keratinocytes in LP,\textsuperscript{1,5} with interferon-\(\gamma\) promoting susceptibility to CD8\textsuperscript{+} T cell attack via major histocompatibility complex I induction.\textsuperscript{3} Shao \textit{et al}. found cytotoxic T-cell-mediated injury to keratinocytes in LP to be dependent upon JAK-2 and signal transducer and activator of transcription 1 signalling, and inhibited by the JAK-1/2 inhibitor baricitinib.\textsuperscript{5} Therefore, it is plausible that baricitinib suppresses the lichenoid tissue reaction in LPP, accounting for the favourable response observed in our patient.

To our knowledge, this is the first case of LPP successfully treated with baricitinib. While further studies are required to support our findings, in the absence of effective treatments for recalcitrant LPP, baricitinib may be a suitable addition to the therapeutic armamentarium.

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Conflict of interest: RS is the Director and Founder of Samson Clinical Pty Ltd, is on the pharmaceutical advisory board of Eli Lilly, Pfizer Inc. and Leo Pharma; is in the speaker bureau of AbbVie and Novartis; and has worked as a principal investigator in clinical trials for Amgen, Novartis, Arcutis Biotherapeutics, Aerotech, Merck and Co., Celgene, Coherus BioSciences, Janssen, Regeneron, MedImmune, Glaxo Smith Kline, Samson Clinical, Boehringer Ingelheim, Oncobiologics, Roche, Ascend, Dermira, AstraZeneca, Akesobio, Reistone Biopharma, UCB, Sanofi, Connect Biopharma, Arena, Sun Pharma, Bristol Myyer Squibb and Galderma. The other authors declare that they have no conflict of interest.

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Data availability: data are available on reasonable request from the corresponding author.

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**Immune response to SARS-CoV-2 mRNA vaccine in patients with psoriasis treated with biologics**

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Dear Editor,

The effect of psoriasis treatment with biologics on the efficacy of COVID-19 vaccines is largely unknown.\textsuperscript{1} Biologics for psoriasis treatment represent one of the most significant therapeutic progresses in dermatology.\textsuperscript{2} The four classes of biologics approved for the treatment of psoriasis include tumour necrosis factor (TNF) inhibitors, interleukin (IL)-12/23, IL-17 inhibitors and IL-23 inhibitors.\textsuperscript{3} Data show that treatment with TNF inhibitors, IL-12/23 inhibitors and IL-17 inhibitors is not associated with lower antibody response to vaccination against pneumococcus, meningococcus, influenza or tetanus; however, large prospective studies assessing immune response against different vaccines are warranted, and the data on IL-23 inhibitors are limited.\textsuperscript{4}

In this study, we aimed to evaluate antibody response against the SARS-CoV-2 virus following two doses of BNT162b2 (Pfizer/BioNTech) in patients with psoriasis receiving biologic monotherapy, and compare it with that of a healthy control (HC) group.

For this prospective observational study, we recruited patients from the Department of Dermatovenerology, University Medical Centre (UMC) Maribor. Patients with a history of prior SARS-CoV-2 infection and positive specific IgG antibodies to SARS-CoV-2 prior to vaccination were excluded. The HC group consisted of healthcare personnel from UMC Maribor.

Blood samples were collected before the initial vaccination and 4 weeks after the second dose (standard two-dose vaccine with a 3-week interval) without interruption in biologic therapy. An indirect chemiluminescence immunoassay (LIASON\textsuperscript{5} SARS-CoV-2 TrimericS; DiaSorin, Saluggia, Italy) for the detection of IgG antibodies to SARS-CoV-2 in human serum or plasma samples was performed in accordance with the manufacturer’s instructions. Total IgG antibodies against SARS-CoV-2 spike protein were quantified and interpreted as positive (\(\geq 33.8\) binding antibody units (BAU)/mL) or negative (< 33.8 BAU/mL).

Statistical analyses were performed using SPSS software (V28.0; IBM Corp., Armonk, NY, USA), and results were expressed as mean ± SD. Differences were analysed statistically by independent samples \(t\)-test, \(\chi^2\) test and one-way ANOVA. \(p < 0.05\) was considered statistically significant.
In total, 32 patients and 22 controls were recruited. The mean age of patients was 55.9 years (range 34–75 years), and the mean age of controls was 46.0 years (range 24–76 years) ($P < 0.01$). Participants were matched for sex (Table 1), and all were of white ethnicity. The mean psoriasis duration was 24.5 years (range 4–70), and the mean therapy duration was 41.1 months (range 1–114 months).

All patients (100%) had positive antibody response, and 1 of 22 HCs was interpreted as negative (4.5%), the difference not being statistically significant ($P = 0.22$). Antibody titres were significantly lower in patients than HCs ($1024.4 \pm 870.3$ vs. $3055.8 \pm 2450.9$, $P < 0.001$). There was no significant difference in antibody titres in patients aged $\leq 55$ years and patients aged $> 55$ years ($1150.7 \pm 966.8$ vs. $898.1 \pm 772.4$, $P < 0.05$), or between different treatment groups ($P = 0.11$) (Table 1, Fig. 1).

A study of antibody titres to SARS-CoV-2 vaccine following an initial dose of either BNT 162b2 (Pfizer/BioNTech) or AZD1222 (AstraZeneca) in patients with psoriasis (107 of 120) and other immune-mediated inflammatory diseases receiving biologic and/or oral non-biologic immunomodulators showed that 15% of patients failed to mount a detectable antibody response; however, this study had no control group. In a recent study of 26 patients with chronic inflammatory diseases (6 patients with psoriasis or psoriatic arthritis) and 42 HCs, antibody

### Table 1 Characteristics of 32 patients and 22 controls.

| Characteristic                  | Patients ($n = 32$) | Controls ($n = 22$) | $P$   |
|--------------------------------|--------------------|--------------------|-------|
| Age, years; mean ± SD          | 55.9 ± 11.3        | 46.0 ± 13.1        | $< 0.01$ |
| Female, n (%)                  | 14 (43.8)          | 13 (59.1)          | 0.27  |
| Disease duration, years; mean ± SD | 24.5 ± 16.5     | NA                 |       |
| Therapy duration, months; mean ± SD | 41.1 ± 33        | NA                 |       |
| Psoriatic arthritis, n (%)    | 10 (31.3)          | NA                 |       |
| Number of responders, n (%)   | 32 (100)           | 21 (95.5)          | 0.22  |
| Antibody titre, mean ± SD (BAU/mL) | 1024.4 ± 870.3    | 3055.8 ± 2450.9   | $< 0.001$ |
| Age group, years; mean ± SD   |                   |                    |       |
| $\leq 55$ (16 of 32; 50.0%)    | 1150.7 ± 966.8     | –                  |       |
| $> 55$ (16 of 32; 50.0%)      | 898.1 ± 772.4      | –                  | $< 0.05$ |
| Drug class, mean ± SD         |                   |                    |       |
| TNF (7 of 32; 21.9%)          | 1044.7 ± 631.8     | –                  |       |
| IL-12/23 (11 of 32; 34.4%)    | 1357.7 ± 1147.6   | –                  |       |
| IL-17 (6 of 32; 8.8%)         | 841.8 ± 679.8     | –                  |       |
| IL-23 (8 of 32; 25%)          | 685.1 ± 682.2     | –                  | 0.11  |

BAU, binding antibody units; IL, interleukin; TNF, tumour necrosis factor.

![Figure 1](image.png)
Our data showed no difference in the rate of seroconversion, but significantly lower titres were observed in patients with psoriasis treated with biologic monotherapy, and there were no significant differences between the four treatment groups. These lower titres suggest the need for the recommended booster shot; however, the exact scheduling for patients with psoriasis on biologics has to be determined.

The limitations of our study include small number of participants, not testing cell-mediated immunity, and not matching patients and controls for age. Further controlled studies including large number of participants, with longitudinal design, and assessing immunological and clinical efficacy simultaneously are needed.

Successful treatment of severe pityriasis rubra pilaris with secukinumab in a 3-year-old boy

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Dear Editor,

Pityriasis rubra pilaris (PRP) is a rare, scaly, keratotic inflammatory skin disease characterized by red scaly patches, keratosis papules, palmoplantar keratoderma and scaling of the scalp. In severe cases, ectropion of the eyelid may occur, and erythroderma may further develop. Recently, it has been reported that secukinumab, a monoclonal anti-interleukin (IL)-17A antibody, may have efficacy in the treatment of PRP. We report a 3-year-old Chinese boy with severe Type III (classic juvenile) PRP who was successfully treated with secukinumab alone.

A 3-year-old Chinese boy presented with a 20-day history of rapidly progressing generalized hyperkeratotic follicular papules with severe pruritus. The patient’s mother reported a history of upper respiratory infection prior to the rash. There was no family history of similar skin disorders.

Physical examination showed keratinizing papules all over the patient’s body, diffuse powdery scales on the scalp, palmar and plantar hyperkeratosis, and ectropion of both upper eyelids (Fig. 1a,c,e,g). Serological tests for HIV were negative.

Histopathological examination of a skin biopsy demonstrated alternating horizontal and vertical parakeratosis, which combined with the clinical presentation confirmed the diagnosis of Type III (classic juvenile) PRP.

Treatment with acitretin 15 mg/day (daily dose of 1 mg/kg/day) was started, combined with antihistamines and topical corticosteroids. After 20 days of treatment, the patient still had intense itching of the skin and presented with erythroderma; consequently, his treatment was switched to secukinumab after excluding relevant contraindications and obtaining written consent from the

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