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Effects of phototherapy on the production of cytokines by peripheral blood mononuclear cells and on systemic antibody responses in patients with psoriasis

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Résumé / Abstract

Exposure to ultraviolet B (UVB) radiation results in the suppression of many cell-mediated immune responses, and recent studies using mice and murine cells in vitro suggest a shift from a T-helper 1 (Th1) to a Th2 type of response on irradiation. Active psoriasis is considered to be a Th1-type disorder, chiefly on the basis of the cytokines produced by inflammatory cells in psoriatic lesions. We investigated the effect of phototherapy in patients with psoriasis on the cytokine profile of mitogen-stimulated mononuclear cells from peripheral blood and the concentration of IgG subclasses and IgE in the plasma. Eight patients were irradiated with a broad-band UV source (Sylvania UV6; 280-400 nm) three times a week and another eight with a narrow-band UVB source (Philips TL-01; 311-313 nm). Peripheral blood was collected before therapy started and after 14 weeks of therapy. Peripheral blood mononuclear cells were stimulated in vitro with phytohemagglutinin; proliferation was measured by incorporation of tritiated thymidine and culture supernatants assayed for interleukin (IL)-2, -4 and -10 and γ -interferon (IFN) by enzyme-linked immunosorbent assays. Lymphoproliferation was not consistently affected by 4 weeks of UV6 therapy, and there was also no consistent change in the production of IL-2, IL-10 or γ -IFN. In contrast, 4 weeks of TL-01 therapy significantly suppressed lymphoproliferative responses. In addition the production of IL-2, IL-10 and γ -IFN was lowered after 1 week of TL-01 therapy, and this was even more apparent after the treatment had been extended to 4 weeks. IL-4 concentrations were below detectable levels in all the samples throughout the study. The amounts of IgG1, -2, -3 and -4 and IgE in the plasma of the patients did not vary with either of the two phototherapies. Thus, although no evidence was obtained to indicate that UV6 exposure affected T-helper subsets in psoriasis, TL-01 inhibited the activity of both Th1 and Th2 subsets while not altering plasma antibody concentrations.

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