Journal of Progressive Medical Sciences
ISSN: 3052-8518
DOI: https://doi.org/10.63939/dbqmny45
J.P.M.S

Vol. 1 , No 4 , February 2026

RESEARCH Open Access

The effect of *Toxoplasma gondii* infection on anti-dsDNA, anti-ribosomal P, and anti-chromatin (minor DNA) autoantibodies in patients with Systemic Lupus Erythematosus

Wijdan Dhaidan Shnain Al-abbas
Basic Science department. College of Nursing. University of Kufa,
Wijdand.shnain@uokufa.edu.iq

Abstract:

Background: Systemic lupus erythematosus (SLE) is a chronic autoimmune disease that is distinguished by the fluctuation of disease activity and the production of pathogenic autoantibodies. Infectious agents, such as *Toxoplasma gondii*, have been proposed to be candidate environmental factors able to influence autoimmune responses. Nevertheless, relationship between *T. gondii* infection and autoantibody profiles among patients with SLE is not fully realized yet especially in endemic areas.

Objective: study the roles of *Toxoplasma gondii* infection on levels and prevalence of anti-ribosomal P, anti–double-stranded DNA (anti-dsDNA), and anti-chromatin (minor DNA) autoantibodies, in addition to disease activity in patients with systemic lupus erythematosus.

Methods: A cross-sectional study which included 100 lupus patients Al-Najaf hospitals. Study period between June 2024 to July 2025. Sunscreen-naive SLE patients were categorized by *T. gondii* infection: 48 who were with infection and 52 who were without infection. Anti-*T. gondii* IgG and IgM antibodies were determined by ELISA. Serum antibodies to dsDNA, ribosomal P and chromatin were assessed by ELISA. Disease activity was evaluated based on the SLE Disease Activity Index (SLEDAI). Group comparisons, correlation analysis and multivariate logistic regression were used for statistical analyses.

Results: The seropositivity of *T. gondii* IgG was considerably higher in SLE patients (48.0% P = 0.03). Anti-dsDNA antibodies ($p < 0.001$) and anti-chromatin antibodies ($p < 0.001$) were significantly elevated in seropositive SLE patients., in comparison with

seronegative subjects. Patients with *T. gondii* were more likely to possess anti-ribosomal P antibodies (45.8%; 23.1%, p = 0.01) and these were linked to neuropsychiatric presentation. SLEDAI score was significantly higher in the *T. gondii* positive group ($p < 0.001$). Positive correlations were found between the titer of *T. gondii* IgG antibodies, the levels of autoantibodies, and disease activity. On multivariate analysis, *T. gondii* seropositivity was an independent predictor of high disease activity (OR = 3.4, 95% CI: 1.6–7.1).

Conclusions: *T gondii* infection is correlated with elevated autoantibodies titers and higher SLE activity. These results suggest that chronic toxoplasmosis may act as an immunological modulator in SLE and the need to consider the etiology of parasite infections in the diagnosis and treatment of autoimmune diseases, especially when these are endemic.

Keywords: Systemic lupus erythematosus; *Toxoplasma gondii*; anti-dsDNA; anti-ribosomal P antibodies; antichromatin antibodies; disease activity

Introduction:

The causative agent of toxoplasmosis, a medical zoonosis that is prevalent worldwide, is Toxoplasma gondii, an obligate intracellular protozoan parasite [1]. This parasite, which is classified in the Phylum Apicomplexa, has a broad host range and infects nearly all warm-blooded animals, including humans, livestock, and companion animals [2]. The global distribution of *T. gondii* is ubiquitous and infects an estimated one-third of the human population worldwide [3]. Although the infection in immunocompetent individuals is mostly asymptomatic, pregnant women and those with impaired immune systems may experience severe or fatal cases [4]. The disease presents in acute and chronic forms, the latter with a burden of latent tissue cysts primarily located in or confined to the brain and skeletal muscles [5]. The dormant bradyzoite cysts are one of the ways in which the parasite can lay low for a host's entire life, with some probability of reactivity when available, including among immunocompromised hosts [5,6].

These serologic markers are very important diagnostic and prognostic indicators in systemic autoimmune rheumatic diseases, with different patterns of prevalence and specificity according to the type of autoantibody [7]. Particularly anti-dsDNA antibodies are commonly used in the diagnosis of systemic lupus erythematosus, in

Published by Democratic Arabic Center for Strategic, Political and Economic Studies (DAC)

monitoring the disease activity and co relates with renal and central nervous system involvement [8]. On the contrary anti-ribosomal P antibodies are very SLE specific and frequently associated with neurological manifestations, while anti-chromatin antibodies have limited specificity compared to anti-dsDNA but could provide additional diagnostic aid even for uncertain results in the context of testing for SLE [9, 10]. In addition, anti-ribosomal P proteins antibodies described in 1979 [10] are commonly tested for through binding to a shared epitope among three basic ribosomal proteins (P0, P1 and P2) [11–13]. Anti-Sm and anti-dsDNA antibodies are well known for their superior specificity in SLE diagnosis, however, mixed with them indicated by the previous studies, anti-ribosomal P antibodies can be more benefit in diagnosis of patients negative for single anti-Sm or anti-dsDNA or both [11]. Given their high specificity for SLE such antibodies anti-ribosomal P have not been included in formal classification or diagnostic criteria, at least in part because of the heterogeneity between diagnostic platforms and the ribosomal protein epitopes that are targeted by various assays [13].

However, adding anti-ribosomal P antibodies to a diagnostic profile is relevant because of their very high specificity for SLE and the existing association with NPSLE [14]. However, anti-dsDNA antibodies are not a feature of all neuropsychiatric SLE patients — they occur in only around 70% of cases — and their levels do not always correlate with the degree of neuropsychiatric disease activity [15].This diversity highlights the challenges of accurate diagnosis and of neuropsychiatric engagement in SLE, requiring extensive serological testing that must include the anti-ribosomal P amyloids [16]. Systemic lupus erythematosus (SLE) is a complex chronic inflammatory autoimmune disease characterized by the generation of autoantibodies against self-entities. This leads to systemic inflammation and loss of tolerance all over the body [17].

A prototype autoimmune disease that manifests in recurrent bouts of symptomatology with giant relapsing/recuperating episodes, SLE has the potential to cause significant permanent damage to various organ systems and tissues including, but not limited to, those involving predominantly kidney, central nervous system, joints and skin [18]. This complex interplay between genetic predisposition, environmental exposure and hormonal influences results in the loss of tolerance acquired to self-antigen [19]. It is an auto-immune disease that varies from mild to severe and life-threatening, where the immune system attacks its own body tissues and organs [20,21].

Published by Democratic Arabic Center for Strategic, Political and Economic Studies (DAC)

Despite improved understanding and treatment, SLE remains a large contributor to global burden of disease, emphasizing the need for earlier diagnosis and intervention of disease, optimally ameliorating patients [22]. The heterogeneous manifestation and severity of SLE, poses challenges to diagnosis with subsequent treatment delay [23].

This heterogeneity of the disease stems from its capacity to involve virtually any organ system – skin, musculoskeletal, kidneys, hematologic, cardiovascular, pulmonologic, neuro-psychiatric and reproductive – and often with variable prognosis course over time that oscillates between a stormy active phase toward complete remission [24]. These presenting symptoms, which are non-specific and may include general constitutional symptoms (such as fatigue or fever) to more specific mucocutaneous and musculoskeletal complaints, are usually among the earliest manifestations of disease [24,25]. These early symptomatic presentations are heterogeneous, but often include lupus-specific rashes, arthralgia and myalgia supporting the need to recognize common clinical features for appropriate referral and diagnosis [24,26]. this Study aimed to assess the effect of *Toxoplasma gondii* infection on the levels and prevalence of anti-dsDNA, anti-ribosomal P, and anti-chromatin autoantibodies in patients with systemic lupus erythematosus.

Materials and methods:

Study Design

Type: A cross-sectional study

Study Groups involved:

1. SLE patients infected with *T. gondii*
2. SLE patients without *T. gondii* infection

Inclusion Criteria: confirmed diagnosis of SLE ACR or SLICC

Exclusion Criteria: Pregnancy Other autoimmune diseases Acute bacterial or viral infection within 3 weeks Prior vaccine therapy (within the previous 3 months) Malignancy

Specimen Collection: Venous blood samples of 5–7 mL, Serum separation and storage at −20°C until analysis

Laboratory Investigations:

Diagnosis of Toxoplasma gondii: Number of anti- *T. gondii* IgG and IgM antibodies by ELISA

Published by Democratic Arabic Center for Strategic, Political and Economic Studies (DAC)

Autoantibody Tests: Anti-dsDNA antibodies: ELISA or Crithidia luciliae immunofluorescence assay, Anti-ribosomal P antibodies: ELISA, Anti-chromatin (minor DNA): ELISA

Clinical Assessment: Disease activity - SLEDAI, Renal and neuropsychiatric involvement recorded

Statistical Analysis

Data are presented as mean ± SD or median (interquartile range) for continuous variables. Univariate analysis was carried out using independent t-test or Mann–Whitney U test for comparison of the groups and Chi-square test for categorical variables. both Pearson and Spearman correlation analyses, Multivariate logistic regression analysis, adjusting for confounding factors. $P < 0.05$ as considered statistically significant

Ethical Issues: The approval was taken by the Institutional Ethical Committee Written informed consent for each patient Stricter confidentiality is maintained by hospital data.

Results:

Demographic and Clinical Characteristics

One hundred patients with SLE were included and separated by *T. gondii* serostatus into groups of seropositive (n = 48) and seronegative (n = 52). There were no significant differences in age, disease duration between the two SLE groups ($p > 0.05$). In contrast, the *T. gondii*–infected group had a higher SLEDAI score ($p < 0.001$).

Table 1. Demographic and Clinical Data of the Study Populations

Parameter	SLE patients with *T. gondii*-infection (n=48)	SLE patients without *T. gondii*-infection (n=52)	p-value
Age (years, mean ± SD)	34.6 ± 9.1	33.2 ± 8.7	0.42
Female (%)	43 (89.6%)	47 (90.4%)	0.88
Disease duration (years)	6.8 ± 3.4	6.2 ± 3.1	0.36
SLEDAI score	15.2 ± 4.6	10.4 ± 3.8	<0.001
Renal involvement (%)	27 (56.3%)	18 (34.6%)	0.03
Neuropsychiatric SLE (%)	14 (29.2%)	7 (13.5%)	0.04

Published by Democratic Arabic Center for Strategic, Political and Economic Studies (DAC)

Seroprevalence of Toxoplasma gondii

Anti-*T. gondii* IgG antibodies were found to be 48% in SLE patients. Anti-T. gondii IgM antibodies were positive in a small proportion of SLE patients, indicating recent or reactivated infection.

Table 2. *T. gondii* Seroprevalence

Patients	IgG Positive (%)	IgM Positive (%)
SLE patients (n=100)	48 (48.0%)	9 (9.0%)
p-value	0.03	0.21

Anti-dsDNA Antibody Levels

SLE patients with *T. gondii* infection had significantly higher mean serum anti-dsDNA levels than seronegative subjects ($p < 0.001$). Both the SLE groups displayed significantly increased frequencies.

Table 3. Anti-dsDNA Antibody Levels (IU/mL)

Patients	Mean ± SD	Range	p-value
SLE patients with *T. gondii* infection	112.6 ± 34.9	45–180	<0.001
SLE patients without *T. gondii* infection	78.4 ± 29.1	30–145	

Anti-Ribosomal P Antibodies

Anti-ribosomal P antibodies were present in 45.8% and 23.1% of *T. gondii* positive and negative SLE patients, respectively ($p = 0.01$). There was a strong association between positivity for anti-ribosomal P antibodies and neuropsychiatric symptoms ($p = 0.02$).

Table 4. Frequency of Anti-Ribosomal P Antibodies

Patients	Positive (%)	Negative (%)	p-value
SLE patients with *T. gondii* infection	22 (45.8%)	26 (54.2%)	0.01
SLE patients without *T. gondii* infection	12 (23.1%)	40 (76.9%)	

Published by Democratic Arabic Center for Strategic, Political and Economic Studies (DAC)

Anti-Chromatin (Minor DNA) Antibody Levels

Anti-chromatin antibody levels were extensively higher in *T. gondii* positive SLE patients when compared with seronegative individuals ($p < 0.001$).

Table 5. Anti-Chromatin Antibody Levels (U/mL)

Group	Mean ± SD	Range	p-value
SLE patients with *T. gondii* infection	96.3 ± 28.7	40–150	<0.001
SLE patients without *T. gondii* infection	64.9 ± 25.2	20–120	

Correlation Analysis

Strong associations were found between *T. gondii* IgG titter and autoantibody levels, as well as disease activity in general.

Table 6. Relationship of *T. gondii* IgG Titers and Clinical/Immunological Features

Parameter	r	p-value
Anti-dsDNA	0.52	<0.001
Anti-ribosomal P	0.41	0.002
Anti-chromatin	0.48	<0.001
SLEDAI score	0.56	<0.001

Multivariate Logistic Regression Analysis

After the adjustment for age, sex, disease duration and corticosteroid treatment, T. gondii seropositivity was independently predicative of high level of disease activity and elevated autoantibody levels.

Table 7. Variables correlated with high disease activity (SLEDAI ≥ 12)

Variable	OR	95% CI	p-value
***T. gondii* IgG positivity**	3.4	1.6–7.1	0.002
High anti-dsDNA titer	2.9	1.4–6.0	0.004
Anti-chromatin positivity	2.6	1.2–5.4	0.01
Disease duration	1.1	0.9–1.3	0.18

Published by Democratic Arabic Center for Strategic, Political and Economic Studies (DAC)

Discussion:

Systemic lupus erythematosus (SLE) is a complex autoimmune disease in which genomic factors, environmental influences and immune dysregulation interplay to promote the production of autoantibodies and consequent organ damage. Infectious agents have been suggested for many years to be a major environmental trigger that can modulate disease activity and Hausa autoimmune responses. In the present study, we investigated a possible role of *T. gondii* infection in autoantibody profiles and disease activity among SLE patients showing a strong correlation between seropositivity of toxoplasmosis infection with enhanced production of autoantibodies and severe disease.

As previously reported, patients with autoimmune disorders have been shown to have increased exposure or susceptibility to chronic infections and the present study showed that seropositivity for *T. gondii* infection was found among SLE patients significantly higher than healthy individuals. This could be suggestive of, immune dysfunction, prolonged iatrogenic immunosuppressive state or shared environment. *T. gondii* seroprevalence has also been observed in SLE patients from endemic areas, thereby demonstrating the importance of parasitic infections among populations affected by autoimmune diseases [27–29].

Among the interesting findings of the current study was a significantly higher titer of anti-dsDNA antibodies in *T. gondii* positive compared with T. gondii–negative SLE patients. Anti-dsDNA forms a significant component of SLE pathogenesis and are significantly associated with the IC deposition, complement activation, and resulting lupus nephritis. The present finding implies that life-long chronic *T.gondii* infection could result in overactivation of autoreactive B cell production due to continuous antigenic stimulation or just polyclonal activation of B cells, or it could be molecular mimicry. Experimental studies have reported that Toxoplasma gondii antigens can elicit strong Th1-polarized immune responses with high-level of IFN-γ and tumor necrosis factor, both known to promote autoantibody production and disease exacerbation in SLE [30–32].

In addition, to anti-dsDNA antibodies, T. gondii–positive patients had a significantly higher frequency of anti-ribosomal P antibodies. These autoantibodies are

Published by Democratic Arabic Center for Strategic, Political and Economic Studies (DAC)

somewhat unique to SLE and highly associated with neurologic disease. Anti-ribosomal P positivity was significantly associated with NP-SLE in the current cohort, as previously described.

This either crossing the blood–brain barrier by *T. gondii*, or its capacity to form cysts in neural tissue, can drive towards local immune activation and epitope spreading with effects on neurotropic autoantibody generation [33–35]. *T. gondii* infected SLE patients had similarly elevated anti-chromatin (anti-nucleosome) antibodies. Antibodies that had been the earliest one to appear compared with anti-dsDNA antibodies are being increasingly recognized as markers of disease activity and kidney involvement. The strong association between *T. gondii* IgG concentration and anti-chromatin antibodies identified in this study is consistent with the notion that chronic infection may enhance persisting apoptotic cellular turnover, as well as defective clearance of nuclear remnants leading to exposure to chromatinic antigens [36–38].

And significantly higher SLEDAI scores and renal involvement and neurologic symptoms were related to the presence of T. *gondii* antibodies. The infection remained a significant independent predictor of high disease activity over multivariate logistic regression analysis, despite controlling for confounding factors. Such observations suggest that, besides being a coincidental infection in patients with cancer already on treatment such as chemotherapy, chronic toxoplasmosis instead acts as an external potent immunomodulator causing heightened disease severity. Other intracellular pathogens such as Epstein–Barr virus and cytomegalovirus have also been reported to reactivate autoimmune disease activity, although this data stems from case reports where infectious flaring is described [39–41].

Several hypothetical immunopathogenic mechanisms can explain this finding. Persistent T. gondii infection leads to prolonged embeddedness of innate immune pathways (e.g., Toll like receptor signaling and inflammasome context) that may lower a threshold for autoimmune processes. Conversely, cross-reactive immune responses due to molecular mimicry between parasitic antigens and host nuclear proteins can lead to epitope spreading and breakdown of self-tolerance mediated by chronic antigen stimulation [42–44].

Published by Democratic Arabic Center for Strategic, Political and Economic Studies (DAC)

This study had several limitations, despite its strengths. A causal relationship cannot be drawn in this cross-sectional study, and longitudinal follow-up is necessary to determine whether T. gondii infection occurs before the elevation of autoantibodies or disease flare. Finally, burden or strain of parasite were not evaluated, which could impact immune responses and ultimate clinical sequelae. However, the uniformity of findings seen does provide evidence for a biologically plausible relationship between chronic toxoplasmosis and SLE pathogenesis.

Conclusions:

In the current study, we demonstrate that *Toxoplasma gondii* infection is linked to an amplified autoantibody response, as well as a more severe disease course in lupus patients. These observations carry implications for the concept of infection-induced autoimmunity and provide a rationale for considering long-term parasitic infections as potential modulators of susceptibility to autoimmune disease in endemic areas.
infection was significantly more frequent among SLE patients. SLE *T. gondii* seropositive patients showed increased antibodies against dsDNA, chromatin and ribosomal P. Seropositivity for *T. gondii* was significantly associated with higher activity of disease and with organ damage. Chronic toxoplasmosis seems to act by itself as an immunomodifier in SLE.

References:

1. Döşkaya M, Liang L, Jain A, Can H, İz SG, Felgner PL, et al. Discovery of new Toxoplasma gondii antigenic proteins using a high throughput protein microarray approach screening sera of murine model infected orally with oocysts and tissue cysts. Parasites & Vectors. 2018 Jul 4;11(1). Available from: https://doi.org/10.1186/s13071-018-2934-1
2. Gao Y, Guo H, Moumouni PFA, Liu M, Li J, Efstratiou A, et al. Development and evaluation of an enzyme-linked immunosorbent assay based on recombinant TgSRS2 for serodiagnosis of *Toxoplasma gondii* infection in cats. Journal of Veterinary Medical Science. 2020 Jan 1;82(11):1662. Available from: https://doi.org/10.1292/jvms.20-0231
3. Abbas M, Nasir A, Kashif M, Hussain K, Bano N, Raza MA, et al. Serodiagnosis of toxoplasma gondii, associated risk factors in domesticated cats: preventing zoonosis in humans and implications for livestock extension. International Journal

Published by Democratic Arabic Center for Strategic, Political and Economic Studies (DAC)

of Agricultural Extension [Internet]. 2021 Apr 7;9(1). Available from: https://doi.org/10.33687/009.01.3428

4. Kato K. How does *Toxoplama gondii* invade host cells? Journal of Veterinary Medical Science. Japanese Society of Veterinary Science; 2018 Jan 1;80(11):1702. Available from: https://doi.org/10.1292/jvms.18-0344
5. Daher D, Shaghlil A, Sobh E, Hamie M, Hassan ME, Moumneh MB, et al. Comprehensive Overview of Toxoplasma gondii-Induced and Associated Diseases. Pathogens. Multidisciplinary Digital Publishing Institute; 2021 Oct 20;10(11):1351. Available from: https://doi.org/10.3390/pathogens10111351
6. Rahman T, Gulshan JE, Rahman A. Impact of Toxoplasma gondii infection on human health. Bioresearch Communications. 2021 Dec 26;8(1):1093. Available from: https://doi.org/10.3329/brc.v8i1.57049
7. Didier K, Bolko L, Giusti D, Toquet S, Robbins A, Antonicelli F, Servettaz A (2018) Autoantibodies Associated with Connective Tissue Diseases: What Meaning for Clinicians? Frontiers in Immunology. https://doi.org/10.3389/fimmu.2018.00541
8. Cozzani E, Drosera M, Gasparini G, Parodi A (2014) Serology of Lupus Erythematosus: Correlation between Immunopathological Features and Clinical Aspects. Autoimmune Diseases 2014:1
9. Parodis I, Lagutkin D, Lindblom J, et al (2025) NEW IGG AND IGA AUTOANTIBODY SPECIFICITIES TARGETING DNA- AND RNA-BINDING PROTEINS DIFFERENTIATE SYSTEMIC LUPUS ERYTHEMATOSUS FROM HEALTHY INDIVIDUALS AND OTHER AUTOIMMUNE DISEASES. The Journal of Rheumatology. https://doi.org/10.3899/jrheum.2025-0390.pv226
10. Sciascia S, Bizzaro N, Meroni PL, et al (2023) Autoantibodies testing in autoimmunity: Diagnostic, prognostic and classification value. Autoimmunity Reviews 22:103356
11. Shi Z, Han Y, Yin J, Zhang Y, Jiang Z, Zheng L, Tan G, Wang L (2020) The diagnostic benefit of antibodies against ribosomal proteins in systemic lupus erythematosus. Advances in Rheumatology. https://doi.org/10.1186/s42358-020-00148-2
12. Frodlund M (2020) Antinuclear and antiphospholipid antibodies versus disease manifestations and clinical outcomes in systemic lupus erythematosus. Linköping University medical dissertations. https://doi.org/10.3384/diss.diva-164957

Published by Democratic Arabic Center for Strategic, Political and Economic Studies (DAC)

13. Choi MY, Fritzler MJ (2019) Challenges and Advances in SLE Autoantibody Detection and Interpretation. Current Treatment Options in Rheumatology 5:147
14. Bizzaro N, Villalta D, Bini V, et al (2022) Multiparametric autoantibody analysis: a new paradigm for the diagnosis of connective tissue diseases. Arthritis Research & Therapy. https://doi.org/10.1186/s13075-022-02980-x
15. Emerson J, Gruenewald SM, Gomes L, Lin M, Swaminathan S (2023) The conundrum of neuropsychiatric systemic lupus erythematosus: Current and novel approaches to diagnosis. Frontiers in Neurology. https://doi.org/10.3389/fneur.2023.1111769
16. Manca E (2021) Autoantibodies in Neuropsychiatric Systemic Lupus Erythematosus (NPSLE): Can They Be Used as Biomarkers for the Differential Diagnosis of This Disease? Clinical Reviews in Allergy & Immunology 63:194
17. Gulsara EK, Sharma A, Arju M, Ashraf Z, Rahaman A. SYSTEMIC LUPUS ERYTHEMATOSUS. International Research Journal of Modernization in Engineering Technology and Science. 2024 Mar 21; Available from: https://doi.org/10.56726/irjmets50763
18. Su X, Yu H, Lei Q, Chen X, Tong Y, Zhang Z, et al. Systemic lupus erythematosus: pathogenesis and targeted therapy. Molecular Biomedicine. Springer Nature; 2024 Oct 30;5(1):54. Available from: https://doi.org/10.1186/s43556-024-00217-8
19. Moulton VR, Suárez-Fueyo A, Meidan E, Li H, Mizui M, Tsokos GC. Pathogenesis of Human Systemic Lupus Erythematosus: A Cellular Perspective. Trends in Molecular Medicine. Elsevier BV; 2017 Jun 13; 23(7): 615. Available from: https://doi.org/10.1016/j.molmed.2017.05.006
20. Klimi E, Wong-Baeza C, Mora AR, Baeza I, Tescucano A, Barrera-Aveleida G, et al. Systemic Lupus Erythematosus - Pathogenesis and Management. IntechOpen eBooks. IntechOpen; 2022. Available from: https://doi.org/10.5772/intechopen.101008
21. Lupus: Public Education. In: Lupus. SAGE Publishing; 2024. Available from: https://doi.org/10.36255/lupus-public-education
22. Hoi A, Igel T, Mok CC, Arnaud L. Systemic lupus erythematosus. The Lancet. Elsevier BV; 2024 Apr 17;403(10441):2326. Available from: https://doi.org/10.1016/s0140-6736(24)00398-2
23. Panwar B, Schmiedel BJ, Liang S, White B, Rodríguez E, Kalunian K, et al. Multi–cell type gene coexpression network analysis reveals coordinated interferon

Published by Democratic Arabic Center for Strategic, Political and Economic Studies (DAC)

response and cross–cell type correlations in systemic lupus erythematosus. Genome Research. 2021 Mar 5;31(4):659. Available from: https://doi.org/10.1101/gr.265249.120

24. Rahman A, Isenberg D. Systemic Lupus Erythematosus [Internet]. Vol. 365, Elsevier eBooks. Elsevier BV; 2016. p. 312. Available from: https://doi.org/10.1016/b978-0-323-37754-6.50094-4
25. Makki K, Ahsan SA, Ali S. Systemic Lupus Erythematosus and its major organ complications: a narrative review on available literature. Liaquat National Journal of Primary Care. 2022 Jan 1; Available from: https://doi.org/10.37184/lnjpc.2707-3521.5.12
26. Primavera D, Fornaro M, Carrà G, Romano F, González CIA, Preti A, et al. Mood Disorder Questionnaire Positivity in Systemic Lupus Erythematosus and Other Chronic Diseases including Screen Bipolar Disorders or Rhythm and Energy Dysregulation Syndromes (DYMERS). Clinical Practice and Epidemiology in Mental Health. 2024 Aug 8;20(1). Available from: https://doi.org/10.2174/0117450179303653240705051227
27. Shoenfeld Y, Agmon-Levin N. "ASIA" – autoimmune/inflammatory syndrome induced by adjuvants. J Autoimmun. 2011;36(1):4–8.
28. Daryani A, Sharif M, Meigouni M, et al. Seroprevalence of *Toxoplasma gondii* in patients with autoimmune diseases. Iran J Parasitol. 2014;9(4):468–474.
29. Shapira Y, Agmon-Levin N, Shoenfeld Y. Defining and analyzing geoepidemiology and human autoimmunity. J Autoimmun. 2010;34(3): J168–J177.
30. Gazzinelli RT, Denkers EY. Protozoan encounters with Toll-like receptor signalling pathways: implications for host parasitism. Nat Rev Immunol. 2006;6(12):895–906.
31. Crispín JC, Tsokos GC. Interleukin-17-producing T cells in lupus. Curr Opin Rheumatol. 2010;22(5):499–503.
32. Munz C, Lunemann JD, Getts MT, Miller SD. Antiviral immune responses: triggers of or triggered by autoimmunity? Nat Rev Immunol. 2009;9(4):246–258.
33. Bonfa E, Golombek SJ, Kaufman LD, et al. Association between lupus psychosis and anti-ribosomal P protein antibodies. N Engl J Med. 1987;317(5):265–271.
34. Hanly JG. Neuropsychiatric lupus. Rheum Dis Clin North Am. 2005;31(2):273–298.

Published by Democratic Arabic Center for Strategic, Political and Economic Studies (DAC)

35. Carruthers VB, Suzuki Y. Effects of *Toxoplasma gondii* infection on the brain. Schizophr Bull. 2007;33(3):745–751.

36. Amoura Z, Koutouzov S, Piette JC. The role of anti-nucleosome antibodies in systemic lupus erythematosus. Curr Opin Rheumatol. 2000;12(5):369–373.

37. Burlingame RW, Boey ML, Starkebaum G, Rubin RL. Anti-chromatin antibodies: a sensitive marker for systemic lupus erythematosus. J Clin Invest. 1994;94(5):1844–1851.

38. Rekvig OP. Autoimmunity and SLE: Factual and semantic evidence-based critical analyses of definitions, etiology, and pathogenesis. Front Immunol. 2020; 11:569234.

39. James JA, Kaufman KM, Farris AD, et al. An increased prevalence of Epstein–Barr virus infection in young patients suggests a possible etiology for systemic lupus erythematosus. J Clin Invest. 1997;100(12):3019–3026.

40. Poole BD, Scofield RH, Harley JB, James JA. Epstein–Barr virus and molecular mimicry in systemic lupus erythematosus. Autoimmunity. 2006;39(1):63–70.

41. Barzilai O, Sherer Y, Ram M, Shoenfeld Y. Epstein–Barr virus and cytomegalovirus in autoimmune diseases: are they truly notorious? A preliminary report. Ann N Y Acad Sci. 2007; 1108:567–577.

42. Oldstone MB. Molecular mimicry and immune-mediated diseases. FASEB J. 1998;12(13):1255–1265.

43. Getts DR, Chastain EM, Terry RL, Miller SD. Virus infection, antiviral immunity, and autoimmunity. Immunol Rev. 2013;255(1):197–209.

44. Rose NR. Negative selection, epitope mimicry and autoimmunity. Curr Opin Immunol. 2017; 49:51–55.

Published by Democratic Arabic Center for Strategic, Political and Economic Studies (DAC)