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LABORATORY OF

HAEMATOONCOLOGY

haematopoiesis, leukaemia, haematopoietic stem cells, transcription factors

All blood cells are derived from a small population of cells called haematopoietic stem cells (HSC). HSC reside in the bone marrow, are pluripotent and can differentiate into all haematopoietic lineages. The processes of HSC maintenance, differentiation, and proliferation are tightly regulated and defects in their regulation are associated with haematopoietic disorders, such as acute myeloid leukaemia (AML). AML is a malignant haematopoietic disease that represents over 90 % of acute leukaemias in adults, and is characterized by an accumulation of immature and non-functional blood cells. AML originates from a single transformed cell, which progressively acquires additional genetic and epigenetic defects and gives rise to leukemic stem cells (LSC). The compilation of aberrations in LSC alters their normal haematopoietic program, giving rise to full-blown leukaemias. As conventional AML therapies are not efficient in eradicating the LSC, better understanding of the mechanisms regulating stem cell fate will be critical to cure this disease. In the laboratory of Haematooncology, we investigate the molecular mechanisms that control HSC and LSC. We use C/EBPα, a key transcription factor regulating HSC maintenance and myeloid differentiation, as a model to identify critical regulators in the stem cell fate. Using well-defined HSC populations and lentiviral gene transfer we investigate the role of the identified genes. We perform in vitro assays (including gene expression profiling and chromatin immunoprecipitation) and in vivo experiments (generation and analysis of murine AML models) to elucidate novel mechanisms and pathways critical for HSC fate, and determine their contribution to leukaemogenesis. Ultimately, our work will contribute to establishing knowledge for the development of better AML therapies.

Selected recent papers:

Polina Zjablovskaja, Miroslava Kardosova, Petr Danek, Pavla Angelisova, Touati Benoukraf, Tomas Kalina, Martin Balastik, Ruud Delwel, Tomas Brdicka, Daniel G. Tenen, Fréderic Fiore, Bernard Malissen,Vaclav Horejsi, and Meritxell Alberich-Jorda: EVI2B is a C/EBPα target gene required for granulocytic differentiation and functionality of hematopoietic progenitors. Submitted.

Alexander Arthur Wurm, Polina Zjablovskaja, Miroslava Kardosova, Dennis Gerloff, Daniela Bråuer-Hartmann, Christiane Katzerke, Jens-Uwe Hartmann, Stephan Fricke, Nadja Hilger, Anne-Marie Müller, Marius Bill, Daniel G. Tenen, Dietger Niederwieser, Meritxell Alberich-Jorda, Gerhard Behre. Disturbance of the C/EBPα-miR-182 balance impairs granulocytic differentiation and promotes development of acute myeloid leukemia. Submitted.

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