The *Pseudomonas aeruginosa* quorum sensing signal molecule *N*-(3-oxododecanoyl) homoserine lactone enhances keratinocyte migration and induces *Mmp13* gene expression *in vitro*

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Abstract	Re-epithelialization is an essential step of wound healing involving three overlapping keratinocyte functions: migration, proliferation and differentiation. While quorum sensing (QS) is a cell density-dependent signaling system that enables bacteria to regulate the expression of certain genes, the QS molecule N-(3-oxododecanoyl) homoserine lactone (AHL) exerts effects also on mammalian cells in a process called inter-kingdom signaling. Recent studies have shown that AHL improves epithelialization in in vivo wound healing models but detailed understanding of the molecular and cellular mechanisms are needed. The present study focused on the AHL as a candidate reagent to improve wound healing through direct modulation of keratinocyte's activity in the re-epithelialization process. Results indicated that AHL enhances the keratinocyte's ability to migrate in an in vitro scratch wound healing model probably due to the high Mmp13 gene expression analysis after AHL treatment that was revealed by real-time RT-PCR. Inhibition of activator protein 1 (AP-1) signaling pathway completely prevented the migration of keratinocytes, and also resulted in a diminished Mmp13 gene expression, suggesting that AP-1 might be essential in the AHL-induced migration. Taken together, these results imply that AHL is a promising candidate molecule to improve re-epithelialization through the induction of migration of keratinocytes. Further investigation is needed to clarify the mechanism of action and molecular pathway of AHL on the keratinocyte migration process.
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