Integrated Multi-omics Unveils the Epigentic Landscape in the Pathogenesis of Keloid

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Keloids are benign skin tumors, but their molecular mechanisms of pathogenesis remain unclear. Through multi−omics analyses, we identified that keloid pathogenesis involves transcriptional dysregulation driven by DNA methylation reprogramming. Compared to normal scars, keloid tissues exhibit a global DNA hypermethylation pattern, which is similarly observed in keloid fibroblasts. RNA−seq analysis revealed significant upregulation of bone and cartilage−related signaling pathways, while hormone−related pathways were markedly downregulated in keloid samples. Furthermore, inhibition of aberrant DNA hypermethylation using a DNA methyltransferase inhibitor suppressed the growth of keloid fibroblasts. These findings indicate that DNA methylation plays a crucial role in keloid pathogenesis, and targeting the regulation of bone, cartilage, and hormone−related signaling pathways may provide novel therapeutic strategies for keloid treatment.