

SARS-CoV-2 vaccines in patients with SLE

Wei Tang ,¹ Anca D Askanase ,¹ Leila Khalili ,¹ Joan T Merrill²

To cite: Tang W, Askanase AD, Khalili L, *et al.* SARS-CoV-2 vaccines in patients with SLE. *Lupus Science & Medicine* 2021;**8**:e000479. doi:10.1136/lupus-2021-000479

Received 15 January 2021
Revised 11 February 2021
Accepted 20 February 2021

ABSTRACT

As the Moderna (mRNA-1273) and Pfizer/BioNTech (BNT162b2) vaccines become available to patients with autoimmune diseases and SLE, practitioners will have to inform them about the safety and efficacy of these vaccines. Here we discuss the challenges of applying vaccine data to patients with autoimmune diseases and the evidence available in the literature that may help in the decision process.

The COVID-19 pandemic has affected nearly every corner of the world and changed the face of medicine. Almost a year into the pandemic, there have been over 91 million cases and more than 1 970 000 deaths globally.¹ During the winter of 2021, there has been a new surge of virulent strains in many parts of the world with no assurances of when lasting relief can be expected. Given these grim statistics, the approval and initial dispersion of the Moderna and Pfizer/BioNTech vaccines are monumental events. There is limited knowledge about the safety and efficacy of the COVID-19 vaccines in patients with SLE. While the overall safety and efficacy of the vaccines are reassuring, reports of immunological adverse reactions to the vaccine may be concerning to people with autoimmune diseases such as SLE.

SLE is characterised by dysregulation in type I interferon pathways.^{2 3} Type I interferons are key components of the innate and adaptive immune responses to new pathogens.⁴ The critical role of type I interferons in antiviral immunity is well known, including responses to SARS-CoV-2.^{5 6} While data are limited, patients with SLE do not seem to be at higher risk of SARS-CoV-2 infections or severe COVID-19 disease compared with the general population.^{7–9} However, patients with SLE may be at higher risk of hospitalisation during the COVID-19 disease course.^{10 11} In addition, it is possible that COVID-19 leads to increased SLE disease activity.^{12 13} COVID-19 has been associated with the development of autoantibodies in the serum of hospitalised patients and features suggestive of severe,

Key messages

What is already known about this subject?

- ▶ There have been previous studies on patients with SLE and COVID-19, and patients with SLE and vaccines; however, there is minimal information about patients with lupus and SARS-CoV-2 vaccinations.

What does this study add?

- ▶ This paper discusses the challenges of applying vaccine data to patients with autoimmune diseases and reviews the evidence available in the literature.

How might this impact clinical practice or future developments?

- ▶ This paper may help practitioners counsel patients with autoimmune diseases who are eligible for vaccination.

uncontrolled autoimmunity appear to be present in those most ill from COVID-19.^{14–16}

Patients with SLE in our cohort experienced COVID-19 infections at a rate similar to that of the general population (27/450 or 6%), and 6/27 (22.2%) experienced a disease flare within 22 days after developing symptoms.¹⁷ Of these six flares, five were mild/moderate and one was severe, using SLE Disease Activity Index Flare Index definitions. The symptoms manifested included arthritis, alopecia, rash, pleurisy and serological worsening (low complement components C3/C4 and antibodies to double-stranded DNA (anti-dsDNA)). Although these observations did not seem to indicate a high risk of serious flares, further long-term longitudinal studies are needed to determine the impact of COVID-19 infection on SLE disease activity.

Both Moderna (mRNA-1273) and Pfizer/BioNTech (BNT162b2) vaccines contain RNA that encodes the stabilised prefusion SARS-CoV-2 Spike (S) protein. The RNA enters host cells and produces non-virulent Spike protein that elicits high titres of neutralising antibodies and robust antigen-specific CD8+ and Th1-type CD4+ T-cell responses.^{18–21} The immunogenic protein includes the full-length S protein, with both the receptor-binding subunit (S1) and the membrane



© Author(s) (or their employer(s)) 2021. Re-use permitted under CC BY-NC. No commercial re-use. See rights and permissions. Published by BMJ.

¹Rheumatology, Columbia University Irving Medical Center, New York, New York, USA

²Arthritis and Clinical Immunology Program, Oklahoma Medical Research Foundation Arthritis and Clinical Immunology Research Program, Oklahoma City, Oklahoma, USA

Correspondence to

Dr Anca D Askanase; ada20@cumc.columbia.edu

fusion subunit (S2). These are the first mRNA-based antiviral vaccines in human use and, as such, produced some public controversy. Phase I trials found systemic side effects in up to 90% of participants.^{18 19} However, the late phase vaccine trials provided reassurance about their safety prior to what is planned to be the largest-scale worldwide vaccine distribution in history.^{22 23}

The efficacy rate in preventing COVID-19 infections for the Moderna (mRNA-1273) and Pfizer/BioNTech (BNT162b2) vaccines was 94.1% and 95.0%, respectively. The phase III trials of both vaccines reported low percentages of serious adverse events (below 0.1%).^{22 23} Local reactions were common and included tenderness, erythema and swelling at injection sites. Transient systemic reactogenicity such as fever, fatigue, headache, and muscle and joint pain were noted. These effects are generally seen within 24–48 hours following the vaccination and one or more may occur in up to 95% of vaccine recipients.^{22 23} Earlier work from Pfizer/BioNTech on a vaccine (BNT162b1) that encodes the secreted trimerised SARS-CoV-2 receptor-binding domain was abandoned because of greater severity of systemic reactions in older adults.^{18 24}

Unfortunately, patients treated with immunosuppressants or immune-modifying drugs for >14 days within 6 months of entry were excluded from all phases of Moderna (mRNA-1273) vaccine trials (NCT04470427).^{19 23} In the Pfizer/BioNTech (BNT162b2) vaccine trials, individuals with a history of autoimmune disease or an active autoimmune disease requiring therapeutic intervention were specifically excluded from phase I (NCT04368728), and patients who received treatment with immunosuppressive therapy, including cytotoxic agents or systemic corticosteroids, were excluded from phases I–III.^{22 24} The applicability of the published data to the safety and efficacy of COVID-19 RNA vaccines in patients with autoimmune disorders is unknown, especially in those patients receiving immunosuppressive treatment.

The dysregulation of the immune systems and concurrent use of immunosuppressive therapies raise theoretical concerns about the efficacy of vaccinations in patients with SLE. To the extent that optimal dosing of immune modulators restores immune set points towards normal, vaccine responses might be enhanced, allowing the development of protective immunity without major immunological side effects. However, since our current knowledge falls short of refined immunological control of the background immune disorder, vaccines could synergise with either incomplete or mistargeted immune modulation to either inhibit effective adaptive immunity or unleash excessive interferon responses. Indeed, as indirectly suggested by the results of a study of influenza vaccine in patients with lupus, greater interferon responses might in turn be associated with decreased neutralising antibodies to vaccinations, along with a greater development of autoantibodies and flares.²⁵ Thus, treatments targeting the immune system could have the potential to enhance or inhibit vaccine responses, depending on a patient's

underlying pathological immune variables and degree of immune suppression.

There are a number of factors at play that may provide unique challenges to the response of patients with lupus to SARS-CoV-2 vaccines. Quantitative and qualitative lymphocyte defects are prevalent in SLE. Lymphopenia is one of the most common haematological manifestations in SLE,²⁶ resulting from a combination of lymphocytotoxic antibodies, complement-mediated cytolysis, impaired lymphopoiesis, and side effects of immunosuppressants and B-cell depleting agents.^{27 28} In addition, CD8+ T-cell responses are dampened in patients with SLE, and the compromised cytotoxicity results in susceptibility to viral infections.^{29–32} Furthermore, decreased immunogenicity and inadequate immune responses have been reported in patients with SLE receiving vaccines against pneumococcus and influenza.^{33–35} Any condition that shortens durability of responses that build immunity or impairs immunological memory might weaken the long-term efficacy of vaccinations in patients with SLE. Some clinical evidence supports this theory. Serial antibody titers to capsular polysaccharides were followed up for 3 years in 19 patients with SLE after immunisation with polyvalent pneumococcal polysaccharide vaccine; at year 3, the antibody levels of vaccinated patients with SLE dropped below the threshold of protection in 8 of 19 (42%) patients, and 1 patient developed a pneumococcal pneumonia.³⁶ Taken together, these data illustrate that much remains to be learnt about vaccine responses in SLE. In the setting of dysregulated cellular and humoral immunity, patients with SLE might require modifications in vaccination strategies to achieve adequate and durable immune responses.

Previous studies have established that similar type I interferon and proinflammatory cytokine pathways are shared between SLE and COVID-19,^{2 3 5 6} which could either potentiate or dysregulate immune responses to SARS-CoV-2 vaccines while triggering disease activity in previously stable lupus. Vaccine-associated enhanced respiratory disease was not observed in either the Moderna (mRNA-1273) or Pfizer/BioNTech (BNT162b2) vaccine trials; the risk of vaccine-associated enhanced respiratory disease was observed in animal models of SARS-CoV and Middle East respiratory syndrome coronavirus infections, and it was associated with vaccine antigens that induce antibodies with poor neutralising activity and Th2-biased responses.^{37 38} The imbalance of Th1/Th2 subsets is important in the pathogenesis of SLE,^{39–41} which raises concern for a potential risk of developing vaccine-associated enhanced respiratory disease in patients with lupus. A second concern is that the SARS-CoV-2 vaccine delivers mRNA encoding S protein that is likely degraded by normal cellular process⁴² and could interact with a number of cytoplasmic RNA-binding proteins involved in the post-transcriptional regulation of inflammation; these pathways play a role in the pathogenesis of autoimmune diseases, as a consequence worsening SLE.⁴³ Similarly, RNA vaccines could trigger Toll-like receptors, setting

off the production of type I interferons.⁴⁴ However, there are no data yet to suggest that the dose and timing of an intramuscular injection of RNA vaccine would have any significant impact on the immune cells that underlie the pathogenic mechanisms of SLE.

The safety of routine immunisations against common pathogens has been well established in patients with SLE. Patients with SLE have been vaccinated against pneumococcus,⁴⁵ hepatitis B virus (HBV),⁴⁶ *Haemophilus influenzae* type B,⁴⁷ human papilloma virus (HPV)^{48,49} and varicella zoster virus with minimal concerning side effects.^{50–51} However, inactivated influenza vaccines were reported to trigger transient increases in a variety of serum autoantibody titres in patients with SLE, including ANA, dsDNA, anticardiolipin, anti-Smith, anti-RNP, anti-Ro and anti-La. This transient increase in autoantibody response is not necessarily or consistently associated with significant increases in clinical disease activity in all studies following these vaccinations.^{25 52 53} On the other hand, prospective monitoring for flares has been inconsistent. SLE flares occurred in only 4 out of 73 patients with SLE (5%) within 3 months of immunisation against poliomyelitis in a nationwide campaign following a 1988 Israeli outbreak.⁵⁴ A body of data is accumulating describing new-onset SLE or lupus-like syndromes following administration of HBV and HPV vaccines, but no causal relationships have been established.^{55–58}

The current mRNA vaccines for COVID-19 possess the advantages of enhanced stability and translational efficacy that potentiate the persistence of immunogen expression⁵⁹; additionally, the high immunogenic potency elicits strong immune responses.^{60 61} These two advantages, when combined together, may cause more immune stimulation than conventional vaccines that could potentially induce exacerbations in patients with existing autoimmune disorders or unmask de novo autoimmune diseases in predisposed individuals. Insights into the complex immune responses to mRNA vaccines in patients with autoimmune disease might provide clues to better treat the disease itself. For example, delivery of mRNA vaccine encoding multiple sclerosis specific autoantigens in a murine model successfully delayed onset and reduced severity of established disease by inducing antigen-specific tolerisation without general immunosuppression.⁶² Understanding the immunogenicity, efficacy, and safety of mRNA vaccines in people with autoimmune conditions could be of value in the therapeutic translation of this mRNA strategy.⁶³ As mRNA vaccines continue to be distributed over the next couple of months across the USA, observational research will be critical in determining the impact of COVID-19 vaccination in patients with autoimmune disease from both an efficacy and safety perspective.

Although the potential risks of COVID-19 vaccination remain unknown for patients with SLE and optimal dosing for efficacy could be different from the general population, the risks of not receiving the vaccine are far greater at the present time. When immunised, it seems

reasonable to take steps so that patients with lupus will be as stable as possible without receiving more immune suppression than needed to control disease activity. After vaccination, it might be worthwhile to monitor both SARS-CoV-2 antibody response and a full array of autoantibody titers. Close follow-up to identify early flares is also prudent. Because efficacy of the vaccine is unclear both in overly immunosuppressed patients and in those with disordered immune activity, patients with lupus should continue high vigilance in social distancing, wearing masks and avoiding unnecessary contact with other individuals.⁶⁴

The American College of Rheumatology COVID-19 Vaccine Clinical Guidance Task Force has just released a guidance document attempting to shed light on the complexities of vaccinating patients with autoimmune rheumatic diseases. The main guidance statements align with our suggestions. Patients with autoimmune rheumatic diseases should receive the COVID-19 vaccines and should be prioritised before the general population. Specific submissions regarding the timing and changes to commonly used medications are included with the caveat that these need to be individualised.⁶⁵

Contributors All authors contributed to data collection, analysis and manuscript preparation.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient and public involvement Patients and/or the public were not involved in the design, conduct, reporting or dissemination plans of this research.

Patient consent for publication Not required.

Provenance and peer review Not commissioned; externally peer reviewed.

Open access This is an open access article distributed in accordance with the Creative Commons Attribution Non Commercial (CC BY-NC 4.0) license, which permits others to distribute, remix, adapt, build upon this work non-commercially, and license their derivative works on different terms, provided the original work is properly cited, appropriate credit is given, any changes made indicated, and the use is non-commercial. See: <http://creativecommons.org/licenses/by-nc/4.0/>.

ORCID iDs

Wei Tang <http://orcid.org/0000-0002-6599-980X>

Anca D Askanase <http://orcid.org/0000-0003-4597-5023>

Leila Khalili <http://orcid.org/0000-0002-1070-0997>

REFERENCES

- 1 Johns Hopkins University coronavirus resource center, COVID-19 dashboard by the center for systems science and engineering (CSSE) at Johns Hopkins University 2021.
- 2 Postal M, Vivaldo JF, Fernandez-Ruiz R, *et al*. Type I interferon in the pathogenesis of systemic lupus erythematosus. *Curr Opin Immunol* 2020;67:87–94.
- 3 Weckerle CE, Franek BS, Kelly JA, *et al*. Network analysis of associations between serum interferon- α activity, autoantibodies, and clinical features in systemic lupus erythematosus. *Arthritis Rheum* 2011;63:1044–53.
- 4 McNab F, Mayer-Barber K, Sher A, *et al*. Type I interferons in infectious disease. *Nat Rev Immunol* 2015;15:87–103.
- 5 Hadjadj J, Yatim N, Barnabei L, *et al*. Impaired type I interferon activity and inflammatory responses in severe COVID-19 patients. *Science* 2020;369:718–24.
- 6 Arunachalam PS, Wimmers F, Mok CKP, *et al*. Systems biological assessment of immunity to mild versus severe COVID-19 infection in humans. *Science* 2020;369:1210–20.

- 7 Ramirez GA, Gerosa M, Beretta L, *et al.* COVID-19 in systemic lupus erythematosus: data from a survey on 417 patients. *Semin Arthritis Rheum* 2020;50:1150–7.
- 8 Favalli EG, Gerosa M, Murgu A. Are patients with systemic lupus erythematosus at increased risk for COVID-19? *Ann Rheum Dis* 2020.
- 9 Gartshteyn Y, Askanase AD, Schmidt NM, *et al.* COVID-19 and systemic lupus erythematosus: a case series. *Lancet Rheumatol* 2020;2:e452–4.
- 10 Fernandez-Ruiz R, Masson M, Kim MY, *et al.* Leveraging the United States epicenter to provide insights on COVID-19 in patients with systemic lupus erythematosus. *Arthritis Rheumatol* 2020;72:1971–80.
- 11 Gianfrancesco M, Hrych KL, Al-Adely S, *et al.* Characteristics associated with hospitalisation for COVID-19 in people with rheumatic disease: data from the COVID-19 global rheumatology alliance physician-reported registry. *Ann Rheum Dis* 2020;79:859–66.
- 12 Raghavan S, Gonakoti S, Asemota IR, *et al.* A case of systemic lupus erythematosus flare triggered by severe coronavirus disease 2019. *J Clin Rheumatol* 2020;26:234–5.
- 13 Kondo Y, Kaneko Y, Oshige T, *et al.* Exacerbation of immune thrombocytopaenia triggered by COVID-19 in patients with systemic lupus erythematosus. *Ann Rheum Dis* 2020. doi:10.1136/annrheumdis-2020-218157. [Epub ahead of print: 05 Aug 2020] (published Online First: 2020/08/08).
- 14 Zuo Y, Estes SK, Ali RA, *et al.* Prothrombotic autoantibodies in serum from patients hospitalized with COVID-19. *Sci Transl Med* 2020;12. doi:10.1126/scitranslmed.abd3876. [Epub ahead of print: 18 11 2020].
- 15 Fujii H, Tsuji T, Yuba T, *et al.* High levels of anti-SSA/Ro antibodies in COVID-19 patients with severe respiratory failure: a case-based review : High levels of anti-SSA/Ro antibodies in COVID-19. *Clin Rheumatol* 2020;39:3171–5.
- 16 Merrill JT, Erkan D, Winakur J, *et al.* Emerging evidence of a COVID-19 thrombotic syndrome has treatment implications. *Nat Rev Rheumatol* 2020;16:581–9.
- 17 Khalili L, Gartshteyn Y, Schmidt NM. COVID-19 Infections May Increase the Risk of SLE Flares [abstract]. *Arthritis Rheumatol* 2020;72.
- 18 Walsh EE, Frenck RW, Falsey AR, *et al.* Safety and immunogenicity of two RNA-based Covid-19 vaccine candidates. *N Engl J Med* 2020;383:2439–50.
- 19 Jackson LA, Anderson EJ, Roupael NG, *et al.* An mRNA Vaccine against SARS-CoV-2 - Preliminary Report. *N Engl J Med* 2020;383:1920–31.
- 20 Anderson EJ, Roupael NG, Widge AT, *et al.* Safety and immunogenicity of SARS-CoV-2 mRNA-1273 vaccine in older adults. *N Engl J Med* 2020;383:2427–38.
- 21 Walsh EE, Frenck R, Falsey AR, *et al.* RNA-Based COVID-19 vaccine BNT162b2 selected for a pivotal efficacy study. *medRxiv* 2020. doi:10.1101/2020.08.17.20176651. [Epub ahead of print: 20 Aug 2020].
- 22 Polack FP, Thomas SJ, Kitchin N, *et al.* Safety and efficacy of the BNT162b2 mRNA Covid-19 vaccine. *N Engl J Med* 2020;383:2603–15.
- 23 Baden LR, El Sahly HM, Essink B, *et al.* Efficacy and safety of the mRNA-1273 SARS-CoV-2 vaccine. *N Engl J Med* 2021;384:403–16.
- 24 Mulligan MJ, Lyke KE, Kitchin N, *et al.* Phase I/II study of COVID-19 RNA vaccine BNT162b1 in adults. *Nature* 2020;586:589–93.
- 25 Crowe SR, Merrill JT, Vista ES, *et al.* Influenza vaccination responses in human systemic lupus erythematosus: impact of clinical and demographic features. *Arthritis Rheum* 2011;63:2396–406.
- 26 Rivero SJ, Díaz-Jouanen E, Alarcón-Segovia D. Lymphopenia in systemic lupus erythematosus. Clinical, diagnostic, and prognostic significance. *Arthritis Rheum* 1978;21:295–305.
- 27 Ramos-Casals M, Sanz I, Bosch X, *et al.* B-Cell-Depleting therapy in systemic lupus erythematosus. *Am J Med* 2012;125:327–36.
- 28 Martin M, Guffroy A, Argemi X, *et al.* [Systemic lupus erythematosus and lymphopenia: Clinical and pathophysiological features]. *Rev Med Interne* 2017;38:603–13.
- 29 Katsuyama E, Suarez-Fueyo A, Bradley SJ, *et al.* The CD38/NAD⁺/SIRTUIN1/EZH2 Axis Mitigates Cytotoxic CD8 T Cell Function and Identifies Patients with SLE Prone to Infections. *Cell Rep* 2020;30:112–23.
- 30 Kis-Toth K, Comte D, Karampetsou MP, *et al.* Selective loss of signaling lymphocytic activation molecule family member 4-Positive CD8⁺ T cells contributes to the decreased cytotoxic cell activity in systemic lupus erythematosus. *Arthritis Rheumatol* 2016;68:164–73.
- 31 Larsen M, Sauce D, Deback C, *et al.* Exhausted cytotoxic control of Epstein-Barr virus in human lupus. *PLoS Pathog* 2011;7:e1002328.
- 32 Tsokos GC. Systemic lupus erythematosus. *N Engl J Med* 2011;365:2110–21.
- 33 Rezende RPV, Ribeiro FM, Albuquerque EMN, *et al.* Immunogenicity of pneumococcal polysaccharide vaccine in adult systemic lupus erythematosus patients undergoing immunosuppressive treatment. *Lupus* 2016;25:1254–9.
- 34 Saad CGS, Borba EF, Aikawa NE, *et al.* Immunogenicity and safety of the 2009 non-adjuvanted influenza A/H1N1 vaccine in a large cohort of autoimmune rheumatic diseases. *Ann Rheum Dis* 2011;70:1068–73.
- 35 Pugès M, Biscay P, Barnetche T, *et al.* Immunogenicity and impact on disease activity of influenza and pneumococcal vaccines in systemic lupus erythematosus: a systematic literature review and meta-analysis. *Rheumatology* 2016;55:1664–72.
- 36 McDonald E, Jarrett MP, Schiffman G, *et al.* Persistence of pneumococcal antibodies after immunization in patients with systemic lupus erythematosus. *J Rheumatol* 1984;11:306–8.
- 37 Jaume M, Yip MS, Cheung CY, *et al.* Anti-Severe acute respiratory syndrome coronavirus spike antibodies trigger infection of human immune cells via a pH- and cysteine protease-independent FcγR pathway. *J Virol* 2011;85:10582–97.
- 38 Graham BS. Rapid COVID-19 vaccine development. *Science* 2020;368:945–6.
- 39 Chan RW-Y, Lai FM-M, Li EK-M, *et al.* Imbalance of Th1/Th2 transcription factors in patients with lupus nephritis. *Rheumatology* 2006;45:951–7.
- 40 Muhammad Yusoff F, Wong KK, Mohd Redzwan N. Th1, Th2, and Th17 cytokines in systemic lupus erythematosus. *Autoimmunity* 2020;53:8–20.
- 41 Akahoshi M, Nakashima H, Tanaka Y, *et al.* Th1/Th2 balance of peripheral T helper cells in systemic lupus erythematosus. *Arthritis Rheum* 1999;42:1644–8.
- 42 Pardi N, Hogan MJ, Porter FW, *et al.* mRNA vaccines - a new era in vaccinology. *Nat Rev Drug Discov* 2018;17:261–79.
- 43 Yoshinaga M, Takeuchi O. RNA binding proteins in the control of autoimmune diseases. *Immunol Med* 2019;42:53–64.
- 44 Karikó K, Buckstein M, Ni H, *et al.* Suppression of RNA recognition by Toll-like receptors: the impact of nucleoside modification and the evolutionary origin of RNA. *Immunity* 2005;23:165–75.
- 45 Elkayam O, Paran D, Burke M, *et al.* Pneumococcal vaccination of patients with systemic lupus erythematosus: effects on generation of autoantibodies. *Autoimmunity* 2005;38:493–6.
- 46 Kuruma KAM, Borba EF, Lopes MH, *et al.* Safety and efficacy of hepatitis B vaccine in systemic lupus erythematosus. *Lupus* 2007;16:350–4.
- 47 Battafarano DF, Battafarano NJ, Larsen L, *et al.* Antigen-Specific antibody responses in lupus patients following immunization. *Arthritis Rheum* 1998;41:1828–34.
- 48 Pellegrino P, Carnovale C, Perrone V, *et al.* Human papillomavirus vaccine in patients with systemic lupus erythematosus. *Epidemiology* 2014;25:155–6.
- 49 David P, Shoenfeld Y. Human papillomavirus vaccine safety in systemic lupus erythematosus patients. *Lupus* 2020;29:1485–6.
- 50 Guthridge JM, Cogman A, Merrill JT, *et al.* Herpes zoster vaccination in SLE: a pilot study of immunogenicity. *J Rheumatol* 2013;40:1875–80.
- 51 Lai YC, Yew YW. Severe autoimmune adverse events post herpes zoster vaccine: a case-control study of adverse events in a national database. *J Drugs Dermatol* 2015;14:681–4.
- 52 Wiesik-Szewczyk E, Romanowska M, Mielnik P, *et al.* Anti-Influenza vaccination in systemic lupus erythematosus patients: an analysis of specific humoral response and vaccination safety. *Clin Rheumatol* 2010;29:605–13.
- 53 Abu-Shakra M, Press J, Sukenik S, *et al.* Influenza virus vaccination of patients with SLE: effects on generation of autoantibodies. *Clin Rheumatol* 2002;21:369–72.
- 54 Schattner A, Ben-Chetrit E, Schmilovitz H. Poliovaccines and the course of systemic lupus erythematosus--a retrospective study of 73 patients. *Vaccine* 1992;10:98–100.
- 55 Agmon-Levin N, Zafrir Y, Paz Z, *et al.* Ten cases of systemic lupus erythematosus related to hepatitis B vaccine. *Lupus* 2009;18:1192–7.
- 56 Geier DA, Geier MR. A case-control study of serious autoimmune adverse events following hepatitis B immunization. *Autoimmunity* 2005;38:295–301.
- 57 Geier DA, Geier MR. A case-control study of quadrivalent human papillomavirus vaccine-associated autoimmune adverse events. *Clin Rheumatol* 2015;34:1225–31.
- 58 Geier DA, Geier MR. Quadrivalent human papillomavirus vaccine and autoimmune adverse events: a case-control assessment of the vaccine adverse event reporting system (VAERS) database. *Immunol Res* 2017;65:46–54.
- 59 Karikó K, Muramatsu H, Welsh FA, *et al.* Incorporation of pseudouridine into mRNA yields superior nonimmunogenic vector

- with increased translational capacity and biological stability. *Mol Ther* 2008;16:1833–40.
- 60 Sahin U, Muik A, Derhovannessian E, *et al.* COVID-19 vaccine BNT162b1 elicits human antibody and T_H1 T cell responses. *Nature* 2020;586:594–9.
- 61 Pardi N, Hogan MJ, Naradikian MS, *et al.* Nucleoside-Modified mRNA vaccines induce potent T follicular helper and germinal center B cell responses. *J Exp Med* 2018;215:1571–88.
- 62 Krienke C, Kolb L, Diken E, *et al.* A noninflammatory mRNA vaccine for treatment of experimental autoimmune encephalomyelitis. *Science* 2021;371:145–53.
- 63 Serra P, Santamaria P. Antigen-Specific therapeutic approaches for autoimmunity. *Nat Biotechnol* 2019;37:238–51.
- 64 Centers for disease control and prevention, COVID-19 information page, 2021. Available: <https://www.cdc.gov/coronavirus/2019-nCoV/index.html>
- 65 American College of Rheumatology (ACR) COVID-19 Vaccine Clinical Guidance Task Force. COVID-19 vaccine clinical guidance summary for patients with rheumatic and musculoskeletal diseases, 2021. Available: <https://www.rheumatology.org/Portals/0/Files/COVID-19-Vaccine-Clinical-Guidance-Rheumatic-Diseases-Summary.pdf>

Correction: SARS-CoV-2 vaccines in patients with SLE

Tang W, Askanase AD, Khalili L, *et al.* SARS-CoV-2 vaccines in patients with SLE. *Lupus Sci Med* 2021;8:e000479. doi: 10.1136/lupus-2021-000479.

The authors want to alert the readers on the three references cited in their paper which are of scientifically doubtful credibility.

Regarding our paper entitled “SARS-CoV-2 vaccines in SLE patients,” it has come to our attention that the authors of references 56–58 have been sanctioned multiple times for misrepresentation of credentials, intellectual dishonesty and violations of good clinical practice and scientific method in clinical and epidemiologic studies. For this reason, references 56–58 must be considered unreliable.



OPEN ACCESS

Open access This is an open access article distributed in accordance with the Creative Commons Attribution Non Commercial (CC BY-NC 4.0) license, which permits others to distribute, remix, adapt, build upon this work non-commercially, and license their derivative works on different terms, provided the original work is properly cited, appropriate credit is given, any changes made indicated, and the use is non-commercial. See: <http://creativecommons.org/licenses/by-nc/4.0/>.

Lupus Sci Med 2021;8:e000479corr1. doi:10.1136/lupus-2021-000479corr1

