

Study: Fountain of youth for your heart?

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An age-related decline in heart function is a risk factor for heart disease in the elderly. While many factors contribute to a progressive age-related decline in heart function, alterations in the types of fuels the heart uses to produce energy also play important roles. Jason Dyck and his research team at the University of Alberta have been studying the types of fuels used by the heart in young and aged mice.

The young healthy heart normally used a balance of fat and sugar to generate energy to allow the heart to beat and pump blood efficiently. However, as the heart ages the ability to use fat as an energy source deteriorates. This compromises heart function in the elderly.

Interestingly, at a time when the heart is using less fat for energy, Dyck has shown that a protein that is responsible for transporting fat into the contractile cells of the heart actually increases. Based on this finding, Dyck proposed that the mismatch between fat uptake and fat use in the heart could lead to an accumulation of fat in the heart resulting in an agerelated decrease in heart function.

Using a genetically engineered mouse that is deficient in a protein that is responsible for transporting fat into the cells of the heart, Dyck studied these mice as they aged. These genetically altered mice have no choice but to mainly use sugar as a fuel source because they lack the protein that allows them to use fat as a primary fuel source. In an exciting new finding, Dyck showed that old genetically modified mice did not accumulate fat in their hearts, as did ordinary mice.



In addition, Dyck and his team showed that these old genetically altered mice out-performed ordinary old mice on a treadmill test, were completely protected from age-related decline in heart function, and in many ways their hearts looked and performed like hearts from a young mouse. His findings suggest that the protein responsible for transporting fat into the contractile cells of the heart may be a candidate for drug inhibition and that this drug could protect the heart from aging.

This research holds great promise for human beings. Dyck hopes it will lead to the development of medications that inhibit the uptake of fatty acids into the heart and prevent and/or reverse the effects of aging on the heart muscle.

Source: University of Alberta

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