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Photobiomodulation Activates WNT/β-catenin Signaling for Wound Healing in an in Vitro Diabetic Wound Model

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Abstract : Diabetic foot ulcers (DFUs) are a complication of diabetes mellitus (DM), a metabolic disease caused by insulin resistance or insufficiency, resulting in hyperglycaemia and low-grade chronic inflammation. Current therapies for treating DFUs include wound debridement, glycaemic control, and wound dressing. However, these therapies are moderately effective as there is a recurrence of these ulcers and an increased risk of lower limb amputations. Photobiomodulation (PBM), which is the application of non-invasive low-level light for wound healing at the spectrum of 660-1000 nm, has shown great promise in accelerating the healing of chronic wounds. However, its underlying mechanisms are not clearly defined. Studies have indicated that PBM induces wound healing via the activation of signaling pathways that are involved in tissue repair, such as the transforming growth factor-β (TGF-β). However, other signaling pathways, such as the WNT/β-catenin pathway, which is also critical for wound repair, have not been investigated. This study aimed to elucidate if PBM at 660 nm and a fluence of 5 J/cm² activates the WNT/β-catenin signaling pathway for wound healing in a diabetic cellular model. Human dermal fibroblasts (WS1) were continuously cultured high-glucose (26.5 mM D-glucose) environment to create a diabetic cellular model. A central scratch was created in the diabetic model to 'wound' the cells. The diabetic wounded (DW) cells were thereafter irradiated at 660 nm and a fluence of 5 J/cm². Cell migration, gene expression and protein assays were conducted at 24- and 48-h post-PBM. The results showed that PBM at 660 nm and a fluence of 5 J/cm² significantly increased cell migration in diabetic wounded cells at 24-h post-PBM. The expression of CTNNB1, ACTA2, COL1A1 and COL3A1 genes was also increased in DW cells post-PBM. Furthermore, there was increased cytoplasmic accumulation and nuclear localization of β-catenin at 24 h post-PBM. The findings in this study demonstrate that PBM activates the WNT/β-catenin signaling pathway by inducing the accumulation of β-catenin in diabetic wounded cells, leading to increased cell migration and expression of wound repair markers. These results thus indicate that PBM has the potential to improve wound healing in diabetic ulcers via activation of the WNT/β-catenin signaling pathway.

Keywords : wound healing, diabetic ulcers, photobiomodulation, WNT/β-catenin, signalling pathway
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