Enhancer hijacking by EVI1 in acute myeloid leukemia

1) The group of AML patients with EVI1 oncogene deregulation caused by 3q-aberrations and hijacking of various enhancers should be considered as a single AML entity (this Thesis).

2) Oncogenic EVI1 over-expression in t(3;8)(q26;q24) AML is driven by the hijacking of and activation by a MYC super-enhancer (this Thesis).

3) CRISPR-Cas9 genome editing technology has greatly enhanced and simplified molecular research.

4) A patient based translocation can ‘simply’ be derived in vitro by cutting two chromosomes using CRISPR-Cas9 technology (this Thesis).

5) Super-enhancers are regulatory regions consisting of multiple enhancer modules that are involved in complex chromatin interactions with promoters, together regulating highly organized gene expression (this Thesis, Whyte et al., Cell 2013).

6) A CTCF bound enhancer-docking site facilitates enhancer hijacking by oncogenes such as MYC and EVI1 and may serve as a common vulnerability in cancer (this Thesis, Schuijers et al., Cell reports 2018).

7) Mechanisms of gene regulation and chromosomal organisation of malignant cells do not necessarily differ from normal cells, however mutations can lead to misuse of the system.

8) Unusual cases often shed light on the biology in health and disease.

9) Science cannot be performed without teamwork and valuable collaborations.

10) Scientific research should be driven by curiosity rather than by the need to publish or get funding.

11) Stay away from negative people, they have a problem for every solution (Albert Einstein).

Sophie Ottema
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