Proceedings Paper:
Katibi, O.S., Andrew, P., Pinnock, A. et al. (2025) P16 Effect of moisturisers on allergen-induced inflammation and skin barrier. In: British Journal of Dermatology. 40th British Society for Paediatric and Adolescent Dermatology Annual Meeting 2025, 07-08 Nov 2025, Nottingham, United Kingdom. ISSN: 0007-0963. EISSN: 1365-2133.

https://doi.org/10.1093/bjd/ljaf465.024

Results

Skin barrier integrity was reduced by all three treatments relative to NTC as indicated by elevated TEWL (OO 30.68 ± 16.08, CO 30.72 ± 15.60 and EC 31.99 ± 16.92 compared to NTC 26.15 ± 13.58 g/m^2/h; $P = 0.0067$). The strongest APT reactions (graded 0–4) were seen following pre-treatment with EC(2.28 ± 1.03) when compared to the NTC (1.70 ± 0.90, $P = 0.0147$), followed by OO (2.00 ± 1.032), then CO (1.93 ± 0.84).

Conclusion

Some moisturisers decrease skin barrier integrity and increase reactions to allergens coming in contact with the skin. Further studies are required to better understand the mechanisms of this effect and the role of moisturiser use in the broader development of atopic diseases.

Abstract citation ID: ljaf465.024

P16 Effect of moisturisers on allergen-induced inflammation and skin barrier

Oludolapo Sherifat Katibi,[1,2] Paul Andrew,[3] Abigail Pinnock,[3] Kirsty Brown,[3] Michael Cork,[1,3] and Simon Danby[3]

[1]Sheffield Children's Hospital, Sheffield, UK; [2]University of Ilorin, Ilorin, Nigeria; and [3]University of Sheffield, Sheffield, UK

Abstract

Introduction

Recent evidence suggests that moisturisers are not beneficial in preventing development of atopic dermatitis (AD) in infants but can increase risk of allergic sensitisation. Given that emollient vehicles can modify the delivery of 'actives' into the skin it is important to examine their effects on epicutaneous sensitization to environmental allergens.

Objectives

To determine the effects of regularly using emollients on cutaneous reactions to house dust mite allergen.

Materials and Methods

Observer-blind study in 40 adults with house dust mite sensitisation. Participants underwent 4 weeks of treatment with olive oil OO, coconut oil CO and a basic emollient cream EC applied on 3 different areas of both forearms with a fourth area left as a no-treatment control (NTC). Skin barrier integrity was assessed with tape-stripping trans-epidermal water loss (TEWL) and atopy patch tests (APT) were performed.

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of patients with PsO achieved **PASI 75 at Week 4**

(vs 1.2% placebo [n=1/86], p<0.0001)*** [2]

76.9%
(N=52)†

of patients with PsO achieved **PASI 100 at 5 years** [3]

51.5%
(n=222/431)

and

50.6%
(n=135/267)

of biologic-naïve and TNFi-IR PsA patients achieved **ACR 50 at Week 104/100,** respectively‡ [1,4–6]

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These data are from different clinical trials and cannot be directly compared.

Co-primary endpoints PASI 90 and IGA 0/1 at Week 16 were met.**Secondary endpoints. †N= mNRI, missing data were imputed with mNRI (patients with missing data following treatment discontinuation due to lack of efficacy or a TRAE were counted as non-responders; multiple imputation methodology was used for other missing data). ‡43.9% (n=189/431), and 43.4% (n=116/267) of biologic-naïve and TNFi-IR PsA patients achieved the primary endpoint of ACR 50 at Week 16 in BE OPTIMAL and BE COMPLETE, respectively (vs 10.0% [n=28/281] and 6.8% [n=9/133] placebo, p<0.0001); 54.5% (n=235/431) and 51.7% (n=138/267) maintained it at Week 52 (NRI).[4–6]

ACR 50, ≥50% response in the American College of Rheumatology criteria; **AS**, ankylosing spondylitis; **CRP**, C-reactive protein; **DMARD**, disease-modifying antirheumatic drug; **HS**, hidradenitis suppurativa; **IGA**, Investigator's Global Assessment; **(m)NRI**, (modified) non-responder imputation; **MRI**, magnetic resonance imaging; **nr-axSpA**, non-radiographic axial spondyloarthritis; **NSAID**, non-steroidal anti-inflammatory drug; **PASI 75/90/100**, ≥75/90/100% improvement from baseline in Psoriasis Area and Severity Index; **PsA**, psoriatic arthritis; **PsD**, psoriatic disease; **PsO**, psoriasis; **TNFi-IR**, tumour necrosis factor-α inhibitor – inadequate responder; **TRAE**, treatment-related adverse event.

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