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Aaron Mendel

Effects of Toxoplasmosis on Human Behavior

Abstract:

Toxoplasma gondii is a parasite that relies on the predation of mice by cats to complete its life cycle. In order to facilitate its life cycle, it has evolved in a way that influences the mouse host's behavior that raises predation chance. Besides mice, *T. gondii* can infect many other mammals as secondary hosts, including humans. While *T. gondii* infection in humans remains relatively asymptomatic, there have been instances of disease caused by the parasite. It is possible that, since *T. gondii* can affect the behavioral traits of mice, that it might have similar effects on humans. This review examines evidence of *T. gondii* effects on human personality, and perhaps also human culture as a whole.

Introduction:

Toxoplasma gondii is a complex parasite. As an obligate, coccidian parasite, it has several different phases to its life cycle (Dubey, 2009). Key to this life cycle is the interactions between cats and mice. Cats serve as the primary host, necessary for the bradyzoite phase. These bradyzoites are then shed into the environment to infect a variety of secondary hosts, with mice being a common one. In order to continue the life cycle, the parasite must return to the cat, which requires the secondary host to be eaten, making mice an important secondary host.

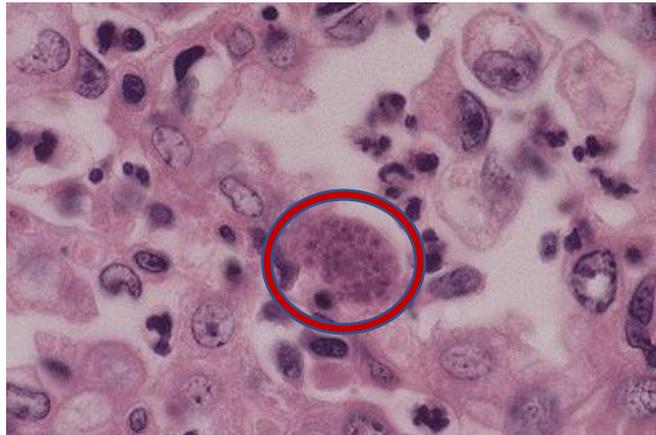
Part of the life cycle requires for the secondary host must be consumed, something the host would actively try to avoid. This would make things more difficult for *T. gondii*. However, in a study done by Justyna et al. (2012), it was found that mice infected by *T. gondii* had altered exploratory behavior and emotional learning. These changes make it easier to be preyed upon by the cat, which benefits the parasite. It would seem that *T. gondii* has adapted to change the behavior of mice in order to aid its transition into cats.

Mice may be the secondary host most commonly used in the life cycle, but many other organisms can serve as one, including humans. And if humans can be infected by a parasite that changes mice behavior, it might also change human behavior as well. J. Flegr and I. Hrdy found in 1994 that people infected with toxoplasmosis had certain personality traits in common,

possibly altered by the parasite. With the possibility of *T. gondii* affecting the personality of individuals, if enough people were infected in a group and pushed towards a certain personality type, it is possible that it could influence culture as a whole.

The Life Cycle:

The life cycle of *Toxoplasmosis gondii* is a complex one, comprised of several stages, as it is a coccidian parasite (Dubey, 2009). Coccidian parasites are spore forming, singular celled, obligate parasites that live in the intestinal walls of animal cells. The stages in the life cycle are marked with incredibly different and distinct genetic expression. In the primary host, cats, the parasite replicates in a sexual manner within the digestive system, forming oocytes that are then shed in their fecal matter. These oocytes survive the external environment by forming sporocysts, which are incredibly resistant, allowing it to contaminate water sources and survive through the digestive system of other organisms. When these sporocysts are taken in by a secondary host, they replicate as tachyzoites.



Tachyzoites replicate asexually, as opposed to in the cat. These tachyzoites are able to cross over and infect the offspring of an infected mother. It is important to note that this vertical transmission only occurs in secondary hosts, and does not occur in cats, making it all the more important for *T. gondii* to find ways of moving between primary and

Figure 1, Eckhaus (1995)
Microscopic slide of a *T. gondii* tissue cyst filled with bradyzoites. When ingested by a cat, the cysts burst and the bradyzoites continue the life cycle

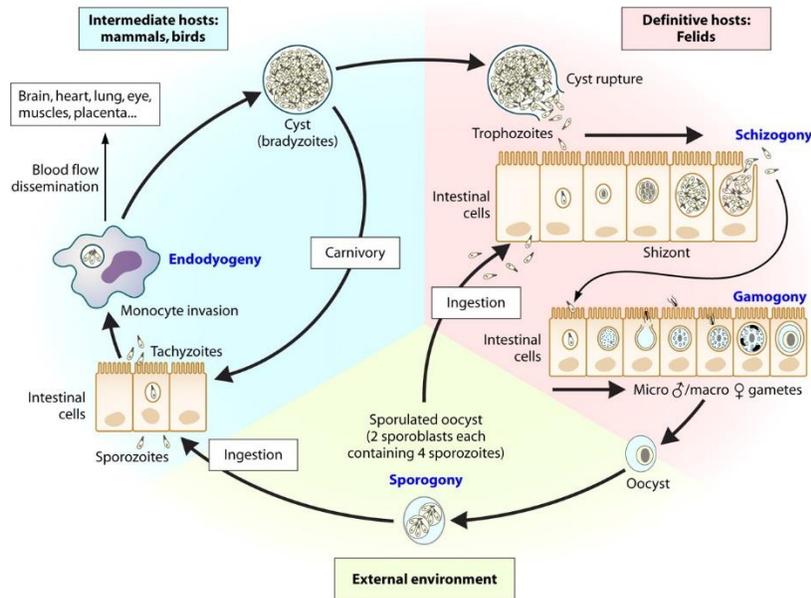


Figure 2, Robert-Gangneux et al, (2012)

The Life Cycle of *T. gondii*. Bradyzoites released into the intestinal walls of obligate host cats reproduce sexually, forming oocysts. Oocysts released into the external environment are taken up by secondary hosts, releasing tachyzoites that reproduce asexually until they form cysts filled with bradyzoites, repeating the cycle

secondary hosts. When this tachyzoite infection becomes chronic, they mass together and form cysts. These cysts contain the bradyzoite phase. When the cysts are ingested by the primary host, cats, again they rupture and release the bradyzoites, which may then begin replicating sexually again and forming more oocytes.

If a cat ingests just one bradyzoite, it can resultantly shed millions of oocytes, though the oocysts themselves are not pathogenic to cats should they end up ingesting any (Dubey, 2009). While cats can shed oocysts after ingesting any phase of the parasite, it occurs far more frequently when it was the bradyzoite ingested. This shows that it is preferential

for the bradyzoite phase to enter the cat in order for reproduction to occur. The other phases are similarly specific. While having a secondary host ingest a sporocyst continues the cycle, having a secondary host ingest a bradyzoite will not lead to infection (Dubey, 2009).

Behavioral effects of Toxoplasmosis Infection in Rodents:

An important part of the life cycle of *T. gondii* is that once it enters the rodent, it must then re-enter the cat. This is usually done when a cat eats the flesh of a rodent under chronic infection, thus ingesting the bradyzoite cysts. The problem with this is that rodents will naturally try to avoid being eaten. For *T. gondii* to require its host to do something so counter to its own existence would be selectively disadvantageous, as it would rely on chance that a cat is not only able to catch a mouse, but also for it to be an infected one. If *T. gondii* would keep this dual host life cycle, it would also be selectively advantageous to make the predation of infected mice more likely.

The hippocampus and amygdala are important parts of the brain in regards to predator invasion behavior. They are involved in conditioned fear and unconditioned anxiety, which guide the rodents to take defensive behaviors around cats, which would hinder the reproductive cycle of the *T gondii*. But Gatkowska et al. (2011) found that the parasite has adapted, and they tend to form cysts in these two areas of the brain. By invading these regions of the brain, it is possible that the parasite could hinder the defensive behavior of their rodent host, allowing easier predation by a feline host, and thus perpetuating the life cycle of *T gondii*. It would seem that this is indeed the case, as infected mice have been shown to have a reduced capacity to learn, and are much more active and mobile than the non-infected controls (Webster, 2007). The impaired learning results in a rodent failing to learn to be afraid of cats, which can result in them instead cozying up to cats. The increased movement is also important, since cats are then more likely to notice the infected rodents. The reduction of defensive behaviors and increase in predation behaviors makes the infected mice more likely to be preyed upon, as well as easier to be preyed upon than non-infected mice, making it selectively advantageous for cats to eat the infected mice. The *T gondii* has adapted itself to alter the behavior of their secondary host to make ingestion by a primary host more likely. Skalova et. al (2006) thought that the mechanism for this might be through altered dopamine levels in the brains of the mice. Other researchers looked to see if similar mechanisms might be occurring in infected humans as well.

Toxoplasmosis Infection of Humans:

While the effects of *T. gondii* on rodents and its effects on their behavior are well known, it does not answer how it affects humans. Firstly, how does *T. gondii* infect humans? *T. gondii* is a zoonotic disease, able to be transferred between humans and animals, and as it turns out, any 3 phases of the life cycle can infect a human, but they cannot do so the same way. Astrid et al. (2000) found that Tachyzoites are incredibly susceptible to the external environment, so it is hard to transmit infection horizontally at this stage. However, they also noted it can be transmitted vertically in this stage, from parent to offspring during birth. This is where the main concern of *T. gondii* is found pathologically, as this vertical transmission can cause infant mortality, such as the case given in Dubey (2009). It can also be found in the milk of infected individuals, but due to their susceptibility to the external environment, infection through this vector is rare (Tenter et

al. 2000). Furthermore, standard pasteurization procedures are more than enough to kill off any Tachyzoites in the milk.

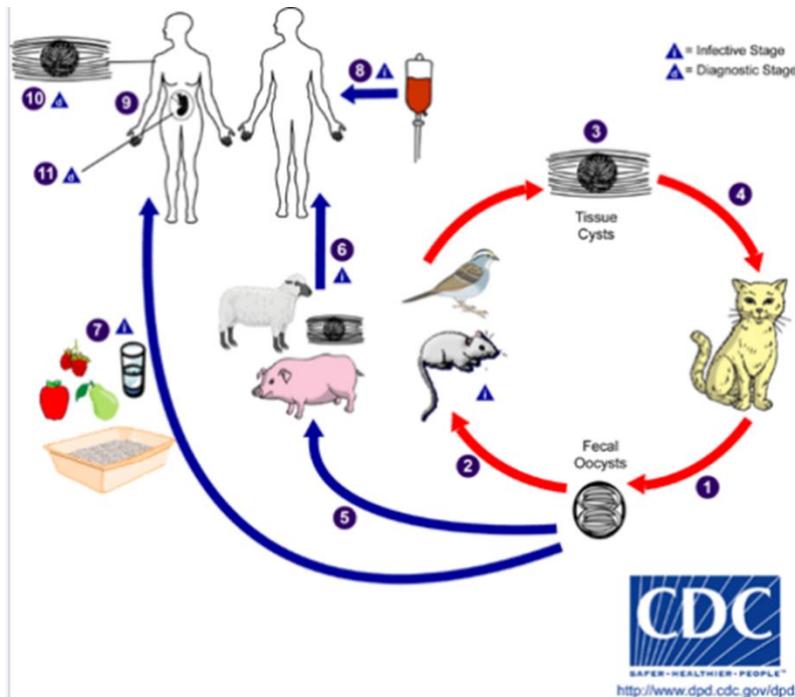


Figure 3, CDC

Paths of Infection. From the obligate host cats, sporocysts are released, infecting secondary hosts. Rodents serve as the main secondary host for the life cycle, but any mammal can serve as one. This can infect a human directly through contact with the sporocysts, but if a different animal serves as a secondary host, eating it can also infect a human. *T. gondii* can also infect a human congenitally if the mother is infected.

The bradyzoite phase is more resistant, able to survive external environments to an extent, and able to survive the digestive enzymes. This makes ingestion a viable vector for infection at this stage. This is the main way it infects cats during its normal life cycle, but can enter various other carnivorous secondary hosts if infected meat is ingested. *T. gondii* does not grow the same in every secondary host, some are more likely to infect humans than others, like pork, where the parasite can form infective cysts in the parts that we eat (Tenter et al, 2000). So eating infected pork can possibly lead infection of the Bradyzoites are not killed off first. While the Bradyzoite phase is more resistant to the external environment, it is not completely immune. It is better at surviving colder temperatures than it is hot ones, so while refrigeration

will not kill the *T. gondii* of infected meat, properly cooking it will (Tenter et al, 2000). Thus, eating undercooked meat dramatically raises one's chances of *T. gondii* infection.

The oocyst phase, which is shed from the cat itself, is the most resistant stage of the *T. gondii* life cycle. These sporocysts are able to survive harsher external environments, allowing them to cause infection in a few different ways. They start off in the fecal matter of cats, and should it manage to contaminate a water supply, any that drink from it have a risk of ingesting

the sporocysts and developing an infection. This can happen often on farms with cats, where the cat contaminates the livestock's water source, causing many of them become intermediate hosts, which can then be eaten and transfer the parasite to humans. *T. gondii* can also transfer to humans by contact with an infected cats fecal matter, such as cleaning out a litter box. This can easily cause an infection, especially if you don't wash your hands right after, as some of the oocysts can get on the skin, and then accidentally get ingested later while eating or touching your face. The fortunate thing about the oocysts is that they do not sporulate right away, so regularly cleaning out the litter box every day and taking the garbage out can be an effective means of preventing infection (Tenter et al, 2000). This removes the potential for contact with sporulated oocysts, which is when they truly become infective, by removing the oocysts from potential contact before they get the chance to sporulate.

Effects of Toxoplasmosis Infection in Humans:

Once the *T. gondii* infects a human, what are the effects of this infection? For the majority of immunocompetent people, it is relatively asymptomatic, and most will not even realize they are infected. However, it can become symptomatic in some immunocompetent individuals and show symptoms. This can occur in people who develop AIDS or are taking cytotoxic drugs which reduce lymphocyte count, allowing a previously asymptomatic infection to develop further (Weiss & Dubey, 2009). This is because immunocompromised individuals are far more susceptible to infection, and are more likely to be symptomatic. The tachyzoite phase of *T. gondii* can also pass congenitally from an infected mother to her child during birth. Since the infant's immune system is not developed, they are likely to develop symptoms. The more common symptoms brought on by significant infection include chorioretinitis, lymphadenitis, myocarditis, and polymyositis. (Weiss & Dubey, 2009). Infected individuals are also at an increased risk of developing brain cancer, likely related to the inflammation in the brain (Mozaffarian et al, 2014). In the case of infants infected congenitally, they will develop a recurrent infection of toxoplasmosis, resulting in encephalitis and chorioretinitis (Weiss & Dubey, 2009). These will usually lead to the infant's death. *T. gondii* can also affect several different neuronal pathways. This could lead to potential symptoms that can affect behavior, similar to how it affects mice.

After a symptomatic episode, *T. gondii* can return to an inactive, asymptomatic state. However, while it may lay dormant, it can still reactivate and cause infection again. Carruthers & Suzuki found in 2007 that an outbreak of reactivated *T. gondii* has been linked to many cases of first-onset schizophrenia (Carruthers & Suzuki, 2007). These authors found elevated anti *T. gondii* IgG antibodies within patients with first-onset schizophrenia, indicating a *T. gondii* infection. They also discovered that while IgG antibodies were present, IgM *T. gondii* antibodies were not. This is significant because IgM antibodies are part of the innate immune response, made when a person is first infected with something new. IgG, on the other hand, is part of the adaptive immune response, and are found when dealing with something that has infected the individual before. Having IgG but not IgM indicates that the schizophrenia found in these individuals did not coincide with a recent infection, but one they likely got congenitally at birth from their mother. Carruthers & Suzuki also noted that congenitally infected patients develop toxoplasmic chorioretinitis at around 20-30 years of age. They speculated that this condition was perhaps caused by reactivated *T. gondii*. Interestingly, this time window coincides with the typical age of schizophrenia of onset. Carruthers & Suzuki proposed that congenital *T. gondii* infection may be part of the etiology for schizophrenia onset.

Evidence for another possible relationship between the effect of toxoplasmosis on human behavior was provided by Flegr & Hrdy (1994). They tested if *T. gondii* had an effect on personality traits, hypothesizing that this might indicate that *T. gondii* was shifting these people's personalities, and thus behavior, in that direction. What they found was that there were several personality traits that were significantly different between infected and non-infected individuals. Some of these differences were gender specific, much like in mice. While many differences were gender specific, there was one change that was consistent across all infected participants, both male and female. The infected participants had higher scores for apprehension factors, as well as the personality trait for neuroticism. People infected with *T. gondii* appear to have a set change in personality, increased neuroticism.

The Role of Dopamine:

The correlation between *T. gondii* and schizophrenia is an interesting one. Understanding how *T. gondii* causes schizophrenia could show how it might also be affecting behavior in general. To understand how *T. gondii* causes schizophrenia, it is best first to

understand how schizophrenia might occur in the first place. Creese et al. in 1976 found that butyrophenones worked as an effective method for treating schizophrenic symptoms. Butyrophenones work as a dopamine inhibitor, indicating that schizophrenia may be caused by an excess of dopamine if inhibiting dopamine helps to treat those symptoms.

In a study by Skalova et al. in 2006, mice were injected with a selective dopamine re-uptake inhibitor, GBR. The result of this is the inability to clear out dopamine released, effectively increasing dopamine release. The researchers found that injecting mice with GBR produced similar effects to *T. gondii* infection, imitating the same reduction in anxiety factors. They also found that GBR affects mice differently depending on gender, just like *T. gondii* does. What makes this significant is that GBR is a dopamine re-uptake inhibitor. If GBR and *T. gondii* have similar effects, it could be that *T. gondii* is also acting as a dopamine reuptake inhibitor. This would explain why *T. gondii* can cause schizophrenia, *T. gondii* is preventing dopamine reuptake, causing an increase in synaptic dopamine.

Another aspect of dopamine that *T. gondii* might affect is the protein Interleukin-2. Pettito et al. (1996) injected Interleukin-2 into mice. Doing this caused mice to show increases in locomotion, one of the main symptoms of *T. gondii* infection that helps cats notice them and more easily prey on them.

Interleukin-2, besides just being a protective cytokine part of the immune response, Pettito et al. found that it also acts as a modulator in the dopamine release. *T. gondii* affecting IL-2 would be another way to cause the excessive dopamine

levels that cause schizophrenia. Mozaffarian et al. (2014) states that IL-2 is found in elevated

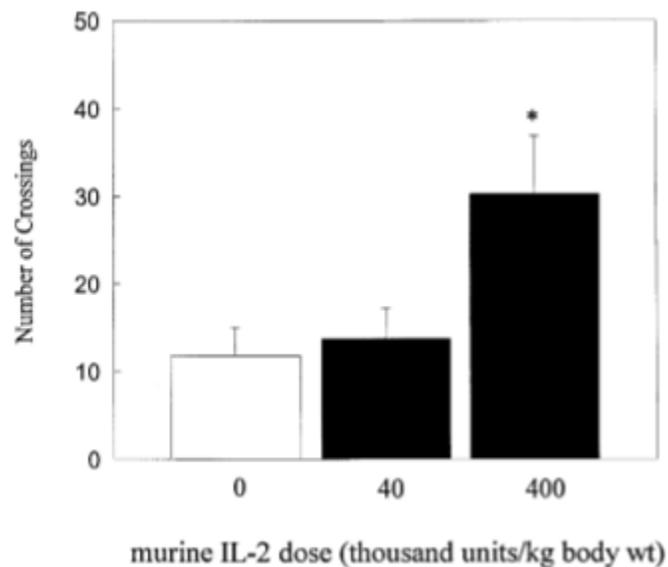


Figure 4, Pettito et al. (1997)

Increased levels of IL-2 correlate with increased locomotion in mice. This is a similar symptom to *T. gondii* infection

levels in people infected with *T. gondii* as part of the immune response, and particularly in those suffering from schizophrenia as well as infection.

Webster (2007) found that There were significant differences in dopamine levels between *T. gondii* infected mice and non-infected mice. This gives credence to dopamine being involved *T. gondii* symptoms. Webster also found that Haloperidol, a dopamine antagonist, could be used to treat *T. gondii* symptoms, further indicating that dopamine is mode for causing symptoms. Another study done by Skallova et al. (2006) also found that mice infected with *T. gondii* had 114% more dopamine in their brains. Through their experiments, they proposed that *T. gondii* increased dopamine in activity in the limbic system, but also decreased dopaminergic activity in the pre-frontal cortex. This could mean that while dopamine in general being increased causes *T. gondii* symptoms, where and how it is affecting dopamine also plays an important role.

Gatkowska et al. (2011) found that in rodents, *T. gondii* tends to infect the amygdala and hippocampus. It is hard to study if it infects the same places in humans, but if it did, affecting dopamine output in these areas, which take part in emotional learning, could influence behavior, much like it does in rodents. One effect found in humans by Skallova et al. (2006) was that men infected with *T. gondii* consistently scored lower on measures of novelty seeking, a measure thought to be negatively correlated with basal dopaminergic activity. The researchers note that while this link between novelty seeking and dopamine has been shown in mice, the dopamine hypothesis for novelty seeking in humans still needs to be confirmed. While excessive dopamine levels may cause schizophrenia, a milder difference might cause less severe symptoms, such as the behavioral changes seen in those infected by *T. gondii*. How *T. gondii* causes this rise of dopamine is still unclear, but it is likely the cytokine interleukin 2 is involved.

Toxoplasmosis' Link to Culture:

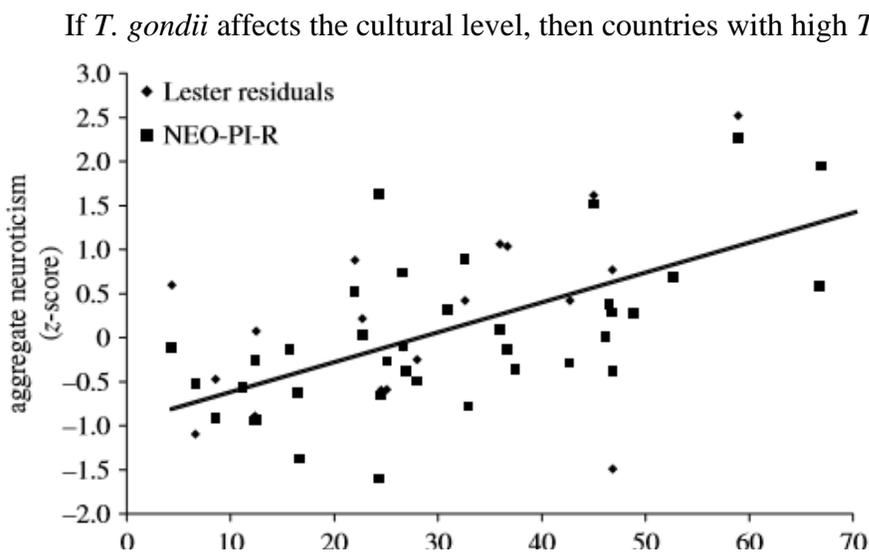
Stepwise Regression Results of Mean NEO-PI-R Factor Scores Against the Four Culture Scores Across 33 Countries

Criterion	Predictors	Cumulative Adj. R ²
Neuroticism	+ Uncertainty avoidance	0.31
	+ Masculinity	0.55
Extraversion	+ Individualism	0.39
	- Masculinity	0.46
Openness to experience	+ Masculinity	0.13
	- Power distance	0.29
	+ Uncertainty avoidance	0.36
Agreeableness	- Uncertainty avoidance	0.28
Conscientiousness	+ Power distance	0.24

Figure 5, Hofstede & McCrae, 2004
Cultural predictors of certain personality types. This shows a moderate correlation of uncertainty avoidance with neuroticism, and a strong correlation between masculinity and neuroticism

if many people had a similar personality shift? The personality trait that *T. gondii* acts on universally is neuroticism (Flegr & Hrdy, 1994). The thing about this particular personality trait is that it is a strong predictor

for several cultural factors. Hofstede & McCrae (2004) found that Neuroticism is positively correlated to traits cultural masculinity and uncertainty avoidance. They defined Masculine cultures as being focused on things like ego, money, and work, and for being more individualistic. They defined Cultures high in uncertainty avoidance as being associated with the expression of emotion, anxiety, as well as stress. Many of the countries Lafferty (2006) found having these traits were western cultures.



would be associated with masculine and uncertainty avoidant cultures. This was indeed the case, with western nations that had high *T. gondii* prevalence reporting more cases of neurotic personality

It has already been shown that *T. gondii* can affect a person's personality individually. Flegr in 2007 found that much like in mice, both motor function and behavior were affected in humans. But what would happen on a cultural level

Figure 6, Lafferty, 2006. Prevalence of *T. gondii* infection plotted against presence of the neurotic personality type. This graph shows a positive correlation between the two factors, indicating the possible link between *T. gondii* and human behavior.

traits, particularly both masculine roles and uncertainty avoidance (Lafferty, 2006). It would seem that *T. gondii* influences the kind of personality and culture found in western society, pointing to the possibility that these cultures were influenced by the parasite.

Conclusion:

Toxoplasmosis gondii is a complex organism. It is an obligate parasite, with several phases in the life cycle that require it to jump between primary and secondary hosts. The primary host, required for sexual reproduction, are cats. Cats shed oocysts into the environment, from which secondary hosts can be infected. Though *T. gondii* is able to infect most mammals, mice are the most common secondary hosts. When the infected mice are preyed by a cat, the *T. gondii* infects the cat, starting the life cycle again. Requiring the secondary host to be preyed upon leaves a lot to chance, and seems to be selectively disadvantageous for an organism. *T. gondii* appears to have gotten around this by evolving “to manipulate” the behavior of its mouse host. Webster in 2007 found that infected mice had reduced capacity to learn and the exhibited the increasing activity and mobility. Pertinent to the possible mechanisms of these behavioral changes, Gatkowska et al. (2011) found that *T. gondii* forms cysts in the murine hippocampus and amygdala, areas that influence these behaviors. It seems that the neurotransmitter dopamine is involved in the mechanism by which the parasite modifies behavior of its secondary host. Consistent with this notion, Skallova et al. (2006) found that injections the dopamine reuptake inhibitor GBR induced behavioral changes similar to those seen in mice infected with *T. gondii*. Furthermore, Petitto et al. (1996) found that increased levels of interleukin 2, a modulator for dopamine release, also produced behaviors similar to those in *T. gondii* infected mice.

While mice are the required secondary *T. gondii* host, this parasite is able to infect other vertebrates, which end up being dead end hosts. Humans are one of the dead-end hosts, and there are many ways they can become infected. Cleaning the fecal matter of infected cats puts people in direct contact with shed oocysts, causing infection. Also, much like when a cat is infected by preying on an infected secondary host, so too can humans. Since possible secondary hosts are cattle and pigs, consuming the meat of infected individuals carries the risk of ingesting infectious cysts. The parasite can also cross over from an infected mother to her child during birth, causing

a congenital infection. Congenital infection can cause many health problems, such as chorioretinitis and myocarditis (Weiss & Dubey, 2009). Important to the main subject of this review Carruthers & Suzuki (2007) also found that congenital *T. gondii* contributed to schizophrenia onset. Interestingly, analogous to the putative mechanisms of *T. gondii*-induced behavioral changes in mice, the schizophrenia-related pathology is also associated with dopamine abnormalities.

Besides representing a risk factor for the schizophrenia, the *T. gondii* is known to affect the human behavior more subtly. Specifically, Flegr & Hrdy (1994) found consistent changes in personality between individuals infected by *T. gondii*, and those that were not. One of the more noticeable changes they found was that infected individuals scored higher for markers of neuroticism. This finding is highly intriguing because of its possible implications for human culture. Pertinent to this point, Hofstede & McCrae (2004) found that the neuroticism correlates with cultural indicators for masculine roles and with uncertainty avoidance. Based on these findings, one could speculate that a widespread *T. gondii* infection could influence personalities and the behavior of a large group of people, pushing them toward creating certain types of cultures. It is possible that through *T. gondii* infection, human civilization was influenced by cats.

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