I would like to thank UKMF for supporting my participation in the 2012 ASH meeting in Atlanta with a Travel Award. I had the opportunity to communicate my work and attend very interested developments in biology and clinical aspects of myeloma.

From this meeting, I would highlight the Ad Hoc Scientific Committee in Plasma Cell Neoplasia, where the epigenome in the context of multiple myeloma was discussed. The session was opened with a state-of-the-art presentation by Dr P Jones, on chromatin structure and epigenetic processes, with emphasis on the methylation of CpG islands in transcriptional start sites of tumour-suppressor genes and the changes in the nucleosome positioning, which affect activation of gene expression. Dr J Licht discussed the role of MMSET overexpression, as a result of t(4;14), a cytogenetic event present in about 15% of the myeloma patients. MMSET, a histone methyltransferase, among other functions induces methylation in H3K36 and demethylasion of H3K27 and is linked to changes of expression levels in approximately 1000 genes in myeloma. New mechanisms of gene expression regulation are proposed, involving EZH2 and displacement of the Polycomb complex. Dr J Bardner discussed the therapeutic potential of a novel class of epigenetic modulators in myeloma. Epigenetic "readers" such as the BET family of bromodomain-containing transcriptional regulators are critical in the recognition of histone acetylation and initiation of transcription. JQ1 is a small molecule inhibitor of the BRD4 member of the BET family, resulting in downregulation of *c-Myc* expression. This provides the rationale for testing JQ1 or similar molecules in MYC-dependent haematological malignancies, including myeloma. BET bromodomain inhibitors are promising, novel agents, and likely to represent a new class of anti-myeloma drugs in the near future, next to proteasome inhibitors and immunomodulatory drugs.

In the clinical field, a number of trials highlighted the transition of the previously investigational drugs pomalidomide and carlfizomib into routine clinical practice. Among those, in a late-breaking abstract Prof M Dimopoulos presented the results of pomalidomide plus low-dose dexamethasone in patients with refractory or relapsed disease after bortezomib and lenalidomide. There is significant increase in both PFS and OS, suggesting that this combination may become the standard of care in a group of patients previously offered palliative care only, in most cases. Also, a trend towards the use of lower dexamethasone dose is proposed to avoid toxicity. Carfilzomib in combination with other agents could be used not only in relapsed disease but also as first-line treatment of myeloma, taking advance of the better toxicity profile of this new drug compared to bortezomib. Dr J Shah reported response in over two thirds of heavily-treated relapsed disease with carfilzomib-pomalidomide-dexamethasone combination, despite poor cytogenetics profiles, including 17p deletion. In would be very interesting in the future to see more works tailoring treatment to the biological profile.

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