Identifying aneuploidy-tolerating genes

Simon, Judith Elisabeth

Stellingen behorende bij het proefschrift

IDENTIYING ANEUPLOIDY-TOLERATING GENES

1. Aneuploidy alone does not lead to tumorigenesis in the mouse, but it is an accelerator for tumour growth in a predisposed background. *(This thesis)*

2. The fact that aneuploidy combined with p53 deficiency leads to aggressive T-ALL with recurrent karyotypes indicates that cancer does not suffer from disorganised, but rather organised chromosomal chaos. (*This thesis and Siddhartha Mukherjee*)

3. PRMT5 accumulation in cytoplasmic granules increases upon aneuploidy in untransformed cells, suggesting that PRMT5 specifically responds to aneuploidy-induced stress. *(This thesis)*

4. PRMT5 is a potential target for the treatment of aneuploid cancer, as it functions as a sensor for methionine to activate mTORC1, which is essential for aneuploid tumour cell growth and metabolism. (*This thesis*)

5. Transposon mutagenesis and Mad2 activation in the haematopoietic system of mice is a powerful method for the identification of aneuploidy-tolerating genes. *(This thesis)*

6. In addition to studying how the cell itself responds to mitotic defects, it should be considered how cells cooperate to promote the growth of 'less-fit' subclones within a tumour. *(e.g. Marusyk et al., 2014)*

7. "The impediment to action advances action. What stands in the way becomes the way" - *Marcus Aurelius*

8. "You will reap what you sow" - *Galatians 6:7*

9. "Zeg wat je denkt, doe wat je zegt" – *Jack Henri Simon*

Judith Elisabeth Simon, september 2018