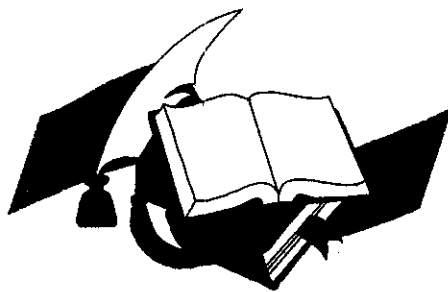


INTRODUCTION AND AIM OF THE WORK



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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.