31st EWRR Meeting in Amsterdam

The 31st EWRR Meeting in Amsterdam was an international workshop of about 300 scientists, focused on cutting-edge rheumatology research. Some of the invited speakers came from other research areas such as diabetes and multiple myeloma, inspiring innovative thinking and interdisciplinary collaborations. Interesting to me were the following aspects.

One exciting project presented is the free access human protein atlas to study cells, tissues and organs in a disease prespective. The first draft of the **human Proteome** will be completed by 2015.

Thousand of papers are published per year on biomarkers. High attention was given on correctly selecting **types of biomarkers of disease**:
- Diagnostic markers should have high specificity, enable detection of specific disease through blood/body fluid sampling (early detection) or tissue sampling (synovial biopsies, importance of the joint sampled, differential diagnostics).
- Prognostic markers should correlate with patient outcome, enable stratification of high versus low risk patients and are a guide for patient information and monitoring.
- Predictive markers are important for specific therapy response, enable stratification of responders versus non-responders and guide selection of therapeutic regime.

A Meta Analysis identified shared loci in **coeliac disease and rheumatoid arthritis.** Evolving areas of research are genome wide associations and SNPs analysis. Careful interpretation of overlaping genetics and epidemiology of diseases is key in chosing clinically relevant therapeutic applications. Validation prospective cohort studies are necessary to prove new concepts.

Another interesting session was the one toward **novel therapeutic targets**. For instance the work presented by my group on PKC412 as a FLT3 inhibitor in in vitro osteoclastogenesis was complementary to another group's work where the FLT3 receptor was found to be significantly overexpressed in rheumatoid arhritis patients, especially in those with high disease activity. This findings are suggestive that the FLT3/CD135 pathway may be implicated in the disease course of rheumatoid arthritis.

The role of **B cells** in rheumatoid arthritis remains unclear. B cell depletion therapies may effectively treat T cell mediated autoimmune diseases. The role of B cells in altering **dendritic cell**-mediated priming of TH1 or **TH17** responses is under investigation. Rheumatoid arthritis patients treated with B cell depleting-Rituximab revealed gene expression changes on the synovial tissue level. Lymphatic migration, recruitment and differentiation are investigated using dynamic contrast MRI scans to find differences with changing disease activity.

Major emphasis was given on the role of **synovial fibroblasts** in chronic inflammation, especially in rheumatoid arthritis. Multiple myeloma was used

as a paradigm to show the role of stromal cells and fibrosis. Molecular profiling from
patient synovial biopsies is being employed to identify key effector molecules.

Finally, the **TNF-trangenic** model remains a key experimental arthritis model for hypothesis testing in the lab.

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