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Increased expression of A-kinase anchoring proteins in T cells from systemic lupus erythematosus patients

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Introduction

Deficient activation of protein kinase A (PKA) is a characteristic of T cells in systemic lupus erythematosus (SLE). A-kinase Anchoring Proteins (AKAPs) associate to and regulate the activity of PKA [1]. Furthermore, some AKAPs are expressed in T cells and influence their function [2]. Therefore, altered expression and/or function of AKAPs can play a role in the deregulated activity of PKA observed in SLE T cells.

Aims

To analyse the expression of different AKAPs in T cells isolated from SLE patients.

Patients and methods

T cells were isolated by negative selection using magnetic beads from SLE patients (n = 12) and healthy controls (HC, n = 12). RNA was purified and levels of AKAP79, AKAP95 and AKAP450 mRNA were quantified by RT-qPCR. Subsequently, the analysis of AKAP79 expression was extended to include a total of 24 SLE patients and19 HC. In addition, AKAP79 protein was detected by Western Blot and quantified with Quantity One software.

Results

Levels of AKAP450 mRNA were comparable in HC and SLE T cells (10.73 +/- 0.73 (HC, n = 12) vs 12.69 +/- 1.09 (SLE, n = 12), P = 0.15). However, T Cells from SLE patients had significantly higher levels of AKAP79 and AKAP95 mRNA than HC (AKAP79: 1.99 +/- 0.29 (HC, n = 19) vs 3.61 +/- 0.72 (SLE, n = 24), P= 0.04; AKAP95: 2.58 +/- 0.18 (HC, n = 12) vs 4.13 +/- 0.31 (SLE, n = 12), P = 0.0005). Analysis of AKAP79 protein levels showed increased levels of AKAP79 in SLE T cells compared to HC T cells (0.38 ± 0.05 (HC) vs 0.75 ± 0.18 (SLE), P = 0.06).

Conclusions

Increased levels of AKAP79 and AKAP95 in T cells from SLE patients can contribute to the deregulation of PKA activity in these cells.

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