Road to the fountain of youth paved with fast food-and sneakers?

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We all know that too much food combined with too little exercise can add up to poor health and disease. But overeating and inactivity also speed up the aging process, right down to our cells. At the end of a cell's lifespan, a process called senescence kicks in—cells lose the ability to divide and begin to secrete substances that damage the surrounding cells. While unhealthy lifestyle habits can accelerate this process, researchers at the Mayo Clinic wanted to know if increased exercise could counteract it. Thomas A. White will present their findings in a poster session on Monday, April 28, at the Experimental Biology meeting.

The research team compared mice fed a fast food diet (FFD) for 5 months with those fed a standard chow diet (control). Unlike the controls, the FFD mice developed insulin sensitivity, impaired glucose tolerance, impaired exercise ability, and heart dysfunction. But when the FFD mice were given a running wheel, the exercise began to counteract the effects of a poor diet. White et al. observed a number of improvements including body weight, metabolism, and cardiac function. They also saw a significant decrease in signs of cell senescence and associated inflammation.

Their results suggest that lifestyle choices do play a major role in cell aging and that exercise may help protect against aging by interfering with cell senescence.

From the authors: "Our data clearly show that poor nutritional choices dramatically accelerate the accumulation of senescent cells, and for the

first time, that exercise can prevent or delay this fundamental process of aging. Despite the need to better understand the role of cellular senescence in aging and disease, our data underscore the profound impact of lifestyle choices on health and successful aging."

Full Abstract

Aging cellular senescence and disease: the influence of diet and exercise

Nutrient excess and physical inactivity are primary drivers of numerous age-related chronic diseases. A growing body of evidence has implicated cellular senescence, a process in which cells lose the ability to divide and concurrently damage neighboring cells by the factors they secrete, as an underlying mechanism of aging and its associated conditions. However, the impact of lifestyle choices on the age-associated accumulation of senescent cells remains poorly understood. Thus, we examined the effects of a fast food diet (FFD) and a voluntary exercise intervention on multiple parameters of healthspan and cellular senescence using a transgenic mouse model that expresses green fluorescent protein (GFP) in p16Ink4a-positive senescent cells. As expected, 12-month old male mice that had received the FFD diet for 5 months demonstrated significantly greater gains in body weight and fat mass compared to mice fed a standard chow diet.

Moreover, mice fed the FFD exhibited hyperinsulinemia impaired glucose tolerance, reduced exercise capacity and diastolic dysfunction. Impressively, providing an exercise wheel (26.9 ± 6.9 km/wk) in parallel with the FFD nullified the deleterious effects of the FFD on body weight and composition, whole-body metabolism, physical performance and cardiac function. Strikingly, our data further suggest that exercise markedly lowers the number of cells in visceral fat depots that express

GFP and stain positively for senescent-associated beta-galactosidase; markers of senescence, that are highly abundant in sedentary mice fed the FFD. Correspondingly, FFD-induced expression of senescence-associated inflammatory factors was also diminished by the exercise intervention. Collectively, our data suggest that lifestyle choices may affect the abundance of senescence cells with advancing age, and exercise may partly mediate its salutary effects on multiple organ systems by preventing their accumulation and/or mediating their removal.

More information: Thomas White, Glenda Evans, Grace Verzosa, Tamara Pirtskhalava, Tamara Tchkonia, Jordan Miller, James Kirkland, and Nathan LeBrasseur. "Aging, cellular senescence and disease: the influence of diet and exercise" (880.7). *FASEB J* April 2014 28:880.7

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