Telomere Immortality

In this report we will talk about optimizing health so as to extend life and remain healthy. But why exactly does better health help you live longer? Put another way, what exactly makes the body grow old and die?

The answer in a word is "telomeres". This is the holy grail for some longevity scientists. Inside the nucleus of a cell, our genes are arranged along twisted, double-stranded molecules of DNA called chromosomes. At the ends of the chromosomes are stretches of DNA called telomeres, which protect our genetic data, make it possible for cells to divide, and hold some secrets to how we age and get cancer.

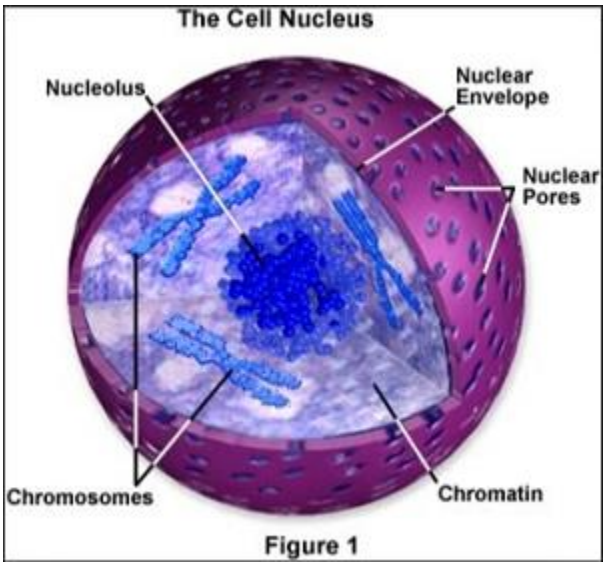


Figure 1

Telomeres have been compared with the plastic tips on shoelaces, because they keep chromosome ends from fraying and sticking to each other, which would destroy or scramble an organism's genetic information.

Yet, each time a cell divides, the telomeres get shorter. When they get too short, the cell can no longer divide; it becomes inactive or "senescent" or it dies. This shortening process is associated with aging, cancer, and a higher risk of death. So telomeres have also been compared with a bomb fuse because once the telomere gets too short the body cell no longer divides and dies.

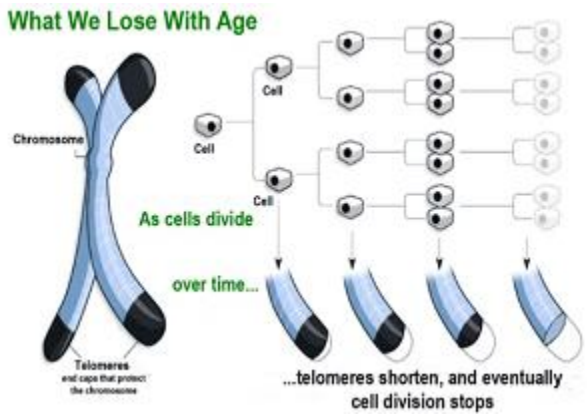
In white blood cells, the length of telomeres ranges from 8,000 base pairs in newborns to 3,000 base pairs in adults and as low as 1,500 in elderly people. (An entire chromosome has about 150 million base pairs.) Each time it divides, an average cell loses 30 to 200 base pairs from the ends of its telomeres.

Cells can normally divide only about 50 to 70 times, with telomeres getting progressively shorter until the cells become senescent or die.

Telomeres do not shorten in tissues where cells do not continually divide, such as heart muscle.

Without telomeres, the main part of the chromosome — the part with genes essential for life — would get shorter each time a cell divides. So telomeres allow cells to divide without losing genes. Cell division is necessary for growing new skin, blood, bone, and other cells.

Without telomeres, chromosome ends could fuse together and corrupt the cell's genetic blueprint, possibly causing malfunction, cancer, or cell death. Because broken DNA is dangerous, a cell has the ability to sense and repair chromosome damage. Without telomeres, the ends of chromosomes would look like broken DNA, and the cell would try to fix something that wasn't broken. That also would make them stop dividing and eventually die.



An enzyme named telomerase adds bases to the ends of telomeres. In young cells, telomerase keeps telomeres from wearing down too much. But as cells divide repeatedly, there is not enough telomerase, so the telomeres grow shorter and the cells age.

As a cell begins to become cancerous, it divides more often, and its telomeres become very short. If its telomeres get too short, the cell may die. But very often these cells escape death by making more telomerase enzyme, which prevents the telomeres from getting even shorter.

Too much telomerase can help confer immortality onto cancer cells and actually increase the likelihood of cancer, whereas too little telomerase can also increase cancer by depleting the healthy regenerative potential of the body. To reduce the risk of cancer we need an ideal level of telomerase, with not a whole lot of room for error.

This clarifies that “telomerase shots” are not the magical anti-aging potion that Faust and so many other humans have sought throughout history.

Many cancers have shortened telomeres, including pancreatic, bone, prostate, bladder, lung, kidney, and head and neck.

Measuring telomerase may be a way to detect cancer. And if scientists can learn how to stop telomerase, they might be able to fight cancer by making cancer cells age and die. In one experiment, researchers blocked telomerase activity in human breast and prostate cancer cells growing in the laboratory, prompting the tumor cells to die. But there are risks. Blocking telomerase could impair fertility, wound healing, and the production of blood cells & immune-system cells.

Geneticist Richard Cawthon and colleagues at the University of Utah found shorter telomeres are associated with shorter lives. Among people older than 60, those with shorter telomeres were three times more likely to die from heart disease and eight times more likely to die from infectious disease.

If telomerase makes cancer cells immortal, could it prevent normal cells from aging? Could we extend lifespan by preserving or restoring the length of telomeres with telomerase? If so, would that increase our risk of getting cancer?

Scientists are not yet sure. But they have been able to use telomerase in the lab to keep human cells dividing far beyond their normal limit, and the cells do not become cancerous.

If we used telomerase to "immortalize" human cells, we may be able to mass produce cells for transplantation, including insulin-producing cells to cure diabetes, muscle cells for treating muscular dystrophy, cartilage cells for certain kinds of arthritis, and skin cells for healing severe burns and wounds. An unlimited supply of normal human cells grown in the laboratory would also help efforts to test new drugs and gene therapies.

Cawthon's study found that when people are divided into two groups based on telomere length, the half with longer telomeres lives an average of five years longer than those with shorter telomeres. This study suggests that lifespan could be increased five years (or more) by increasing the length of telomeres in people with shorter ones.

People with longer telomeres still experience telomere shortening as they age. How many years might be added to our lifespan by completely stopping telomere shortening? Cawthon believes 10 years and perhaps 30 years.

After age 60, the risk of death doubles every 8 years. So a 68-year-old has twice the chance of dying within a year compared with a 60-year-old. Cawthon's study found that differences in telomere length accounted for only 4% of that difference. And while intuition tells us older people have a higher risk of death, only 6% is due purely to chronological age. When telomere length, chronological age, and gender are combined (women live longer than men), those factors account for 37% of the variation in the risk of dying over age 60. So what causes the other 63%?

A major cause of aging is "oxidative stress." It is the damage to DNA, proteins, and lipids (fats) caused by oxidants, which are highly reactive substances containing oxygen. These oxidants are produced normally when we breathe, and also result from inflammation, infection, and consumption of alcohol and cigarettes. In one study, scientists exposed worms to two substances that neutralize oxidants, and the worms' lifespan increased an average 44%.

Another factor in aging is "glycation." It happens when glucose, the main sugar we use as energy, binds to some of our DNA, proteins, and lipids, leaving them unable to do their jobs. The problem becomes worse as we get older, causing body tissues to malfunction, resulting in disease and death. Glycation may explain why studies in laboratory animals indicate that restricting calorie intake extends lifespan. Their extended life is likely to be a result of eating less sugary foods rather than overall calorie restriction.

It is likely that oxidative stress, glycation, telomere shortening, and chronological age (along with various genes) all work together to cause aging.

What are the prospects for human immortality?

Human lifespan has increased considerably since the 1600s, when the average lifespan was 30 years. By 2012, the average US life expectancy was nearly 79. Reasons for the increase include sewers and other sanitation measures, antibiotics, clean water, refrigeration, vaccines and other medical efforts to prevent children and babies from dying, improved diets, and better health care. Life expectancy varies considerably in different parts of the world.

Some scientists predict that average life expectancy will continue to increase. A few say vastly longer lifespans are possible. Geneticist Richard Cawthon says that if all processes of aging could be eliminated and oxidative stress damage could be repaired, "one estimate is people could live 1,000 years."

Although we cannot slow down the degradation of our telomeres as a result of aging (the passage of time), there are two very effective things we can do straight away to preserve our telomeres. As already touched upon, we can reduce oxidative stress and we can reduce glycation in our body. These are the two biggest things we can do to extend longevity and remain healthy.

Summary: Preserving the finite telomere capacity of the body is the key to staying healthy and living longer. This is not beyond our control. We do it by minimizing oxidative stress and glycation.

Source: The Science of Longevity by Russell Eaton.

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