The adverse effects of smoking on wound healing of the skin are known clinically. Recently, an endogenous cholinergic pathway for angiogenesis mediated by endothelial nicotinic acetylcholine receptors was discovered. The objective of this study was to investigate the appropriate concentration of nicotine that accelerated angiogenesis and wound healing. Experiments on tube formation were conducted using an Angiogenesis Kit. Basic fibroblast growth factor (10ng/ml) and nicotine (10⁻¹⁰M, 10⁻⁹M, 10⁻⁸M, 10⁻⁷M, 10⁻⁶M, 10⁻⁴M, 10⁻²M) were added to the conditioned medium. The conditioned medium was used as a control. The area and length of each tube were calculated using an Angiogenesis Image Analyzer. Full-thickness skin defects (8mm) were created on the dorsum of C57BL mice and a silicone sheet (8mm) was sutured. PBS (10⁻¹l), bFGF (1µg), and nicotine (10⁻¹M, 10⁻⁴M, 10⁻⁷M) were topically injected for seven days (n=5). Significant differences in area and length of newly formed tubes were seen between the control group and bFGF and the 10⁻⁴M nicotine-added groups. The wound area was significantly decreased in the wounds treated with bFGF and 10⁻⁴M of nicotine. The epithelium length was significantly longer in the wounds treated with bFGF and 10⁻⁴M of nicotine. In this study, nicotine accelerated angiogenesis and promoted wound healing.

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Introduction

**Key words**
Nicotine promotes tube formation of HUVECs.

Nicotine accelerates wound healing.