Stellingen behorende bij het proefschrift

Studies on Genetic Aberrations in Acute Myeloid Leukemia

1. The gene encoding the B-cell CLL/Lymphoma 11B acts as an oncogene in acute myeloid leukemia (this thesis).

2. The fact that mutations that impair the proper splicing of exons 8 of Cas-Br-M (murine) ecotropic retroviral transforming sequence gene (*CBL*) are exclusively associated with core binding factor leukemias suggests that *CBL* mutations contribute to the pathogenesis of this type of leukemia (this thesis).

3. In acute myeloid leukemia nonsense-mediated RNA decay is responsible for degradation of mutated transcripts of the Wilms Tumor 1 oncogene as they often carry premature termination codons (this thesis).

4. Mutation analysis of genes containing premature termination codons should not be performed using RNA but genomic DNA (this thesis).

5. Somatic mutations in the isocitrate dehydrogenase genes have an only moderate prognostic value in acute myeloid leukemia (this thesis).

6. Mutations in the isocitrate dehydrogenase genes are present in pre-leukemic conditions suggesting that isocitrate dehydrogenase gene mutations represent early transformation events in these diseases (this thesis).

7. How to distinguish driver mutations that determine malignant transformation from passenger alterations that coincidently arise and accumulate in the malignant clone will be one of the major challenges of genome-wide profiling of gene mutations in cancer.

8. The plausible explanation for the numerous gene aberrations in acute lymphoblastic leukemia and the relative lack of such mutations in acute myeloid leukemia relates to the fact that precursor B and T-cells are prone to the physiological process of receptor rearrangements.

9. "That's the nature of research - you don't know what in hell you're doing". Harold Edgerton

10. A rock is heavier in its own resting place. A Kurdish proverb

11. "Wise men talk because they have something to say; fools, because they have to say something". Plato

Saman Abbas
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