

Toxoplasma gondii

- [Taxonomy](#)
- [Brief facts](#)
- [Infection pathways](#)
- [Life cycle image](#)
- [Predominant genotypes](#)
- [Developmental stages](#)
- [Tissues](#)
- [References](#)

Brief facts

cellular organisms - Eukaryota - Alveolata - Apicomplexa - Coccidia - Eucoccidiorida - Eimeriorina - Sarcocystidae - Toxoplasma - *Toxoplasma gondii*

Brief facts

- *Toxoplasma gondii* is an obligate intracellular parasite capable of infecting a wide variety of mammals and birds. This single-celled protozoan organism might cause a disease known as **toxoplasmosis**.
- Cats (***Felis catus***, [domestic cat at GeoChemBio: taxonomy, brief facts, digestive system, development](#)) are **primary hosts** of *Toxoplasma gondii*. About sexual cycle of the parasite and its distribution by cats' feces please see section Developmental stages below.

- Between 15 and 85% of the world adult human population is chronically infected with *Toxoplasma gondii* depending on geographical location.
- Toxoplasmosis is a potentially fatal disease of the developing human fetus and immunocompromised (e.g., AIDS and transplant) patients and can cause severe ocular disease in otherwise healthy individuals.
- Most infections remain asymptomatic and treatment is not prescribed, however, toxoplasmosis is strongly linked with development of psychosis, depression, and anxiety disorders, reckless behavior and impulsivity in youth. Unfortunately, all above mentioned disorders are quite common and their etiology often is not explored fully.
- Experiments on rats and mice, natural prey of cats, had shown that infected rats and mice exhibit hyperactivity, fearlessness toward cat urine odor, and novelty seeking behavior - types of conduct that most likely make them victims of cat predation. It is speculated that the pathogen targets brain of an intermediate host to increase its exposure and vulnerability to its natural predators. Although some hosts, such as humans, cannot be considered to become a prey of a cat, it can be postulated that all hosts can be affected by similar pathways.
- According to most recent studies, it seems that these pathogens can secrete protein kinases into host cells to subvert host-cell signalling pathways and that this explains many of the differences in virulence among the three dominant clonal lineages.
- Recent population genetic studies have identified a remarkably limited number of *Toxoplasma gondii* genotypes in nature, the vast majority of which fall into one of only three distinct lineages. They can be associated with different types of toxoplasmosis as a disease (ocular, brain, muscle, necrosis, etc.).
- Genetic analysis of strains indicates that *Toxoplasma gondii* sexual recombination between different strains of the parasite is very rare in natural populations of the host (felines).

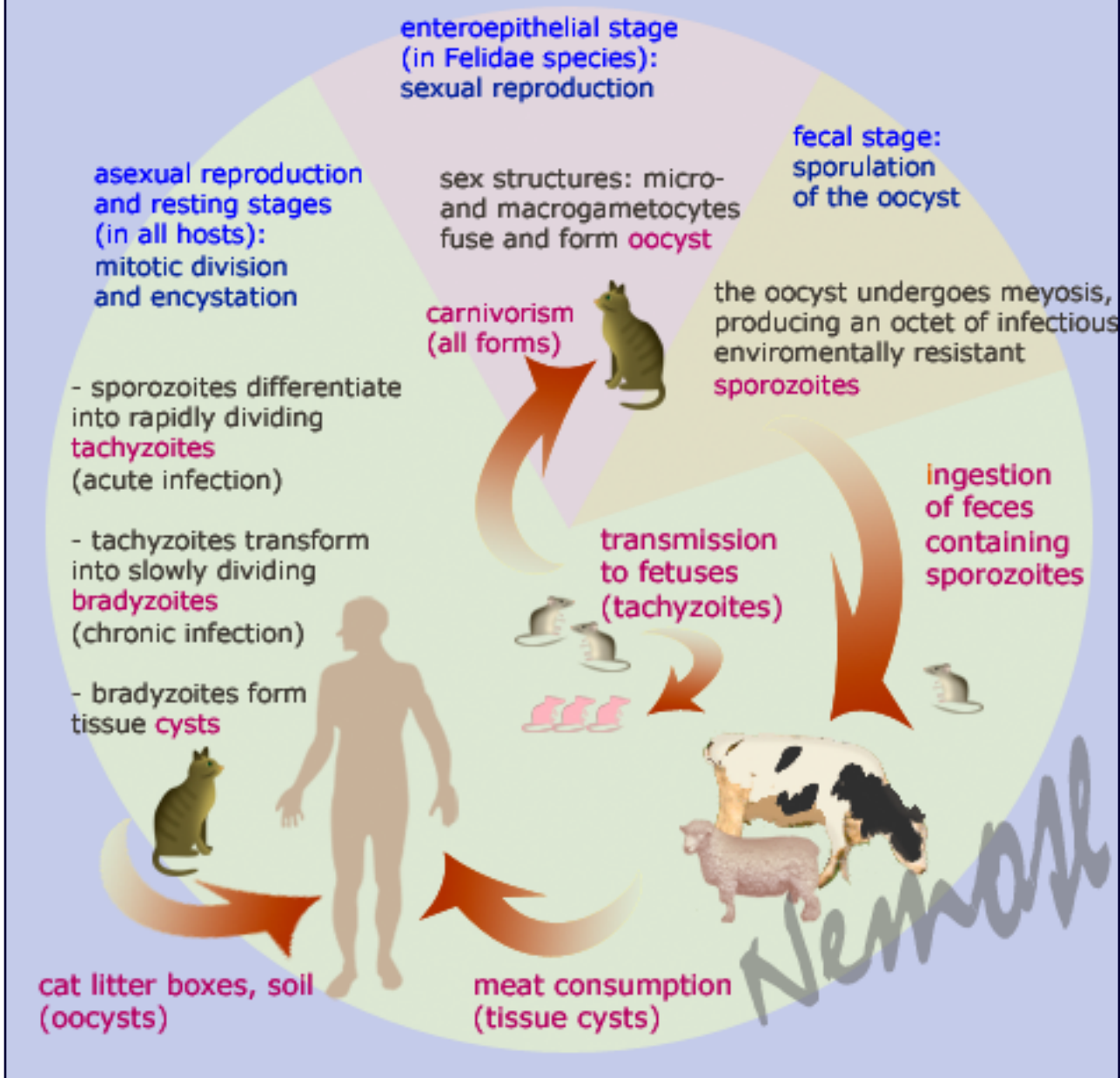
Infection pathways

- from mother to fetus during pregnancy
- inhaling oocysts (from litter boxes of domestic cats, from soil)
- contamination of hands with feline feces while gardening, playing in sandboxes, etc.
- ingestion of toxoplasma cysts contained in contaminated undercooked meat

Predominant genotypes of *T. gondii*

- Type I is highly virulent in mice and, perhaps, in human.
- Type II predominates in infections of immunocompromised patients; relatively avirulent in mouse. Type II strains are most often associated with human toxoplasmosis, especially in Europe.
- Type III is not highly virulent in mouse, and causes chronic infections in humans.

Life cycle of *Toxoplasma gondii*



Developmental stages

- **sexual**

this part of the life cycle takes place only in members of the *Felidae* family (domestic and wild cats), after ingestion of flesh of infected animal (any warm-blooded animal including birds)

- **enteroepithelial stage**

after ingestion of tissue cysts, the parasites invade the cat enterocytes, undergo several rounds of division and differentiate into microgametocytes and macrogametocytes

- **division**

proliferation of the parasites in the cat enterocytes

- **differentiation**

parasites differentiate into sexual structures, **microgametocytes** and **macrogametocytes**

- **zygote**

the gametocytes fuse to form a zygote or oocyst that is shed into the environment with the cat feces

- **fecal stage**

- **meiosis**

the oocyst undergoes meiosis, producing an octet of highly infectious sporozoites that are resistant to environmental damage and may persist for years in a moist environment

- **asexual**

asexual reproduction begins after ingestion of the cat feces by a secondary host such as a mouse

- acute infection stage

sporozoites differentiate into the rapidly dividing tachyzoite form, which establishes and sustains the acute infection

- chronic infection stage

a chronic phase of the disease ensues, as the tachyzoite changes into a slowly dividing form known as the bradyzoite

- resting stage

latent bradyzoite tissue cysts persist for the life of the host mainly within the muscles and brain; the cysts are very difficult to eradicate entirely because they rest inside the host cells

Tissues

- cyst

a small thick-walled sac that encloses dormant bradyzoites; usually resides in muscle or brain tissues; resting stage

- oocyst

zygote produced by gametocytes fusion; mature oocyst consists of wall surrounding two sporocysts, each of which is comprised of a sporocyst wall surrounding four sporozoites; fecal stage

- unsporulated oocyst

- sporulated oocyst

mature oocyst containing sporozoites

- sporozoite

highly infectious form of *T. gondii* produced by meiosis of the oocyst; resistant to environmental damage and may persist for years in a moist environment

- tachyzoite

sporozoites differentiate into tachyzoites - motile haploid, asexually reproducing form of the parasite - that establish and sustain the acute infection; upon host cell penetration tachyzoites form the **parasitophorous vacuole (PV)**; PV membrane (PVM) surrounds dividing parasites and protects them from acidification by host's endocytic vesicles; *T. gondii* presents three organelles that are involved in host's cells invasion, formation of PV, nutrient acquisition, and, above all, subversion of host's defenses, including suppression of apoptosis: **microneme proteins (MICs)**, **rhoptry proteins (ROPs)**, and **dense granules**

- bradyzoite

a slowly dividing form of the parasites; latent bradyzoite tissue cysts persist for the life of the host, re-emerging occasionally, thus sustaining chronic infection

References

PubMed articles

- Saeij JP et al. Polymorphic secreted kinases are key virulence factors in toxoplasmosis. *Science*. 2006 Dec 15; 314(5806): 1780-3. **PMID: 17170306**

- Sibley LD, Boothroyd JC. Virulent strains of *Toxoplasma gondii* comprise a single clonal lineage. *Nature*. 1992 Sep 3;359(6390):82-5. **PMID: 1355855**
- Laliberté J. and Carruthers VB. Host cell manipulation by the human pathogen *Toxoplasma gondii*. *Cell Mol Life Sci*. 2008 Jun;65(12):1900-15. **PMID: 18327664**
- Yolken RH, Torrey EF. Are some cases of psychosis caused by microbial agents? A review of the evidence. *Mol Psychiatry*. 2008 May;13(5):470-9. Epub 2008 Feb 12. **PMID: 18268502**
- Webster JP. The effect of *Toxoplasma gondii* on animal behavior: playing cat and mouse. *Schizophr Bull*. 2007 May;33(3):752-6. Epub 2007 Jan 11. **PMID: 17218613**

Websites

- [James W. Ajioka, Jennifer M. Fitzpatrick and Christopher P. Reitter \(2001\) *Toxoplasma gondii* genomics: shedding light on pathogenesis and chemotherapy.](#)
-



Last updated 03/15/09
nemose@live.com

[Privacy policy](#)

©Nemose 2008 - 2009 All rights reserved