Telomeres and telomerase in aging, disease, and cancer - molecular mechanisms of adult stem cell ageing

Springer - Cancer and ageing: convergent and divergent mechanisms

Description: -

Criminal behavior.

Crime -- Social aspects.

Telomerase -- physiology

Neoplasms -- genetics

Cell Aging -- genetics

Aging -- genetics

Adult Stem Cells

Telomere -- physiology

Cancer -- Molecular aspects

Telomerase

Telomere

Aging -- Molecular aspects Telomeres and telomerase in aging, disease, and cancer - molecular mechanisms of adult stem cell ageing

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Notes: Includes bibliographical references and index.

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Tags: #Telomeres #in #Disease

Telomere length, stem cells and aging

In all four mouse models, telomere dysfunction and the retention of a p53-dependent DNA damage response resulted in impaired growth of tumors. Physical activity appears to protect against metabolic and psychological stress and maintain TEL.

Pan

Thus, either progressive telomere shortening or acute telomere uncapping can lead to non-reciprocal translocations through a fusion-breakage mechanism.

Adult stem cells in aging, diseases and cancer

Short telomere syndromes have a predominant degenerative phenotype marked by organ failure that most commonly manifests as pulmonary fibrosis and are associated with a relatively low cancer incidence. Mo Agric Exp Res Stn Res Bull.

Telomerase, telomerase function, telomerase in cancer & aging

Using these neonatal SMCs, they were able to create reliable human vessels, and then it becomes possible to create coronary arteries for coronary artery bypass grafting. The bar plots show the recalibrated mutation frequencies after propensity score weighting.

Biological immortality

But what is the evidence that telomere dysfunction is a relevant mechanism driving epithelial cancers in humans? In November, 2019, the first telomere-lengthening gene therapy clinical trial has started, which aims to reverse aging by at least 20 years.

Cancer and ageing: convergent and divergent mechanisms

Furthermore, rare mutations in shelterin genes coding for the proteins that protect telomere structure can produce severe dyskeratosis but do not alter telomerase repair capacity. For instance, in a murine model of telomerase deficiency and accelerated telomere attrition, researchers found that certain intracellular pathways involved in mitochondrial function and glucose metabolism were deregulated, a common occurrence in aging individuals, ultimately causing heart muscle disease.

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