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If skin is a potential host of SARS-CoV-2, IL-17 antibody could reduce the risk of COVID-19

To the Editor: In the era of the coronavirus disease 2019 (COVID-19) pandemic, debates have emerged on whether biologics might increase the risk of contracting the disease. Interleukin (IL) 17 is a biologic that is widely used in dermatology. There were reports that viral reactivation, although extremely low, could be detected during the use of IL-17 antibody (160 mg subcutaneously at Week 0, followed by 80 mg at Weeks 2, 4, 6, 8). This led to concerns in using the IL-17 antibody because it was believed that it could make patients more susceptible to severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). When we read the article by Sun et al, in a recently published issue, a question occurred to us: If skin is a target of SARS-CoV-2, what might be the consequence of using the IL-17 antibody?

Angiotensin-converting enzyme 2 (ACE2) is the main entrance receptor for SARS-CoV-2. Its expression is associated with the risk of making the target tissue susceptible to infection by SARS-CoV-2. Therefore, downregulating the expression of ACE2 could decrease the risk of COVID-19. To evaluate the influence of IL-17 antibody on skin ACE2 expression, we randomly selected 5 psoriasis patients who were treated with IL-17 antibody (Taltz, Eli Lilly and Company, Indianapolis, IN). The skin lesions of these patients were biopsied on week 0 and week 8 and prepared for RNA sequencing. The skin ACE2 expression of patients who underwent the antibody therapy for 8 weeks (0.36 ± 0.10; n = 5) was downregulated compared with that at week 0 (1.24 ± 0.50; n = 5), when the IL-17 antibody treatment had just begun (P < .05, paired t test). To confirm the result, we also selected 3 patients to compare the skin ACE2 expression at weeks 0 and 8 with immunofluorescence. Immunofluorescence staining revealed that the fluorescence intensity of ACE2 was downregulated in the skin at week 8 (0.84 ± 0.26; n = 3) compared with that before the IL-17 antibody treatment (9.23 ± 2.33; n = 3; P < .05; unpaired t test). Hence, either the messenger RNA or protein of ACE2 obtained from psoriasis patients can reveal that IL-17 antibody treatment remarkably reduces ACE2 expression.

Our above-mentioned work proves that IL-17 antibody treatment during the COVID-19 pandemic is not contraindicated. Elevated ACE2 expression and detection of SARS-CoV-2 in the skin of COVID-19 patients implied skin was a potential host of SARS-CoV-2. After IL-17 antibody treatment, the skin ACE2 expression was downregulated, which meant IL-17 antibody could decrease the risk of COVID-19 through lessening the cells that could interact with SARS-CoV-2. Additionally, IL-17 antibody could reverse the deteriorated barrier and inflammatory status in the skin of psoriasis patients, which meant less microbe infection. Hence, the specific microbe could be SARS-CoV-2. To our knowledge, until now there has been no evidence that COVID-19 can be spread by contact with skin. However, SARS-CoV-2 could survive on skin for about 9 hours, which indicates that it might be transmitted through skin in certain skin conditions such as psoriasis. Thus, whether IL-17 antibody could reduce the COVID-19 risk through reversing the inflammatory skin status with a deteriorated barrier and preventing SARS-CoV-2 transmission should be further discussed.

Qiannan Xu, MD, Libong Chen, MD, PhD, Xia Li, MD, PhD, and Jie Zheng, MD, PhD
From the Department of Dermatology, Ruijin Hospital, School of Medicine, Shanghai Jiao Tong University, Shanghai, People’s Republic of China.

Funding sources: None.

Conflicts of interest: None disclosed.

IRB approval status: Not applicable.

Reprints not available from the authors.

Correspondence to: Jie Zheng, MD, PhD, No. 197 Ruijin Rd Number Two, Shanghai, China
E-mail: jie-zheng2001@126.com

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https://doi.org/10.1016/j.jaad.2020.10.084