Spermidine found to lengthen lifespan in mice and to promote cardiovascular health

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Emmental cheese. Credit: Wikipedia

(Medical Xpress)—A large team of researchers with members from several Europeans countries and the U.S. has found that mice fed a compound called spermidine lived longer than ordinary mice and also had better cardiovascular heath. In their paper published in the journal *Nature Medicine*, the researchers describe experiments they carried out

with the compound and mice, what they found and why they believe the compound might provide benefits for humans.

Prior research has found that ingestion of spermidine—which was first discovered in semen samples, hence its name—led to longer lifespans in simple organisms such as fruit flies, yeast and roundworms. In this new study, the researchers sought to find out if the same would prove true for more complex creatures.

The researchers chose mice as their target, feeding some groups water with spermidine mixed in, while other groups received plain water. After observing the rodents over the course of their lifespans, the researchers discovered that those who had been given spermidine lived longer than those who had not—even if the supplement was not given to them until middle age. Closer examination of the rodents revealed that those given the supplement also had better heart function and lower blood pressure. They also found that rats fed a high-salt diet, which causes high blood pressure, had lower pressure readings when given spermidine.

Prior research had also suggested that the means by which spermidine extended lifespan was by inducing autophagy in heart cells, which is where cells naturally disable parts of themselves that are dysfunctional or no longer necessary. To find out if this might be the case for rodents, the researchers conducted the same experiments using mice that had a genetic defect that prevented autophagy from taking place and found that feeding them spermidine did not cause them to live longer or to have improved cardiovascular health, suggesting that autophagy may, indeed, be involved in the process.

The researchers acknowledge that there is thus far little evidence that suggests humans might receive the same benefits from consuming the compound, but note that they did conduct a survey of approximately 800 people regarding their diets and found that those that reported eating

foods that contained a fair amount of the compound (mushrooms, whole grains, aged cheese, etc.) had fewer cardiovascular disease symptoms including lower blood pressure. They suggest a much larger study should be undertaken before any real conclusions can be made.

More information: Tobias Eisenberg et al. Cardioprotection and lifespan extension by the natural polyamine spermidine, *Nature Medicine* (2016). DOI: 10.1038/nm.4222

Abstract

Aging is associated with an increased risk of cardiovascular disease and death. Here we show that oral supplementation of the natural polyamine spermidine extends the lifespan of mice and exerts cardioprotective effects, reducing cardiac hypertrophy and preserving diastolic function in old mice. Spermidine feeding enhanced cardiac autophagy, mitophagy and mitochondrial respiration, and it also improved the mechano-elastical properties of cardiomyocytes in vivo, coinciding with increased titin phosphorylation and suppressed subclinical inflammation. Spermidine feeding failed to provide cardioprotection in mice that lack the autophagy-related protein Atg5 in cardiomyocytes. In Dahl salt-sensitive rats that were fed a high-salt diet, a model for hypertension-induced congestive heart failure, spermidine feeding reduced systemic blood pressure, increased titin phosphorylation and prevented cardiac hypertrophy and a decline in diastolic function, thus delaying the progression to heart failure. In humans, high levels of dietary spermidine, as assessed from food questionnaires, correlated with reduced blood pressure and a lower incidence of cardiovascular disease. Our results suggest a new and feasible strategy for protection against cardiovascular disease.