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BETA-ADRENERGIC BLOCKADE IN ATOPIC DERMATITIS. EVIDENCE OF AN ABNORMALITY OF T-LYMPHOCYTE BETA-RECEPTORS

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β-adrenergic blockade in atopic dermatitis (AD): evidence of an abnormality of T-lymphocyte β-receptors.

During the last few years many data have been accumulated supporting the Szentivanyi theory of β-adrenergic blockade in atopic diseases. In particular it has been demonstrated that the responsiveness to isoproterenol of keratinocytes and leukocytes (evaluated as inhibition of DNA synthesis or as concentration of cAMP) is depressed in AD.

It is also known that intracellular levels of cAMP play a crucial role in the maturation and function of lymphocytes. Therefore it may hypothesized that T-cells of atopic patients undergo some disturbance in the stages of their maturation; this might explain the many recent reports of T-cell disfunction in AD.

We have presented evidence that T-cell differentiation in AD is likely to be disturbed: the percentage of lymphocytes forming rosettes with SRBC (E-rosettes) is lower, whereas the percentage of lymphocytes forming rosettes with autologous erythrocytes (H-rosettes) is higher then normal. It has been claimed that H-rosetting is a property of relatively immature T-cells (so-called T_1 cells), while further maturation to the more differentiated T_2 cells is accompanied by loss of this property. Isoproterenol inhibits the formation of H-rosettes, when added to lymphocytes of normal subjects, while it clearly fails to affect the percentage of H-rosetting lymphocytes from AD patients. These data suggest that the β-blockade could be responsible for the defect in the maturation of T cells. Moreover it has been demonstrated that isoproterenol inhibits the proliferation of lymphocytes induced by PHA, due to the increased concentration of cAMP.

We have shown that the percentage of inhibition induced by isoproterenol is lower in AD patients; the difference is statistically significant.

Propanolol antagonized the effect of isoproterenol, while PGE_1 produced the same effect in AD as in controls.

These data suggest that the β-receptor blockade affects the maturation, differentiation and function of T cells.