WOUND HEALING

SUSTAINED SECRETION OF THE ANTIMICROBIAL PEPTIDE PSORIASIN IS MEDIATED BY THE SAME CELLULAR MACHINERY THAT REGULATES THE WOUND HEALING RESPONSE

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Introduction: Maintaining a prolonged antimicrobial response is an important element of cutaneous innate immunity. A major component of this immunity is the secretion of antimicrobial peptides (AMPs), such as psoriasin (S100A7), which is released from epidermal keratinocytes in response to bacterial infection and bacterial components like flagellin. The signalling cascade which leads to psoriasin secretion in skin keratinocytes during infection is not clearly understood.

Objective: The main goal of this work is to elucidate the signalling mechanism that leads to S100A7 secretion and investigate the potential impact of skin cleansing on longer term AMP psoriasin levels.

Methods: Differentiated primary skin keratinocytes were used for treatments in vitro. Target genes and proteins were measured by qPCR, ELISA and western blots. Skin biopsies from healthy volunteers were used and, target biomarkers were evaluated by IHC ex-vivo.

Results: The data showed that skin keratinocytes secreted S100A7 in a biphasic manner. The acute phase required activation of TLR-5 signalling; the chronic secretion of S100A7 appeared to utilize the same signalling pathways, mediated by the downregulation of caspase 8, which occurs during wound healing. Skin cleansing with a cosmetic soap bar containing thymol, terpineol, and silver had no effect on S100A7 expression and it did not modulate the caspase 8 signalling either.

Conclusion: Results showed downregulation of caspase 8, which happens during wound healing plays a critical role in AMP secretion during infection. Skin cleansing with soap containing thymol, terpineol, and silver does not modulate caspase 8 levels, and hence does

not impact the S100A7 levels on skin.