signaling pathways were measured by Western blot, enzyme activity was measure using fluorescent substrates, ATP production was measured with luminescent method.

Results: Our results show that though both types of LPS-activated MFs secrete IL-1β, in the case of M-MFs IL-1β is released rapidly and only for a short time period, while IL-1β secretion by GM-MFs is sustained. We found striking differences in Nlrp3 and caspase-1 expression, also in caspase-1 activation. We measured substantial differences in the activation of signaling pathways, as well as in the effect of IL-10 neutralizing antibody, and in the expression of IL-1Ra and that of the ecto-ATPases on Nlrp3 inflammasome activation.

Conclusion: Due to intensive studies, the general mechanism of Nlrp3 inflammasome activation is well characterized, nevertheless our results demonstrate that the actual inflammasome activation and IL-1β secretion is substantially determined by the molecular characteristics of a given cell.

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STUDIES ON THE EFFECT OF PROPIONIBACTERIUM ACNES ON THE BARRIER PROPERTIES OF HUMAN *IN VITRO* CULTURED KERATINOCYTES

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The human skin is heavily colonized by a specialized microbial community called the microbiome, which plays a complex role in the protection from the attack of external pathogens. This microbial flora can interact with the cells in the healthy skin and play a role in the maintenance of skin homeostasis, but also known to contribute to the pathogenesis of different diseases.

Our aim was to analyze whether the Propionibacterium acnes (P. acnes) bacterium, a member of the skin microbiome, or the bacterium induced pro-inflammatory mediator, TNFα has any effect on the barrier properties of our epider-mis.

For that, a confluent monolayer of in vitro cultured human immortalized keratinocytes (HPV-KER cells) were treated with different P. acnes strains and external TNFα in different doses, and changes in the barrier properties were analyzed in real time using the xCELLIgence system. We also analyzed the effect of the bacterium on the mRNA expression changes of tight junction proteins claudin 1, 2, 4 (CLDN1, 2, 4), ocludin1 (OCL1) and ZO1 in these cultures using real-time RT-PCR.

Our results suggest that the bacterium induced an elevation, followed by a drop of the measured impedance values in the keratinocyte monolayers, possibly due to dynamic alterations of the barrier properties. The extent of these changes depended on the used P. acnes strain and the applied doses. Addition of TNFα (1, 5, 10 ng/ml), a cytokine that is a know mediator of the P. acnes-induced innate im-mune and inflammatory events in keratinocytes also lead to a marked decrease of the measured impedance of the HPV-KER monolayers.

Real-time RT-PCR analysis of tight junction genes suggested that CLDN2 and 4 mRNAs were not present in these cells. However, the expression of CLDN1 decreased, whereas ZO1 and OCL1 mRNA levels increased in response to the bacterial treatment.

Our results suggest that our microbiome can modulate the barrier properties of the epidermis. It is possibly achieved, in one hand, through the direct regulation of genes playing a key role in the formation of cell-to-cell contacts. On the other hand, secreted factors, such as the TNFα pro-inflammatory mediator, may also have a direct effect and can loosen the epidermal barrier, possibly leading to the easier penetration of keratinocyte- as well as bacterial-derived factors to deeper tissue compartments. These findings strengthen the importance of a balanced interaction among the epidermal cells and our microbiome for the maintenance of healthy skin functions.

BACTERIAL SEPSIS INCREASES SURVIVAL IN METASTATIC MELANOMA: CLAMIDOPHYLA PNEUMONIAE INDUCES MACROPHAGE POLARIZATION AND TUMOR REGRESSION IN VIVO

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Background: It has been recognized for over 100 years that