CEBPA IN NORMAL BLOOD CELL DEVELOPMENT AND IN MYELOID MALIGNANCIES

(1) The human *CEBPA* locus is organized into regulatory modules that comprises at least fourteen tissue-specific enhancers (this thesis).

(2) A single enhancer located at +42kb of the *CEBPA* gene acts as an autonomous switch to activate *CEBPA* and prime myeloid differentiation (this thesis).

(3) A non-cell autonomous mechanism reduces hematopoietic stem cell numbers in neutropenic bone marrows of *Cebpa*-enhancer deleted mice (this thesis).

(4) Oncoproteins compromise *CEBPA* transcription in AML by reversing the active chromatin state and topology of the +42kb enhancer in myeloid progenitors (this thesis).

(5) The *CEBPA* locus serves as a paradigm to study transcriptional control in cell lineage differentiation and disease.

(6) The concept of topological associated domains in nuclear organization is a definition of chromatin architecture rather than a technical artefact.

(7) The advent of the CRISPR/Cas9 genome editing technology has superseded conventional tools to generate *in vitro* and *in vivo* models in a cheaper and less time-consuming manner.

(8) Bone marrow heterogeneity limits the study of rare cell populations, which is circumvented by the advent of single-cell genome wide technologies.

(9) The application of single-cell technology combined with lineage tracing experiments in myelopoiesis is diverging the concept of branching hematopoiesis to unilineage hematopoiesis.

(10) Progress in science seeks to combine multi-disciplinary knowledge from physics, engineering and biology to develop technologies and answer biological questions.

(11) Every great and deep difficulty bears in itself its own solution. It forces us to change our thinking in order to find it (Niels Bohr).

Roberto Avellino
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