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FABP and Some Related Diabetic Parameters Among Adolescents with *Toxoplasma gondii*

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Abstract

Recently, it has been revealed that Toxoplasmosis may be associated with some factors related to type 2 diabetes, such as glucose, insulin, the Homeostatic Model Assessment for Insulin Resistant (HOMA-IR), and Fatty acid binding protein (FABP). Therefore, the current study aimed to specify how *Toxoplasma gondii* (*T.gondii*) infection affects glucose, insulin, HOMA-IR, and FABP among adolescents. From October to December 2022, this study was carried out at Al Madain Hospital in Baghdad. For a group of adolescents visiting the hospital, an ELISA test was performed to check their anti-*T.gondii* antibodies. Ninety adolescents were selected to participate in the study on the basis of this examination. They were divided into two groups: those who tested positive for the parasite (n = 45) and those who tested negative (n = 45), the control group. Measurements were performed on each participant's adolescence: weight for age percentile, weight for age z score, glucose, insulin, HOMA-IR, and FABP. Results showed that *the T. gondii* positive group had a significantly ($P<0.05$) higher weight for age percentile (86.84±2.66) and z score (1.29 ±0.11) versus the control group. Results also showed that those overweight adolescents with seropositive antibodies had significantly ($P<0.05$) greater levels of each glucose, insulin, HOMA-IR , and FABP than did a seronegative group of adolescents.

Keywords: adolescents, FABP, HOMA-IR, insulin, overweigh, Toxoplasmosis**.**

1. Introduction

Toxoplasma gondii (*T. gondii*), an intracellular tissue protozoan parasite that may infect both humans and animals, is the parasite that causes toxoplasmosis. A quarter of the world's population is thought to be at risk of contracting *Toxoplasma gondii*, which is known as one of the globally distributed parasites [1]. Toxoplasmosis is also one of the most common parasite infections in Iraq [2,3]. *T. gondii* has three infectious stages: a tachyzoite that divides quickly, a bradyzoite that develops slowly in tissue cysts, and an environmental stage known as a sporozoite that is shielded inside an oocyst [4]. Mammals and birds serve as intermediate hosts for *T. gondii*, while cats serve as the parasite's definitive host. Humans typically become infected by eating contaminated raw or undercooked meat, drinking contaminated water, or coming into contact with cats [5]. Rare mechanisms of *T.gondii* transmission include transplacental

transmission from infected mothers, organ donation, and blood transfusion from infected donors [5, 6].

Infection is seriously important for women when they get it for the first time during their pregnancy [7]. It is significant to note that *T. gondii* may affect the risk of obesity by altering the inflammatory pathways linked to weight gain [8]. By interfering with the host's lipid metabolism, as shown by tachyzoites, which are auxotrophic for low-density lipoprotein-cholesterol (LDL-C) from host cells via LDL receptors that induce a process of endocytosis, *T. gondii* may also have direct metabolic consequences other than weight gain [9, 10]. On the other hand, there have been cases of toxoplasmosis linked to diabetic people [11]. El araby *et al. (*2015 showed a significant elevation in the average percent of brain parasite load and IgM/IgG titers. All values exceed and are parallel to the progression of corticosteroids in the infected treated group compared to the infected-untreated group. In conclusion, long-term corticosteroid therapy may opportunistically increase the *T. gondii* brain parasite load and induce encephalitis in a latently infected murine model, imitating this serious condition in *T. gondii*-infected patients who received corticosteroid therapy [12]. It is unclear, nevertheless, whether an infection might raise the risk of diabetes or whether diabetes can raise the risk of parasitic infections [13]. Further research has revealed a connection between toxoplasmosis and elements linked to diabetes, including insulin, glucose, and the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) [11,13,14]. Several molecules are involved in the development of obesity and diabetes. One such compound is a fatty acid binding protein (FABP). FABP increases intracellular lipid accumulation, impairs glucose absorption in some cells, and induces insulin resistance [15]. FABPs are a class of 14–15 kDa lipocalin proteins that have a role in intracellular lipid homeostasis as well as metabolic and inflammatory pathways [16]. The liver, intestine, heart, adipose tissue, epidermis, ileum, brain, myelin, and testis are just a few of the body organs and tissues where they are widely expressed [17]. Recent years have seen a firm understanding that chronic systemic inflammation and obesity are related. Adipocyte tissues also contain T cells, macrophages, natural killer cells, and a large number of lymphocytes in the stromal vascular fraction. Metabolic and systemic inflammation are impacted by molecular and cellular alterations in adipocyte tissues in obese people [18]. However, the association of FABP with *T. gondii* infection in adolescents has only been reported in very little research [19]. It is necessary to investigate FABP levels and diabetic-related parameters in seropositive *T. gondii* adolescents compared to seronegative adolescents.

2. Materials and Methods

2.1 Individuals and study design

In Al-Madain Hospital in Baghdad, Iraq, this case control research was conducted from October to December of 2022. An ELISA test was run on a group of adolescents who were patients at this hospital to look for anti-*T. gondii* antibodies. The "IgG and IgM" enzyme immunoassay kit for anti-*T. gondii* IgG and IgM antibodies was used to analyze the serum samples they provided (Bioassay Technology Laboratory, China). The procedure was carried out in accordance with the manufacturer's instructions. Ninety participants in the study were chosen based on this test. Participants were split into two groups: those who tested positive for the parasite (n = 45) and those in the control group who tested negative (n = 45). Between the ages of 14 and 19, there were 45 (50%) boys and 45 (50%) girls. The study protocol was approved by the regional ethics committee of the College of Science at the University of Baghdad (Ref.:

CSEC/1022/0124). Each subject also gave their agreement to participate in the study, undergo all tests, and provide the required data.

2.2 Weight –for- age

A free-standing portable stadiometer and an electronic weighing scale were used to measure the height and weight of all participants. On the scale, each subject was asked to stand still while their weight was recorded. The individuals were not wearing shoes when they were weighed. Then, using the online calculator at:
https://www.msdmanuals.com/professional/multimedia/clinical-calculator/, the weight-for-age percentile and weight-for-age Z score were determined.

2.3 Blood collection and serum preparation

Each subject was venipunctured in order to get about 4-5 mL of blood. After that, 1–1.5 mL of the drawn blood sample was immediately added to EDTA tubes for anticoagulation, kept cool in a cooler, and sent within an hour to the lab for examination. The remaining blood sample was put into evacuated tubes with a clot gel activator and allowed to coagulate for about 30 minutes at room temperature. All samples underwent a 10-minute centrifugation process at 3000 rpm. Many portions of each serum sample were separated out. Each component was put into a sterile Eppendorf tube using a sterile micropipette for biochemical analysis.

2.4 Glucose

A glucose kit was used to analyze the glucose (Linear Chemicals, Spain) for all participants. The test was conducted in accordance with the manufacturer's guidelines.

2.5 Insulin and FABP

The Human Insulin and FABP sandwich enzyme-linked immunosorbent assay (ELISA) kits (Bioassay Technology Laboratory, China) were used for the quantitative measurement of insulin and FABP in both *T.gondii* infected group and the control group. The procedure was based on the manufacturer's instructions. All samples were analysed on a 96-well plate. After that, the plate was examined with an ELISA reader operating at 450 nm wavelength.

2.6 HOMA-IR calculation

The HOMA-IR was calculated after measuring insulin and glucose, as mentioned previously. The following formula was used for the calculation [20]: HOMA-IR=fasting insulin (microU/L) × fasting glucose (nmol/L)/22.5.

2.7 Statistical Analysis

The student T-test was used to compare the mean values of the weight-for-age percentile, weight-for-age Z score, glucose, HOMA-IR, insulin, and FABP between the *T. gondii* positive groups and the control group. A P-value of less than 0.05 was used to indicate statistical significance. The data were expressed as the mean and standard deviation (mean±SD).Results showed that both the weight-for-age percentile and weight-for-age Z score differed between the T. *gondii*-positive group and the control group. Weight-for-age percentile was significantly ($P<0.05$) higher (86.84±2.66) in those adolescents who had positive antibodies against *T. gondii* versus the low weight-for-age percentile in the control group (55.97±4.22) **Table 1and 2** illustrates the results of the weight-for-age Z score. They did not show any difference from the weight-for-age percentile, as the result of the weight-for-age Z score was significantly higher in *T. gondii*-positive groups (1.29 ±0.11) compared to the lower weight-for-age Z score among the control group (0.14 ±0.1).

Table 1.Weight for age percentile of *Toxoplasma gondii* positive group and the control group

Groups	Weight for age percentile (Mean ± SD)	T-test	P-value
Toxoplasma positive	86.84±2.66	6.1	<0.05
Toxoplasma negative	55.97±4.22		

Table 2. Weight for age Z-score of *Toxoplasma gondii* positive group and the control group

Groups	Weight for age Z-score (Mean ± SD)	T-test	P-value
Toxoplasma positive	1.29±0.11	6.05	<0.05
Toxoplasma negative	0.14±0.1		

The results showed that the glucose levels between the *T. gondii* positive group and the control group were significantly different (P<0.05) **Table 3**. The highest mean glucose levels were found in subjects who tested positive for anti-*T.gondii* antibodies (137.6 ± 0.6 mg/dL), compared to (118.07± 1.4 mg/dL) in the control group.

Table 3. Glucose levels of *Toxoplasma gondii* positive group and the control group

Groups	Glucose (Mean ± SD) mg/dL	T-test	P-value
Toxoplasma positive	137.6±0.6	12.6	<0.05
Toxoplasma negative	118.07±1.4		

A significant difference was also found between the two groups regarding insulin levels. The insulin level was significantly higher in the *T. gondii* positive group versus the control group, as the insulin levels were (2.15±0.09 ng/mL) and (1.56±0.07 ng/mL) respectively **Table 4**.

Table 4. Insulin levels of *Toxoplasma gondii* positive group and the control group

Groups	Insulin (Mean ± SD) ng/ml	T-test	P-value
Toxoplasma positive	2.15±0.09	5.06	<0.05
Toxoplasma negative	1.56±0.07		

The results also showed a significant difference between the *T. gondii* positive group and the control group regarding HOMA-IR level. It was found that HOMA-IR was significantly (P<0.05) higher among the *T. gondii* positive group compared to the control group, as the levels of HOMA-IR were (0.73±0.03) and (0.42±0.01) respectively **Table 5**.

Table 5. HOMA-IR levels of *Toxoplasma gondii* positive group and the control group

Groups	HOMA-IR(Mean ± SD)	T-test	P-value
Toxoplasma positive	0.73±0.03	7.52	<0.05
Toxoplasma negative	0.45 ±0.01		

FABP levels also varied between the two groups, as their levels were shown to be significantly higher in the *T. gondii* positive group compared to the control group. Their levels were (11 ±0.4 ng/mL) and (5.17±0.1 ng/mL) respectively **Table 6**.

Table 6. FABP levels of *Toxoplasma gondii* positive group and the control group

Groups	FABP (Mean ± SD) ng/mL	T-test	P-value
Toxoplasma positive	11 ±0.4	11.87	<0.05
Toxoplasma negative	5.17 ±0.1		

3. Discussion

The findings of the current study indicated that both the weight-for-age percentile and weight-for-age Z score could be correlated with *T.gondii* positive status. Animal studies have provided evidence that suggests a link between obesity and the survival of parasites. Researchers hypothesized that behavioral modifications, such as a high food intake associated with tissue cysts containing bradyzoites in the brain infected with *T. gondii* or directed dominant effects, such as modifications in hypothalamic functions, including appetite regulation, as a result of peripheral tissue inflammations, are responsible for increased weight [21]. Overweight and anti-*Toxoplasma* seropositive status were found to be related. This finding is consistent with that of Reeves et al. (2013), who revealed that individuals with positive *T. gondii* IgG results had nearly double the likelihood of being overweight compared to individuals with negative results [7]. However, other studies found no evidence of a link between toxoplasmosis and overweight [11, 22]. The results from the current investigation also corroborated *T. gondii*'s modulation of a few diabetes-related indicators, such as glucose, insulin, and HOMA-IR. Glucose levels increased significantly in those who had positive *T.gondii* antibodies. An animal study achieved by Modrek et al. (2015 revealed a significant rise in blood glucose levels in the *T. gondii*-infected diabetic group when compared to the uninfected diabetic group.Both insulin and HOMA-IR (insulin resistance) were shown to be higher in *the T.gondii*-positive group compared to the control group. Since the group of *T.gondii*-positive patients had a significant increase in weight, which is one of the most prominent risk factors for the development of diabetes, Inflammation and insulin resistance are exacerbated by the infiltration of classically activated inflammatory macrophages into adipose tissue [23]. Hyperinsulinemia and insulin resistance in humans are correlated with the development of inflammatory adipose tissue macrophages [23, 24]. The results of glucose and HOMA-IR in the current study agreed with the results of Moudgil *et al*. (2019), who showed that hyperglycemia and a high level of HOMA-IR are correlated with Toxoplasmosis [13]. The high insulin levels found in this study may be due to insulin resistance. In this instance, an excess of insulin does not result in low blood sugar. Hyperinsulinemia and insulin resistance factors can lead to persistent hyperglycemia [25]. Compared to the control group, the FABP level in the *Toxoplasma* positive group was considerably higher in this current study. Obese individuals' adipose tissue exhibits elevated FABP, and body mass index is favorably correlated with FABP's enhanced bloodstream release. FABP is an adipokine that has been associated with metabolic syndrome development, obesity, insulin resistance, and poor glucose metabolism [26]. According to the results of the current investigation, people who were seropositive for *T. gondii* had higher weight for age percentile and z score values than seronegative people. So, it makes sense that obese people with seropositive antibodies have greater FABP levels than do seronegative people, but much more study is still needed to support these claims for *T.gondii* and for other parasites [27-31].

4. Conclusion

Toxoplasma gondii seropositive status can be correlated with weight gain among adolescents, and changes in some diabetic related biochemical parameters may be involved.

Conflict of Interest

The authors declare that they have no conflicts of interest.

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Ethical Clearance

The study protocol was approved by local ethics committee of the College of Science - University of Baghdad (Ref.: CSEC/1022/0124) which were issued on October 15th 2022. Each subject also gave their agreement to participate in the study, undergo all tests, and provide the required data and this is consistent with the instructions of the Iraqi Ministry of Health and Environment.

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