

# Additional ESDR Abstracts

The following are additional abstracts of presentations of the 24th Annual Meeting of the European Society for Dermatological Research in Vienna, September 24-27, 1994. Because of a clerical error, these abstracts were not submitted with the abstracts published in the September issue of the *Journal*.

## QUANTITATION OF A MAJOR HISTOCOMPATIBILITY COMPLEX CLASS I MOLECULE AFTER 8-METHOXYPORALEN AND UVA PHOTOINDUCTION.

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The response of cutaneous T cell lymphoma to extracorporeal phototherapy with 8-methoxypsoralen (8-MOP) and UVA may be due to the photoinduction of new antigenic determinants on the surface of malignant cells with a subsequent elicitation of an immune response by the patient after reinfusion. To determine whether photoactivated 8-MOP enhances the cell surface expression of MHC class I molecules, the murine T cell lymphoma cell line RMA was treated with 300 ng/ml 8-MOP and 1 J/cm<sup>2</sup> UVA. After the treatment, cells were cultured at 26°C for 24 hours and subsequently divided into two groups: to one group was added the MHC class I H-2K<sup>b</sup>-specific peptide FAPGNYPAL, the other group was incubated in the absence of peptide. After exposure to 37°C for 3 hours to denature MHC class I molecules without peptide, RMA cells were immunostained with the monoclonal antibody Y-3. Calibration beads with a known number of antibody binding sites were used for the cytofluorometric analysis to quantitate the number of photoinduced cell surface MHC class I H-2K<sup>b</sup> molecules. At 24 hours, an increase of 81% in H-2K<sup>b</sup> molecules in the treated cell population has been observed in comparison with 41% from the untreated control group corresponding to a total increase of  $1.8 \times 10^5$  H-2K<sup>b</sup> molecules. Therefore, the enhancement of antigenicity as a result of the increased MHC class I antigen presentation may be responsible for the immune augmenting effects observed in patients after photopheresis.

IMMUNOLABELLING STUDIES FOR KERATINS IN NORMAL HUMAN PALM AND SOLE SKIN: ANOTHER PREREQUISITE TO STUDYING PALMOPANTAR KERATODERMA. Ole Swensson, J.R. McMillan,\*L.J. Churchill,\*T. Egelrud,\*I.M. Leigh, and R.A.J. Eady. Dept. of Cell Pathology, St. John's Institute of Dermatology, St. Thomas's Hospital; \*Experimental Dermatology Lab., The Royal London Hospital, London, U.K.; ^Dept. of Dermatology, Umea University, Umea, Sweden.

Knowledge of the keratin (K) expression in palm and sole skin should prove useful for studying palmoplantar keratoderma which we now know may be caused by mutations in K genes, including the site-specific K9. To investigate the pattern of K expression in normal human palm and sole epidermis we performed immunolabelling studies using indirect immunohistochemistry.

A panel of monoclonal antibodies (Abs) directed against K5 [RaK5], K14 [LL001] K1 [LL017], K10 [LH2], K9 [FE1], K6 [KA12], K16 [LL025], K17 [E3], and K19 [LP2K] were used on snap frozen biopsy material from clinically normal palm and sole skin of seven subjects (3 female, 4 male; aged 6 mo. to 62 yrs.).

Basal keratinocytes showed immunoreactivity with Abs against K5 and K14. In addition, small groups of one to four basal cells were immunoreactive to anti-K17. Suprabasally, K1, K10, and K9 labelling was regularly seen. K6 and K16 staining was present especially in suprabasal cells of epidermal ridges but was less pronounced in cells above dermal papillae. K19 expression was observed in eccrine glands, but not in interappendageal epidermis.

Our data indicate a complex pattern of keratin expression with at least 5 different K-subtypes being expressed suprabasally in palm and sole epidermis. The expression of K6 and K16 might indicate a physiologically increased proliferative activity of normal ridged epidermis compared with other sites. The nature of anti-K17 positive cells and their relation to putative epidermal stem cells have to be established.

## MODULATION OF KERATINOCYTE ACTIVATION PROCESS BY PLASMIN. Imre Szabo, Miklos Simon and Janos Hunyadi, Departments of Dermatology, Medical School of Debrecen, Hungary and Erlangen, Germany.

Keratinocytes have been found to exert plasminogen activator capacity resulting plasmin formation and initiation of fibrinolysis in vitro. The role of plasmin in wound healing, however, is not well understood. In this study we attempted to analyze plasmin effect on various keratinocyte activities using freshly isolated keratinocytes from healthy human skin and HaCaT cells as well. 0.025 U/ml plasmin induced 156 % rise in keratinocytes chemotactic migration which was completely blocked by the plasmin inhibitor epsilon-amino-caproic acid at the concentration of 400 mg/ml. On the other hand, spontaneous proliferation of HaCaT keratinocytes incubated for 24 or 48 h in the presence of 0.025 U/ml plasmin was reduced by 21% and 13.5%, respectively as determined by 3H-Thymidine uptake. Adherence of keratinocytes was not affected by plasmin treatment. These results suggest, that plasmin might regulate wound healing process by increasing motility and decreasing proliferation rate of keratinocytes.

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