

2 Promising Developments in Alzheimer's Treatment

Alzheimer's is a devastating disease that has no cure. In the United States, more than 5.3 million Americans currently have the condition, and unless new treatments are developed, the number of people suffering from the disease will almost triple by 2050.

With the depressing statistics firmly in mind, researchers are rushing to develop treatments that will at least slow—but hopefully cure and even prevent the disease.

Here are two promising developments:

Telomerase treatment. Dr. Michael Fossel, author of *The Telomerase Revolution* and co-founder of the bio-tech company Telocyte, is petitioning the FDA to start human trials on a treatment he believes will cure Alzheimer's. Fossel, a former emergency room physician, has studied aging for 30 years, and he says that an enzyme called telomerase is key to conquering the diseases of aging, including Alzheimer's.

Telomeres are the pieces of DNA that act as protective caps on the ends of chromosomes. Chromosomes, which protect our genes, get shorter every time a cell divides. Once a chromosome gets too short, it dies. Many studies have shown that shortened chromosomes are associated with the diseases of aging, especially cardiovascular disease, dementia and diabetes.

Some cells, however, don't age and continue to reproduce because they produce telomerase, which lengthens telomeres and keeps the cells young.

Fossel believes that treatments involving telomerase will extend life spans and conquer the diseases of aging.

Other researchers are also studying how to use telomerase to lengthen telomeres as a way of extending life and treating age-related disorders. Scientists at Stanford University are investigating a modified RNA protein that extends the length of telomeres.

RNA, or ribonucleic acid, helps transfer genetic information. The Stanford researchers modified RNA to contain the coding for telomerase. When they treated skin cells with the modified RNA, the treated cells acted like much younger cells, multiplying as many as 40 times more than untreated cells.

“We have found a way to lengthen human telomeres by as much as 1,000 nucleotides, turning back the internal clock in these cells by the equivalent of many years of human life, said Helen Blau, Ph.D., professor of microbiology and immunology at Stanford.

They hope that their research will one day be used to treat humans. “This study is a first step toward the development of telomere extension to improve cell therapies and to possibly treat disorders of accelerated aging in humans,” said study co-author John Cooke, M.D., Ph.D.

Ultrasound technology. Researchers at Australia’s Queensland Brain Institute at the University of Queensland have developed a noninvasive ultrasound that breaks up the brain-clogging amyloid plaques associated with Alzheimer’s disease.

The technique hasn’t been used on humans yet, but when it was used on mice bred to develop Alzheimer’s, 75 percent recovered their memory function. The procedure did not harm normal brain tissue.

The technique uses a specific type of relatively inexpensive ultrasound called focused therapeutic ultrasound, which sends sound waves into brain tissue.

The waves move very quickly, and gently unlock the blood-brain

barrier, which normally protects the brain. But it also prevents drugs from entering, which is a barrier to using traditional medicine.

The sound waves pass through the blood-brain barrier and activate the brain's microglial cells, which remove waste from brain cells. When activated, they break down and remove the toxic beta-amyloid clumps.

"We're extremely excited by this innovation of treating Alzheimer's without using drug therapeutics," said professor Jürgen Götz. "The ultrasound waves oscillate tremendously quickly, activating microglial cells that digest and remove the amyloid plaques that destroy brain synapses.

"With our approach the blood-brain barrier's opening is only temporary for a few hours, so it quickly restores its protective role," Götz said in a statement.

"This treatment restored memory function to the same level of normal healthy mice," he said.

"We're also working on seeing whether this method clears toxic protein aggregates in neurodegenerative diseases other than Alzheimer's, and whether this also restores executive functions, including decision-making and motor control."

The researchers hope to test the technique on larger animals, such as sheep, and to start human trials in 2017.

"The word 'breakthrough' is often misused, but in this case I think this really does fundamentally change our understanding of how to treat this disease," Götz said. "I foresee a great future for this approach."

Other treatments for Alzheimer's are also on the horizon. Scientists at the University of California discovered that immune cells found outside the brain—B-cells, T-cells, and NK-cells—help the brain rid itself of memory-destroying plaque.

Additional studies have indicated that gene therapies, including preventing the development of amyloid plaque in the brain, may become new treatments to prevent Alzheimer's.

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