

Response to: 'Are patients with systemic lupus erythematosus at increased risk for COVID-19?' by Favalli *et al*

We thank Favalli *et al* for their interest in our study reporting the course of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) disease 2019 (COVID-19) in a case series of 17 patients with systemic lupus erythematosus (SLE) under long-term treatment with hydroxychloroquine (HCQ).^{1 2} As mentioned in our study, we did not aim at reporting the incidence rate and the severity of COVID-19 in SLE, because our cohort most likely over-represents the most symptomatic and severe cases, as a result of a bias in the selection procedure of the patients used by the physicians.

Favalli *et al*, by studying the nasopharyngeal carriage of SARS-CoV-2, confirm the low prevalence of COVID-19 in their cohort of patients with SLE, similar to that reported in the general population even in epicentres of the outbreak. By May 11, when France eased the COVID-19 lockdown, Salje *et al* projected that only 5.7% (range 3.5 – 10.3) of the general French population had been infected and that this proportion was likely to be 12.3% (range 7.9 – 21.3) in Ile-de France, which includes Paris, and 11.8% (range 7.4 – 20.5) in Grand Est, the two most affected regions of the country.³ Furthermore, in the general population, as probably in patients with SLE, most infected patients display only mild symptoms, if any, without the need for hospital care, whereas even in the case of hospitalisation, death occurs in less than 6% of cases during the course of the disease.⁴ Therefore, it is not surprising that Favalli *et al*, in a series of 62 patients with SLE, did not observe cases of nasopharyngeal swab positivity for SARS-CoV-2 and that only eight (13%) patients reported symptoms consistent with viral infection. Only larger studies describing the incidence and severity of COVID-19 in patients with SLE, based on the detection of SARS-CoV-2, as well as specific antiviral antibodies, will help decipher the prevalence and the risk factors of severe COVID-19 in this fragile population suffering from comorbidities such as cardiovascular or chronic kidney disease. However, even if their cohort size is rather limited, the authors' observation that patients with SLE respecting strict contagion prevention rules do not show an increased risk of developing COVID-19 is very encouraging.

Data collected through the COVID-19 Global Rheumatology Alliance registry recently confirmed that patients with lupus on baseline therapy with HCQ are not universally protected from COVID-19.⁵ Therefore, we commend Favalli *et al* to emphasise the primordial role of physical distancing and the adoption of strict rules for the prevention of contagion, because of the uncertain protection of patients with SLE from severe SARS-CoV-2 infection by HCQ treatment.

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