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## Events at the ends

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The [telomere position effect \(TPE\)](#), first described in yeast, causes the reversible silencing of genes near telomeres by a mechanism that depends on telomere length and the distance to the gene. Evidence for TPE in human cells might explain the altered cellular phenotypes associated with [replicative aging](#) and telomere shortening. In the June 15 [Science](#), Baur *et al.* report the first demonstration of TPE in human cells (*Science* 2001, **292**:2075-2077). They created new telomere structures in HeLa cells by introducing a plasmid containing a luciferase reporter gene next to a stretch of telomeric repeats. Cell clones with a telomeric reporter expressed ten times less luciferase than clones with a random internal integration site. The telomere silencing effects could be overcome by treating cells with an inhibitor of histone deacetylase (Trichostatin A). Baur *et al.* found that when they elongated the telomere length, using the catalytic subunit of human telomerase reverse transcriptase (hTERT), there was an additional 2-10-fold decrease in telomeric luciferase expression. The authors suggest that TPE provides a mechanism by which the expression of subtelomeric human genes could increase with replicative age.

## References

1. Position effect at *S. cerevisiae* telomeres: reversible repression of Pol II transcription.
2. Cellular senescence as a tumor-protection mechanism: the essential role of counting.
3. *Science* , [<http://www.sciencemag.org>]