

## Epidermal Calmodulin Levels in Psoriasis

To the Editor:

Recently, we measured the calmodulin level in lesional psoriatic skin vs normal epidermis [1]. The protein content of epidermal homogenates was estimated by the method of Lowry et al [2]. The values were as follows: (1) Using 0.1 mM CaCl<sub>2</sub> containing 50 mM Tris-HCl buffer: in normal skin the calmodulin activity was  $1.05 \pm 0.31 \mu\text{g calmodulin mg}^{-1}$  epidermal protein (mean  $\pm$  SEM,  $n = 9$  volunteers), in psoriatic lesions  $2.28 \pm 0.69$  ( $n = 7$  patients); (2) using 0.5 mM EGTA containing 50 mM Tris-HCl buffer:  $1.3 \pm 0.30$  and  $2.28 \pm 1.08$ , respectively.

This underlines the findings of Mac Neil and Tucker [3] indicating that the Lowry protein assay is recommended. Furthermore, changing the calcium content of the buffer used does not result in changing the calmodulin activity significantly.

Biologically active calmodulin in the psoriatic plaque is elevated 2-fold. The striking difference to earlier reports by Tucker et al [4] and van de Kerkhof and van Erp [5] is caused by methodologic discrepancies.

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### REPLY

We are pleased that Wollina, Klinger, and Wetzker confirm a 2-fold elevation in epidermal calmodulin activity in patients with psoriasis.

This is now the fourth independent group (from as many countries) to report an increase in epidermal calmodulin levels in psoriasis of the order of 2- to 3-fold: Mac Neil et al (36 patients) [1], Fairley et al (9 patients) [2], and Mizumato et al (8 patients) [3].

Similar values are obtained whether calmodulin levels are determined by radioimmunoassay or by activity assay for calmodulin. (Our earlier preliminary report of a 6-fold elevation in calmodulin we now know to be an overestimate based on an inappropriate choice of protein assay for the skin samples [4]. Also the original observation of a 30-fold elevation in epidermal calmodulin [5] has not been confirmed to our knowledge.)

In the face of such good agreement among the different research groups, it would now seem to be time to investigate the relevance of this finding to the etiology of psoriasis. Our data so far show a decrease in epidermal calmodulin following successful treatment for psoriasis [6] and that one of the common treatments for psoriasis, dithranol, possesses calmodulin antagonist activity [7]. With more groups now studying calmodulin in psoriasis we hope that the investigation of the possible role of this protein in psoriasis proceeds rapidly over the next few years.

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