Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

reproducible with rapidly available results, they will not be able to guide clinical decision making in real time. IL-18 and S100A12 have the potential to be important tools in the paediatric rheumatologist's arsenal for combatting cytokine storm syndromes, but further efforts are needed to maximise these biomarkers so that they can be deployed to the front lines and improve patient outcomes.

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Lauren A Henderson
lauren.henderson@childrens.harvard.edu

Karp Family Research Building, Boston, MA 02115, USA

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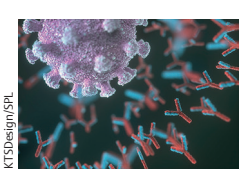
Systemic lupus erythematosus does not prevent antibody responses to SARS-CoV-2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See **Articles** page e585

From the beginning of the COVID-19 pandemic, patients with systemic lupus erythematosus (SLE) were particularly concerned about their risks following exposure to the virus and so were the physicians caring for them. After all, it seemed entirely possible that SLE and its therapy might reduce the immune response to SARS-CoV-2 and increase the risk of severe COVID-19 outcomes. Owing to immunoglobulin and immune complex formation and the ensuing release of interferons,[1] SLE mimics much of the immune response against viral infection. Accordingly, therapeutic action in SLE is designed to disrupt these feedback loops and to reduce the formation of antibodies. In March–April, 2020, patients with SLE were more commonly admitted to hospital for COVID-19 than the general population,[2] and in a study[3] done between April 13 and June 1, 2020, in New York City, four of 41 patients with SLE and RT-PCR-confirmed SARS-CoV-2 infection died, although it should be noted that three of these four patients refused intubation.[3]

As more data became available, most of the fears surrounding COVID-19 outcomes in patients with SLE were alleviated. The proportion of hospital admissions for patients with SLE diagnosed with COVID-19 was not significantly increased compared to patients with other rheumatic diseases (odds ratio [OR] 1·80; 95% CI 0·99–3·29; p=0·06),[4] which might be explained in part by increased caution. Compared to patients with rheumatoid arthritis, a diagnosis of SLE was not associated with an increased odds of death from COVID-19 (OR 1·2; 95% CI 0·70–2·04),[5] despite a higher prevalence of organ damage in this group.

In *The Lancet Rheumatology*, Amit Saxena and colleagues[6] fill another relevant gap in our knowledge, by investigating whether patients with SLE are able to mount a sufficient antibody response to SARS-CoV-2.

By analysing data from 329 patients with SLE from the New York City area between April 29, 2020, and Feb 9, 2021, Saxena and colleagues[6] identified 51 (16%) patients who had a positive SARS-CoV-2 antibody test.[6] 24 of these patients had a positive RT-PCR result, one had a negative RT-PCR result, and 26 did not undergo RT-PCR testing (similarly to many other patients with COVID-19 during the early stages of the pandemic). The calculated seroprevalence of 16% was not far off from the estimated seroprevalence of 20% found in repeated cross-sectional seromonitoring studies of the New York City population during the same period.[7] A seroprevalence of 20%, which would suggest that around 1·6 million New Yorkers had COVID-19, is also in line with the 17 000 deaths from COVID-19 reported in the city during this period. The authors' argument that the lower seroprevalence in New York City's SLE population might be a consequence of extra caution exercised by patients with SLE appears convincing.

There was a notable difference between ethnicities, in that 26% (24 of 91) of Hispanic patients with SLE were positive for SARS-CoV-2 antibodies, compared with 11% (27 of 238) of non-Hispanic patients. These differences are likely to be due to factors such as family size and workplace environment, as well as differences in social behaviour, which could have an impact on the rate of infection.

This said, the by far most relevant finding reported by Saxena and colleagues[6] relates to the development of antibodies against SARS-CoV-2. When focusing on the 29 patients with symptomatic COVID-19 subsequently confirmed by RT-PCR, 24 (83%) were positive for SARS-CoV-2 IgG antibodies when tested, and five were formally antibody negative. One of the five antibody-negative patients had mild disease, which might or might not result in SARS-CoV-2 antibodies. In three other patients, who all had a moderate course with fever and pneumonia or nausea, this finding is less clear. All three of these patients were receiving immunosuppression, namely mycophenolate mofetil and tacrolimus for transplantation, and cyclophosphamide or mycophenolate mofetil plus the B-cell-depleting antibody obinutuzumab for active lupus nephritis. The fifth patient presented without upper respiratory symptoms.

However, two other patients treated with mycophenolate mofetil, one with cyclophosphamide, and three with the B-cell depleting antibody rituximab, were able to mount an antibody response. The same was true for three patients receiving belimumab, and hydroxychloroquine and prednisone did not appear to inhibit the antibody response either, although maximum prednisone doses did not exceed 10 mg daily.[8] These findings should encourage patients with SLE to continue their prescribed therapy. Given the absence of a pattern in immunosuppression in the three more severe patients who did not develop SARS-CoV-2 antibodies, it is tempting to speculate that lupus nephritis might have played a role, since three patients had this most common severe organ manifestation. After all, lupus nephritis is proteinuric by nature, and proteinuria might also include immunoglobulin loss.

Overall, however, patients with SLE were not only able to produce sufficient amounts of IgG antibodies against SARS-CoV-2 to reach the defined positive range, they also mostly maintained positive antibody levels for up to 40 weeks. Although there was an apparent decline over time, this decline did not exceed what has been reported in other populations;[9] moreover, the long-term impact of SARS-CoV-2 on the immune system is still not yet fully understood.

It remains to be confirmed whether these SARS-CoV-2 antibodies are protective in patients with SLE. However, at present there are no convincing arguments that patients with SLE who recover from COVID-19 should differ from other patients who recover from COVID-19, and the tests used generally correlated well with SARS-CoV-2 IgG antibodies. Patients with SLE should therefore be able to maintain protective antibodies for around half a year.

After 6 months, at the latest, they should be vaccinated. Many patients with SLE in New York City are likely to have been vaccinated by now. In other regions, vaccination of all people or all vulnerable groups at the very least might not yet be feasible, but must remain an important goal. This leaves us with one remaining question: will patients with SLE respond to vaccination as well as other individuals? Again, the variety of approaches targeting autoantibody formation, from cyclophosphamide to mycophenolate mofetil, and from belimumab to B-cell depletion with rituximab or obinutuzumab, might dampen the immune response.

However, the data presented by Saxena and colleagues seem to indicate that the humoral immune response of patients with SLE is more robust than perhaps thought, and initial data[10] from a cohort of 26 patients with a

variety of treated autoimmune conditions suggest that vaccination will also be protective in these patients. Although we do not have the complete picture yet, these initial findings should be reassuring for patients with SLE.

I declare no competing interests.

Martin Aringer
martin.aringer@uniklinikum-dresden.de

Division of Rheumatology, Department of Medicine III, University Medical Center and Faculty of Medicine Carl Gustav Carus at the Technische Universität Dresden, 01307 Dresden, Germany

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Patient and public involvement in rheumatology research: embracing the wave of change


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Patient partnership in research is crucial to ensure that research priorities align with the priorities of patients. There are various ways in which patients can be integrated into research.[1] Three facets are important to ensure successful, patient-centred research: patient involvement, patient engagement, and patient participation. All three require long-term investment to nurture patient–researcher and patient–clinician relationships, supported by resources developed by organisations such as INVOLVE, a national advisory group funded by the National Institute for Health Research (NIHR) that supports public involvement in research.[1] While patient and public involvement has been shown to promote higher standards of research[2] and improve patient outcomes,[3] there is a need to enhance the consistency, quality, and frequency of activities around patient and public involvement.[4]

Patient and public involvement is particularly crucial in rheumatology research, as most rheumatic and musculoskeletal conditions are lifelong and life-altering. To foster high-quality, patient-focused research and ensure that patient and public involvement activities have a positive impact on research, these activities should be undertaken in a structured manner, guided, for example, by the UK Standards for Public Involvement.[5] The beneficial effects of patient and public involvement on health research have been reported, not only from the researcher perspective,[6] but also from the patient and public perspective,[6,7] where it has been described to positively influence the research process; this influence extends from the initial stages of research, with the development of research questions, research design and delivery, data collection, and analysis, through to dissemination.[8] In addition, patient and public involvement has been described to have a positive impact on enrolment of patients in research studies, as reported in a systematic review and meta-analysis.[9]

Rheumatic conditions and therapeutics can rapidly evolve and world events, such as the COVID-19 pandemic, can bring unanticipated issues and new research priorities that require appropriate and timely responses that are best facilitated through established frameworks, supported by academic institutional infrastructure. Here, we detail the experiences and patient and public involvement models of three academic centres in the UK.