PP-06

마우스 피부 상처 모델에서 GV1001의 상처 치유 촉진 효과

GV1001 Enhances Cutaneous Wound Healing in a Murine Model

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Purpose:

GV1001 is a telomerase-derived peptide reported anti-inflammatory and cytoprotective properties. However, its potential role in cutaneous wound healing has not been fully elucidated. This study evaluated its effects on cutaneous wound healing in a murine excisional model.

Methods:

Full-thickness 6-mm dorsal excisional wounds were created in male C57BL/6 mice. Animals were randomly assigned to receive daily subcutaneous injections of saline or GV1001 (1 mg/kg). Wound closure was evaluated on days 0, 3, 6, 10, and 12. Pro-inflammatory cytokines (IL-6, TNF-α, IL-1β) were quantified by ELISA. Collagen I/III, TGF-β1, phospho-Smad2, VEGF-A, and CD31 expression were analyzed by Western blotting. Collagen architecture was assessed using Masson's trichrome staining. In vitro keratinocyte viability and migration were evaluated using CCK-8 and scratch assays.

Results:

GV1001-treated mice demonstrated accelerated wound closure compared with controls, with significant differences observed at later time points(Fig. 1.). Early expression of IL-6, TNF-α, and IL-1β was significantly reduced on day 3 in the GV1001 group. Collagen I and III expression was increased on day 12, accompanied by denser and more organized collagen deposition histologically. TGF-β1 levels were decreased, whereas phospho-Smad2 showed no significant difference between groups. VEGF-A and CD31 expression were elevated in GV1001-treated wounds(Fig. 2.). In vitro, GV1001 enhanced keratinocyte migration without affecting cell viability.

Conclusion:

GV1001 enhances cutaneous wound repair in a murine model and is associated with modulation of early inflammatory responses, increased collagen deposition, and elevated angiogenesis-related markers. These findings suggest that GV1001 may represent a potential therapeutic candidate for improving wound healing.

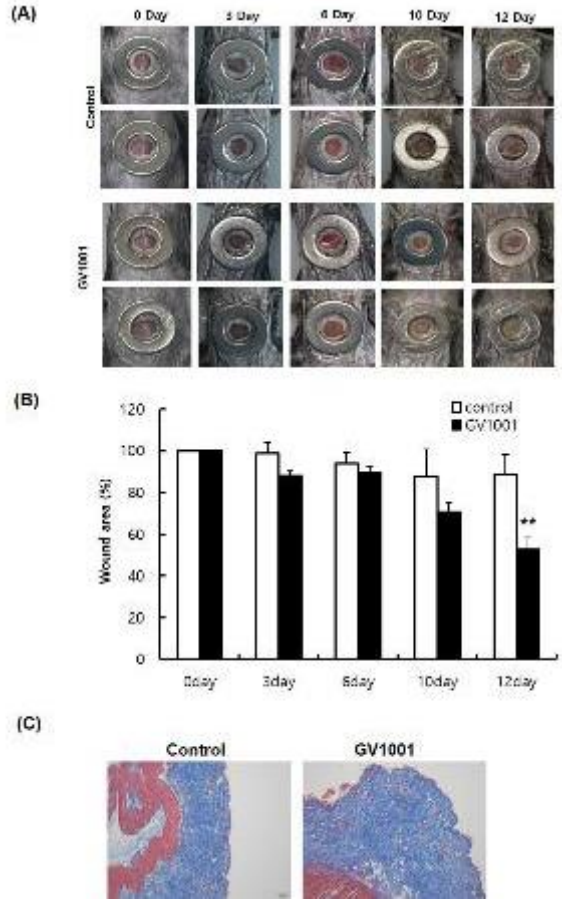

Fig. 1. : Representative wound area images and quantitative analysis over time. (a) Representative images of wound areas in mice from both the control and GV1001 treatment groups. Images were captured to monitor the progression of wound healing in each group. (b) Quantitative analysis of wound areas at the 0, 3, 6, 10, and 12 days post-wounding. Between-group differences were observed from day 3 and reached statistical significance at day 12. (c) Masson's trichrome staining on day 12 post-wounding. GV1001-treated wounds exhibited denser and more organized collagen fiber deposition, suggesting regulated matrix remodeling rather than pathological fibrosis.

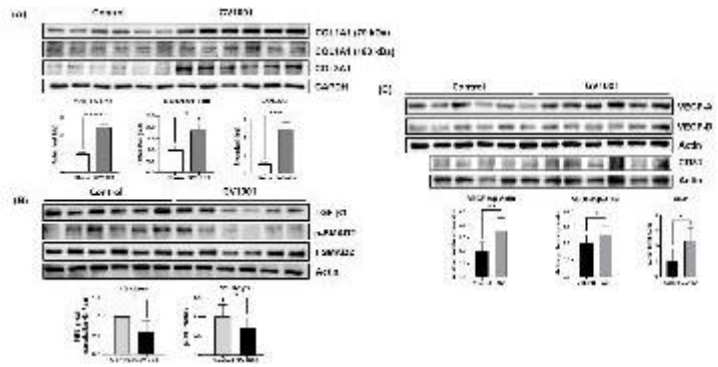

Fig. 2. : Impact of GV1001 on collagen expression and TGF-β signaling in wound tissues on day 12 post-wound development. (a) Western blot analysis showing the upregulation of collagen types I and III in skin wound tissues from GV1001-treated mice. (b) Western blot analysis of TGF-β1 and p-SMAD2 levels in wound tissues. GV1001 treatment resulted in a significant decrease in TGF-β1 protein levels on day 12 post-wound development. (c) Western blot analysis of angiogenic markers (VEGF-A, VEGF-D, and CD31) in wound tissues on day 12 post-wounding. GV1001 treatment significantly increased VEGF-A and CD31 levels, whereas VEGF-D showed no significant difference. Representative blots are shown with loading controls (GAPDH in A; β-actin in B and C).