Stellingen

1- Endogenous stochastic DNA damage due to ERCC1 deficiency diminishes but not functionally exhausts the stem cell population of the small intestine *(this thesis)*.

2- DNA damage accumulation and not mutations is primarily responsible for adult stem cell and subsequently organ ageing phenotype *(this thesis)*.

3- Rapidly cycling intestinal stem cells are not efficient in NER-mediated repair, with many NER components downregulated compared to liver stem cells, which adeptly repair the same lesions *(this thesis)*.

4- Tissue and tissue-residing cells' self-renewal rates influence damage accumulation and ageing-related pathology *(this thesis)*.

5- The Translesion Synthesis (TLS) pathway synergizes with global genome NER to protect rapidly proliferating intestine from replication stress and mutagenesis (*this thesis)*.

6- A single stem/progenitor cell population can control systemic ageing in the mouse *(Zhang et al., Nature 2017)*.

7- The heterogeneity of the senescence phenotype(s) and the complexity of the (bio)markers that need to be observed simultaneously make the simple task of just examining this cell fate really challenging. In fact, few publications actually present all the necessary experimental evidence.

8- Regular exercise and a daily cup of coffee sound as the most reasonable anti-ageing intervention(s) so far *(Jurdana M., 2017, Furman et al., 2017, Vujic et al., 2018)*.

9- Adult Stem Cell (ASC)-derived organotypic cultures (organoids) are valuable tools to replace animal studies on (anti-) ageing research. Ironically, the organoid technology is made possible thanks to animal studies.

10- There are serious considerations regarding the social consequences of a significant prolongation of human lifespan.

11- 'Έχεις τα πινέλα, έχεις και τα χρώματα, ζωγράφισε τον παράδεισο και μπές μέσα / You have the brushes you have the colors, draw the paradise and get in – Nikos Kazatzakis.