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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 Caucasians and darker skinned individuals, including in Asia and Africa¹.

Sunscreen has been a popularly hypothesised 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 reports²⁻⁶. Limitations 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 FFA². Increased sunscreen use among patients with FFA may actually reflect increased protection of exposed skin or higher economic status⁷.

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Some have hypothesised that topical products may trigger FFA as endocrine disruptors⁵; 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 allergic².

We think that a causative allergic mechanism is highly unlikely. In allergic contact dermatitis (ACD) 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 applied¹.

Chemical sunscreen allergy is very uncommon. Allergic contact dermatitis from titanium dioxide (TiO₂) in sunscreens has not been reported. A study identified TiO₂ along hair shafts of both FFA patients and controls, and hence is meaningless⁸. Lichenoid, photo-lichenoid or follicular allergic reactions to sunscreens have not been reported.

Studies which have patch tested patients with FFA have consistently compared their results to a general patch test population, rather than patch testing controls². 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 (22.5% compared with 9.8%, $p = 0.016$) and *Myroxylon pereirae* (12.5% compared with 3.7%, $p = 0.017$)². They compared these reactions with a patch test centre population tested between 2010-2013. It is not clear whether the subjects in their study were patch tested in this same time frame, which is important, because sensitisation 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 sensitisation rates are in fact comparable to other general patch tested populations¹ including the UK (56%) and in our Australian patch testing centre (66.5%) (unpublished).

As experienced contact allergy experts (MT 24 years, RN 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 Caucasians with photo damaged skin¹.

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