



Alterations in adipokine and cytokine profiles during *Toxoplasma gondii* infection and their role in chronic inflammation

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Toxoplasma gondii infection can modulate host immune and metabolic pathways, leading to alterations in adipokine and cytokine profiles that may contribute to persistent inflammation and the development of chronic immunological responses. This study aimed to evaluate alterations in adipokine (leptin, adiponectin, and resistin) and cytokine (IL-6, TNF- α , and IL-10) profiles in patients with *T. gondii* infection and to investigate their potential role in the development of chronic inflammatory responses associated with the infection. This case-control study investigated alterations in adipokine and cytokine profiles in patients with *Toxoplasma gondii* infection and their role in chronic inflammation. The study was conducted between 23 May 2025 and 11 January 2026 in diagnostic laboratories in Thi-Qar Health Directorate, Al-Habbobi Teaching Hospital, and included 120 participants: 90 infected patients and 30 healthy controls matched by age and sex. Infection was confirmed using serological detection of anti-*Toxoplasma* IgG and IgM antibodies. Venous blood samples were collected, serum was separated, and adipokines (leptin, adiponectin, resistin) and cytokines (IL-6, TNF- α , IL-10) were measured using ELISA. The sociodemographic characteristics showed no significant differences between patients with *T. gondii* infection and controls in age, gender distribution, BMI, residence, or smoking status, indicating comparable baseline characteristics. Infected patients exhibited significantly higher leptin and resistin levels and lower adiponectin levels compared with controls. Acute infection showed higher leptin, resistin, IL-6, and TNF- α , while adiponectin and IL-10 were higher in chronic infection. Significant correlations were observed between adipokines and inflammatory cytokines. *Toxoplasma gondii* infection significantly alters adipokine and cytokine profiles, promoting a pro-inflammatory state characterized by increased leptin, resistin, IL-6, and TNF- α and reduced adiponectin. Such alterations are probably due to the activation of immunometabolic dysregulation and immune activation due to parasites and the inclusion of parasites as factors in the development of chronic inflammation.

Keywords: *Toxoplasma gondii* infection; adipokines; cytokines; chronic inflammation; immunometabolic regulation.

Introduction

Toxoplasma gondii is an intracellular protozoan obligate parasite of cats, capable of infecting a broad spectrum of warm-blooded animals such as human beings. It has been estimated that about one-third of the world population has been infected with *T. gondii* and as a result, toxoplasmosis is among the most common parasitic infections in the world. Human infection is usually caused by the intake of inadequately cooked meat, intake of food or water contaminated by oocysts dropped by infected cats or by the perineal infection of the mother and fetus. Even though a majority of the infections in immunocompetent individuals are asymptomatic, *T. gondii* may cause a lifelong chronic infection whereby the parasite is present as cysts in the body tissues in regions like the brain, skeletal muscles, and retina. Chronic inflammatory reactions and long term immunological changes in the host may occur because of this persistent infection.

The immune response to *T. gondii* is complicated and entails both the innate and adaptive immunity. At an early phase of infection, pattern recognition receptors allow the recognition of the parasites by innate immune cells, including macrophages, dendritic cells, and neutrophils, which activate the secretion of pro-inflammatory cytokines (Sasai et al., 2019). Interleukin-6 (IL-6) and tumor necrosis factor- α (TNF- α) are cytokines that are important when it comes to the reduction of parasite replication by increasing macrophages, and enhancing the production of T-helper-1 (Th1) immune responses. Nevertheless, a high level of these cytokines can lead to tissue destruction and prolonged inflammation. To offset this influence, the anti-inflammatory cytokines like interleukin-10 (IL-10) are generated to control immune responses and restrict host tissue damage in the case of infection (Brasil et al., 2017; Lima et al., 2019). Consequently, over the last several years there has been a growing focus on how adipose

tissue derived mediators, so called adipokines, play a role in immune regulation during infectious diseases. Adipokines are bioactive substances released by adipose tissue and are involved in the regulation of metabolism, immune regulation, and inflammation. The adipokines that have been studied most are adiponectin, resistin, and leptin. The molecules are known to play key roles as connectors between metabolism and immune activity and have been progressively identified to play critical roles in host responses during infection (López-Ortega et al., 2022; Trim et al., 2022).

Leptin is a pro-inflammatory adipokine that plays an essential role in immune activation. It promotes the proliferation and activation of immune cells, enhances macrophage phagocytic activity, and stimulates the production of pro-inflammatory cytokines. Elevated leptin levels have been associated with various inflammatory and infectious diseases, suggesting that leptin may contribute to the amplification of immune responses during pathogen invasion (Kiernan et al., 2021). In contrast, adiponectin generally exerts anti-inflammatory effects by suppressing the production of pro-inflammatory cytokines and promoting anti-inflammatory signaling pathways. Reduced adiponectin levels have been observed in several inflammatory conditions and may contribute to persistent immune activation. Resistin, another adipokine with pro-inflammatory properties, has been shown to stimulate the expression of inflammatory cytokines through activation of nuclear factor kappa-B (NF- κ B) signaling pathways (Monteiro et al., 2019).

Recent studies suggest that parasitic infections, including toxoplasmosis, can significantly alter adipokine secretion and cytokine production. The interaction between *T. gondii* and host immune cells may lead to changes in both metabolic and inflammatory signaling pathways (Salem et al., 2021). These alterations may play an important role in the development of chronic inflammation associated with

persistent infection. Chronic immune activation during toxoplasmosis has been implicated in various pathological conditions, including neurological disorders, ocular toxoplasmosis, and immune-mediated tissue damage (Saftawy et al., 2025).

Although it is becoming clear that infectious diseases have metabolic and immunological interrelations, there is inadequate understanding of the association of adipokines and cytokines in cases of *T. gondii* infection. Specifically, the pathways through which changes in adipokines can mediate regulation of inflammatory responses to acute and chronic toxoplasmosis are yet to be explored. The knowledge of such interactions can be quite useful in making decisions on the immunopathogenesis of toxoplasmosis and also in determining possible biomarkers of disease progression and target therapies (Ray et al., 2023; Nuszkiwicz et al., 2024).

Thus, the current research aimed to assess the changes in adipokine and cytokine profiles of the patients infected with *T. gondii* and investigate their possible contribution to the pathogenesis of chronic inflammatory reactions to this parasitic infection. Through the examination of major inflammatory and metabolic mediators, the study aims at enhancing our knowledge on the intricate immunometabolic interplay that takes place when toxoplasmosis develops.

Materials and methods

The research was carried out in accordance to the principles of the Declaration of Helsinki. The objectives of the study were explained to all of the participants and informed consent was signed by all the participants. Participants declared that they were confident that their personal data and lab findings would not be disclosed to any third parties and could only be utilized in the context of research.

The present case-control study was designed to examine the changes in the adipokine and cytokine profiles in the patients infected with *T. gondii* and also to determine its involvement in the chronic inflammatory processes. The study was conducted from 23 May 2025 until 11 January 2026 in a number of diagnostic laboratories and medical centers in Thi-Qar Health Directorate, Al-Habbobi Teaching Hospital. One hundred and twenty subjects were recruited in the study and they were separated into two groups. 90 patients who were diagnosed with *T. gondii* infection and the 30 clinically healthy people formed the first and second groups respectively. The control group was composed of people who visit the same medical facilities to check their health regularly and were as similar as possible to patients in terms of age and sex.

The patients recruited into the patient group were 18 years old or above and were proved by laboratory tests to be infected with *Toxoplasma gondii*. Exclusion criteria for the study included diagnosis with chronic inflammatory diseases and autoimmune disorders, malignancies and metabolic diseases, and other infectious diseases, as these factors could influence cytokine and adipokine levels. Further exclusion criteria were pregnancy, immunosuppressive therapy or anti-parasitic therapy within the last three months. The control group was composed of clinically healthy individuals who received negative serological responses indicating infection with *T. gondii* and none of whom had clinical signs of inflammatory or infectious diseases.

The diagnosis of *T. gondii* infection was established using serological testing for anti-*Toxoplasma gondii* antibodies. Blood samples were analyzed for the presence of specific IgM and IgG antibodies using commercially available serological diagnostic kits according to the manufacturer's instructions. Individuals with positive serological results were considered infected and included in the patient group, whereas individuals with negative serological results were included in the control group.

Approximately 5 mL of venous blood was collected from each participant under sterile conditions using disposable syringes. The collected blood samples were placed in plain tubes and allowed to clot at room temperature, after which they were centrifuged at 3000 rpm for 10 minutes to obtain serum. The separated serum samples were stored at -20°C until further laboratory analysis.

Serum levels of adipokines (leptin, adiponectin, and resistin) and cytokines interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF- α),

and interleukin-10 (IL-10) were measured using enzyme-linked immunosorbent assay (ELISA) kits according to the manufacturer's instructions. All assays were performed under standardized laboratory conditions and samples were analyzed in duplicate to ensure the accuracy and reliability of the results.

Statistical analysis was performed using SPSS software (version 26). Continuous variables were expressed as mean \pm standard deviation (SD). Differences between patients and controls were assessed using one-way analysis of variance (ANOVA). Pearson's correlation coefficient was used to measure associations between adipokines and cytokines. The P-value that was taken as significant was 0.05.

Results

Table 1 shows the sociodemographic background of the study population. The average age of the *Toxoplasma gondii* infection patients was 36.4 ± 9.2 years, similar to that of the control group (35.1 ± 8.7 years), and there was no significant difference between them ($P = 0.52$). Gender distribution did not differ significantly in either the patient group or the control group, whereby males constituted 45.6% and 46.7% respectively ($P = 0.91$). The body mass index (BMI) between the patients ($27.6 \pm 4.3 \text{ kg/m}^2$) and the controls ($26.9 \pm 3.8 \text{ kg/m}^2$) did not differ significantly ($P = 0.38$). Similarly, no significant differences between the two groups in terms of residence and smoking status were observed ($P > 0.05$), meaning that the two groups were similar in terms of sociodemographic features at the baseline.

Table 1
Sociodemographic characteristics of patients with *Toxoplasma gondii* infection and healthy controls

Variable	<i>Toxoplasma</i> patients (n = 90)	Controls (n = 30)	P-value
Age (mean \pm SD), years	36.4 ± 9.2	35.1 ± 8.7	0.52
Male, n (%)	41 (45.6%)	14 (46.7%)	0.91
Female, n (%)	49 (54.4%)	16 (53.3%)	–
BMI (mean \pm SD), kg/m^2	27.6 ± 4.3	26.9 ± 3.8	0.38
Urban residence, n (%)	55 (61.1%)	18 (60.0%)	0.92
Rural residence, n (%)	35 (38.9%)	12 (40.0%)	–
Smoking, n (%)	28 (31.1%)	9 (30.0%)	0.89

Table 2 shows the level of adipokines in the serum of the patients with *T. gondii* infection and healthy controls. The findings proved that the level of leptin in infected patients ($18.9 \pm 6.7 \text{ ng/mL}$) was much greater than in the control group ($12.4 \pm 4.8 \text{ ng/mL}$, $P = 0.001$). In a similar way, the levels of resistin were significantly higher in the patient group ($11.6 \pm 3.5 \text{ ng/mL}$) compared to the control group ($7.4 \pm 2.3 \text{ ng/mL}$, $P = 0.0001$). Conversely, the adiponectin level was also reduced considerably in the infected patients ($6.1 \pm 2.0 \text{ ug/mL}$) relative to the healthy individuals ($8.3 \pm 2.5 \text{ ug/mL}$, $P = 0.002$). These results suggest that there is an evident change in the profile of adipokines related to *T. gondii* infection.

Table 2
Serum adipokine levels in patients with *Toxoplasma gondii* infection and healthy controls (mean \pm SD)

Biomarker	Patients (n = 90)	Controls (n = 30)	P-value
Leptin, ng/mL	18.9 ± 6.7	12.4 ± 4.8	0.001
Adiponectin, $\mu\text{g/mL}$	6.1 ± 2.0	8.3 ± 2.5	0.002
Resistin, ng/mL	11.6 ± 3.5	7.4 ± 2.3	0.0001

Table 3 shows the serum cytokines of patients who have *T. gondii* infection in comparison to the healthy controls. The findings showed that the IL-6 level was very high in the infected patients ($14.8 \pm 5.2 \text{ pg/mL}$) compared to the control group ($6.7 \pm 2.4 \text{ pg/mL}$, $P < 0.001$). On the same note, the TNF- α levels were significantly higher in affected patients ($22.5 \pm 7.3 \text{ pg/mL}$) relative to controls ($11.3 \pm 4.1 \text{ pg/mL}$, $P < 0.001$). Moreover, the anti-inflammatory cytokine IL-10 was also significantly higher in the patients group ($9.1 \pm 3.0 \text{ pg/mL}$) in comparison with control ($5.8 \pm 2.1 \text{ pg/mL}$, $P = 0.004$). These results denote a triggered inflammatory and defense immune response with *Toxoplasma gondii* infection.

Table 3
Serum cytokine levels in patients with *Toxoplasma gondii* infection and healthy controls (mean ± SD)

Cytokine	Patients (n = 90)	Controls (n = 30)	P-value
IL-6, pg/mL	14.8 ± 5.2	6.7 ± 2.4	<0.001
TNF-α, pg/mL	22.5 ± 7.3	11.3 ± 4.1	<0.001
IL-10, pg/mL	9.1 ± 3.0	5.8 ± 2.1	0.004

The results show that the levels of adipokine and cytokines differ in patients with acute and chronic infections of *T. gondii* as indicated in Table 4. The findings yielded more significant levels of leptin and resistin in patients with acute infection (21.2 ± 6.5 and 12.8 ± 3.7 ng/mL, respectively) than in chronic infection patients (17.4 ± 6.2 and 10.8 ± 3.1 ng/mL, respectively). On the same note, pro-inflammatory cytokines, IL-6 and TNF-α were also found to be significantly increased in the acute infection group (17.6 ± 4.8 and 25.4 ± 6.9 pg/mL), as compared to the chronic infection group (13.1 ± 4.9 and 20.4 ± 6.8 pg/mL, P ≤ 0.003). Conversely, the level of adiponectin and IL-10 was more pronounced in the chronic infection patients, implying greater control of anti-inflammatory processes in the chronic stage of the disease.

Table 4
Comparison of adipokine and cytokine levels between acute and chronic *Toxoplasma gondii* infection patients (mean ± SD)

Biomarker	Acute infection (n = 35)	Chronic infection (n = 55)	P-value
Leptin, ng/mL	21.2 ± 6.5	17.4 ± 6.2	0.02
Adiponectin, μg/mL	5.4 ± 1.8	6.6 ± 2.1	0.03
Resistin, ng/mL	12.8 ± 3.7	10.8 ± 3.1	0.04
IL-6, pg/mL	17.6 ± 4.8	13.1 ± 4.9	0.001
TNF-α, pg/mL	25.4 ± 6.9	20.4 ± 6.8	0.003
IL-10, pg/mL	7.9 ± 2.7	10.0 ± 3.1	0.01

The correlation analysis between adipokines and pro-inflammatory cytokines in patients with *T. gondii* infection is expressed in Table 5. The findings showed that there were positive significant relationships between leptin and IL-6 (r = 0.58, P = 0.001) and leptin and TNF-α (r = 0.49, P = 0.002). On the same note, resistin was found to be positively correlated with IL-6 (r = 0.62, P < 0.001) and TNF-α (r = 0.55, P < 0.001). Conversely, adiponectin showed a strong negative relationship with IL-6 (r = -0.41, P = 0.004) and TNF-α (r = -0.38, P = 0.006). Also, there was a significant positive correlation between IL-6 and TNF-α (r = 0.67, P < 0.001), which demonstrated the simultaneous stimulation of inflammatory processes in the course of *T. gondii* infection.

Table 5
Correlation between adipokines and pro-inflammatory cytokines in patients with *Toxoplasma gondii* infection

Variables	r	P-value
Leptin vs IL-6	0.58	<0.001
Leptin vs TNF-α	0.49	0.002
Resistin vs IL-6	0.62	<0.001
Resistin vs TNF-α	0.55	<0.001
Adiponectin vs IL-6	-0.41	0.004
Adiponectin vs TNF-α	-0.38	0.006
IL-6 vs TNF-α	0.67	<0.001

Discussion

The current paper addresses the changes in the profiles of adipokines and cytokines as they occur during *T. gondii* infection and their possible involvement in the development of chronic inflammatory reactions. The results show that there are numerous differences in the inflammatory biomarkers between infected individuals and healthy controls, which implies that *T. gondii* infection could cause systemic impacts of immune-metabolic changes that might lead to chronic inflammation. The sociodemographic data did not differ significantly in patients and controls, which indicates that most of the differences in biomarker levels could have been connected with the infection status

and not demographic and lifestyle aspects. This has also been noted in other studies in the past where demographic factors were not significant in determining immunological responses to *T. gondii* infection (Hussein et al., 2020; Vueba et al., 2020).

The results of the present study revealed significantly elevated serum levels of leptin and resistin in infected patients compared with healthy individuals, while adiponectin levels were significantly decreased. These findings are consistent with those of previous studies demonstrating that adipokines are closely involved in immune regulation during parasitic infections. Leptin, a pro-inflammatory adipokine, plays a crucial role in the activation of innate and adaptive immune responses by promoting macrophage activation and T-helper-1 (Th1) immune responses, which are essential for controlling intracellular pathogens such as *T. gondii* (Hassan et al., 2020). Increased leptin levels observed in the current study may therefore reflect activation of immune mechanisms aimed at limiting parasite replication. Similar elevations of leptin in parasitic infections have been reported by several researchers who demonstrated that leptin acts as an immunomodulatory hormone linking nutritional status and immune activation (Salem et al., 2021).

Resistin levels were also significantly higher in infected individuals. Resistin is known to stimulate the production of pro-inflammatory cytokines through activation of nuclear factor kappa-B (NF-κB) signaling pathways, thereby contributing to chronic inflammatory states. Previous studies have suggested that elevated resistin levels may be associated with increased macrophage activation and inflammatory cytokine production during infectious diseases (Hassanein et al., 2024). The increase in resistin levels observed in the present study may therefore reflect an inflammatory response induced by parasite invasion and host immune activation (Al-Halbousi et al., 2024).

Conversely, adiponectin levels were significantly reduced in infected patients. Adiponectin is widely recognized as an anti-inflammatory adipokine that suppresses the production of pro-inflammatory cytokines such as TNF-α and IL-6 while enhancing anti-inflammatory signaling pathways. Decreased adiponectin levels during infection may therefore promote a pro-inflammatory environment that facilitates immune responses against intracellular pathogens (Iskandar et al., 2016). Similar reductions in adiponectin levels during inflammatory conditions have been documented in several immunometabolic studies (Muwafaq et al., 2025). The identified reduction of adiponectin can thus be the cause of the chronic inflammation state related to long-term *T. gondii* infection.

The present research showed in addition to adipokines, substantially higher levels of pro-inflammatory cytokines, such as IL-6 and TNF-α, in patients with the infection than in controls. These cytokines play important roles as mediators of host defense against intracellular parasites. IL-6 is extremely important in causing acute-phase responses and cytokine-mediated control of the immune cell activation, and TNF-α is activated by macrophages and assists in the clearance of parasites (El-Sherbini et al., 2019). High concentrations of these cytokines in cases of toxoplasmosis have been already reported and are believed to be key elements of defence mechanism against the parasite (Santose et al., 2021). These elevated levels in the current study are therefore probably due to the activation of the host immunity to counter proliferation of the parasites.

Interestingly, the anti-inflammatory cytokine IL-10 was also elevated in infected individuals. IL-10 plays an essential regulatory role by limiting excessive inflammatory responses that may cause tissue damage during infection. The increase in IL-10 levels may represent a compensatory mechanism to balance pro-inflammatory cytokine activity and prevent excessive immune-mediated pathology (Raouf-Rahmati et al., 2021). Similar patterns of concurrent elevation of both pro-inflammatory and regulatory cytokines have been reported in chronic toxoplasmosis, suggesting the existence of a complex immune regulatory network during infection (Salomão Lopes et al., 2025).

The comparison between acute and chronic infection stages revealed that IL-6, and TNF-α were significantly elevated during acute infection and IL-10 was comparatively elevated during chronic infection. Such results are in line with the established immunopathogenesis of toxoplasmosis. The acute stage is characterized by a high pro-inf-

lammatory response developed by the immune system in order to contain the fast replication of the parasite. The regulatory cytokines including IL-10 take preeminence as the infection advances to the chronic stage to keep down tissue devastation as well as control immune homeostasis (Marchioro et al., 2018; Marino Ana et al., 2020).

Correlation analysis also showed that there were strong positive relationships between pro-inflammatory adipokines and cytokines. There were significant positive correlations between leptin and resistin and IL-6, and TNF- α and this indicates that adipokines could be significant in intensifying the effects of inflammatory responses in infection. The connections between adipokines and inflammatory cytokines have been observed to have similar associations with other chronic inflammatory disorders and parasitic infections (Ansari-Lari et al., 2024). Adiponectin on the other hand showed negative results with inflammatory cytokines, which favors its reported anti-inflammatory effects (Iskandar et al., 2016).

Despite the fact that the current results are broadly consistent with the already published results, there can be a number of different discrepancies between various studies. These differences can be explained by the differences in study populations, genetic backgrounds, nutritional status, the variability of parasite strain, and methodology employed in the detection of the biomarkers. As an illustration, the response of cytokines differed in the different geographic populations because of the differences in the host immune genetics and the exposure to the environment (De Haan et al., 2021). Also, the stage of disease, parasite load and host metabolic status could play a role in adipokine and cytokine expression patterns.

Conclusion

The findings of the present study, in general, assist the idea that *T. gondii* infection causes the significant change in the adipokine and cytokine phenotype, which is a part of multifaceted immunometabolic network, which controls chronic inflammation. These data point to the possibility of using adipokines as one of the biomarkers of the inflammation processes in the case of toxoplasmosis and can provide answers to questions regarding the processes of parasite-induced chronic inflammation.

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