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240 Serum polyclonal free light chains: possible markers of immune activation in psoriasis

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The aim of this study was to investigate the role of polyclonal free light chains (FLCs) of the immunoglobulins as markers of immune activation in the management of psoriatic patients on treatment with biologics. The overall study population included 30 patients affected by mild-to-severe psoriasis with either ongoing biological treatment or without any current systemic therapy and 10 healthy controls. FLCs, which include κ and λ light chains, were determined by an assay specific for immunoassay. Their concentrations were measured as the sum of intact and monoclonal light chains (κ and λ FLCs) compared to healthy controls. Interestingly, κ and λ FLCs values were significantly increased only in psoriatic patients with ongoing biological treatment and in responder subjects. Furthermore, correlation of both κ and λ FLCs with duration of therapy showed significant results. Patients with FLC levels above the normality range and under biological treatment showed higher odds to be ANA+ respect to patients with FLC levels above the normality range but without biological treatment for less than 12 months. Increased FLCs levels would seem to act as markers of immune reactivation in psoriatic patients on treatment with biologic agents suggesting that the determination of FLCs could have clinical relevance in psoriasis clinical management.

242 High potency activities of a Tangerine extract to target Rosacea imprints

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Rosacea, a chronic skin disorder, is characterized by inflammation and vascular abnormalities of the central facial skin. Cathelicidin LL-37 and VEGF are both specific contributors to pathophysiology of Rosacea and involved in inflammatory and angiogenesis pathways, respectively. The aim of this work was to assess the soothing and anti-redness properties of a dermo-cosmetic ingredient in several models exposed to the different components of the rosacea environment. Firstly, the use of human normal keratinocyte primary cell line (NHK) after a tangerine extract pre-treatment allowed us to show a decrease of VEGF and MMP9 gene expression and ILB cytokine release after an stimulation by Rosacea-like cocktail (TLR agonist, LL17, TNFa) in the same way, after a stimulation with Substance P on this same cell line, an inhibition of the production of cytokines IL-1β and TNFa could be demonstrated. Then, the potency on angiogenesis axis of this compound could be confirmed on a cocultured NHDF/HAVEC based VEGF stimulated Pseudohydra model as well as on the CA2+ production of a Au5MC model. Moreover, this compound once formulated in a dermo-cosmetic cream was able to inhibit the production of chemokines involved in neurogenic inflammation and EGFR pathways in a rosacea reconstructed human epidermis model. These data suggest that a topical Dermo-Cosmetic product with this tangerine extract could be useful to protect against Rosacea burden in targeting inflammatory and vascular scales.

244 Comprehensive Approach to Addressing Skin Inflammaging

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One of skin’s major functions is to serve as a protective barrier to our environment. The skin is exposed to many external aggressors on a daily basis, including but not limited to various wavelengths of light (UV, blue light, etc.), thermal stress, and pollutants (shale and cigarette smoke, etc.). Over the past few decades, environmental exposures have been extensively studied and coined the “exposure”. The skin exposure, which comprises the total daily exposure that our skin endures, can lead to damaging inflammatory cascades within the skin. These inflammatory cascades can be activated by various alarmins such as key inflammatory mediators (e.g. IL-1) as well as reactive oxygen species (ROS). Alarmins perpetuate a strong inflammatory cascade, which can ultimately result in neutrophil and macrophage recruitment and lead to damaged extracellular matrix structures and appearing, over time, as visible lines, wrinkles, loss of firmness, and sagging. Furthermore, inflammation can induce other visible issues such as redness and hyperpigmentation. While many cosmetic ingredients address phases during the resolution phase of inflammation, there is limited information about how to address the third phase of inflammation (resolution). In fact, it was relatively recently discovered that the skin can actively resolve inflammation during the resolution phase, whereby the skin produces specific mediators that help to resolve inflammation. The resolution phase utilizes lipid mediators which act to coordinate the resolution of inflammation.

Here, we propose a novel technique to more comprehensively address exposure-induced “inflammaging” (inflammation-induced aging) by utilizing both and other powerful active ingredients to help mitigate the inflammatory response and to aid in the resolution of inflammation through their interactions with the appropriate systems to ensure delivery to skin. Finally, we employ an innovative combination of ingredients to address skin’s own natural resolution process via boosting the production of specialized pro-resolution mediators (maresins and lipoxins).

245 Metabolic reprogramming defines myeloid cell function in skin repair

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The skin provides a life-sustaining structural and immunological outer barrier of the body and harbors immune mechanisms that protect the organism from diverse external threats including infections and mechanical injuries. Skin injury induces a complex, dynamic cellular program proceeding in sequential stages of inflammation, tissue growth and differentiation. Cells of the monocyte/macrophage lineage are an integral component of an effective repair response. Blood monocytes that are recruited to the side of tissue damage sense a variety of environmental cues of injured tissue and integrate those into a host protective wound healing response. Given that inappropriate macrophage activation underlies a broad range of pathophysiological processes, from chronic inflammatory skin diseases, type 2 diabetes and cancer, macrophages have emerged as an important target to treat disease. The molecular determinants that precisely control the dynamics of macrophage functional plasticity during healing progression are largely unknown and are just beginning to emerge. Previously we have identified the tumor necrosis factor-α (TNFα) as an endogenous inhibitor of the polarization process from a pro-inflammatory and angiogenic phenotype in the early healing phase towards a type 2 cytokine-activated pro-healing phenotype in the late phase of healing. We aim to understand the functional relationship between cellular metabolism and the execution of specific repair functions in injury-associated monocytes/macrophages. We will present novel findings showing that different metabolic functions of mitochondria are required to support phase-specific injury-towards-repair programs in wound macrophages. Shaping macrophage-mitochondria functions by targeting mitochondria could offer a new approach to pharmacologically boost regenerative capacity in inflammatory skin diseases.