

Keloids and Inflammation: The Crucial Role of IL-33 in Epidermal Changes

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BACKGROUND:

Keloids are benign fibroproliferative disorders characterized by excessive collagen deposition and inflammation that extend beyond the original wound boundaries.

METHODS:

In this study, through clinical sample and high-throughput analysis, we investigate the role of interleukin-33 (IL-33) in the pathogenesis of keloids, highlighting its expression in keratinocytes and its interactions with infiltrating lymphocytes.

RESULTS:

Our results demonstrate that IL-33 levels are significantly elevated in the epidermis of keloid tissues, where it functions as an alarmin, promoting a chronic inflammatory response. We further reveal a feedback loop between IL-33 and interferon-gamma (IFN- γ), whereby IL-33 induces IFN- γ production in lymphocytes, which in turn stimulates keratinocytes to produce more IL-33. This loop contributes to impaired keratinocyte differentiation and skin barrier dysfunction, exacerbating the inflammatory environment.

CONCLUSION:

By elucidating the role of the IL-33/ST2 axis in keloid formation, this research provides valuable insights into potential therapeutic targets for managing this challenging condition.