Far-infrared promotes burn wound healing by suppressing NLRP3 inflammasome caused by enhanced autophagy

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Abstract

Understanding the underlying molecular mechanisms in burn wound progression is crucial to providing appropriate diagnoses and designing therapeutic regimens for burn patients. When inflammation becomes unregulated, recurrent or excessive, it interferes with burn wound healing. Autophagy, which is a homeostatic and catabolic degradation process, was found to protect against ischemic injury, inflammatory diseases, and apoptosis in some cases. In the present study, we investigated whether far-infrared (FIR) could ameliorate burn wound progression and promote wound healing both *in vitro* and in a rat model of deep second-degree burn. We found that FIR induced autophagy in differentiated THP-1 cells (human monocytic cells differentiated to macrophages). Furthermore, FIR inhibited both the NLRP3 inflammasome and the production of IL-1β in lipopolysaccharide-activated THP-1 macrophages. In addition, FIR induced the ubiquitination of ASC, which is the adaptor protein of the inflammasome, by increasing tumor necrosis factor receptor-associated factor 6 (TRAF6), which is a ubiquitin E3 ligase. Furthermore, the exposure to FIR then promoted the delivery of inflammasome to autophagosomes for degradation. In a rat burn model, FIR ameliorated burn-induced epidermal thickening, inflammatory cell infiltration and the loss of distinct collagen fibers. Moreover, FIR enhanced autophagy and suppressed the activity of the NLRP3 inflammasome in the rat skin tissue of burn model. Based on these results, we suggest that FIR-regulated autophagy and inflammasomes will be important for the discovery of novel therapeutics to promote the healing of burn wounds.

Key words: far-infrared, burn wound healing, autophagy, NLRP3 inflammasome