IL-1β induces rapid secretion of the antimicrobial protein IL-26 from Th17 cells

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The potential link between the microbiota of CD and PSO

The pro-inflammatory microbes in the gut of psoriatic patients compared to healthy controls. Conversely, microbes known for their anti-inflammatory characteristics that allow them to resist or avoid host phagocytic and immune responses. Acne vulgaris is a multi-factorial inflammatory skin disorder with phagocytosis of Cutibacterium acnes (C. acnes) as an important part in disease pathogenesis. Previous studies have indicated that C. acnes-induced cytokine secretion, and other studies have observed immune cell phagocytic activity associated with TLR2 expression. In this study, we aimed to observe the effects of TLR2 functionality on C. acnes phagocytosis.

Stimulation of TLR2-wild type (WT) and TLR2 knock-out (KO) THP-1 monocytes with C. acnes showed a decreased secretion of pro-inflammatory cytokines IL-1β, IL-6, IL-12p70, and TNF-α in TLR2-KO cells. Flow cytometry analysis of TLR2-WT and TLR2-KO cells stimulated with fluorescein labeled C. acnes revealed decreased phagocytosis in TLR2-KO cells. These findings suggest involvement of other co-receptors in C. acnes phagocytosis. Single-cell RNA sequencing of C. acnes showed increased expression of ITGAM, ITGB2, and CSAR1 genes of the complement system when compared to non-leukosial cells, suggesting that immune cell phagocytosis of C. acnes does not completely depend on TLR2 and may involve activation of the complement system. Further analysis revealed that in both TLR2-WT and TLR2-KO cells, monocytes phosphorycuted more anti-inflammatory and mTOR signaling associated strains (Cm). While both Cm and Cm induce similar pro-inflammatory cytokine secretion levels, Cm are phagocytosed more than Cm, suggesting the possibility of a structural or biochemical difference between Cm and Cm that may influence immune cell pathogen recognition, phagocytosis, and anti-microbial activity.

Psoriatic fungal and bacterial microbiomes identify patient endotypes

C. acnes colonization on lesional skin that correlates with disease severity. We previously reported that, in normal human epidermal keratinocytes, S1PR1 and 2 control neutrophil hyperproliferation. We report identification of pro- and anti-inflammatory intestinal LC precursors, are the sole dendritic cell subpopulation in the epidermis and potent regulators of immune surveillance and tolerance. Unlike conventional DCs, LCs follow unique patterns of development and maintenance after birth and LCs repopulation remain unknown. To address if CBF2 is required for LC hemostasis and repopulation but not required for its embryonic development.

Skin-induced IL-36 triggers plasma cell IgG class switching and allergic disease

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363 Systemic sphingosine 1-phosphate receptor 2 (SIPPR2) deficiency facilitates dermal neutrophil infiltration against S. aureus infection

The lack of S1pr2: The lack of S1pr2 dramatically blocked BM-derived MHCII+ DCs from entering the skin and increased the number of LCs in adult mice compared to WT littermates, while there was no significant decrease in filaggrin2. These data suggest that, although the lack of S1pr2 causes more severe dermal inflammation with higher proinflammatory cytokine expressions. These data were accompanied by an increased skin barrier permeability and a decrease in filaggrin2. These data suggest that, although the lack of S1pr2 in the epidermis decreases the proinflammatory cytokines response, the increased barrier permeability drives a strong dermal inflammation due to bacteria penetration. Conclusions. The lack of S1pr2 decreases S1PR2 expression and activity, which is a negative regulator of IL-36 production. S1PR2 deficient mice showed more severe dermal inflammation with higher proinflammatory cytokine expressions. These data were accompanied by an increased skin barrier permeability and a decrease in filaggrin2. These data suggest that, although the lack of S1pr2 in