After 48 hrs, supernatants (SUPs) were harvested and cytokine content quantified by ELISA. Pretreated with a CD126-IL-6 chimeric molecule and washed x 4 prior to setting up cultures. Set up identically except that LCs were not pretreated but, rather, responding T cells were pretreated with medium alone. (3) Cultures were set up identically except that LCs were pretreated with medium alone. (4) Cultures were set up identically except that LCs were pretreated with the chimeric molecule showed similar changes in IFNγ and IL-17A production. These results strongly indicate that the effect of IL-6 treatment of LCs on biasing the outcome of Ag presentation results from trans-presentation of IL-6 by LCs to responding T cells.

In vitro genetic reprogramming increases MHC-I expression and ameliorates resistance to an antitumor immune response in Merkel cell carcinoma

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Merkel cell carcinoma (MCC) is a rare but aggressive skin cancer with high levels of unresponsiveness to immune checkpoint inhibitors (ICIs). A primary resistance mechanism driving IC1 resistance in MCC is MHC class I expression. Understanding the mechanisms regulating MHC class I expression may lead to the identification of novel therapeutic targets for MCC.

To investigate the regulation of MCC MHC class I expression, we used a transgenic mouse model in which MCC cells are transplanted into the skin of immuno-compromised mice. This model allows for the study of MCC MHC class I expression in vivo. We found that MCC cells expressing a dominant-negative mutant of the transcription factor T-bet, which is involved in the regulation of MHC class I expression, exhibited a significant decrease in MHC class I expression compared to wild-type MCC cells.

In vitro, we found that MCC cells cultured with CD4+ T cells exhibited a significant decrease in MHC class I expression compared to MCC cells cultured without T cells. This decrease in MHC class I expression was not observed in MCC cells cultured with CD8+ T cells. These results suggest that the decrease in MHC class I expression observed in MCC cells cultured with CD4+ T cells is mediated by CD4+ T cells, possibly through the release of cytokines or other factors that inhibit MHC class I expression.

In conclusion, our results suggest that the decrease in MHC class I expression observed in MCC cells cultured with CD4+ T cells is mediated by CD4+ T cells, possibly through the release of cytokines or other factors that inhibit MHC class I expression. These findings may have implications for the development of novel therapeutic strategies for MCC.

In vitro signaling in non-T and B cell population is important for suppression of interface dermatitis in mouse

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Skin commensals are known to play an important role in the modulation of skin immune responses. Recent studies have shown that skin commensals can induce tolerance to self-antigens, which is important for the maintenance of skin barrier function and the prevention of autoimmunity.

In this study, we investigated the role of skin commensals in the suppression of interface dermatitis in a murine model of colitis. We found that skin commensals, specifically S. epidermidis, were able to induce tolerance to self-antigens, which was associated with reduced inflammation and a decrease in the number of inflammatory cells. These findings suggest that skin commensals play an important role in the suppression of interface dermatitis and may provide a new target for the treatment of this condition.

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