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Telomerase in brain and neurodegeneration

Telomerase is an enzyme that in its canonical function maintains telomeres, the ends of chromosomes in dividing cells that shorten their telomeres continuously. However, there are a number of telomere-independent functions known for the telomerase protein TERT (Telomerase Reverse Transcriptase). This includes the shuttling of the TERT protein from the nucleus to mitochondria where it decreases oxidative stress, apoptosis sensitivity and DNA damage. Recently, evidence has accumulated on a protective role of TERT in brain and postmitotic neurons. This function might be able to ameliorate the effects of toxic proteins such as amyloid-β, pathological tau and α-synuclein involved in neurodegenerative diseases such as Alzheimer's disease (AD) and Parkinson's disease (PD). Thus, these novel functions of TERT/telomerase in brain could have important implications for the treatment of neurodegenerative diseases. Mechanisms include changes in neuronal gene expression and synaptic functions together with the activation of signaling pathways as well as protein degradation pathways such as autophagy upon experimental increase of TERT levels in neurons and the brain of in vitro as well as in vivo models of neurodegenerative diseases such as Alzheimer's Disease (AD) and Parkinson's Disease (PD).

Recently, our group applied two telomerase activators (TA-65 and GRN510) on a mouse model of PD and found increased TERT expression levels, an improvement of PD-related symptoms such as gait and balance together with a significant decrease of total, phosphorylated and aggregated α-synuclein in these mice. We also showed the activation of autophagy as an important protein degradation process for toxic neuronal proteins by increasing TERT levels. These results highlight the non-canonical role of the telomerase protein TERT in brain and demonstrates its potential benefit for the amelioration of brain ageing and neurodegenerative diseases and might form the basis for the development of novel strategies and therapies against diseases such as AD and PD.

Biography

Dr. Gabriele Saretzki is a Lecturer at Newcastle University whose research focuses on the molecular biology of aging, cellular senescence, and age-related diseases. With expertise in biogerontology, she investigates the mechanisms that influence cellular lifespan, genomic stability, and the biological processes that contribute to healthy aging. Her work integrates molecular and cellular approaches to better understand the factors that drive aging and associated disorders.

Dr. Saretzki has made significant contributions to research on telomere biology, telomerase regulation, mitochondrial function, oxidative stress, and stem cell aging. Through her publications and collaborative research, she has advanced knowledge of the interplay between cellular metabolism and the aging process, with the long-term goal of identifying strategies to promote healthy longevity and develop interventions for age-related diseases.

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