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Stellingen
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Discovery of genetic defects in unexplained colorectal cancer syndromes

1. During tumor formation, MMR-deficiency can develop secondary to a POLE hyper-mutated phenotype. (this thesis)

2. Intrinsic MMR variants are rarely the cause of Lynch Syndrome cancers. (this thesis)

3. Formalin fixation of cells enables the detection of aberrant RNA transcripts that would otherwise be subject to nonsense-mediated decay. (this thesis)

4. The presence of multiple adenomas can be explained by field cancerization, a mechanism in which a tumorigenic clone can spread throughout the colon. (this thesis)

5. The majority of variants in a cancer cell are a result of random DNA replication errors. (Most cancers arise from 'bad luck', Tomasetti, Li and Vogelstein, Nature, 2017).

6. Colorectal cancer is a heterogeneous disease, not a single entity.

7. Targeting DNA repair processes can trigger neoantigen generation, and has the potential to be exploited as a therapeutic approach. (Germano et al, Nature, 2017)

8. Early-onset tumors are clinically, pathologically and molecularly distinct from late-onset CRC. (Yeo et al, Clinical Colorectal Cancer, 2017)

9. Science achieves too little if it stays in the lab.

10. There is no absolute truth in science.

11. Newsworthiness does not always mean worthy science.

12. The pressure to publish promotes inferior science.

Anne Jansen, 2018