Chromosome's Guardians Susceptible to UV Radiation, Scientists Find

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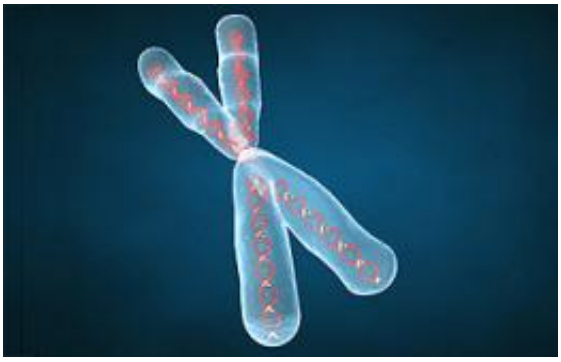

A chromosome with DNA

(PhysOrg.com) -- The molecular caps at the ends of chromosomes that protect humans against cancer and premature cellular aging show a surprising inability to protect themselves against ultraviolet radiation, a new Yale School of Medicine study has found.

Telomeres—the repeat sequences of DNA at the end of chromosomes that act like plastic tips at the end of a shoelace—are much more likely to be damaged by UV radiation than are other common cellular structures, researchers report in the study published online April 29 in the journal PLoS Genetics.

“This damage is not repaired. It is as if the cell has decided to defer maintenance to the telomeres,” said Douglas Brash, professor of

therapeutic radiology, genetics and dermatology, a researcher for the Yale Cancer Center, and senior author of the study.

As cells divide over a lifetime, telomeres tend to wear down, and the resulting instability of chromosomes can lead to problems such as increased risk of cancer. As telomeres shorten, cells begin to age, deteriorate and eventually die.

Given their importance, scientists expected telomeres to possess robust defense mechanisms. Brash and Yale postdoctoral researcher Patrick Rochette, now assistant professor at Laval University, Quebec, tested the hypothesis by bombarding human cells with ultraviolet radiation. They found 10 times more DNA damage in telomeres than to the p53 gene, to a gene encoding a subunit of the cell's ribosome or to mitochondrial DNA. And the damage to the telomeres was not repaired.

"There may be many reasons for this, but it looks like the medicine might be worse than the disease," Brash said.

An overly robust response to fix damage at the tips of the chromosome might trigger even bigger problems for the cell - such as causing breaks within double strands of DNA, Brash speculated.

The strategy, however, is not without risk. Over many years, the accumulating damage may make the telomeres harder to copy when the cell divides, eventually leading to cell aging and death.

Provided by Yale University