SNC 2D

Cell Division and Aging

Most scientists now agree that aging is, at least in part, the result of accumulating damage to the molecules—such as proteins, lipids, and nucleic acids (DNA and RNA)—that make up our cells. If enough molecules are damaged, our cells will function less well, our tissues and organs will begin to deteriorate, and eventually, our health will decline. So in many respects, we appear to age much like a car does: Our parts start to wear out, and we gradually lose the ability to function.

How do our cells know when to retire? Do cellular clocks have a big hand and a little hand and go, "Tick, tock?" Not exactly. It turns out that each cell has 92 internal clocks—one at each end of its 46 chromosomes. Before a cell divides, it copies its chromosomes so that each daughter cell will get a complete set. But because of how the copying is done, the very ends of our long, slender chromosomes don't get copied. It's as if a photocopier cut off the first and last lines of each page.

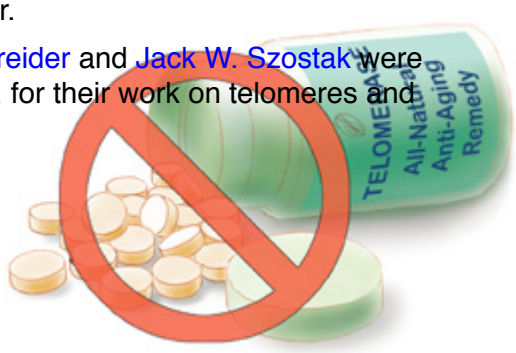
As a result, our chromosomes shorten with each cell division. Fortunately, the regions at the ends of our chromosomes—called **telomeres**—spell out the genetic equivalent of gibberish, so no harm comes from leaving parts of them behind. But once a cell's telomeres shrink to a critical minimum size, the cell takes notice and stops dividing.

In 1985, scientists discovered **telomerase**. This enzyme extends telomeres, rebuilding them to their former lengths. In most of our cells, the enzyme is turned off before we're born and stays inactive throughout our lives. But theoretically, if turned back on, telomerase could pull cellular retirees back into the workforce. Using genetic engineering, scientists reactivated the enzyme in human cells grown in the laboratory. As hoped, the cells multiplied with abandon, continuing well beyond the time when their telomerase-lacking counterparts had stopped.

Could reactivating telomerase in our cells extend the human lifespan? Unfortunately, the exact opposite—an untimely death from cancer—could occur. Cancer cells resurrect telomerase, and by maintaining the ends of the cell's chromosomes, the enzyme enables the runaway cell division that typifies cancer. It may, therefore, be a good thing that shrinking telomeres mark most of our cells for eventual retirement.

Nonetheless, scientists still have high hopes for harnessing telomerase. For instance, the enzyme could be used as a tool for diagnosing cancer.

Three scientists, Elizabeth Blackburn, Carol W. Greider and Jack W. Szostak were awarded the 2009 Nobel Prize in Physiology or Medicine, for their work on telomeres and telomerase.



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Questions

1. What happens to our chromosomes with each cell division?

2. What are telomeres?

3. What happens to a cell when telomeres shrink to a critical size?

4. How does telomerase work?

5. Is reactivation of telomerase a good idea, why or why not?