Sebaceous glands are actively and differentially involved in the pathogenesis of atopic dermatitis and psoriasis as revealed by spatial transcriptomics

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Atopic dermatitis (AD) is a chronic, relapsing, inflammatory skin disease characterized by pruritus and eczematous skin lesions. It has been described that the repeated topical application of the hapten oxazolone to the ears of sensitized mice cause lesions that mimic some of the principal features of the most of the published AD models. The incidence was significantly high (6 to 12), which translates into a long duration of the assay and causing a significant distress to the animals. In the present study we have characterized a model of dermatitis induced by 4 challenges of oxazolone applied on days 7, 9, 11, and 14 post-sensitization. Ear samples were obtained on day 15. Using this protocol, the main clinical signs consists of an increase in ear skin thickness and erythema. Histological analysis reveals the presence of a diffuse, moderate acanthosis with frequent areas of parakeratosis and mounds of macrophages, lymphocytes and lymphoproliferative foci. Well as proliferation of fibroblasts is also observed. Skin biopsies show increased levels of mRNA expression of 11 cytokines, including the Th2 markers IL-4, IL-13, IL-22 and IL-31, and a reduced expression of loricin, indicating barrier disruption. To further validate the model, we assessed the effect of commercial formulations of the anti-inflammatory drugs betamethasone, tacrolimus and crisaborole. The three topical treatments were able to reduce mRNA expression of IL-31 and IL-22, and increase the expression of loricin. Of note, betamethasone inhibited IL-22 expression whereas tacrolimus increased it and crisaborole had no effect at all. Overall, our results show that this model replicates several AD features and respond to approved topical drugs, making it suitable for screening purposes.

Characterization of a murine model of dermatitis induced by oxazolone

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Tobacco smoke is a mix of thousands of chemicals, many of which are known to cause devastating diseases. More than 60% of smokers are smokers, while 10% of children are smokers. But the evidence on the effect of smoking on the skin is still limited. The skin is the largest and most complex organ of the body, and it is exposed to environmental factors, such as tobacco smoke, that can alter its function and structure. Smoking has been associated with a number of skin diseases, including acne, rosacea, and psoriasis. In addition, smoking has been linked to an increased risk of skin cancer, including melanoma and non-melanoma skin cancer. The exact mechanisms by which smoking affects the skin are not fully understood, but it is believed that smoking can alter the skin's barrier function, promote inflammation, and affect the immune system. Furthermore, smoking can affect the skin's response to UV radiation, leading to an increased risk of skin cancer. In conclusion, smoking has a significant impact on the skin and its health, and it is crucial to reduce smoking to improve skin health and prevent the development of skin diseases.