical variables enables individualized disease trajectory prediction. Such models can identify patients requiring intensified monitoring or early prophylactic intervention, improving the efficiency of clinical resource allocation and potentially reducing preventable morbidity in high-risk populations.

Accumulating experimental and clinical evidence implicates gut microbiome composition in shaping susceptibility to *T. gondii* infection and the severity of subsequent disease. Acute toxoplasmosis disrupts intestinal microbial community structure, and specific bacterial taxa appear capable of modulating the magnitude of anti-parasitic immune responses (Cong *et al.*)^[27]. Computational microbiome profiling may ultimately identify dysbiotic signatures associated with elevated disease risk, opening avenues for probiotic or dietary interventions to restore protective microbial configurations before or during infection.

One Health Prevention Strategies

Reduction of environmental oocyst contamination requires coordinated intervention across both owned and feral cat populations. Responsible pet ownership practices-including housing cats indoors, preventing access to hunting prey, and hygienic disposal of litter contents-substantially reduce oocyst environmental loading. Trap-neuter-return programmes for feral colonies provide a humane mechanism for limiting population growth and cumulative shedding risk, with population-level modelling informing optimal intervention densities and spatial targeting to maximize public health benefit.

Foodborne toxoplasmosis prevention relies on adequate cooking of meat to internal temperatures sufficient to inactivate tissue cysts (at least 66 °C throughout), blast-freezing of preparations intended for raw consumption, and consumer education regarding the risks of uncooked pork, lamb, and game. Supply-chain analytics can identify high-risk product streams warranting enhanced inspection protocols (Shapiro *et al.*)^[28]. In commercial food processing, high-pressure treatment represents a validated non-thermal cyst-inactivation technology increasingly available to the industry.

T. gondii oocysts are not reliably inactivated by standard chlorination concentrations employed in municipal water treatment, underscoring the importance of physical filtration and ultraviolet irradiation in surface water management. Individuals engaged in soil contact activities-including gardening and agriculture should wear gloves and wash hands thoroughly. Pregnant women and immunocompromised individuals warrant explicit risk counselling encompassing these environmental exposure pathways.

Effective toxoplasmosis control requires coordinated surveillance spanning human clinical, veterinary, and environmental domains. Automated anomaly-detection algorithms applied to integrated data streams-encompassing notified human cases, cat infection prevalence data, environmental oocyst monitoring, and meat inspection results-can identify unusual patterns warranting investigation before conventional threshold-based surveillance would trigger a response (Mackenzie and Jeggo)^[2].

Challenges and Future directions

Notwithstanding the substantial advances described throughout this review, several fundamental scientific and operational challenges persist. The absence of any therapeutic agent capable of eliminating bradyzoite-stage parasites represents the most critical unmet clinical need, and identifying safe, cyst-active compounds with acceptable systemic tolerability remains a priority. The development of a human toxoplasmosis vaccine similarly requires resolution of unresolved questions regarding optimal antigen selection, the relative contributions of humoral and cellular immune effectors to protection, and the duration of vaccine-induced immunity.

On the computational and operational side, deployment of advanced diagnostic algorithms in low-resource settings-where the burden of congenital and cerebral toxoplasmosis is often greatest-faces constraints of data quality, infrastructure, and health equity. Models trained predominantly on data from high-income settings may perform inadequately across diverse epidemiological and

genetic contexts (Rudin) ^[29]. Intentional inclusion of geographically and demographically representative training datasets, alongside rigorous prospective validation in varied clinical environments, is essential to ensure that technological advances serve all affected communities rather than further concentrating diagnostic advantage in well-resourced settings.

Clarification of the mechanism underlying associations between latent *T. gondii* infection and neuropsychiatric outcomes represents a third high-priority domain. Establishing or refuting causality-through longitudinal cohort analyses, Mendelian randomisation studies, and mechanistic experimental models carries direct implications for population mental health strategy. Finally, regulatory frameworks governing clinical deployment of advanced diagnostic tools must continue evolving to balance the imperative of innovation access with rigorous safety assurance, incorporating prospective validation requirements and robust post-deployment performance monitoring (Rudin) ^[29].

Conclusion

The domestic cat, despite its cherished status as a companion animal, sustains a biologically diverse portfolio of zoonotic risks at the human-animal interface, of which *Toxoplasma gondii* is unquestionably the most globally consequential. The parasite's capacity to infect virtually any warm-blooded host, establish lifelong latency, and cause severe disease in pregnant women, immunocompromised individuals, and congenitally infected neonates renders it a persistent public health concern of the first order. The emerging recognition of neuropsychiatric associations with chronic infection further extends the public health relevance of this organism beyond traditionally recognized clinical domains.

Advances in molecular diagnostics, multivariable risk stratification, advanced neuroimaging analysis, and systems-biology-guided drug discovery have collectively transformed both understanding and management of toxoplasmosis. The translational pipeline for bradyzoite-active therapeutic compounds and novel vaccine platforms offers genuine prospect of durable improved outcomes for affected patients. These scientific gains must, however, be operationalized within a One Health framework that integrates environmental management, animal population control, food safety intervention, and equitable access to diagnostic and therapeutic innovations across all geographies and patient populations.

The future of toxoplasmosis control lies not in any single intervention but in intelligent, sustained integration across the entire epidemiological continuum from environmental management reducing oocyst exposure, through enhanced early detection and risk stratification, to personalized therapeutics aiming at parasitological cure. Achieving this vision demands interdisciplinary collaboration among clinicians, veterinarians, epidemiologists, computational scientists, food safety authorities, and public health policymakers. It represents both a scientific imperative and an ethical obligation to protect the populations most vulnerable to this globally ubiquitous, perennially underestimated pathogen.

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Conflict of Interest

The authors declare that there are no conflicts of interest.

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