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Selected Biomarkers of Systemic Lupus Erythematosus

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ABSTRACT

Systemic lupus erythematosus (SLE) is a disease with diverse clinical manifestations and variable prognosis. The search for reliable SLE biomarkers is useful to enable earlier diagnosis, better assessment of organ involvement, and more accurate monitoring of disease activity. This paper reviews the latest findings on non-organ-specific and selected organ-specific biomarkers of SLE in various biological specimens, including blood, urine, and cerebrospinal fluid (CSF). Much progress has been made in identifying SLE biomarkers, though many remain inadequately validated for everyday clinical practice. The complicated pathogenesis of SLE and varied clinical presentation result in the need for multiple biomarkers from different biological fluids like blood, urine, or CSF. Large-scale, prospective multicenter studies with modern technologies are needed to discover new molecules. The search for new biomarkers can help create personalized treatment approaches for SLE patients and enhance treatment outcomes.

Keywords: biomarkers, disease activity, organ-specific biomarkers, non-organ-specific biomarkers, systemic lupus erythematosus

1. INTRODUCTION

Biomarkers are commonly used in clinical practice for diagnosis, disease activity assessment, and prognostic stratification of various diseases. Most commonly, they are assessed from biological materials obtained from patients, such as blood, urine, synovial fluid, or cerebrospinal fluid (Colina and Campana, 2025). The classification is based on the type of molecule detected, including deoxyribonucleic acid, ribonucleic acid, proteins, metabolites, and cellular subpopulations. Clinical utility is also a key aspect, because they can serve as measurable indicators of physiological or pathological processes, including diagnosis, prognosis, monitoring of disease activity, pharmacodynamics, and assessment of treatment response (Ding et al., 2024). An optimal biomarker should have analytical reliability, high prognostic precision, reproducibility, and wide clinical applicability. It should also be cost-effective in use and have minimal influence from variable factors (González et al., 2021).

When describing systemic lupus erythematosus, it is important to mention that it is an autoimmune disease, characterized by chronic inflammation and

multisystem involvement, leading to progressive tissue and organ damage. The areas requiring attention right now are early diagnosis, reliable monitoring of disease activity, and ensuring an objective assessment of treatment efficacy.

Aim

This paper reviews the latest findings from scientific literature on non-organ-specific and selected organ-specific biomarkers of SLE in various biological specimens, including blood, urine, and cerebrospinal fluid (CSF).

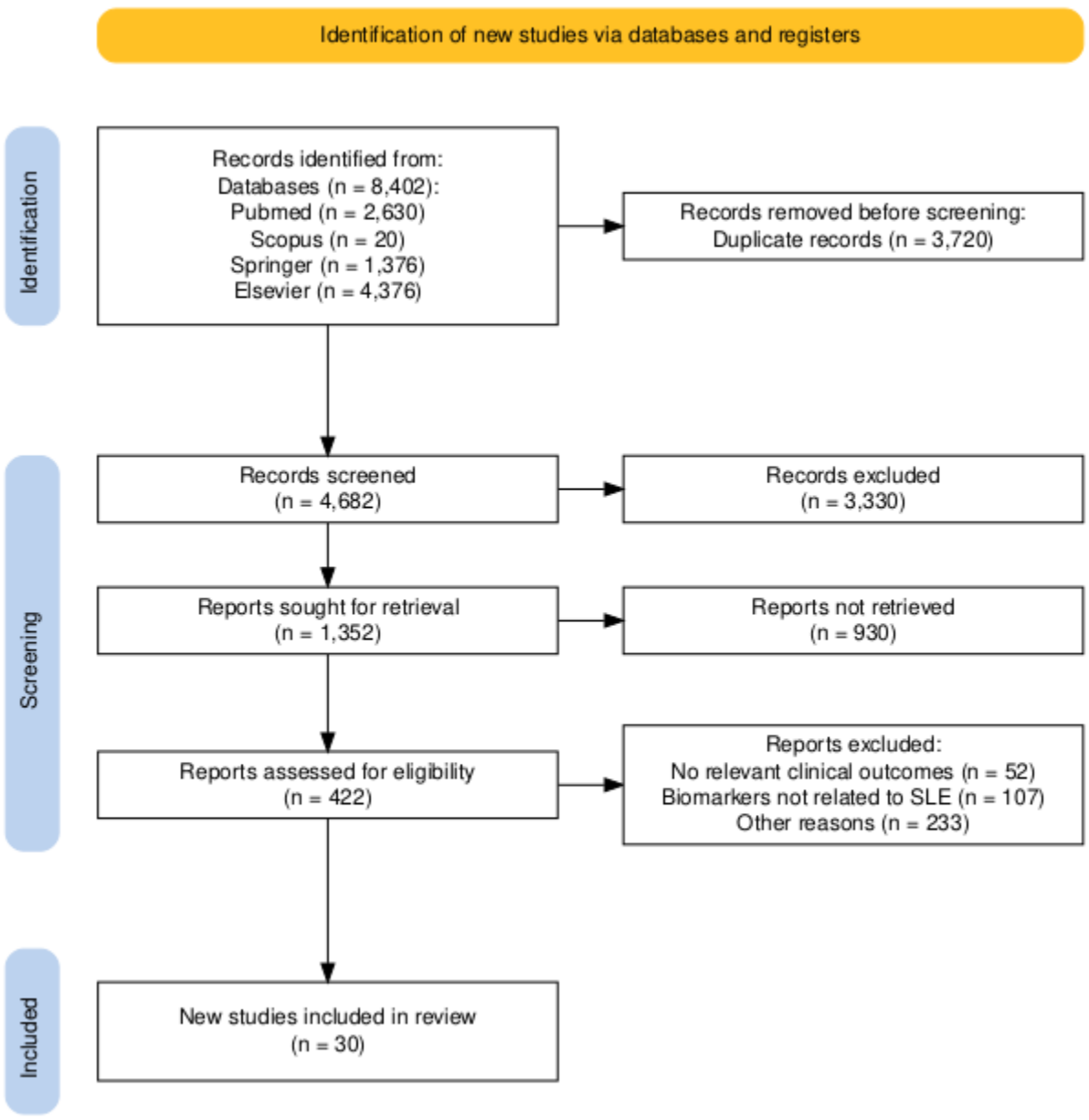

Figure 1. PRISMA Flowchart

2. REVIEW METHODS

The study selection process was carried out in accordance with the PRISMA guidelines and in two stages. In the first stage, titles and abstracts were evaluated, and in the second, the full texts of the articles were analyzed.

The selection was conducted by four independent reviewers. In the event of a disagreement, a decision was reached through discussion among the reviewers. The search covered publications released from January 2015 to December 2025. A literature review was conducted using electronic databases such as PubMed, Scopus, Springer, and Elsevier, using several Medical Subject Headings

(MeSH) terms, and a combination of keywords and the logical operators "AND" and "OR". Keywords included biomarkers, disease activity, organ-specific biomarkers, non-organ-specific biomarkers, and systemic lupus erythematosus, identifying a total of 8,402 records. We included only articles written in English and published in the last 10 years in the analysis. After removing duplicates (n = 3,720), 4,682 publications were selected for further analysis. During the evaluation of titles and abstracts, 3,330 studies were excluded as not meeting the inclusion criteria. Each article was rigorously assessed for relevance, and the references cited were examined to identify additional relevant sources. A total of 422 articles were eligible for full-text review, of which 392 were excluded, most commonly due to "No relevant clinical outcomes" or "Biomarkers not related to SLE". Specific exclusion criteria excluded articles without abstracts, conference proceedings, errata, retracted articles, and studies that did not address the possible association between biomarkers and SLE. Ultimately, 30 studies were included in the review. We assessed the methodological quality of the included studies using the Cochrane Risk of Bias Tool. The selection process is illustrated in the PRISMA diagram presented in Figure 1.

3. RESULTS & DISCUSSION

Non-organ-specific biomarkers of SLE

Non-organ-specific markers are indicative of SLE presence but are not restricted to damage or involvement of a particular organ or system, for example: antinuclear antibodies (ANA), anti-double-stranded DNA antibodies (anti-dsDNA), anti-Smith antibodies (anti-Sm), and complement components. ANA shows high sensitivity up to 90-98% and low specificity, approximately 20% (Ameer et al., 2022). It can be assumed that an increase in ANA titer is associated with increased plasma cell activity and, consequently, higher SLE activity, but this relationship has not been confirmed in many cases. ANA titer may fluctuate throughout the natural course of the disease and may also be influenced by immunosuppressive drugs. The routine assessment of ANA titer for monitoring SLE activity is not recommended, as its correlation with clinical disease activity is limited. In some patients, negative ANA seroconversion was associated with a reduced risk of disease exacerbation, whereas in others it has been associated with impending disease flares (Frodlund et al., 2020). Anti-dsDNA antibodies, characterized by low sensitivity (37%) and high specificity (100%) for SLE diagnosis, in some patients correlate well with disease activity (Wichainun et al., 2013). They show heterogeneity and can cross-react with different endogenous proteins. This molecular mimicry contributes to target-organ pathology, for example, cross-reactivity with α-actinin represents one of the pathogenetic mechanisms of lupus nephropathy (LN). In contrast, cross-reactivity with the N-methyl-D-aspartate receptor (NMDA) is associated with neuropsychiatric manifestations of SLE. Anti-dsDNA antibodies may be present for many years before SLE diagnosis.

A significant increase in anti-dsDNA levels may foreshadow an exacerbation of the disease, especially with renal involvement (Pisetsky and Lipsky, 2020). The clinical significance of anti-Sm antibodies remains unclear. These antibodies demonstrate high specificity (98.6%) but low sensitivity (39.7%) for SLE and may be detectable before the onset of clinical symptoms. Associations between anti-Sm antibodies and specific SLE manifestations have been demonstrated in several studies, for example, renal and central nervous system involvement, as well as hemolytic anemia and vasculitis. ANA and anti-dsDNA were found in the urine of 33% of SLE patients.

The complement system serves as a critical serological parameter in SLE, with both diagnostic and pathogenic significance. Hereditary deficiencies in classical pathway components, particularly C1q, are among the strongest genetic risk factors for SLE. Complete deficiency of one of the early components of the classical pathway of complement system activation is very rare. The classical pathway includes compounds like C1q, C1r, C1s, and C4. Partial deficiency is more common, especially C4A deficiency, which is present in 30-40% of patients. The assessment of C3 and C4 levels may be useful in monitoring disease activity, but their prognostic value is limited (Coss et al., 2023). Both hypocomplementemia and elevated anti-Sm antibody titer have been identified as predictive markers for the development of LN. Notably, patients with isolated hypocomplementemia of the C3 component and renal involvement are at increased risk of progression to end-stage renal disease. Decreased C3 and C4 concentrations in pleural fluid help differentiate lupus pleuritis from pleural effusion of other etiology. Complement components and anti-dsDNA titer are used as biomarkers of immune dysfunction in SLE. Patients with severe exacerbations of neuropsychiatric lupus tend to have low C4 levels, while decreased C3 concentrations may be observed in patients with complications such as lupus enteritis or myocarditis. It is important to emphasize that assessment of SLE activity is mainly based on a global evaluation.

Erythrocyte sedimentation rate (ESR) is a nonspecific marker of systemic inflammation and is frequently used in the assessment of SLE. Due to multifactorial influences like hypoalbuminemia, hypercholesterolemia, hypergammaglobulinemia, anemia, and elevated fibrinogen levels, its diagnostic specificity is limited. Elevated ESR shows a strong correlation with overall disease activity. In contrast,

C-reactive protein (CRP) levels are normal in uncomplicated SLE patients, elevated CRP may indicate serosal inflammation, musculoskeletal involvement, or infection. Elevated ESR coupled with normal or minimally elevated CRP is considered characteristic of SLE-associated inflammation and can be used to monitor disease activity (Yu et al., 2021).

Organ-specific biomarkers of SLE

Skin

Skin lesions occur in approximately 70% of SLE patients, with several biomarkers implicated in their pathogenesis. Type I interferon plays an important role in the pathogenesis of skin lesions in systemic lupus erythematosus. Notably, IFN-I-associated proteins such as annexin-1 and interleukin-18 have demonstrated diagnostic utility in differentiating cutaneous lesions of both cutaneous lupus erythematosus (CLE) and SLE from those observed in other dermatological conditions. Skin lesions in SLE are characterized by upregulated expression of two IFN-γ-related genes (IFNα10 and IFNκ) concurrent with elevated levels of IFN-γ-induced proteins, including myxovirus resistance protein A (MxA), C-X-C motif chemokine receptor 3 (CXCR3), and guanylate-binding protein-1 (GBP-1) (Zhu et al., 2021). Various autoantibodies and protein biomarkers have been identified as associated with specific subtypes of lupus skin lesions. Subacute cutaneous lupus erythematosus (SCLE) is a subtype that is clinically defined by the presence of non-scarring, photosensitive lesions typically distributed on the trunk and upper extremities.

Anti-SS-A antibodies serve as an important serological marker for identifying SCLE and are found in about 63% of patients with this subtype of the disease. are a crucial serological marker for distinguishing SCLE and are detectable in approximately 63% of patients with this subtype. Among patients with skin lesions, anti-annexin antibody levels were considerably higher than in healthy individuals. The increase is especially pronounced in patients with discoid lupus erythematosus. The Cutaneous Lupus Erythematosus Disease Area and Severity Index was used to assess the disease activity in this patient population, and it showed a positive correlation with TNF-α levels in peripheral blood mononuclear cells (PBMCs) and IgG anti-ribonucleoprotein antibodies in serum. Studies have shown that an increased population of myeloid dendritic cells with higher TNF-α expression may predict a poor response to hydroxychloroquine (HCQ) treatment in patients with CLE. Finally, the presence of anti-dsDNA and anti-Sm antibodies, along with an accelerated ESR, has been associated with an increased risk for progression from CLE to SLE (Zhu et al., 2021).

Kidneys - Lupus Nephritis

Renal involvement occurs in approximately 70% of SLE patients, often remaining clinically silent. The key diagnostic method for Lupus Nephritis (LN) is renal biopsy. However, due to its invasiveness, there is a need for non-invasive biomarkers. It has been shown that certain cytokines and chemokines can serve as biomarkers and can be used to determine the activity and severity of LN, distinguish between the histological subtypes, and monitor response to treatment (Alduraibi and Tsokos, 2024). Urine is an easily accessible potential source of diagnostic material. Monocyte chemoattractant protein-1 is a chemokine that recruits leukocytes to sites of inflammation. Elevated MCP-1 levels correlate with inflammatory infiltration in renal interstitial tissue, fibrosis, and renal tubular atrophy. Elevated urinary MCP-1 levels can accurately differentiate between active and inactive LN. Urinary levels of interleukin-17 and transforming growth factor beta 1 correlate with the severity of renal involvement in SLE. Likewise, elevated levels of IL-12p40, IL-15, thymus-regulated chemokines, TWEAK, and UMCP-1K are also associated with active LN. It is worth noting that both TWEAK and UMCP-1K markers have strong prognostic value in predicting progression to end-stage renal disease (Bergkamp et al., 2023).

Elevated urinary osteoprotegerin levels in patients with LN also correspond to the activity of renal involvement and appear to be capable of predicting treatment failure and disease recurrence. Blood biomarkers also show diagnostic potential. Among serum biomarkers, Axl, a cell-surface tyrosine kinase receptor, regulates innate immunity and efferocytosis. It shows potential as a serum biomarker, correlating with kidney disease activity. Its persistently elevated levels may indicate the need to intensify immunosuppressive treatment (Parodis et al., 2019). A proliferation-inducing ligand is a TNF superfamily member and is responsible for regulating B-cell survival. It is also associated with severe proliferative histopathological variants, demonstrating an association with characteristic histopathological features revealed in renal biopsy, such as neutrophilic infiltrates, fibrinoid necrosis, and crescent formation. Table 1 shows selected biomarkers of LN.

Anti-dsDNA antibodies remain the most significant in assessing LN activity. Also, anti-C1q antibodies demonstrate high specificity and moderate sensitivity for LN, serving as discriminative serological markers to differentiate LN from non-lupus nephritis. Antibodies targeting chromatin/nucleosomes are strongly associated with proliferative lupus nephritis forms. Recently, neutralizing anti-DNase1L3 antibodies have been identified as a novel biomarker associated with a more severe LN phenotype.

Table 1. Clinical relevance of selected biomarkers of LN

Biomarker	Biological material	Clinical significance
Galectin-3 binding protein	urine	assessment of LN activity; significantly elevated in class III, IV, and V LN
MCP-1	urine	assessment of LN activity; predictive value of LN exacerbation; significantly elevated in LN class III and IV; marker of kidney interstitial tissue involvement; monitoring the effectiveness of treatment
TGF-β1	urine	assessment of LN activity
TWEAK	urine	assessment of LN activity; predictive value of renal failure progression
UMCP-1K	urine	assessment of LN activity, predictive value of renal failure progression
MP	urine	an early marker of glomerular injury in LN
Axl AaA	blood	assessment of LN activity
sTNFR2	blood	assessment of LN activity, renal damage, and response to treatment
Adipokines	blood	associated with LN activity, insulin resistance, and endothelial dysfunction
APRIL	blood	assessment of LN activity monitoring the effectiveness of treatment
IL-17 and IL-18	urine, blood	assessment of LN activity
suPAR	urine, blood	assessment of LN activity monitoring the effectiveness of treatment
MicroRNAs (miRs)	urine, blood, kidney	assessment of LN activity monitoring the effectiveness of treatment

AaA - Anti-actinin antibodies; APRIL - A Proliferation-Inducing Ligand; Axl - Axl receptor tyrosine kinase; IL-17 - Interleukin-17; IL-18 - Interleukin-18; MCP-1 - Monocyte Chemoattractant Protein-1; MP - Microparticles from podocytes; sTNFR2 - Soluble Tumor Necrosis Factor Receptor 2; suPAR - Soluble urokinase plasminogen activator receptor; TGF-β1-Transforming Growth Factor Beta 1; TWEAK - TNF-like Weak Inducer of Apoptosis; UMCP-1K - Urinary MCP-1/Creatinine ratio

Neuropsychiatric SLE

Neuropsychiatric systemic lupus erythematosus is not a singular disease, but a group of neurological and psychiatric symptoms. It may affect from 12% to 95% of patients and significantly impair their quality of life and general prognosis. The confirmation of definitive diagnosis remains a challenge, as neither neuroimaging nor neuropsychological evaluations provides conclusive results. That is why biomarkers indicating nervous system involvement may play a key role in diagnosing, monitoring disease activity, and predicting treatment response in patients with neuropsychiatric systemic lupus erythematosus. Cerebrospinal fluid (CSF) is a particularly

valuable biological material for this purpose. A crucial biomarker for NPSLE is the anti-ribosomal protein antibody. Another significant factors in the pathogenesis and assessment of neuropsychiatric systemic lupus erythematosus activity include anti-UCH-L1 (ubiquitin carboxyl-terminal hydrolase L1) and anti-NMDA receptor antibodies. Aquaporin-4 (AQP4) is an astrocyte protein essential for regulating water flow between neural cells. IgG-class autoantibodies against AQP4 (anti-AQP4) are the main cause of neuromyelitis optica, an inflammatory, autoimmune demyelinating central nervous system disease. In some patients, anti-AQP4 antibodies is associated with an increased risk of nervous system involvement (Kopp et al., 2023). Proinflammatory cytokines play an important role in the pathogenesis of neuropsychiatric symptoms by increasing blood-brain barrier permeability and facilitating the entry of autoantibodies into the central nervous system. Increased levels of Interleukin 6, interleukin 8, monocyte chemoattractant protein-1, interferon gamma-induced protein 10, granulocyte colony-stimulating factor, and granulocyte-macrophage colony-stimulating factor have also been found in CSF of patients with SLE and neuropsychiatric manifestations (Yoshio et al., 2016; Ding et al., 2024).

Pulmonary Involvement

Data on biomarkers of pulmonary involvement in patients with systemic lupus erythematosus is limited. Increased levels of chemokines CCL21 and IP-10 have been observed in SLE patients with lung involvement. They are negatively correlated with lung function parameters like diffusing capacity of the lung for carbon monoxide, FVC (forced vital capacity), and FEV1 (forced expiratory volume in one second) (Osman et al., 2022). Cysteine-rich angiogenic inducer 61 is a significant marker for identifying SLE patients with pulmonary arterial hypertension (Fan et al., 2019).

Cardiovascular System

The first biomarker associated with cardiovascular events in SLE patients was high-sensitivity troponin T, whose increased levels correlated with cardiovascular disease (CVD) risk (Chezel et al., 2021). Low levels of high-density lipoprotein (HDL) are a well-known risk factor for CVD. SLE patients exhibit reduced HDL levels and dysfunctional HDL, significantly increasing the risk of atherosclerosis (Kim et al., 2020). Elevated levels of antibodies against HDL and paraoxonase 1 (PON1) have been found in SLE patients and correlated with increased carotid intima-media thickness and reduced cerebral artery blood flow velocity in patients with clinical and subclinical CVD. Elevated numbers of low-density granulocytes (LDG) and monocytes, as well as an abnormal LDG/HDL ratio, may also serve as useful biomarkers of CVD risk (López et al., 2020).

Selected genetic biomarkers

Emerging evidence underscores the significant heritable contribution to SLE pathogenesis, identifying rare pathogenic variants enriched in lupus cohorts. Notably, the majority of these risk-associated alleles localize to non-coding regulatory elements, and the strongest links were observed in the HLA region. The majority of the identified genes are related to apoptosis, activation of B and T lymphocytes, or upregulation of type I interferon pathways. In the course of recent years, many new genetic markers of SLE have been discovered: the ABCB1 gene, CD247, DSC1, KIR2DL3, and MX2 (Zhao et al., 2023). Several new genes (PHACTR2, GOT2, CMC4, MAP2K1, CMPK2, ECPAS, and SRA1) have been associated with the risk of exacerbation of this disease (Li et al., 2022).

4. CONCLUSION

The significant clinical heterogeneity of systemic lupus erythematosus makes it difficult to identify and develop specific biomarkers for this disease. Variability in assessment is a key factor that influences biomarker research. Many SLE biomarkers have not yet demonstrated sufficient sensitivity, specificity, or predictive power for clinical use. Furthermore, the lack of validation cohorts makes it challenging to determine the value of any identified biomarkers. As no single biomarker is sufficiently sensitive or specific for SLE, a mathematical analysis of multiple biomarkers could be beneficial. Further large-scale observational and multicenter studies are required to confirm the role of potential biomarkers in the pathogenesis, assessment of activity, and treatment of SLE. The prognosis of SLE patients depends on prompt diagnosis and treatment tailored to organ changes and disease activity. To this end, new, organ-specific, low-cost biomarkers of the disease are being sought. Because of the complexity of the pathogenesis and the heterogeneous clinical picture, multiple biomarkers of different biological materials are needed. Prospective, multicenter studies using modern technology are needed to detect novel molecules.

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Authors' Contributions
All authors take full responsibility for the integrity and accuracy of all aspects of the work.
MD, PhD Dorota Suszek: study conception and design
Magdalena Popławska: data collection, analysis, and interpretation of results, literature screening
Karolina Przeniosło: analysis and interpretation of results, literature screening, and quality assessment
Katarzyna Prośniak: review and editing, literature screening and quality assessment
Jakub Prośniak: methodology, project administration
Marcin Kaniewski: draft manuscript preparation
Aleksander Oskroba: draft manuscript preparation
Weronika Janczylik: analysis and interpretation of results
Prof., MD, PhD Bożena Targońska – Stępniak: study conception and design
All authors reviewed the results and approved the final version of the manuscript.

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Conflict of interest
The authors declare that they have no conflicts of interest, competing financial interests or personal relationships that could have influenced the work reported in this paper.

Data and materials availability
All data associated with this study will be available based on the reasonable request to corresponding author.

REFERENCES

1. Alduraibi FK, Tsokos GC. Lupus Nephritis Biomarkers: A Critical Review. Int J Mol Sci 2024;25(2):805. doi:10.3390/ijms25020805
2. Ameer MA, Chaudhry H, Mushtaq J, Khan OS, Babar M, Hashim T, Zeb S, Tariq MA, Patlolla SR, Ali J, Hashim SN, Hashim S. An Overview of Systemic Lupus Erythematosus (SLE) Pathogenesis, Classification, and Management. Cureus 2022;14(10):e30330. doi:10.7759/cureus.30330
3. Bergkamp SC, Wahadat MJ, Salah A, Kuijpers TW, Smith V, Tas SW, van den Berg JM, Kamphuis S, Schonenberg-Meinema D. Dysregulated endothelial cell markers in systemic lupus erythematosus: a systematic review and meta-analysis. J Inflamm Lond Engl 2023;20(1):18. doi:10.1186/s12950-023-00342-1
4. Chezel J, Costedoat-Chalumeau N, Laouénan C, Rouzaud D, Chenevier-Gobeaux C, Le Guern V, Mathian A, Belhadi D, de Almeida Chaves S, Duhaut P, Fain O, Galicier L, Ghillani-Dalbin P, Kahn JE, Morel N, Perard L, Pha M, Saidoune F, Sarrot-Reynauld F, Aumaitre O, Chasset F, Limal N, Desmurs-Clavel H, Ackermann F, Amoura Z, Papo T, Sacre K. Highly sensitive serum cardiac troponin T and cardiovascular events in patients with systemic lupus erythematosus (TROPOPLUS study). Rheumatol Oxf Engl 2021;60(3):1210-1215. doi:10.1093/rheumatology/keaa434

5. Colina M, Campana G. Precision Medicine in Rheumatology: The Role of Biomarkers in Diagnosis and Treatment Optimization. J Clin Med 2025;14(5):1735. doi:10.3390/jcm14051735
6. Coss SL, Zhou D, Chua GT, Aziz RA, Hoffman RP, Wu YL, Ardoin SP, Atkinson JP, Yu CY. The complement system and human autoimmune diseases. J Autoimmun 2023;137:102979. doi:10.1016/j.jaut.2022.102979
7. Ding H, Shen Y, Hong SM, Xiang C, Shen N. Biomarkers for systemic lupus erythematosus - a focus on organ damage. Expert Rev Clin Immunol 2024;20(1):39-58. doi:10.1080/1744666X.2023.2260098
8. Fan Y, Zhao J, Qian J, Hao Y, Wang Q, Gao L, Li M, Zeng X, Zhang Z. Cysteine-rich protein 61 as a novel biomarker in systemic lupus erythematosus-associated pulmonary arterial hypertension. Clin Exp Rheumatol 2019;37(4):623-632
9. Frodlund M, Wetterö J, Dahle C, Dahlström Ö, Skogh T, Rönnelid J, Sjöwall C. Longitudinal anti-nuclear antibody (ANA) seroconversion in systemic lupus erythematosus: a prospective study of Swedish cases with recent-onset disease. Clin Exp Immunol 2020;199(3):245-254. doi:10.1111/cei.13402
10. González LA, Ugarte-Gil MF, Alarcón GS. Systemic lupus erythematosus: The search for the ideal biomarker. Lupus 2021;30(2):181-203. doi:10.1177/0961203320979051
11. Kim SY, Yu M, Morin EE, Kang J, Kaplan MJ, Schwendeman A. High-Density Lipoprotein in Lupus: Disease Biomarkers and Potential Therapeutic Strategy. Arthritis Rheumatol Hoboken NJ 2020;72(1):20-30. doi:10.1002/art.41059
12. Kopp CR, Prasad CB, Naidu S, Sharma V, Misra DP, Agarwal V, Sharma A. Overlap syndrome of anti-aquaporin-4 positive neuromyelitis optica spectrum disorder and systemic lupus erythematosus: A systematic review of individual patient data. Lupus 2023;32(10):1164-1172. doi:10.1177/09612033231191180
13. Li Y, Ma C, Liao S, Qi S, Meng S, Cai W, Dai W, Cao R, Dong X, Krämer BK, Yun C, Hocher B, Hong X, Liu D, Tang D, He J, Yin L, Dai Y. Combined proteomics and single cell RNA-sequencing analysis to identify biomarkers of disease diagnosis and disease exacerbation for systemic lupus erythematosus. Front Immunol 2022;13:969509. doi:10.3389/fimmu.2022.969509
14. López P, Rodríguez-Carrio J, Martínez-Zapico A, Pérez-Álvarez ÁI, Suárez-Díaz S, Mozo L, Benavente L, Caminal-Montero L, Suárez A. Low-density granulocytes and monocytes as biomarkers of cardiovascular risk in systemic lupus erythematosus. Rheumatol Oxf Engl 2020;59(7):1752-1764. doi:10.1093/rheumatology/keaa016
15. Osman HM, Omar GM, Elameen NF, Abdel-Nasser AM. CCL21 and IP10 as serum biomarkers for pulmonary involvement in systemic lupus erythematosus. Lupus 2022;31(6):706-715. doi:10.1177/09612033221093493
16. Parodis I, Ding H, Zickert A, Cosson G, Fathima M, Grönwall C, Mohan C, Gunnarsson I. Serum Axl predicts histology-based response to induction therapy and long-term renal outcome in lupus nephritis. PloS One 2019;14(2):e0212068. doi:10.1371/journal.pone.0212068
17. Pisetsky DS, Lipsky PE. New insights into the role of antinuclear antibodies in systemic lupus erythematosus. Nat Rev Rheumatol 2020;16(10):565-579. doi:10.1038/s41584-020-0480-7
18. Wichainun R, Kasitanon N, Wangkaew S, Hongsongkiat S, Sukitawut W, Louthrenoo W. Sensitivity and specificity of ANA and anti-dsDNA in the diagnosis of systemic lupus erythematosus: a comparison using control sera obtained from healthy individuals and patients with multiple medical problems. Asian Pac J Allergy Immunol 2013;31(4):292-298. doi:10.12932/AP0272.31.4.2013
19. Yoshio T, Okamoto H, Kurasawa K, Dei Y, Hirohata S, Minota S. IL-6, IL-8, IP-10, MCP-1 and G-CSF are significantly increased in cerebrospinal fluid but not in sera of patients with central neuropsychiatric lupus erythematosus. Lupus 2016;25(9):997-1003. doi:10.1177/0961203316629556
20. Yu H, Nagafuchi Y, Fujio K. Clinical and Immunological Biomarkers for Systemic Lupus Erythematosus. Biomolecules 2021;11(7):928. doi:10.3390/biom11070928
21. Zhao X, Duan L, Cui D, Xie J. Exploration of biomarkers for systemic lupus erythematosus by machine-learning analysis. BMC Immunol 2023;24(1):44. doi:10.1186/s12865-023-00581-0v
22. Zhu JL, Black SM, Chong BF. Role of biomarkers in the diagnosis and prognosis of patients with cutaneous lupus erythematosus. Ann Transl Med 2021;9(5):429. doi:10.21037/atm-20-5232