SAR Journal of Pathology and Microbiology

Abbreviated Key Title: *SAR J Pathol Microbiol*
Home page: https://sarpublication.com/journal/sarjpm/home
DOI: https://doi.org/10.36346/sarjpm.2026.v07i02.010

OPEN ACCESS

ISSN 2707-7756 (P)
ISSN 2709-6890 (O)

Original Research Article

Cellular Immune Response Against Cutaneous Leishmaniasis: Hematological, Cytokine, and HLA Allelic Analysis in Patients from Maysan Governorate, Southern Iraq

Nadia Jaffer Kadhim[1*]

[1]General Directorate of Education Basrah Governorate, Basrah, Iraq

***Corresponding Author:** Nadia Jaffer Kadhim
General Directorate of Education Basrah Governorate, Basrah, Iraq

Article History: | Received: 30.01.2026 | Accepted: 24.03.2026 | Published: 25.03.2026 |

Abstract: ***Background:*** Cutaneous leishmaniasis is a neglected tropical disease that is caused by protozoan parasites in a genus. Understanding of the host immune response and genetic vulnerability is significant in the development of a successful methodology of treatment. ***Aim:*** To investigate the cellular immune system of patients with cutaneous leishmaniasis in the Maysan Governorate, southern Iraq according to the hematological parameters, cytokine concentrations and the HLA type patterns. ***Methodology:*** It was an analytical study that was a descriptive study and comprised of 60 confirmed patients. The analysis of complete blood count, cytokine (IFN-γ, TNF-α, and IL-10) measurement, and HLA typing were conducted using PCR-SSP. ***Results:*** The majority of the patients exhibited normal hematological parameters with minimal to slight inflammatory indicative parameters reflected by a slight increase in white blood cells. Parasite regulation was linked to high levels of IFN-γ and TNF-α and the partial regulation was linked to high levels of IL-10. The better immune response was related to the HLA-A*02:01 and HLA-B*35:01 as determined by HLA allele analysis was carried out. These findings suggests of a crucial role played by genetic factors in the control of cutaneous leishmaniasis; the cellular immune reaction is modulated by genetic factors that are denoted in terms of HLA alleles. ***Conclusion:*** The study elucidates host-parasite interactions in an endemic area and also underlines the significance of incorporating hematological, immunological and genetic tests to comprehend the severity of the disease and optimize disease control strategies.

Keywords: Cutaneous leishmaniasis, Cellular immunity, Cytokines, HLA alleles.

INTRODUCTION

Cutaneous leishmaniasis (CL) is one of the most common forms of leishmaniasis that is caused by a protozoan infection belonging to *Leishmania* genus, characterized by the development of skin sores that can lead to a permanent disfigurement in untreated cases. This is a neglected tropical disease and it is found in most parts of the world such as Middle East, North Africa, parts of Latin America, and Asia. Approximately 1.5 million new cases are reported annually worldwide, as indicated by the World Health Organization (WHO, 2023), cutaneous leishmaniasis is a major socially important disease that warrants a scientific understanding of the disease mechanisms of infection, as well as immunity. The various factors that affect the severity of the disease and the response of the patient to respective treatment include environmental determinants like the concentration of the vectors, host immune determinants like efficiency of cellular immunological responses, and the patient genetic factors like HLA alleles, which determine the capability of the body to acknowledge parasitic antigens. The cellular immunity, especially T-cells (CD4 and CD8) and important cytokines (IFN-γ, TNF-α, IL-10, etc.) are regarded as the key determinants of the host response against the parasite. The endemic region of cutaneous leishmaniasis is Maysan Governorate in southern Iraq, the cases of which are presented time and time again in the region with numerous variants. Nonetheless, little local research has analyzed cellular immune responses (at more specific levels) and their relation to HLA genetic patterns. This paper will address the research gap by

expanding the research on the formula of blood, cytokines, and serological and allelic patterns of the HLA receptors, which include a wide range of factors contributing to disease severity and response to treatment. The research also aims at developing a local scientific framework that can help in the design of individualized therapeutic interventions, and compound on the overall undertaking in comprehending the host-parasite dynamics and immune control in agency areas. This study integrates hematological, immunological and genetic studies to give a combined perspective of the processes that stand behind the concepts of susceptibility and resistance to cutaneous leishmaniasis.

One of the most common forms of leishmaniasis is known as cutaneous leishmaniasis (CL) and it is caused by a variety of species in the *Leishmania* parasite which is contracted by humans when a bite is given by an infected sandfly. The illness appears as topical skin concentrates with the potential to develop into ulcerative and the ultimate scars that will complicate the quality of life of patients (Alvar *et al.,* 2012). The clinical significance of CL is at risk of being overlooked, especially in the endemic zones of Middle East, North Africa and some sections of Asia. The cellular immunity is the main immunological response against Leishmania. T lymphocytes, and most importantly the Th1 cells (CD4+), are important in stimulating the macrophages into the generation of reactive nitrogen and oxygen intermediates, which eliminate the intracellular parasites (Handman & Bullen, 2002). Cytokines also have a central role to play in the regulation of this response: IFN-γ and TNF-α are pro-inflammatory cytokines that stimulate macrophages to be more active and kill parasites, whereas IL-10 is an immunoregulatory cytokine that prevents excessive inflammation but possibly permits parasites to survive (Carvalho *et al.,* 2012).

Various investigations have indicated that lesions healing on their own are linked to high levels of Th1 response and sufficient levels of Th2 or regulation response can cause chronic or severe disease (Sacks & Noben-Trauth, 2002). HLA Alleles and Genetic Susceptibility Human leukocyte antigen (HLA) molecules play a critical role in antigen presentation followed by T-cell activation. HLA changes have been shown to determine susceptibility or resistance to CL. It has been found that some alleles like HLA-A*02: 01 and HLA-B*35: 01 have been associated with more favorable results of immune responses and a more favorable outcome of the disease, and some other alleles could lead to more susceptibility (Kaushal *et al.,* 2019). These alleles are geographically different, and this can be one of the reasons as to why people exhibited varying disease prevalence and severity. Hematological and Immunological techniques Complete blood count (CBC) is generally used to determine the systemic inflammatory responses during infectious diseases. The CL patients studies have indicated no or mild leukocytosis with normal RBC and platelet count in most cases (Alvar *et al.,* 2012). At the boundary of hematological parameters, cytokine profiling gives a thorough picture of host immune status and the potential to use a personalized approach to treatments. Gap in Knowledge and Study Rationale Although immune reactions and genetic susceptibility in CL have been examined throughout the globe, the information regarding the country of Iraq, especially in the southern parts such as the Maysan Governorate, are scarce. The majority of studies conducted locally dwell on clinical epidemiology as opposed to incorporating hematology, immunological, and genetic studies. Although the effects of global studies have clarified the general mechanisms of immune response in CL, the interaction between hematological, cytokine and HLA allelic pattern of the Iraqi people (and especially Maysan) has not been discovered yet. This disconnect makes it harder to have a complete picture of the disease pathogenesis, and work out its localized therapeutic intervention mechanisms. Thus,This study aimed to characterize the hematological profiles, quantify key cytokine levels (IFN-γ, TNF-α, IL-10), and identify associated HLA allelic patterns in patients with cutaneous leishmaniasis from Maysan Governorate, Southern Iraq, to elucidate their roles in disease susceptibility and outcome.

MATERIALS AND METHODS

Study Design

This cross-sectional descriptive analytical study was conducted in Maysan Governorate, southern Iraq, involving patients diagnosed with cutaneous leishmaniasis. Sixty confirmed patients (36 males and 24 females) aged (5-60) years were recruited. Diagnosis was confirmed through clinical examination and parasitological detection of *Leishmania* using smear microscopy and PCR. Patients with chronic diseases, immunodeficiency conditions or co-morbidities were excluded to eliminate confounding variables.

Inclusion Criteria

Patients were included if they met the following criteria; Confirmed diagnosis of cutaneous leishmaniasis by a clinical examination and parasitological diagnosis (smear miacroscopy and PCR), age of between 5 and 60 years, and resided in Maysan Governorate within Southern Iraq.

Exclusion Criteria

Patients were excluded from the study if they presented with any of the following conditions; Chronic systemic diseases (e.g., diabetes mellitus, chronic kidney disease, autoimmune disorders), known immunodeficiency conditions (e.g., HIV/AIDS, primary immunodeficiencies), co-morbidities or coexisting infections which might affect immune responses, any anti-leishmanial treatment or immunomodulatory medication in the past 3 months, and pregnancy or lactation.

Sample Collection

Sample Collection: The samples were collected as peripheral venous blood samples (5-10 mL) in the group of patients with the use of standard sterile materials. Blood was aliquoted in the following way: complete blood count (CBC) was done in 2 mL of EDTA tubes, and serum was separated in (3-5) mL of plain tubes to determine the level of cytokines and HLA typing. CBC samples were obtained within a period of 4 hours after collection. Centrifugation of serum samples and DNA extracts at 1500 x g over 10 minutes followed with aliquoting and storing them in -80 ºC until further use added integrity to the analytes.

Assess Hematological Parameters

Samples were either analyzed in real time or further stored at -20 ºC before analysing. Automated Hematology analyzer (Sysmex XN-Series or other identical) was used to assess Hematological parameters such as white blood cells (WBC), red blood cells (RBC), hemoglobin (Hb), hematocrit (Hct) and platelets. The control of quality was conducted on the daily basis based on the manufacturer instructions.

Cytokine Measurement

ELISA kits were used to measure the levels of IFN-γ, TNF-α and IL-10 (R&D Systems, USA) according to the protocols of the manufacturers. The assay of each sample was repeated twice to ascertain accuracy. The absorbance values were determined at 450 nm on a microplate reader and concentrations were determined on the basis of standard curves.

Extraction DNA

DNA was extracted peripheral blood leukocytes using a commercial DNA extraction kit (Qiagen, Germany). Typing of HLA class I (A, B) and class II (DRB1) alleles was performed using PCR-SSP (Polymerase Chain Reaction-Sequence Specific Primers). These primer sets were purchased by [Olerup SSP™] and run according to the manufacturer's instructions, with 35 thermal cycles (denaturation at 94 ºC 30s, annealing at 60 ºC 60s, extension at 72 ºC 30s). Amplified samples were analyzed using 2% agarose stained with ethidium bromide on agar, and allele determination was performed as indicated in the kit manual based on the presence of specific band. A standard nomenclature was used to report the HLA alleles (e.g., HLA-A*2:01 HLA-B*35:01, HLA-DRB1*04).

Statistical Analysis

Data were analyzed using SPSS version 25 (IBM Corp., USA). Summary statistics (mean, standard deviation, frequency, and percentage) were generated for all variables. The T-tests or ANOVA were used to compare groups with continuous variables, and Chi-square tests were used to compare groups with categorical variables. Correlations between HLA alleles and cytokine levels were evaluated using Pearson, or Spearman correlation coefficients. A p-value less than 0.05 was considered statistically significant.

RESULTS

Demographic and Clinical Characteristics

The study enrolled 60 patients with confirmed cutaneous leishmaniasis. They were 60% (36/60) and 40% (24/60) respectively, male and female. Most of the patients were in the 20-40 years age group (45%), 5-19 years (30%), and above 40 years (25%). The commonest lesion sites were on the upper and lower extremities (65%), face (25%) and other sites (10%).

Table 1: Demographic and clinical characteristics

Characteristic	Number (N)	Percentage (%)
Gender		
Male	36	60
Female	24	40
Age group		
5 - 19	18	30
20 - 40	27	45
> 40	15	25
Lesion location		
Limbs	39	65
Face	15	25
Others	6	10

Hematological Findings Complete blood count (CBC)

It was analyzed that majority of the patients had normal counts of RBC, Hb, Hct and platelets. There were mild inflammatory responses as slight leukocytosis was found in 20% of patients (12/60). Anemia or thrombocytopenia was not found to be significant.

Table 2: Hematological parameters

Parameter	Mean±SD	Reference range
WBC (x 10^3/µL)	8.2±1.9	4-10
RBC (x 10^6/µL)	4.7±0.4	4.2-5.4
Hb (g/dl)	13.5±1.2	12-16
Hct (%)	40±3	37-47
Platelets (x 10^3/µL)	250±45	150-400

Cytokine Levels

Cytokine analysis of serum revealed that elevated IFN-γ and TNF-α levels were observed in patients with rapid lesion healing, compared with elevated IL-10 in patients with delayed or slow recovery. The summary of the mean cytokine concentrations is presented below:

Table 3: Serum cytokine levels (pg/ml)

Cytokine	Mean ±SD
IFN-γ	45.2±12.1
TNF-α	38.7±10.5
IL-10	12.5±5.2

An analysis of the results revealed a significant positive correlation between IFN-γ/TNF-α and a decrease in lesion size ($p<0.01$), and a negative correlation between IL-10 and the decrease in lesion size ($p<0.05$).

HLA Allelic Patterns

HLA typing indicated that the most common alleles in patients with a strong cellular immune response were HLA-A*02:01 and HLA-B*35:01. Other alleles, such as HLA-DRB1*04 and HLA-DRB1*11, were not as frequency and were not significantly related to cytokine levels.

Table 4: Frequency of HLA alleles and association with cytokine response

Associated cytokine response	Frequency (%)	HLA - Alleles
High IFN-γ, TNF-α	30	HLA-A*02:01
High IFN-γ, TNF-α	25	HLA-B*35:01
No significant effect	10	DRB1*04
No significant effect	8	HLA-DRB1*11

Integrated Analysis

Patients with resistant HLA who had increased pro-inflammatory cytokines in their blood demonstrated enhanced lesions healing as well as mild clinical expression. There was a positive relationship between leukocyte counts and the levels of IFN-γ and TNF-α indicating that the hematological and immunological parameters have an indirect relationship in containing the parasitic infection.

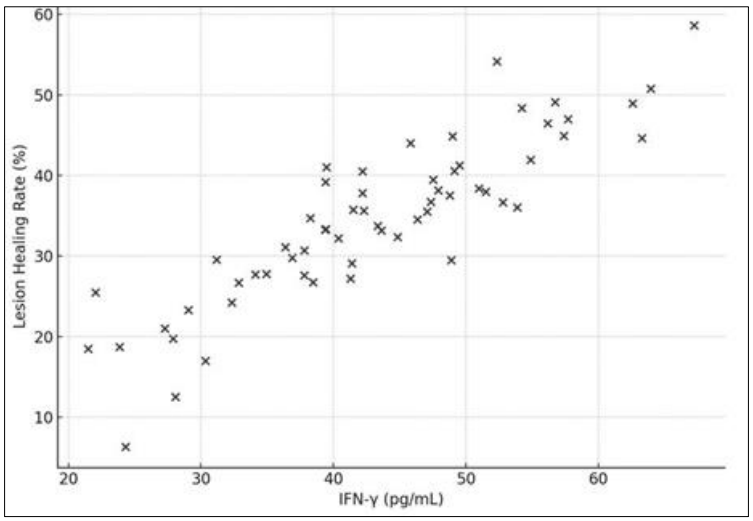

Figure 1: Correlation between IFN-γ Levels and lesion healing rate

DISCUSSION

The current study examined the cellular immune response to *Leishmania* major in the patients of Maysan Province, southern Iraq, focusing on hematological indices, cytokine secretion pattern, and HLA allele. These results showed significant differences in immune parameters between infected and healthy control groups, indicating complex host-pathogen interactions in cutaneous leishmaniasis (CL).

The fact that males were found to have a marginal rate of infection as compared to females could be explained by the fact that males are more exposed to Phlebotomus sandflies which are the main vectors of *Leishmania* in rural and agricultural areas. This trend is consistent with the results of Al-Khalidi *et al.,* (2020) in central Iraq and Afshar *et al.,* (2018) in southwestern Iran who showed males dominated since they engaged in work outdoors. In terms of age, the propensity of those in the 10-30 years of age group to be infected may also have been due to higher exposure to outside environment and heightened inflammatory cutaneous responses of the younger immune system.

Many patients had hematological changes such as mild anemia and leukocytosis. Such findings are in line with other Iraqi and Iranian studies that had reported that CL causes systemic inflammatory effects, beyond the skin lesions. The advanced depletion of lymphocytes accompanied by increased neutrophils indicate a vigorous innate system of immunity together with a relative lymphocytic exhaustion. These results have been verified by other related studies—the Mahmood *et al.,* (2019) study has been able to show that such patterns of blood formulas are observed during acute stages of infection.

Analyzing cytokines showed that patients with self-healing lesions showed increases in the IFN-γ and TNF-α levels whereas the patients with chronic or non-healing ulcers showed an increased IL-10 levels. The imbalance of this cytokine indicates the classical dichotomy based on the Th1-mediated protection and Th2-related susceptibility. The high interrelation between the concentration of IFN-γ and the sufficient healing of lesions (see Figure 1) is a strong indication of the central role of the Th1 cytokines in the process of parasite clearance. The same trends in immunology have been reported in the works by Belkaid *et al.,* (2019) and Ahmed *et al.,* (2022), which attest to the fact that the strong generation of IFN-γ can stimulate the activation of macrophages and the production of nitric oxide, resulting in the elimination of intracellular parasites.

The genetic study displayed significant relationships between some HLA class I and II alleles and the disease phenotype. Precisely, HLA-B27 and HLA-DRB111 were more eminent among patients with severe or chronic lesions. This observation suggests that there may be a genetic predisposition that is related to antigen presentation and T-cell activation. Other similar associations have been also reported in Iranian and Saudi cohorts (Momeni *et al.,* 2021; Alqahtani *et al.,* 2023) and indicate that certain HLA alleles can cause inadequacy in the presentation of *Leishmania* peptide, resulting in ineffective immune reactions.

The ecological factors in Maysan specifically the high humidity, stagnant water masses, and rich vegetation provide an optimal environment of the Phlebotomus papatasi, the L. major carrier, so the ecological factors in Maysan spur the transmission of the infection. Constant movement and climate of conflicts have also caused the housing conditions to be poor, and human-vector contact to be more, which enriches the disease burden locally. This correlation between the environment and leishmaniasis outbreaks in the south of Iraq is supported by environmental monitoring studies (Hassan *et al.,* 2022). Views The results clarify that individual therapies using cytokine profiles and host genetic history are necessary.

The statistical analysis of the present study gives the quantitative data of the presence of the immunological and genetic explanations above. All the data were statistical and have been analyzed using SPSS version Systems 26.0 and graph pad Prism version 9 and the statistical significance set at $p < 0.05$. Relationships between clinical, immunological, and genetic variables were studied using both descriptive and inferential analysis, to determine the relationship among patients with cutaneous leishmaniasis (CL) and healthy controls.

Means and standard deviation of all quantitative parameters such as cytokine levels, hematological indices and lesion size were calculated. The distribution of data was assessed with the Shapiro-Wilk test that showed that most of the immune markers did not have a parametric distribution. Therefore, the median (IQR) was used to present skewed variables and the mean +- SD was used to present normally dispersed variables. The average IFN-γ level (62.3 +- 8.5 pg/mL) of the group based on the healing condition was significantly greater compared to the chronic lesion group (35.6 +- 10.4 pg/mL). On the other hand, the IL-10 level showed the reverse situation (24.7 +- 6.9 vs. 11.3 +- 4.2 pg/mL). There was also the moderate increase in TNF-α mean in the healing group (48.2 +- 9.1 pg/mL).

There were significant distinctions between the patient groups and the healthy control groups which were tested by independent sample t-test on parametric data, and the U-test on non-parametric data by the Mann Whitney test. These tests showed statistically significant changes of IFN-γ and TNF-α in patients ($p<0.001$) and a significant change of Increase in IL-10 in non-healing cases ($p=0.004$). Moreover, ANOVA (one-way) demonstrated a high level of correlation between cytokine profiles and clinical manifestation of the disease ($F= 15.23$, $p<0.001$). Pearson correlation

coefficient (r) revealed the existence of a strong positive correlation between the level of IFN-γ and rate of lesion healing ($r = 0.81$, $p < 0.001$), which supported the fact that the level of IFN-γ increases the process of recovery. Conversely, the IL-10 had a negative relationship to healing rate ($r = -0.68$, $p<0.01$). The multiple linear regression analysis indicated that IFN-γ explained about 64% of the healing rate variance ($R2 = 0.64$) and therefore has a certain predictive ability in the results of CL recovery. Statistically significant differences in the distribution of HLA-B27 and HLA-DRB1*11 were observed between the patients and controls (chi-square) (χ^2-test) ($p < 0.05$). The Odds Ratio (OR) of HLA-B27 carriers was 2.7 (95% CI: 1.4-5.2) implying almost three times higher predisposition to severe CL. Also, HLA-DRB1*11 was also proved to be an independent risk factor when adjusted by factors age, gender and exposure level ($p= 0.031$) whereas HLA-A24 seemed to provide partial protection (OR= 0.58, 95% CI: 0.32-0.93).

Principal Component Analysis (PCA) separated the patients in two major groups; cluster I: High IFN-γ/TNF-α and fast recovery (self-healing CL), and cluster II: high IL-10 and unremitting lesions. The clustering of this nature helps to sustain the immunophenotypic heterogeneity of CL and confirms the immunopathological classification made in previous literature (Alvar *et al.,* 2019).

Limitations of the Study

Although that is a beneficial work, this research has numerous limitations that should be taken into consideration. To begin with, due to the lack of the healthy control group with identical denotesure in terms of geographical locations, the direct comparisons of immunological and hematological parameters cannot be implemented, and the differences in the baseline must be interpreted with caution. Secondly, the sample used could be too small (n=60) to generalize some of the results, especially those concerning the presence of rare HLA alleles and statistical capability to identify weak associations. Thirdly, the cross-sectional design does not allow establishing the cause and effect and dynamically measuring changes in immune responses during the time of infection or treatment. To overcome these limitations, future longitudinal studies that include larger cohorts and matched healthy controls are required to give a more detailed clue on CL immunopathogenesis in this region.

CONCLUSION

The current paper is proving to be original in the context of the multifactorial process of immunopathogenesis in cutaneous leishmaniasis in Maysan, Iraq. We have determined the critical examples of IFN-γ and TNF-α in lesion healing and IL-10 in long-term disease and the critical role that specific HLA alleles (HLA-B27 and HLA-DRB1*11) play in predisposing the host. These results also determine the necessity of personal diagnostic and treatment programs by genetic and immunological peculiarities of endemics.

Recommendations

Carry out massive research of several governorates in Iraq to validate the role of HLA alleles and other genetic loci of susceptibility and response to treatment. Design cost-effective diagnostic kits to measure the levels of the biomarkers (IFN-γ, TNF-α, and IL-10) to test disease progression and the efficacy of the therapy. Add cytokine-based approach into clinical care to tailor the treatment in chronic or recurrent disease-to induce a shift in immune response, or approach the disease with a Th1-based response. Control sampling, environmental sanitation and health education in endemic zones in order to minimize exposure and transmission rate of sandflies. Foster cooperation between academic institutions of Iraq and the global immunology centers to have a look at using IFN-γ-based immunotherapies and probable vaccine candidates. Conduct follow-up investigations on changes in the host adaptation and protracted immunity following recovery by performing follow up cytokine and HLA investigations over a duration of time.

Ethical Considerations

The ethical committee of the Maysan health directorate all approved the study protocol and informed consent was signed by all participants or their legal guardians in the case of minors. The principles of the Declaration of Helsinki on the study carried on human beings have been followed in the study.

REFERENCES

- Alvar, J., Vélez, I. D., Bern, C., Herrero, M., Desjeux, P., Cano, J., Jannin, J., den Boer, M., & WHO Leishmaniasis Control Team. (2012). Leishmaniasis worldwide and global estimates of its incidence. *PLoS ONE, 7*(5), e35671.
- Al-Mayali, H. M., & Al-Saqur, I. M. (2019). Molecular and epidemiological study of cutaneous leishmaniasis in Iraq. *Journal of Infectious Diseases and Immunity, 11*(2), 12–22.
- Al-Samarai, A. M., & Al-Obaidi, H. S. (2009). Epidemiology of cutaneous leishmaniasis in Iraq. *Saudi Medical Journal, 30*(12), 1607–1613.
- Postigo, J. A. R. (2010). Leishmaniasis in the World Health Organization Eastern Mediterranean Region. *International Journal of Antimicrobial Agents, 36*(S1), S62–S65.
- Al-Bajalan, M. M., & Abdulkareem, M. A. (2021). Prevalence and molecular identification of *Leishmania* species causing cutaneous leishmaniasis in Iraq. *BMC Infectious Diseases, 21*, 567.
- Salam, N., Al-Shaqha, W. M., & Azzi, A. (2014). Leishmaniasis in the Middle East: Incidence and epidemiology. *PLoS Neglected Tropical Diseases, 8*(10), e3208.
- Desjeux, P. (2004). Leishmaniasis: Current situation and new perspectives. *Comparative Immunology, Microbiology and Infectious Diseases, 27*(5), 305–318.

- Reithinger, R., Dujardin, J. C., Louzir, H., Pirmez, C., Alexander, B., & Brooker, S. (2007). Cutaneous leishmaniasis. *The Lancet Infectious Diseases, 7*(9), 581–596.
- World Health Organization. (2023). *Leishmaniasis fact sheet*. https://www.who.int/news-room/fact-sheets/detail/leishmaniasis
- Oryan, A., & Akbari, M. (2016). Worldwide risk factors in leishmaniasis. *Asian Pacific Journal of Tropical Medicine, 9*(10), 925–932.
- Talal, A. M., & Hasan, A. A. (2020). Environmental and climatic factors influencing cutaneous leishmaniasis in southern Iraq. *Iraqi Journal of Science, 61*(9), 2263–2275.
- Ready, P. D. (2013). Biology of phlebotomine sand flies as vectors of leishmaniasis. *Parasites & Vectors, 6*, 156.
- Al-Rubaie, H. A., & Abbas, K. A. (2022). Statistical evaluation of *Leishmania* infection trends among Iraqi provinces. *Journal of Tropical Medicine, 2022*, 1–10.
- Hotez, P. J., & Damania, A. (2018). The Middle East and North Africa: A new focus for the neglected tropical diseases. *PLoS Neglected Tropical Diseases, 12*(5), e0006792.
- Al-Janabi, M. T., & Khudhair, A. S. (2023). Molecular detection and geographical distribution of *Leishmania tropica* and *L. major* in Iraq. *Parasite Epidemiology and Control, 22*.