Diabetic Wound Repair: From Mechanism to Therapeutic Opportunities

ABSTRACT

Diabetic wound healing, characterized by persistent inflammation, impaired angiogenesis, and dysfunctional cellular responses, remains a major clinical challenge due to its complex pathophysiology. This challenge is most evident in diabetic foot ulcers (DFUs), which carry high risks of infection, recurrence, and amputation, contributing substantially to patient morbidity, mortality, and healthcare costs. Despite multidisciplinary care, debridement, and advanced dressings, healing outcomes are often suboptimal, highlighting an urgent need for deeper pathophysiological insights and more effective therapeutic strategies. This review synthesizes current understanding of DFU pathogenesis, emphasizing how sustained metabolic dysfunction disrupts fibroblast and immune cell function, thereby perpetuating chronic wounds. We also critically examine commonly used animal models and their limitations in replicating the complexity of human DFUs and discuss emerging therapeutic approaches with translational promise. Advancing our understanding of these mechanisms and validating innovative interventions may ultimately reduce DFU-related amputations and mortality, improve healing outcomes, and enhance patient quality of life. This review aims to catalyze future research and therapeutic innovation in diabetic wound care

Diabetic wound healing is compromised by chronic metabolic dysfunction that disrupts essential cellular processes. This review examines key predisposing factors, including neuropathy and vascular disease, characterizes dysfunction in critical cell types such as macrophages and fibroblasts, evaluates limitations of current models, compares established and emerging therapies, and outlines future research and clinical strategies to advance management for diabetic foot ulcers.

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