REPORT
Society for Investigative Dermatology Annual Meeting, Scottsdale, AZ
11 – 14.05.16

Dr Richard Woolf
NIHR Academic Clinical Lecturer in Dermatology, King's College London

I am very grateful to the British Association of Dermatologists and National Institute for Health Research for jointly supporting my attendance at the Society for Investigative Dermatology Annual Meeting 2016, which was held in Scottsdale, Arizona. It was a well-organised meeting, attended by over 1300 delegates, held over four days, with a mix of plenary sessions, poster sessions and more specialised parallel mini-symposia. In addition to the core programme there were lunchtime and evening sessions organised by special interest groups.

At the meeting, my abstract had been selected for a presentation at the Innate Immunology mini-symposium. This was great exposure for my work, which I had completed during my PhD. This focused on human skin-resident γδ T cells, which are a small and poorly understood subset of lymphocytes in human tissues. γδ T cells had only been discussed a couple of times earlier in the meeting, and in these discussions they were largely disregarded as a 'mouse skin phenomena' as they are much more numerous in murine tissues. So, at my talk, there seemed genuine interest in that we had sought to address their potential in human skin biology, and had found that they had distinct innate-like potential. This lead to some interesting questions from senior investigators, such as; in what physiological context do we hypothesise that γδ T cells may proliferate in the way that we see them respond *in vitro* to culture with IL2 and IL15; if these cells activate independently of the TCR, what do we think the TCR is reactive to; do we see any evidence of early recruitment of γδ T cells to neonatal/child skin as is seen in murine skin; and, do we see localisation of Vδ1 and Vδ3 cells to certain anatomical sites within the skin i.e. epidermis vs. dermis/appendageal structures, as in murine skin? In addition to my oral presentation, I had a poster summarising this work, and had further discussions at that session. For me, these were highly constructive experiences, as we are currently writing a manuscript on this work, so I appreciated the early peer review. It was good to get positive feedback regarding the work, and this was useful as I look to develop further questions to build on this work.

Specifically regarding my interest in innate lymphocytes, and more specifically T cell biology, not much else was presented. A group from Manchester/Italy presented work on human Vδ1 ($\gamma\delta^+$) T cells in alopecia areata. Using histological specimens, they demonstrated that these cells are present around healthy scalp hair follicles at different stages of the hair cycle. In addition, during alopecia areata disease, Vδ1 T cells are found within the inflammatory infiltrate and they presented some functional work showing their cytotoxic potential towards the hair follicle. Therefore, they hypothesised that such lymphocytes may contribute to early loss of immune privilege within the hair follicle. The only other γδ T cells project that I saw presented was from Yasmin Belkaids group at the NIH, looking at the skin-gut-microbiome axis in mice. Interestingly, they showed that in germ-free conditions, the imiquimod psoriasis model has a very mild phenotype; however, when the gut was colonised with gut-specific flora the skin phenotype became much more severe/as published. They showed that this was due Vγ4 T cells in the skin, indicating that these cells had migrated to the skin following activation in the gut. This is interesting, as murine Vγ4 T cells seem to behave more like human Vδ2 T cells, which are predominantly found in the blood, and have been previously implicated in psoriasis.

More broadly, there were many additional abstracts presented looking at adaptive αβ T cell biology in skin pathology. This area seems dominated, at least in the U.S. dermatology community, by the work done by both Thomas Kupper and Rachael Clark at Harvard medical school. They presented several studies in which they are now routinely using high throughput sequencing of TCRβ CD3 regions to follow clonal T cell populations following challenge. Using this approach in mouse skin, for example, they demonstrated that in a graft-versus-host disease model donor T cells persist within the graft and may be key in mediating subsequent pathological inflammation. In a human study, they demonstrated the presence of what may be oligoclonal T cell populations in psoriasis. This arises the question of what these cells are reactive to, and whether the development of such oligoclonal populations is a disease-initiating event. It would be interesting to apply such an approach to other T cell populations, namely γδ T cell subsets that we have studied, to understand their biology better.

So, in conclusion, I was really happy to have attended the Society for Investigative Dermatology Annual Meeting. It was interesting, with a diverse range of high quality data presented, and I had an invaluable opportunity to both present and discuss my work. I am very grateful to the British Association of Dermatologists and National Institute for Health Research for financially supporting me in attending this meeting.