From May 10th to May 13th, I attended the 8th International Congress on Autoimmunity in Granada, Spain. This congress has as goal to update physicians, immunologists, rheumatologists, researchers and clinicians interested in autoimmune diseases with the latest available diagnostic tools and new therapeutic avenues. As a consequence, this meeting brings together a broad range of international experts in different fields of autoimmunity. Each day started with a plenary session, in which 4 oral presentations were given. After that, 3 parallel sessions were planned each day. In between, there was time to visit posters. Since this is too much to describe in detail, I will only focus on the highlights of this meeting.

The first plenary session started with a lecture from A.K. Abbas, giving an introduction on the effector/regulatory T cell balance. In this lecture, the dual role of IL-2 was discussed. IL-2 is mainly known for its role to drive clonal expansion of effector T cells in response to antigen. However, IL-2 knockout actually leads to autoimmunity in a mouse model. It was postulated that different subtypes of cytokines exist, of which IL-2, so-called superkines, each stimulating a specific lymphocyte population. The second lecture was from P. Youinou. In this lecture, the role of B cells in autoimmunity was discussed. Special attention was given to regulatory roles of B cells. CD19<sup>IgD</sup><sup>low</sup>CD38<sup>low</sup>CD24<sup>low</sup>CD27<sup>-</sup>B cells were shown to impair DC function by either cell-cell contact or by soluble IL-12 production. In addition, B cells can also regulate T cell polarization, either by cell-cell contact (CD86) or by soluble IL-10.

On Friday, one of the parallel sessions dealt with B cells in autoimmunity, starting with a keynote lecture from E. Toubi. Human regulatory B cells are a source of the inhibitory molecules IL-10 and TGF-β. Although no specific marker is known for Breg, they defined Breg were as CD19<sup>+</sup>CD25<sup>high</sup>CD86<sup>high</sup>CD1d<sup>high</sup>IL-10<sup>high</sup>TGF-β<sup>high</sup>. It was shown that this subpopulation of B cells decreased significantly the proliferative capacity of CD4<sup>+</sup>T cells. In addition, FoxP3 and CTLA-4 expression were enhanced after co-culture with stimulated Breg.

On Sunday, a plenary session on vitamin D was scheduled. In this session I had to give myself an oral presentation about the association between vitamin D and depression and fatigue in Multiple Sclerosis patients. It was a great opportunity to present my work to a broad international audience. Therefore I would like to thank again the NVVI for the financial support giving me the opportunity to visit this congress. The 8<sup>th</sup> International meeting on Autoimmunity was a very nice meeting, with a lot of good international speakers in a very pleasant city. I ended this meeting with some interesting suggestions for future research.