Report on the EMBO workshop on RUNX transcription factors in development and disease, held in Oxford on 16-19 August 2009

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This workshop was a great success. All participants were unanimously positive in their questionnaire responses (see attachment). The scientific program, including the poster session was very highly rated as was the size and duration of the meeting and the time available for networking and socializing. The location was also greatly enjoyed, and the concentrated conference location gave ample opportunity for networking and discussions.

The sessions of RUNX proteins in cell fate decisions revealed that Runx function is increasingly found to be context specific, i.e. subject to the specific cell lineage and developmental stage, as well as the received environmental stimuli. An example of this is the critical requirement for Runx1 in hematopoietic stem cell (HSC) generation, but not for the maintenance of adult HSCs. These insights have arisen partly from the analysis of conditional knock out and rescue mouse models. These models also increasingly reveal non-redundant roles for Runx proteins outside of the organ systems in which they were originally identified. A good example of this is a novel role for Runx1 in bone development in the sternum, but not in other bones. Thus, the family of Runx proteins appears to work together in many organ systems (e.g. Runx1 and Runx2 in bone, Runx1, Runx2 and Runx3 in blood), adding complexity to the original assignment of Runx1 to blood, Runx2 to bone and Runx3 to neural tissues. New and exiting fields of research are the roles of Runx proteins in mammary gland formation and its pre-neoplastic changes, and in the etiology of inflammatory bowel disease, which appears to be leukocyte driven.

Studies in lower model organisms continue to provide information on the how RUNX proteins affect basic cellular biology such as growth and survival. In addition, the phylogeny of RUNX genes is being studied in order to identify and study the most basic and conserved transcriptional network downstream of RUNX genes. Once identified, these networks may shed light on what goes awry with the basic cell machinery in human diseases such as cancer. Several studies reported on new cellular pathways in which Runx proteins are involved, such as ties with the Wnt pathway, MDM2 ubiquitination, necdin and quiescence, shingolipid metabolism and NFkB. New co-factors of Runx proteins were also reported, as well as specific mechanisms by which it may regulation transcription of downstream target genes.

Exciting developments in the leukemia field were the identification of new collaborating genes and mutations, and the development of inhibitors of leukemic fusion proteins. Interestingly, a screen in drosophila identified new modulators of AML-ETO, potentially opening the possibility for new avenues of research in AML therapy.

Part of the outcomes of this meeting will be published in Blood Cells, Molecules and Diseases early next year as individual and collaborative contributions. For example, one such collaborative paper will focus on clarifying Runx gene terminology to the many investigators new to this gene family. Finally, the enthusiasm for RUNX workshops is still strong and three future meetings have been planned, in Tokyo (2010), California (2011) and Montreal (2012).