Crosstalk between skin inflammation and adipose tissue-derived products: pathogenic evidence linking psoriasis to increased adiposity.

Chiricozzi A[1], Raimondo A[2], Lembo S[3], Fausti F[4], Dini V[1], Costanzo A[5], Monfrecola G[2], Balato N[2], Ayala F[2], Romanelli M[1], Balato A[6].

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Abstract

INTRODUCTION:

Psoriasis is a chronic skin disorder associated with several comorbid conditions. In psoriasis pathogenesis, the role of some cytokines, including TNF-α and IL-17, has been elucidated. Beside their pro-inflammatory activity, they may also affect glucose and lipid metabolism, possibly promoting insulin resistance and obesity. On the other hand, adipose tissue, secreting adipokines such as chemerin, visfatin, leptin, and adiponectin, not only regulates glucose and lipid metabolism, and endothelial cell function regulation, but it may contribute to inflammation.

AREAS COVERED:

This review provides an updated 'state-of-the-art' about the reciprocal contribution of a small subset of conventional cytokines and adipokines involved in chronic inflammatory pathways, upregulated in both psoriasis and increased adiposity. A systematic search was conducted using the PubMed Medline database for primary articles. Expert commentary: Because psoriasis is associated with increased adiposity, it would be important to define the contribution of chronic skin inflammation to the onset of obesity and vice versa. Clarifying the pathogenic mechanism underlying this association, a therapeutic strategy having favorable effects on both psoriasis and increased adiposity could be identified.

KEYWORDS:

IL-17; IL-6; Psoriasis; TNF-α; adiponectin; chemerin; leptin; metabolic syndrome; obesity; visfatin