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Reply

Our answer to the Letter entitled "Superantigens in T cell mediated skin diseases – more than a coincidence!" is rather simple and goes back to criteria established already by Robert Koch for the attribution of a particular disease to a particular microbial pathogen. *Staphylococcus aureus* was not detected in 37% of the study group and therefore this microorganism could not account for atopic dermatitis in these patients (Jappe *et al*, 1998). Furthermore, only 45% of the *S. aureus* isolates from the 63% of the study group carrying this microorganism on their skin had the genotype for the

superantigens analyzed. This would clearly indicate that these superantigens were not involved in the pathology. The design of the investigation does allow the hypothesis of cause and effect, *S. aureus* and atopic dermatitis, to be tested. Assuming that genetic disposition of the individual is essential combined with an extraneous environmental factor, in this case *S. aureus* superantigen producer, then atopic dermatitis would occur only when *S. aureus* was present on the skin. The hypothesis that *S. aureus* skin colonization is a prerequisite for atopic dermatitis is greatly weakened by our data. This investigation strengthens the hypothesis that other environmental factors, apart from *S. aureus*, are important in atopic dermatitis.

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***In Vitro* Determination of Erythema and Immunologic Protection Afforded by Sunscreens do not Accord with *In Vivo* Assessments**

To the Editor:

As members of the cosmetic industry involved in sunscreen research and development, and currently working on photoimmunologic protection afforded by sunscreens, we would like to add our comments to those from Gasparro (1998) and Wolf and Kripke (1998) on Davenport *et al*'s publication (1997), particularly after reading Chu *et al*'s (1998) responses to these comments.

The statement "sunscreens protect against immunosuppression beyond their designated sun protection factor (SPF)" is incorrect. Re-analysis of the data shows exactly the opposite. In addition, we want to state that SPF determined *in vitro* by the Diffey method often do not correlate with the *in vivo* SPF (Diffey and Farr, 1991). For example, based on our extensive experience, sunscreen A (2% octyl-methoxy cinnamate) would be expected to have an *in vivo* SPF around 2 (5.7 found *in vitro*), sunscreen B (2% o-PABA) a SPF of 2.5 (4.5 found *in vitro*), and sunscreen E (6% ZnO) a SPF of 5 (3.8 found *in vitro*). So, the conclusion that cream A, which provided the highest immune protection, is the cream that had the highest *in vitro* SPF is valid only for an *in vitro* situation and this model of evaluation.

We also disagree with one of the other conclusions of this work: "Protection by creams D and E, broad-spectrum sunscreens, is lower than protection afforded by pure UVB sunscreens (creams A and B)." Indeed, the published *in vivo* results issued from our laboratory and from other international teams have demonstrated just the opposite: sunscreens containing both UVA and UVB filters are more effective against photoimmunosuppression than pure

UVB formulations (Bestak *et al*. 1995; Damian *et al*. 1997, Serre *et al*. 1997, Gueniche and Fourtanier, 1997; Moyal, 1998). We have also demonstrated that the higher the UVA protection level, the better the immune system is protected, and that sunscreen protection factor against immunosuppression are lower than their *in vivo* SPF.

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