Cutaneous lupus erythematosus (CLE) is a disfiguring manifestation of systemic LE (SLE). The pathophysiology of CLE is unclear. However, regulation of skin inflammation and apoptosis contribute, potentially in a female-biased manner. Differential DNA methylation is important in the sex-specific manifestations of SLE and may underlie dysregulated apoptosis and female bias of CLE. Differentially methylated sites with 924 hypomethylated genes and 519 hypermethylated genes in lupus KC compared to controls. The top canonical pathway was Hippo signaling, suggesting that serum protein values may be useful as potential biomarkers for separating CLE patients from controls. The top canonical pathway was Hippo signaling, which could promote cell death. Further, methylation of FCGR2B was significantly correlated (r^2 = 0.35, p < 0.0115) with the percentage of circulating proinflammatory intermediate monocytes. In addition, total costs were also significantly correlated with the number of therapies patients failed (r = -0.378, p = 0.0056) and total burden of disease (r = -0.394, p < 0.0035). Thus, total charges may accurately reflect correlative biomarkers with psoriasis severity.

Interleukin-27 alleviates psoriatic inflammation by suppressing interleukin-17A production from T17 cells

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Interleukin-17A (IL-17A) has emerged as one of the major pro-inflammatory cytokines in psoriasis. Genetic and clinical studies have shown that IL-17A plays a crucial role in the pathogenesis of psoriasis. IL-17A production by T cells in the skin is regulated by multiple factors, including IL-23 and IL-27. In this study, we measured the effect of IL-27 on the IL-17A production from T17 cells and γδT cells and used IL-27 receptor A deficiency (Il17a^-/-) mice to determine the role of IL-17A in psoriasis. The expression of IL-27 in psoriasis was determined using immunofluorescence staining. The expression of IL-17A in T cells was determined using flow cytometry and enzyme-linked immunosorbent assay. Imiquimod (IMQ)-induced psoriasis model was used to investigate the role of IL-27 in psoriasis in vivo. Our results show that IL-27 is expressed in the lesional skin of psoriasis patients. IL-27 downregulates IL-23-induced IL-17A expression in psoriatic skin. The mechanism by which IL-27 inhibits IL-17A production remains unclear. However, the role of IL-27 in psoriasis remains controversial. In this study, we measured the effect of IL-27 on the IL-17A production from T17 cells and γδT cells and used IL-27 receptor A deficiency (Il17a^-/-) mice to determine the role of IL-17A in psoriasis. The expression of IL-27 in psoriasis was determined using immunofluorescence staining. The expression of IL-17A in T cells was determined using flow cytometry and enzyme-linked immunosorbent assay. Imiquimod (IMQ)-induced psoriasis model was used to investigate the role of IL-27 in psoriasis in vivo. Our results show that IL-27 is expressed in the lesional skin of psoriasis patients. IL-27 downregulates IL-23-induced IL-17A expression in psoriatic skin. The mechanism by which IL-27 inhibits IL-17A production remains unclear. However, the role of IL-27 in psoriasis remains controversial.

Psoriasis patient serum biomarkers may be useful for separating response to therapy endotypes

S Chu1, G Damiani1, B Richardson1, M Cameron1, T McCormick1 and K Cooper2

Psoriasis is a chronic inflammatory disease affecting skin, joints, and nails. It is characterized by erythema and well-demarcated scaling plaques. The pathogenesis of psoriasis is complex and multifactorial, involving genetic, environmental, and immunological factors. Current therapeutic strategies include topical treatments, phototherapy, and systemic medications. Accurate prediction of treatment response is crucial for optimal management of psoriasis. A recent study aimed to identify serum biomarkers that could predict response to therapy endotypes. The study utilized a serum proteome-based approach to analyze a cohort of psoriasis patients treated with 17α-allyl-19-nortestosterone (17AAT) and compared them to a control group. The results showed that several serum proteins were significantly differentially expressed between responders and non-responders, with some proteins showing potential as biomarkers for predicting treatment response. The findings suggest that serum protein values may be useful as potential biomarkers for separating psoriasis patient endotypes.

**Correlation of psoriasis severity with burden of disease cost in psoriatic patients**

S Chu1, G Damiani1, B Richardson1, M Cameron1, T McCormick1 and K Cooper2

Psoriasis is a chronic inflammatory disease affecting skin, joints, and nails. It is characterized by erythema and well-demarcated scaling plaques. The pathogenesis of psoriasis is complex and multifactorial, involving genetic, environmental, and immunological factors. Current therapeutic strategies include topical treatments, phototherapy, and systemic medications. Accurate prediction of treatment response is crucial for optimal management of psoriasis. A recent study aimed to identify serum biomarkers that could predict response to therapy endotypes. The study utilized a serum proteome-based approach to analyze a cohort of psoriasis patients treated with 17α-allyl-19-nortestosterone (17AAT) and compared them to a control group. The results showed that several serum proteins were significantly differentially expressed between responders and non-responders, with some proteins showing potential as biomarkers for predicting treatment response. The findings suggest that serum protein values may be useful as potential biomarkers for separating psoriasis patient endotypes.

**IL-27 induces IL-15 production to facilitate T cell survival in allergic contact hypersensitivity (CHS) model using IL-27p28eGFP receptor knock-in mouse**

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Interleukin (IL)-27 has been widely reported to inhibit IL-17A production in many diseases. Moreover, in the murine allergic contact hypersensitivity (CHS) model using IL-27p28eGFP receptor knock-in mouse (IL-27p28eGFP-/-RECA mice), we found increased IL-27 production in CD172a+MAC. Functionally, IL-27p28fl/fl;LysM-cre transgenic mice demonstrated less DNFB-induced ear thickening and dermal HF-associated CD8 T cells compared to WT mice (p < 0.001). Thus, we concluded that IL-27p28 induces IL-15 production to facilitate T cell survival in allergic contact hypersensitivity (CHS) model using IL-27p28eGFP receptor knock-in mouse (IL-27p28eGFP-/-RECA mice).