

Caffeine from four cups of coffee protects the heart with the help of mitochondria

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The caffeine in four cups of coffee triggers multiple protective effects. Credit: Nathan Dumlao, Pixabay

Caffeine consumption has been associated with lower risks for multiple diseases, including type II diabetes, heart disease, and stroke, but the

mechanism underlying these protective effects has been unclear. A new study now shows that caffeine promotes the movement of a regulatory protein into mitochondria, enhancing their function and protecting cardiovascular cells from damage. The work, publishing 21 June in the open access journal *PLOS Biology*, by Judith Haendeler and Joachim Altschmied of the Medical Faculty, Heinrich-Heine-University and the IUF-Leibniz Research Institute for Environmental Medicine in Duesseldorf, Germany, and colleagues, found that the protective effect was reached at a concentration equivalent to consumption of four cups of coffee, suggesting the effect may be physiologically relevant.

The authors have previously shown that at physiologically relevant concentrations (i.e. levels reached after four or more cups of coffee) caffeine improved the functional capacity of [endothelial cells](#), which line the interior of blood vessels, and that the effect involved mitochondria, the cell's energy powerhouses.

Here, they showed that a protein called p27, known mainly as an inhibitor of the cell cycle, was present in mitochondria in the major cell types of the heart. In these cells, mitochondrial p27 promoted migration of endothelial cells, protected [heart muscle cells](#) from cell death, and triggered the conversion of fibroblasts into cells containing contractile fibers—all crucial for repair of heart muscle after myocardial infarction. They found that caffeine induced the movement of p27 into mitochondria, setting off this beneficial chain of events, and did so at a concentration that is reached in humans by drinking four cups of coffee. Caffeine was protective against heart damage in pre-diabetic, obese mice, and in aged mice.

"Our results indicate a new mode of action for caffeine," said Haendeler, "one that promotes protection and repair of heart muscle through the action of mitochondrial p27. These results should lead to better strategies for protecting [heart muscle](#) from damage, including

consideration of coffee consumption or [caffeine](#) as an additional dietary factor in the elderly population. Furthermore, enhancing mitochondrial p27 could serve as a potential therapeutic strategy not only in cardiovascular diseases but also in improving healthspan."

More information: Ale-Agha N, Goy C, Jakobs P, Spyridopoulos I, Gonnissen S, Dyballa-Rukes N, et al. (2018) CDKN1B/p27 is localized in mitochondria and improves respiration-dependent processes in the cardiovascular system—New mode of action for caffeine. *PLoS Biol* 16(6): e2004408. doi.org/10.1371/journal.pbio.2004408

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