Smart Life Forum

SmartLifeForum.org Presents

Bill Andrews, Ph.D.

on

The Quest for the Cure for Aging

Thurs., Oct 15, 2009, 7 PM
LOCATION: Cubberley Community Center – Room H1
4000 Middlefield Rd
Palo Alto, CA

FUTURE SPEAKERS
Nov. 19 -- Adiel Tel-Oren, MD, on Functional Medicine
Dec. 17 -- Dawson Church, PhD, on Genes
Jan 21 -- John Dommisse, MD, on Psychotherapy

Short Presentation: "Want to change your life for the better or for ever?" Eric Olsen and Charlene Arrigale, a health and fitness team will speak on lifestyle and dietary choices with emphasis on Acid/Alkaline body pH balance. Charlene Arrigale, C.N.C, the previous owner of Elk Grove Vitamins, currently consults at Apple Health Foods and Back in Health Chiropractic, both in Redwood City. Eric Olsen, is a 30 year health and fitness advocate/motivational speaker with a passion for living free of all sickness and disease. Change in lifestyle and dietary choices initiates better eating, thinking, and living when combined with fitness, for balanced wellness and energy. Eric and Charlene will demonstrate how to create easy pH balanced food choices focusing around green vegetable and real food recipes.

FMBR, October 23: Dr. Dawson Church will speak on "Your Happy Gene: The Science of Inducing Spiritual States of Heart." This lively presentation will talk about the significance of genes and their relation to the states people experience such as happiness, spirituality and love. For location and more information see www.FMBR.org.

Meet Bill Andrews

Dr. William H. Andrews has worked in the biotech industry for 28 years, focusing the last 15 years on finding ways to extend human lifespan through the intervention of telomere shortening in human cells.

Dr. Andrews earned his Ph.D. in Molecular and Population Genetics at the University of Georgia in 1981. He was a Senior Scientist at Armos Corporation and Codon Corporation, Director of Molecular Biology at Codon and at Geron Corporation, and Director of Technology Development at EOS Biosciences. He is presently the founder, President and CEO of Sierra Sciences, a biotech company in Reno, Nevada focused exclusively on finding drugs that will transiently induce the expression of endogenous telomerase in human cells. Sierra Sciences has already identified more than fifty such drugs and is presently characterizing their mechanism of action.

While Director of Molecular Biology at Geron Corporation, Dr. Andrews was one of the principal discoverers of both the RNA and protein components of human telomerase and was awarded 2nd place as "National Inventor of the Year" in 1997 for this work. He is presently a named inventor on 37 US issued telomerase patents.

Main Presentation

In search of the cure for aging, medical science is examining technologies that will not merely extend our life expectancy by a few years, but may actually extend it indefinitely. Although our life expectancy has increased tremendously over the last century, there is still a theoretical 125-year limit on our lifespan, and no medical therapy available today has been able to break through this barrier. In the last three decades, there has been a tremendous upsurge of scientific knowledge of how and why we age.

There is a clock that ticks inside every dividing cell of our bodies. This clock is found at the tips of our chromosomes, in a region of the chromosome called the telomere. When human cells divide, telomeres shorten, and the length of the telomeres correlates with the age of these cells. This telomere clock may be the clock of aging.

Inside every one of our cells is a gene that produces an

enzyme called telomerase. Telomerase stops the telomere clock from ticking, and can give cells the potential to divide forever. The gene for telomerase is turned on only in our reproductive cells, and turned off in almost all other cells. A small-molecule compound could possibly turn it back on to prevent the shortening of our telomeres.

Control of telomere length may be the most important step in eliminating the 125-year limit on our lifespan and taking the first crucial steps toward allowing us to live young, healthy lives indefinitely.

Is Aging a Disease?

References to "the disease of aging" still make many people uncomfortable. After all, aging is a natural process that has existed forever – so how can it be a disease?

In fact, aging has not existed forever. Approximately 4.5 billion years ago, a cell came into existence on Earth that was the progenitor of every living organism that has since existed. This cell had the ability to divide indefinitely. It exhibited no aging process; it could produce a theoretically infinite number of copies of itself, and it would not die until some environmental factor killed it. When the ancestry of any given cell is traced back to this very first living cell, this lineage is called the cell's "germ line."

Much later – perhaps three billion years later – some cells of the germ line began to form multicellular organisms: worms, beetles, lobsters, humans. The germ line, however, was still passed on from one generation to the next, and remained immortal. Even with the inclusion of multicellular organisms, the germ line itself exhibited no aging process.

But, in some multicellular organisms, such as humans, certain cells strayed from the germ line and began to exhibit signs of aging. These cells aged because they became afflicted with a disease: their ability to reproduce themselves indefinitely became broken. The cause of this disease is still speculative, but many scientists are searching for cures.

The fact that a disease has existed in the genetic code of an animal for a very long time does not mean that it is not a disease. Thousands of diseases, from hemophilia to cystic fibrosis, have lurked in our genes for far longer than recorded history. These diseases should be cured, and aging is no exception.

The Cause of Aging

The root cause of aging is very straightforward: we age because our cells age.

In 1961, Leonard Hayflick, a researcher at the Wistar Institute in Philadelphia, discovered that there was a limit to the number of times a human cell could divide. After about 70 divisions, a cell derived from embryonic tissue enters a stage where its ability to divide slows and eventually stops. This stage is called cellular senescence. Hayflick also observed that the number of times a cell could divide was governed by the age of the cells: cells from a twenty-year-old could divide more times than cells from a fifty-year-old, which in turn would divide more times than cells from a ninety-year-old.

Hayflick discovered that, in essence, there is a clock ticking inside every dividing cell of our body. Our aging process isn't simply a consequence of accumulated damage: there is a specific property of our cells that limits how long we can live.

The nature of this property was proposed independently in the early 1970s by both Soviet and American scientists. When a cell divides, the genetic material inside that cell needs to be copied. This process is called DNA replication. These scientists suggested that the limitation on cell division is rooted in the very nature of DNA replication. The enzymes that replicate a strand of DNA are unable to continue replicating all the way to the end, which causes the loss of some DNA.

As an analogy, think of a DNA as a long row of bricks, and of DNA replication as a bricklayer walking backwards on top of a brick wall laying a new layer on top of that row. When the end of the wall is reached, the bricklayer finds himself standing on top of the brick he's supposed to replicate. Since he can't put down a brick where his feet are, he steps back and falls off the wall - leaving the very end of the wall missing a brick. As a result, the new copy of the wall is shorter.

Just like this brick wall was copied imperfectly, our DNA is unable to perfectly copy itself; when a strand is replicated, the new strand is shorter than the old strand.

If we lost portions of the information encoded in our DNA every time it replicated, human life would be impossible. Our cells couldn't even divide enough times to allow us to be born. Fortunately, we are born with long, repetitive sequences of DNA at the end of each of our chromosomes, which later shorten during the normal DNA replication process. These repetitive sequences are called "telomeres."

Telomeres, like all DNA, are made up of units called nucleotides, arranged like beads on a string. The nucleotides in human telomeres are arranged in the repeating sequence TTAGGG (two thymine nucleotides, one adenine nucleotide, and three guanine nucleotides). This sequence is repeated hundreds of times in tandem in every telomere.

Each time our cells divide and our chromosomes replicate, our telomeres become shorter. When we are first conceived, the telomeres in our single-cell embryos are approximately 15,000 nucleotides long. Our cells divide

rapidly in the womb, and by the time we are born, our telomeres have decreased in length to approximately 10,000 nucleotides. They shorten throughout our lifetime, and when they reach an average of about 5,000 nucleotides, our cells cannot divide any further, and we die of old age.

Leonard Hayflick had discovered that there was a clock ticking in every dividing cell of our body; telomere shortening explains what makes that clock tick.

The time remaining on this "telomere clock" can be measured from our blood cells. When such measurements are taken, a significant correlation is found between a person's age and the number of "ticks" remaining on the person's clock.

Telomerase

Obviously, there must be a way for our bodies to re-lengthen telomeres. Otherwise, our sperm and egg cells would contain telomeres the same length as the rest of our cells, which would yield embryos as old as we are. Because so much cell division takes place in the womb, our children would then be born much older than us. Humanity could not exist more than a generation or two if this were the case.

However, our reproductive cells do not exhibit telomere shortening, and show no signs of aging. They are essentially immortal. They are our germ line – the same one that has been dividing since the beginning of life on this planet.

The reason these cells are immortal is that our reproductive cells produce an enzyme called telomerase. Telomerase acts like an assembly line inside our cells that adds nucleotides to the ends of our chromosomes, thus lengthening our telomeres.

In a cell that expresses telomerase, telomeres are lengthened as soon as they shorten; it's as though every time the "telomere clock" inside our cells ticks once, telomerase pushes the hands of the clock back one tick.

Telomerase works by filling the "gap" left by DNA replication. Returning to the analogy of the bricklayer that can't lay the last brick on the brick wall, telomerase would be like an angel that flies in and puts the last brick in place.

Thus, by restoring telomere length to enable cells to continue to divide and replicate, telomerase might be the key to extending the human lifespan.

Proofs of Principle

There is a plan in place for inducing telomerase in all our cells. But will that plan work? Will it cure aging? That's the trillion-dollar question, and scientists have been trying to answer it for more than a decade. So far, all the signs point to yes: telomerase is a very likely cure for aging.

In 1997, scientists inserted the telomerase gene into normal human skin cells grown in a Petri dish. When they observed that the telomerase enzyme was being produced in the cells, as hoped, they also observed that the skin cells became immortal: there was no limit to the number of times these cells could divide. When the lengths of the telomeres in these "telomerized" cells were examined, the scientists were surprised to see that the telomeres didn't just stop shortening: they got longer. The critical question, then, was whether the cells were becoming younger.

A few years later, scientists inserted the telomerase gene into human skin cells that already had very short telomeres. These cells were then grown into skin on the back of mice. As one would expect, skin from cells that hadn't received the telomerase gene looked like old skin. It was wrinkled, blistered easily, and had gene expression patterns indicative of old skin.

The skin grown from cells that had received the telomerase gene, on the other hand, looked young! It acted like young skin, and, most importantly, its gene expression patterns, as analyzed by DNA Array Chip analysis, were almost identical to the gene expression patterns of young skin. For the first time ever, scientists had demonstrably reversed aging in human cells.

So much for individual cells. Now, would the concept apply to whole living organisms? In November 2008, scientists published a paper describing how they had created cloned mice from mouse cells containing the inserted telomerase gene, which continually produced the telomerase enzyme. These mice were shown to live 50% longer than cloned mice created from cells that didn't contain the inserted telomerase gene.

It's becoming increasingly clear that prevention of telomere shortening might be the best way to extend human lifespan beyond the theoretical 125-year maximum lifespan. How long this can extend the human lifespan is anyone's guess, but living a healthy, youthful life to 250, 500, or even 1,000 years is not outside the realm of possibility. More research needs to be done to answer that question.

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