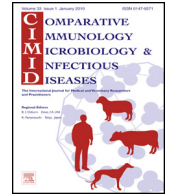




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Review

Molecules to modeling: *Toxoplasma gondii* oocysts at the human–animal–environment interface

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ABSTRACT

Environmental transmission of extremely resistant *Toxoplasma gondii* oocysts has resulted in infection of diverse species around the world, leading to severe disease and deaths in human and animal populations. This review explores *T. gondii* oocyst shedding, survival, and transmission, emphasizing the importance of linking laboratory and landscape from molecular characterization of oocysts to watershed-level models of oocyst loading and transport in terrestrial and aquatic systems. Building on discipline-specific studies, a One Health approach incorporating tools and perspectives from diverse fields and stakeholders has contributed to an advanced understanding of *T. gondii* and is addressing transmission at the rapidly changing human–animal–environment interface.

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1. Introduction

Understanding the ecology and epidemiology of disease in the context of ecosystems is critical for preserving human and animal population health. Over 7 billion people now depend upon the earth's resources [1], and global environmental change has made disease migration to new hosts and landscapes a reality rather than a potential threat [2,3]. Anthropogenic activities, in particular, reshape landscapes, climate, and species distributions and interactions across the globe, with significant potential to alter patterns of pathogen emergence and spread [4–7]. Habitat conversion, introduction of non-native species, and increased contact between human, domestic animal, and wildlife populations have been linked to emerging viral, bacterial, and parasitic diseases [8]. Climate change also has the potential to alter cycles of disease transmission by influencing vector and host ranges, pathogen survival, and dynamics of water-borne transmission [8,9]. Of the 1415 organisms documented as human pathogens, over 60% are believed to have come from domestic or wild animal reservoirs [10]. Examining animal, human, or environmental health alone ignores the vital links between these components.

Historical biological and geographic boundaries of disease transmission will likely continue to shift significantly with changing environmental conditions, making a more holistic, One Health approach to pathogen research and management essential. By linking diverse non-academic stakeholder communities and researchers from different disciplines, the One Health approach builds a synergistic base from which to study and address the unique health challenges emerging at the human–animal–environment interface in a changing global environment [11,12].

Toxoplasma gondii, a globally distributed, zoonotic, protozoan parasite capable of infecting a wide range of warm-blooded animals [13], provides a broadly applicable example of the complexity of pathogen transmission among diverse hosts and environments and illustrates the need for a One Health approach to better understand disease ecology and epidemiology. Although long-studied in terrestrial landscapes, *T. gondii* has also emerged as a significant aquatic pathogen linked to marine mammal infection and water-borne outbreaks of disease in humans around the world [14]. Oocysts, the exceptionally hardy free-living environmental stage of the parasite, play a key role in transmission of *T. gondii* to newly recognized hosts and ecosystems. As wild and domestic felids are the only known hosts capable of shedding *T. gondii* oocysts in their feces [15–17], infection of people and animals through contaminated terrestrial and aquatic sources emphasizes the need to jointly examine human, domestic animal, and wildlife populations. While parasitologists, physicians, veterinarians, ecologists, and molecular biologists have studied *T. gondii* independently, understanding how a traditionally terrestrial pathogen is emerging in new environments requires more integrated knowledge. For *T. gondii* and other pathogens, creating a more collaborative approach to research and management from molecular to landscape levels has enhanced our understanding of health at the human–animal–environment interface.

2. Importance of oocysts in transmission of *T. gondii* infections

Warm-blooded animals, including humans, are typically infected with *T. gondii* through one of three pathways: ingesting oocysts from the environment (through contaminated water, soil, or food), eating an infected intermediate host with *T. gondii* cysts in its tissues, or congenital transmission from infected mothers to offspring [13,18]. Additional potential routes of transmission, including *T. gondii* tachyzoite-contaminated sperm and unpasteurized milk, have been demonstrated, but are thought to be rare sources of infection [19–21]. Uncommon cases of human infection following blood transfusion or organ transplantation from *T. gondii*-infected donors have also been reported [22,23]. However, oocyst-induced infections are increasingly recognized as a significant route of *T. gondii* transmission (Fig. 1) [14,24].

A key feature of oocyst-borne infections is that the majority of human cases with clinical symptoms were reported in immunocompetent individuals, which contrasts with the traditional dogma that 90% of acquired cases of toxoplasmosis in healthy hosts are asymptomatic [25]. Clinical disease from acquired toxoplasmosis has been linked to strain virulence (as reviewed by [26]), and may be more severe when intermediate hosts acquire the infection through the oocyst versus tissue stages of *T. gondii* [27–29]. As reviewed by Grigg and Sundar [30], a number of studies link oocyst infections to symptomatic disease in immunocompetent individuals, with signs ranging from mild flu-like illness to more disseminated and severe outcomes, such as chorioretinitis, neurologic deficits, aborted fetuses, and even death. While outbreaks associated with consumption of bradyzoite tissue cysts in undercooked meat have been reported, the numbers of individuals affected are minimal as compared with documented oocyst outbreaks [30].

2.1. Terrestrial oocyst transmission

Widespread infection in chickens, grazing livestock, and herbivorous wildlife species from diverse geographic locations provides evidence of environmental exposure to *T. gondii* oocysts [31,32]. Omnivorous animals may acquire *T. gondii* infection through ingestion of tissue cysts, but infection from oocyst-contaminated food or water has also been reported [33]. Additionally, human infection has been repeatedly linked to terrestrial environmental sources including oocysts in domestic cat (*Felis catus*) litter boxes and soil as well as ingestion of contaminated unwashed fruits and vegetables [34–36]. Oocysts have been recovered from soil under natural conditions [37,38], and epidemiological studies indicate that there is an increased risk of acquired *T. gondii* associated with soil exposure (Tables 1 and 2 [39]). While differentiating routes of *T. gondii* acquisition has been historically difficult, a recently recognized oocyst-specific antigen [27,40,41] applied in a study of mothers of congenitally infected infants in the United States demonstrated that 78% of these women (59 of 76), had oocyst-acquired infections [41].

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