

Toxoplasmosis in focus – how a small parasite conquers the world

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Summary

Toxoplasma gondii is one of the most important parasites globally in both veterinary and human medicine, with significant impacts on livestock. Approximately one-third of the global population is latently infected. In humans, infection is typically asymptomatic or causes mononucleosis-like symptoms, but primary infection during pregnancy and reactivation in immunocompromised individuals are of particular concern. Latent infection may be associated with personality and neuropsychiatric disorders or neurodegenerative diseases. Despite a sound rationale, many congenital toxoplasmosis surveillance programmes have been discontinued due to limited effectiveness. Current One Health research focuses on developing new therapies and vaccines to prevent transmission in humans and animals.

Introduction

Toxoplasmosis is among the most common zoonoses worldwide. The infection is caused by the protozoan parasite *Toxoplasma gondii*, one of the most successful parasites globally. A significant portion of the world's population, as well as many animals, is latently infected without being aware of it, but most people become infected later in life through contaminated food.

The initial infection is almost always asymptomatic. In rare cases, it may present with flu-like symptoms, including fever, peripheral lymphadenopathy, and prolonged fatigue. Thereafter, the pathogen remains dormant as a tissue cyst within the host. Of particular concern is primary infection during pregnancy in humans and in animals, which can lead to miscarriage or foetal damage. Reactivation of latent toxoplasmosis can be extremely dangerous for immunocompromised individuals. Moreover, *T. gondii* has been implicated in behavioural changes in infected animals and may play a significant role in the development of neurological and psychiatric disorders in humans.

Global patterns and transmission pathways of *Toxoplasma gondii*

History and seroprevalence

T. gondii was first identified in 1907 by the French physicians and parasitologists Charles Nicolle and Louis H. Manceaux. Its name derives from the Greek words «*toxon*» (bow, arc) and «*plasma*» (formation, shape), as well as from the North African rodent *gundi*, in which the parasite was first discovered. It was not until 1948 that the parasite was identified in humans [1, 2].

Humans, like other warm-blooded animals, act as intermediate hosts for *T. gondii* worldwide. Members of the family *Felidae* (domestic and wild cats) are the definitive hosts. The expansion of agriculture over the past 11,000 years, along with the establishment of the cat–mouse transmission cycle, likely facilitated the global spread of *T. gondii* and the selection of specific parasite lineages. [3]. Although only a limited number of *T. gondii* genotypes predominate in the Northern Hemisphere, a wide variety of genotypes coexist in South America. Moreover, South American strains tend to exhibit higher virulence than those found in the Northern Hemisphere [3].

The parasite is one of the most important foodborne zoonoses globally and a major cause of production losses in agriculture [1, 2]. Depending on the region, 5–70% of humans are latently infected, alongside livestock and rodents [1, 2, 4, 5]. Human seroprevalence is estimated to be around 32% in Europe, 15% in North America, 45% in South America, 54% in Australia, 42% in Africa, and 25% in Asia. Within a single country, prevalence can vary widely across demographic groups depending on culture, socioeconomic status, and diet [4, 5].

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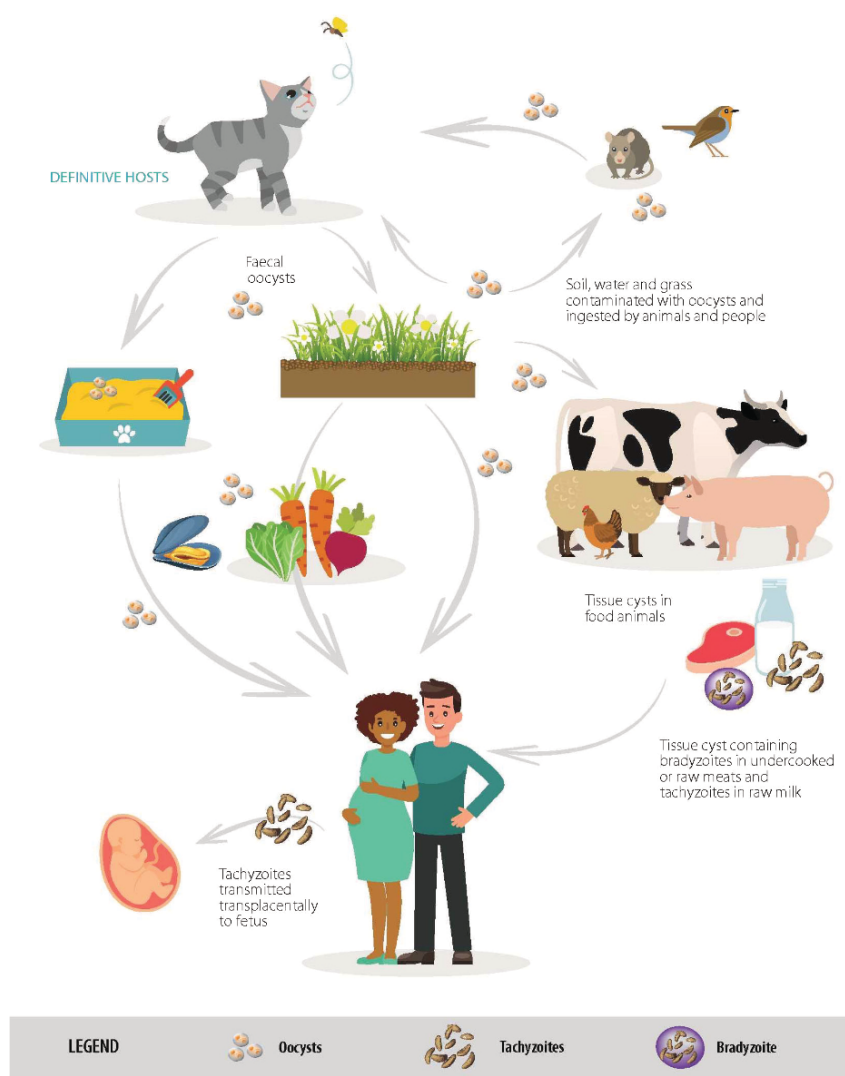
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Over recent decades, *T. gondii* seroprevalence has declined in industrialised countries, likely due to reduced environmental exposure [4, 5]. Among women of childbearing age, rates dropped from 53% to 35% in Switzerland (1982–1999), from 84% to 23% in France (1960–2006), and from 43.3% to 31.5% in Austria (1995–2012) [4–6]. Similar declines have occurred in indoor-raised livestock, while prevalence has increased in free-range animals [1, 2, 10]. Despite improved diagnostics, global data remain fragmented, with substantial variation by region, time, and methodology. In many countries, surveillance is limited, and prevalence data are outdated or incomplete [4].

Pathogenesis and environmental resistance

Toxoplasma gondii belongs to the phylum *Apicomplexa*, which is closely related to *Plasmodium* and *Cryptosporidium*. *Apicomplexa* are single-celled eukaryotic parasites with a complex life cycle involving sexual reproduction in definitive hosts and asexual reproduction in intermediate hosts. They exploit host cells for efficient propagation [1, 2].

Figure 1: Foodborne transmission pathways for *Toxoplasma gondii*. From: EFSA Panel on Biological Hazards (BIOHAZ); Koutsoumanis K, Allende A, Alvarez-Ordóñez A, Bolton D, Bover-Cid S, Chemaly M, et al. Public health risks associated with foodborne parasites. EFSA J. 2018 Dec 4;16(12):e05495. doi: 10.2903/j.efsa.2018.5495, published under the license CC BY-ND 4.0.

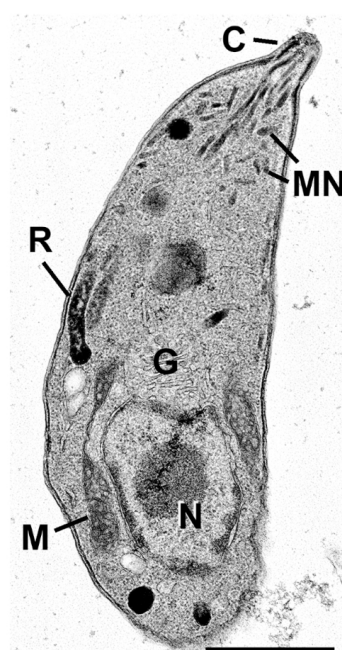


Cats, the definitive hosts, shed vast numbers of oocysts in their faeces (3 to 810 million), which contaminate soil, plants, and water [8]. In warm, moist environments, the process of sporulation is initiated, and infectious sporozoites develop within the oocysts [9]. Sporozoites are resistant to

heat and desiccation and can survive for months in the environment [10]. Intermediate hosts ingest these sporulated oocysts through contaminated food or prey. Humans can acquire the parasite through contaminated water, unpasteurised milk, vegetables, and undercooked meat, or through contact transmission during gardening, cleaning cat litter, or playing in sandboxes [10] (figure 1). Transmission via human breast milk has not been demonstrated (though tachyzoites are excreted in the milk of infected livestock), and direct human-to-human transmission is not possible, except through organ transplants or blood transfusions [1].

In the stomach and intestines, the oocysts dissolve, releasing tachyzoites that invade host cells. A characteristic apical complex, the conoid, aids in cell invasion (figure 2). Current research, including research conducted in Switzerland, focuses on understanding this mechanism [7].

Figure 2: Extracellular tachyzoite with a fully extruded conoid (C, conoid; G, Golgi body; M, micronemes; Mi, mitochondrion; Nu, nucleus; R, rhoptry). The apical complex is used by the parasite to penetrate the host cell. Electron microscopy, scale bar = 1 µm. (Courtesy to Prof. David Ferguson, Nuffield Department of Clinical Laboratory Science, University of Oxford, John Radcliffe Hospital, Oxford, United Kingdom. Source: Koreny L, Zeeshan M, Barylyuk K, Tromer EC, van Hooft JJE, Brady D, et al. Molecular characterization of the conoid complex in *Toxoplasma* reveals its conservation in all apicomplexans, including *Plasmodium* species. *PLoS Biol.* 2021 Mar 11;19(3):e3001081. doi: 10.1371/journal.pbio.3001081.)



Once in the bloodstream, the parasite spreads throughout the host's body. Upon reaching tissues, it becomes less active and forms tissue cysts (bradyzoites), particularly in the skeletal muscles, heart, central nervous system, and retina. These tissue cysts result in chronic, latent infections. Host immune responses, both humoral and cellular, cannot eradicate the parasite but keep it in check, allowing it to persist unnoticed unless immunosuppression triggers reactivation. Only in definitive hosts does the parasite complete its sexual cycle, releasing new infectious oocysts through feline faeces and restarting the cycle.

Clinical manifestations – who falls ill?

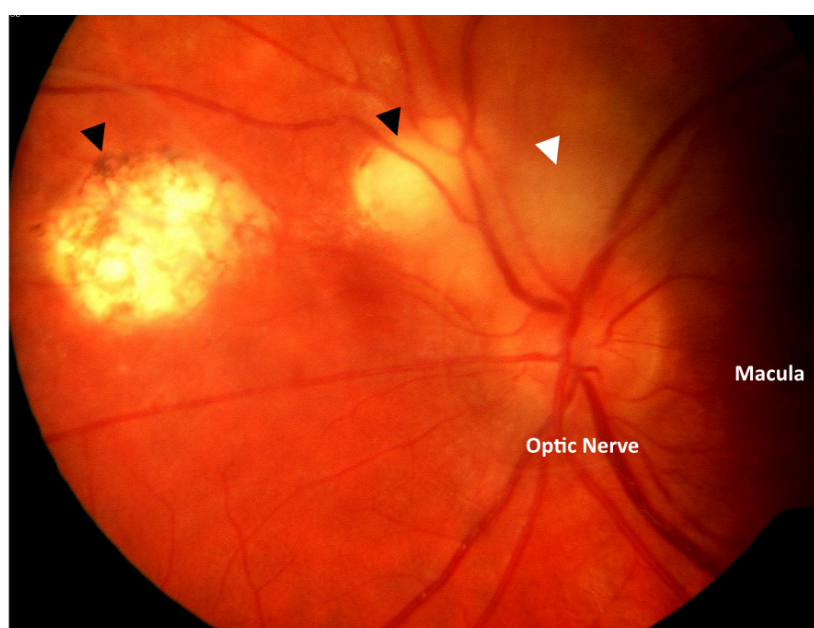
Despite the millions of people globally being exposed to *T. gondii*, clinically significant infections are rare. In immunocompetent individuals, primary infection is often asymptomatic or mild, occasionally presenting with mononucleosis-like symptoms such as fever, peripheral lymphadenopathy, hepatosplenomegaly, and fatigue [1, 2]. Cervical or nuchal lymph node enlargement can persist for extended periods, prompting further medical investigations. Similar presentations can arise from EBV, CMV, HIV, bartonellosis, tularaemia, lymph node tuberculosis, and lymphoma. Severe cases in immunocompetent patients are more common in South America due to different, more virulent *Toxoplasma* strains [1, 3].

Ocular toxoplasmosis and recurrences

Toxoplasmosis may primarily affect the eye and is the leading cause of posterior uveitis. Around 1.5–3% of primary *T. gondii* infections result in ocular toxoplasmosis (OT). Initially, OT was thought to result from congenital infection, but most cases are now believed to be acquired postnatally [11]. It typically presents as unilateral central retinal necrosis (figure 3). If it involves the macula, the area of sharpest vision, significant visual impairment or blindness may result. Persistent vitreous opacities, strabismus, cataracts, and glaucoma can also occur [11, 12].

Four out of five affected individuals experience recurrences, often within two years of the primary infection. Recurrence can follow trauma, stress, or hormonal fluctuations [12]. The specific immunological factors responsible remain unclear. Unlike VZV reactivation, ageing alone is not sufficient to trigger *T. gondii* reactivation. This differs from reactivation in immunocompromised patients, which can lead to severe, necrotising *Toxoplasma* retinitis.

Figure 3: Recurrence of ocular toxoplasmosis (discrete brightening and blurred disc margin, white arrow) nasally above the disc. The black arrows point to old OT scars. (Courtesy to Prof. Dr. med. J. G. Garweg, Bern Eye Clinic.)



Primary infection in pregnancy

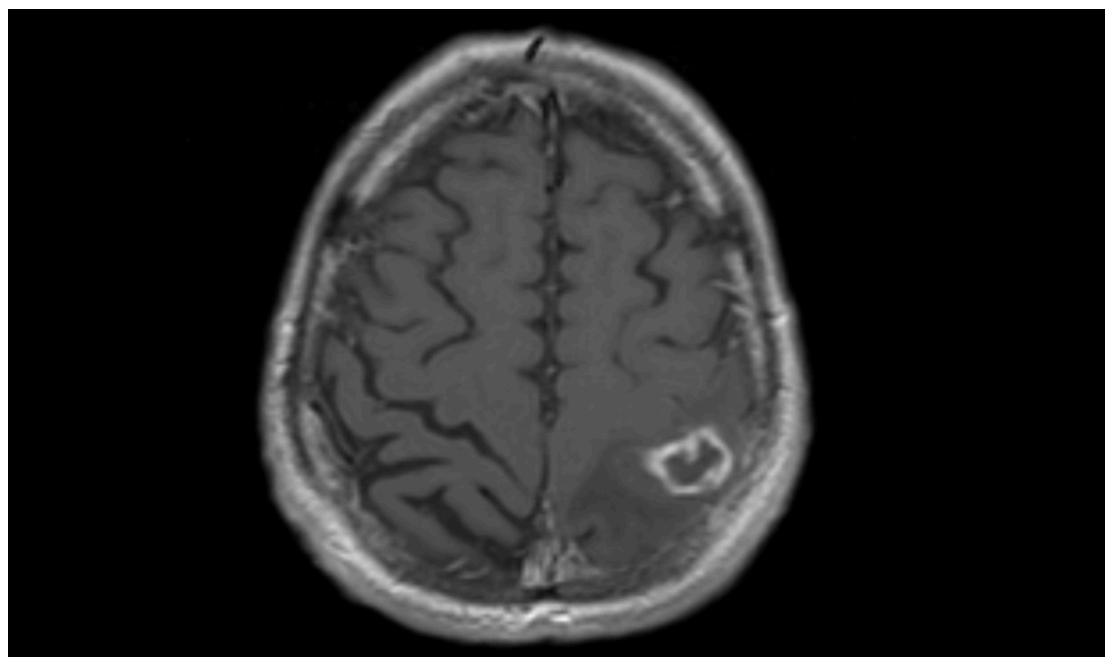
Infection with *T. gondii* can also be acquired vertically through a primary infection during pregnancy or shortly before conception. This feared infection can, in the worst case, lead to miscarriage or permanent damage to the foetus. However, the effects on the child are often less severe, and congenital infection is fortunately rare. In 2022, 173 cases of congenital toxoplasmosis were reported to the ECDC, corresponding to a rate of 5.8 per 100,000 live births [13]. Of congenitally infected infants, 10–25% are symptomatic at birth. The earlier the mother becomes infected, the lower the risk of foetal infection (14%), but the more severe the potential damage to the child. The clinical spectrum is broad, including early miscarriage, hydrocephalus, cerebral calcifications, ocular lesions, hepatosplenomegaly, jaundice, and anaemia, among others [14, 15]. By contrast, infection during the last trimester results in foetal infection in nearly 60% of cases, though it tends to be milder. Some affected children are not diagnosed until adolescence, when they present with impaired vision, hearing or learning difficulties, or epilepsy.

Reactivation during immunosuppression

Reactivation of latent infection occurs when the immune system is weakened and can no longer control the parasite. This can result from highly immunosuppressive therapy, cellular immune dysfunction in haematological cancers, or advanced untreated HIV infection (CD4 <100 cells/μl). In these cases, the disease re-emerges as an opportunistic infection, causing severe damage and

potentially fatal outcomes without treatment. Reactivation can manifest locally (e.g. in the eye, lungs, or CNS) or be disseminated. Patients with encephalitis may exhibit altered consciousness, headaches, seizures, or focal neurological deficits that correlate with multiple ring-enhancing lesions seen on CT or MRI scans (figure 4). Severe, disseminated cases are observed particularly in patients following allogeneic stem cell transplantation (allo-HSCT) [1, 15]. In patients with HIV, these feared reactivations have become rare with the advent of highly active antiretroviral therapy and prophylactic treatment.

Figure 4: MRI of the neurocranium of a patient with HIV and toxoplasmosis encephalitis. A centrally necrotic lesion is visible in the left parietal region with ring enhancement. Axial T1 with contrast.



Toxoplasma gondii as a subtle manipulator?

In humans and rodents, *T. gondii* primarily encysts in the CNS, prompting research into its effects on behaviour and neurodegenerative diseases [16]. *T. gondii* appears to influence its intermediate hosts, such as mice and small rodents, by reducing their ability to detect predators, making them more prone to predation. This loss of predator fear does not seem to be species-specific [16]. Other animals, such as wolves, also show increased risk-taking behaviour following *T. gondii* infection. While it remains unclear whether these effects benefit or harm the host, the parasite undoubtedly benefits by facilitating its global spread.

The implications become unsettling when considering research on *T. gondii*'s potential effects on humans. The parasite may not simply lie dormant in host tissue without leaving traces. Recent studies suggest that *T. gondii* might influence human personality and decision-making [16–19]. Correlations have been reported between *T. gondii* seroprevalence and authoritarian behaviour, tribalism, conservatism, and risk-taking entrepreneurship [17, 18]. Furthermore, chronic CNS inflammation caused by *T. gondii* is thought to contribute to psychiatric or neurodegenerative diseases (e.g. schizophrenia, personality disorders, addiction, and dementia) [18]. Possible underlying mechanisms include chronic neuroinflammation; altered neurotransmitter metabolism, particularly dopamine; and immune-mediated changes. Causality, however, remains unproven, and other factors may contribute to a similar extent [16, 19]. While many questions remain unanswered, it is plausible that *T. gondii* and other parasites have shaped human evolution more profoundly than previously recognised.

Laboratory diagnosis

The Sabin-Feldman dye test, introduced in 1948, was the first specific test to detect the parasite. It revealed the widespread presence of *T. gondii* among warm-blooded animals [1, 15]. Today, various immunoassays are available for serological diagnostics. Diagnosis is based on the detection of specific IgM and IgG antibodies or on evidence of seroconversion. IgM appears within 1–2 weeks and peaks at 4–8 weeks, whereas IgG rises after 2–3 weeks, reaching maximal levels approximately 6–8 weeks after infection [20]. Antibody levels decline following therapy or natural infection, with IgG persisting lifelong, whereas IgM typically disappears within 6–9 months. However, IgM may persist for up to two years [20, 21], making it challenging to distinguish recent infection from prior infection. IgG avidity testing aids in this differentiation, as high avidity rules out infection within the past 3–4 months [20].

Ocular toxoplasmosis diagnosis depends on the characteristic retinal appearance and positive serology. Atypical presentations involving isolated eye infection without detectable serum antibodies may require additional tests (e.g. PCR of the aqueous or vitreous humour and antibody comparison between the aqueous humour and serum) [11, 12]. Negative results from a single test should not rule out the diagnosis, as test sensitivity varies.

Since infection during pregnancy can pose a threat to the unborn child but is difficult to detect, several countries, particularly France, have established surveillance programs with the goal of identifying maternal seroconversion early and preventing transmission to the foetus [6, 13]. According to the EU One Health 2023 Zoonoses report, six European countries, namely Austria, Belgium, France, Greece, Slovakia, and Slovenia, had ongoing active surveillance of congenital toxoplasmosis with mandatory screening of pregnant women [13]. Over the past two decades, these resource-intensive programmes have come under increasing criticism for the following reasons: cost constraints (e.g. in settings with lower or declining prevalence), uncertainties about test reliability, unwarranted anxiety and invasive diagnostics due to persistent IgM antibodies, and insufficient evidence of clinical effectiveness [6, 15]. As a result, many countries, including Switzerland, have discontinued or never implemented routine screening programmes [6, 13].

Neonatal evaluation for toxoplasmosis at birth requires a great deal of attention, as maternal infection is often asymptomatic. If congenital infection is suspected, umbilical cord blood serology (IgA, IgM, IgG) is the preferred method. PCR testing of cord blood or amniotic fluid is also possible, though prenatal amniocentesis is discouraged due to the associated risks and low PCR sensitivity (40–70%) [15].

Reactivation of latent infection in immunosuppressed *T. gondii*-positive patients can be challenging. This applies particularly to patients following stem cell transplantation (HSCT). In this setting, repeated qPCR testing of the blood has become an established follow-up marker [15]. To confirm tissue infection in suspected CNS toxoplasmosis or other focal infections, a biopsy for PCR and histopathological analysis is often needed.

Treatment and pharmacologic prophylaxis

Toxoplasmosis in immunocompetent individuals is typically self-limiting and does not require treatment. Therapy is necessary for severe cases, disseminated infections, encephalitis, pneumonitis, myocarditis, or chorioretinitis [15]. Since the 1950s, the gold standard for non-pregnant patients has been the combination of pyrimethamine and sulfadiazine (Pyr-Sulf). This combination inhibits folic acid metabolism, necessitating concurrent folic acid supplementation. Alternatives include pyrimethamine combined with clindamycin, azithromycin, dapsone, or the antimalarial drug atovaquone. Co-trimoxazole, either alone or in combination with pyrimethamine or clindamycin, also has supporting data. Corticosteroids may be indicated for encephalitis with increased intracranial pressure or for chorioretinitis but are otherwise not recommended [11, 15].

In early pregnancy (first trimester), the macrolide spiramycin was traditionally the drug of choice because it does not cross the placental barrier and is thought to prevent foetal transmission. However, as mentioned above, many experts now discourage early treatment due to a lack of efficacy [6, 15]. From the second trimester onwards and/or in cases of suspected foetal infection, Pyr-Sulf alongside folic acid may be used [15]. It should be noted that spiramycin is no longer available in the Swiss market, and sulfadiazine must be imported. All therapeutic agents target the metabolically active tachyzoite form of the parasite. Currently, no drugs can eliminate dormant tissue cysts.

Severe infections in immunosuppressed patients require prolonged treatment (>6 weeks), ideally extending for 1–2 weeks beyond symptom resolution. In cases of persistent immunodeficiency, patients need prophylaxis after therapy. Co-trimoxazole (TMP-SMX) remains the standard prophylaxis for *Toxoplasma gondii* in severely immunosuppressed patients, including those with advanced HIV, organ or stem-cell transplantation, or intensive immunosuppressive therapy, while also providing concurrent protection against *Pneumocystis jirovecii* [15].

Preventive measures and novel approaches

Avoiding toxoplasmosis entirely may seem almost impossible given the parasite's efficient spread and its robust oocysts. *T. gondii* is nearly ubiquitous, found in food, water, and the environment [1, 10]. Nevertheless, seropositivity correlates with sociocultural factors, dietary habits, and access to adequate sanitation and clean drinking water [1]. Safe food, water, and hygiene practices can reduce the risk of infection. Key measures include thoroughly cooking meat, washing fruits and vegetables, and maintaining good hand hygiene after handling raw meat, soil, and cat litter.

Developing new therapeutic and preventive strategies to protect the global population from this cunning parasite remains a significant challenge. One Health research initiatives aim to create a multisectoral vaccine to protect both humans and livestock while reducing oocyst excretion by cats [22, 23]. Conditions are favourable for such advances. For instance, an attenuated live vaccine already prevents miscarriages in sheep. However, key challenges remain, particularly the parasite's complex biology, poor cross-species protection, limited translatability of animal models, and persistent regulatory and economic obstacles [22, 23]. Another promising approach involves therapeutics that prevent the parasite from invading host cells and agents that are capable of eliminating tissue cysts [3, 24, 25, 26]. If these initiatives succeed, the world could be relieved of a significant burden.

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