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Loss of telomere function can induce cell cycle arrest and apoptosis but the processes that trigger cellular responses to telomere dysfunction remain largely unknown. In the October 5 issue of Cell, Michael Hemann and colleagues from Johns Hopkins University School of Medicine show that the shortest telomere, not average telomere length, is critical for cell viability and chromosome stability (*Cell* 2001, **107:**67-77).

Hemann *et al.* crossed telomerase-deficient mice having short telomeres with mice heterozygous for telomerase activity, which have longer telomeres. They found that the phenotype of the telomerase-null offspring was similar to that of the late generation parent, although only half of the chromosomes were short. In addition, spectral karyotyping showed that loss of telomere function occurred preferentially on chromosomes with critically short telomeres.

"Our evidence suggests that once a telomere becomes very short, the cell recognizes it as a DNA break," said Carol Greider the senior author of the paper. The break signals the cell to enter apoptosis, as a protective mechanism to prevent chromosome rearrangement and the development of cancer.

These results hint that the main task of telomerase is not to maintain average telomere length but rather to maintain function for critically short telomeres.

References

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