BIOCHEMICAL AND HISTOLOGICAL STUDIES OF A MODEL OF PSORIASIS

Rydlewski, M.[1], Cruz-Silva, I.[1], Praxedes-Garcia P.[1], Caetano A.L.[2], Viel, T.A.[2], Buck H.S.[2], Sampaio, M.U.[1], Araujo, M.S.[1]

[1]Departamento de Bioquímica–UNIFESP/EPM, [2]Departamento de Fisiologia da FCMSCSP, São Paulo, Brazil

Proteases are commonly related to some inflammatory diseases. Among them, tissue kallikrein (TK), a kinin-releasing related protease, is also responsible for controlling the turnover of the stratum corneum on skin, which is affected by human neutrophil elastase (HNE), a highly harmful when liberated without control. HNE also causes hyperproliferation of epidermal cells and inflammatory cell infiltration. Both proteases are related to several inflammatory skin diseases, such as psoriasis, which is characterized by thickened epidermis, hyperkeratosis and dermal inflammatory infiltrate. The aims of the present study were to establish a model of psoriasis in mice by HNE application on skin and evaluate its effect on endogenous elastase and TK. The animals' skin treated with 0.1-10nM HNE showed a higher protein concentration and a dose-dependent hydrolysis of an elastase specific chromogenic substrate. There were also an increased number of epidermal layers and vessels, and a 2-fold thickening of stratum corneum compared to the control group. The group treated with 10nM of HNE, followed by a 24 hour later application of 0.5-200nM SBTI, a protease inhibitor, showed decreased hydrolysis of the elastase substrate, and the histological markers were reverted. 10nM HNE also increased the kinin releasing of low molecular mass kininogen by TK. Our results suggested that the interaction between TK and HNE is important in the psoriasis development.

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