Name: Ke Wang
Thesis title: The Role of JunB in Exhausted CD8 T Cell Populations in Tumors

Research aim:

To understand the involvement of JunB in the regulation of the maintenance of Tpex cells and the Tpex to Ttex transition under tumor challenge.

Material and method:

Materials: $JunB^{flox}$ $CD4^{Cre}$ mice, $JunB^{flox}$ $CD4^{Cre}$ OT-I mice, dTAG-JunB mice, dTAG-JunB OT-I mice, B6SJL mice, OT-I mice. Flag-JunB retrovirus. B16-OVA tumor cells.
Methods: Inoculation of B16-OVA to T cell Junb-deficient mice or mice transferred with Junb-deficient CD8 T cells or mice transferred with activated CD8 T cells with JunB overexpression. Co-culture of dTAG-JunB OT-I cells with tumor-infiltrated DCs.

Result:

Junb-deficient mice receiving tumor challenge had uncontrolled tumor growth due to lack of tumor-infiltrated CD8 T cells. Junb-deficient Tpex cells showed decreased Ly108 expression while increased expression of Tim-3. Junb-deficient Tpex cells also had more closed chromatin structure of key Tpex-related genes including *Myb*. Ttex population lacking JunB expression showed strikingly elevated cell death and deficiency on cytokines expression. Importantly, retrovirus induced JunB overexpression could accelerate intratumoral tumor-specific CD8 T cells number thus enhancing anti-tumor ability.

Conclusion:

JunB is necessary for the maintenance of Tpex phenotype and governing the survival and cytokines expression of Ttex population. And JunB may be a target to improve CD8 T cell-dependent cancer therapy.