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Cytokine Profiling and Immune Response in Atopic Dermatitis Patients with *Sarcoptes scabiei*

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ABSTRACT

Background and Aim: Scabies, caused by *Sarcoptes (S.) scabiei*, is associated with immune dysregulation and elevated immunoglobulin E (IgE) levels. Differentiation between scabies and atopic dermatitis (AD) may be challenging because of overlapping clinical and immunologic features. This study aimed to evaluate serum total IgE, Interleukin (IL) 4 (IL-4), IL-5, IL-13, and Interferon gamma (IFN-γ) levels in scabies affected patients and to assess their diagnostic performance.

Materials and Methods: In this cross-sectional study, 50 participants were included, comprising 26 AD patients with scabies infestation and 24 uninfected controls. The diagnosis of scabies was verified using polymerase chain reaction (PCR). ELISA was used to assess the serum levels of total IgE, IFN-γ, IL-4, IL-5, and IL-13. The statistical analysis was performed using an independent t-test. The diagnostic performance was analyzed using receiver operating characteristic (ROC) curves, with a statistical significance level of $P<0.05$.

Results: Serum total IgE levels were significantly higher in patients compared with controls ($P<0.05$). IFN-γ and IL-5 levels showed significant differences between study groups ($P<0.05$). ROC curve analysis demonstrated statistically significant area under the curve values for IgE and cytokines in differentiating affected patients from controls. The results indicate that the immune response in AD patients was significantly altered by scabies infestation, resulting in a more complex cytokine profile than control.

Conclusion: The elevated level of IgE and altered INF-γ and IL-5 cytokine profiles are associated with scabies and atopic dermatitis. These immunologic markers may assist in differential diagnosis and provide insight into disease-related immune responses.

Keywords: Atopic Dermatitis, Cytokines, Immunoglobulin E, Interferon-gamma, Interleukin-4, *Sarcoptes scabiei*

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1. Introduction

Sarcoptes (S.) scabiei are ectoparasites that are the major cause of scabies, a very infectious skin mite that afflicts a high proportion of the population in most parts of the world, particularly in resource-deprived regions **(1)**. An infestation by *S. scabiei* has been linked with severe pruritus, popular eruptions, and secondary bacterial infections owing to skin excoriation **(2)**. The mite is known to secrete proteolytic enzymes and allergens that invade stratum corneum and activate both adaptive and innate immune responses **(3)**.

Th2-mediated cytokines effect and incompetency of the epidermal barriers interrelate complicatedly in the

pathophysiology of atopic dermatitis (AD) and among these, interleukin-13 (IL-13) is a crucial type-2 cytokine that exacerbates inflammation and the severity of AD in patients by contributing to immune dysregulation and disruption of skin barrier proteins like filaggrin. It has been demonstrated that IL-13 expression levels in peripheral blood and lesional skin correlate with both disease severity and immunoglobulin E (IgE) levels in AD patients, underscoring its involvement in the pathophysiology of the disease **(4)**. Another essential Th2 cytokine that plays a major role in eosinophil maturation, proliferation, and activation is interleukin-5 (IL-5). In allergic skin conditions, IL-5-stimulated eosinophils migrate into the skin and release cytotoxic granules and inflammatory mediators, exacerbating tissue damage and inflammatory reactions **(5)**.

It has been illustrated that mite antigens can induce a dominant Th2 immune profile, characterized by increased interleukins IL-13, IL-4, and IL-5, alongside increased serum IgE levels and eosinophilia **(6)**. Scabies infestation has been implicated in the dysregulation of host cytokine networks, including altered expression of IL-17, IFN-γ, and IL-10 in different disease severities **(7)**.

The ectoparasite *S. scabiei*, which causes scabies, causes the skin to mount a strong Th2 immune response. After mite invasion, antigen presentation encourages naïve T cells to release IL-13, IL-4, IL-5, and other type-2 cytokines in comparison with Th2 effector cells. These cytokines promote pruritic inflammation at infestation sites, increase mast cell and eosinophil recruitment, and assist in B cell class switching to IgE. By encouraging eosinophil survival and effector functions, IL-5 further maintains the local Th2 inflammatory milieu. In scabies, the combined action of IL-13 and IL-5 contributes to pathological inflammation, which shows up clinically as severe itching and eczematous skin changes, as well as host defense mechanisms against the parasite **(8)**.

Atopic dermatitis is a chronic inflammatory skin disease that has been characterize by its typical eczematous lesion, and severe itching with variable clinical progression **(9)**. A type 2 immune profile has been closely linked to the disease, whereby IL-13 and IL-4 play key roles as epidermal barrier dysfunction, IgE production, and chronic inflammation **(10)**.

Epidermal alarmins including thymic stromal, lymphoblastic/lymphocytic leukemia IL-33, IL-25 have also been found to be involved in amplification of Th2 inflammation in lesions of AD **(11)**. An association has been observed between scabies and worsening of atopic dermatitis symptoms, which is particularly notable in individuals with a predisposition to allergies **(12)**. In both scabies and atopic dermatitis, immunoreactivity has been ascribed to similar cytokine expression and immune cell activation mechanisms **(13)**. It has been proposed that co-infection or concurrent presence of *S. scabiei* in AD patients may amplify type 2 inflammation, promoting more severe or treatment-resistant disease phenotypes **(14)**. This study was therefore conducted to investigate the cytokine responses, particularly the levels of IL-4, IL-5, IL-13 and IFN-γ in patients with atopic dermatitis infested with *S. scabiei* at Al-Diwaniyah Hospital, Iraq.

2. Materials and Methods

2.1 Sample Size Determination

G*Power version 3.1 was utilized to perform a post-hoc power analysis. The statistical power was determined as 0.87 (87%) based on the moderate expected effect size (r = 0.40), alpha level of 0.05, and overall sample sizes of 50 participant, which indicates that the calculated statistical power was sufficient to demonstrate clinically significant relationship between cytokine levels and number of scabies parasites.

2.2 Study Design and Population

A cross-sectional observational study was command at the Dermatology Unit of Al-Diwaniyah Teaching Hospital, Iraq. The study period was defined in February 2025 to June 2025. Among outpatients and inpatients presenting to the Dermatology Department during this time frame, 50 participants were recruited consecutively. The study population was composed of female and male patients with the age ranged from 10 to 45 years. The inclusion criteria were established according to dermatological standards and included individuals who had been clinically diagnosed with atopic dermatitis based on the Hanifin and Rajka diagnostics criteria, which require the presence of at least three major and three minor clinical features (Pruritus, Flexural lichenification in adults, family history of atopy and recurrent skin infections). Those patients who were additionally confirmed with *S. scabiei* infestation were also included as verified through clinical evaluation and microscopic examination of skin scrapings. When mites such as larvae and eggs were identified, scabies was diagnosed through an optical microscope. Patients who had received systemic corticosteroids, immunosuppressive therapy, or anti-parasitic treatment within four weeks prior to enrollment were excluded to eliminate confounding immune alterations. Also, patients with suspected scabies but negative microscopic findings were excluded to ensure diagnostic specificity.

2.3 Parasite Diagnosis

The diagnosis of *S. scabiei* infestation was confirmed through both clinical and laboratory criteria, via typical clinical signs, including pruritic papules, burrows, and nocturnal itching that were assessed by a certified dermatologist during physical examination. Skin scrapings were obtained from active lesions, particularly intense pruritus, burrows, and papulovesicular areas, using sterile scalpel blades after applying a drop of mineral oil. Collected samples were placed on glass slides and examined under light microscope at 10X and 40X magnifications. The presence of any developmental stage of the mite—including eggs, larvae, or adult forms—was considered diagnostic of active infestation.

2.4 Molecular Detection

A single swab was rubbed across the fronts of both wrists, and on any suspected scabies lesions (burrows, nodules, and papulovesicular). DNA was extracted from the skin samples using Tissue DNA Isolation Kit (Genaid/Korea). Scabies DNA was amplified using ThermalAce™ DNA polymerase kit (Thermo Fisher Scientific, Waltham, MA, USA). For molecular detection, a specific set of universal primers were used to target a partial fragment of *S. scabiei* genome with an estimated size of 130 bp. The PCR product mixtures included $MgCl_2$ (25 mmol/L) (2 µL), enzyme mix containing Taq DNA polymerase, deoxyribonucleotide triphosphates (dNTPs), and reaction buffer (10X) (2.5 µL), and forward primer (GGGTCTTTTTGTCTTGGAATAAA) and reverse primer (CTAAGGTAGCGAAATCATTAGC) (0.5 µL each) **(15)**. The thermal cycling protocol used in PCR amplification was as follows; denaturation at 95ºC (3 min), followed by 35 cycles of denaturation at 95ºC (30 sec), annealing at 58ºC (30 sec) and extension at 72ºC (1 min), and finally extension at 72ºC (7 min). Negative control included all components in PCR product preparation except nuclease free water replacement of DNA.

2.5 Cytokines and Immunoreactivity Analysis

A total of 5 mL of peripheral venous blood was collected from each participant using an aseptic method with EDTA and serum-separating gel tubes. The samples were processed within 1 hr of collection, and the serum was separated by centrifugation at 3500×g for 10 min before being stored at -8°C until immunoassay analysis. The serum cytokine amounts were identified by commercially available enzyme-linked-immunoadsorbent assays (ELISA), which detected recombinant aggregates of human IL-13, IL-4, IL-5, and IFN-γ based on manufacturer's instructions (Sunlong/ China). The absorbance was detected at 450 nm using microplate reader (ELx800, BioTek Instruments Inc., Winooski, VT, USA). Each sample was tested in duplicate to obtain analytical reliability. The cytokine levels were determined based on conventional curves developed using standard recombinant cytokine concentrations. Total serum IgE levels were also measured using an ELISA-based immunoassay. Quality control was maintained by including internal controls and calibrators with each assay batch. The results were compared between patients with atopic dermatitis infested with *S. scabiei* and those with atopic dermatitis alone.

2.6 Statistical Analysis

The data were analyzed using the Statistical Package for the Social Sciences (SPSS) Software Version 25.0 (IBM Corp., Armonk, NY, USA). Categorical variables were reported as frequencies, percentages, and continuous data as mean ± standard deviation (SD). The Shapiro-Wilk test was used to determine whether the data distribution was normal. All cytokine variables followed a normal distribution (P>0.05). Levene test was used to assess the homogeneity of variance. The independent sample t-test was performed to compare the groups. To determine the links between category variables, the independent t-test was used to compare the mean values of continuous measures such as cytokine levels between patients with and without scabies infestation.

3. Results

The distribution of skin lesions was analyzed among patients with and without *S. scabiei* infestation. A total of 50 patients with atopic dermatitis were included, of whom 26 were confirmed infested with *S. scabiei*, while 24 were not. Burrows were observed in 16 patients with and 6 patients without infestation, accounting for the most frequent lesion type among the scabies group. On the other hand, the papulovesicular lesions were noted in 7 patients with and 11 patients without infestation. Finally, the intense pruritus was recorded in 3 infested individuals compared to 7 in the non-infested group.

A statistically significant relationship was identified between type of skin lesion and the presences of *S. scabiei* infestation (χ^2 = 6.965, P=0.03), suggesting that burrows were significantly more prevalent among infested patients (Table 1).

3.1 Molecular Conformation of Scabies

Positive scabies diagnoses were obtained for all skin scraping samples using both diagnostic approaches, namely microscopic examination and PCR assay. Microscopic analysis revealed the presence of scabies-related structures, confirming infestation in all examined specimens.

Molecular confirmation was further achieved through PCR amplification of scabies-specific DNA. Agarose gel electrophoresis was performed to verify the PCR products, and distinct bands corresponding to the expected amplicon size of 130 bp were consistently observed in all PCR-positive samples. Electrophoresis was conducted at 70 V for 1 hr, and no nonspecific bands were detected, indicating high specificity of the assay (Figure 1).

3.2 Cytokines and Immunoreactivity Analysis Results

Serum cytokine concentrations and total IgE levels were compared between patients with AD alone and those with both AD and *S. scabiei* infestation. An elevation in IL-4 levels was observed among patients with scabies + AD (176.38 ± 9.27 pg/mL) compared to those with AD alone (154.63 ± 11.45 pg/mL), although the differences were not reaching statistically significances (*P*=0.143) Data are shown in Table 2.

Similarly, IL-13 levels were higher in scabies-infested group (201.71 ± 19.93 pg/mL) than in non-infested group (188.42 ± 22.93 pg/mL), but the difference was not statistically significant (*P*=0.066). A significant increase in IL-5 concentration was detected in scabies + AD group (101.34 ± 11.38 pg/mL) compared to AD group (65.78 ± 8.93 pg/mL) (*P*=0.043). IFN-γ levels were markedly elevated among AD patients co-infected with scabies (837.62 ± 19.74 pg/mL) in contrast to those with AD alone (482.93 ± 24.84 pg/mL), showing statistical significance (*P*=0.031). Total serum IgE concentration was also significantly higher in scabies-infested group (371.87 ± 38.65 ng/mL) compared to AD-only group (288.94 ± 45.83 ng/mL) (*P*=0.028). These findings suggest an enhanced Th2 and Th1 immune response in patients with concurrent scabies infestation and AD.

3.3 ROC Curve Analysis Results

The ROC analysis was made, after which the subcurved area (AUC) of IFN-γ was calculated at 0.870 with sensitivity 86%, specificity 88% (95%CI: 0.78-0.94), indicating differentiation between patient groups. A high AUC value of 0.850 was observed for IL-5 (sensitivity 78%, specificity 82%) (95%CI: 0.76-0.92), reflecting strong diagnostic ability. For this reason, a diagnostic evaluation of immune signals and differentiation between patients suffering from chronic scabies and atopic inflammation was performed. Moderate performance was recorded for IL-13, with an AUC of 0.730 with sensitivity 68%, specificity 73% (95%CI: 0.62-0.82), suggesting a fair ability to differentiate between AD and scabies + AD cases. In contrast, total IgE demonstrated a poor diagnostic capacity with an AUC of 0.547 with sensitivity 52%, specificity 58% (95%CI: 0.44-0.65), which was only slightly above the chance line. The IL-4 marker yielded the lowest AUC value of 0.476 with sensitivity 45%, specificity 50%) (95%CI: 0.38-0.57), indicating no useful discriminative value. These findings suggest that IL-5, IFN-γ, and to a lesser extent IL-13, may serve as useful immunological markers in identifying scabies infestation among patients already diagnosed with AD (Figure 2).

The relationships between Th2- and Th1-related cytokines and total IgE were evaluated in patients with AD alone and in patients with scabies associated with AD. The correlation analysis demonstrated distinct immunological interaction patterns between the two patient groups (Table 3).

In patients with AD, positive correlations were observed between Th2 cytokines, particularly IL-4, IL-5, and IL-13, and total IgE levels. This finding connotes that the production of IgE was tightly coupled with that of Th2 immunization, which is known to be a symptom of atopic dermatitis. The IL-4 and IL-13 are known to promote class switching of B-cells to IgE, and IL-5 is a key player in the activation of eosinophil and survival. Thus, the noted correlations indicate that allergic inflammation and Th2-mediated immune response were largely responsible for the pathogenesis in AD response.

In contrast, patients with scabies associated with AD exhibited stronger and more significant correlations between Th2 cytokines and total IgE levels. This enhancement suggests that infestation with *S. scabiei* intensified Th2 polarization. Parasitic antigens are known to stimulate exaggerated Th2 responses, leading to increased IgE synthesis and eosinophilia. Consequently, the immune response in Scabies + AD patients appeared to be amplified beyond that observed in AD alone.

Table 1. Relationship between type of skin lesion and the presence of *S. scabiei* infestation.

Skin lesion	Patient With Scabiei	Patient without Scabiei	Total
Burrows	16	6	22
Popularvascular	7	11	18
intense pruritis	3	7	10
Total	26	24	50
Statistical analysis		X^2= 6.965; Df= 2; P=0.0376	

X^2= chi square test; Df=degree of freedom; P=probability

Table 2. Serum cytokine concentrations and total IgE levels in patients with AD and those with scabies + AD

Marker	Atopic Dermatitis (AD)	Scabies + AD	P-value
IL-4 pg/mL	154.63± 11.45	176.38 ± 9.27	0.143
IL-13 pg/mL	188.42± 22.93	201.71±19.93	0.066
IL-5 pg/mL	65.78± 8.93	101.34± 11.38	0.043
IFN-γ pg/mL	482.93±24.84	837.62±19.74	0.031
Total IgE ng/mL	288.94 ± 45.83	371.87± 38.65	0.028

Table 3. Correlation of immunological markers in AD and scabies + AD patients.

Marker Pair	AD patients (r)	P-value	Scabies + AD patients (r)	P-value
IL-4 vs IgE	0.42	0.031*	0.61	0.004**
IL-5 vs IgE	0.48	0.019*	0.69	0.001**
IL-13 vs IgE	0.36	0.067	0.54	0.012*
IFN-γ vs IgE	−0.29	0.112	−0.47	0.028*
IL-4 vs IL-5	0.51	0.014*	0.73	<0.001**

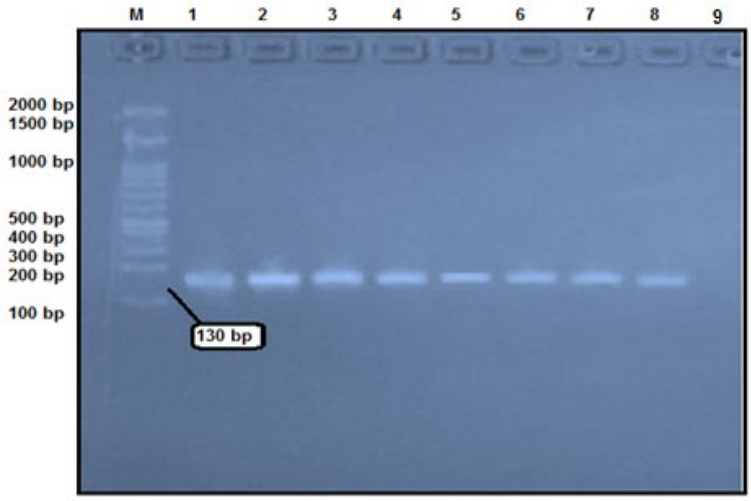

Figure 1. Agarose gel electrophoresis for *S. scabiei* detection; M represents DNA marker 100 bp; Wells 1-8 represent positive PCR products for *S. scabiei* with 130 bp. The well 9 represents negative control sample **(Prepared by Authors, 2026)**.

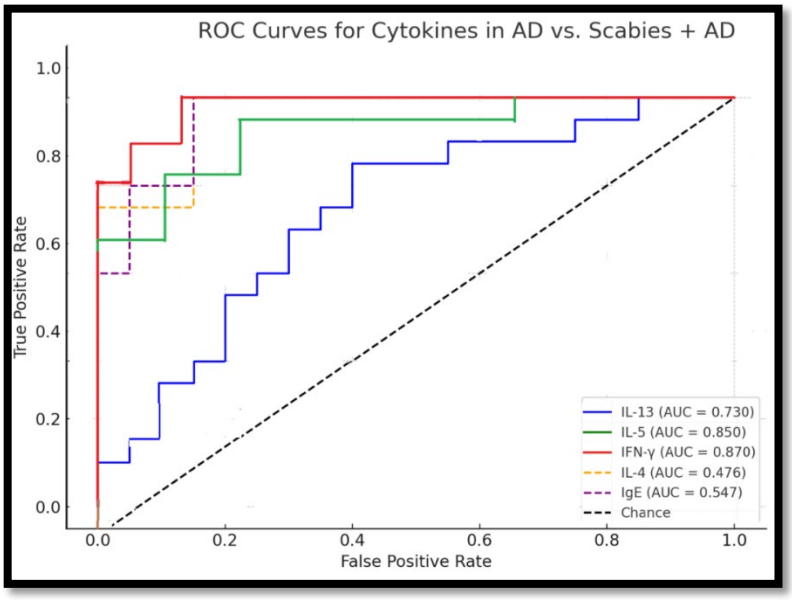

Figure 2. Roc curve analysis for the cytokines and IgE levels. The ROC curve shows true positive rates for AD vs. scabies + AD groups **(Prepared by Authors, 2026)**.

4. Discussion

In the present study, scabies infestation was successfully confirmed in all skin scraping samples using both conventional microscopic examination and molecular PCR-based detection. The concordance between the two diagnostic methods indicates a high level of diagnostic accuracy and supports the reliability of combining morphological and molecular techniques for scabies identification **(16)**. Microscopic examination remains a widely used diagnostic approach due to its simplicity and low cost; however, its sensitivity has been reported to vary depending on parasite burden and operator expertise **(17)**.

This study measured the immunological characteristics of AD patients that were co-infested with *S. scabiei*, with particular emphasis on key cytokines and IgE. It is worth noting that co-infected group showed increased immunoreactivity, especially in the shape of increased Th2 and Th1 cytokine responses **(18)**. The levels of IL-5 were tremendously high among scabies-infected patients, and this is consistent with the action of IL-5 in recruiting eosinophils. A feature characteristic of helminth and ectoparasite-induced is activation of inflammation **(19)**. The co-infected group also demonstrated an increase in the IFN-γ level. At the same time, Th1-mediated response to scabies infestation is a mixed-type (Th1/Th2) response **(20)**. This dual exacerbation of the skin pruritus and inflammation can be the result of response pattern in co-infected patients. Though the IL-13 difference level was not significant, its moderate AUC and greater mean concentration in scabies + AD group indicate a possible contributory. Contributing to the intensification of allergic inflammation, IL-13 has been involved in skin barrier dysfunction as well as fibrosis that may aggravate the clinical expression of infested patients with AD **(21)**.

To the contrary, the level of total IgE did not significantly vary across the groups though it was high in co-infected patients. This observation implies that IgE can be increased because of chronic allergic stimulation but it is not specific enough to differentiate scabies infestation in the presence of AD **(22)**. Likewise, IL-4 did not show a significant diagnostic value, which is indicated by low AUC values and insignificant difference **(22, 23)**.

The ROC analysis also contributed to the diagnostic significance of IL-5, IFN-γ and to a lesser degree, IL-13, pointing to their possible use as biomarkers in the detection of scabies infestation in AD patients. These findings are in agreement with earlier studies that have demonstrated the presence of both Th1 and Th2 pathways during scabies infestation **(24)**, which makes immunological situation more difficult in those patients who have underlying conditions of atopy **(25, 26)**.

Furthermore, a significant correlation between IFN-γ and IgE was observed in the scabies + AD group, whereas a weaker or non-significant association was noted in AD patients. The elevated IFN-γ observed in our AD patients with scabies may also be a direct reflection of the host's immune response to *S. scabiei* var. hominis.

The immune response to scabies is not static; it evolves over the course of infection. Early infestation

is marked by Th1-polarized response, which is critical for initial mite control. IFN-γ is classically considered a Th1 cytokine that counter-regulates Th2 responses **(27)**; however, chronic parasitic infections have been shown to induce a mixed Th1/Th2 immune profile. Therefore, the association of IFN-γ with IgE in scabies + AD patients may reflect immune dysregulation caused by persistent antigenic stimulation, rather than effective Th1-mediated suppression **(28)**.

Additionally, stronger inter-cytokine correlations (e.g., IL-4 with IL-5) were detected in scabies + AD group. This finding suggests a more synchronized cytokine network, possibly induced by continuous immune activation due to mite infestation. Such immune amplification may contribute to disease severity, prolonged inflammation, and poor clinical outcomes in the patients with concurrent scabies and AD **(29)**. The combined use of microscopic examination and PCR-based detection is particularly valuable in epidemiological and clinical studies, as it ensures the accurate diagnosis and strengthens the interpretation of immunological findings associated with scabies infestation **(30, 31)**.

Overall, the findings indicate that the immune response in AD patients is significantly altered by scabies infestation, resulting in a more complex cytokine milieu. The immune changes evaluation helps to develop accurate diagnostic methods and determine treatment.

This study has several limitations. The relatively small sample size and recruitment of participants from a single teaching hospital in Al-Diwaniyah, Iraq, may limit the generalizability of the findings. In addition, challenges associated with patient recruitment and sample collection in the study setting may have affected the study population. Furthermore, the cross-sectional design and single-time-point measurement of cytokines preclude assessment of temporal changes and causal relationships between *Sarcoptes scabiei* infestation and immune responses in patients with atopic dermatitis. Therefore, larger multicenter prospective studies are warranted to validate these findings.

5. Conclusion

The present study demonstrated that patients with atopic dermatitis co-infected with *S. scabiei* exhibited significantly elevated levels of IL-5, IFN-γ, and total IgE as compared to the patients with atopic dermatitis alone. ROC analysis further indicated that IL-5 and IFN-γ showed good discriminative ability, while IL-13 demonstrated moderate diagnostic performance. These findings suggest that scabies infestation may intensify immune dysregulation and promote a mixed Th1/Th2 cytokine profile in affected patients. Measurement of selected cytokines, particularly IL-5 and IFN-γ, may assist in identifying scabies infestation in individuals with underlying atopic dermatitis. However, larger multicenter studies are recommended to validate these immunological markers.

6. Declarations

6.1 Acknowledgment

The authors would like to thank University of Al-Qadisiyah, Al Diwaniyah, Iraq, for its support in the present work.

6.2 Ethical Considerations

The ethical approval was granted by the local institutional review board in Department of Medical and Basic Science, College of Nursing, University of Al-Qadisiyah (Approval No. REC201/2025, dated September 11, 2025). Written informed consent was obtained from all participants or their legal guardians prior to their inclusion in the study.

6.3 Authors' Contributions

H.M.K: Conceptualized the study, collected patient and control samples, performed laboratory analyses, and contributed to data interpretation and initial drafting. A.K.M. and H.R.F: Assisted in laboratory and statistical analyses, contributed to data interpretation, methodology development, and manuscript editing. All authors read and approved the final manuscript.

6.4 Conflict of Interests

The authors declare no conflict of interest.

6.5 Financial Support and Sponsorship

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6.6 Using Artificial Intelligence Tools (AI Tools)

The researchers refrained from employing artificial intelligence tools.

References

1. Lin Y, Fang K, Zheng Y, Wang HL, Wu J. Global burden and trends of neglected tropical diseases from 1990 to 2019. J Travel Med. 2022; 29(3):taac031. [DOI:10.1093/jtm/taac031] [PMID]
2. Chosidow O. Scabies. N Engl J Med. 2006; 354(16):1718-27. [DOI:10.1056/NEJMcp052784] [PMID]
3. Walton SF, Currie BJ. Problems in diagnosing scabies, a global disease in human and animal populations. Clin Microbiol Rev. 2007;20(2): 268-79. [DOI:10.1128/CMR.00042-06] [PMID] [PMCID]
4. Napolitano M, Di Vico F, Ruggiero A, Fabbrocini G, Patruno C. The hidden sentinel of the skin: An overview on the role of interleukin-13 in atopic dermatitis. Front Med. 2023;10: 1165098. [DOI:10.3389/fmed.2023.1165098] [PMID] [PMCID]
5. Fagundes BO, de-Sousa TR, Victor JR. Gamma-delta (γδ) T cell-derived cytokines (IL-4, IL-17, IFN-γ and IL-10) and their possible implications for atopic dermatitis development. Int J Dermatol. 2023;62(4):443-8. [DOI:10.1111/ijd.16364] [PMID] [PMCID]
6. Bhat SA, Mounsey KE, Liu X, Walton SF. Host immune responses to the itch mite, Sarcoptes scabiei, in humans. Parasit Vectors. 2017;10(1): 385. [DOI:10.1186/s13071-017-2320-4] [PMID] [PMCID]
7. Engelman D, Yoshizumi J, Hay RJ, Osti M, Micali G, Norton S, et al. The 2020 international alliance for the control of scabies consensus criteria for the diagnosis of scabies. Br J Dermatol. 2020;183(5):808-20. [DOI:10.1111/bjd.18943] [PMID] [PMCID]
8. Torres T, Ferreira EO, Gonçalo M, Mendes-Bastos P, Selores M, Filipe P. Update on atopic dermatitis. Acta Med Port. 2019;32(9):606-13. [DOI:10.20344/amp.11963] [PMID]
9. Guttman-Yassky E, Krueger JG, Lebwohl MG. Systemic immune mechanisms in atopic dermatitis and psoriasis with implications for treatment. Exp Dermatol. 2018;27(4):409-17. [DOI:10.1111/exd.13336] [PMID]
10. Tsakok T, Woolf R, Smith CH, Weidinger S, Flohr C. Atopic dermatitis: the skin barrier and beyond. Br J Dermatol. 2019;180(3):464-74. [DOI:10.1111/bjd.16934] [PMID]
11. Cohen PR. Scabies masquerading as bullous pemphigoid: scabies surrepticius. Clin Cosmet Investig Dermatol. 2017;10:317-24. [DOI:10.2147/CCID.S145494] [PMID] [PMCID]
12. Gazi U, Taylan-Ozkan A, Mumcuoglu KY. Immune mechanisms in human Sarcoptes scabiei (Acari: Sarcoptidae) infestations. Parasite Immunol. 2022;44(1-2):e12900. [DOI:10.1111/pim.12900] [PMID]
13. Al-Mendalawi MD, Ibrahim JG. Pattern of dermatoses in Iraqi children. East Mediterr Health J. 2012;18(4):365-371. [DOI:10.26719/2012.18.4.365] [PMID]
14. Abd El-Aal AA, Hassan MA, Gawdat HI, Ali MA, Barakat M. Immunomodulatory impression of anti and pro-inflammatory cytokines in relation to humoral immunity in human scabies. Int J Immunopathol Pharmacol. 2016;29(2):188-94. [DOI:10.1177/0394632015627464] [PMID] [PMCID]
15. Gavrilita E, Silion SI, Bitca ML, Tatu AL. Insights into Intrinsic Atopic Dermatitis: immunogenicity, Dysbiosis, and Imaging (Reflectance Confocal Microscopy, Optical Coherence Tomography). Clin Cosmet Investig Dermatol. 2024;17:1377-86. [DOI:10.2147/CCID.S459096] [PMID] [PMCID]
16. Abidov K, Khaitov K, Abidova Z, Abidov A. Diagnostic value of dermatoscopy in atopic dermatitis in children. Sci Innov. 2023;2(D7):48-54.
17. Taylor S, Hales BJ, Thomas WR. Host immune response to scabies. InScabies 2023 Oct 26 (pp. 45-73). Cham, Switzerland: Springer International Publishing. [DOI:10.1007/978-3-031-26070-4_4]
18. Siddig EE, Hay R. Laboratory-based diagnosis of scabies: a review of the current status. Trans Royal Soc Trop Med Hygiene. 2022;116(1):4-9. [DOI:10.1093/trstmh/trab049] [PMID] [PMCID]
19. Mounsey KE, Murray HC, Bielefeldt-Ohmann H, Pasay C, Holt DC, Currie BJ, et al. Prospective study in a porcine model of Sarcoptes scabiei indicates the association of Th2 and Th17 pathways with the clinical severity of scabies. PLoS Negl Trop Dis. 2015;9(3):e0003498. [DOI:10.1371/journal.pntd.0003498] [PMID] [PMCID]
20. Upadhyay PR, Seminario-Vidal L, Abe B, Ghobadi C, Sims JT. Cytokines and epidermal lipid abnormalities in atopic dermatitis: a

systematic review. Cells. 2023;12(24):2793. [DOI:10.3390/cells12242793] [PMID] [PMCID]

21. Tamamoto-Mochizuki C, Santoro D, Saridomikelakis MN, Eisenschenk MN, Hensel P, Pucheu-Haston C, et al. Update on the role of cytokines and chemokines in canine atopic dermatitis. Vet Dermatol. 2024;35(1):25-39. [DOI:10.1111/vde.13192] [PMID]
22. Sugaya M. The role of Th17-related cytokines in atopic dermatitis. Int J Mol Sci. 2020;21(4): 1314. [DOI:10.3390/ijms21041314] [PMID] [PMCID]
23. Szymański Ł, Cios A, Ciepielak M, Stankiewicz W. Cytokines and apoptosis in atopic dermatitis. Adv Dermatol Allergol. 2021;38(1): 1-3. [DOI:10.5114/ada.2019.88394] [PMID] [PMCID]
24. Nomura I, Goleva E, Howell MD, Hamid QA, Ong PY, Hall CF, et al. Cytokine milieu of atopic dermatitis, as compared to psoriasis, skin prevents induction of innate immune response genes. J Immunol. 2003;171(6):3262-9. [DOI:10.4049/jimmunol.171.6.3262] [PMID]
25. Katsarou S, Makris M, Vakirlis E, Gregoriou S. The role of tight junctions in atopic dermatitis: a systematic review. J Clin Med. 2023;12(4): 1538. [DOI:10.3390/jcm12041538] [PMID] [PMCID]
26. Scheurer J, Sauer B, Focken J, Giampetraglia M, Jäger A, Schürch CM, et al. Histological and functional characterization of 3D human skin models mimicking the inflammatory skin diseases psoriasis and atopic dermatitis. Dis Model Mech. 2024;17(1):dmm050541. [DOI:10.1242/dmm.050541] [PMID] [PMCID]
27. Choi Y, Park KY, Han HS, Lee MK, Seo SJ. Comparative analysis of cutaneous fungi in atopic dermatitis patients and healthy individuals. Ann Dermatol. 2022;34(2):118-25. [DOI:10.5021/ad.2022.34.2.118] [PMID] [PMCID]
28. Yamamura Y, Nakashima C, Otsuka A. Interplay of cytokines in the pathophysiology of atopic dermatitis: insights from Murin models and human. Front Med. 2024;11:1342176. [PMID] [DOI:10.3389/fmed.2024.1342176] [PMCID]
29. Kim HO. Targeting cytokines and signaling molecules related to immune pathways in atopic dermatitis: therapeutic implications and challenges: H.-O. Kim. Arch Pharm Res. 2022; 45(12):894-908. [PMID] [DOI:10.1007/s12272-022-01421-2]
30. Santamaria-Babi LF. Atopic dermatitis pathogenesis: lessons from immunology. Dermatol Pract Concept. 2022;12(1):e2022152. [DOI:10.5826/dpc.1201a152] [PMID] [PMCID]
31. Krupka-Olek M, Bozek A, Drewienkowska M, Dryjka K, Medrala A. Immunological differences between atopic dermatitis, psoriasis, and their combination in adult patients. Allergol Immunopathol. 2022;50(4):143-6. [DOI:10.15586/aei.v50i4.557] [PMID]