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Report NVVI Travel Grant
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Keystone symposium "**B cell development and function**" February 10-15 2013
Keystone, Colorado

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The keystone symposium on B cell development and function is a biannual meeting that was held this year in Keystone Colorado from February 10-15. This year the symposium was joined with the HIV vaccines symposium, allowing participants to attend sessions in both meetings. Moreover, there were also 3 joint sessions of the two meetings that dealt with topics concerning both B cell activity and its implications for HIV vaccine development strategies.

In the morning and evening of each day plenary sessions were organized in which experts in the field showed their most recent data. In the afternoon parallel workshops were held in which mostly postdocs showed their recent, often unpublished, results. After dinner, poster sessions were organized in which workshop presenters but also others that were not selected for workshops showed their data. These poster sessions were really well attended and interactive and I really enyojed them since it allowed you to informally discuss results, also with people of whom you had attended the talk. On Wednesday evening I also had a poster presentation; many people came by to dicuss and gave suggestions and it was really nice to discuss my own data with experts within my own field.

For my own work, in which I study regulation of plasma cell differentiation by FOXP1, the workshop on plasma cell and memory B cell responses was very interesting. In this workshop, several new regulators of plasma cell differentiation were discussed, like zbtb20 (promotes plasma cell differentiation) and TSC1 (deletion promotes plasma cell differentiation).

One of my other interests is the difference between IgG^+ and IgM^+ B cells.
There were also several talks and poster presentations regarding this issue. Susan Pierce showed that IgG BCRs show enhanced signaling due to a motif in their cytoplasmic tail to which sap97 binds. Niklas Engels, on the other hand showed that differences in IgM^+ and IgG^+ B cell signaling might indeed be due to the cytoplasmic tail but he ascribed this to another motif to which grb2 can bind. He did not find effects when mutating the SAP97 binding site. Tomohiro Kurosaki showed that differences between IgG^+ and IgM^+ B cell differentiation might also rely on differences in the expression of transcription factors between these two B cell subsets and might not (only) be dependent on differences in the BCR.

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