

Telomere length influences cancer cell differentiation

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Researchers from the Japanese Foundation for Cancer Research in Tokyo have discovered that forced elongation of telomeres (extensions on the end of chromosomes) promotes the differentiation of cancer cells, probably reducing malignancy, which is strongly associated with a loss of cell differentiation. They report their findings in a manuscript published online ahead of print, in the journal *Molecular and Cellular Biology*.

"Cancer cells may maintain short telomeres to maintain their undifferentiated state," says Hiroyuki Seimiya, a researcher on the study.

Telomeres are protective extensions on the ends of chromosomes, which shorten as cells age, like an hourglass running down. They protect the end of the chromosome from deterioration or from fusion with neighboring chromosomes. Without telomeres chromosomes would progressively lose genetic information as cells divide and replicate.

Cancer cells have shorter telomeres compared to healthy cells, but they guard their immortality by maintaining these telomeres' length.

In the study, the forced elongation of [cancer cells'](#) telomeres suppressed a number of genes and proteins that appear to be involved in tumor malignancy, according to the report. For example, one of these factors, N-cadherin, is involved in prostate cancer metastasis.

Based on their results, the investigators now propose that telomeres also modulate the behavior of cells by controlling [gene expression](#), by as yet

unknown mechanisms, says Seimiya. His research, he says, may ultimately lead to new types of treatments for cancer.

More information: [www.asm.org/images/Communicati ...
ps/2013/0613telo.pdf](http://www.asm.org/images/Communications/2013/0613telo.pdf)

Provided by American Society for Microbiology

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