THE NON-CODING RNA, PRINS AFFECTS AIM2 INFLAMMASOME ACTIVATION IN KERATINOCYTES

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The non-coding RNA, PRINS was previously described by our research group as a differentially expressed transcript in psoriatic uninvolved and healthy skin. The expression level of PRINS in cultured keratinocytes is altered after exposure to various stress factors and silencing of it decreases the viability of keratinocytes during stress stimulations suggesting its role in stress response of the cells. A potential stress signal in psoriatic skin may be the extracellular DNA, which activates the AIM2 inflammasome. The activated inflammasome cleaves the precursor proIL-1β form into mature, functioning IL-1β. The role of the AIM2 inflammasome and the IL-1β cytokine in psoriasis has been described recently.
The aim of our study was to investigate if the PRINS non coding RNA affects the expression and activation of the inflammasome members and IL-1β in normal human epidermal keratinocytes (NHEK) after exposure to extracellular DNA.
The expression of PRINS was transiently silenced by a vector based RNA interference method in cultured NHEK cells. Silenced and non-silenced NHEK cells were primed with the cytokines TNF-α and IFN-γ and transfected with the synthetic DNA analogue poly(dA:dT). The expression of PRINS and inflammasome members was detected by real-time RT-PCR and the secreted IL-1β was measured by ELISA.
Poly(dA:dT) treatment caused a moderate increase in PRINS expression and IL-1β secretion as well, whereas priming with a combination of TNF-α and IFN-γ before poly(dA:dT) transfection resulted in a highly elevated PRINS expression and higher secreted IL-1β levels. The silencing of PRINS decreased the amount of secreted IL-1β, but did not affect the expression of the proIL-1β or AIM2.
Our results suggest that the PRINS non-coding RNA regulates the IL-1β production of NHEK cells, but not through the regulation of proIL-1β expression, rather contributing to inflammasome-activation.

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