University of Thi-Qar Journal of Science (UTJsci)
E-ISSN: 2709-0256, P-ISSN: 1991-8690, Vol. 13, No. 1, Jun. 2026

IFNα and Galectin-9 as Emerging Predictors of Disease Activity in Systemic Lupus Erythematosus

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Received: 29-08-2025, Revised: 11-10-2025, Accepted: 18-10-2025, Published: 01-06-2026

***Abstract*— Systemic lupus erythematosus (SLE) is a multisystem autoimmune disease marked by the loss of immune tolerance to nuclear antigens, predominantly affecting women of reproductive age. The present study aims to assess disease activity in SLE patients and serum concentrations of chemokine (IFNα, Galectin-9) in relation to disease severity. A case-control study was conducted on 100 SLE patients and 25 healthy controls between January 7 and April 5, 2025. Serum IFNα, Galectin-9 levels were measured using a sandwich ELISA. Interferon-alpha levels were significantly higher in SLE patients than in controls ($p < 0.001$), with severe cases showed the highest values (354.8±159.7pg/ml) was elevated in active SLE, with severe cases demonstrated the highest levels compared to mild–moderate disease (4.8±1.2 ng/ml , $p < 0.01$). Clinical manifestations according to SLEDAI revealed that mucocutaneous involvement (54%), musculoskeletal (48%), hematologic (42%), and renal involvement (38%). indicating that chemokine upregulation reflects both systemic immune activation and specific organ involvement. Increased serum levels of IFNα, Galectin-9, especially in severe SLE, underscore their potential role as reliable biomarkers. Their Strong correlation with SLEDAI supports their role in assessing disease activity and monitoring progression.**

Keywords*— *Systemic lupus erythematosus (SLE); Disease activity (SLEDAI-2K); IFNα ;Galectin-9.

I. INTRODUCTION

Systemic lupus erythematosus (SLE) is a chronic autoimmune illness that causes inflammation and immune-mediated harm to various organ systems, including the mucocutaneous, musculoskeletal, hematologic, and renal systems[1]. Its occurrence is more common among women of reproductive age, exhibiting a greater female predominance of 9:1[2]. The precise cause of this illness is not fully comprehended. The development of SLE involves exposure to environmental triggers like UVB rays, infections, and harmful substances causes a breakdown of immune tolerance in individuals with genetic susceptibility and results in unusual activation of autoimmune disease [3] ,leading to the excessive generation of autoantibodies by the B cells and imbalance of cytokines resulting in tissue and organs damage[4-5]. Dendritic cells, especially plasmacytoid dendritic cells (pDCs), are the primary producers of type I interferon (IFN) cytokines, playing a key role in the immunopathogenesis of SLE [6]. Impaired activation or loss of tolerance in DCs not only results in abnormal production of inflammatory mediators and type I interferons, contributing to pathogenic innate immunity and auto inflammation, but also creates an imbalance between effector and regulatory T cell responses and persistent auto-antibody production from B cells, resulting in a perpetually amplified autoimmune pathogenesis in SLE [7].

Interferon alpha (IFNα) plays a crucial role in regulating the immune system. Plasmacytoid dendritic cells play a unique role in generating IFNα. IFNα has the capacity to significantly impact the development, progression, and pathogenesis of SLE, as it can alter the function and activation status of various key immune cell subsets and serve as a link between innate and adaptive immunity[8]. Interferon alpha (IFN- α) belongs to the type I IFN family. It became evident that the overexpression of genes induced by type I IFN was a prevalent dominant trend in human SLE[9]. The interaction of the dendritic cell with IFNα plays a role in T cell polarity. When CD4+ T cells are stimulated alongside IFNα-secreting dendritic cells, their orientation shifts towards producing IFN-γ instead of IL-4, potentially fostering autoimmunity or immune imbalance [10]. In lupus patients, T-reg function is reduced, partially due to the effects of IFNα , suggesting that elevated IFNα levels in these individuals likely play a role in the onset or persistence of autoimmunity by inhibiting T-reg cells [11]. IFN-α stimulates BLyS, thus offering assistance for B cell differentiation, and aids in immunoglobulin class switching to produce possibly disease-causing autoantibodies [12].

Galectin-9 (Gal-9) is a β-galactoside binding lectin recognized for its immunomodulatory function in numerous microbial infections. Gal-9 is found in all organ systems and is situated in the nucleus, on the cell surface, within the cytoplasm, and in the extracellular matrix. It facilitates interactions between hosts and pathogens and modulates cell

https://doi.org/10.32792/utq/utjsci/v13i1.1471

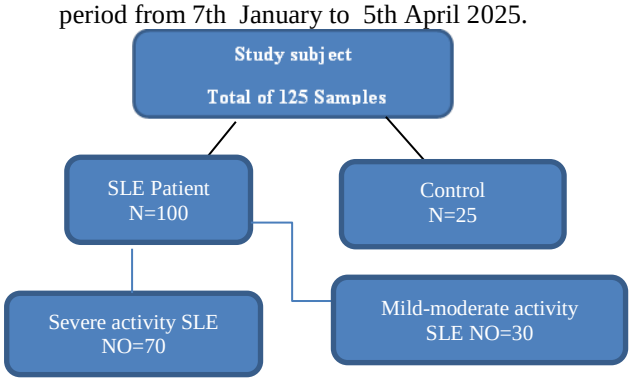
signaling by attaching to its receptors [13]. Galectins primarily reside in lipid rafts on the cell surface, facilitating their binding to glycan receptors or ECM ligands, thus engaging in various biological functions such as inflammation, immune responses (both innate and adaptive), and interactions between cells and ECM [14]. Galectin-9 can affect the differentiation of T cells, especially inhibiting the activation of effector T cells. Galectin-9's capacity to trigger T cell apoptosis may also play a role in the breakdown of self-tolerance [15]. Galectin-9 has been demonstrated to regulate the IFN-α/β pathway, recognized for its impact on several facets of immune cell activity, such as the activation of T cells and B cells. Increased levels of galectin-9 might enhance the inflammatory reactions caused by the type I IFN pathway [16].

The aim of the study is to determine the activity of SLE using SLEDAI-2K, measure serum levels of (IFN-α, Galectin-9) and compare study groups according to disease Activity, as well as the correlation between (IFN-α, Galectin-9) levels and disease activity in patients with systemic lupus erythematosus .

II. METHODOLOGY

A. *Study design:*

This case-control study was conducted during the period from 7th January to 5th April 2025.



B. *Study population:*

Patient samples and data were collected from the Rheumatology Unit of Baghdad Teaching Hospital, and the Rheumatology Unit of Surgical Specialties Hospital in Medical City, Baghdad, Iraq, The diagnosis was established by a rheumatologist using the American College of Rheumatology's (ACR) updated criteria for SLE, as well as the criteria for The Systemic Lupus Erythematosus Disease Activity Index (SLEDAI-2K), which are based on clinical examination and laboratory testing(17).The Exclusion criteria involved (Pregnancy or lactation, and any other inflammatory or Autoimmune diseases, infectious diseases, hematologic disorders and malignancies).

C. *Ethical approval:*

The study was conducted with the permission of the Middle Technical University, College of Health and Medical Technology (Baghdad),2/1/2025, MEC; 104

D. Samples *collection:*

A disposable sterile syringe was utilized to collect five milliliters of blood from a peripheral vein in 100 Iraqi patients across various age groups. Five milliliters of blood samples were deposited into glass tubes containing a gel activator. The blood was allowed to coagulate and subsequently centrifuged at 4000 rpm for ten minutes to separate pure serum. The serum collected from each patient was divided into four aliquots, transferred to sterilized Eppendorf tubes, and stored at -20℃ until utilized for the assessment of different indicators. Each sample was employed just once to avert freezing and thawing.

E. *Quantitative determination of serum CXCL-10, SIGLEC-1:*

Serum biomarkers were evaluated via Enzyme-linked immunosorbent assay (ELISA, FineTest(catalog NO: EH3252, EH0148) of IFN-α and Gal-9, respectively. These kits employ the Double Antibody-Sandwich ELISA detection method. The microplate pre-coated with (anti-IFN-α, anti-Gal-9) antibody. The biotinylated detecting antibody bind to (IFN-α, Gal-9) which is linked to the precoated antibody, add HRP-Streptavidin Conjugate (SABC). Adding the TMB substrate solution, catalyzed by HRP to produce a blue-colored compound that transitioned to yellow upon the introduction of a stop solution. The optical density absorbance was determined at 450 nm via a microplate reader. Ascertain the concentration of (IFN-α, Gal-9) in the sample by plotting the standard curve. The concentration of the target chemical is correlated with the OD450 value. The disease activity was determined by the Systemic Lupus Erythematosus Disease Activity Index 2000 (SLEDAI-2K).

F. *Statistical analyses*

All statistical analyses were performed using R version 4.5.0 (18), and R Studio(19). Quantitative data are presented as Mean ± SD or Median (Range), while qualitative data are expressed as number (percentage). Spearman correlation analysis was performed to assess pairwise associations between variables. Results were visualized using lower-triangle and scatter plots. For comparisons involving three groups, the Kruskal–Wallis test followed by pairwise Mann–Whitney U tests with Bonferroni correction was applied. ROC also helps compare parameters and find the optimal cutoff for sensitivity and specificity.

III. RESULTS

A. *Characteristics of the study population*

A total of 100 SLE patients and 25 healthy people as control were included. Among the SLE patients, 30 (30%) had mild–moderate disease activity (mean SLEDAI=7.2±2.4) and 70 (70%) had severe activity (mean SLEDAI=16.8±4.6, $p < 0.001$ vs. mild–moderate). As shown in Table 1 a significant difference was observed in sex distribution, with a female predominance among SLE patients (96%) compared with controls (80%, $p = 0.016$). Body mass index (BMI) was higher in SLE patients (26.4±4.1 kg/m²) than controls (24.8±2.5 kg/m²). When categorized, overweight and obesity were more frequent

among SLE patients (45% and 19%, respectively) compared with controls (48% and 0%, respectively), with this distribution showing a statistically significant difference (p = 0.037).

TABLE 1. Demographic and clinical characteristics of the study population

Characteristic		***Healthy Controls N = 25***	***SLE Patients N = 100***	***p-value****
Age (years)	Mean ± SD	34.3 ± 9.1	34.4 ± 9.4	0.978
	Median (Min, Max)	33.0 (22.0, 55.0)	34.0 (20.0, 60.0)	
Sex, n (%)	Female	20 (80.0%)	96 (96.0%)	**0.016**
	Male	5 (20.0%)	4 (4.0%)	
BMI (kg/m²)	Mean ± SD	24.8 ± 2.5	26.4 ± 4.1	0.052
	Median (Min, Max)	25.0 (21.0, 29.8)	26.0 (15.6, 40.4)	
BMI Category, n(%)	Normal	13 (52.0%)	33 (33.0%)	**0.037**
	Obese	0 (0.0%)	19 (19.0%)	
	Overweight	12 (48.0%)	45 (45.0%)	
	Underweight	0 (0.0%)	3 (3.0%)	

B. *Characteristic of patients*

More than half of the patients (57.6%) had a disease duration of ⩽ 3 years, while 42.4% had disease duration > 3 years. A positive family history of autoimmune disease (AID) was documented in 30% of patients, whereas 70% reported no family history. Among those with a positive family history, rheumatoid arthritis (RA) was the most frequent (70%). Among the SLE patients, 30 (30%) had mild–moderate disease activity (mean SLEDAI = 7.2 ± 2.4) and 70 (70%) had severe activity (mean SLEDAI = 16.8 ± 4.6, p < 0.001 vs. mild–moderate).

TABLE 2. SLE Duration and Family history among SLE Patients

Characteristic		***SLE Patients (N = 100)***
SLE Duration (years)	**Mean ± SD**	3.5 ± 2
	Median (Min, Max)	3.0 (1, 8.0)
SLE Duration Category	**≤ 3 years**	57 (57.6%)
	> 3 years	42 (42.4%)
Family History of AID	**Negative**	70 (70.0%)
	Positive	30 (30.0%)
Type of AID among Positive Family History	**LN**	1 (3.3%)
	Psoriasis	1 (3.3%)
	RA	21 (70.0%)
	SLE	6 (20.0%)
	SS	1 (3.3%)

AID = Autoimmune Diseases. ,LN =Lupus nephritis ,RA = Rheumatoid arthritis, SS = Sjögren's syndrome

C. *SLEDAI among SLE patients:*

As shown in Table 3 the total SLEDAI score among patients was 16.3±6.0, Among the SLE patients, 30 (30%) had mild–moderate disease activity (mean SLEDAI=7.2 ±2.4) and 70 (70%) had severe activity (mean SLEDAI=16.8±4.6, p < 0.001 vs. mild–moderate).

TABLE 3. SLEDAI-2K among SLE Patients

Total SLEDAI Score	Mean ± SD	16.3 ± 6
	Median (Min, Max)	16.5 (4, 31.0)
SLE Disease Severity, n (%)	Mild-Moderate Activity (SLEDAI-2K <= 10)	30 (30.0%)
	High Activity (SLEDAI-2K > 10)	70 (70.0%)

D. *SLEDAI-2K Components among SLE Patients*

Fig. 1 shows Bar Chart of Clinical Feature Frequencies in SLE Patients, The most observed clinical components of SLEDAI in the figure were arthritis (87.0%), new rash (72.0%), mucosal ulcers (49%), fever (31%), vasculitis (28%), alopecia (23%), lupus headache (20%), proteinuria (15%), Leukopenia (11%), pyuria(10%), visual disturbance (9 %), pleurisy (7%), Urinary casts(5%), pericarditis(5%), Hematuria(5%), Thrombocytopenia(3%) ,myositis (3%), Organic brain syndrome(2%).

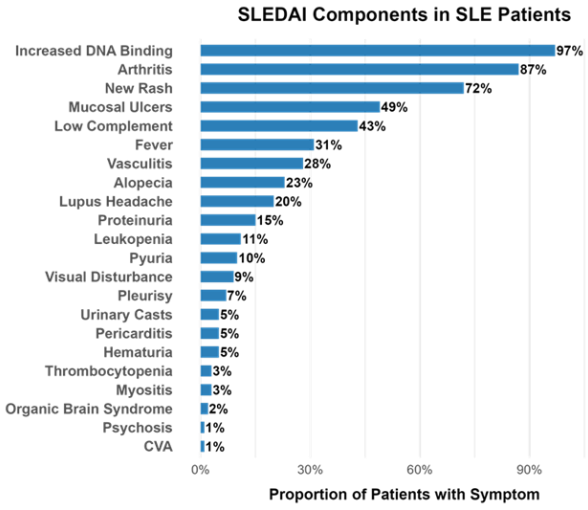


Fig. 1: SLEDAI-2K components among SLE patients

E. *Chemokine levels*

Table 4 showed the Serum levels of the studied chemokines (IFN-α and Galectin-9) significantly elevated in SLE patients compared to healthy controls, with a progressive increase from mild–moderate to severe disease activity. In IFN-α Patients with mild–moderate activity exhibited a significant elevation(290.0±138.0 pg/ml), while those with severe activity had the highest concentrations (354.8±159.7 pg/ml). The differences among groups were highly significant (p < 0.001) compared to the controls (33.0±18.6 pg/ml). Galectine-9 was significantly higher in SLE patients, particularly in severe cases (4.8±1.2ng/ml) compared to mild–moderate disease (3.7±0.8ng/ml, p < 0.01) and controls (0.8±0.4ng/ml).

Table 4: Chemokine levels among study groups

Characteristic		*Control N = 25*	*Mild-Moderate Activity N = 30*	*Severe Activity N = 70*	*p-value**
IFN-α (pg/ml)	***Mean ± SD***	33.0 ± 18.6	290.0 ± 138.0	354.8 ± 159.7	**<0.001**
	Median (Min, Max)	27.5 (12.8, 94.5)	282.0 (82.8, 682.0)	367.5 (89.3, 930.0)	
Galectine-9 (ng/ml)	***Mean ± SD***	0.8 ± 0.4	3.7 ± 0.8	4.8 ± 1.2	**<0.001**
	Median (Min, Max)	0.8 (0.2, 1.4)	3.6 (2.3, 6.5	4.6 (2.3, 8.1)	

In Fig. 2 IFN-α showed weak correlation(ρ = 0.37) with disease activity .a statistically significant correlation existed between serum IFNa levels and SLEDAI. Galectin-9 show moderate strong correlation with disease activity in SLE patients as measured by the SLEDAI-2K (ρ = 0.57) .

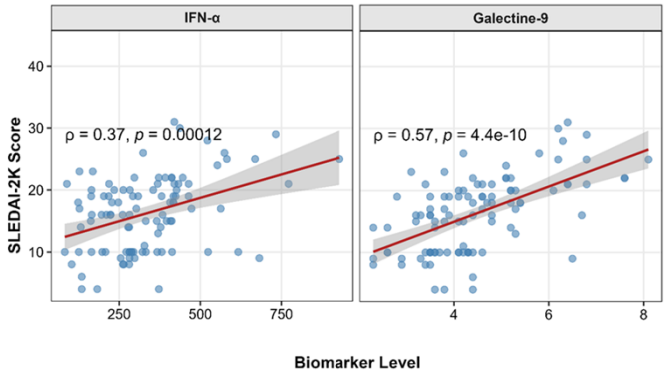


Fig. 2: Spearman Correlation between SLEDAI Score and Chemokine among Study group

F. Receiver Operative *Curve of Chemokine levels among Studied Groups:*

As shown in Fig. 3 that IFN-α in identifying severe disease activity showed an AUC of 0.64, indicating moderate discriminatory ability. (p = 0.021) . The sensitivity was 57.1% and specificity was 73.3%, indicating that increased IFN-alpha levels are a fairly good indicator of sever disease activity.

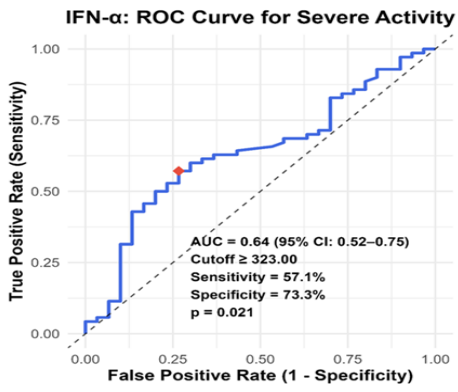


Fig. 3: IFN-α ROC curve for sever Activity

Figure 4 illustrates that Galectin-9 in identifying severe disease activity shows an AUC of 0.76, indicating fair diagnostic accuracy. (p < 0.001) . The sensitivity was 55.7% and specificity was 93.3%, indicating that increased Galectin-9 levels are a fairly strong indicator of serious disease activity.

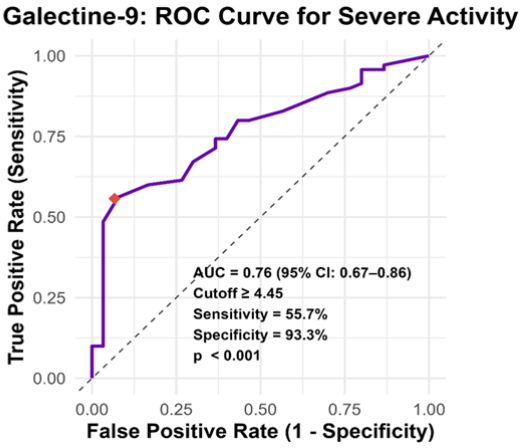


Fig. 5: Galectin-9 ROC curve for sever Activity

IV. DISCUSION

The noted higher prevalence of females in SLE indicates a significant influence of sex hormones [17-18]. Approximately 45% of the patients were classified as overweight based on their body mass index (BMI). Overweight and obesity lead to negative physical health and social performance following treatment in SLE [19]. It was discovered that first-degree relatives possess a 17 times higher risk of SLE in comparison to the general population and that genetic relatedness correlates with the level of risk for SLE [20]. Disease activity is typically classified at a certain moment or over a duration as follows: no disease (remission), low (mild) or minimal disease activity, moderate disease activity, and high (severe) disease activity [21].The capacity to differentiate between mild/moderate and severe illness is clinically significant; individuals with severe illness experience more organ participation, associated conditions, and death rates in contrast to individuals with mild illnesss [22]. IFN-α increase strongly in sever SLE patients comparing to healthy control (354.8 ± 159.7 pg/ml) and less in mild-moderate SLE (290.0±138.0pg/ml). Shahin *et al.* have proposed that IFN-α is a valuable biomarker for recognizing SLE patients [23].Specifically, IFN-α is crucial in the pathogenesis of SLE, being continuously and consistently overexpressed in 50–70% of SLE patients, regardless of treatment and variations in disease activity [24] . The results showed that a correlation was found between serum levels of IFN-α and the clinical symptoms, laboratory investigations revealed a notable relationship with IFN-α serum levels, similar to SLE disease severity (SLEDAI)[25]. Also Galectin-9 show Significant difference between healthy control and two other groups of SLE (Mild-Moderate activity) mean =3.7±0.8 ng/ml , (Sever activity) mean = 4.8 ± 1.2 ng/ml at p-value <0.001. serum Gal-9 levels were notably elevated in patients with organ involvement related to SLE compared to SLE patients without such involvement[26]. Galectin-9 may be co-regulated through interferon signaling. Glycosylation on

the cell surface acts as a binding site for endogenous lectin families like galectins (Gals) that have established immunoregulatory roles. Numerous studies have demonstrated that Gals are crucial in the pathogenesis of SLE[27].

V. CONCLUSION

The research focused on developing dependable biomarkers for evaluating disease activity in systemic lupus erythematosus. Serum IFN-α and Galectin-9 were considerably raised in patients with SLE, particularly in severe cases, and linked with SLEDAI , supporting their role as indicators of disease progression. To improve the study, the authors might increase the sample size, incorporate longitudinal follow-up, and do multivariate analysis to substantiate the predictive usefulness of the biomarkers.

Abbreviations

AID	Autoimmune disease
ECM	Extra cellular membrane
ELISA	Enzyme-Linked Immunosorbent Assay
SLE	Systemic Lupus Erythematosus

G. SLEDA Systemic Lupus Erythematosus Disease Activity Index

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

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