

Correspondence

Reply

To the Editor:

We thank Mohiuddin et al¹ for their comments about the value of our finding that vitamin D insufficiency (VDI) increases the risk of food allergy and that this appears to be independent of the risk of eczema and read with interest their confirmatory findings in their own cohort of eczematous children. We strongly support their conclusions that more work in this field is warranted, in particular that randomized controlled trials on the use of vitamin D for the prevention of food allergy would be timely. Vitamin D supplementation and food chain fortification are already universal in Europe and North America, which poses significant feasibility issues for supplementation trials in these settings. Thus there is an urgent need to evaluate the effect of vitamin D supplementation in countries that are yet to implement routine vitamin D supplementation, food chain fortification, or both (eg, Australia). Our own findings also demonstrate the importance of using challenge-proved food allergy (as opposed to proxy measures) as the primary outcome.²

The authors raise some fascinating points about the biological plausibility of the vitamin D and food allergy association. In particular, they discuss the importance of an altered skin barrier in food allergy development and whether low vitamin D levels might lead to an impaired skin barrier. We certainly agree that skin barrier dysfunction might play a role in the development of food allergy. We have previously reported that the filaggrin gene (*FLG*), a potent mediator of epidermal barrier integrity, appears to play a role in increased risk of food sensitization (although it did not have an effect on food allergy over and above sensitization), and this was independent of the role it plays in the risk of eczema development.³ However, the hypothesis that vitamin D might play a role in food allergy through altering the skin barrier seems to be contrary to our finding that vitamin D was not associated with eczema,⁴ another allergic condition known to be associated with skin barrier dysfunction.

We have now extended our investigation of the HealthNuts cohort to explore whether there was any evidence to support the hypothesis of Mohiuddin et al¹ that the role of vitamin D in food allergy development might be mediated through skin barrier dysfunction. First, we examined whether VDI might be associated with severe eczema because patients with severe eczema are most likely to have an impaired skin barrier.⁵ Among those with eczema, VDI was not associated with the use of topical steroids for more than 10 consecutive days (odds ratio, 1.1; 95% CI, 0.4-3.0; $P = .86$).

Subsequently, we also investigated whether the relationship between vitamin D and food allergy was modified by the presence of *FLG* null mutations and found that this was not the case. VDI was associated with increased risk of food allergy in both those with and without *FLG* null mutations ($P < .05$ for both, P for interaction = .40).

Taken together, these findings suggest that impaired skin barrier function is not essential for VDI to have an effect on food allergy.

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