



## Behavioral Study of Mice Experimentally Infected with *Toxoplasma gondii*

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**T**OXOPLASMA *gondii* parasite is a parasitic protozoan that has the ability to infect a wide range of warm-blooded intermediate hosts, including humans. The parasite invades the host's central nervous system of the host, causing damage to the brain and muscles and may cause an imbalance in neurotransmitters, which causes abnormal behavior changes of the host. This study aimed to measure the behavioral changes in laboratory mice which infected experimentally with *Toxoplasma gondii*. The parasite was isolated from placenta samples in women infected with toxoplasmosis from Al-Salam Teaching Hospital in Mosul during September 2022, and injected into peritoneal cavity of laboratory mice, the behavioral changes were measured in mice open field (OF) and determined the effect of infection on concentration of dopamine and acetylcholinesterase in mice serum. As the results showed a decrease in the locomotors activity of the mice in the open field, as well as a decrease in the rate of movement in the open field in males and females mice after 6 weeks p.i. Serological tests in males and females showed an increase in dopamine concentration after 3 and 6 weeks p.i. while caused a significant decrease in the concentration of acetylcholinesterase in serum of infected males and females mice. This result indicates that infection with *Toxoplasma gondii* leads to an increase and impairment in the ability to explore, and the cysts of the parasite can destroy neurons in the brain, alter the concentration of neuroimmune and interfere with the process of regulating neuroimmunity in the host.

**Keywords:** Behavioral disorders, Cholinesterase, Dopamine, *Toxoplasma gondii*, Toxoplasmosis.

### Introduction

*Toxoplasma gondii*, a neurotropic parasite of the phylum Apicomplexan unicellular eukaryotic, cause worldwide zoonotic diseases called toxoplasmosis. Prevalence levels vary widely, human is a secondary hosts in the transmission cycle of *T. gondii* in the environment, about 30% – 80% of the human population are infected [1]. Cats mainly become contaminated by ingesting animals encysted with *T. gondii* (mouse, bird) [2]. Toxoplasmosis is one of the most frequent infections of the central nervous system (CNS) which associated with several neuropsychiatric conditions. The parasite infects direct invasion

of the brain as well as effector on the peripheral immune system and causes CNS inflammation by altering dopamine metabolism which is responsible for inflammation of the central nervous system and may lead to chronic neurological lifelong in the CNS [3]. *Toxoplasma gondii* is able to induce direct modifications in the infected CNS cells, lead to changes in the behavioral of the host [4], schizophrenia pathogenesis in animal models might be related to toxoplasmosis, if the infection is not resolved for years [5]. The most fascinating effect of *T. gondii* on mice behavior that is the increase of attraction of cat [6, 7]. Toxoplasmosis effect on human personality and behavior is wide range and revealed several

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changes in their psychology including psychotic-like symptoms, suicide attempts, mixed anxiety, self-directed violence, depressive disorder and depression during pregnancy. Some studies suggest that parasite clearance by promoting systemic immunity alleviates neuroinflammation [8] and may be changing in the neuronal activity caused by the parasite, an adaptation towards the inflammatory environment [9].

The aim of this research was to study the influence of *T. gondii* infection on behavior mice models infected experimentally.

## **Material and Methods**

### *Experimental Design*

A total of 48 mice were used in this study, The experimental groups comprised of:

- Group (1), consisted of 12 female mouse, injected with *T. gondii* isolated from Placenta and the behavioral changes were studied including 6 mouse after 3 weeks p.i. and 6 mouse after 6 weeks p.i
- Group (2) consisted of 12 male mouse injected with *T. gondii* isolated Placenta and behavioral changes were studied including 6 mouse after 3 weeks p.i. and 6 mouse after 6 weeks p.i.
- Group 3(Control), consisted of 12 mouse male and 12 female without any treatment (including 6 mouse after 3 weeks p.i. and 6 mouse after 6weeks p.i of each treatment ).

### *Sample collection*

Five Placenta samples were collected from aborted women from Al-Salam Teaching Hospital in Mosul during September 2022, (standard ethical issues were considered through the manipulation of human tissues). The samples were kept in clean and sterile containers containing phosphate buffer saline (PBS) and placed in a refrigerated container until the samples arrived at the laboratory to be used for parasite isolation.

### *Parasite isolation*

In order to isolate the pure parasite from infected placentas which collected from aborted women previously, the placenta was cut into small pieces and suspended with 5 ml of pepsin enzyme at a temperature of 37 °C. The suspension was incubated at 37 °C for 10 minutes then filter with gauze. The mixture was centrifuged at 3000 rpm for 10 minutes, then the precipitate was suspended in a PBS solution with a pH of 7.2[10].

### *Microscopic examination*

About 10 µl of suspension placing on a slide and stained with Giemsa for 10 minutes then washed with distilled water, examined under light microscope with 100x magnification Figure (1).

### *Molecular Diagnosis of the Parasite*

#### *A. DNA extraction from the blood of cats*

Whole blood samples were collected in EDTA tubes , were used to extracted DNA from blood cats, depending on method manufacturer's instructions kit of the Korean company: Add Bio (add prepGenomic DNA) Extraction kit.

#### *B. Amplification B1gene region by conventional PCR*

The polymerase chain reaction technique was used to detect *B1*gene region from in order to the diagnosis of *T.gondii* in cat blood samples, by dilution with Borat, EDTA buffer Tris solution to obtain the required concentration for conducting the PCR reactions at 50 ng/microliter for each sample.

The primers targeting *B1* gene were used according to Mohammed et al.[11] .The master mixture reaction volume were done in a final volume PCR reaction fixed to 20 µl, the reaction mixture contained 4µl DNA template , 4µl DW and 1µl of 10pmol of each of the forward and reverse primers. The PCR cycler condition was installed according to the guide as shown in (Table 2) [11].

#### *C. Agarose gel electrophoresis of DNA products:*

Separation of the PCR products was done in 2% agarose gel electrophoresis, The electrophoresis samples were prepared by mixing 5 µl of DNA sample with 3 µl of loading solution then 4 µl of DNA ladder (100 bp) were add . Finally, PCR product were electrophoresed with 80 volts for 60 minutes and the results were visualized under UV light [11] .Table (1), and according to the program shown in Table (2).

#### *How to prepare the injection dose*

The injection dose was prepared and enumerated about 100 tissue cysts from a drop of 10 µl from suspension prepared previously, placed on the slide and stained with Giemsa dye. Then, the dose was prepared by adding physiological saline solution with a pH of 7.2 and antibiotic "Pen & Strep" of 0.1 mg, each mice injected with about 0.1ml suspension with 100 tissue cysts [12].

#### *Injected animals*

Swiss albino mice(males and females), aged

**TABLE 1. The nucleotide sequence of the primers and the temperature used to detect the B1 gene of the *Toxoplasma gondii* parasite**

No.	The sequence of the nitrogenous bases	Temperature	Length	Primer
1	TTTTGACTCGGGCCAGC	60	18	Forward
2	GTCCAAGCCTCCGACTCT	58	18	Revers

**TABLE 2. Steps of the PCR program to detect the B1 gene**

No.	Stage	Temperature	Time	Cycle number
1.	Initial denaturation	95	6 min.	1
2.	Denaturation	95	45 sec.	
3.	Annealing	56	1 min.	35
4.	Extension	72	1 min.	
5.	Final extension	72	5 min.	1

21-25 days, and their weight ranged between 17 to 20 grams were used as in experimental design. The mice were given an intraperitoneal injection (IP) with a 0.1 ml dose containing approximately 100 tissue cyst.

#### *Collection of blood samples*

Blood samples were collected from infected mice from the retro-ocular vein for serum after determined behavioral test, the animals were manipulated within a suitable standards and ethical issues considered to mice by veterinarian. The serum was withdrawn and placed in small Eppendorf tubes and stored at -20° C until the required tests are performed later [13].

#### *Brain impression smear*

All mice used in the experiment were dissected and the brain extracted and impression smear of brain was done on a glass slide and stained with Giemsa dye, then it was confirmed that the experimental infection with the parasite occurred by observing the tissue cyst in these prints.

#### *Serological tests - ELISA Test*

The DA, ACHE- ELISA Kit from the Chinese Company SUNLONG was used to measure the concentration of acetylcholinesterase and dopamine in the serum samples of the mice according to the method described in the User Manual, after which the absorbance optical density (OD) was read at (450 nm).

#### *Behavior test*

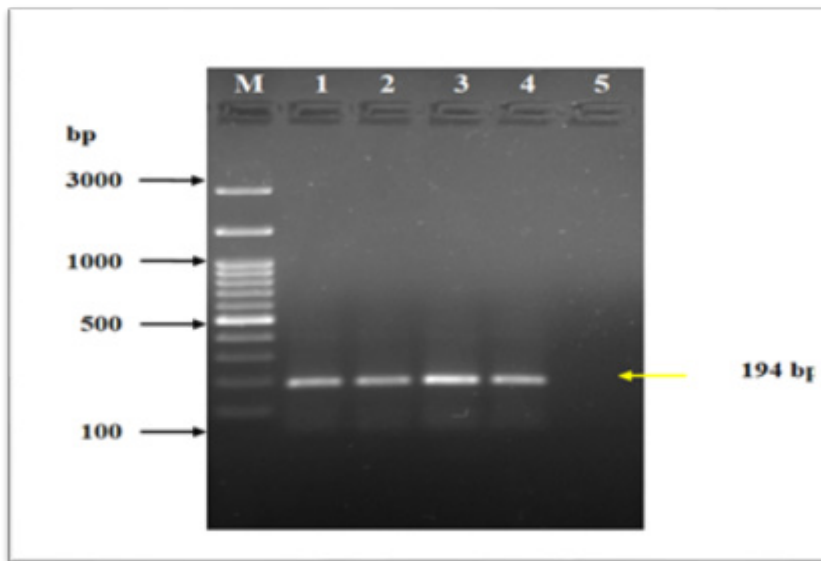
Mice behavior of for each mouse was measured inside a quiet room using the open field (OF), the cage made of wood and a white floor divided into 25 squares with dimensions of 59 × 59 × 30 cm with sterilizing the box with 70% ethyl alcohol before starting to test each animal by gently placing each mouse in the middle of the box and following up on the number of cut squares, the number of climbs, rest, the number of pauses, the number of times of presence in the center of the box, and the type of animal movement. It was recorded in a 3-minute video and follow-up of these measurements three and six weeks after the experimental infection with the parasite. Mice behavior evaluated after 3 weeks p.i. which represents acute infection and 6weeks p.i as a chronic infection [14].

#### *Statistical analysis*

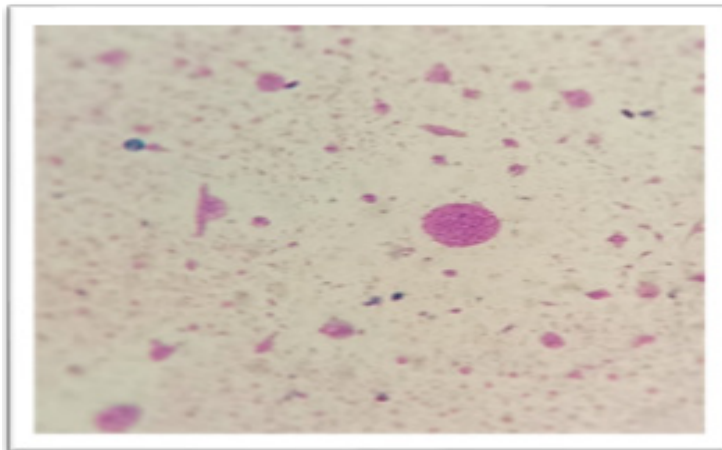
The statistical program SPSS was used to analyze the results of the study Tow- way Analysis of Variance at a significant level of  $P \leq 0.05$ .

#### **Results**

The results of the molecular examination study using polymerase chain reaction by targeting and amplifying the B1 gene showed that toxoplasmosis infection was confirmed in placenta samples from which the parasite was isolated Fig. (1 and 2) .



**Fig. 1.** Shows the electrophoresis of agarose gel and the bands resulted from the polymerase chain reaction (PCR) for the detection of *Toxoplasma gondii*. The path M: Marker shows size of 100 bp. The path 1-4 represent positive blood samples from infected placenta with a size of 194 bp, The path 5 represents the negative control.



**Fig. 2.** Tissue cyst of *T. gondii* from placenta suspension

#### *Behavioral changes*

##### *Passing through the middle*

By following up the locomotor activity of the mice in the open field, the results showed that there was a significant difference, represented by a significant decrease in the presence of experimentally infected males in the middle, six weeks after infection, at a rate of  $0.8 \pm 0.37$  times. As for females, there was a significant decrease six weeks after infection, at a rate of  $1.0 \pm 0.0$  times (Table 3).

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##### *The duration of the stopping or resting*

By following up on the results of the period of inactivity of the mice in the open field, the infected males showed that there was no significant difference between the infected group and the control group after three weeks and six weeks p.i. While the results of females showed a significant difference between the female groups (infected and control) after six weeks p.i, represented by a decrease in the rate of stopping movement by  $3.8 \pm 0.37$  seconds (Table 4).

**TABLE 3.** The average number of present times in the middle of the box for males and females experimental infected with *Toxoplasma gondii* after 3 week p.i. (acute infection) and 6week p.i (chronic infection) .

Group/ Period	Control males (n=6)	Infected males (n=6)	Control females (n=6)	Infected females (n=6)
3 weeks p.i. (Acute infection)	3.0 ± 0.31 <sup>b</sup>	1.4 ± 0.2 <sup>a</sup>	2.0 ± 0.3 <sup>ab</sup>	1.2 ± 0.48 <sup>a</sup>
6 weeks p.i. (Chronic infection)	3.0 ± 0.3 <sup>c</sup>	0.8 ± 0.37 <sup>a</sup>	2.0 ± 0.31 <sup>b</sup>	1.0 ± 0.0 <sup>a</sup>

\*The alphabet letters indicate that there is no significant difference, and different letters indicate that there is a significant difference.

\* Values represent the mean ± error of measurement for each 5 mice in the group at a significant level of 0.05.

**TABLE 4.** The average period stopped moving in seconds for males and females experimental infected with *Toxoplasma gondii* after 3 weeks p.i. (acute infection) and 6 weeks p.i (chronic infection)

Group/ Period	Control males (n=6)	Infected males (n=6)	Control females (n=6)	Infected females (n=6)
3 weeks p.i. (acute infection)	3.25±1.31 <sup>a</sup>	6.20±1.69 <sup>a</sup>	4.28±1.16 <sup>a</sup>	7.0±2.002 <sup>a</sup>
6 week p.i. (chronic infection)	3.25±1.31 <sup>a</sup>	7.59±1.39 <sup>a</sup>	5.6±0.51 <sup>a</sup>	3.8±0.37 <sup>b</sup>

\*The alphabet letters indicate that there is no significant difference, and different letters indicate that there is a significant difference.

\* Values represent the mean ± error of measurement for each 5 mice in the group at a significant level of 0.05.

#### Mice movement

Our finding in the results rate of spontaneous locomotor activity moving in the open field, that there was no significant difference in males and females experimental infected with *Toxoplasma gondii* after 3 weeks p.i. While there was a significant decrease in the locomotor activity of the mice after 6weeks p.i, which was represented by a decrease in the speed of movement in the infected mice in the open field, as the lowest rate of movement in male mice was recorded at a rate of 0.8±0.37, and the same was the case in females at a rate of 1.0±0.0 compared to the control group (Table 5).

#### Climbing

The results of climbing and jumping across the wall in experimentally of infected males and females in the open field, were close to the control group after 3 week p.i. (acute infection) in males at a rate of 19.7±1.45 times and in females at a rate of 19.2±2.24 times. A significant difference appeared, represented by a decrease in the number of climbing times in infected males after six weeks

1.0±0 times as in Table (6).

#### The number of times of stopping movement

The results revealed that the number of times of stopping movement for mice in the open field, showed that there was no significant difference in males between the control group and the infected group after 3 weeks p.i. (acute infection) and 6 weeks p.i (chronic infection).

While the results showed a significant difference represented by a decrease number of stops with in the females after 3 weeks p.i. (rate of 2.8 ± 0.48 times), (Table 7).

#### Number of squares (distance crossed)

By following up the locomotor activity of the mice in the open field, the results showed a decrease in the number of squares crossing by in the infected males, this decrease was significant after three weeks p.i at a rate of 77.4±8.4 times, while in females the significant decrease was great after sixth week p.i at a rate of 69.6±1.7 squares (Table 8).



**TABLE 5. The rate of spontaneous locomotor activity moving in seconds for males and females experimental infected with *Toxoplasma gondii* after 3 weeks p.i. (acute infection) and 6 weeks p.i (chronic infection)**

Group/ Period	Control males (n=6)	Infected males (n=6)	Control females (n=6)	Infected females (n=6)
3 weeks p.i. (acute infection)	3.0±0.31 <sup>a</sup>	3.0±0.31 <sup>a</sup>	3.0±0.36 <sup>a</sup>	2.4±0.5 <sup>a</sup>
6 weeks p.i. (chronic infection)	3.0±0.0 <sup>c</sup>	0.8±0.37 <sup>a</sup>	2.0±0.31 <sup>b</sup>	1.0±0.0 <sup>a</sup>

\* The alphabet letters indicate that there is no significant difference, and different letters indicate that there is a significant difference.

\* Values represent the mean ± error of measurement for each 5 mice in the group at a significant level of 0.05.

**TABLE 6. The average of the number of climbing times for males and females experimental infected with *Toxoplasma gondii* after 3 weeks p.i. (acute infection) and 6 weeks p.i (chronic infection)**

Group/ Period	Control males (n=6)	Infected males (n=6)	Control females (n=6)	Infected females (n=6)
3 weeks p.i. (acute infection)	23.2±0.6 <sup>a</sup>	19.7±1.45 <sup>a</sup>	19.4±0.74 <sup>a</sup>	19.2±2.24 <sup>a</sup>
6 weeks p.i. (chronic infection)	22.7±0.6 <sup>a</sup>	16.1±3.7 <sup>a</sup>	2.33±0.3 <sup>a</sup>	1.0±0 <sup>b</sup>

\* The alphabet letters indicate that there is no significant difference, and different letters indicate that there is a significant difference.

\* Values represent the mean ± error of measurement for each 5 mice in the group at a significant level of 0.05.

**TABLE 7. The average number of stopped moving for males and females experimental infected with *Toxoplasma gondii* after 3 weeks p.i. (acute infection) and 6 weeks p.i (chronic infection).**

Group/ Period	Control males (n=6)	Infected males (n=6)	Control females (n=6)	Infected females (n=6)
3 weeks p.i. (acute infection)	4.0±0.31 <sup>a</sup>	4.0±0.70 <sup>a</sup>	5.8±0.374 <sup>b</sup>	2.8±0.48 <sup>a</sup>
6 weeks p.i. (chronic infection)	3.8±0.37 <sup>a</sup>	5.4±1.28 <sup>a</sup>	5.8±0.48 <sup>a</sup>	4.0±0.36 <sup>a</sup>

\* The alphabet letters indicate that there is no significant difference, and different letters indicate that there is a significant difference.

\* Values represent the mean ± error of measurement for each 5 mice in the group at a significant level of 0.05.

#### Serological test results

##### Acetylcholinesterase concentration in serum mice:

Experimental infection with *Toxoplasma gondii* in mice caused a significant decrease in the concentration of acetylcholinesterase in serum of infected males, females' mice, decrease was significant compared to the control group, as in Table (9).

##### Dopamine concentration (DO) in serum mice

The results of serological tests in males and females showed an increase in dopamine concentration after 3 weeks p.i. (acute infection), while the increasing was significant in the chronic phase in males with a mean of 313.7±55.1 pg/ml and in females with a mean of 316.5 ±89.4 pg/ml.

**TABLE 8. The average number of squares crossed for males and females experimental infected with *Toxoplasma gondii* after 3 weeks p.i. (acute infection) and 6 weeks p.i (chronic infection).**

Group / Period	Control males (n=6)	Infected males (n=6)	Control females (n=6)	Infected females (n=6)
3 weeks p.i.(acute infection)	126.8±0.4 <sup>c</sup>	77.4±8.4 <sup>a</sup>	99.4±0.92 <sup>b</sup>	99.4±0.9 <sup>b</sup>
6 weeks p.i.(chronic infection)	87.0±0.7 <sup>ab</sup>	64±18.07 <sup>a</sup>	103.8±0.37 <sup>b</sup>	69.6±1.7 <sup>a</sup>

\*The alphabet letters indicate that there is no significant difference, and different letters indicate that there is a significant difference.

\* Values represent the mean ± error of measurement for each 5 mice in the group at a significant level of 0.05.

**TABLE 9. The average concentration of acetylcholinesterase in serum of males and females experimental infected with *Toxoplasma gondii* after 3 weeks p.i. (acute infection) and 6 weeks p.i (chronic infection).**

Groups	Males	Females
Control animals (n=6)	387.24±43.3 <sup>B</sup>	289.9±17.08 <sup>C</sup>
3 weeks p.i. (acute infection) , (n=6)	43.75±8.9 <sup>A</sup>	26.32±4.2 <sup>A</sup>
6 weeks p.i. (chronic infection), (n=6)	70.63±19.2 <sup>A</sup>	89.54±8.6 <sup>B</sup>

\* The alphabet letters indicate that there is no significant difference, and different letters indicate that there is a significant difference between the groups.

\*The values represent the mean ± standard error for all 5 mice in the group at a probability level of P≤0.05.

-The mark (\*) indicate the significant difference between the control group and the infected group.

**TABLE 10. The rate of dopamine concentration in serum males and females experimental infected with *Toxoplasma gondii* after 3 weeks p.i. (acute infection) and 6 weeks p.i (chronic infection).**

Groups	Males	Females
Control animals (n=6)	81.2± 28.3 <sup>A</sup>	119.1±0.9 <sup>A</sup>
Infected in the acute phase (n=6)	151.5±36.9 <sup>A</sup>	160.003±17.79 <sup>A</sup>
Infected in the chronic phase (n=6)	313.7±55.1 <sup>B*</sup>	316.5±89.4 <sup>B*</sup>

-The alphabet letters indicate that there is no significant difference, and different letters indicate that there is a significant difference between the groups.

-The values represent the mean ± standard error for all 5 mice in the group at a probability level of P ≤ 0.05.

-The mark (\*) indicate the significant difference between the control group and the infected group.

## Discussion

Toxoplasmosis, which affects about 30% - 80% of population globally, is caused by infection with the parasite *Toxoplasma gondii* that can infect the central nervous system (CNS), promoting neuroinflammation and neurotransmitter imbalance, as well as behavioral changes although it is considered asymptomatic in its latent stage, it has been found different effects between the sexes on personality behavior [15]. Studies have stated that infection with the parasite may be associated with neurological and psychological disorders such as schizophrenia [16]. There is mounting evidence confirming that infection with the parasite radically changes the behavior of rodents and is associated with the occurrence of certain psychological and neurological conditions in humans [9].

The infection spreads to neurons such as astrocytes and microglia [17]. These tissue cysts destroy neurons, alter neurotransmitter concentrations, and interfere with neuroimmunity regulation in the host [18]. Affected mice showed significant behavioral disturbances such as diminished exploratory activity [19] and neurological and behavioral abnormalities in affected mice during the chronic period [20]. Studies have reported that the parasite has the ability to subtly alter host behaviors either through manipulation to promote transmission to the definitive host in cats or as a side effect of infection [21].

The results of the current study showed a decrease in the presence of infected mice in the middle of the box, and this is consistent with what was stated by many authors [22, 23]. In his study of the behavior of infected females after 8 weeks of infection, as they noticed a decrease in the rate of presence of experimentally infected mice in the middle of the box. It is consistent with the results of Soares et al.[13] with a decrease in the presence of infected mice in the middle of the box after 12 and 22 days of infection. Boillat et al.[24] also reported a similar result with a decrease in the presence of infected mice in the central quadrant. This result indicates an increase in the level of anxiety. As Gatkowska et al.[14] mentioned during his study that the mice were less present in the central part of the box compared to the uninfected mice in

the acute and chronic phase, he noticed that the change in the locomotor activity of infected mice during the acute phase in the peripheral parts was more evident, as all infected mice showed less likely to be present in the central part, usually in unfamiliar surroundings, and the mice first explore the peripheral parts and then move to the central parts in the open environment [25,26]. Studies indicated that mice with toxoplasmosis tend to remain in the peripheral region of the box, especially during acute toxoplasmosis [27]. This may be due to the differences resulting from histopathological changes resulting from the proliferation of tissue cysts of the *Toxoplasma gondii* parasite in the brains of infected mice. It may be a pathological immune response due to the presence of tissue cysts in the brain. Studies have stated that infection may contribute to a marked deficit in behavior and may make infected mice more vulnerable to predation by the domestic cat, which is the definitive host of *Toxoplasma* [27]. The results showed a significant difference represented by the increase in the duration of stopping, and this is consistent with what was indicated by Soares et al.[13]. The largest decrease in the ability to move was 22 days after infection, as Bezerra et al. [28] indicated that infection with toxoplasmosis leads to an increase in the period of stopping or resting, especially in the period of chronic infection. Although studies aimed at evaluating the behavioral effect that was conducted on the behavior of animals infected with different strains of *Toxoplasma gondii* are recent and rare, they reinforce the importance of the influence of genetic factors, metabolism and immune response to the development of changes in rodent behavior [29].

The results showed a decrease in the number of climbing times in infected mice compared to the control group. Gatkowska et al.[14] indicated a significant decrease in the number of climbing times in male mice infected with *Toxoplasma gondii* in the acute phase and the chronic phase. The results showed a decrease in the number of squares crossed by the affected animals in general, and this is consistent with the results shown by Skallova et al.[ 30] that infected male and female mice crossed fewer squares compared to the control group. It may be due to neurological disorders caused by *Toxoplasma gondii* infection



due to an increase in the number of parasitic cysts in the central nervous system [31].

The presence of the parasite has been associated with behavioral changes in various hosts, including humans, although this relationship is not clearly understood, as it is believed that these behavioral changes represent a change in the behaviors of the parasite to promote the transition from the intermediate host to the definitive host in cats [32,19]. By following up the results of the current study, a decrease in dopamine concentration is observed in the acute phase, then it begins to increase in the chronic phase, and this is consistent with what was mentioned by Omidian et al. [33]. A previous study also found an increase of 14% in dopamine concentration in chronic phase [34,35], also they mentioned that increased dopamine release is associated with *Toxoplasma gondii* in males with high rates. Wang et al. [36] stated that infection with the parasite leads to a defect in neurotransmitters and activation of NF-B and dopamine signaling pathways in infected mice. Studies confirmed the existence of evidence that the *Toxoplasma gondii* parasite causes neurological and psychological disorders in humans [37] it also reduces motor function in the chronic condition [38,1] and there are many studies that showed the association of chronic Alzheimer's disease and Huntington's disease in humans [39] and Wang et al. [36] mentioned through disruption of the dopamine neurotransmitter and activation of the NF-B signaling pathway. Excessive activation of inflammation in the brain can lead to neuronal apoptosis [40]. It promotes neuroinflammation, neuronal loss, neurotransmitter imbalance, and behavioral changes [41]. Wana et al. [42] stated that each strain of the parasite can cause changes in behavior to varying degrees.

Cholinergic receptors are particularly involved in anti-inflammatory reactions [43] AChE is a membrane-bound enzyme present mainly in brain, muscle, erythrocytes, lymphocytes, and cholinergic neurons ACh is known to be produced within lymphocytes [44, 45]. Therefore, an increase in the number of lymphocytes definitely increases the concentration of free ACh and thus increases the activity of AChE as an inflammatory procedure [46].

## Conclusion

By following up the results of the current study, it was found that the *Toxoplasma gondii* parasite has a major role in changing the behavior of infected animals, which was reflected in the inability to concentrate in movement in the open field. The effect was greater in females than in males. We suggest conducting a study on the neurological effect of each strain of the parasite on humans and its association with neurological and psychological diseases.

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## Conflict of Interest

Authors declare that there have no conflict of interest about this article.

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## دراسة سلوكية للفئران المصابة تجريبياً بالتوكسوبلازما جوندي

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يعتبر طفيل التوكسوبلازما جوندي من الاوالي الطفيلية التي لها القدرة على اصابة مجموعة واسعة من العوائل الوسطية من ذوات الدم الحار منها الانسان، بسبب داء المقوسات الذي يصيب مدى واسع الانتشار من المضائف بضمنها الانسان اذ سجلت نسب الاصابة تقدر بأكثر من ثلث سكان العالم. يغزو الطفيل الجهاز العصبي المركزي للمضيف مسبباً أذى للدماغ والعضلات وقد يكون وجود الطفيلي في الدماغ سبباً في حدوث خلل في النواقل العصبية والتي تسبب حدوث التغيرات السلوكيات غير الطبيعية للمضيف. هدفت هذه الدراسة الى قياس التغيرات السلوكية في الفئران المختبرية بعد أحداث اصابة تجريبية بالطفيل ، اذ تم عزل الطفيلي من عينات المشيمة لنساء مصابات بداء المقوسات جمعت من مستشفى السلام التعليمي في مدينة الموصل وحقت في التجويف البريتوني للفئران المختبرية . قيمت التغيرات السلوكية (التواجد في المنتصف , مدة التوقف , عدد مرات التسلق ) في الفئران المصابة بعد مرور 3 و6 أسابيع بعد الاصابة ، اذ اظهرت النتائج انخفاض لتواجد الفئران المصابة في المنتصف فضلاً عن انخفاض في معدل الحركة في الميدان المفتوح بشكل عام في مجاميع الذكور والاناث في حين اظهرت الفئران المصابة زيادة مدة التوقف او السكون و انخفاض في عدد مرات التسلق. تشير هذه النتيجة الى زيادة مستوى القلق مما يعني أن الاصابة أعاققت المسار الطبيعي للاستكشاف. تشير هذه النتيجة الى أن الاصابة بالتوكسوبلازما جوندي أحدثت أعاققة في القدرة على الاستكشاف، ويمكن لأكياس الطفيلي أن تدمر الخلايا العصبية في الدماغ وتغير تركيز النواقل العصبية وتتداخل مع عملية تنظيم المناعة العصبية في المضيف.

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