

Toxoplasma gondii - only species of the genus

> clonal population structures – different genotypes



In the life cycle – several infection stages – relevant for parasite transmission

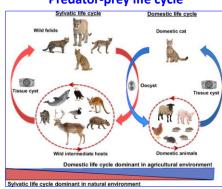
Toxoplasma gondii

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

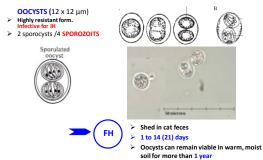
- > cosmopolitan distribution
- > seropositive prevalence rates vary ≽generally 20-75%
- > generally causes very benign disease in immunocompetent adults
- > tissue cyst forming coccidia

 - predator-prey life cycle
 felines are definitive host
 - infects wide range of birds and mammals (intermediate hosts)

Predator-prey life cycle

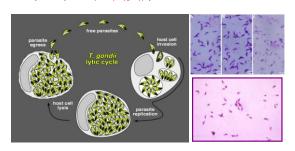


PARASITE FORMS



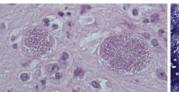
Tachyzoites

- ➤ Most common in brain, skeletal muscle, and cardiac muscle ➤ Rapid multiplication (endopolygeny) lyses cell

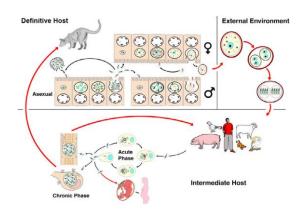


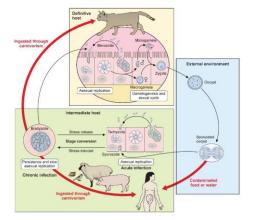
Bradyzoite Stage

- > dormant, slowly replicating (endodyogeny)
- > due to host immune response
- \triangleright chronic or latent infection 50 150 μ m (1000 3000 bradyzoits) (mainly in the brain, skeletal muscle, and cardiac muscle)
- > Remain for the life of the host
- > Recrudescence of infection in immunocompromised hosts





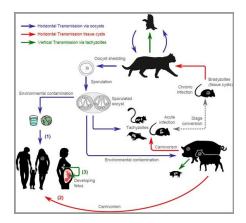




Bradyzoite cysts are highly infective if ingested



- Bradyzoites marks the beginning of the chronic phase of infection; Tissue cysts show very little evidence of
- inflammation or immune cell infiltrates;
- Bradyzoites (not tachyzoites) are resistant to low pH and digestive enzymes during stomach passage;
 - Protective cyst wall is finally dissolved and bradyzoites infect tissue and transform into tachyzoit;



Feline Clinical Toxoplasmosis

- > High prevalence, but very rare
- > Effects young and immunocompromised
- > Reactivation of infection in older cats
- ➤ Most common in < 2 year olds
- Infects all nucleated cells; no erytrocytes!
 Cats support both sexual and asexual development
- Enteritis, lymphadenopathy, pneumonia (esp. in FIV + cats), encephalitis, nephritis, anorexia, weight loss, lethargy,dyspnoea, ocular signs
- Most cats asymptomatic
- > Transplacental transmission rare in cats

Feline Clinical Toxoplasmosis



Pre-patented period: 20-24 days (with oocysts) 7-10 days (when consuming tachyzoites) 3-5 days (when chewing the right / bradyzoites)

Excretion oocyte - 1x for life, 5-7 days, max. 14- 21

days;

> eve form - retinitis (retinal inflammation), uveitis, chorioretinitis; > lethal course in cats with impaired immune system;

Diagnosis - Felines

Detection of oocysts

- Difficult, shedding done before clinical signs
- Dif. dg: Hammondia hammondi

Serology

- IgG-class immunoglobulins,
- IgM-class, higher titers





Seropositive cat is epidemiologically safe - only in immunosuppressed cats can secondary infection and oocyst production occur;

Oocysts produced into the environment from DH - cats are not immediately infectious because they are not shed; therefore, direct contact with a cat does not usually result in infection.

Clinical signs - dogs (IH)

- > Acute increased temperature, lymph node enlargement, nose and eye discharge, tonsillitis, dyspnoea, diarrhea, progressive weight loss, pneumonia (most commonly);
- Chronic hepatitis, myocarditis, pneumonia; in dogs frequent nerve symptoms during CNS involvement - apathy, tremor, ataxia, hemiparesis, paresis, paralysis;

Toxoplasma gondii and other IH - food animals

- Large ruminants Aborts
- Small ruminants sheep, goats abortions, CNS disorders
- Pigs as with dogs temperature, enlarged lymph nodes, nose and eye discharge
- ➤ Horses
- ➤ Race game
- Poultry

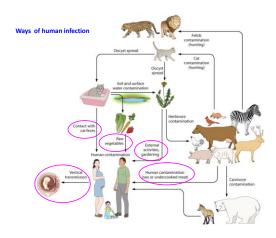




Treatment

- Toltrazuril cats (20 mg/kg b.w.)
- Clindamycin (15 mg/kg b.w.) + trimetoprim (10 mg/kg b.w.) - dogs, cats with systemic clinical toxoplasmosis;
- Sulfonamids + trimetoprim in dogs at regular doses;

Toxoplasmosis Zoonotic Potential Human Transmission



Leisure activities on contaminated sand/ lawn Gardening in contaminated soil Gardening in contaminated soil Consumption of contaminated raw or undercooked meat, unpasteurized goat's milk Embryo Infection Contact with surfaces contaminated by cat feces Contaminated water Consumption of contaminated raw or undercooked meat, unpasteurized goat's milk

Pathogenesis

The **tachyzoites directly destroy host cells**, especially parenchymal and reticuloendothelial cells.

Clinical Features

- > Prenatal (congenital) toxoplasmosis
- Postnatal toxoplasmosis

Prenatal toxoplasmosis may cause:

- > Stillbirths
- ➤ Chorioretinitis
- > Intracerebral calcification
- > Hydrocephaly/microcephaly
- Neurological DamageLearning Difficulties

Congenital Toxoplasmosis

- > acute infection of the expectant mother. Severity depends on the stage of pregnancy
- > severity varies with age of fetus
 - > more severe early in pregnancy
 - > more frequent later in pregnancy
- > Spontaneous abortions or neurological disorders such as blindness and mental retardation can result.
- stillbirth, premature birth, or full-term ± overt disease, chorioretinitis, Intracerebral calcification, Hydrocephaly/microcephaly, Neurological Damage, Learning Difficulties

Congenital toxoplasmosis

- If a women is infected for the first time during pregnancy the parasite can transverse the placenta and cause fetal disease;
- Both the probability and severity of the disease depend on when the infection takes place during pregnancy (early: low transmission, but severe disease, late: high transmission, more benign symptoms);





Ocular Toxoplasmosis

- >chorioretinitis: likely due to both active proliferation and immune parasite hypersensitivity
- >generally a recrudescence rarely from primary infection
- congenital infection
- >20% exhibit ocular symptoms at birth
- >82% by adolescence
- >most lesions are focal and self-limiting
- >rapidly destructive in AIDS patients

Clinical Features

Acquired Postnatal toxoplasmosis

Postnatal toxoplasmosis may cause:

- > Lymph node infection similar to mononucleosis
 - > Local hypersensitivity
- ➤ Blood vessel blockage
- > Cell death near the cyst
- > All of these symptoms are rare in healthy human patients;
- ➤ 1-2 week incubation period
 ➤ acute parasitemia persists for several weeks until development of tissue cysts
 - ➤often asymptomatic (>80%)
 - >a common symptom is lymphadenopathy without fever
 - >occasionally mononucleosis-like (fever, headache, fatigue, myalgia)
- > likely persists for life of patient
- immunosuppression can lead to reactivation (eg., organ transplants)

Diagnosis

- > Because methods for direct evidence of toxoplasma are very difficult, basic laboratory diagnosis relies primarily on indirect diagnostic methods for evidence of toxoplasma antibodies in serum.
- > Investigative procedures consist of a combination of basic and complementary (supra-basic) investigative methods.
- > By combining appropriate tests allowing the determination of individual classes of immunoglobulins and monitoring the dynamics of antibody production, fresh infection can be distinguished from latent, inactive infection.

Diagnosis

- In acute toxoplasmosis, high titers of specific IgM and IgA antibodies can be detected in the serum, while IgG levels are initially low.
- > A rise in antibody titer is indicative of an active infection, stable high titer of a recent resolving infection and stable low titer of a chronic, latent infection.

Prevention of toxoplasmosis (human)

Primary prevention - adherence to strict hygiene rules, especially pregnant women and immunosuppressed patients:

- > Wearing gloves when working with soil, processing raw meat
- > Washing fruit and vegetables before consumption
- > Eating only sufficiently cooked meat (71.1 °C);
- > Regular cleaning of cat boxes
- > Preventing insect contamination of food (house fly)
- > Serological examination in the pre-gestation perio

Secondary prevention - the aim is to prevent infection of the fetus or to prevent

- Learning prevention the aim is to prevent infection of the fetus or to prevent the consequences if infection has occurred

 Regular serological screening once every trimester (Expert Guideline of the Ministry of Health of the Slovak Republic on the diagnosis of toxoplasmosis No. 106/2006);
- 100/2006);
 -Women's health education on the possibility of infection and its prevention;
 -Surveillance and monitoring for Toxoplasma gondii especially sheep and goats,
 organic and pasture-raised pigs;
 -Surveillance and monitoring of food and food products (meat, milk and milk
 products, but also water);

Hammondia - Hammondiosis

- > There are two species in domestic animals:
- > H. hammondi in cats (FH), mice, rat (IH)
- H. heydorni in canines (FH), cattle, sheep, goat, dog (IH)
- > Both are obligatory two-host parasites with life cycles similar to Toxoplasma.
- > The generalized life cycle involves the intermediate hosts becoming infected by eating sporulated oocysts.
- > Sporozoites excyst and invade intestinal cells, become tachyzoites and multiply. The parasite eventually encysts in muscles.

Hammondia hammondi

- Hosts:
- Definitive: Cats
- Intermediate: Rodents
- ➤ Life Cycle:
- The unsporulated oocyst leaves the cat in the feces.
- ➤ It takes 3 days in the environment to develop to the infective, sporulated stage (which contains 8 sporozoites).
- When a rodent ingests the infective oocyst, the sporozoites invade intestinal cells and multiply as tachyzoites.



Hammondia hammondi

- > Hammondia hammondi The life cycle and structure are essentially the same as T. gondii with the following exceptions:
- > (1) H. hammondi does not have an extraintestinal cycle in the cat;
- (2) Intermediate hosts are infected only by ingestion of oocysts; (3) Definitive hosts are infected only by ingestion of tissue cysts of intermediate hosts.

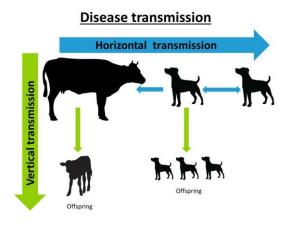


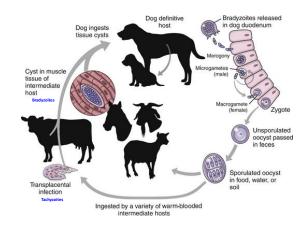
Clinical Findings

- > There is loss of hair and epidermis.
- In addition to the skin lesions, there may be focal, disseminated myositis, keratitis, periostitis, endostitis, lymphadenitis, pneumonia, periorchitis, orchitis, epididymitis, arteritis, and perineuritis.
- > Severely affected animals become emaciated.
- ➤ Common Diagnostic Test:
- > Faecal flotation
- > Treatment:
- None known. Try treating as for *Toxoplasma*: Clindamycin.

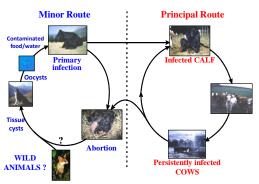
Neospora caninum

- > Toxoplasma like (dog, cattle, sheep)
- ➤ Life cycle similar to *T. gondii* dog is definitive host
- Can differentiate using serology (antibody specific rx to differentiate from T. gondii) morphology of organisms slightly different
- ➤ Route of natural transmission is trans-placental and via ingestion of oocysts infection life long?





Transmission of Bovine Neosporosis







> prenatal infection – young dogs – focal necroses – brain, spinal cord and nerves:

nerves:
> muscular atrophy, ataxia, paresis, ascending paralysis, muscular pain in lumbal pelvic region, torticollis, dyphagia, incontinence;







Clinical signs

- Abortion is the only clinical sign in adult cows (they may abort from 3 moths of gestation to term)
- > Fetuses may die in utero, be absorbed,
- Mummified, autolyzed, stilborn, born alive but diseased, or born clinically normal but chronically infected
- > Within herds, abortion may be clustered, sporadic or epidemic
- > N. caninum induced abortions occur year round
- Cows with N. caninum antibodies (seropositive) are more likely to abort than seronegative cows

Clinical signs

- > Only in individual calves younger than two months
- N. caninum infected calves may be born underweight, unable to rise and with neurologic signs
- Hind limbs and / or forelimbs may be flexed or hyper-extended and neurologic examination reveal ataxia, decreased patellar reflexes, and loss of conscious proprioreception.
- Exophtalmia or an asymmetrical appearance of the eyes may also be observed

Diagnosis

- Clinical findings
- Serology
- > Immunohistochemistry
- ► PCR
- Histopathology
- > Examination of the foetus is necessary for a definitive diagnosis
- Differential diagnosis





Occurrence: cosmopolitan

➤ Affects dairy cows and fattening cattle Europe: UK 1996, Germany 1996, Belgium 1997, Denmark 1997, Italy 1998, Spain 1998, Hungary 1998, Czech Republic 1999; SLOVAKIA: since 2007 in north-eastern Slovakia, average seropositivity up to 39,8 %;

➤ Clinical signs: neurological signs in calves younger than 2 months: ataxia, flexion or hyperextension of limbs, exophtalmia in adults, abortions from 3 months of gestation, in some geographical prevalence up to 45 % in some areas;





Bovine neosporosis Control: management

- > Dogs can produce oocysts, persist in the environment
- > Prevent animal contamination of feed
- Prevent bovine/canine contact with placenta or abortion tissues
- > Embryo transfer

Bovine neosporosis Control: pharmaceuticals

- Sulfonamides, pyrimethamine and clindamycin, toltrazuril: canine neosporosis
- > No drug effective against tissue cysts
- No effective treatment to prevent vertical transmission in cattle
- Potential problem of drug residues in the milk of lactating cows

CARYOSPORA

- > Coccidia of the genus *Caryospora* are found primarily **in birds** and **snakes**.
- > Species of *Caryospora* have two-host life cycles, in which the hosts have a predator-prey relationship.
- > There are owl-mouse and snake-mouse cycles.
- > Caryospora bigenetica and C. simplex

Caryospora

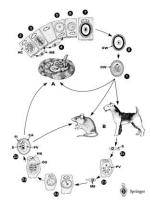
- ➤ Unsporulated oocysts are shed in the faeces of the predatory or definitive host (birds and reptiles).
- Sporulated oocysts contain a single sporocyst with eight sporozoites.
- ➤ Ingestion of sporulated oocysts by the **definitive host** results in *schizogony*, *gamogony*, *fertilization*, and *formation of oocysts* in the intestinal epithelium.

Pathogenesis

- ➤ Formation of pyogranulomes in subcutaneous tissues
- ➤ In immunocompromised hosts fatal

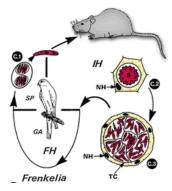
Symptoms

- ➤ Oedema around eyes, nose, inflammation interdigital spaces, abdominal region,
- pyogranulomes;



Life cycle of Caryospora bigenetica

➤The life cycle is nearly identical to that of Sarcocystis spp.



FRENKELIA

- > Coccidia of the genus Frenkelia have an obligatory two-host life cycle involving a rodent intermediate host (prey) and a raptorial bird definitive host (predator).
- $\,\blacktriangleright\,\,$ The life cycle is nearly identical to that of Sarcocystis spp.

- Rodents are infected by ingestion of sporulated oocysts or sporocysts. Schizogony occurs in the rodent liver and tissue cysts are found in CNS.
 Mature tissue cysts can be macroscopic.
 They are multilobulated and surrounded by a thin wall, with many thousands of slender bradyzoites, which are infectious to the definitive book.
- After ingestion, bradyzoites enter the intestinal cells and form gametes, which develop into oocysts after fertilization. Oocysts sporulate in situ and are shed in the faeces.