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persistent group were higher than that of sham control at 12th week, while the concentrations of IL-2 were lower compared with antigen transient stimulation control. Following IL-2 treatment, the concentrations of IL-2 in BM increased while IFN-γ got declined. IL-2 treatment could restore the expression levels of those transcription factors and the proliferation of LK cells. Our results indicated that *M. tuberculosis* antigen persistent stimulation decreased the proliferating activity of LK cells, promoted myelopoietic differentiation, and repressed lymphopoietic differentiation as a consequence of elevated IFN-γ production. IL-2 complementation contributed to maintaining the homeostasis of hemopoiesis.

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Understanding the tumor immune escape and the immune modulatory function of cancer stem cells

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Cancer therapy is experiencing a paradigm shift due to the clinical success of immune checkpoint inhibitors (ICI), yet efficacy remains limited to a few tumor types and a minority of patients. In an increasing number of cancers, tumor populations called cancer stem cells (CSCs) or tumor-initiating cells (TIC) have been defined. Strong evidence is supporting the immunoresistance feature of CSCs. CSCs can also assist immunosuppression in the Tumor microenvironment (TME) through interaction with other tumor stroma cells. Understanding the mechanisms leading to the immunotolerance of CSCs will be essential for treatment of immune-resistant and metastatic tumors. The identification of such mechanisms faces challenges when applying the widely used transplanted tumor models since they fail to recapitulate the tumor microenvironment as it develops during the progression of human tumors. We previously generated a transgenic model of autochthonous melanoma, named TiRP, which express murine MAGE-type antigen P1A, and can be used as a more faithful preclinical model for immunotherapy. In this model, the CSCs and CTCs are immune resistant. They are devoid of surface MHC I expression and are resistant to both antigen-specific T-cells- and NK-cells-mediated killing. In contrast to non-CSC tumor clones from the TiRP tumor (T429.11), CSCs/CTCs, when transplanted into recipient mice, generate a highly immunosuppressive TME with high numbers of PMN- and Monocytic-MDSCs, and are resistant to immune therapy.

O323

Myeloid Response Score (MRS): a simple immune signature that reflects intratumoral myeloid contexture and predicts prognosis of hepatocellular carcinoma

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Myeloid cells, including monocytes/macrophages (Mo/Mφ), neutrophils, dendritic cells, and myeloid-derived suppressor cells (MDSCs), can marshal divergent immune responses according to different pathological conditions. In cancer, the myeloid immune components play important roles in disease progression as well as in therapy, suggesting that appraising the overall and exact intratumoral myeloid response can provide valuable information that may help to predict cancer prognosis and tailor more precise therapeutic strategies. In this study, we constructed Myeloid Response Score

