PublisherInfo		
PublisherName	:	BioMed Central
PublisherLocation	:	London
PublisherImprintName	:	BioMed Central

Senescence tale

ArticleInfo		
ArticleID	:	4436
ArticleDOI	:	10.1186/gb-spotlight-20020402-01
ArticleCitationID	:	spotlight-20020402-01
ArticleSequenceNumber	:	102
ArticleCategory	:	Research news
ArticleFirstPage	:	1
ArticleLastPage	:	2
ArticleHistory	:	RegistrationDate : 2002–4–2 OnlineDate : 2002–4–2
ArticleCopyright	:	BioMed Central Ltd2002
ArticleGrants	:	
ArticleContext	:	130593311

Jonathan B Weitzman
Email: jonathanweitzman@hotmail.com

Replicative senescence is associated with telomere shortening and the loss from the ends of chromosomes of about 100 bp per population doubling. In the March 19 Science, Jan Karlseder and researchers at Rockefeller University claim that the state of the ends, rather than telomere loss, determines the induction of senescence (*Science* 2002, **295**:2446-2449). They studied primary human fibroblasts expressing TRF2, a sequence-specific DNA-binding protein that binds to telomeric repeats. TRF2 overexpression caused accelerated telomere shortening, increasing the rate of loss to 165-181 bp per end per population doubling. TRF-dependent telomere shortening required cell division and was independent of the p53 or pRb pathways. TRF2-overexpressing cells did not exhibit premature senescence, but they continued to grow and underwent senescence with telomeres that were considerably shorter than control cultures. The elevated TRF2 levels caused a reduction in chromosomal-end fusions and chromosomal damage. The authors propose that TRF2 protects critically short telomeres.

References

1. Extension of life-span by introduction of telomerase into normal human cells.

2. *Science*, [http://www.sciencemag.org]

3. Rockefeller University , [http://www.rockefeller.edu]

4. Human telomeres contain two distinct Myb-related proteins, TRF1 and TRF2.