

Allergy to sunscreen and leave-on facial products is not a likely causative mechanism in frontal fibrosing alopecia: perspective from contact allergy experts

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The pathogenesis of frontal fibrosing alopecia (FFA) is complicated and incompletely understood, and the cause for its increasing incidence over the last 10–15 years is unknown. It has been reported to affect both white and darker-skinned individuals, including in Asia and Africa.¹

Sunscreen has been a popularly hypothesized environmental trigger for FFA, partly because of its relatively recent introduction, which is thought to fit with the reported epidemiology of FFA. Studies detecting an association between increased sunscreen use and FFA comprise retrospective questionnaire studies and case reports.^{2–6} Limitations of these studies include recall bias, control selection bias and temporal ambiguity – with lack of specification in either the questionnaire or study regarding whether the increased sunscreen use preceded the diagnosis of FFA.² Increased sunscreen use among patients with FFA may actually reflect increased protection of exposed skin or higher economic status.⁷

Some have hypothesized that topical products may trigger FFA as endocrine disruptors,⁵ or that sunscreen cessation and the subsequent increase in ultraviolet exposure may improve FFA via anti-inflammatory and immunomodulatory effects.^{3,6} Others have suggested that the mechanism is allergic.²

We think that a causative allergic mechanism is highly unlikely. In allergic contact dermatitis to face cream ingredients, there is often a margin of sparing below the hairline. FFA in some cases also affects the occipital scalp, where sunscreen is not usually applied.¹ Chemical sunscreen allergy is very uncommon. Allergic contact dermatitis from titanium dioxide in sunscreens has not been reported. A study identified titanium dioxide along hair shafts of both patients with FFA and controls, and hence is meaningless.⁸ Lichenoid, photolichenoid or follicular allergic reactions to sunscreens have not been reported.

Studies that have patch tested patients with FFA have consistently compared their results with a general patch test population, rather than patch testing controls.² This is a problem because patients with FFA are on average older and more likely to be female compared with a general patch test population, and may have had more exposures over time, such as to fragrances.

Aldoori *et al.* patch tested a subcohort of women with FFA, and found a higher frequency of positive patch tests not to sunscreens, but to the fragrances hydroperoxides of

linalool (23% vs. 9.8%, $P = 0.016$) and Myroxylon pereirae (13% vs. 3.7%, $P = 0.017$).² They compared these reactions with a patch test centre population tested between 2010 and 2013. It is not clear whether the participants in their study were patch tested in this same time frame, which is important because sensitization rates to different allergens are known to change over time, even within a few years. The relevance of their positive patch tests was not commented on. We would like to highlight that such high sensitization rates are in fact comparable with those in other general patch-tested populations,¹ including in the U.K. (56%) and in our Australian patch testing centre (66.5%) (R. Nixon and A. Palmer, unpublished).

As experienced contact allergy experts (M.T. 24 years; R.L.N. 31 years), we have diagnosed a large number of patients with contact allergy to fragrances, hair dyes, preservatives and, less commonly, sunscreens. FFA has never been recorded as a coexisting diagnosis in those patients.

In summary, we think that there is no conclusive evidence that sunscreens cause FFA by a contact allergic mechanism. We are concerned that implicating sunscreen as a cause of FFA could lead to sunscreen avoidance, especially in white populations with photodamaged skin.¹

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Conflicts of interest: none to declare.