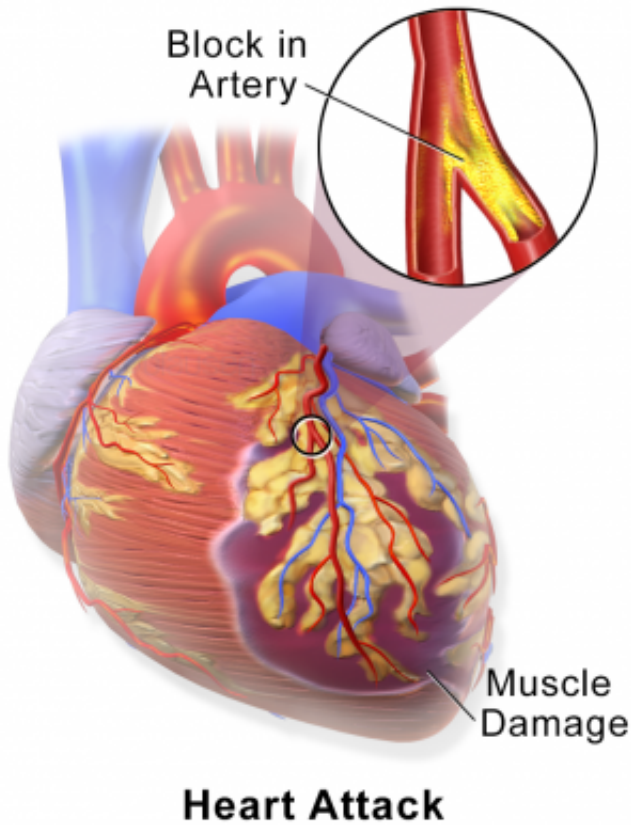


Beta-blockers promote heart muscle cell survival following a heart attack

15 September 2015



Myocardial Infarction or Heart Attack. Credit: Blausen Medical Communications/Wikipedia/CC-A 3.0

A commonly prescribed drug for heart disease may do more good than previously thought. Researchers at York University have found that β -blockers may prevent further cell death following a heart attack and that could lead to better longer term patient outcomes.

Human hearts are unable to regenerate. When [cardiac muscle cells](#) die during and immediately after a [heart attack](#), there is no way to bring the organ back to full health, which contributes to the progression towards eventual heart failure.

Preventing the cells from dying in the first place is the focus of the paper, "Pro-survival Function of MEF2 in Cardiomyocytes is Enhanced by β -blockers", published yesterday in the Nature Publishing Group journal *Cell Death Discovery*.

The paper's corresponding author, York University biology Professor John McDermott, says "An initial clue was the accumulating evidence that the primary function of the MEF2 protein complex in neurons is to protect them from dying off. We suspected that these proteins might play a similar role in the heart."

McDermott and co-authors, PhD students Sara Hashemi, Jahan Salma and Stephanie Wales, subsequently found that MEF2, known for its role in the development of the cardiovascular system, is also linked to heart cell survival and the control of gene expression.

The researchers found that cardiac [cell death](#) increased when MEF2 was suppressed, but when β -blockers, such as Atenolol, were used, MEF2 activity was enhanced and promoted cell survival under conditions when their survival was compromised.

More research is still needed, says McDermott, to fully understand whether β -blocker treatment immediately after a heart attack in human hearts will help recovery and ward off heart failure progression.

More information: *Cell Death Discovery*, www.nature.com/articles/cddiscovery201519

Provided by York University

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