To the editor,

We would like to report on the synaptophysin-like immunoreactivity of Merkel cells in fetal rat skin at day 20 of gestation, as detected by double immunostaining [1]. Unfixed, frozen sections obtained from the barbate portion of fetal rats were first incubated with an MC-specific antikeratin antibody (RCK-102, Bio-Science Products AG), confirmed by immunoelectron microscopy [unpubl. data], and thereafter immunostained using an immunogold-silver staining technique. Washed in PBS, the sections were then incubated with a monoclonal antibody against synaptophysin (Boehringer, Mannheim). Using the ABC immunostaining technique, the final reaction product was developed with DAB.

For negative controls the antisynaptophysin antibody was replaced by PBS. Single cells, scattered in the basal layer of the epidermis, as well as cells clustered in the outer root sheath of hair follicles, stained positively with the Merkel cell-specific antikeratin antibody applied in the first step. Incubation with the antisynaptophysin antibody revealed a positive staining reaction within the cytoplasm of these cells (fig. 1). Negative controls were negative.

We thus could extend the findings of Ortonne et al. [2], who demonstrated a synaptophysin-like immunoreactivity of adult Merkel cells in human, mammalian, and rodent skin.
known to produce photosensitivity, it is possible that similar mechanisms may be operative in the
case presented.
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References
immunohistochemical technique using immunogold-silver staining and the avidin-biotin-

Photosensitivity due to Alprazolam
Sir,
Alprazolam is one of the newer benzodiazepines now being recommended for the treatment of
anxiety disorders [1,2]. It is more effective and has lesser side effects as compared to the older
benzodiazepines such as diazepam and chlordiazepoxides [3,4]. Although rare, the latter are
known to produce photosensitivity and other skin rashes [4, 5]. However, cutaneous reactions
due to alprazolam have so far not been observed. The present communication describes a case of
photosensitivity due to alprazolam.

Case: A 41-year-old farmer presented with itching, redness and scaling over the face of 2 days’
duration. The patient had taken a single dose of 1 mg alprazolam a day prior to the onset of the
eruption. Past history revealed a similar episode a month back following ingestion of the same
drug.

Examination revealed bright red erythema and scaling over the forehead, cheeks and chin. There
was no oozing or crusting. Other areas of the body were normal. The patient was advised
photoprotec-tion and topical corticosteroids with which the rash subsided over a period of 3-4
days. After the lesions had subsided, a challenging dose of 0.5 mg alprazolam was given. The
rash recurred at the same sites within 12 h.

Comments: The positive provocation test confirms that the eruption was caused by alprazolam.
This drug has been introduced only recently as one of the most promising and effective drugs for
the treatment of anxiety disorders. The reported side effects pertain to central nervous system,
gastrointestinal tract and autonomic nervous systems [1, 4]. To the best of our knowledge , there
has been no earlier report of any cutaneous side effects due to this drug. As the basic chemical
structure of alprazolam resembles the other benzodiazepines which are

References
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Sir,

We read with great interest the paper by Gandolfo et al. [1] published in your journal. We fully agree with the authors that their findings of normal erythrocytic urodecarboxylase (UD) activity in patients with familial porphyria cutanea tarda (PCT) and the recently published analogous cases [2] contradict the currently widely accepted view, which strictly separates the sporadic form of PCT from the familial form on the basis of the UD deficiency [4]. Held et al. [3] pointed out this contradiction too on the basis of their finding that in 5 out of 32 PCT patients without a positive family history of PCT the UD activity was approximately half the normal level. Further, symptom-free persons with a significantly decreased UD activity are also known. These cases lead us to another conception which predicts polygenic heredity for the disease and supposes that the sporadic and familial forms of PCT are the two end-poles of one and the same disease, which differ from each other only with regard to the severity of the genetic defect [5].

Polygenic heredity is characterized by genetic defects which, together with environmental factors, are responsible for the disease. The genetic background involves several genes and the alteration of a certain number of this set of genes (limit point of genetic determination) results in the genetic determination of the disease. The main characteristics of polygenic heredity were described by Carter [6].

In our investigations of the role of genetic and hepatotoxic factors in the development of PCT in 51 patients we have found that the rules of polygenic heredity fit this disease well [5]. The present findings