Telomere shortening during replicative exhaustion

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15/05/2024

Introduction

Reactome is open-source, open access, manually curated and peer-reviewed pathway database. Pathway annotations are authored by expert biologists, in collaboration with Reactome editorial staff and cross-referenced to many bioinformatics databases. A system of evidence tracking ensures that all assertions are backed up by the primary literature. Reactome is used by clinicians, geneticists, genomics researchers, and molecular biologists to interpret the results of high-throughput experimental studies, by bioinformaticians seeking to develop novel algorithms for mining knowledge from genomic studies, and by systems biologists building predictive models of normal and disease variant pathways.

The development of Reactome is supported by grants from the US National Institutes of Health (P41 HG003751), University of Toronto (CFREF Medicine by Design), European Union (EU STRP, EMI-CD), and the European Molecular Biology Laboratory (EBI Industry program).

Literature references

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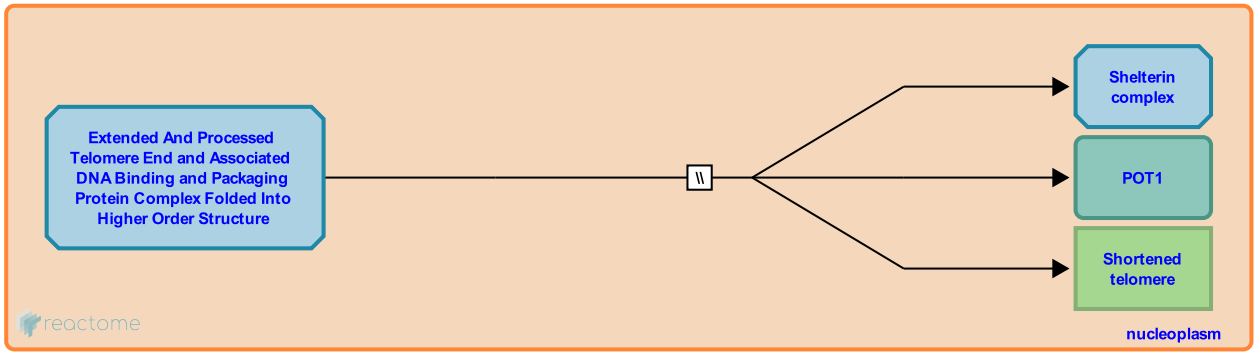
Stable identifier: R-HSA-3785711

Type: omitted

Compartments: nucleoplasm



In somatic cells where telomerase is not active, telomeric DNA shortens with each cell division (Harley et al. 1990, Hastie et al. 1990). This may be especially pronounced in cells undergoing replicative exhaustion due to oncogenic signaling-driven cell division. The shelterin complex, which protects telomeres from being recognized as double strand DNA breaks (reviewed by de Lange 2005), binds telomeric DNA through interaction of its subunits TREF1 (TRF1) and TREF2 (TRF2) with long TTAGGG repeat tracts (Smogorzewska et al. 2000). Telomere shortening due to replicative exhaustion results in a decreased number of TTAGGG repeats, which negatively impacts shelterin binding to telomeric DNA.

Literature references

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Editions

2013-07-15	Edited	D'Eustachio, P., Matthews, L.
2013-07-15	Authored	Orlic-Milacic, M.
2013-09-03	Reviewed	Samarajiwa, S.