INTRODUCTION
AND AIM OF THE WORK

Beta-endorphin (B-END) is one of the most important mediators of stress (Olsen et al., 1986) and is known to be generated up on stimulation of pituitary-adrenal axis (Morley, J.E., 1981).

B-END is a 31-residue amino-acid peptide which belongs to the endogenous opiate family (Wallengren et al., 1987).

Farber et al. (1986) suggested that this neuropeptide is of pathogenic importance in psoriasis and atopic dermatitis and serves as a neuro-modulatory factor underlying exacerbation of psoriasis and/or persistence of psoriatic lesions.

Dermal sensory nerve C fibres have been shown to be much more numerous in psoriatic lesional skin and resolution of persistent psoriatic lesions as a result of surgical damage to cutaneous sensory nerves has been reported. It has been suggested that exacerbation of psoriasis after stress might be related to the release of neuro-peptides and that the bilateral distribution of cutaneous peripheral nerves might account for the symmetry of psoriatic lesions (Naukkarinen et al., 1989).

B-END level may increase in other inflammatory skin diseases in which there is extensive T-cell infiltration such as in atopic dermatitis (Dewing, 1971).
Aim of the work:

To study the concentration of B-END in sera of patients with psoriasis, atopic dermatitis and healthy subjects.