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Non-cell autonomous roles of telomere shortening in zebrafish

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Background: Cancer incidence increases with age. Due to the end-replicative problem, telomeres shorten with each cell division throughout our lives. Telomere shortening has consequences beyond the cellular level. As we age, cells approach replicative senescence and DNA damage emanating by short telomeres initiate a cascade of events that expands to the extracellular environment. Senescent cells were shown to release a set of molecules including inflammatory cytokines, termed senescence-associated secretory phenotype (SASP). Indeed, an inflammatory environment generates a pro-tumorigenic environment that supports tumor invasiveness.

Aim of the study: Investigate the causal relation between short telomeres and cancer incidence. Determine whether this effect has a non-cell autonomous component by using zebrafish embryo chimeras and tumor transplants.

