LANGERHANS CELLS AND EPIDERMAL MICROENVIRONMENT

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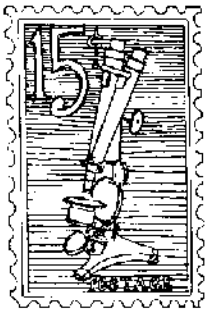
Letters to the Editor



Langerhans Cells and Epidermal Microenvironment

To the Editor:

We read with great interest the article by Dr. Azizi and co-workers (1) on Langerhans cells (LCs) in basal cell carcinomas (BCCs). In particular, we appreciated the careful usage of fine quantitative and reproducible methods in evaluating number, distribution, and morphology of LCs in BCCs.

Concerning the conclusions, however, some critical points require further discussion. In fact, in the three cases in which both BCC and perilesional sun-exposed skin were examined, no difference was found in the number of LCs/mm^2, while a highly significant difference was found in the dendricity index. Based upon these findings, the authors conclude that the morphologic and—probably—functional perturbation of LCs observed within BCCs may contribute to the growth of this epithelial tumor; alternatively, they suggest the possibility that the growth of tumor cells produces local perturbation in the morphology of the LCs. We have now to consider the following points: (a) A normally differentiated epidermis (2,3) as well as an epidermoid metaplastic epithelium (4) provide a suitable microenvironment for the differentiation of LCs. (b) Pathologic alterations of the epidermal differentiation, either inherited (ichthyosis) or subsequent to viral infection (warts), are associated with a perturbation in LC morphology (5–7). (c) The density and morphology of LCs in epithelial skin tumors have been reported to be related to the degree of epithelial differentiation (8,9), but not to the intraepithelial T-cell density and the amount of peritumoral infiltrate (8). In particular, the smallest number and the most perturbed morphology of LCs were found in BCCs; through morphometric methods, a significant inverse correlation was demonstrated between mean nuclear area of the epithelial tissue and LC density (8). (d) Immunohistologic and ultrastructural studies performed in different pathology models revealed that LCs acquire their complete and—probably—functional characteristics only following their permanence in the epidermal microenvironment (10,11).

In conclusion, while a correlation between number and differentiation of LCs and degree of differentiation of the epithelial microenvironment seems at present reasonably acceptable, it is not possible to define any relationship between perturbation of LC morphology and distribution and creation of epithelial skin cancers, even from a merely speculative point of view. Further studies are clearly needed to elucidate this problem.

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To the Editor:

We appreciate the comments of Drs. Pimpinelli and Bani and agree with them completely.

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