## The Number of Mast Cells Is Highly Increased in Non-involved Skin in Psoriasis

Sir,

The presence of mast cells in non-involved and involved psoriatic skin was compared with that in skin from healthy, nonpsoriatic persons.

Punch biopsies (3 mm) were taken from lesional (n = 11) and non-involved skin (n = 12) in patients with psoriasis. One patient was treated with etretinate 40 mg/day but the others had only topical treatment with emollients. From 2 of the patients biopsies were taken only from non-involved skin, as they had only scalp psoriasis, not considered suitable for biopsies. For comparison biopsies were taken from normal skin of 5 healthy persons and also from 3 positive prick tests to Dermatophagoides pter onyssinus 15 min as well as 5 h after the test. Acetone-fixed, 6 μm thick cryostat sections were stained with the use of a 3-step monoclonal antibody peroxidase antiperoxidase (PAP) technique. Endogenous peroxidase was blocked by incubation for 15 min in 0.3% H<sub>2</sub>O<sub>2</sub> in phosphate-buffered saline (PBS). The sections were then allowed to react with normal rabbit serum (diluted 1/10) for 10 min to reduce non-specific staining. They were then incubated with the primary mouse anti-human mast cell tryptase monoclonal antibody, dilution 1/5,000 (MAB 1222, Chemicon, Temecula, CA, USA), which binds to mast cell tryptase. Rabbit anti-mouse IgG (diluted 1/40; Dakopatts) was used as a secondary antibody. Finally the sections were incubated in a third step with horseradish peroxidase-mouse monoclonal anti-horseradish peroxidase (diluted 1/250; Dakopatts). The peroxidase reaction was developed with 3-amino-9ethylcarbazole. The sections were counterstained with Mayer's haematoxylin. Controls without the primary antibodies gave no

Table I. Tryptase-positive mast cells, number/section (mean  $\pm$  SD)

Healthy subjects $(n=5)$	6.8± 1.6
Mite-positive prick test 15 min $(n=3)$	5.3± 4.0
Mite-positive prick test 6 h $(n=3)$	18.7± 4.5
Non-involved skin, psoriasis $(n = 11)$	41.2±20.9
Psoriatic lesions $(n = 12)$	74.5±24.6

staining. Tryptase-positive cells/section were counted in the upper part of the dermis (corresponding to 5 units depth on a grid scale, Zeiss Axiomat  $(10 \times 10)$ .

All psoriasis lesions contained large numbers of tryptase-positive cells, particularly in the papillary dermis. The number of positive cells in non-involved skin was, however, also highly increased, when compared with the number observed in the biopsies from healthy persons and in biopsies from mite-positive prick tests. In some biopsies from psoriasis patients with many positive cells – sometimes present in clusters – the exact number of cells was not possible to establish and the figures given are approximations. In some of the biopsies from psoriasis patients positively stained areas were also observed without a clear relation to a defined mast cell, which may indicate a previous release of tryptase into the dermis. This was observed particularly in biopsies with large numbers of mast cells.

A marked increase of mast cells in psoriasis lesions has been reported in several papers by Naukkarinen et al. (1 for ref.). The main purpose of their studies was to characterize the distribution and the interrelationship between tryptase- and chymase-positive mast cells in the lesions as well as the contact between sensory nerves and mast cells, with particular reference to the pattern of neuropeptides.

We have, however, not found any data indicating an elevated number of tryptase-positive cells in non-involved psoriatic skin, in comparison with that of healthy skin. Larger groups of patients should be studied to see if the pronounced mast cell increase is a regular finding in psoriasis. If so, it might be of importance in the pathogenesis of psoriasis.

## REFERENCE

 Naukkarinen A, Harvima IT, Aalto ML, Horsmanheimo M. Mast cell tryptase and chymase are potential regulators of neurogenic inflammation in psoriatic skin. Int J Dermatol 1994; 33: 361–366.

Accepted November 11, 1994.

Gerd Michaëlsson, Eva Hagforsen, Inger Pihl Lundin, Department of Dermatology, University Hospital, S-75185 Uppsala, Sweden.