TOXOPLASMA GONDII

THE ORGANISM/TOXIN

Toxoplasma gondii is an obligate intracellular parasite that occasionally causes serious illness in immunocompromised individuals and unborn babies. It is estimated that one third of all humans have been exposed to *T. gondii*, but most instances of toxoplasmosis are mild or asymptomatic.

Humans can contract foodborne toxoplasmosis from eating raw/undercooked meat containing parasitic cysts or from consuming food contaminated by infected cat faeces.

Cats are the 'definitive hosts' of *T. gondii* and are the only animal in which the parasite replicates sexually. An infected cat can shed between 300,000 and 100 million oocysts in faeces over a 10 to 20 day period (Frenkel, 1990), but will develop immunity to further infection. Oocysts are able to infect humans, other mammals, and some birds, which are all 'intermediate hosts'. *T. gondii* forms cysts in the muscle and neural tissue of these intermediate hosts which generally cause no harm. However, if an infected food animal is eaten without adequate cooking, the parasites can be transmitted to the human consumer and cause infection.

The organism has a life cycle with three infectious stages – sporozoites, tachyzoites and bradyzoites. Cats can become infected after eating any of the three forms. Intermediate hosts are infected by the sporozoite, which is an oocyte excreted by a cat that has 'sporulated' to contain sporozoites. Unsporulated oocysts require only 24 hours outside the host to sporulate and become infectious, and the sporozoite is extremely resilient (Ortega, 2007).

LIFE-CYCLE OF *T. GONDII*

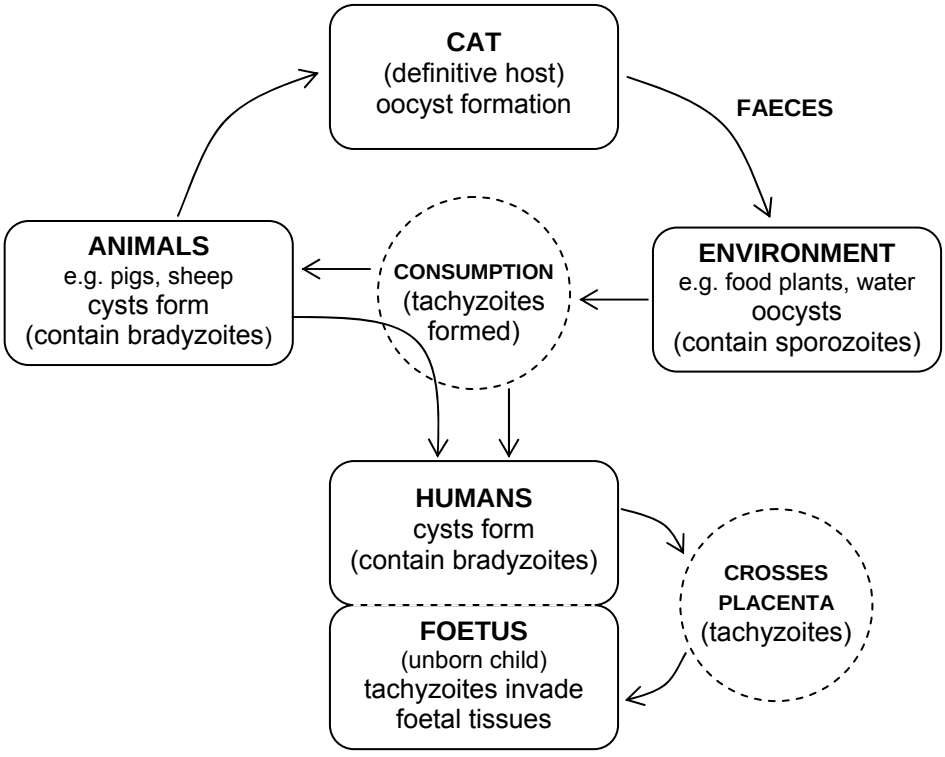

Diagram created using information presented by Ortega, 2007.

GROWTH AND CONTROL

Growth
Does not grow outside of a suitable host.

Survival
Oocysts can survive in the environment.

Temperature
Oocysts survive and remain infective in water and faeces for months at 20°C to 37°C (Dumètre and Dardé, 2003).

Disinfectants
Oocysts are extremely resistant to many detergent and disinfectant solutions, and can remain viable in 2% sulphuric acid, 2.5% potassium dichromate and sodium hypochlorite.

Inactivation
Oocysts and cysts exhibit different characteristics.

***T. gondii* oocysts (sporozoites)**
Temperature
Sporulated oocysts (i.e. those containing sporozoites) are killed by constant freezing at –21^{o}C over 28 days, but not fluctuating temperature between -21°C and room temperature. Unsporulated oocysts are killed within 1 and 7 days of constant freezing at -21°C and -6°C respectively. Under fluctuating temperatures (to room temperature), unsporulated oocysts survive at -6°C, but not at -21°C (Frenkel and Dubey, 1973). Oocysts were inactivated in water when heated to 60°C for 1 minute (Dubey, 1998).

Disinfectants / Sanitisers
Inactivated by:

- 28% ammonia for 10 min
- 7% iodine gas and 5% potassium iodide for 30 min
- 1-10% formaldehyde for 24h
- 5% -10% ammonia for 30 and 10 minutes respectively (Dubey *et al*., 1970).

***T. gondii* cysts (bradyzoites)**
Cysts in meat are inactivated by cooking, freezing, preservatives, irradiation, and pressure.

Temperature
Cooking: Meat temperatures >66°C inactivate cysts (Goldsmid *et al*., 2003). In pork, $D_{67°C}$ = 1 second (Dubey *et al.,* 1990). Microwave cooking may leave residual viable cysts because of uneven heating (Lundén and Uggla, 1992).
Freezing: Cysts lose their infectivity between -6° and -40°C. Kotula *et al.,* (1991) studied commercial freezing effects on cysts in pork. Cysts were rendered non-infective at -9.4°C or colder. Thermal death data predict that cysts should be inactivated instantaneously at -12.4°C or colder.

Preservatives
The following preservatives inactivated cysts in meat:

- Salt and sugar-cured mutton stored at 4°C for 64 hours (Lundén and Uggla (1992).
- 2% salt + ≥ 1.4% potassium/sodium lactate, 8 hours at 4°C in pork loins (Hill *et al.,* 2006).
- 2 to 6% NaCl at 4°C (Dubey, 1997; Hill *et al.,* 2004).

Commercial curing and smoking are thought to inactivate *T. gondii* cysts. However, cured pork consumption was cited as a risk factor in a case-control study in Italy (Buffolano *et al.,* 1996).

Pressure
Cysts in minced pork were inactivated at 300 to 400 Mpa but remained viable at lower pressures (Lindsay *et al.,* 2006).

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CLINICAL PICTURE

Incubation: 3-25 days, mean 11 days.
Symptoms: Vary depending on immunity status. In 80-90% of healthy humans infection is asymptomatic and clinical toxoplasmosis is rare. In the remaining 10-20% cases, a viral-like febrile illness occurs with swollen lymph nodes, a rash, malaise and 'flu' like symptoms. Usually self-limiting (Mead *et al.,* 1999; Smith, 1997).
If infection occurs for the first time in pregnancy, the mother may remain asymptomatic but tachyzoites cross the placenta to the foetus. About 60% of babies born to infected mothers remain uninfected, 26% have subclinical infections, 10% clinical infections and 4% die. Of those infected babies born asymptomatic, about 85% will develop symptoms in later life (Smith, 1997). HIV/AIDS patients can develop toxoplasmic encephalitis.
Condition: Toxoplasmosis.
Dose: Not established, and ethics prohibit human trials. In animal studies of pigs, one sporulated oocyst was infective (Dubey *et al.,* 1996).
At Risk Groups: Immuno-compromised groups and pregnant women. People receiving immuno-suppressive therapy, the very young and the very old are especially at risk. Women who are seropositive for the parasite before pregnancy do not transmit *T. gondii* to the unborn child (Smith, 1997).
Long Term Effects: Where acquired postnatally, infection in healthy humans rarely leads to death. However, in immuno-compromised groups, reactivation from a previous subclinical infection may lead to disease and, possibly, death.
Treatment: No human vaccine is currently available. Usually no treatment is needed for healthy hosts. Spiramycin is commonly used to prevent placental infection. For immuno-compromised patients, depending on immunity status, a combination of pyrimethamine is administered with sulphadiazine and folinic acid (to prevent bone marrow complications). Clindamycin is used for ocular toxoplasmosis.

SOURCES

Human: Except in the case of mother-to-baby transplacental transmission (Montoya and Liesenfeld, 2004), person-to-person spread has not been described. Organ transplant and blood transfusion transmission has been reported (Smith, 1997).
Animal: Cats are the primary hosts, and the only animals known to shed oocysts. Flies, earthworms and cockroaches that have come into contact with infected faeces may harbour and spread oocysts (Dubey, 1996). There is a wide range of intermediate hosts including pigs, sheep and goats, birds, horses, game animals (e.g. deer), mice and rats, marsupials and dogs. Piglets can be killed by toxoplasmosis, but older infected pigs are asymptomatic. Most sheep abortions in New Zealand are caused by *T. gondii*, *Salmonella* Brandenburg or *Campylobacter fetus* infection (MAF Biosecurity, 2010). A live *Toxoplasma* vaccine is commonly used to reduce the risk of abortion in ewes. Disease in cattle is rarely reported (Smith, 1991).
Food: Meat can contain *Toxoplasma* cysts; particularly pork, as well as sheep and goat meat. Beef is rarely contaminated. Unpasteurised goat milk has been implicated in outbreaks (Skinner *et al.,* 1990; Sacks *et al.,* 1982). Although cysts have been found in chickens and pigeons there is no evidence for transmission to humans by these foods.
Environment: Oocysts from cat faeces can contaminate the environment (e.g. soil, sand pits and water supplies) and may be transmitted to humans.
Transmission Routes: There are five recognised modes of transmission to humans (Leroy and Hadjichristodoulou, 2005):
1) ingestion of the oocyst – directly from cat's faeces or indirectly e.g. via unwashed vegetables,
2) consumption of the cyst originating from animal tissue, e.g. from undercooked pork,
3) human organ transplant or blood transmission,
4) accidental inoculation e.g. contaminated needles from work with infected animals, and
5) congenital transmission.

OUTBREAKS AND INCIDENTS

NZ Incidence: The disease was notifiable in New Zealand from 1987 to 1996 and only one case of congenital toxoplasmosis was reported. Between 2000 and 2006, acquired toxoplasmosis (i.e. excluding congenital transmission) cases totalled 84. Congenital transmission during the same period was recorded as the primary diagnosis in 3 fatalities and 7 hospital discharges (Lake *et al*., 2008). No further information publicly available. Studies of seroconversion of various age and community groups suggest that hospital discharge and surveillance records do not reflect the actual incidence of clinical toxoplasmosis in New Zealand.

Overseas outbreaks

Foodborne outbreaks usually involve raw or undercooked meats (Smith,1993).

Pork offal: Korea, two outbreaks of 5 and 3 cases. Offal was uncooked (Choi et al, 1996).

Kangaroo meat: Australia, 12 cases plus one congenital case. The meat was rare (Robson *et al.,* 1995).

Goat's milk: USA and UK, 10 cases. Milk was not pasteurised (Sacks *et al.,* 1982; Skinner *et al.,* 1990).

Municipal water: Canada, 100 cases (congenital infection excluded). Open surface water reservoir, no water filtration and the chloramination treatment (chlorine and ammonia) was insufficient to destroy oocysts. Domestic cats and wild cougars were probably shedding oocysts in the catchment area (Bowie *et al.,* 1997).

Municipal water and commercial ice cream: Brazil, 176 cases. Ice cream made with contaminated water that was unfiltered, not flocculated and inadequately chlorinated. Kittens lived on top of the water tank and the tank roof was in poor condition. Rainfall may have carried oocysts into reservoir water (de Moura *et al.,* 2006).

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