Do microorganisms control our behaviour?

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Abstract

Introduction Can microorganisms infect particular parts of the brain and do they control our behaviour? *Toxoplasma gondii* can only reproduce sexually in cats' intestines. Asexual reproduction can take place in humans, rodents, cats, and birds. In humans the brain and muscle cells are mostly infected. Chronic infection is thought to cause behavioural changes. Rats with the *T. gondii* infection are way more active, more novelty seeking, and have higher interest in cat-smelling areas than the control group of non-infected rats. One may conclude that *T. gondii* does influence the behaviour of their host. According to the researchers this may be because of the need of the parasite to be transmitted to the cat's intestines to fulfil their life cycle. The success of the parasite through manipulation by the parasite is called the behavioural manipulation hypothesis. Data shows that *T. gondii* is located primary in the amygdala. This is remarkably, because it indicates that the parasite can determine very specifically which part of the brain it will infect. This specificity indicates that the behavioural manipulation hypothesis is plausible. Does the parasite have similar effects on human behaviour? One way of investigating the human behaviour is via personality questionnaires. Infected persons showed higher anxiousness, and a decrease in novelty seeking. Infected men showed lower rule consciousness, higher cautiousness, jealousy, suspiciousness, and expedience. Women who are infected showed higher warmth and, unlike men, higher rule consciousness. Humans infected with *T. gondii* rated the pleasantness of cat urine different than non-infected humans, there is a gender-specific difference. A different way of investigating human behaviour is via behavioural testing. Men showed lower self-control and clothes tidiness, lower relationships/warmth, and higher mistrust in rural environment. Women showed a trend for higher self-control and clothes tidiness. After infection people have a slower reaction time and a lower ability to concentrate, which is problematic in daily life, since these things result in a higher change of (traffic) accidents. There is also a correlation with psychiatric disorders, which have dopamine dysfunction in common. So there might be a relation between toxoplasmosis and the dopamine pathway. **Discussion** There is a big chance that microorganisms do affect our behaviour. In rats the results are more convincing than in human beings. General medical investigations can be a good way investigating behavioural changes. Since mechanisms are unknown, medication can be antibiotics or dopamine inhibitors. When researchers investigate other microorganisms more, they may achieve more insight in the problems surrounding the question if and how microorganisms affect our behaviour.

Introduction

Robert Sapolsky wrote an article named “Bugs in the Brain”. He wrote about microorganisms and the infecting of human’s and other animal’s brain. He hypothesized that microorganisms may know more about the brain than humans do, because microorganisms can infect parts of the brain which are specifically important to what these microorganisms want to achieve. [1] This essay is about whether microorganisms can infect particular parts of the brain and whether they can control our behaviour this way.

Annually there are about four hundred to four thousand new cases of toxoplasmosis (the disease caused by *Toxoplasma gondii*) in the United States. [2] However, about eleven percent of the Americans are infected with the parasite, but the people who are not ill have a sufficient immune system which can clear the infection. [3] Recently researchers have found that microorganisms such as *Toxoplasma gondii* infect the brain of humans and animals, and after infection their behaviour is changed. [4] That is how the question is raised; Do microorganisms control our behaviour?

Symptoms

In table 1 and table 2 one can see respectively the independent variables and the behavioural variables for *Toxoplasma gondii* infection. In general infection with *Toxoplasma gondii* can happen due to the eating of infected meat, by accidental ingestion of the oocysts or in utero infection. [5] Humans infected with *Toxoplasma gondii* oocysts have enlarged lymph nodes, fever, listlessness, and sometimes inflammation of the heart when the disease is in its active phase. Pregnant women who are infected via the uterus where the infection can get to the foetus have a big chance of abortion. [6] This happens early in pregnancy; later in pregnancy the infant can survive the infection, but there may occur other things, like serious or fatal congenital defects, such as hydrocephalus (“water in the brain”), blindness, and mental retardation. [7] Also here it applies that not all infected people do have to get the symptoms. Diagnosis of this infection can be made through the testing of serological blood samples. [8]

Variable	Cases (n = 64)	Controls (n = 128)	OR (95% CI)	p
Age (years)				
Mean ± SD	35.9 ± 9.5	35.8 ± 7.7	1.0 (0.9-1.0)	0.902
Gender				
Male	37	78	0.9 (0.5-1.6)	0.677
Female	27	50		
Previous cerebral toxoplasmosis				
Yes	11	16	1.5 (0.6-3.4)	0.380
No	53	112		
CD4 T-cell count (cells/μL)				
Mean ± SD	71.4 ± 72.9	61.5 ± 0.6	1.0 (0.9-1.0)	0.284
Use of prophylaxis at admission				
Yes	3	23	0.2 (0.1-0.8)	0.019
No	61	105		
IgG anti-*T. gondii*				
≥ 1:16	58	72	7.5 (3.0-18.7)	< 0.001
< 1:16	6	56		
IgG anti-*T. gondii*				
≥ 1:1024	52	41	9.2 (4.4-19.1)	< 0.001
< 1:1024	12	87		
T. gondii PCR in blood				
Positive	51	3	163 (45-597)	< 0.001
Negative	13	125		

Table 1.
Univariate analysis for the identification of independent variables associated with the diagnosis of cerebral toxoplasmosis [9]

Behavior	Yes		No	
	No.	%	No.	%
Handled cats	128	73.6	46	26.4
Cleaned cat litter boxes	24	13.8	150	86.2
Handled raw meat	160	92.0	14	8.0
Gardening	112	64.4	62	35.6

	Always/Daily		Sometimes		Never	
	No.	%	No.	%	No.	%
Ate food inside pig facilities	18	10.3	52	29.9	104	59.8
Washed hands after leaving pig facilities	101	58.0	38	21.8	35	20.1
Washed hands after handling raw meat	117	67.2	26	14.9	31	17.8
Washed hands before eating	90	51.7	84	48.3	0	0.0
Handled pig feed	56	32.2	96	55.2	22	12.6

Table 2
Descriptive statistics on behavioural risk factors for Toxoplasma gondii infection in Illinois swine farmers [10]

Toxoplasmosis

Toxoplasma gondii is a parasite which can be present in humans, rodents and a variety of other animals. [11] One way of infection of *T. gondii* in humans is due to consumption of raw meat. [10] Infection with this parasite does not always lead to disease. Many humans and other animals are able to induce a normal immunological response. Infection during pregnancy is more dangerous, because *Toxoplasma gondii* can infect the foetus or it can induce abortion. Individuals who have an impaired immune system often cannot survive the infection. [14]

Toxoplasma gondii can reproduce sexually and asexually (see *Figure 1*). It can only reproduce sexually in cats' intestines, where it can be excreted with the faeces or urine. [12] Asexual reproduction can take place in humans, rodents, cats, and birds. [11] Infection of a cell happens through the formation of a vacuole by the parasite. In this vacuole *Toxoplasma gondii* forms bradyzoites (slow-growing form of *T. gondii*) which form cysts in the muscles and the brain of the host. This way the parasite cannot be attacked by the immune system, since the immune system does not recognize cysts as foreign matter. *T. gondii* can replicate itself in the vacuoles until the cell is filled with the parasite. When it is entirely filled, the cell can burst and this way tachyzoites (moving form of *T. gondii* which is asexually reproduced) are released. These tachyzoites can be attacked by the immune system, but since they often are not cleared entirely, they can infect other cells and produce bradyzoites. This shows that it is a very infectious disease which is hard to get rid of. [6]

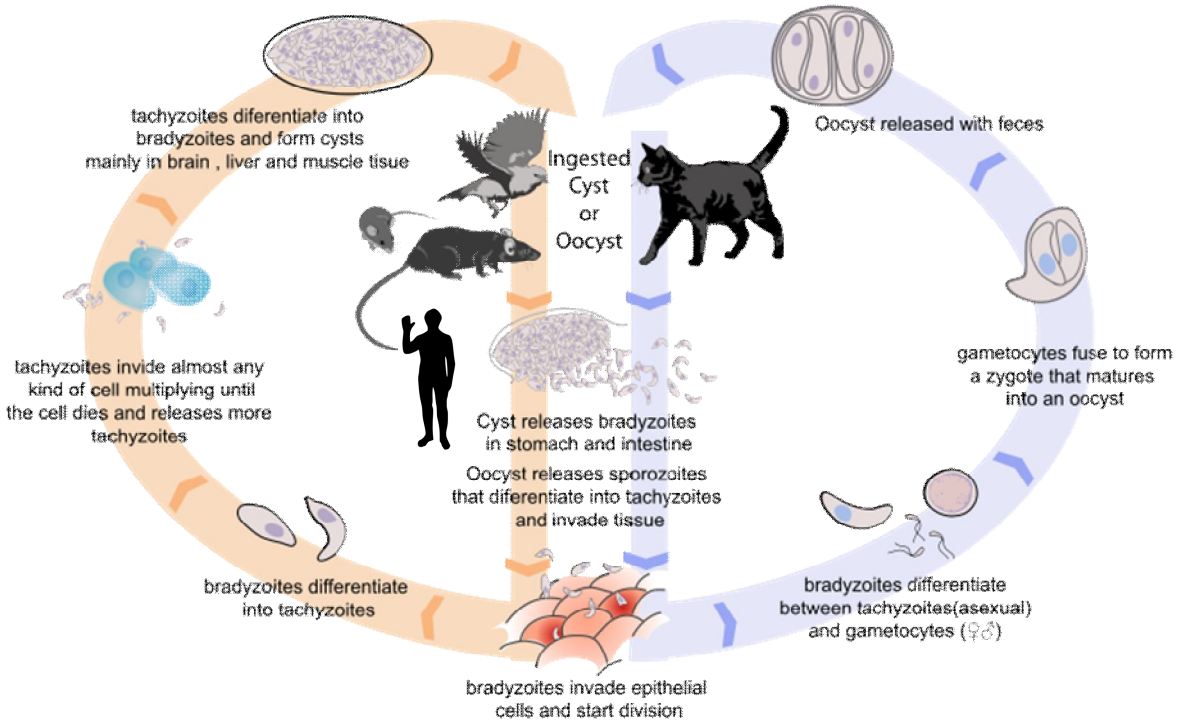

Figure 1.
Life cycle Toxoplasma gondii *[15]*

In humans the bradyzoite cysts are mostly in the brain and muscle cells. [6] That is why there is mainly neurological and/or ocular damage after an acute infection. Chronic infection is thought to cause behavioural changes. [13] This chronic infection stays infective when the immunity of the host is not high enough compared to the infectivity of the parasite. There are also a lot of cases were the infection in humans is thought to be latent. In this case the infection is asymptotic. However, in combination with AIDS, which weakens the immune

system, the pathogen is able to make the bradyzoites and thus the cysts as described above. The latent infection seems to be not that innocent as first has been thought, since the presence of neurological changes is in consistence with the appearance of the *T. gondii* infection. In animal models behavioural changes occur after infection. There has also been some research done which investigates the relationship between *T. gondii* infection and neurological disorders as for example schizophrenia. [16]

Animal model

To investigate whether behavioural changes occur after a human Toxoplasmosis infection an animal model is needed. Since rodents have to deal with the infection in nature itself, they are a suitable animal model. Behaviour can be observed quite well in these animals. Mice and rats are most suited, since there is a lot known about rats and mice and their behaviour and metabolism.

Experiments influenced behaviour

Researchers have found that rats with the *T. gondii* infection are way more active than the control group with non-infected rats. [17] To investigate this, they used brown rats, *Rattus norvegicus*, to look at the relationship between parasite load and host activity. They formed the following hypothesis: Parasites with indirect life cycles will increase the activity of the host so that the chance that the host will be predated is increased and the parasite can reproduce sexually. This means they expected infected rats to have a higher activity level than non-infected rats. They compared rats infected with microorganisms characterized by a direct life cycle with rats infected with closely related microorganisms with indirect life cycle and they used a non-infected control group. They looked at night activity. They found that *T. gondii* was the only parasite they used which required predation to get to their definitive host to produce sexually and that it was the only parasite which was associated with higher activity levels in infected rats compared to uninfected rats. One may conclude that *T. gondii* does influence the behaviour of their host. According to the researchers this may be because of the need of the parasite to be transmitted to the cat's intestines to fulfil their life cycle. [17]

Other research showed that infected rats are more novelty seeking when a novel object is presented compared to uninfected rats. [13] In figure 2 the differences in curiosity between infected and non-infected rats are shown. Only the curiosity for places which smell like cat differs between these groups; here infected rats have a higher interest in the cat-smelling areas than non-infected rats.

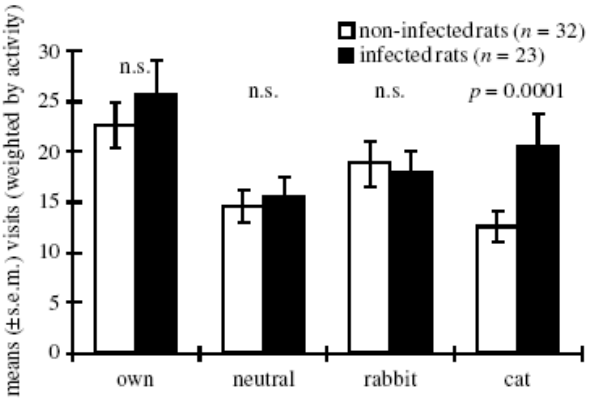

Figure 2.
Mean number of visits to the four scented areas in the outdoor pens over one night. [13]

A part of this experimental group also shows a potentially sexual attraction to areas which smell like cats. [13,18]

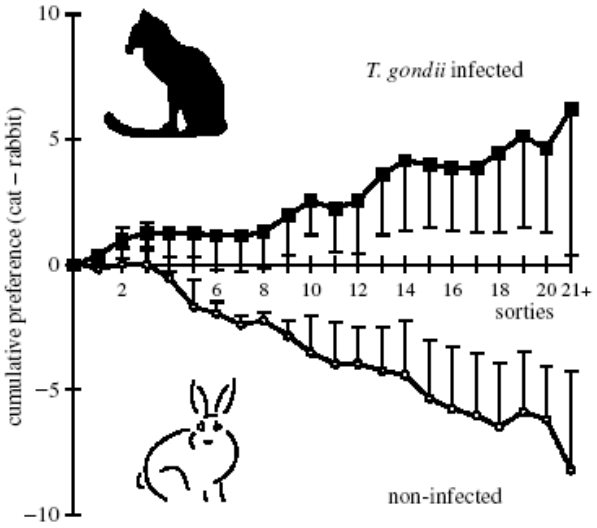

Figure 3.
Development of preference or avoidance throughout the night of infected and non-infected rats. [13]

In figure 3 there is another result shown from the same experiment. The researchers counted the amount of visits to areas with a cat smell and the amount of visits to areas with a rabbit smell. They found that there are significant differences between the rats with and without *Toxoplasma gondii* infection, where infected rats are more attracted to areas which smell like cat, and non-infected rats are more attracted to areas which smell like rabbit (non-cat).

Different researches investigating the same thing showed similar results. In figure 4 and figure 5 one can see that also in these studies infected rats are more attracted to cat odour than uninfected rats.

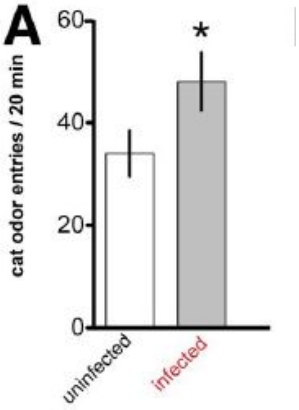

Figure 4.
Toxoplasma *infected rats spent more time exploring cat urine than uninfected rats. [18]*

Also these researchers believe that manipulation of the behaviour of the host is due to the parasite because of the received advantage of the parasite. The success of the parasite through manipulation by the parasite is called the behavioural manipulation hypothesis. [19,20]

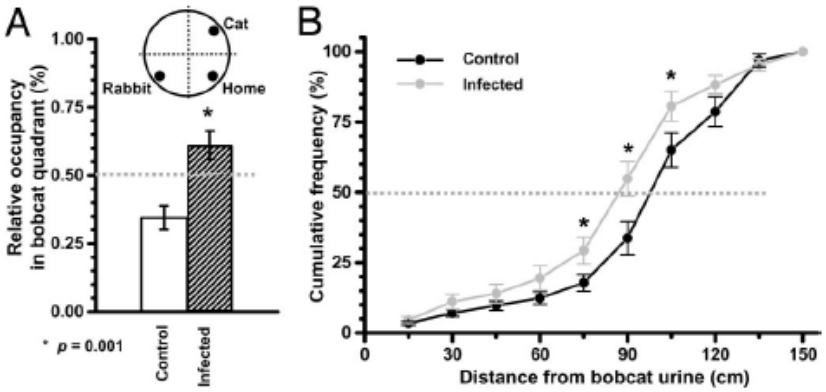

Figure 5.
Attraction/aversion to different scents in rats. (A) Travelling/ being in different quadrants containing rabbit, cat, human, or neutral smell. (B) Infected rats spent more time near the bobcat smell compared to control rats. [21]

More than one behavioural change has been noticed. Infected individuals seem to have changes in learned fear, innate fear, and anxiety. Combinations of these changes in behaviour occur a lot, so there might be a nonspecific pathway leading to this changed behaviour. [21] This might be due to findings of a higher density of *T. gondii* cysts in the amygdala compared to other brain areas such as the hippocampus, prefrontal cortex, etc. In fact, the concentration of cysts is almost doubled in the amygdala. [21] This is in consistence with the changes in fear behaviour, since the amygdala is the most important area of the brain where processes of memory and emotions take place. Fear is one of those emotional reactions regulated in the amygdala.
This may explain the attraction to cat odour. The behavioural manipulation hypothesis ventilates that the reversal of innate fear to cat odour is due to the capacity of the parasite, whereas it can leave other domains unchanged. [22] The mechanism in the brain causing the behavioural changes is not known yet.

When you search for *Toxoplasma gondii* there has a lot of research been done investigating infected rats and their preference for cat odour. As said before, *T. gondii* changes the innate behaviour of rats when they get in touch with cat urine; in stead of their innate response to avoid this smell, infected rats are attracted to the pheromone which is present in cat urine. This way the chance of getting predated by a cat is increased, which is in advantage for *T. gondii*, since it can reproduce sexually in the cat's intestines. This is called adaptive, behavioural manipulation. This means that a parasite will specifically manipulate the behaviour of the host which is essential for increasing their own transmission. In the case of *Toxoplasma gondii* there are multiple neural circuits damaged, implicating innate fear, learned fear and anxiety. This leads to the thought that *T. gondii* damages these neural circuits non-specifically. These hypotheses are somewhat contradictory, because where first has been thought that the parasite is very specific on which brain area it will infect, the second one says that *T. gondii* is not that specific as you might think.

These hypotheses are investigated in the following research project. [21] Researchers used female mice and male rats (tested serologically negative for *T. gondii*) and infected them with a latent *Toxoplasma* infection. To look at the parasite division mice were injected with luciferin, so the luciferase in the parasite will luminescence. About 4-5 weeks after infection the behavioural experiments took place. They used a lot of different behavioural testing, such as open-field exploration, aversion to bobcat urine, and aversion to cat collar and towel for rats. They also did a fear conditioning and Morris water maze test, but for these tests naïve animals were used. The behavioural tests in mice were avoidance of novel food, social transmission of food preference, and aversion to bobcat urine. Naïve mice were used for aversion to cat collar.

They found that the latent *Toxoplasma* infection in mice and rats changed the aversion to odours of cats to an attraction to these odours. This indicates a loss of fear. This loss of fear is very specific, whereas the infection did not change learned fear, anxiety, olfaction, and non-aversive learning. This is remarkable, because it indicates that the parasite can determine very specifically which part of the brain it wants to infect. Also in this research the researchers found that the specific brain area which the parasite infects is the amygdala. Specific means in this case that there are way more cysts in the amygdala than in other regions of the brain. The researchers also investigated other types of behaviour to look at the specificity of the infection, and they also found that the behavioural changes are very specific. [21]

In figure 6 the results of this study are showed. The researchers found that infected animals showed a higher luminescence flux, which means that there is a higher amount of parasites in the brain (figure 6a). With the luminescence method they measured the density of cysts in different brain areas. The result was that amygdalar areas contain a higher density of cysts than other brain areas (figure 6b+c).

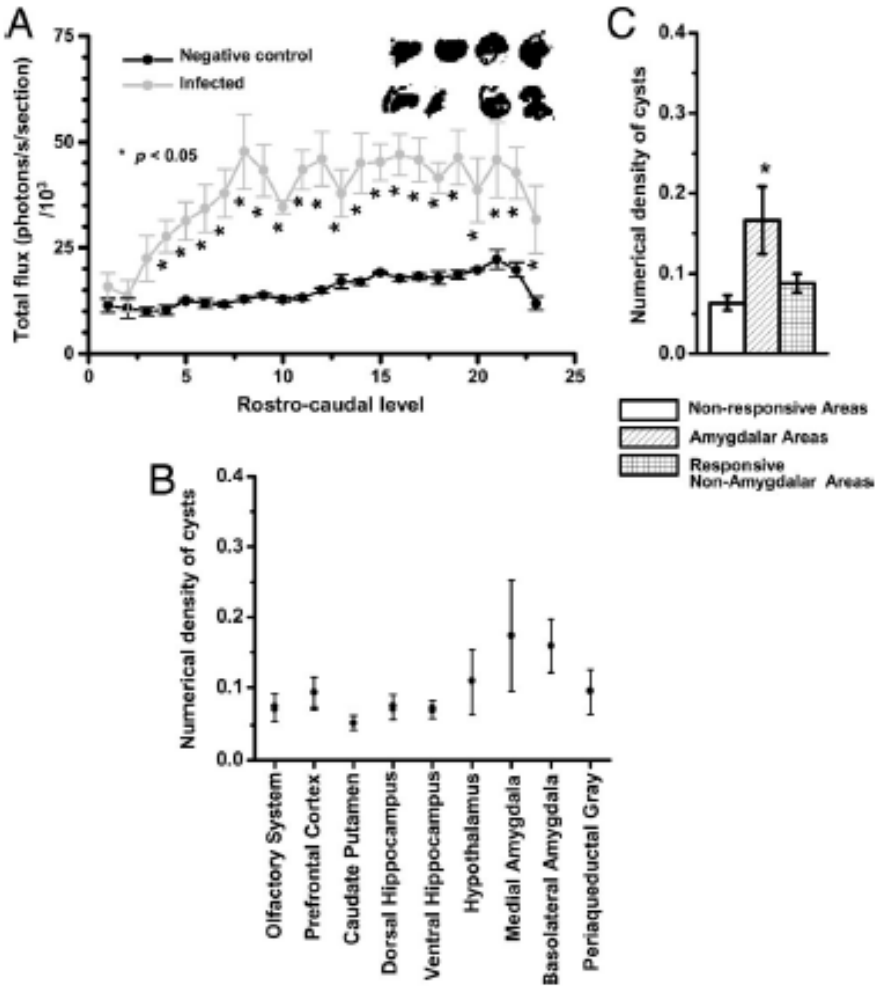

Figure 6.
Localization of tissue cysts in the brain. (A) Detection of parasites in brain slices using bioluminescence imaging. The presence of parasites is revealed on a broad range of coronal levels. (B) Tissue cysts were present in different brain areas. (C) Determination of tissue cyst density in different brain regions. [21]

This research indicates that the first hypothesis (the behavioural manipulation hypothesis) is more plausible than the one where they believed that the infection is non-specific. [21] The proximate mechanisms are still not known.

Brain and neuronal anatomy

Looking at the response of cells to the *Toxoplasma gondii* infection (table 3) one can see that the infection mostly considers neuron, microglia, endothelium, and astrocyte cells. The infection infects mainly neurons and muscle cells. The infectious stage of the parasite is mainly the tachyzoite stage, where there are also some parasitic cells in bradyzoite stage.

Brain cell type	Parasite stage	Activity
Neuron	Tachyzoite	Parasites can encyst in neurons
Neuron	Tachyzoite	Infection induces cytokine and chemokine production; stimulated neurons are unable to inhibit parasite growth
Neuron	Bradyzoite	Neurons containing parasite cysts avoid scrutiny by $CD8^+$ T cells
Neuron, microglia	Tachyzoite	Murine $Nramp1^{-/-}$ models are affected in stress response and mortality following *Toxoplasma gondii* infection
Microglia	Tachyzoite, bradyzoite	Microglial cells are preferentially infected, but most effectively inhibit parasitic growth within CNS cells
Microglia	Tachyzoite	Upon *Toxoplasma* infection, microglia produce IL-1 beta, IL-10, and tumor necrosis factor-alpha
Microglia, endothelium	Tachyzoite	Murine model infection induce an upregulation of CD200R & CD200, which control CNS inflammation
Microglia, astrocyte	Tachyzoite	Infection downregulates MHC class II expression
Microglia	Tachyzoite	Toxoplasmic encephalitis induces IL-12p40, iNOS, IL-1beta, TNF-alpha largely due to $CD8^+$ T cell interaction. MHC classes I and II, ICAM-1, and leukocyte function-associated antigen-1 are also upregulated
Endothelium	Tachyzoite	Toxoplasmic encephalitis induces vascular cell adhesion molecule, ICAM-1, and MHC classes I and II. Induction depends on IFN-gamma receptor
Endothelium	Tachyzoite	Infection induces ICAM-1, IL-6, and MCP-1 Induction levels vary depending on parasite strain
Astrocyte, neuron	Tachyzoite	Astrocytes are preferentially infected compared to neurons
Astrocyte, microglia	Tachyzoite	Intracellular infection reduces expressed MHC II
Astrocyte	Tachyzoite	Interferon-gamma-activated indoleamine 2,3-dioxygenase (IDO) induction inhibits parasite growth
Astrocyte	Tachyzoite	IFN- gamma induced parasite growth inhibition is independent on reactive oxygen intermediates
Astrocyte	Tachyzoite, bradyzoite	Tissue Inhibitor of Metalloproteinases-1 (TIMP-1) is induced by infection
Astrocyte	Tachyzoite	Autophagy may be involved in the elimination of the degraded parasite material from the astrocyte host cell cytoplasm
Astrocyte	Tachyzoite	IGTP is required for IFN-gamma-induced inhibition of parasite growth

Table 3.
The response of CNS-resident cells to Toxoplasma gondii *infection [16]*

Figure 7 shows the presence of the infection in rats. Remarkable is that the chronic infection stage shows no luminescence, which can be explained as having no presence of *Toxoplasma gondii* anymore. That is probably not the truth; it might be that the *Toxoplasma* infection is still present, but in a very small amount.

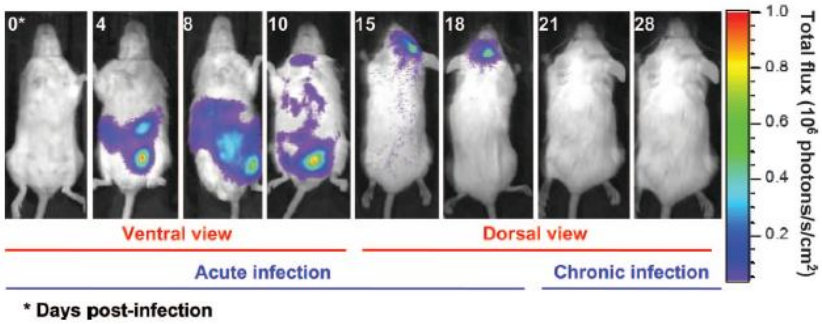

Figure 7
Infection caused a transient increase in luminescent signals emanating from parasites. The series of images reflects the spread of luminescent signal at successive days after infection. [21]

Mechanisms
Data shows that *T. gondii* is located primary in the amygdala. This is the area in the brain that regulates emotions, such as fear. It is known that the parasite can infect this specific area, but how the parasite can be so specific and which mechanisms underlie this are not known yet.

Human

Behavioural changes

Toxoplasma gondii is thought to induce behavioural changes in the host. [1] This is probably in advantage of the parasite to have a better distribution of their own cells. In animal studies infected rats were less fearful of cats and the smell of a cats urine than the control group. [6,18,19,21] Now the question arises: does the parasite have similar effects on human behaviour? And are these behavioural changes due to the immune response to the parasite of the host or are they the effect of the parasite by itself? The behavioural changes can also be due to the side effects of the symptoms of the disease. Or they can be a side effect to compensate the symptoms of the infection.

Cat odour

Toxoplasmosis in rats causes attraction to cat odour, but does the same thing happen in humans? Czech researchers investigated this. They found that humans infected with *T. gondii* rated the pleasantness of cat urine different than non-infected humans. Also there is a gender specific difference; infected men rated the pleasantness of cat urine higher than non-infected men, while non-infected women rated this pleasantness higher than infected women. They also investigated the smell of other animals' urine, but these findings showed no significance. [23]

Questionnaires

One way of investigating the human behaviour is via personality questionnaires. Different researchers use this method. They found that after *Toxoplasma* infection the behaviour changed compared to non-infected people. Infected persons showed higher anxiousness, and a decrease in novelty seeking (figure 8). However, they also found gender-specific differences. Infected men showed lower rule consciousness, higher cautiousness, jealousy, suspiciousness, and expedience. Women who are infected showed higher warmth and, unlike men, higher rule consciousness. They were listed more warm-hearted, persistent, moralistic, outgoing, and conscientious. [24,25,26]

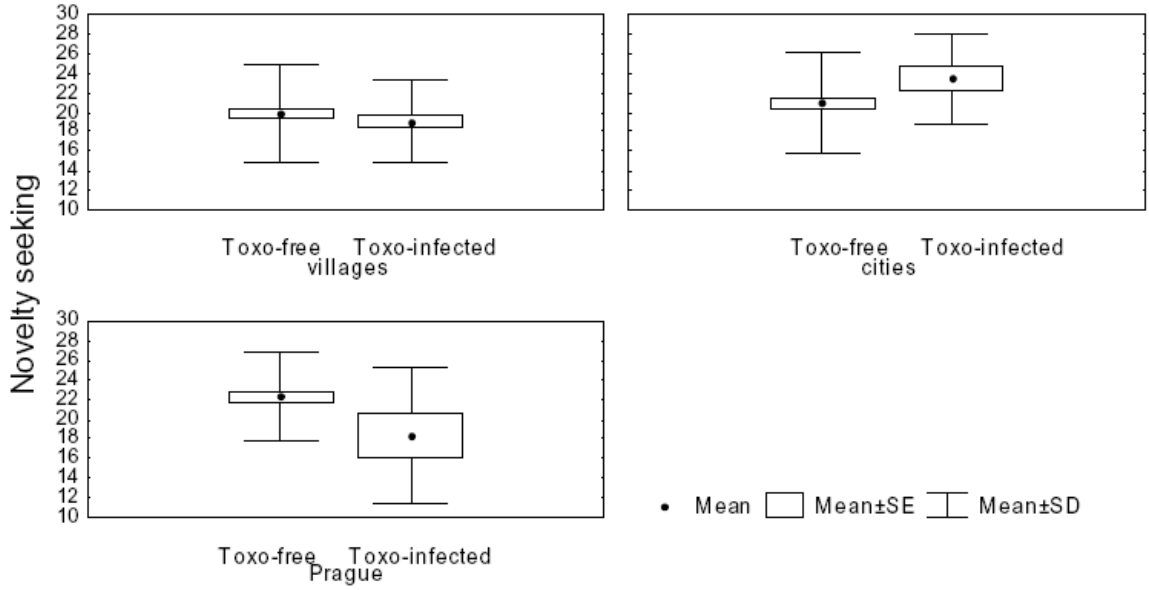

Figure 8.
Effects of toxoplasmosis and size of place of residence on the TCI dimension novelty seeking. [25]

Behavioural tests

A different way of investigating human behaviour is via behavioural testing. Personality questionnaires are a good way of investigating human behaviour because of the variety and amount of questions asked, but the reliability is influenced by self reflection. That is why behavioural testing is a good alternative.

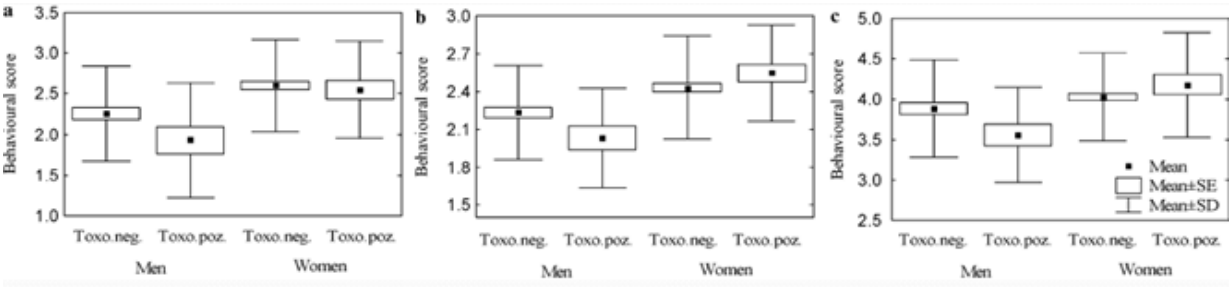

Figure 9.
(A) Relationships, (B) Self Control, and (C) Clothes Tidiness in infected and non-infected men and women. [27]

For this behavioural test Lindová *et al* used male and female students. They used questionnaires, interview techniques, clothing assessment and simple experiments. Their results (figure 9) were that men showed lower self-control and clothes tidiness, lower relationships/warmth, and higher mistrust in rural environment. Women showed a trend for higher self-control and clothes tidiness, but no differences in relationships and mistrust.
Remarkable is that the questionnaires show similar results. This makes the data more reliable.

Other researchers have found that after infection people have a slower reaction time. They also have a lower ability to concentrate. This is problematic in daily life, since these things result in a higher change of (traffic) accidents. They have even found that the chance of being involved in an accident in traffic situations increases with duration of infection. [28,29] Table 4 shows the number of subjects with or without *Toxoplasma gondii* infection, Rh blood group positive or negative, and the amount of accidents in traffic. It also shows the mean age per group. Here one may see that toxoplasmosis patients without Rh blood group relatively were involved in most accidents. This implies that the Rh blood group may have a protective effect on toxoplasmosis. That is probably why not every one who has *Toxoplasma gondii* infection shows symptoms.

Toxoplasmosis	Rh	Accident No		Accident Yes		Row	
No	negative	526 (13.52%)	20.23	14 (0.36%)	20.64	540 (13.88%)	20.24
No	positive	2391 (61.47%)	20.17	69 (1.77%)	20.29	2460 (63.24%)	20.17
Total		2917 (74.99%)	20.18	83 (2.13%)	20.35	3000 (77.12%)	20.18
Yes	negative	170 (4.37%)	19.98	11 (0.28%)	19.91	181 (4.65%)	19.97
Yes	positive	692 (17.79%)	20.02	17 (0.44%)	20.65	709 (18.23%)	20.04
Total		862 (22.16%)	20.01	28 (0.72%)	20.36	890 (22.88%)	20.02
Column Total		3779 (97.15%)	20.14	111 (2.85%)	20.35	3890 (100%)	20.15

Table 4.
Number of subjects and age in particular categories. [29]

Toxoplasma gondii and schizophrenia

After reading and searching for articles about *Toxoplasma gondii* and behavioural changes, you will find that a lot has been written about the correlation between *Toxoplasma gondii* and mental disorders, such as schizophrenia. Evidence has been found that *toxoplasma* infection also involves depression, obsessive compulsive disorder, Parkinson's disease, Alzheimer's disease, autism, epilepsy, and bipolar disorders. Drugs used in schizophrenia treatment inhibit behaviour also found in *Toxoplasma* patients. [30,31,32] The question arises whether *T. gondii* causes schizophrenia or the other way around; that schizophrenic patients are more sensitive to *T. gondii* infections. The first scenario seems to be the truth. Remarkable is that all psychiatric disorders mentioned above have dopamine dysfunction in common. So there might be a relation between toxoplasmosis and the dopamine pathway. More research have to be done to look at how these two influence each other.

Conclusion

Brain sciences are very popular these days, but even after all these research, there is still a lot not known about the brain. It seems that microorganisms 'know' more about the brain and about which area they want to infect than what researchers have discovered so far. However, researchers discover more and more, so in time the specific effect of toxoplasmosis will become clear.

In humans there are a lot of correlations investigated, and there are correlations, but there is not one strong one that stands out and which can convince people that the behavioural changes are due to the *Toxoplasma gondii* infection. To answer the main question: "Do microorganisms control our behaviour?", the answer is that microorganisms might control our behaviour, but the evidence is not that convincing. Further research can show whether the research results are coincidence or whether microorganisms do control our behaviour.

Discussion

The behavioural manipulation hypothesis has supporting data and results through researches. After more investigations in this research field it will become clear whether the behavioural manipulation hypothesis can change into a behavioural manipulation theory. The hard part here is that if researchers will find more supporting data in mice and rats, will this also be true for other animals and even humans?

Likely also in humans

Although there is not one very convincing result about whether microorganisms affect our behaviour, individually all researches are pointing to the same thing, that is that there is a big chance that they do affect our behaviour. In rats the results are more convincing than in human beings, but that is also due to the fact that researchers can give rats the infection, and in humans they can only look at people already having the infection. This way one can only look at the behaviour before the infection, and compare it to the behaviour occurring after the infection, but this way the judgement about the behaviour has already been influenced by later experiences.

General medical investigations can solve this problem, because data have already been collected before the possible infection, so it can be compared to the behaviour after this infection, and this way it is more reliable. However, general medical investigations have started not that long time ago, so the results of this have to be waited for.

The uncertainty about whether microorganisms do or do not affect our behaviour does not stimulate the suppression of the parasite. Since the mechanisms are not known yet, medication can be given, but which medicine works best still needs to be discovered. One may say to give patients antibiotics, because it is an infection, but because of the production of cysts the behaviour might be affected anyhow. Another one may say to give patients dopamine inhibitors, because of the parasite's possible working on the dopamine pathway.

Other microorganisms

Now the question arises whether other microorganisms have similar effects on behaviour. Since there has less research been done investigating other microorganisms, there cannot be said a lot about this. However, when researchers investigate other microorganisms more, they might find consistent results and that way they can achieve more insight in the problems surrounding the question if and how microorganisms affect our behaviour.

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