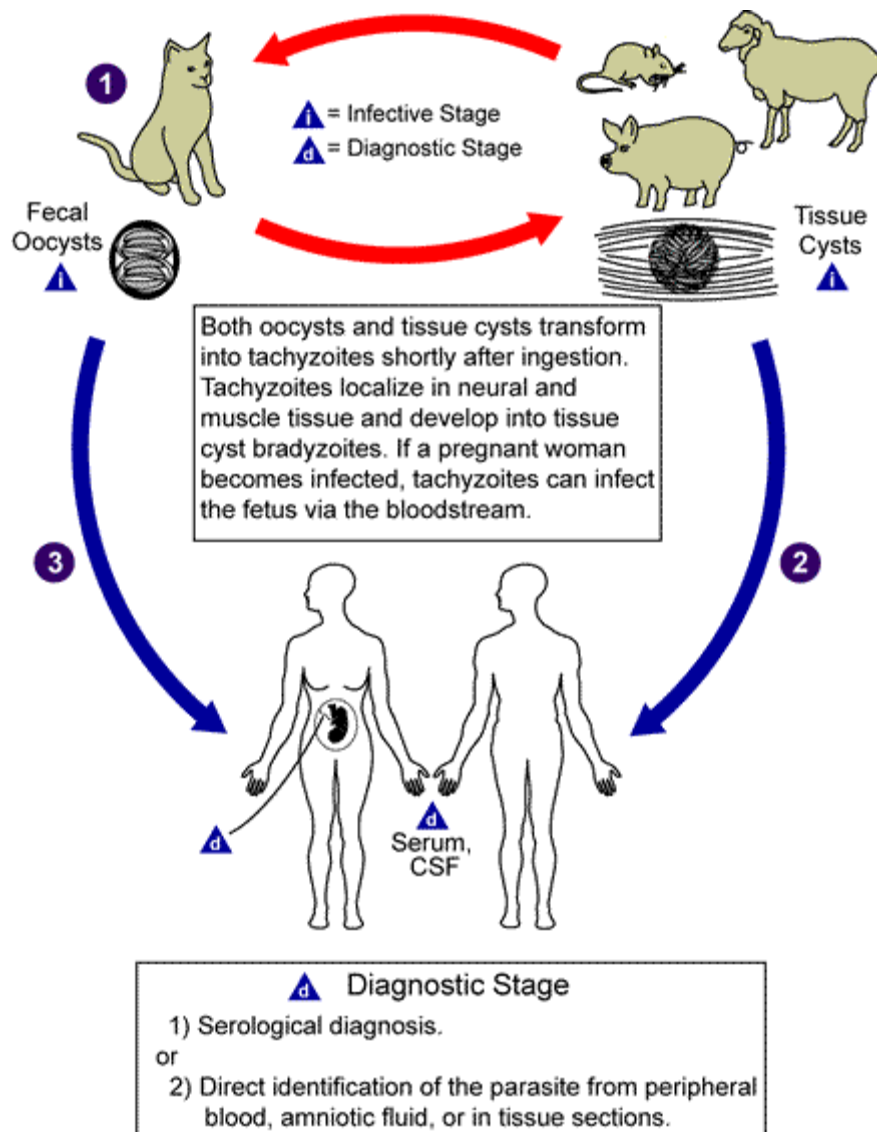


Toxoplasmosis

Causal Agent:

Toxoplasma gondii is a protozoan parasite that infects most species of warm blooded animals, including humans, causing the disease toxoplasmosis.



<http://www.dpd.cdc.gov/dpdx>

Life Cycle:

Members of the cat family (Felidae) are the only known definitive hosts for the sexual stages of *T. gondii* and thus are the main reservoirs of infection. Cats become infected with *T. gondii* by carnivorousism ❶. After tissue cysts or oocysts are ingested by the cat, viable organisms are released and invade epithelial cells of the small intestine where they undergo an asexual followed by a sexual cycle and then form oocysts, which are excreted. The unsporulated oocyst takes 1 to 5 days after excretion to sporulate (become infective). Although cats shed oocysts for only 1 to 2 weeks, large numbers may be shed. Oocysts can survive in the environment for several months and are remarkably resistant to disinfectants, freezing, and drying, but are killed by heating to 70°C for 10 minutes.

Human infection may be acquired in several ways: A) ingestion of undercooked infected meat containing *Toxoplasma* cysts ❷; B) ingestion of the oocyst from fecally contaminated hands or food ❸; C) organ transplantation or blood transfusion; D) transplacental transmission; E) accidental inoculation of tachyzoites. The parasites form tissue cysts, most commonly in skeletal muscle, myocardium, and brain; these cysts may remain throughout the life of the host.

Geographic Distribution:

Serologic prevalence data indicate that toxoplasmosis is one of the most common of humans infections throughout the world. Infection is more common in warm climates and at lower altitudes than in cold climates and mountainous regions. High prevalence of infection in France has been related to a preference for eating raw or undercooked meat, while high prevalence in Central America has been related to the frequency of stray cats in a climate favoring survival of oocysts. The overall seroprevalence in the United States as determined with specimens collected by the third National Health and Nutritional Assessment Survey (NHANES III) between 1988 and 1994 was found to be 22.5%, with seroprevalence among women of childbearing age (15 to 44 years) of 15%.

Clinical Features:

Acquired infection with *Toxoplasma* in immunocompetent persons is generally an asymptomatic infection. However, 10% to 20% of patients with acute infection may develop cervical lymphadenopathy and/or a flu-like illness. The clinical course is benign and self-limited; symptoms usually resolve within a few months to a year. Immunodeficient patients often have central nervous system (CNS) disease but may have retinochoroiditis, or pneumonitis. In patients with AIDS, toxoplasmic encephalitis is the most common cause of intracerebral mass lesions and is thought to be caused by reactivation of chronic infection. Toxoplasmosis in patients being treated with immunosuppressive drugs may be due to either newly acquired or reactivated latent infection. Congenital toxoplasmosis results from an acute primary infection acquired by the mother during pregnancy. The incidence and severity of congenital toxoplasmosis vary with the trimester during which infection was acquired. Because treatment of the mother may reduce the incidence of congenital infection and reduce sequelae in the infant, prompt and accurate diagnosis is important. Most infants with subclinical infection at birth will subsequently develop signs or symptoms of congenital toxoplasmosis unless the infection is treated. Ocular *Toxoplasma* infection, an important cause of retinochoroiditis in the United States, is frequently a result of congenital infection. Patients are often asymptomatic until the second or third decade of life, when lesions develop in the eye.

Laboratory Diagnosis:

The diagnosis of toxoplasmosis may be documented by:

- Observation of parasites in patient specimens, such as bronchoalveolar lavage material from immunocompromised patients, or lymph node biopsy.

- Isolation of parasites from blood or other body fluids, by intraperitoneal inoculation into mice or tissue culture. The mice should be tested for the presence of *Toxoplasma* organisms in the peritoneal fluid 6 to 10 days post inoculation; if no organisms are found, serology can be performed on the animals 4 to 6 weeks post inoculation.
- Detection of parasite genetic material by PCR, especially in detecting congenital infections in utero.
- Serologic testing is the routine method of diagnosis, because the techniques described above are technically complex and generally not rewarding.

Diagnostic findings

- Microscopy
- Antibody detection

Treatment:

Treatment is not needed for a healthy person who is not pregnant. Symptoms will usually go away within a few weeks. Treatment may be recommended for pregnant women or persons who have weakened immune systems.