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Drug induced modulation of T cell activation and differentiation in atopic dermatitis patients

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Atopic dermatitis (AD) is a T cell dependent chronic relapsing inflammatory skin disorder. Cyclosporine A (CsA) has been shown to be an effective treatment for severe AD. We studied the effect of low-dose CsA therapy on T cell activity and T cell differentiation in AD patients. Using a whole blood assay we demonstrated that TcR signalling in peripheral blood T cells of CsA treated AD patients is reduced to 42% ± 18 but not totally blocked. Such partial inhibition of TcR signalling allowed regulatory T cell induction under in vitro conditions. Therefore we asked is there an *in vivo* regulatory T cell induction, too. Indeed AD patients under low-dose CsA therapy have higher numbers and frequencies of functional regulatory T cells compared to untreated AD patients. To evaluate the causal connection between low-dose CsA treatment and higher regulatory T cell numbers we studied individual patients before and during CsA therapy. The data clearly indicate increased numbers and frequencies of regulatory T cells after onset of CsA therapy which remained stable during the therapy. The therapeutic effect of low-dose CsA therapy in AD patients could be due to both, inhibition of T cell hyperactivity and induction of suppressor T cells.