

³H-THYMIDINE LABELLING OF EPIDERMIS AND DERMAL INFILTRATE IN PSORIATIC ERYTHRODERMA

M. de la Brassinne and J. M. Lachapelle

*From the Department of Dermatology, Hôpital de Bavière, Liège University, Liège,
and the Unit for Occupational Dermatoses, Louvain University, Brussels, Belgium*

Abstract. The comparative pattern of ³H-thymidine (³H-T) pulse labelling in lesions of psoriatic erythroderma (3 cases) and of psoriasis vulgaris (20 cases) has been studied by means of radioautography. In the epidermis, no difference was noted between the two types of lesions concerning the epidermal ³H-T labelling index (L. I.) and the localization of labelled cells. The ³H-T L. I. of "round" cells infiltrating the dermis was much higher in lesions of psoriatic erythroderma ($4.03 \pm 0.56\%$) than in lesions of psoriasis vulgaris ($0.62 \pm 0.21\%$), and the difference is highly significant ($p < 0.0001$). The findings indicate that most of these labelled cells could well be lymphoblasts, suggesting that an immunological process is possibly involved in the pathogenesis of psoriatic erythroderma.

Key words: Autoradiography; Psoriasis; Erythroderma; Epidermis; Dermal infiltrate

Psoriatic erythroderma is usually considered as an eruption that appears suddenly and is usually linked with severe general symptoms. This "unstable" form of erythroderma (15) is widespread and does not spare any area of normal skin. The clinical features of psoriasis are often lost (15).

In many cases, the histological picture cannot be distinguished from that of other types of exfoliative dermatitis (11). The epidermis may still show enough of the characteristics of psoriasis to admit of such a diagnosis, though spongiosis and exocytosis are often found, as in eczema (10). Dermal vessels are more dilated and tortuous than in psoriasis vulgaris; they are surrounded by a more dense infiltration, mainly of mononuclear "round" cells (15).

By using the tritiated thymidine (³H-T) incorporation technique, it has been demonstrated that, in psoriasis vulgaris, the epidermis displays an accelerated turnover (5) since the labelling index is increased (3, 4, 13) and the transit time of cells through

the epidermis is shortened (5, 14). Very few "round" cells of the dermal infiltrate are ³H-T labelled (7).

In this study, our goal was to describe the pattern of ³H-T pulse labelling of the epidermal cells of and "round" cells infiltrating the dermis in psoriatic erythroderma. The results will be compared with those obtained in psoriasis vulgaris.

MATERIAL AND METHODS

This study was made in three adult male patients with untreated psoriatic erythroderma. General symptoms, such as fever or joint pain, were present in each patient; two of them suffered from a severe cardiac failure. In each case, a biopsy was performed on the trunk. The classical features of psoriasis were evident in all the histological sections. Skin specimens obtained from the trunk of 20 patients with untreated psoriasis vulgaris were used as controls.

The skin samples were cut into thin slices (< 1 mm) which were incubated for one hour at 37°C in a culture medium containing 2 μ Ci of 6-³H-thymidine (specific activity: 10 Ci/mM; CEN, Mol, Belgium) according to the technique of Lachapelle & Gillman (9). After washing, the specimens were fixed in 4% buffered formalin, paraffin embedded, cut at 5 μ m and processed for light microscopy radioautography. The radioautographs were stained with haematoxylin-eosin or PAS-haemalum.

In order to define the type of labelled cells infiltrating the dermis, an intradermal injection of 5 μ Ci of ³H-T was performed in one of the three erythrodermic patients. A 4 mm punch biopsy of the injected site was obtained 1 hour later. Imprints were prepared according to Degos & Ossipovsky (2). They were treated histochemically for Naphthol AS-D acetate esterase(s) identification (1), fixed in 4% buffered formalin for 10 min, radioautographed, and stained with haematoxylin.

The epidermal labelling index was obtained by counting 2 000 interannexial basal cells in each slide. The labelling index of "round" cells was determined as previously described (6, 7, 8). Data are expressed as mean $\pm$ 1 standard error of the mean. Statistical significance is analysed by the Student's *t*-test for paired data.

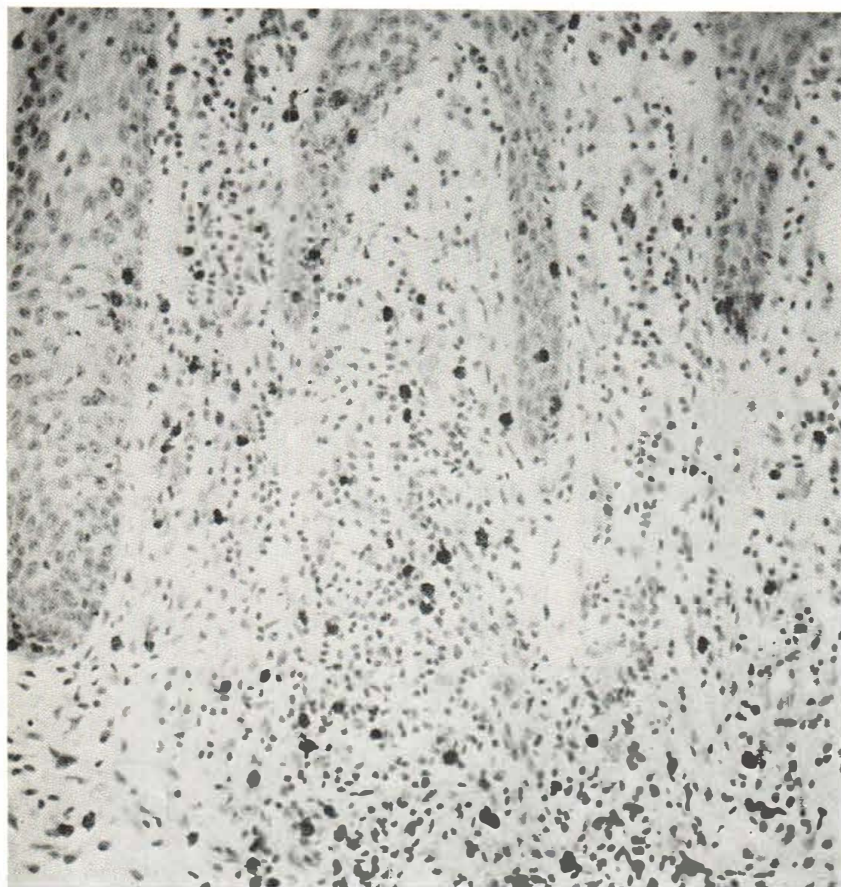


Fig. 1. In vitro $^3\text{H-T}$ labelling of psoriatic erythroderma. Many "round" cells of the dermal infiltrate are labelled. (Radioautograph; H. and E.; $\times 220$.)

RESULTS

Epidermis

Numerous labelled cells are observed in the deep layers of the epidermis, both in erythroderma (Fig. 1) and in psoriasis vulgaris (Fig. 2). No difference can be found between both diseases concerning the topography of labelling. The mean basal L. I. of erythroderma ($8.52 \pm 3.85\%$) is not significantly different ($p > 0.10$) from that of psoriasis vulgaris ($13.3 \pm 7.8\%$).

Dermal infiltrate

In both conditions, the dermal infiltrate consists predominantly of mononuclear cells, but it is heavier in psoriatic erythroderma. Many mononuclear "round" cells of the infiltrate are labelled (Fig. 1) in psoriatic erythroderma. As shown in Fig. 3, the $^3\text{H-T}$ L. I. ($4.03 \pm 0.56\%$), is significantly different ($p < 0.0001$) from that of psoriasis vulgaris ($0.62 \pm 0.21\%$). Radioautographs of imprints treated

histochemically for Naphthol-AS-D acetate esterase(s) identification in one of the three cases of psoriatic erythroderma show that all the $^3\text{H-T}$ labelled "round" cells contain very few, if any, cytoplasmic (specific for esterase) granules. Round cells, which are strongly positive for Naphthol-AS-D acetate esterase(s) are not labelled.

Since this technique allows the distinction between cells of lymphoid origin (negative) and histiocytes (positive), the ^3HT -labelled cells are probably lymphoblasts.

DISCUSSION

It is generally accepted that the "unstable" form of psoriatic erythroderma has to be distinguished from psoriasis universalis (15). The first condition is a widespread rash that appears suddenly and is associated with general symptoms, whilst the latter is a slow and progressive generalization of psoriatic lesions that become confluent (15). According to

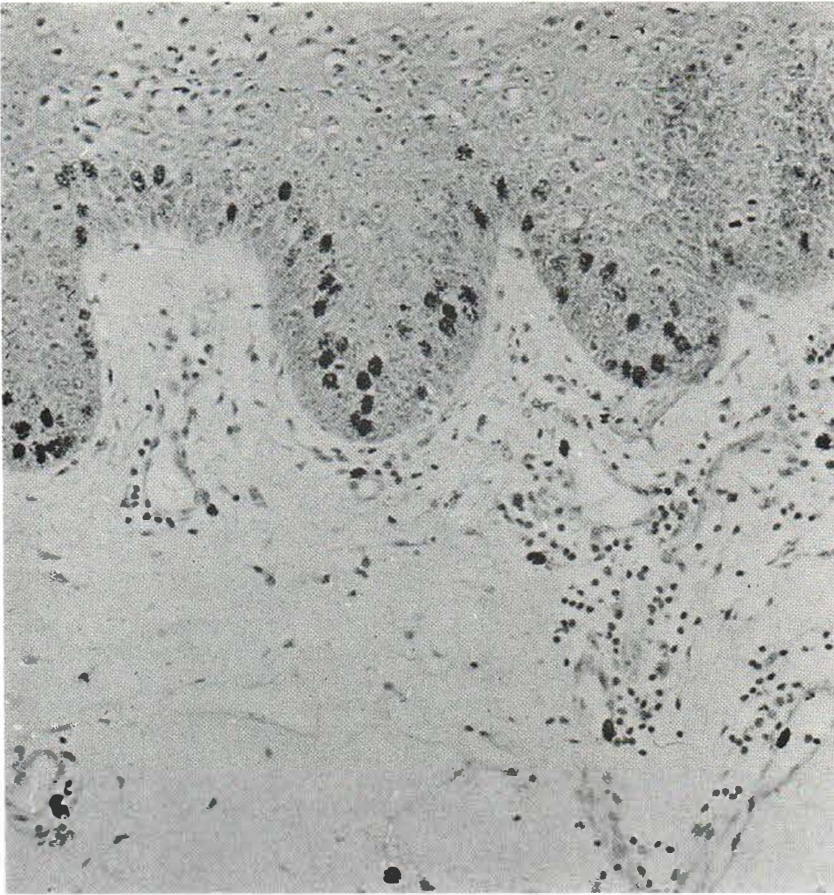


Fig. 2. In vitro ^3H -T labelling of a lesion of psoriasis vulgaris. Very few "round" cells of the dermal infiltrate are labelled. The labelling of many endothelial cells signifies an important angiogenesis. (Radioautograph; H. and E.; $\times 220$.)

these distinctive criteria, our 3 patients can be considered as suffering from "true" psoriatic erythroderma.

It has to be stressed that no statistical difference in the basal epidermal L. I. exists between lesions of psoriatic erythroderma and of psoriasis vulgaris. In both conditions the proliferative activity is increased. Wide variations appear between individual cases of psoriatic erythroderma, as previously observed for lesions of psoriasis vulgaris in different patients (3).

In psoriatic erythroderma, many endothelial cells of blood capillaries are ^3H -T-labelled. A similar observation had been made in psoriasis vulgaris (7). This angiogenesis, encountered in different types of psoriatic lesions, had already been demonstrated by other methods (12).

The ^3H -T L. I. of "round" mononuclear cells infiltrating the dermis in psoriatic erythroderma, is high, and similar to that obtained in lesions of allergic contact dermatitis (6) or lichen planus (8). On

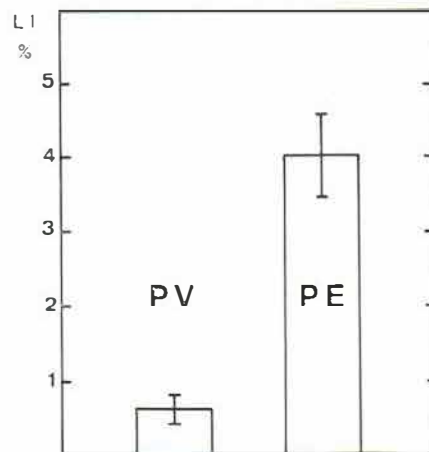


Fig. 3. ^3H -T labelling index (L. I.) of "round" cells infiltrating the dermis in psoriasis vulgaris (PV) and in psoriatic erythroderma (PE). Mean $\pm$ S.E.

the other hand, the ^3H -T L. I. is very low in psoriasis vulgaris (7), and identical with that found in primary irritant dermatitis (6).

By combining cytochemical and radioautographic techniques on the same imprints in psoriatic erythroderma, it has been found that the "round" cells which are proliferating in the infiltrate are probably lymphoblasts. Indeed, ^3H -T-labelled cells are negative (or very weakly positive) for Naphthol AS-D acetate esterase(s), as observed in lichen planus (8). The presence of the lymphoblasts still remains unexplained, but it suggests that an immunological process could be involved in the pathogenesis or in the course of psoriatic erythroderma. In this respect, psoriatic erythroderma could be considered as a generalized Köbner phenomenon consecutive to an immunological event developed in patients who are coded for psoriasis.

ACKNOWLEDGEMENTS

We are very grateful to Miss F. D. M. de Cock and Mr N. Vrancken for technical assistance.

REFERENCES

1. Burstone, M. S.: Esterase activity of developing bones and teeth. *Arch Pathol* 63: 164, 1957.
2. Degos, R. & Ossipowski, B.: Le dermogramme. *Dermatologica (Basel)* 115: 482, 1957.
3. Brassinne, de la. M. & Pierard, G.: Interprétation pathogénique de l'extension des placards de psoriasis. *Dermatologica (Basel)* 147: 161, 1973.
4. Goodwin, P., Hamilton, S. & Fry, L.: Comparison between DNA synthesis and mitosis in involved and uninvolved psoriatic epidermis and normal epidermis. *Br J Dermatol* 89: 613, 1973.
5. — The cell cycle in psoriasis. *Br J Dermatol* 90: 517, 1974.
6. Lachapelle, J. M.: Comparative study of ^3H -thymidine labelling of the dermal infiltrate of skin allergic and irritant patch test reactions in man. *Br J Dermatol* 87: 460, 1972.
7. Lachapelle, J. M. & de la Brassinne, M.: Activité proliférative de l'infiltrat cellulaire dans le psoriasis. *Arch Belg Derm Syph* 28: 231, 1972.
8. — The proliferation of cells in the dermal infiltrate of lichen planus lesions. *Br J Dermatol* 89: 137, 1973.
9. Lachapelle, J. M. & Gillman, T.: Tritiated thymidine labelling of normal human epidermal cell nuclei. A comparison, in the same subjects, of *in vivo* and *in vitro* techniques. *Br J Dermatol* 81: 603, 1969.
10. Lever, W. F.: Histopathology of the skin, 653 pp. J. B. Lippincott Co, Philadelphia, 1961.
11. Nicolis, G. D. & Helwig, E. B.: Exfoliative dermatitis. A clinicopathologic study of 135 cases. *Arch Dermatol* 108: 788, 1973.
12. Telner, P. & Fekete, E.: Capillary response in psoriatic skin. *J Invest Dermatol* 36: 225, 1961.
13. Weinstein, G. D. & Frost, P.: Abnormal cell proliferation in psoriasis. *J Invest Dermatol* 50: 254, 1968.
14. Weinstein, G. D. & Van Scott, E. J.: Autoradiographic analysis of turnover times of normal and psoriasis epidermis. *J Invest Dermatol* 45: 257, 1965.
15. Wilkinson, D. S.: Psoriasis. In Rook, A., Wilkinson, D. S. & Ebling, F. J. G.: Textbook of Dermatology. Blackwell Scientific Publications, Oxford and Edinburgh, 1972.

Received September 9, 1974

M. de la Brassinne, M.D.
Department of Dermatology
Hôpital de Bavière
B-4000 Liège
Belgium