

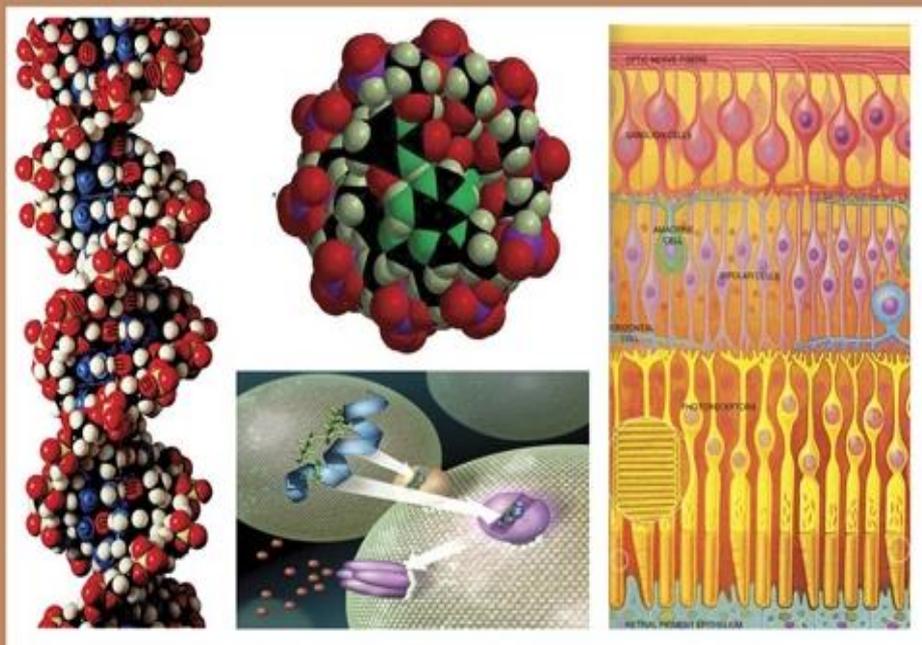


C

EGYPTIAN ACADEMIC JOURNAL OF

# BIOLOGICAL SCIENCES

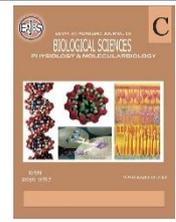
PHYSIOLOGY & MOLECULAR BIOLOGY



ISSN  
2090-0767

WWW.EAJBS.EG.NET

Vol. 14 No. 2 (2022)



## Relationship Between *Toxoplasma gondii* and Sex Hormone

Rwaid Dhari Hussein and Aysir Saleh Mohammed

Department of pathological analysis, College of Applied Sciences, University of Samarra

E-mail : rawed.dari.iq@gmail.com- dr.aysaralsamarrai@uosamarra.edu.iq

### ARTICLE INFO

#### Article History

Received:27/8/2022

Accepted:28/10/2022

Available:31/10/2022

#### Keywords:

*Toxoplasma gondii*,

Sex Hormone,

Baghdad.

### ABSTRACT

The study was conducted between August 2021 and April 2022, and samples were collected as outpatients at a fertility laboratory in Baghdad Governorate, whether they were infected or not.

A total of 150 blood samples were collected from men participating in the Baghdad Provincial Fertility Laboratory by drawing 5 mL of venous blood using a tourniquet and a 5 mL wine syringe and placing them in two types of laboratory tubes for testing. Information about these people obtained through a paper a questionnaire that provides a wealth of information about the subject of the study, including name, age, education, date, place of residence, and whether there are cats in the home.

The effects of parasites on hormone levels (LH, FSH, prolactin, and testosterone) and parasites were examined after the samples were split into four groups (control group, *Toxoplasma* infection group, infertility group, parasite infection group, and infertility group).

### INTRODUCTION

Monoparasite intracellular parasite *Toxoplasma gondii* is found in all cells. This parasite causes toxoplasmosis, often known as cat disease, which is a disease that affects both humans and animals and is a worldwide opportunistic disease, especially in immunocompromised people, while being asymptomatic in immunocompetent people, depending on the detection of anti-toxoplasma antibodies in the serum, the worldwide infection rate ranges from less than 10% to around 90%. (Torgerson and Mastroiacovo, 2013), due to the fact that domestic and wild cats constitute the parasite's primary means of transmission, which involves passing the oocysts of *Toxoplasma gondii* through feces general health, (Cenci-Goga *et al.*, 2011), The fact that the clinical symptoms in the acute phase are mild and similar to a cold and disappear within days and change into the chronic phase, where the patient may not exhibit any clinical symptoms, contributes to the parasite's spread and keeps the animal or person infected for the duration of its life (Dabritz *et al.*, 2010). (AL-Khafaji *et al.*, 2020).

#### Aim of the Study:

1. A study of the incidence of toxoplasmosis among males visiting outpatient clinics in Baghdad and a study of how the parasite affects men's ability to conceive.
2. By doing joint immunological testing between partners, it is possible to determine whether there is a chance of sexual transmission of the parasite.
3. Understanding how the *Toxoplasma* infection affects the concentration of male hormones such (Testosterone, Prolactin, LH, FSH).

## MATERIALS AND METHODS

### Sample Collection:

#### Blood Samples

In order to gather 150 blood samples from males visiting the fertility lab in the Baghdad governorate, 5 ml of venous blood was drawn using a Tourniquet ring and a 5 ml medical wine syringe, and were then placed in two different types of laboratory tubes to perform the following tests, detection by AiA TOSOH.

1. disposable tube was used to separate the blood serum and retain it frozen (-20 c) for the evaluation of toxoplasmosis, the blood was put in the tube and centrifuged for 5 minutes at 3000 cycles per minute.
2. A gel tube was used to test the sex hormones LH, FSH, Prolactin, and Testosterone, the blood was deposited in a tube and centrifuged for 5 minutes at a speed of 3000 revolutions per minute.

## RESULTS AND DISCUSSION

### Luteinizing Hormone:

The current results show a significant increase in the LH hormone of  $6,000 \pm 2,188$  in patients infected with *Toxoplasma gondii* compared to a control group of  $5,100 \pm 1,738$ , and the results also show a hormonal increase of  $6,295 \pm 2,086$  in germ-free patients compared to a control group of  $5,100 \pm 1,738$ , the results also showed that, at the probability level,  $5,836 \pm 2,150$  of parasitic and infertile patients were not significantly different from  $5,100 \pm 1,738$  of controls (Table 1 and Fig. 1).

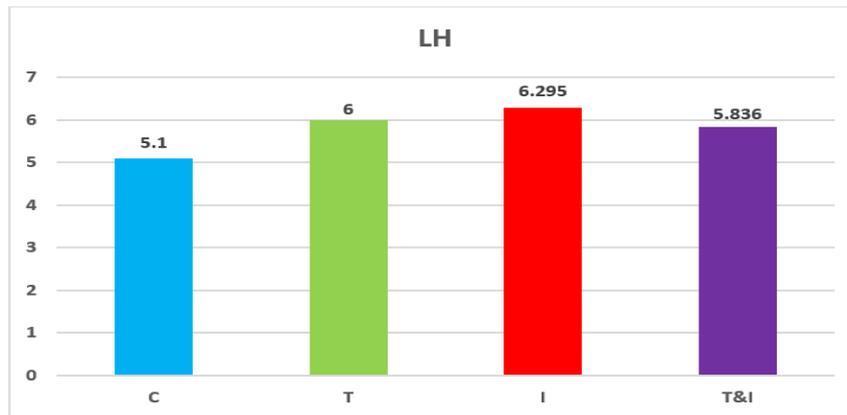
The pituitary gland produces luteinizing hormone (LH), which is released. The hypothalamus, a portion of the brain, produces this hormone. The release of gonadotropin hormones, including LH, is regulated by this gland. LH is crucial for maintaining the health of the reproductive system in the body. It also controls the release

of estrogen and progesterone from the ovaries in women and of testosterone from the testicles in men. LH also promotes the production of testosterone in the testicles. Sperm production and enhancement of male traits like beard hair growth and voice deepening are both brought about by testosterone. One of the reasons for elevated LH levels is (Klinefelter syndrome - a genetic disorder that affects men - damage to the testicle due to alcohol abuse and chemotherapy. (Babu *et al.*, 2004) observed increased levels of FSH and LH with lower testosterone concentration in toxoplasmosis patients, attributing this result to the main hypogonadism that manifested in these individuals, which is similar to the current observation. Lower urine LH levels, variations in the acid methylation pattern Nuclear of the testicular epigenome, and substantial differences in reproductive parameters were found in the Toxo-positive group (Toxo+) compared to the uninfected (Toxo) controls in the Toxo-positive group (Toxo+).

The first theory, according to (Terpsidis *et al.*, 2009; Dong *et al.*, 2004). suggests that *T. gondii* may activate the hypothalamic-pituitary-adrenal (HPA) stress axis and subsequently modulate the hypothalamic-pituitary-gonadal (HPG) axis, resulting in modified release of gonadotropins. Urine LH level was tested to address this hypothesis. As a result, this result conflicts with the present result. Since LH is one of the primary regulators of the development of germ cells, any non-physiological reduction in the amount of LH may result in a severe disruption of the entire spermatogenic process. Due to the fact that GnRH in the hypothalamus controls how much LH is produced from the pituitary gland (Vyas, 2013).

**Table 1:** LH Hormone in healthy people, patients with the *Toxoplasma gondii* parasite and sterile.

Parameter Groups	LH
	Mean ± SD
C	5.100±1.738b
T	6.000±2.188ab
I	6.295±2.086a
T&I	5.836±2.150ab



**Fig.1:** LH Hormone in healthy people, patients with the *Toxoplasma gondii* parasite and sterile.

**Follicle Stimulating Hormone:**

The present results show that, compared to controls, patients infected with *Toxoplasma* had  $7.237 \pm 2.404a$  and  $6.036 \pm 2.393b$  increases in FSH hormone, and the results also showed that, compared with controls, germ-free patients increased FSH hormone by  $7.264 \pm 2.609a$ . The control group was  $6.036 \pm 2.393b$ , and the results also showed that in the parasitic and infertile patients, the increase in FSH was  $7.264 \pm 2.609a$ , while the control group was  $6.036 \pm 2.393b$  (Table 2 and Fig. 2).

The causes for elevated FSH levels in males as we previously discussed, males may also experience high amounts of follicle-stimulating hormone. This increase is brought on by a variety of factors, all of which are connected to the testicles' declining capacity for function, most notably the following: (tests for radiation or trauma-related or traumatic injury damage) use of chemotherapy or alcohol Menopause and aging. Treatment for hormonal problems. using some prescription drugs, such as steroids and analgesic drugs. Having certain

medical diseases, such as type 2 diabetes, pituitary tumours, the AIDS-causing human immunodeficiency virus, etc. Genetic problems, most notably Klinefelter syndrome. (Dongmei *et al.*, 2005). verified that apoptosis of spermatozoa occurred in all different (fertile and infertile) male subjects, and there was a negative correlation between the rate of apoptosis and the ratio of the density of spermatozoa to the forward motility. The two issues—sperm apoptosis and male infertility—are closely related. So, spermatogenic cells may undergo apoptosis as a result of toxoplasma infection. Men with toxoplasmosis and toxoplasma-free controls showed the same levels of FSH. This result was inconsistent with the present finding by (Makker *et al.*, 2009). Elevated levels of FSH and LH were shown with lower levels of totally testosterone in men with *Toxoplasma gondii*. Thus, this result is in agreement with the present finding. (Babu *et al.*, 2004) found that toxoplasmosis patients had lower testosterone concentration and higher levels of FSH and LH; they ascribed this finding to the patients' underlying hypogonadism, which

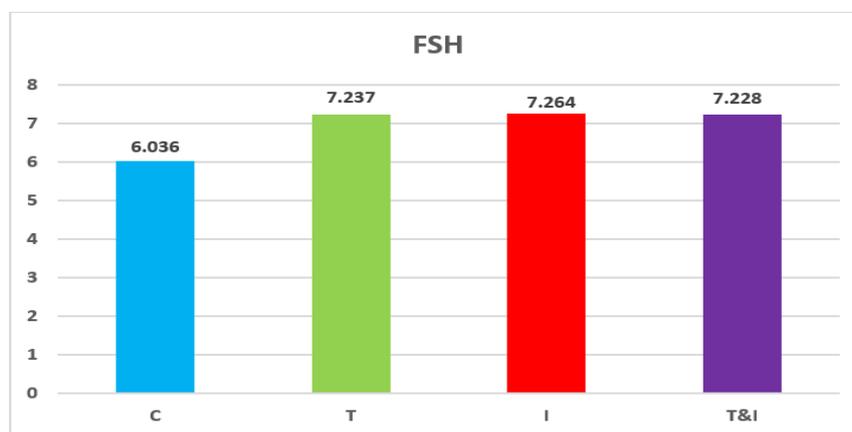
is consistent with the current data. FSH levels were often elevated in both disease processes. The current findings were different from those (AL-Karboli, 2011).

The reasons for height FSH levels in males as we previously discussed, males may also experience high amounts of follicle-stimulating hormone. This increase is brought on by a variety of factors, all of which are connected to the testicles' declining capacity for function, most notably the following: (tests for radiation or trauma-related or traumatic injury damage) use of chemotherapy or alcohol Menopause and aging. Treatment for hormonal problems. using some prescription drugs, such as steroids and analgesic drugs. Having certain medical diseases, such as type 2 diabetes, pituitary tumors, the AIDS-causing human immunodeficiency virus, etc. Genetic problems, most notably Klinefelter syndrome. (Dongmei *et al.*, 2005). verified that apoptosis of spermatozoa occurred in all different (fertile and infertile) male subjects,

and there was a negative correlation between the rate of apoptosis and the ratio of the density of spermatozoa to the forward motility. The two issues—sperm apoptosis and male infertility—are closely related. So, spermatogenic cells may undergo apoptosis as a result of toxoplasma infection. Men with toxoplasmosis and toxoplasma-free controls showed the same levels of FSH. This result was inconsistent with the present finding by (Makker *et al.*, 2009). Elevated levels of FSH and LH were shown with lower levels of totally testosterone in men with *Toxoplasma gondii*. Thus, this result is in agreement with the present finding. (Babu *et al.*, 2004) found that toxoplasmosis patients had lower testosterone concentration and higher levels of FSH and LH; they ascribed this finding to the patients' underlying hypogonadism, which is consistent with the current data. FSH levels were often elevated in both disease processes. The current findings were different from those (AL-Karboli, 2011).

**Table 2:** FSH Hormone in healthy people, patients with the *Toxoplasma gondii* parasite and sterile.

Parameter Groups	FSH
	Mean $\pm$ SD
C	6.036 $\pm$ 2.393b
T	7.237 $\pm$ 2.404a
I	7.264 $\pm$ 2.609a
T&I	7.228 $\pm$ 2.955a



**Fig.2:** FSH Hormone in healthy people, patients with the *Toxoplasma gondii* parasite and sterile.

**Prolactin Hormone:**

The present results showed in patients infected with *Toxoplasma gondii* an increase in PRL hormone  $13.150 \pm 4.392a$  compared to the control group  $9.195 \pm 3.273b$ , and the results also showed in sterile patients also an increase in the hormone PRL  $11.691 \pm 3.677a$  compared to the control group  $9.195 \pm 3.273b$ , and the results showed Also, in sterile and parasite patients, an increase in PRL hormone was  $12.976 \pm 4.722a$  compared to the control group,  $9.195 \pm 3.273b$  (Table 3 and Fig.3).

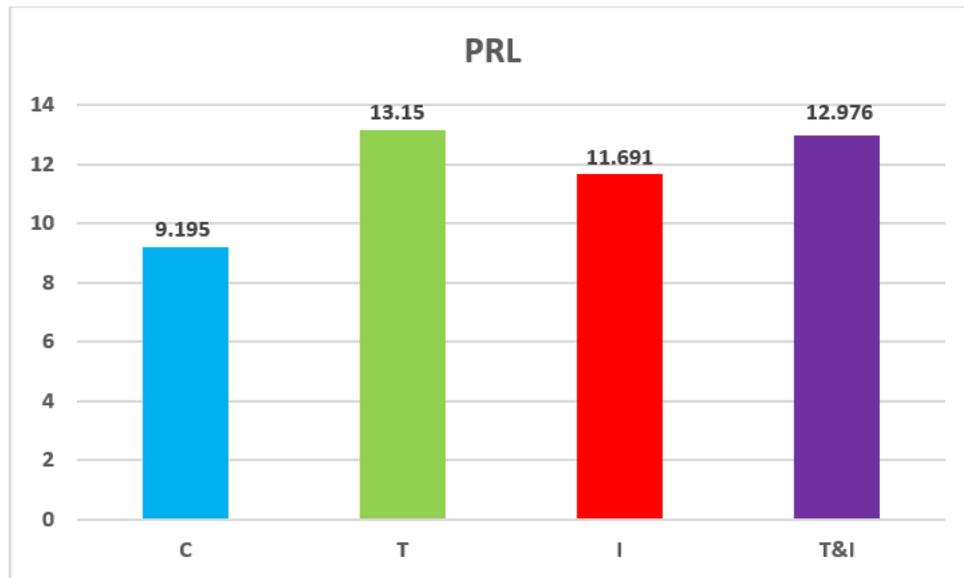
High prolactin, also known as high prolactin or hyperprolactinemia, is a medical disease in which men or women who are not pregnant have higher levels of the milk hormone. One of the hormones produced by the pituitary gland is prolactin, which aids in the development of breast tissue in females and the production of milk in females during pregnancy and after delivery. As a result, the level of the milk hormone is higher in pregnant or nursing women than in men and non-pregnant females. Prolactin is a hormone that causes men and women who are not pregnant to produce breast milk, and a high level of prolactin in the blood may be an indication that a prolactinoma, a tumour of the

pituitary gland, is present. There are numerous symptoms of high prolactin hormone that can affect an individual. There are a variety of causes for elevated milk hormone in males, as well as virgins, and mono, or non-pregnant women, including the following:

Kidney illness, hypothyroidism, Cushing's syndrome, pituitary adenoma, hypopituitarism, and some types of antidepressants, blood pressure drugs, or psychostimulants, revealed that pre-incubation of *Toxoplasma* tachyzoites with recombinant human prolactin (rhPRL) in vitro caused a significant reduction (up to 36.15%) in the reproductive abilities of the parasite. Prolactin (PRL) is one of the most important hormones involved in immune regulation in the host (Dzitko *et al.*, 2010), and the generated prolactin can stimulate antiparasitic activity They disagreed with the results of the current study and argued that the low number of infected cells in their investigation showed that the host cells were not sufficiently penetrated by the parasites to cause the suppression of reproduction. (Dzitko *et al.*, 2012).

**Table 3:** PRL Hormone in healthy people, patients with the *Toxoplasma gondii* parasite and sterile.

Parameter Groups	PRL
	Mean $\pm$ SD
C	<b>9.195<math>\pm</math>3.273b</b>
T	<b>13.150<math>\pm</math>4.392a</b>
I	<b>11.691<math>\pm</math>3.677a</b>
T&I	<b>12.976<math>\pm</math>4.722a</b>



**Fig. 3:** PRL Hormone in healthy people, patients with the *Toxoplasma gondii* parasite and sterile.

#### Testosterone Hormone:

The current findings demonstrated a decrease in TES hormone in *Toxoplasma gondii*-infected patients of 244.894 73.021b compared to the control group of 384.153 72.111a. They also demonstrated a decrease in TES hormone in sterile patients of 196.700 47.569d compared to the control group of 384.153 72.111a. Finally, they demonstrated a decrease in TES hormone in sterile.

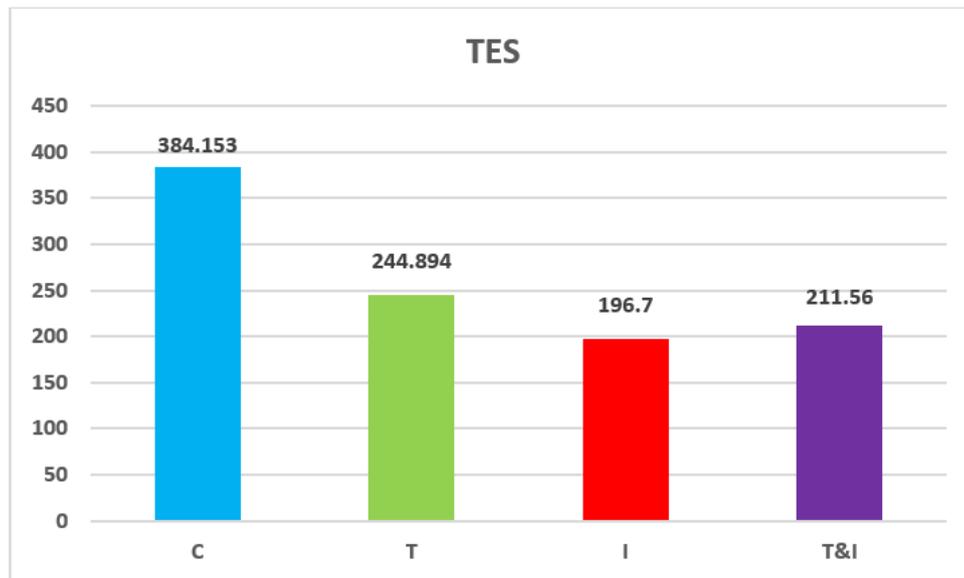
The findings of the present study did not support the findings reported (Afshari *et al.*, 2013), which indicated that rats experimentally infected with the parasite *Toxoplasma* experienced an increase in T hormone levels. This increase was noted for the presence of significant differences from the control group. However, numerous studies concurred with ours in observing a significant drop in serum hormone levels and in numerous samples from the experimental animals examined, as (Khaki *et al.*, 2011) noted a drop in T hormone levels in the blood serum of experimentally infected rats with the parasite with high significant differences compared to the control group, as confirmed (Abdoli *et al.*, 2012.) In his experimental

work, low levels of the T hormone were associated with substantial disparities. When mice were experimentally infected with the parasite *Toxoplasma*, (Rui *et al.*, 2009) noted that the levels of T hormone decreased. They emphasized that this infection causes the apoptosis of sperm-producing cells, particularly spermatocytes, which is associated with low levels of T hormone. Similarly, (Kankova *et al.*, 2011) recorded a decrease in T hormone levels in both males and females, highlighting the fact that there is numerous parasites.

T-hormone is the most significant of these hormones due to its significant involvement in cases of male infertility, and as a result, we concur that parasite infection causes cases of hormonal imbalance that may encompass numerous hormones that contribute to poor male reproductive efficiency (Dalimi & Abdoli, 2013). Regarding behavioural modifications, it was noted that some experimental animals lost their capacity to move and turn around in their cages, and we concurred with that observation (Torres *et al.*, 2013).

**Table 4:** Testosterone hormone in healthy people, patients with the *Toxoplasma gondii* parasite and sterile

Parameter Groups	TES
	Mean ± SD
C	384.153±72.111a
T	244.894±73.021b
I	196.700±47.569d
T&I	211.560±52.612cd



**Fig. 4:** Testosterone Hormone in healthy people, patients with the *Toxoplasma gondii* parasite and sterile

**REFERENCES**

AL-Karboli, A. R. (2011). *Immunological and physiological study of women infected with Toxoplasma* (Doctoral dissertation, Dissertation, College of Science, University of Baghdad).

AL-Khafaji, M. N. J., Muhamed, S. R., & Abdl-Kareem, S. F. (2020). Occurrence of Toxoplasmosis in Baquba City, Diyala, Iraq. *Tikrit Journal of Pure Science*, 25(1), 34-41.

Benedetto, N., Folgore, A., Romano-Carratelli, C., & Galdiero, F. (2001). Effects of cytokines and prolactin on the replication of *Toxoplasma gondii* in murine microglia. *European cytokine network*, 12(2), 348-58.

Boepple, P. A., Hayes, F. J., Dwyer, A. A., Raivio, T., Lee, H., Crowley Jr, W. F., & Pitteloud, N. (2008). Relative roles of inhibin B and sex steroids in the negative feedback regulation of follicle-stimulating hormone in men across the full spectrum of seminiferous epithelium function. *The Journal of clinical endocrinology & metabolism*, 93(5), 1809-1814.

Cenci-Goga, B. T., Rossitto, P. V., Sechi, P., McCrindle, C. M., & Cullor, J. S. (2011). *Toxoplasma* in animals, food, and humans: an old parasite of new concern. *Foodborne Pathogens and Disease*, 8(7), 751-762.

Dabritz, H. A., & Conrad, P. A. (2010). Cats and *Toxoplasma*: implications for public health. *Zoonoses and public health*, 57(1), 34-52.

Dalimi, A., & Abdoli, A. (2013). *Toxoplasma gondii* and male reproduction impairment: a new aspect of

- toxoplasmosis research. *Jundishapur* Rui, Y.; QiAng X. and MingZhe, J. (2009). *Journal of Microbiology*, 6(8).
- Dong, Q., Salva, A., Sottas, C. M., Niu, E., Holmes, M., & Hardy, M. P. (2004). Rapid glucocorticoid mediation of suppressed testosterone biosynthesis in male mice subjected to immobilization stress. *Journal of andrology*, 25(6), 973-981.
- Dongmei, X., Zhou, Y., & Diao, W. (2005). Preliminary investigation on relationship between spermatogenic cells apoptosis and infection of *Toxoplasma gondii* in male in fertility. *Chinese Journal of Schistomiasis*, 6, 1-20.
- Dzitko, K., Gatkowska, J., Płociński, P., Dziadek, B., & Długowska, H. (2010). The effect of prolactin (PRL) on the growth of *Toxoplasma gondii* tachyzoites in vitro. *Parasitology research*, 107(1), 199-204.
- Dzitko, K., Ławnicka, H., Gatkowska, J., Dziadek, B., Komorowski, J., & Długowska, H. (2012). Inhibitory effect of prolactin on *Toxoplasma* proliferation in peripheral blood mononuclear cells from patients with hyperprolactinemia. *Parasite immunology*, 34(6), 302-311.
- Khaki, A.; Farzadi, L. ; Ahmadi, S.; Ghadamkheir, E. ; Khaki, A. shojaee, S. and Sahizadeh, R.(2011). Recovery of spermatogenesis by *Allium cepa* in *Toxoplasma gondii* infected rats . *African Journal of Pharmacy and Pharmacology*, 5(7), pp.903-907.
- Makker, K., Agarwal, A., & Sharma, R. (2009). Oxidative stress & male infertility. *Indian Journal of Medical Research*, 129(4), 357.
- Effect of abnormal gonadal hormone secretion caused by *Toxoplasma gondii* in male fertility descent. *Modern Preventive Medicine*, 36(21), pp.4130-4135.
- Sadhnani, M., Swarna, M., Padmavathi, P., & Reddy, P. (2004). Evaluation of bacterial levels in different subgroups of infertile males. *Indian Journal of Clinical Biochemistry*, 19(1), pp.45-49.
- Shelly, S., Boaz, M., & Orbach, H. (2012). Prolactin and autoimmunity. *Autoimmunity reviews*, 11(6-7), A465-A470.
- Terpsidis, K. I., Papazahariadou, M. G., Taitzoglou, I. A., Papaioannou, N. G., Georgiadis, M. P., & Theodoridis, I. T. (2009). *Toxoplasma gondii*: reproductive parameters in experimentally infected male rats. *Experimental parasitology*, 121(3), 238-241.
- Torgerson, P. R., & Mastroiacovo, P. (2013). The global burden of congenital toxoplasmosis: a systematic review. *Bulletin of the World Health Organization*, 91, 501-508.
- Torres, M., Guiton, R., Lacroix-Lamandé, S., Ryffel, B., Leman, S., & Dimier-Poisson, I. (2013). MyD88 is crucial for the development of a protective CNS immune response to *Toxoplasma gondii* infection. *Journal of Neuroinflammation*, 10(1), 1-12.
- Vyas, A. (2013). Parasite-augmented mate choice and reduction in innate fear in rats infected by *Toxoplasma gondii*. *Journal of Experimental Biology*, 216(1), 120-126.