

The telomerase activator TA-65 elongates short telomeres and increases health span of adult/old mice without increasing cancer incidence.

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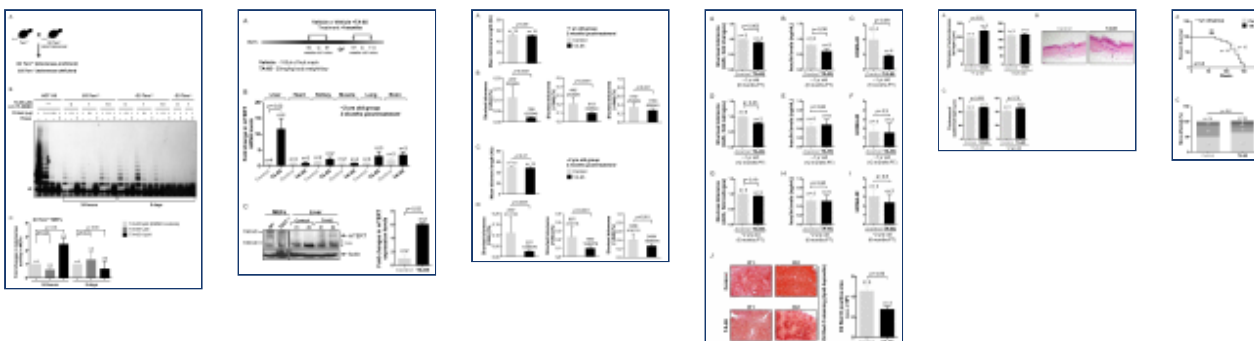
Abstract

Here, we show that a small-molecule activator of telomerase (TA-65) purified from the root of *Astragalus membranaceus* is capable of increasing average telomere length and decreasing the percentage of critically short telomeres and of DNA damage in haploinsufficient mouse embryonic fibroblasts (MEFs) that harbor critically short telomeres and a single copy of the telomerase RNA *Terc* gene (G3 *Terc*(+/-) MEFs). Importantly, TA-65 does not cause telomere elongation or rescue DNA damage in similarly treated telomerase-deficient G3 *Terc*(-/-) littermate MEFs. These results indicate that TA-65 treatment results in telomerase-dependent elongation of short telomeres and rescue of associated DNA damage, thus demonstrating that TA-65 mechanism of action is through the telomerase pathway. In addition, we demonstrate that TA-65 is capable of increasing mouse telomerase reverse transcriptase levels in some mouse tissues and elongating critically short telomeres when supplemented as part of a standard diet in mice. Finally, TA-65 dietary supplementation in female mice leads to an improvement of certain health-span indicators including glucose tolerance, osteoporosis and skin fitness, without significantly increasing global cancer incidence.

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