Response to "New-onset pustular psoriasis in the setting of severe acute respiratory syndrome coronavirus 2 infection causing coronavirus disease 2019"

To the Editor: We read with interest the recent report of a new-onset of pustular psoriasis in the setting of SARS-COV-2 infection by Matthieu et al.[1] Before the SARS-COV-2 pandemic context, a spectrum of respiratory viruses (RV) has been identified for the first time by our group as triggering factors of different psoriasis endotypes flares, including 3 generalized pustular psoriasis (GPP), 2 of which carryied homozygous IL36RN mutations.[2] In our study, several RNA viruses, mainly Rhinovirus and Coronavirus were detected using multiplex polymerase-chain reaction on nasopharyngeal swabs.

These observations support the contribution of viral triggers in predisposing specific genetic backgrounds in psoriasis pathogenesis. Regarding patients with Deficiency in Interleukin-36 receptor antagonist, it is worth emphasizing that the expression of IL-36γ and CXCL8 cytokines is enhanced in primary keratinocytes following exposure to poly-inosinic-polycytidilic acid, a Toll-Like-Receptor 3 agonist, which mimics respiratory virus double stranded RNA.[3] Furthermore, several studies reported a type-I interferon (IFN-I) signature in GPP and psoriasis vulgaris, with some correlation with a deregulation of the IL-36 pathway.[4] In addition, IL-36 inflammatory cytokines have been shown to be produced and released by bronchial epithelial cells after stimulation with dsRNA. Altogether, and taking into account the major role of the IFN-I pathway immune responses to RV, including toward SARS-COV-2,[5] these data argue for an involvement of IL-36 and IFN-I pathways in RV infection-induced flares of psoriasis, and question the mechanistic links between respiratory and skin inflammatory effectors.

Finally, the short time interval between RV infection and the onset of psoriasis flare-ups raises the challenge of immunosuppressive drug intervention in a context of ongoing viral infection, especially in the case of SARS-COV-2. This paradigm also applies to recently developed IL-36-blocking strategies, which appear appealing in GPP.

The report by Matthieu et al brings up SARS-CoV2, among other RV, as a potent trigger of the skin immune system in GPP. Dissecting the immunogenotypic architecture underlying these models might not only help understand psoriasis pathophysiology, but also drive precision therapeutic approaches for the benefit of these patients.

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Conflicts of interest

None disclosed.

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