**Introduction**

- Vδ2 T cells is the specific subset of human γδ T cells (a minor subpopulation of T lymphocytes) enriched in the peripheral blood of normal adults where they represent 1-2% of total T lymphocytes.
- γδ T cells have the particularity to be a link between the innate and the adaptive immunity. In fact, as γδ T cells gd T lymphocytes express gd TCR (T-cell receptor), however, their activation are MHC-independent.
- Recently, several studies demonstrated high anti-tumor potential of human Vδ2 T cells in vitro expanded from the peripheral blood of both healthy donors and different cancer patients. In particular, Zoledronate is a molecule which blocks the mevalonate pathway leading to an increase of phosphoantigen from stimulated lymphocytes that bind and activate Vδ2 TCR.
- Therefore, the aim of the project is to study the susceptibility of different Vδ2 T cell subsets to apoptosis upon *in vitro* stimulation with IL-2 and Zoledronate in order to better understand the activating and inhibitory phenotype of Vδ2 T cells.
- Thus, we want to have more informations about the impact of IL-2 and Zoledronate stimulation on the Vδ2 T cell apoptosis in correlation with their activating and inhibitory phenotype.

**Methods**

We use peripheral blood mononuclear cells (PBMCs) obtained from the blood of healthy donors, enriched with T lymphocytes.

**Results**

- 1) Comparison of the apoptosis of non-stimulated and stimulated Vδ2 T cells.
- 2) Comparison of the apoptosis of non-stimulated Vδ2 T cells and stimulated Vδ2 T cells.
- Activating phenotype
- Inhibitory phenotype
- Day 0 (non-stimulated cells)
- Day 1 (stimulated cells)

**Conclusion**

- IL-2 and Zoledronate stimulation leads to an increase of Vδ2 T cells apoptosis.
- Inhibitory or activating phenotype have no differences on apoptosis induced by IL-2/Zoledronate
- The next step will be the use of the specific IL-2/phosphoantigen to stimulate Vδ2 TCR instead of IL-2/Zol to avoid non-specific effects of mevalonate pathway inhibition on Vδ2 TCR stimulation.