ABSTRACT

The Cytokine Pattern in Psoriasis

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The pathomechanisms of various inflammatory disorders are revealed by distinct cytokine patterns. To elucidate the pathogenesis of psoriasis vulgaris (PV) we therefore have compared the profiles of cytokines expressed in affected skin with those of lesional psoriatic T cell lines (TCL) and clones (TCC) which had been activated via CD2 (2–3)/CD28 in the absence of monocytes, mRNA specific for TNF-α/β, TGF-α/β, IL-2/3/4/5/6/8, and GM-CSF, was determined by PCR and specific hybridisation. The PV-biopsies tested ($n = 4$) showed a combined transcription of IL-2/3/5/6/8, TNF-α/β, TGF-β and GM-CSF, but not of TGF-α, with a very faint signal for IL-4 in 2 samples. A similar cytokine pattern was found in 2 TCC, the supernatants of which enhanced keratinocyte proliferation in vitro, while 2 TCC suppressing keratinocyte growth as well 6 TCL showed also a strong signal for either or both IL-4 and TGF-α. No cytokine mRNA could be amplified from unaffected skin ($n = 2$). Collectively, psoriatic skin lesions express a complex cytokine profile uncharacteristic of a TH1- or TH2-response. This psoriatic pattern can fully be accomplished by psoriatic T lymphocytes with mitogenic effect on keratinocytes. The role of the psoriasis-associated cytokines for the clinical features of PV now needs to be determined. TGF-α as a cytokine mitogenic for keratinocytes, however, is appatently not involved in the increased epidermal turnover of psoriasis.

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