Telomere shortening limits the proliferative capacity of human fibroblast to 50-75 cell divisions by inducing senescence or apoptosis. There is growing evidence that telomere shortening has an impact on human ageing, diseases and cancer: 
(i) Telomere shortening occurs in almost all human tissues during human ageing; 
(ii) Telomere shortening is accelerated in chronic human disease; (iii) Telomere shortening and telomerase reactivation occur in the vast majority of human cancers; (iv) Telomerase mutations are associated with a shortened lifespan, organ failure and increased cancer risk in humans.

Using telomerase knockout mice as a model system, we were able to show that telomere-shortening impairs the maintenance of the organ system with high rates of cell turnover, which is associated with a shortened lifespan. Besides, we were able to demonstrate that telomere shortening limits stress responses and organ regeneration in response to injury, it induces chromosomal instability as well as it increases the rate of cancer initiation while suppressing the progression of tumours.
We are currently analyzing the influence of ageing and telomere shortening on changes in cell-to-cell variation in the gene expression of stem cells and differentiated organ cells (PhD Project Sarah-Fee Katz). Furthermore, we are trying to identify target genes for telomerase activation in the crisis stage of hepatocarcinogenesis (PhD Project Daniel Hartmann). Regarding the role of p27, we are investigating its impact on maintaining stem cell function and organ homeostasis during ageing in the context of telomere dysfunction (PhD Project Parisa Eshraghi).

Selected Publications:


