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PIGMENTATION

## A CHRONIC INFLAMMATORY MICRO-ENVIRONMENT IS REVEALED IN ACTINIC LENTIGINES

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Introduction & Objectives: Actinic lentigines (AL), or age spots, are skin hyperpigmented lesions associated with age and chronic sun exposure, thus representing a hallmark of photoaged skin. In order to better characterize their physiopathology, an exploration of the inflammatory background was conducted in AL from European and Japanese subjects through genomic and histological analysis.

Materials & Methods: AL from the dorsal side of hands were selected using dermatoscopic imaging. 3mm punch biopsies of AL and adjacent non-lesional (NL) skin were performed on 35 women (15 French, 20 Japanese; 50-70 y.o.) and processed for gene expression profiling using full genome Affymetrix® chips or for immunostaining of inflammatory/immune cells with human antibodies against HLA-DR, CD14, CD68, CD11c, FXIIIa, CD117, CD4.

Results: Up to 10% of the total modulated genes in AL versus NL were associated with an inflammation/immune response of the skin. Genes modulated in both European and Japanese AL evidenced a pro-inflammatory environment characterized by the activation of arachidonic acid pathway (PLA2,PTGS2/COX2). Furthermore, the overexpression of several chemokines genes (CCL13, CCL18, CCL19, CCL27) suggested that a chemo-attraction of inflammatory cells could take place in AL. Indeed, the immunostaining of various cell subsets revealed a significant increase in HLA-DR+ cells, CD68+ macrophages and CD4+ T-cells in the dermis of AL vs NL.

Conclusions: Prostaglandins and chemokines release, associated with infiltration of immune cells, may create a chronic inflammatory microenvironment in AL that could contribute to the alteration of skin homeostasis and hyperpigmentation. Targeting this inflammatory loop may contribute to skin normalization and improve the efficacy and sustainability of age spot treatment.





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