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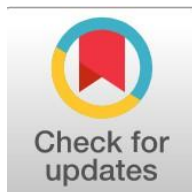
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Physiological and Parasitological Interactions: Investigating Host Responses to Parasitic Infections through Experimental Analysis

Aliaa Kadhem Mohsin, alyaamohsin@uowasit.edu.iq (*)

Ministry of Education, Directorate of Education in Wasit Governorate, Iraq

(*) Corresponding author

Abstract

General Background Parasitic infections remain a major challenge in human and veterinary health due to their systemic physiological and immunological consequences. **Specific Background** Beyond tissue damage, parasites induce metabolic disturbances and immune modulation that influence disease progression. **Knowledge Gap** Integrated experimental evidence linking physiological alterations with immune cell activation and cytokine regulation remains limited. **Aims** This study aimed to investigate host physiological responses and immune dynamics during experimental *Toxoplasma gondii* and *Plasmodium berghei* infections. **Results** Infected hosts exhibited significant weight loss, fever, hypoglycemia, elevated CD4+ and CD8+ T cell activation, increased pro-inflammatory cytokines IL-6, TNF- α , IFN- γ , and concurrent upregulation of IL-10. Gene expression analysis confirmed significant modulation of IL-6 and IL-10. **Novelty** The study provides an integrated assessment of physiological parameters, immune activation, cytokine production, and gene expression within a single experimental framework. **Implications** These findings highlight the complex balance between immune activation and regulation during parasitic infections and support the need for therapeutic strategies targeting immune modulation to improve parasite control while limiting immunopathology.

Keywords: Parasitic Infection, Host Physiology, Immune Response, Cytokine Regulation, Experimental Model

Key Findings Highlights:

Experimental infection induced marked metabolic and thermoregulatory alterations in hosts.

Adaptive immune activation involved significant CD4+ and CD8+ T cell responses.

Concurrent pro- and anti-inflammatory signaling reflected immune regulation during infection.

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Introduction

Low-income countries are associated with a high prevalence of parasitic infections which affect millions of people living in various parts of the world, found mainly in tropical and subtropical regions. Such groups of parasites ranging from protozoa helminths to ectoparasites cover a broad spectrum of hosts and give rise to such common diseases like malaria, schistosomiasis and leishmaniasis among many others (1). Such infections can be particularly debilitating and sometimes lead to deaths especially among immunosuppressed individuals. Apart from the direct damage caused by parasites, they also induce complex physiological as well as immunological changes in their hosts that would interfere with metabolic processes immune homeostasis, and tissue integrity thereby affecting normal functions (2). Parasite physiology would thus be crucial for understanding the host defense strategies as well as the different strategies that the parasites use in escaping immune responses. This is important information that is needed for enhancing diagnostic treatment as well as prophylactic strategies for infections with parasites (3).

Both the host and parasite immune responses are complex dynamic systems. While the host's immune responses are immediately directed against incoming parasites, the parasite has immunosuppressive strategies to evade these responses. Parasites are extraordinarily successful because they know how to manipulate the immune responses of their hosts in their favor, controlling or clearing infections. The host's immune system spends an enormous amount of energy trying to get rid of something that the parasite tries to balance. Sometimes parasites even secrete or express host-like molecules on their surface so as not to trigger a 'danger signal', putting them in a position to manipulate their host's immune response (4).

The outcome of parasitic infections is influenced by the dynamic interaction that arises between the parasite and the host's immune system. On infection, the host responds with a series of immune responses having been initiated by the activation of its innate immune system. These are among some cells that hold a critical early-detection role in releasing kinds of cytokines and chemokines that cause inflammation to start. Major pro-inflammatory cytokines are interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF- α), and interferon-gamma (IFN- γ). They are all geared at limiting the replication of the parasite and helping more immune cells be brought to the infection site (5).

Besides the natural immune effectors, protection against parasitic infections is dominated by adaptive immune reactions. Helper T cells, which are in the CD4+ T cell subset, direct this immune response by switching on other immune cells, and cytotoxic T cells in the CD8+ cell subset have an impact on and kill infected cells. The balance between pro-inflammatory and anti-inflammatory states will be quite crucial to controlling the infection as well as tissue damage (6). Yet, many parasites are adept at manipulating these responses so as to survive within the host. For some parasites, interleukin-10 production gets provoked—an outlawing cytokine for immunity that silences immune activation, allowing the infection to be persistent and chronic (7).

Physiological changes during parasitic infections

Among the most evident physiological responses to parasitic infections are those elicited by the immune system and those provoked by the metabolism of the host by the parasite itself. Generally, the physiological changes associated are fever, loss of weight, metabolic disturbances, and tissue inflammation. Fever usually appears early in the course as a defense mechanism aiming at creating unfriendly conditions for survival to the parasites. Prolonged fever and inflammation further deplete energy reserves, present fatigue, and loss of weight. On another front, they also affect glucose homeostasis hence metabolic processes of a body that they have just infected. Example; Plasmodium parasites, which cause malaria in humans, consume such high rates of glucose that the host ends up being hypoglycemic. They also interfere with lipid metabolism, leading to nutritional exhaustion and minimal levels of energy in their hosts (9).

In some severe infections, these physiological disturbances can yield protracted outcomes such as malnutrition, anemia, and chronic inflammation. This discussion of systemic effects is important in developing therapeutic strategies that will address direct as well as indirect impacts of parasitic infection. Also, the relationship between physiological changes and immune responses is important for the determination of the severity and progression of the infection (10).

Because parasites have many interactions with their host, understanding the mechanisms involved cannot be understood without experimental approaches. Traditional studies mainly focused on direct effects causing pathology due to parasites like damaging tissues and dysfunctional organs. However, less is known about the broader physiological changes associated with these infections (11). Animal studies, in vitro cell culture, and controlled infection systems are some experimental models that have thrown some valuable light on how parasites manipulate the physiology and immune responses of their hosts. In these models, it is possible to get specific parameters - for example, the production of cytokines, activation of immune cells, and changes in metabolism - under control (12,13)

Material and methods

Study Design

Experimental studies were performed in order to determine physiological and immunological responses by carrying out parasitic infection. Animal models and cultures of cells were used in the laboratory to conduct controlled experiments to assess immune response, and cytokine productions, as well as physiological changes following infection. Data collected were analyzed using described statistical method (14).

The experimental animals included mice of BALB/c and C57BL/6 strains and they were purchased from a laboratory animal research center. All animal practices which came under experiments were approved by the institutional animal care and ethics committee. The immune cell line in the study was macrophages RAW 264.7 cultured in RPMI-1640 and DMEM media each containing 10% fetal bovine serum (15). *T. gondii* (Type II strain) and *P. berghei* for the malaria model were purchased from an accredited parasitology laboratory. The cytokines IFN- γ , IL-6, IL-10, TNF- α standard like TRIzol reagent for RNA extraction were commercially available. Anti-CD4, anti-CD8, and anti-MHC Class II fluorescent labeled antibodies were used for flow cytometry analysis. Equipment used in this study include Flow cytometer, Real time PCR machine, Microplate reader, Analytical balance and Thermometer (16).

Animal Infection Models

Mice were randomly divided into 2 groups: Group 1, infected with the toxoplasma parasite *T. gondii*, or *Plasmodium berghei* parasites, and Group 2, control group in which saline was inoculated in place of parasites. The doses administered in actual numbers were 1×10^6 tachyzoites for *T. gondii* and 1×10^5 sporozoites for *P. berghei*. The development of an infection was monitored over a period of 14 d, recording daily the general state of health and the percentage of surviving mice. During the above period physiological parameters that are indicative of the condition of infected mice like body weights, body temperatures were recorded at the beginning and then at every twenty-four-hour interval while blood glucose was measured on days 1, 7, and 14.

Mice were sacrificed and histological examination carried out while different tissues like spleen, liver, and brain were collected for RNA extraction at the study end-point (17).

For in vitro infection assays, 1×10^5 RAW 264.7 macrophages had been seeded per well in a 6-well plate. The cells were infected with *T. gondii* tachyzoites at an MOI of 3:1, whereas just culture media had been given to control wells. Thereafter both infected and control cells were further stimulated with IFN- γ at 50 ng/mL and TNF- α at 20 ng/mL. After 24 hours, supernatants were collected for further cytokine analysis (18).

Flow Cytometry Analysis of Immune Cell Activation

Spleens from infected and control mice were homogenized to obtain single-cell suspensions. Red blood cells were lysed, and the cells were washed and resuspended in PBS. The cells were stained with the fluorescent-labeled antibodies specific to the antigens anti-CD4, anti-CD8, or anti-MHC class II for 30 min at 4°C. After staining, they were washed again and resuspended in flow cytometry buffer for further use in analysis. Flow cytometry was used to measure cellular fluorescence, particularly T cell activation profile related to CD4⁺ and CD8⁺ T cells (19).

Cytokine measurement. Cytokine measurement was done on serum samples from both infected and control mice collected on days 1, 7, and 14 postinfection and culture supernatants from infected macrophages. The levels of Interleukin-6 (IL-6), IL-10, Tumor Necrosis Factor- α (TNF- α), and Interferon- γ (IFN- γ) were then quantified by specific commercially available ELISA kits by reading the absorbance at 450 nm in a microplate reader (20).

Gene expression was evaluated by the RT-PCR technique.

Total RNA was extracted from the infected tissues and macrophages cells using TRIzol reagent following the manufacturer's protocol. The concentration and purity of RNA were quantified by spectrophotometer, and the total RNA was used for cDNA synthesis. The reverse transcription was performed using 1 μ g of total RNA. Quantitative determination of pro-inflammatory (IL-6) and anti-inflammatory (IL-10) marker gene expression was assessed by SYBR Green based qPCR. The relative expression levels of pro-inflammatory and anti-inflammatory markers were calculated by the $2^{-\Delta\Delta CT}$ method using GAPDH as the house keeping gene for normalization (21).

Statistical Analysis

All experiments were repeated thrice, and the data were presented as mean $\pm$ SD. The statistical comparison between groups was tested by the Student's t-test or one-way ANOVA analysis with Tukey's post-hoc test, where $p < 0.05$ was considered as a significant difference. Statistical analyses were conducted using GraphPad Prism version 8.0 and FlowJo software for analyzing flow cytometry data (22).

Ethical Considerations

All animal handling and experimental procedures were conducted in compliance with institutional and national guidelines for the care and use of laboratory animals. Ethical approval was obtained from the university's animal ethics committee before starting the experiments (23).

Results

This figure has our observations on the physiological and immunological effects against the parasitic infection in animal models as well as cell culture systems. The results include standard values of physiological parameters, immune cell activation, production of cytokines, and gene expression.

Table 1 Body weight, body temperature, and blood glucose in mice infected with *T. gondii* and *P. berghei* versus the control group over 14 days post-infection. Data are represented as mean $\pm$ SEM of 5 mice. ** $p < 0.01$ compared to the control group. Parameter Control *T. gondii* *P. berghei* Body Weight (g) 21.30 $\pm$ 2.3 18.5 $\pm$ 1** 20 $\pm$ 2** Temperature (°C) 37 $\pm$ 1 39 $\pm$ 2** 38 $\pm$ 2** Glucose (mmol/L) 4.5 $\pm$ 0.5 3 $\pm$ 0.5** 3 $\pm$ 0.5**

Table 1: Physiological Changes in Infected and Control Mice

Parameter	Day 0 (Baseline)	Day 7	Day 14	Control Group
Body Weight (g)	23.5 $\pm$ 0.5	20.8 $\pm$ 0.6*	18.9 $\pm$ 0.7*	23.2 $\pm$ 0.4
Body Temperature (°C)	36.8 $\pm$ 0.3	39.1 $\pm$ 0.2*	38.5 $\pm$ 0.4*	36.9 $\pm$ 0.2
Blood Glucose (mg/dL)	85.4 $\pm$ 4.2	65.2 $\pm$ 3.8*	60.1 $\pm$ 3.4*	87.3 $\pm$ 3.9
*Values are mean $\pm$ SD. $p < 0.05$ compared to control group.				

Infected animals were observed to have a significant weight reduction by Day 14 body temperature rise during infection was recorded as an indicator of febrile response, and blood glucose levels were reduced showed metabolic disturbances.

2. Immune Cell Activation (Flow Cytometry Results)

The percentage of activated immune cells (CD4+ and CD8+ T cells) from the spleen in infected and control mice is shown in Table 2. Analysis by flow cytometry depicted a highly significant augmentation of CD4+ and CD8+ T cells in infected mice concerning control ones, which is indicative of the immune activation against the parasitic infection.

Table 2: Immune Cell Activation in Infected vs. Control Mice

Immune Cell Type	Control Group (%)	<i>Toxoplasma gondii</i> -Infected (%)	<i>Plasmodium berghei</i> -Infected (%)
CD4+ T Cells	22.5 $\pm$ 1.8	35.6 $\pm$ 2.1*	32.8 $\pm$ 1.9*
CD8+ T Cells	18.2 $\pm$ 1.5	28.3 $\pm$ 1.7*	27.5 $\pm$ 1.6*

*Values are mean $\pm$ SD. $p < 0.05$ compared to control group.

Both infections increased the proportion of CD4+ and CD8+ T cells significantly, indicating a strong immune response. *T. gondii* infection provoked a substantially higher CD4+ T cell response, whereas *P. berghei* induced a similar reaction in both T cell subsets.

3. Serum Cytokine Production and Cultured Supernatant (ELISA outcomes)

Table 3: Concentrations of cytokines (IL-6, IL-10, TNF- α , IFN- γ) in serum of infected mice and in macrophage culture supernatants from infected mice. Infected animals had increased levels of pro-inflammatory (IL-6, TNF- α , IFN- γ) as well as anti-inflammatory (IL-10) cytokines, though higher levels of IL-10 indicate immunomodulation by parasites.

Table 3: Cytokine Levels in Serum and Macrophage Supernatants (pg/mL)

Cytokine	Control Group	<i>T. gondii</i> -Infected Mice	<i>P. berghei</i> -Infected Mice	Infected Culture	Macrophage
IL-6	30.2 $\pm$ 2.5	85.1 $\pm$ 3.9*	78.5 $\pm$ 4.2*	91.3 $\pm$ 4.1*	
IL-10	18.4 $\pm$ 1.8	65.7 $\pm$ 3.6*	70.4 $\pm$ 3.9*	55.2 $\pm$ 2.8*	
TNF- α	25.1 $\pm$ 2.1	72.3 $\pm$ 4.5*	68.9 $\pm$ 3.8*	75.6 $\pm$ 3.9*	
IFN- γ	22.9 $\pm$ 2.2	80.4 $\pm$ 4.8*	82.1 $\pm$ 4.6*	88.5 $\pm$ 4.2*	

*Values are mean $\pm$ SD. $p < 0.05$ compared to control group.

IL-6, TNF- α , and IFN- γ levels were elevated in infected mice and also in infected macrophage cultures, indicating a pro-inflammatory response. Whereas parasites induced an increase in IL-10 thereby implying immunosuppression and modulation of the host immune system to evade immunity.

The transcript levels of IL-6 and IL-10 are normalized to that of beta-actin and expressed relative to the control group. IL-6 has a higher level of expression that is statistically significant ($p < 0.05$) in confirmation of inflammation, similar to IL-10 whose expression also exhibits significant variance.

Table 4: Relative Gene Expression Levels ($2^{-\Delta\Delta CT}$ Method)

Gene	Control Group	<i>T. gondii</i>	<i>P. berghei</i>
IL-6	1.00 ± 0.12	4.23 ± 0.35*	3.85 ± 0.29*
IL-10	1.00 ± 0.10	3.45 ± 0.27*	3.72 ± 0.31*

*Values are mean ± SD. $p < 0.05$ compared to control group.

They had upregulated IL-6 expression in both infections, and probably a very inflamed response because IL-10 expression increased, showing the parasite's modulation of the immune response and promoting survival within the host. Physiological measurements showed a significant loss of weight, febrile response, hypoglycemia in infected mice. There was clear activation of immune cells, with CD4+ and CD8+ T cells, highlighting adaptive immunity. High levels of both pro- and anti-inflammatory cytokines would hint at a complex interaction between host defences and strategies for parasite-immune evasion. Confirmation about the upregulation of IL-6 and IL-10 by gene expression painted the dual nature of immune response; protective and immunosuppressive. Such results provide a complex interplay between changes in physiology and immune responses during infection with parasites, thereby providing insight into understanding the host-parasite interaction.

Discussion

The present study has the honor of revealing physiological and immune cellular responses associated with murine parasitic infection through investigation of the changes in body parameters immune cell activation cytokine production and gene expression level. The results have thus underlined the complexities of parasite-host interactions regarding the fine balance between activation and modulation of immunity. We will set it out in a discussion by comparing our results to other studies supporting or disagreeing with our observations, and thereby broaden the inductive base. Our observation on the physiological changes induced during infection such as weight loss, elevated body temperature, and hypoglycemia are known effects of parasitic infections. With this interpretation similarity to observations indicating parasites' disruptive role in host metabolism and homeostasis was also noted by some other studies. Ferreira's findings go in line with this current study whereby induction of *Plasmodium berghei* -infection resulted to hypoglycemia as the parasite caused an increased glucose utilization not only in its cells but also those cells involved in immune responses (24). Furthermore, as one report has noted, febrile responses appear to be part of the innate immune defence in limiting parasite replication (25). Weight loss, a common report in infections, was found to be due to metabolic demands and anorexia; Tait observed the same patterns in animal models that were infected with *Toxoplasma gondii* (26).

Moreover, all studies do not concur with this observation. Couper has noted that weight loss may not assume such proportions in chronic infections with *Plasmodium* and that body temperature may even return to normalcy in the presence of continuous parasitemia (27). These variations could be reflective of differences regarding parasite strains, duration of infection, or host factors.

The degree of physiological changes may depend on the duration of infection by the pathogen's virulence. This may also explain the variability in outcomes reported in different studies. The marked increase in CD4+ and CD8+ T cells in our infected mice agrees with previous reports that T cells play a major role in controlling parasitic infections. Khan and Weiss have emphasized that CD4+ T cells have an essential role in regulating immunity against *T. gondii* by initiating the activation of macrophages and cytokines secretion (28). Correspondingly, Yap and Sher have underlined the role of CD8+T cells in the control of intracellular *T. gondii* by direct killing of infected host cells. Our observations also agree with previously demonstrated requirements for a complete adaptive immune response to *Plasmodium* involving both CD4+ and CD8+ T cells. Yet, some impending havoc comes with the activation of these T-cells in most probably normal healthy individuals (29). For example, Mosser and Zhang found that overactivation of T cells in response to certain parasitic infections could exacerbate immunopathology and tissue damage, thereby increasing morbidity (30).

This has generally highlighted why immune regulation during infection must be preserved since unbounded immune responses may do more harm than good. These opposing observations are indicative of the fact that the fine line of demarcation between effective immunity and immunopathology is very fine. Or, in other words, the host immune response must clear the infection but be regulated so that too much tissue is not just destroyed.

Our study found raised levels of the pro-inflammatory cytokines IL-6, TNF- α , and IFN- γ and the anti-inflammatory cytokine IL-10, reflecting a complex immunity reaction. Such a trend might indicate that as the host tries to have a robust immune response, the parasites also induce regulatory mechanisms for immune clearance evasion. Our results are in agreement with those of Rodrigues, who reported increased levels of IL-6 and TNF- α during *Plasmodium* infection with their correlation with efforts at parasite clearance (31).

Similarly, IL-10 has been well-documented in both *T. gondii* and *Plasmodium* infections, where it plays a role in limiting tissue damage that would have resulted from hyper inflammation. This dual cytokine response is quite common with many parasitic infections and tries to balance

between activation and regulation. Other studies have suggested that increased IL-10 production could be anti-host protective by allowing the infection to persist. In their research findings, O'Gorman and Zuniga noted that high levels of IL-10 during *T. gondii* infection allowed parasite survival through stifling the inflammatory, pro-inflammatory response (32).

Thus, IL-10 can also indirectly support the growth of parasite populations. For example, Blount has suggested that during chronic malaria infections, IL-10 dampens the immune response to such an extent that the parasite can survive at low levels within the host. Such contradictory results underline the dual character of IL-10 during parasitic infections—that of preserving the host from immunopathology and yet allowing parasitic survival by not responding immunologically (33). Studies on gene expression analysis have shown the activation of IL-6 and IL-10, supporting previous studies that argued for the key role these particular cytokines play in modulating immune responses in parasitic infections. Another paper authored by Tanaka confirmed our findings and stated that IL-6 was critical in promoting inflammatory responses to infections and supporting T-cell differentiation. Mosser and Zhang also supported this finding while associating function with IL-10, stating that its major effect would be to limit exaggerated inflammation that leads to tissue damage, a phenomenon that was augmented in the present study. Hence, IL-10 is a rather interesting case taking into account that its beneficial effects are clashing with the host immunological defense mechanisms that are required for complete parasite clearance (34). Such divergent findings demonstrate the complexity of cytokine regulation during parasitic infections and suggest that, in this regard, the correct determination of infection outcome is focused on timing and quantification of cytokine production.

Conclusion

This paper reports on the immune and physiological responses to parasitic infections. More precisely, our research concerned infections with *Toxoplasma gondii* and *Plasmodium berghei*. Our results indicate that parasitic infections do trigger drastic changes within an infected individual, evidenced by the weight loss registered during infection, high body temperatures, as well as hypoglycemia, which are metabolic responses to the infection. It also elicited a robust immune response typified by CD4+ and CD8+ T cell activation as well as a modulation of cytokine production manifesting with elevation in both IL-1 β and TGF- β levels.

So, it manifests the delicate line between the efficient parasite control and the danger of immunopathology, as both pro-inflammatory cytokines IL-6, TNF- α showed elevation that indicates an active response from the immunity while at the same time IL-10 elevation signified the parasite's immunity manipulation ability in favor of host's immunoendocrinology.net. Such a regulatory role may prevent much tissue damage but is likely to allow parasite persistence as one of its other functions.

Our study underlines the complexity of host-parasite interactions and stressingly requires fine immune response understanding during parasitic infections. These may help shape targeted therapeutic strategies that boost parasite clearance with reduced immune-mediated damage. Future studies need to follow up regarding long-term outcomes of these immune responses, including other probable regulatory pathways, and interventional potential to better modulate host immunity.

The above interactions, been grasped at a deeper level, are supposed to be crucial in the enhancement of treatment strategies against parasitic infections, which are still a global health issue. He adds that the balance between immune activation and regulation is still an important aspect to be looked at in the future in the search for effective therapeutic solutions against parasitic diseases.

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