Oxidative stress and mitochondrial alterations in Kindler Syndrome

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Kindler Syndrome (KS) is an autosomal recessive skin disorder caused by mutations in the COL7A1 gene. Mutations in this gene lead to skin blistering, photosensitivity, and premature cancer predisposition, but the mechanisms involved remain to be fully understood. Here, we present data on the role of oxidative stress and mitochondrial dysfunction in KS skin. We observed increased levels of reactive oxygen species (ROS), mitochondrial membrane potential (ΔΨm), and mitochondrial architecture in KS skin samples compared to control skin. Furthermore, we found a significant decrease in the levels of mitochondrial DNA (mtDNA) and an increase in the levels of mitochondrial damage, such as mitochondrial DNA deletions and strand breaks. These results suggest that oxidative stress and mitochondrial dysfunction play a crucial role in the pathogenesis of KS. The findings of this study provide new insights into the mechanisms underlying the pathogenesis of KS and may lead to the development of novel therapeutic strategies.

Regulation of IL-33 expression by normal human epidermal keratinocyte

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IL-33 is a cytokine produced by keratinocytes and is involved in the regulation of immune responses. However, the regulation of IL-33 expression in normal human keratinocytes remains largely unknown. In this study, we investigated the regulation of IL-33 expression in normal human epidermal keratinocytes (NHEKs) treated with TNFα and IL-1β. We found that treatment with TNFα and IL-1β upregulated IL-33 expression, while TGFβ1 treatment had no effect. We also found that the expression of IL-33 was dependent on the NF-κB and STAT3 pathways. In addition, we observed that IL-33 expression was downregulated by the treatment with the NF-κB inhibitor Bay11-7082. These findings provide new insights into the regulation of IL-33 expression in normal human epidermal keratinocytes and may help in developing new therapeutic strategies for skin diseases.

Regulation of IL-33 transcription and expression in psoriasis

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IL-33 is a cytokine involved in the regulation of immune responses and is known to play a role in psoriasis. However, the mechanisms responsible for the upregulation of IL-33 expression in psoriasis are not fully understood. In this study, we investigated the regulation of IL-33 transcription and expression in psoriasis. We found that the expression of IL-33 was upregulated in psoriasis lesions compared to normal skin. We also observed that the expression of IL-33 was dependent on the NF-κB and STAT3 pathways. In addition, we observed that the expression of IL-33 was downregulated by the treatment with the NF-κB inhibitor Bay11-7082. These findings provide new insights into the regulation of IL-33 expression in psoriasis and may help in developing new therapeutic strategies for this disease.

Splicing regulation disturbances in psoriasis pathogenesis

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Psoriasis is a chronic inflammatory skin disorder characterized by the upregulation of IL-33 expression. However, the mechanisms responsible for the upregulation of IL-33 expression in psoriasis are not fully understood. In this study, we investigated the regulation of IL-33 transcription and expression in psoriasis. We found that the expression of IL-33 was upregulated in psoriasis lesions compared to normal skin. We also observed that the expression of IL-33 was dependent on the NF-κB and STAT3 pathways. In addition, we observed that the expression of IL-33 was downregulated by the treatment with the NF-κB inhibitor Bay11-7082. These findings provide new insights into the regulation of IL-33 expression in psoriasis and may help in developing new therapeutic strategies for this disease.