was set up identically except that LCs were pretreated with medium alone. (3) Cultures were cell death in MCC13 and UISO (p<0.003) and 3.6-, 1.6-, and 2.8-fold CD8+ T-cell expansion, respectively, as well as significant cancer infection. Without nanoparticle-based reprogramming, co-culture of MCC13 and UISO with MHC-I expression (mean fluorescence intensity) in MCC13 and UISO 3 days after transfection was demonstrated low baseline MHC-I expression in 2 of the 3 cell lines (MCC13 and UISO), with ICI resistance in MCC in particular and cancer immune evasion in general is downregulation of tumor MHC class I (MHC-I) expression, thereby limiting cytotoxic cellular immune response. Assessment of three patient-derived MCC cell lines, MCC13, MCC26, and UISO, demonstrated low baseline MHC-I expression in 2 of the 3 cell lines (MCC11 and UISO), with higher expression in MCC26. We used biodegradable polymeric nanoparticles based on poly-beta-amino ester to co-deliver DNA plasmids encoding a co-stimulator molecule (4-1BB) and a CD40 ligand (4-1BBL) to co-stimulate the cDC1 by IL-6 pre- activation of the microparticles showed some 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

Merkel cell carcinoma (MCC) is a rare but aggressive skin cancer with half of patients unresponsive to immunotherapeutic treatments. A primary resistance mechanism driving MCC resistance in MCC is IFNγ resistance in particular and cancer immune evasion in general is downregulation of tumor MHC class I (MHC-I) expression, thereby limiting cytotoxic cellular immune response. Assessment of three patient-derived MCC cell lines, MCC13, MCC26, and UISO, demonstrated low baseline MHC-I expression in 2 of the 3 cell lines (MCC11 and UISO), with higher expression in MCC26. We used biodegradable polymeric nanoparticles based on poly-beta-amino ester to co-deliver DNA plasmids encoding a co-stimulator molecule (4-1BB) and a CD40 ligand (4-1BBL) to co-stimulate the cDC1 by IL-6 pre-activation of the microparticles showed some 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 (IVGR) is a promising strategy to reprogram cancer cells and enhance their immunogenicity for immune targeting. However, the effects of IVGR on IBD-associated colitis have not been extensively investigated. Here, we aimed to evaluate the therapeutic potential of IVGR on IBD-associated colitis. We generated an IBD-associated colitis model using Rag2−/− mice and administered in vivo treatment with CD40L-expressing tumors. We observed significant reduction in colitis severity and improved immune cell infiltration in IVGR-treated mice compared to controls. These results suggest that IVGR could be a promising strategy for treating IBD-associated colitis.

Proprotein convertase subtilisin/kexin type 9 (PCSK9) is a novel psoriasis susceptibility locus

Psoriasis is an inflammatory disease that is characterized by erythematous plaques with silvery scales. It is a common autoimmune disease affecting 3% of the global population. The pathogenesis of psoriasis is complex and involves interactions between genetic and environmental factors. One significant genetic factor identified in psoriasis susceptibility is the proprotein convertase subtilisin/kexin type 9 (PCSK9).

Opin expression in human Langerhans cell-like cell line, ELD-1

Opin expression in human Langerhans cell-like cell line, ELD-1

PCSK9 is a novel psoriasis susceptibility locus

Proprotein convertase subtilisin/kexin type 9 (PCSK9) is a novel psoriasis susceptibility locus

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