LOVE how it starts

"Memo to mature, health-minded vampires: You might want to consider limiting your treats to victims under age 30."

Jee was right al along!!!!!!!!!!!

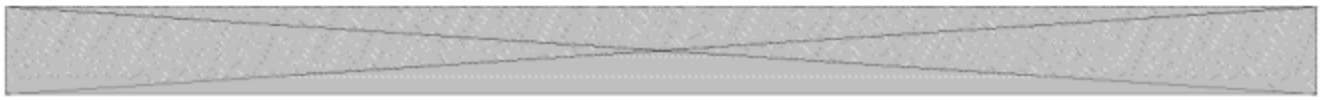
From: Jeffrey Epstein [mailto:jeevacation@gmail.com]
Sent: Saturday, November 05, 2011 9:59 PM
To: Boris Nikolic
Subject: Re: you were right!!!

http://med.stanford.edu/ism/2011/august/aging-brain.html

On Sun, Nov 6, 2011 at 3:01 AM, Boris Nikolic <[redacted]> wrote:

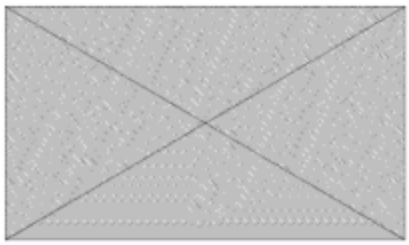
Being around old – even if these are your own cells can't be good for you!

It is a breakthrough…



Cellular Spring Cleaning Slows Aging

by Sarah C.P. Williams on 2 November 2011, 2:04 PM |



Staying young. The mouse on the left has aged normally and shows a curved spine and loss of muscle mass. The mouse on the right was treated with drugs that remove senescent cells from its body, keeping it more youthful.

Credit: Van Deursen Lab

The accumulation of old, stagnant cells in the body is to blame for some age-related diseases, a new study has found. When researchers removed such cells from mice, they were able to delay the onset of cataracts and slow age-related muscle loss.

"This is really a technical tour de force," says geneticist Norman Sharpless of the University of North Carolina School of Medicine in Chapel Hill, who was not involved in the study. "And then they went beyond this technical feat and made findings that are really important to understanding the basic science of aging."

Most cells in the body can't continue dividing forever. After a cell has duplicated itself a number of times—around 50 is the average—a genetic switch turns off the division program. A cell that's no longer dividing is known as senescent; it continues to live but no longer functions as it once did. While most senescent cells continue to behave as whatever cell type they started as, they also begin to secrete immune proteins that scientists hypothesize could cause age-related changes in the surrounding tissues. In elderly humans, at least 5% of the total cells in the body are thought to be senescent. The cells accumulate in places particularly affected by aging—the eyes and muscles, for example.

"It has been hypothesized, since these cells are found at sites of age-related pathologies, that they are related to the development of these pathologies," says biologist Jan van Deursen of the Mayo Clinic in Rochester, Minnesota, lead author of the new paper. But the connection hasn't been fully fleshed out, he says.

Van Deursen and colleagues developed a way to kill senescent cells in mice, clearing them from the body. They engineered mice so that when cells flipped on a gene called $p16^{Ink4a}$, a marker for senescence, the cell would also turn on the production of inactive cellular death genes, not normally produced by senescent cells. Then, when the researchers gave the mice a drug, the death pathway would be activated in all senescent cells. "Our method allowed us to look at the consequences of removing senescent cells at different stages of the mouse life cycle," van Deursen says. "We didn't just block senescence altogether."

First, the researchers cleared senescent cells from the mice throughout their lives—giving the drug every 3 days beginning as soon as the animals were weaned. Although the mice did not have an increased life span, the onset of cataracts was delayed by about 100 days, the treated mice had

twice as wide muscle fibers, and their spine curvature and fat deposits resembled those of youthful mice. Next, the researchers gave the drug to older mice that already showed signs of aging, such as muscle loss. After 5 months, the treated mice showed better improvement in treadmill tests than untreated mice. Their muscle and fat cells did not show signs of aging, although the treatment didn't reverse aging that had already happened, the team reports online today in *Nature*.

"I think the results are quite striking," Sharpless says. But he cautions that further research is needed to understand the effects of removing senescent cells. Although they may promote some age-related disorders, they could prevent others. "Whether there are any unintended results of this has to be studied further," he says. "Yes, we might make cataracts better, but will it come with the risk of cancer or infections?"

Since the work relied on genetically engineered mice, it's not directly translatable into humans, van Deursen says. Researchers, however, can now screen drugs to find compounds that might activate cell death in senescent cells, he says, or that might turn the immune system against senescent cells.

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