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Will the Hayflick limit keep us from living forever?

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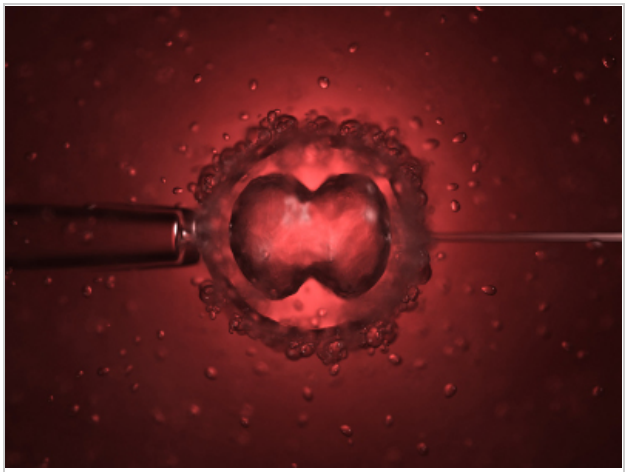

A cell undergoing division.
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Will the Hayflick limit keep us from living forever?

In a small laboratory in Philadelphia, Penn., in 1965, a curious young biologist conducted an experiment that would revolutionize the way we think about aging and death. The scientist who conducted that experiment, Dr. Leonard Hayflick, would later lend his name to the phenomenon he discovered, **the Hayflick limit**.

Dr. Hayflick noticed that cells grown in cultures reproduce by dividing. They produce facsimiles of themselves (by a process known as **mitosis**) a finite number of times before the process stops for good and the cell dies. In addition, cells frozen during their lifetimes and later returned to an active state had a kind of cellular memory: The frozen cells picked up right where they left off. In other words, interrupting the cells' life span did nothing to lengthen it.

Hayflick found that cells go through three phases. The first is rapid, healthy cell division. In the second phase, mitosis slows. In the third stage, **senescence**, cells stop dividing entirely. They remain alive for a time after they stop dividing, but sometime after cellular division ends, cells do a particularly disturbing thing: Essentially, they commit suicide. Once a cell reaches the end of its life span, it undergoes a programmed cellular death called **apoptosis**.

When a new cell is born from an older one through cell division, it begins its own life span. This span appears to be governed by DNA, located in the nucleus of a cell. A student of Hayflick's later found that when he removed the nucleus of an old cell and replaced it with the nucleus of a young cell, the old cell took on a new life. The old cell's life span took on that of a young cell. Like any other cell (except for stem cells), it divided most rapidly early in its lifetime, eventually slowing cellular division as it aged, before stopping altogether and undergoing apoptosis.

The implications of the Hayflick limit are staggering: Organisms have a **molecular clock** that's inexorably winding down from the moment we're born. We'll explore that idea further on the next page.

THE ULTIMATE HAYFLICK LIMIT
When all of the cells created in the human body before birth (and all of the cells these cells produce) are multiplied by the average time it takes for cells to reach the end of their lives, you get roughly 120 years. This is the ultimate Hayflick limit -- the maximum number of years that a human can possibly live. What's strange is that the Biblical book of Genesis (6:3) explicitly states that humankind's days "shall be one hundred years and twenty" [source: Cramer]. It's worth mentioning, though, that this life span is later amended in Psalms 90:10, which says we can live to age 70; 80 years at the most [source: Bible Gateway].

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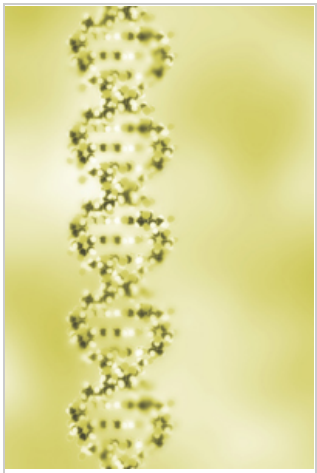
Why do cells commit suicide?

When Dr. Leonard Hayflick performed his experiments using human cells grown in a culture, he managed to pull back the curtain on an ancient process that essentially prevents immortality. The process of cellular death exists within our genetic code. The nucleus of a **diploid cell** (a cell with two sets of chromosomes) is comprised of DNA information contributed by each of an organism's parents. Since the key to the Hayflick limit is found in the cell's nucleus, we are basically programmed to die. Why is this?

There are several reasons why a cell should be programmed to die after a certain point. In the developmental stages, for example, human fetuses have tissue that creates some webbing between our fingers. As we gestate, this tissue undergoes apoptosis that ultimately allows our fingers to form. Menstruation -- the monthly process of shedding the lining of the uterus -- is also carried out through apoptosis. Programmed cellular death also combats cancer (defined as uncontrolled cellular growth); a cell that turns cancerous still has a life span like any other cell and will die out eventually. The drugs used in chemotherapy are meant to accelerate this process by triggering apoptosis in cancerous cells.

Apoptosis is the result of several signals from both inside and outside a cell. When a cell stops receiving the hormones and proteins it needs to function or sustains enough damage to stop functioning properly, the process of apoptosis is triggered. The nucleus explodes and releases chemicals that act as signals. These chemicals attract **phospholipids** that engulf the cell fragments, degrade the individual chromosomes and carry them out of the body as waste.

Clearly, apoptosis is an intensely regulated and highly refined process. How, then, could we ever possibly thwart it? Let's find out on the next page.



Telomeres are non-replicating strands of DNA at the ends of chromosome pairs that allows cell division to be carried out.
Thomas Northcut/Getty Images

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Telomerase and the Possibility of Cellular Immortality

The discovery of the Hayflick limit represented a radical change in the way science looked at cellular reproduction. Before the doctor's discovery, cells were thought to be capable of immortality. Although the phenomenon of the Hayflick limit has been studied only in vitro, it eventually came to generally be accepted in the scientific community as fact. For decades, it looked like the limit was insurmountable, and it still appears that way. In 1978, however, the discovery of a segment of non-replicating DNA in cells called **telomeres** shed light on the possibility of cellular immortality.

Telomeres are repetitive strings of DNA found at the ends of chromosome pairs within diploid cells. These strings are usually compared to the plastic ends of shoelaces (called aglets) that keep the laces from fraying. Telomeres provide the same protection to chromosomes, but the telomere on the end of each chromosome pair is shortened with each cellular division. Eventually, the telomere is depleted, and apoptosis begins.

The discovery of telomeres supported the Hayflick limit; after all, it was the physical mechanism by which cells entered senescence. Just under a decade later, however, another breakthrough in cellular aging was uncovered. **Telomerase** is a protein that's found in all cells, but in normal cells, it's turned off -- it doesn't do anything. In abnormal cells like tumors and germ cells, however, telomerase is quite active: It contains an RNA template capable of producing new telomeres on the ends of chromosomes in aging cells.

Telomerase has the aging research community excited for two reasons. First, since it's naturally active in tumors and can be detected in urine samples, testing for the presence of telomerase can lead to more effective testing of cancer patients. Second, researchers have figured out how to extract telomerase and synthesize it. Potentially, if active telomerase is added to normal adult cells, they'll continue to replicate long beyond their Hayflick limit. In one study that supports this notion, researchers reported that cells to which they'd introduced telomerase had replicated 20 more times than their normal life span would indicate -- and were still dividing [source: Cherfas].

Science has yet to definitively prove that telomerase can produce cellular immortality. There seems to be myriad factors involved in programmed cellular death beyond the destruction of telomeres. As long as humans fear death, though, there will always be research into overcoming these natural obstacles to our immortality, cellular or otherwise.

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