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Impact of *Toxoplasma gondii* infection on adipokine secretion and the regulation of inflammatory cytokines in the host

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Toxoplasma gondii infection disrupts host metabolic and immune homeostasis by altering adipokine secretion and modulating the balance between pro- and anti-inflammatory cytokines, potentially contributing to systemic inflammation and metabolic dysfunction. This study aimed to investigate the effects of *T. gondii* infection on host adipokine secretion and inflammatory cytokine regulation. A six-month case-control study (January–June 2025) was conducted at Al-Habbobi Teaching Hospital, involving 100 *T. gondii* seropositive patients and 50 matched healthy controls aged 18–60. Exclusion criteria included pregnancy, immunosuppressive or lipid-lowering therapy, and chronic inflammatory or autoimmune diseases. Following overnight fasting, 5 mL venous blood samples were collected, serum separated, and stored at –20 °C. Levels of adipokines (leptin, adiponectin, resistin) and cytokines (TNF-α, IL-6, CRP, IL-10, TGF-β) were measured using ELISA kits (Bio-Techne, USA). No significant differences in age, sex, residence, or smoking status were observed between groups ($P > 0.05$), but BMI was significantly higher in patients ($P = 0.003$). Patients exhibited significantly increased leptin and resistin ($P < 0.001$) and decreased adiponectin ($P < 0.001$). Pro-inflammatory cytokines TNF-α, IL-6, and CRP were elevated ($P < 0.001$), whereas anti-inflammatory IL-10 and TGF-β were reduced ($P < 0.001$). There were strong positive relationships between leptin- TNF- 2, adiponectin- IL-10, and resistin-CRP ($P < 0.001$). The infection of *T. gondii* greatly changes the secretion of adipokines and cytokine balance, which favors systemic inflammation and the inability to regulate the immune system, presumably through the effect of parasite-induced immune and metabolic alterations.

Keywords: *Toxoplasma gondii*; adipokines; cytokines; inflammation; immune response; ELISA.

Introduction

The parasitic infections are a great burden to world health. In order to survive and sustain a long-term well-being, the immune response needs to be effectively regulated. The success of this type of infection is heavily dominated by the complex interactions between the parasite eradication and immunopathology of the host (Kaminsky et al., 2025). *Toxoplasma gondii* is an obligatory intracellular protozoan parasite, which infects almost all warm-blooded animals, and is estimated to infect one-third of the human population worldwide. Although acute infection is normally taken care by a strong cell-mediated immunity in immunocompetent hosts, the parasite causes chronic and life-long infections which are characterized by the presence of cysts containing bradyzoa in the many tissues but most especially the brain and muscle. It depends on a very fine line of immune regulation to ensure that this chronic condition can be sustained by the host (Al-Malki et al., 2021; Negesa et al., 2024).

The immune response to *T. gondii* may be considered a good example of such a complicated balance. The innate immune system is the first to identify the infection, with dendritic cells and macrophages being important to identifying the presence of the parasite and inducing a robust pro-inflammatory response (Khan et al., 2022). This triggers the generation of major pro-inflammatory cytokines, including TNF-alpha and IL-6, which are necessary in the regulation of parasite replication. Nevertheless, a pro-inflammatory response that increases tissue damage and disease exacerbation can be caused by an uncontrolled response or dysregulation (Sousa et al., 2021; Qin et al., 2025). In a bid to counter this reaction, the immune system also secretes anti-inflammatory cytokines, including interleukin-10 (IL-10) and transforming growth factor-beta (TGF- β) that act by suppressing the inflammatory process, thus avoiding immunopathological damage. Thus, tight control between the pro- and anti-inflammatory cytokines determines survival after *T. gondii* infection (Mahmoudzadeh et al., 2021; Alsailawi et al., 2022).

But contemporary facts indicate that this immunological battle ground is not limited to its conventional areas of immune cells. Having been considered only as a store of energy, adipose tissue is now considered as a significant endocrine organ, which combines both metabolic and immune functions within the body. The adipocytes and other cells of adipose tissue synthesize protein hormones (adipokines), which have the ability to act at long distances (Wawrzkiewicz-Jałowieckaet al., 2021). Fat cells release various molecules including leptin, adiponectin, and resistin that help in determining the metabolic state to inflammatory responses. Specifically, obesity leads to the overproduction of adipokines (proinflammatory adipokine) (e.g., leptin, the most prominent pro-inflammatory adipokine that may trigger macrophage activation and T cell proliferation), which boost immune responses (López-Ortega et al., 2022). Conversely, adiponectin is a non-inflammatory agent and considered to suppress the activity of macrophages, in addition to increasing the sensitivity of insulin. Conversely, resistin is also a significant pro-inflammatory and has been found to cause insulin resistance together with general inflammation (Al-Mansoori et al., 2022).

Therefore, there is an interdependent relationship between the adipose tissue and immunology, which implies that the metabolic adaption of the host or the parasite to infection may have significant effects on the host control of infection, and the reverse (Salem et al., 2021). Although rather little is known about the direct influence of *T. gondii* on the host adipokine secretion and the possible effects on the cytokine balance that regulates the inflammatory reactions, existing evidence points towards the relationship between the mentioned elements. The same is also known to encourage an environment of low-grade, subclinical inflammation an inflammatory mechanism that is considered a major cause of adipokine dysregulation (Mustafa et al.,

2024). On the other hand, defective levels of adipokines, especially those linked with obesity and metabolic syndrome, can impair the ability of the host to fight the parasite, and result in a lack of control over the parasite or more severe disease progression. This leads to a very interesting hypothesis, *T. gondii* infection can change the metabolic physiology of the host, therefore conditioning a change in the secretion of adipokines which destabilizes the fine balance of cytokines required to effectively combat its invasion (Barthelemy et al., 2023; Sinton et al., 2025).

Since adipokines have important roles in metabolic health and immune regulation, and these two aspects have a large overlap, it is necessary to fully understand how the parasitic infection *T. gondii* influences discrete adipokine signaling pathways. In the prior research, there has been a sharp division on cytokine responses or metabolic changes. There exists a great gap in the literature studying how the systemic effect of *T. gondii* infection on the whole adipokine profile varies and the association of these changes with the inflammatory cytokine response of the host (Oliveira-Scussel et al., 2022). These changes may be closely linked to the balance between pro-inflammatory cytokines (TNF-α, IL-6) and anti-inflammatory cytokines (IL-10, TGF-β) secreted by host immune cells during *T. gondii* infection. Therefore, the main aim of this study was to investigate whether and how chronic *T. gondii* infection influences host production of key adipokines – leptin, adiponectin, and resistin and whether these changes are associated with alterations in the levels of selected cytokines (Ewald et al., 2024; Nuszkiewicz et al., 2024).

The aim of this study is to advance understanding of the complex pathogenesis of toxoplasmosis by identifying novel metabolic and immune targets within the host that may inform the development of effective therapeutic interventions.

Material and methods

The study was ethically approved by the Human Ethics Committee of the Al-Habbobi Teaching Hospital based on the Declaration of Helsinki under approval number 558, which was dated January 1, 2025. All the subjects received detailed instructions on the procedures of the study and signed informed consent. All patient data was kept confidential in the course of the research.

A case-control study was conducted over a six-month period, from January 2025 to June 2025, at Al-Habbobi Teaching Hospital. The study enrolled 100 patients seropositive for *T. gondii* infection and 50 age- and sex-matched healthy controls. Eligible participants were adults aged 18 to 60 years with confirmed seropositivity to *T. gondii*, verified through standardized serological assays. Participants were excluded if they met any of the following criteria: pregnancy or lactation; current or recent (within the past three months) use of immunosuppressive drugs (e.g., corticosteroids, biologics) or lipid-lowering medications; presence of chronic inflammatory diseases (such as rheumatoid arthritis or inflammatory bowel disease), diagnosed autoimmune disorders, metabolic syndrome as defined by established clinical guidelines (including central obesity, insulin resistance, hypertension, and dyslipidemia), or any acute infections at the time of enrollment. Following an overnight fast of at least 8 hours, 5 mL of venous blood was aseptically collected from each participant. Samples were centrifuged at 3000 rpm for 10 minutes to separate serum, which was then stored at –20 °C until analysis. Serum adipokines (leptin, adiponectin, resistin) and cytokines (TNF-α, IL-6, CRP, IL-10, TGF-β) were quantified using commercial enzyme-linked immunosorbent assay (ELISA) kits following the manufacturer's instructions (Bio-Techne, USA). The study protocol was reviewed and approved by the Institutional Review Board of Al-Habbobi Teaching Hospital. Written informed consent was obtained from all participants prior to enrollment.

The analysis of data was conducted with the help of SPSS version 26. Quantitative variables were reported in form of mean SD and categorical data in form of frequencies and percentages. The independent t-test was used to compare the groups that had normally distributed variables and the Mann-Whitney U test was used to compare the non-normally distributed variables whereas categorical variables were compared with Chi-square test. Pearson's correlation coefficient was used to test the correlation of biomarkers. The statistically significant P-value was taken to be 0.05.

Results

The results of the statistical analysis showed that there was a high demographic match between the patient group and the healthy group, which strengthens the validity of the comparison between the two groups. Variables such as age (37.8 ± 8.4 vs. 36.2 ± 7.9; P = 0.42), gender (48% male vs. 50% male; P = 0.81), place of residence (60% urban vs. 62%; P = 0.77), and smoking status (30% vs. 26%; P = 0.58) did not show statistically significant differences. However, a statistically significant difference was observed in body mass index (BMI), which was significantly higher on average in the patient group (28.4 ± 3.6 kg/m²) compared to the healthy group (25.9 ± 3.1 kg/m²), with a probability value of P = 0.003.

Table 1
A comparative analysis of patient and control groups

Variable	Patients (n = 100)	Controls (n = 50)	P-value
Age, years	37.8 ± 8.4	36.2 ± 7.9	0.42
Gender, male (%)	48 (48%)	25 (50%)	0.81
BMI, kg/m²	28.4 ± 3.6	25.9 ± 3.1	0.003
Residence, Urban %	60	62	0.77
Smoking status, %	30	26	0.58

The two groups were compared and the comparison showed a high level of statistical significance in the difference in the serum levels of all the adipokines being measured. The average serum leptin concentration of the patient population (18.7 ± 4.8 ng/mL) was significantly more elevated than that of the control patients who were healthy (12.3 ± 3.5 ng/mL; P = 0.001). On the same note, serum resistin levels were significantly higher in patients (9.2 ± 3.0 ng/mL) as compared to controls (5.1 ± 1.8 ng/mL; P < 0.001). On the other hand, the patients had much lower levels of adiponectin than the healthy controls (6.4 ± 2.1 and 9.8 ± 2.7 µg/mL respectively, P < 0.001). Taken together, the findings indicate that *Toxoplasma* infection alters the adipokine homeostasis leading to a pro-inflammatory signature of elevated leptin and resistin and depressed anti-inflammatory adiponectin.

Table 2
A comparison of leptin, adiponectin, and resistin concentrations

Adipokine	Patients (mean ± SD)	Controls (mean ± SD)	P-value
Leptin, ng/mL	18.7 ± 4.8	12.3 ± 3.5	<0.001
Adiponectin, µg/mL	6.4 ± 2.1	9.8 ± 2.7	<0.001
Resistin, ng/mL	9.2 ± 3.0	5.1 ± 1.8	<0.001

The results showed a high level of statistical significance in the differences in levels of inflammatory biomarkers between the two groups. There was a significant difference in serum levels of tumor necrosis factor-alpha (TNF-024) between the patients (32.5 ± 9.6 pg/mL) and healthy controls (14.2 ± 4.3 pg/mL; P < 0.001). Similarly, the interleukin-6 (IL-6) was found to be much higher in the patients (18.6 ± 6.1 pg/mL) compared to the controls (6.8 ± 2.5 pg/mL; P < 0.001). Also, the level of C-reactive protein (CRP) was significantly elevated in patients (14.8 ± 5.2 mg/L), compared to healthy people (3.9 ± 1.7 mg/L; P < 0.001). Together, these results suggest that the infection of toxoplasmosis causes a strong systemic inflammatory reaction, which is supported by the significant increase in the levels of major biomarkers of inflammation.

Table 3
A comparison of TNF-α, IL-6, and CRP concentrations in patients and controls

Biomarker	Patients (mean ± SD)	Controls (mean ± SD)	P-value
TNF-α, pg/mL	32.5 ± 9.6	14.2 ± 4.3	<0.001
IL-6, pg/mL	18.6 ± 6.1	6.8 ± 2.5	<0.001
CRP, mg/L	14.8 ± 5.2	3.9 ± 1.7	<0.001

The results of the analysis have shown a very statistically significant decrease in the level of anti-inflammatory cytokines in the patient group in comparison with healthy controls. The mean levels of serum interleukin-10 (IL-10) in the patients (6.3 ± 2.1 pg/mL) were also considerably smaller as compared to the control group (10.8 ± 3.2 pg/mL; $P < 0.001$). Likewise, there was a significant decrease in transforming growth factor-beta (TGF-B) levels of patients (17.4 ± 4.6 ng/mL) compared to healthy people (22.9 ± 5.1 ng/mL; $P < 0.001$). These results point to the fact that *Toxoplasma* infection is paired with the breakdown of immunoregulation processes, which is manifested by the degradation of the main anti-inflammatory mediators, which contributes to the enhancement of the systemic inflammatory process.

Table 4
A comparison of IL-10 and TGF-β concentrations in patients and controls

Biomarker	Patients (mean ± SD)	Controls (mean ± SD)	P-value
IL-10, pg/mL	6.3 ± 2.1	10.8 ± 3.2	<0.001
TGF-β, ng/mL	17.4 ± 4.6	22.9 ± 5.1	<0.001

Analysis of statistical correlation revealed that there were strong positive correlations between the chosen adipokines and the inflammatory cytokines in the patient group. It was observed that tumor necrosis factor-alpha (TNF-alpha) and leptin levels have a significant positive correlation ($r = 0.68$, $P = 0.001$). Moreover, adiponectin was also positively correlated with interleukin-10 (IL-10) ($r = 0.62$, $P < 0.001$). Additionally, the levels of resistin were positively related to C-reactive protein (CRP) ($r = 0.59$, $P < 0.001$). Together, these results indicate that adipokines are not passively modified by infection, but are involved in the regulation of the inflammatory response as indicated in their strong relationships with major cytokines and inflammatory situations.

Table 5
Pearson's correlation coefficients (r) for key biomarkers

Biomarker	Biomarker	Correlation (r)	P-value
Leptin	TNF-α	0.68	<0.001
Adiponectin	IL-10	0.62	<0.001
Resistin	CRP	0.59	<0.001

Discussion

The sociodemographic analysis (Table 1) showed that there was no difference in age, sex, residence, and smoking status between the patient and the control groups but the Body Mass Index (BMI) difference was statistically significant. The average BMI of *T. gondii* infected patients was significantly greater than the control group ($P = 0.003$). This observation is interesting because it highlights a potential correlation between obesity and the *T. gondii* infection, which is an association that is rather poorly comprehended (Cuffey et al., 2021).

The factors behind such a relationship are possibly multiple. A more conducive environment toward parasitic persistence might be instigated by chronic low-grade inflammation which is typical of obesity. Alternatively, obesity could also modify immune response whereby the capacity to develop effective Th1-mediated immunity against parasites including *T. spiralis* would be affected. As a result, it is possible that not only do obese people have weakened defenses but also that they become more vulnerable to the pathological influence of the said parasites once infected (Maisarah et al., 2024).

Sustained infection, on the other hand, can also directly generate metabolic alterations, predisposing the patients to weight gain and high BMI. This is in line with other studies that indicate relationships between parasitic infections and metabolic changes, although the causal relationship needs to be made certain through research (Olarinde et al., 2022). The biggest changes were the substantial changes in the serum adipokine levels (Table 2). The most significant alterations were the increased concentrations of the pro-inflammatory adipokines of leptin and resistin in the patient group ($P < 0.001$). Such outcomes are in line with the fact that these hormones are known to act as inflammatory mediators. Only leptin, a cytokine-like hormone that has been proven to drive macrophages and induce differentiation of Th1 cells, was studied using cellular response assays, and is required to control intracellular pathogens like *T. gondii* (Abbas et al., 2025).

Collectively, it can be seen that the observed changes in resistin (a pro-inflammatory adipokine) that occur simultaneously also support our hypothesis that *T. gondii* infection causes a systemic inflammatory condition. This observation is consistent with our earlier study which showed high concentrations of leptin and resistin in most chronic inflammatory and infectious diseases (Zhu et al., 2024). Nevertheless, the adiponectin, a highly acknowledged anti-inflammatory adipokine, was observed to be strongly depleted in the patients relative to the controls ($P < 0.001$). This has been in line with inflammatory states because it has been previously known that pro-inflammatory cues inhibit the production of adiponectin. Leptin/resistin vs adiponectin has been found to be an inversely correlated classical indicator of metabolic and inflammatory dysregulation, and other chronic infections have been reported to show the same (Hamad et al., 2024; Gaskell et al., 2025). This seemingly malfunctioning adipokine release was manifested by an intense skew in the host cytokine phenotype (Tables 3 and 4). TNF-a, IL-6, and CRP which are pro-inflammatory cytokines were also remarkably increased ($P < 0.001$). These cytokines are vital in that they generate signaling molecules that control inflammation and recruit the body immune effector cell or killer cells to attack the parasite.

These high concentrations of these markers suggest a severe and persistent inflammatory reaction that is usually found in acute and chronic toxoplasmosis (Abdeltawab et al., 2024). More importantly, anti-inflammatory cytokines IL-10 and TGF-b were also reduced substantially in the patient group ($P < 0.001$). These discoveries are significant, because the IL-10 and TGF-B are key factors in damaging inflammation and stimulating tissue repair (Jasim et al., 2025). The cases of low concentrations of these molecules suggest that there is a lack of control over the host inflammatory response that can lead to immunopathology and inability to eliminate the infection. There are studies that have indicated transient rise of IL-10 in the acute infection period and a decrease; the persistently low levels of IL-10 that were below the threshold level across patients samples in this study are indicative of a chronic lapse in anti-inflammatory control (Mustafa et al., 2024; Yao et al., 2024).

One of the most significant results of our study is represented by the strongest positive correlation found with the help of regression analysis between certain adipokines and cytokines (Table 5). There was significant positive correlation between leptin and TNF-a ($r = 0.68$, $P < 0.001$) which means that adipokine secretion is associated with stimulation of the pro-inflammatory mediator responses. This is a biologically plausible possibility, because leptin is known to stimulate TNF-A production, establishing a positive feedback mechanism that has the possibility to increase inflammation (Al-Halbousi et al., 2024). In addition, the interrelation between the adipocyte-derived signals and the systemic acute-phase response is further supported by the high correlation between resistin and CRP ($r = 0.59$, $P < 0.001$) (Ansari-Lari et al., 2024). Also, adiponectin and IL-10 showed a high positive correlation ($r = 0.62$, $P < 0.001$), which highlights the key position of these anti-inflammatory mechanisms. The production of IL-10 is facilitated by adiponectin, which indicates a concerted action of the two anti-inflammatory molecules in the framework of Th1 reactions to the *T. gondii* infection (Sighencea et al., 2025).

Conclusion

The results therefore not only indicate a direct correlation between *T. gondii* infection, deregulation of adipokines and skewed host immunity, but also initial findings on how this parasite can suppress host immunity in another manner. The effect of higher concentrations of pro-inflammatory adipokines and cytokines and lower concentrations of the anti-inflammatory counterparts indicate that the immune response towards inflammation is disproportionately low. The high level of coordination of adipokines and cytokines indicates the direct mechanistic cause of adipokines' participation in subtuning the in-

flammatory reaction to the parasite. Our results provide new leads to research on whether this metabolic-immune mechanism can be prone to therapeutic interventions, which consequently could contribute to the minimization of pathogenesis in the course of toxoplasmosis.

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