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ORIGINAL RESEARCH MICROBIOLOGY

Immunological evaluation of serum CXCL1 and interleukin-34 and their correlation with IL-34 gene polymorphism in patients with systemic lupus erythematosus

Athraa Abd ALnabi Musa [1], Aseel Shakir Mahmood [2]

Authors affiliations:

1. Athraa Abd ALnabi Musa, Department of Biotechnology, College of Science, University of Baghdad, Baghdad, Iraq; Email: azraa.abdulnabi1706b@sc.uobaghdad.edu.iq; ORCID: https://orcid.org/0009-0003-7064-8638
2. Aseel Shakir Mahmood, Department of Biotechnology, College of Science, University of Baghdad, Baghdad, Iraq; Email: Aseel.mahmood@sc.uobaghdad.edu.iq; ORCID: https://orcid.org/0009-0007-1607-5073

Correspondence: Athraa Abd ALnabi Musa**; E-mail:** azraa.abdulnabi1706b@sc.uobaghdad.edu.iq

ABSTRACT

Background & objective: Systemic lupus erythematosus (SLE) is a heterogeneous autoimmune disease. The chemokine CXCL1 and interleukin-34 (IL-34) are implicated in inflammatory and immune responses, but their combined role and genetic regulation in SLE remain unclear. This study aimed to investigate serum levels of CXCL1 and IL-34, and the association of IL-34 gene polymorphism (rs7193968) with these markers and disease parameters in SLE patients.

Methodology: A case-control study was conducted on 50 SLE patients and 50 healthy controls. Serum levels of CXCL1 and IL-34 were measured by ELISA. Genotyping for the IL-34 rs7193968 G/C polymorphism was performed using PCR and sequencing. Statistical analyses included t-tests, chi-square tests, ROC analysis, and logistic regression.

Results: Serum levels of CXCL1 (125.42 ± 97.55 vs. 53.36 ± 34.60 pg/mL, (P < 0.0001) and IL-34 (23.30 ± 14.16 vs. 12.58 ± 9.89 pg/mL, P < 0.0001) were significantly elevated in SLE patients. The GG genotype and G allele of rs7193968 were significantly more prevalent in patients (P = 0.0020) and correlated with elevated levels of CXCL1, ANA, and anti-dsDNA. ROC analysis demonstrated significant diagnostic capability for CXCL1 (AUC=0.85) and IL-34 (AUC=0.81). Multivariate regression analysis identified all three biomarkers as autonomous predictors of SLE.

Conclusion: Elevated serum CXCL1 and IL-34 are significantly associated with SLE, and the IL-34 rs7193968 G allele is linked to increased disease susceptibility and severity. These molecules represent promising biomarkers and highlight a potential genetic-immunological axis in SLE pathogenesis.

Keywords: Chemokine CXCL1; Genetic Polymorphism. Interleukin-34; Lupus Erythematosus;

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1. INTRODUCTION

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease and a multifactotial systemic inflammation that can strike various organ systems. The clinical picture of SLE is characterized by a rather unpredictable course consisting of alternating remission and disease flares.[1] Differences in clinical phenotype of the disease may be explained by a complicated mechanism including autoantibodies production,

complement activation and immune-complexes deposition into tissues. [2] It is generally recognized that the pathogenesis of SLE includes genetic susceptibilities and environmental triggers.[3,4]

Cytokines and chemokines are key signaling molecules involved in the induction of the inflammatory and immune responses driving autoimmunity such as that seen in SLE.[5] Interleukin-34 (IL-34) is a new cytokine which has been found in relatively recent years, considered as crucial player in the initiation and resolution of immune-inflammatory responses. [6,7] In addition, Chemokine (C-X-C motif) ligand 1 (CXCL1), one of the members of chemokine family, exerts regulatory effects on a number of biological activities including inflammation, angiogenesis and tissue repair.[8,9]

In SLE, there is growing evidence that CXCL1 could help drive production of a wide range of autoantibodies, particularly during active disease. Dysregulation of chemokines such as CXCL1 may thus be a candidate biomarker for the identification of individuals at risk or to monitor disease activity in SLE.[10,11] Speculations have also been made to the possible involvement of CXCL1 in lupus nephritis (LN), a severe SLE complication. This has been suggested to involve recruitment of immune cells in the kidney and activation of local effector immune cells with marked renal damage. An amplification circuit has also been created in which the leukocyte infiltrating serves as a secondary source of chemokine production, continuing the cycle of inflammation.[12,13]

Based on the roles of these mediators in separate parts of immune response, this work was to synchronously analyze the serum levels of CXCL1 and IL-34 in SLE patients. The primary goal was to assess whether these levels are higher than those found in healthy subjects and investigate the potential relationship between these cytokine concentrations and a particular genetic polymorphism located in the IL-34 gene.

2. METHODOLOGY

This study was designed as a case–control investigation. The study included 100 patients who were divided into two groups: group (A) which comprised of 50 patients with confirmed diagnosis of SLE, and group (B) which included 50 healthy matched controls regarding age and sex. SLE diagnosis of each patient was affirmed by a consultant dermatologist according to the 2023 ACR classification criteria. The study was approved by the Research Ethical Committee of University of Baghdad and the Iraqi Ministry of Health (Approval number: CSEC/0725/0084). Written informed consents were obtained from all participants prior to enrollment in the study.

The study was carried out in the Department of Biotechnology, College of Science, University of Baghdad. Patient recruitment and sample collection were conducted from November 2024 to March 2025 at two major teaching hospitals in Baghdad: Baghdad Teaching Hospital and Al-Yarmouk Teaching Hospital.

The patient group included adult individuals (≥18 years) with a confirmed diagnosis of SLE. Exclusion criteria for both patients and controls encompassed the presence of chronic disorders (e.g., hepatic, renal, thyroid, endocrine, or cardiac diseases), active infections, other types of malignancies, and cases with incomplete clinical or demographic data.

2.1. Sample collection and serum preparation

Three milliliters of venous blood were taken from each participant. Blood was put into a plain tube, let it clot at room temperature, and then spun at 3000 rpm for 15 minutes at 4°C to separate the serum. The serum samples were kept at -20°C until they were ready to be tested.

2.2. Measurement of serum CXCL1 and IL-34 levels

The serum concentrations of CXCL1 and Interleukin-34 (IL-34) were quantified using ELISA kits (FineTest®, Wuhan Fine Biotech Co., Ltd, China; Catalogue No. EH0005 for CXCL1 and EH2043 for IL-34). All assays were performed in strict accordance with the manufacturer's protocols.

2.3. Genetic analysis

Genomic DNA was extracted from whole blood collected in EDTA tubes using the EasyPure® Blood Genomic DNA Kit (Cat. No. EE121). The purity and concentration of the extracted DNA were assessed prior to polymerase chain reaction (PCR) amplification. The primers for genotyping the IL-34 gene were designed as follows: forward primer 5'-TCAGGGGTGTTGTGGTAGAC-3' and reverse primer 5'-CTGGACGTGCATTTTCCCA-3'. The PCR reaction was prepared in a total volume of 25 µL, which included 8.5 µL of nuclease-free distilled water, 2 µL of the DNA sample (at a concentration of 50 ng), 1 µL of each primer (10 µM), and 12.5 µL of GoTaq® Green Master Mix. The PCR cycling conditions were as follows: an initial denaturation step at 95°C for 5 minutes, followed by 35 cycles of denaturation at 95°C for 30 se, annealing at

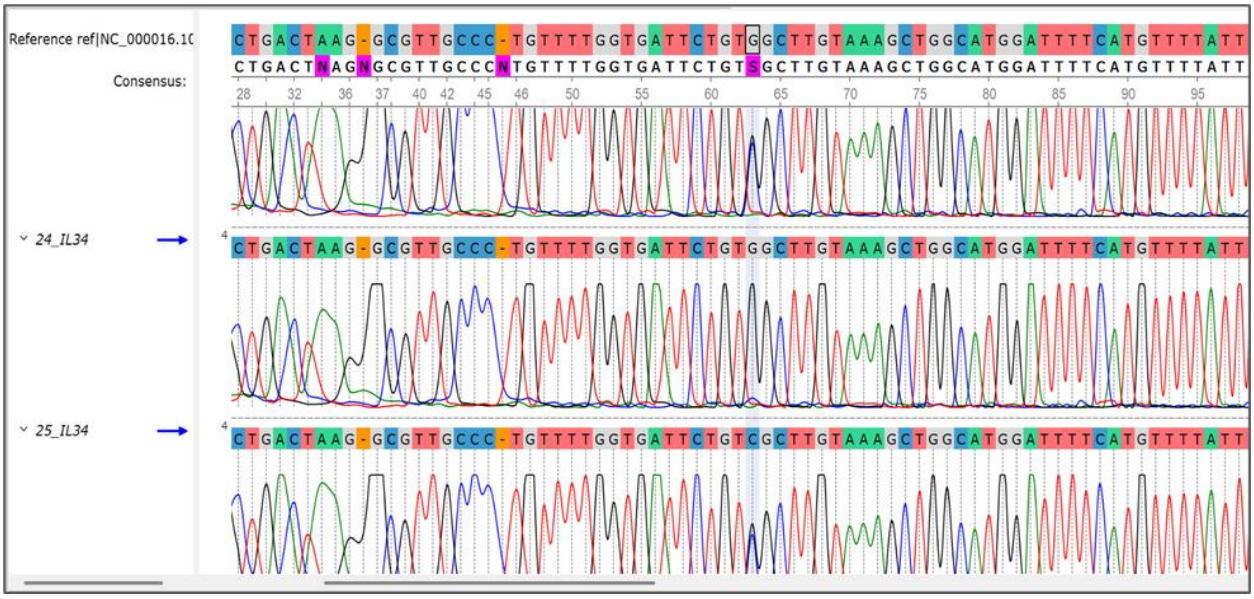

Figure 1: IL-34 rs7193968 G/C SNP DNA sequence chromatogram displaying GG ,GC and CC genotypes.

Table 1: Baseline Characteristics of Study Participants

Characteristic	SLE Group (n=50)	Control Group (n=50)	P-value
Age (years)	34.7 ± 10.9	33.0 ± 11.5	0.398
Sex, n (%)			
Female	42 (84.0%)	38 (76.0%)	0.453
Male	8 (16.0%)	12 (24.0%)	
BMI (kg/m²)	27.87 ± 5.75	25.45 ± 3.31	0.089
Smoking status, n (%)			
Non-smoker	42 (84.0%)	40 (80.0%)	0.795
Smoker	8 (16.0%)	10 (20.0%)	
Disease duration (years)	8.88 ± 7.87	N/A	—

Chi-square test. Mann-Whitney U test

60°C for 30 se, and extension at 72°C for 30 se. The last step of the process was to extend the time at 72°C for 5 min. The amplified PCR products were then sequenced using an ABI 3730xl DNA Analyzer (Macrogen Corporation, Korea) to determine the genotype.

2.4. Statistical analysis

All statistical analyses were conducted using GraphPad Prism software (version 9.4.1). Shapiro-Wilk tests were utilized to examine normality with continuous variables. The data were normally distributed (independent samples t-test) and expressed as mean ± SD for the two groups. For non-normal data the non-parametric Mann-Whitney U test was performed and median (IQR) is presented. Comparison of categorical variables was done by χ^2 test. The diagnostic value of the biomarkers for distinguishing SLE patients from health controls was assessed using receiver operating characteristic (ROC) curve. The above diagnostic precision of AUC was evaluated by 95 % confidence interval (CI). The independent predictors of SLE were further analyzed by multivariable logistic regression model. All tests had a statistical significance level of $P < 0.05$.

3. RESULTS

Demographic and clinical characteristics of the patient group (50 patients with SLE matched by age and sex) and control group (50 healthy individuals from the general population) are shown in Table 1. The average age of SLE group was 34.7 ± 10.9, that of control group was 33.0 ± 11.5, which had no statistical significance between two groups (P = 0.398). Most patients and controls were females (84.0% of the SLE patients vs 76.0% of the controls, P = 0.453). Body mass index (BMI) and smoking were also similar among the two groups (p = 0.089, P = 0.795; respectively). The mean duration of the disease in SLE patients was 8.88 ± 7.87 years.

Significant differences were observed in most hematological parameters (Table 2). SLE patients exhibited lower levels of hemoglobin (10.41 ± 2.86 vs. 12.40 ± 0.90 g/dL, P = 0.0008), red blood cell count (3.61

Table 2: Comparison of Hematological Parameters Between Groups

Parameter	SLE Group (n=50)	Control Group (n=50)	Mean Difference	P-value
Platelet count (×10³/μL)	169.52 ± 47.26	194.42 ± 30.86	-24.90	0.0147
Hemoglobin (g/dL)	10.41 ± 2.86	12.40 ± 0.90	-1.99	0.0008
Red blood cell count (×10⁶/μL)	3.61 ± 0.75	4.10 ± 0.40	-0.49	0.0001
Hematocrit (%)	0.37 ± 0.08	0.41 ± 0.04	-0.04	0.0860
MCV (fL)	85.57 ± 7.09	89.73 ± 4.39	-4.16	0.0044
MCH (pg)	28.55 ± 3.29	29.89 ± 1.67	-1.34	0.1249
MCHC (g/L)	325.74 ± 30.78	337.40 ± 11.43	-11.66	0.3087
White blood cell count(×10³/μL)	10.00 ± 2.04	6.32 ± 1.43	3.69	<.0001
Lymphocyte count (×10³/μL)	3.32 ± 0.83	2.40 ± 0.66	0.92	<.0001
Granulocyte count (×10³/μL)	5.46 ± 1.00	4.71 ± 1.04	0.75	0.0001
Mid-range cell count (×10³/μL)	1.37 ± 0.31	1.01 ± 0.36	0.36	0.0001

Table 3: Comparison of Immunological and Inflammatory Markers

Biomarker	SLE Group (n=50)	Control Group (n=50)	Mean Difference (95% CI)	P-value	Effect Size (Cohen's d)
IL-34 (pg/mL)	23.30 ± 14.16	12.58 ± 9.89	10.72 (5.93, 15.51)	<.0001	0.878
CXCL1 (pg/mL)	125.42 ± 97.55	53.36 ± 34.60	72.06 (43.37, 100.75)	<.0001	0.985
Anti-dsDNA (IU/mL)	61.86 ± 18.56	22.38 ± 10.46	39.48 (33.57, 45.39)	<.0001	2.620
ESR (mm/hr)	41.72 ± 22.33	13.35 ± 4.03	28.37 (22.08, 34.66)	<.0001	1.768
ANA (U/mL)	2.48 ± 1.61	0.55 ± 0.25	1.93 (1.48, 2.38)	<.0001	1.677

Table 4: IL-34 rs7193968 G/C Genotype Distribution and Association with SLE

Genotype/Allele	SLE Group n (%)	Control Group n (%)	Odds Ratio	P-value
Genotype				
GG	20 (40.0%)	6 (12.0%)	1.00 (Reference)	
GC	24 (48.0%)	28 (56.0%)	0.26 (0.09-0.74)	0.0192*
CC	6 (12.0%)	16 (32.0%)	0.11 (0.03-0.42)	
Allele Frequency				
G	0.640	0.400	1.00 (Reference)	0.0011**
C	0.360	0.600	0.38 (0.21-0.66)	
Overall Test	$\chi^2 = 12.392$	P = 0.0020	df = 2	

± 0.75 vs. 4.10 ± 0.40 ×10⁶/μL, P = 0.0001), platelet count (169.52 ± 47.26 vs. 194.42 ± 30.86 ×10³/μL, P = 0.0147), and mean corpuscular volume (85.57 ± 7.09 vs. 89.73 ± 4.39 fL, P = 0.0044). Conversely, white blood cell count (10.00 ± 2.04 vs. 6.32 ± 1.43 ×10³/μL, P < 0.0001), lymphocyte count (3.32 ± 0.83 vs. 2.40 ± 0.66 ×10³/μL, P < 0.0001), granulocyte count (5.46 ± 1.00 vs. 4.71 ± 1.04 ×10³/μL, P = 0.0001), and mid-range cell count (1.37 ± 0.31 vs. 1.01 ± 0.36 ×10³/μL, P = 0.0001) were significantly elevated in SLE patients.

As shown in Table 3, All evaluated markers were significantly elevated in the SLE group. Specifically, IL-34 levels were 23.30 ± 14.16 pg/mL in SLE patients compared to 12.58 ± 9.89 pg/mL in controls (P < 0.0001,

Table 5: Diagnostic Performance Characteristics of Biomarkers

Biomarker	AUC (95% CI)	Optimal Cutoff	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	Accuracy (%)
IL-34	0.81 (0.73-0.90)	15.29	78.0	82.0	81.2	78.8	80.0
CXCL1	0.85 (0.77-0.93)	66.84	90.0	72.0	76.3	87.8	81.0
Anti-dsDNA	0.96 (0.93-1.00)	29.00	98.0	94.0	94.2	97.9	96.0

Cohen's d = 0.878). CXCL1 concentrations were also

Table 6: Association Between IL-34 rs7193968 Genotype and Biomarker Levels in SLE Patients

Biomarker	GG (n=20)	GC (n=24)	CC (n=6)	P-value
Interleukin-34 (pg/mL)	20.36 ± 8.94	25.03 ± 18.06	26.18 ± 10.01	0.4702
CXCL1 (pg/mL)	102.51 ± 40.63	116.65 ± 62.28	236.82 ± 227.53	0.4223
Anti-dsDNA (IU/mL)	58.43 ± 20.36	64.71 ± 17.70	61.90 ± 16.59	0.5741

markedly higher in SLE patients (125.42 ± 97.55 vs.

Table 7: Multivariate Logistic Regression Analysis for SLE Prediction

Predictor Variable	β Coefficient	Odds Ratio	95% CI for OR	P-value
Interleukin-34 (pg/mL)	0.4845	1.623	(1.000, 2.635)	0.05*
CXCL1 (pg/mL)	1.4655	4.330	(1.000, 18.747)	0.05*
Anti-dsDNA (IU/mL)	1.7907	5.994	(1.000, 35.926)	0.05*
*Model Performance: Accuracy = 97.0%, Precision = 98.0%, Recall = 96.0%, F1-Score = 97.0%, AUC = 0.997. Significance levels: * (P ≤ 0.05).*				

53.36 ± 34.60 pg/mL, P < 0.0001, Cohen's d = 0.985). Anti-dsDNA antibody levels showed the largest effect size (Cohen's d = 2.620), with SLE patients exhibiting

Table 6 Relationship between IL-34 rs7193968 genotypes and serum biomarker levels in patients with SLE. For IL-34 (PP = 0.4702), CXCL1 (p = 0.4223), anti-dsDNA (P = 0.5741), no statistically significant differences between genotypes GG, GC, CC were observed.

The results of a multivariate logistic model that was constructed to identify independent predictors of SLE are shown in Table 7. There were three biological markers (IL-34, CXCL1, and anti-dsDNA) as predictors. All three were identified as significant independent predictors of SLE (P = 0.05). The odds ratios were 1.623 for IL-34 (95% CI, 1.000–2.63), 4.330 for CXCL1 (95% CI, 1.000–18.747), and 5.994 for anti-dsDNA (95% CI: 1.000–35:926). The model achieved good performance indexes (accuracy = 97.0%, precision = 98.0%, recall = 96.0%, F1-score = 97.0% and AUC - 0,997).

4.

DISCUSSION

The present study provides a significant contribution to the understanding of the immunopathology of SLE by concurrently evaluating serum levels of chemokine CXCL1 and IL-34, and exploring the influence of an IL-34 gene polymorphism (rs7193968). The principal findings reveal that both CXCL1 and IL-34 are significantly elevated in SLE patients compared to healthy controls, and importantly, that the G allele of the rs7193968 polymorphism is associated with an increased risk of SLE and correlates with higher levels of CXCL1, ANA, and anti-dsDNA antibodies.

Our findings of significantly increased CXCL1 in SLE patients corroborate an increasingly convincing hypothesis that this chemokine is a critical mediator in the pathogenesis of lupus. A large study by Zeng et al. (2021) also reported that serum CXCL1 levels were higher in active SLE, and correlated tightly with both the SLE disease activity index (SLEDAI), the presence of LN, and anti-dsDNA antibodies.[14] The current study corroborates these findings, reinforcing the potential of CXCL1 as a robust biomarker for disease activity.

Mechanistically, CXCL1, a potent neutrophil chemoattractant, exerts its effects primarily through the CXCR2 receptor.[15] More recent studies point to the role of neutrophil dysregulation in the pathogenesis of SLE. SLE patient-derived neutrophils have been shown to produce an array of pro-inflammatory cytokines and are the main cellular origin of NETs, which leads to the exposure of autoantigens and further amplification of this autoimmune pathway.[16] The raised circulating level of CXCL1 in our patients may at least partly account to the recruitment and activation of these cytotoxic neutrophils into target organs, especially the kidneys, which has been implicated in pathogenesis of lupus nephritis.[17,18] The discovery of markedly elevated levels of CXCL1 in LN patients further confirms the biological relevance of this chemokine in programming the pro-inflammatory milieu responsible for mediating renal injury.

The increase in serum IL-34 levels in SLE patients described here is supporting evidences that IL-34 appears to be a pivotal cytokine associated with autoimmune diseases. Xie et al. (2018) reported that IL-34 could be detected in most SLE patients, and its levels were positively linked to the SLEDAI score, IgG titer, anti-dsDNA antibody levels and negatively with C3 and hemoglobin.[19] Our observations echo these associations that support a close relationship of IL-34, with the humoral auto-immune response and systemic inflammation in driving the disease activity.

IL-34 functions as an alternative ligand for the colony-stimulating factor-1 receptor (CSF-1R), a key regulator of the survival, differentiation, and function of macrophages and other myeloid cells.[20] By activating CSF-1R, IL-34 can promote the differentiation of monocytes into pro-inflammatory macrophages, which in turn produce other inflammatory mediators like TNF-α and IL-6, thereby amplifying the inflammatory cascade in SLE.[21] Interestingly, while some studies, including ours, show a clear link between IL-34 and overall disease activity, the association with lupus nephritis is less consistent. This may reflect the pleiotropic nature of IL-34, which has been reported to have both pro- and anti-inflammatory functions depending on the specific tissue microenvironment and disease context.[22]

A particularly novel and significant finding of this study is the association of the IL-34 gene polymorphism rs7193968 with SLE. The higher prevalence of the GG genotype and the G allele in the patient group suggests this variant confers susceptibility to the disease. Crucially, the study demonstrates a functional consequence of this polymorphism: a significant correlation with increased serum levels of CXCL1, ANA, and anti-dsDNA. This provides a genetic basis for the observed immunological phenotype in a subset of patients.

Genetic predisposition is a well-established factor in SLE, with numerous polymorphisms in cytokine and chemokine genes being linked to disease risk.[23] These genetic variations can alter protein expression or function, leading to a dysregulated immune response. The data from this study suggest that the rs7193968 polymorphism may lead to increased IL-34 expression or activity, which in turn could drive the production of CXCL1 and promote the B-cell hyperactivity responsible for ANA and anti-dsDNA production. This finding bridges the gap between genetic susceptibility and the specific inflammatory pathways active in SLE, offering a more nuanced understanding of disease heterogeneity.

These results, corroborated by recent literature, provide insight to a pathogenic axis in SLE from predisposing genotype to cytokine abnormalities and cell infiltration. An IL-34 polymorphism could serve as an upstream signal that creates a pro-immune condition. This leads to an induction of IL-34 and CXCL1, allowing for the recruitment and activation of some critical effector cells (e.g., macrophages, neutrophils), in addition to promoting the autoantibody responses characteristic of SLE.

The high accuracy of both CXCL1 and IL-34 (with AUC 0.85 for each) implies them to be a good candidate as components of biomarker panels, improving the currently used anti-dsDNA marker to distinguish SLE patients from healthy individuals (AUC: 0.96). Though anti-dsDNA is a rose and not a mixed blessing in that it will rarely be positive without some lupus activity, CXCL1 (8 and IL-34 could potentially add value to the monitoring of disease activity, prediction of flares and identification of patients particularly likely to benefit from targeted therapies at these pathway.[11]

5. LIMITATIONS

Although the utility of this work is enlightening, there are shortcomings in this study. Because our study is cross-sectional, it does not allow us to establish the causal relationship between risk factors and metabolic syndrome; in addition, the relatively small sample population must be confirmed in larger multiethnic groups. Prospective long-term monitoring of these biomarkers with disease relapses and remission needs to be investigated in longitudinal studies.

6. CONCLUSION

In conclusion, this study provides compelling evidence for the roles of CXCL1 and IL-34 in the pathogenesis of

SLE and highlights their potential as valuable biomarkers for disease activity. The novel identification of an association between the IL-34 rs7193968 polymorphism and key immunological features of SLE offers a significant advancement in our understanding of the genetic underpinnings of the disease. These findings pave the way for further research into personalized medicine approaches for SLE, where therapeutic strategies could be tailored based on the specific genetic and immunological profile of the patient.

7. Availability of data

The datasets used and analyzed during the current study are available from the corresponding author on reasonable request.

8. Acknowledgements

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9. Ethical Clearance

The Research Ethical Committee must give its approval before scientific studies can go ahead in Iraq. The University of Baghdad and the Ministry of Health have both given this work the green light (CSEC/0725/0084).

10. Financial support

Nil.

11. Conflicts of interest

There are no conflicts of interest.

12. Author contributions

A.A.A: Study design, carried out experimental work, acquisition of data, Study conception, analysis of data, proposal writing and drafting and revising the manuscript and submitting the final manuscript. A.S.M: Study design, planned methodology, read, revised and approved the manuscript.

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