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Keywords
Telomerase – ageing – neurons - stress

Science
Mammalian telomeres are highly organized structures at the end of chromosomes, made by an array of nucleotide sequences (TTAGGG)n and some DNA-binding proteins. One of these proteins is Telomerase, a ribonucleoprotein containing a small RNA and a catalytic subunit (Telomerase Reverse Transcriptase, TERT) which uses the RNA as a template for the elongation of telomeres.

In proliferative cells, the well-known role of Telomerase is to prevent telomere shortening and, thereby, the onset of replicative senescence, which are prerequisites for cellular immortality; on the other side, its function in post-mitotic neurons is almost obscure.

Previous experiments of TERT over-expression in both, pseudoneuronal cells PC12 and neuronal primary cultures, showed a clear anti-apoptotic role of the protein against oxidative stress independent from its role of telomere elongation: increased oxidative stress induces Telomerase exclusion from the nucleus and a shuttling of the enzyme to cytosol and mitochondria, where its non-canonical activity leads to an improved mitochondrial function.

On the other hand, it is becoming more and more evident that certain neurodegenerative diseases arise as consequence of abnormal stress-related apoptosis. It therefore becomes
essential to understand the mechanisms neurons put to work to sustain survival under stress and how their strength evolves during senescence.

I am studying the mechanism behind the pro-survival function of the cytosolic TERT to counteract stress-induced apoptosis in ageing neurons in culture and in vivo mouse model since neurodegenerative disease arise as a consequence of abnormal stress-related apoptosis.

Selected publications