Role of telomere dysfunction in aging and its detection by biomarkers.

Abstract:
Aging is a complex process that has been shown to be linked to accumulation of DNA damage. Telomere shortening represents a cell-intrinsic mechanism leading to DNA damage accumulation and activation of DNA damage checkpoints in aging cells. Activation of DNA damage checkpoints in response to telomere dysfunction results in induction of cellular senescence—a permanent cell cycle arrest. Senescence represents a tumor suppressor mechanism protecting cells from evolution of genomic instability and transformation. As a drawback, telomere shortening may also limit tissue renewal and regenerative capacity of tissues in response to aging and chronic disease. In aged organs, telomere shortening may also increase the cancer risk by initiation of chromosomal instability, loss of proliferative competition of aging stem cells, and selection of aberrant growing clones. Consequently, aged individuals are more susceptible and vulnerable to various diseases and show an increased cancer risk. Recently, proteins were discovered, which are induced by telomere dysfunction and DNA damage. It was shown that these proteins represent new biomarkers of human aging and disease. Here, we review the scientific background and experimental data on these newly discovered biomarkers.
2009

Band: 87

Heft / Issue: 12

Seiten: 1165-71

Sprache: eng


Print-ISSN: 0946-2716

TUM Einrichtung: Chirurgiesche Klinik und Poliklinik

Occurrences:
- Einrichtungen > Fakultäten > Fakultät für Medizin > Kliniken und Institute > Chirurgische Klinik und Poliklinik > 2009

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