LONG TELOMERE SYNDROME

Shortening of telomere with aging has been associated with shortening of lifespan itself. Maintenance of long telomeres, therefore, has been regarded as a cure for aging. Centenarians and their offspring have indeed been reported to maintain longer telomeres compared with controls[1]. The common notion is that critical telomere shortening, the consequent onset of telomeric DNA damage and cellular senescence are a general determinant of the life span of a species[2]. Furthermore, "Telomerase reactivation reverses tissue degeneration in aged telomerase-deficient mice"[3].

Things, however, are more complex, almost paradoxical. In neuroblastomas, the reactivation of telomerase or ALT (alternative lengthening of telomeres), point to a poor prognosis for the patient[4]. In line with this, DeBoy et al. [5] recently reported a familial *POT1* mutation causing excessively long telomeres, along with an inherited capacity to lengthen telomeres. The mutation "conferred a predisposition to a familial clonal hematopoiesis syndrome that was associated with a range of benign and malignant solid neoplasms. The risk of these phenotypes was mediated by extended cellular longevity and by the capacity to maintain telomeres over time".

1. https://www.pnas.org/doi/10.1073/pnas.0906191106?url_ver=Z39.88-2003&rfr_id=ori%3Arid%3Acrossref.org&rfr_dat=cr_pub++0pubmed
2. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6660761/
3. https://www.nature.com/articles/nature09603
4. https://www.science.org/doi/full/10.1126/science.aat6768
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