

Protecting the heart: Cardiac heme oxygenase regulates injury response

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The constant beating of the heart requires an enormous output of energy. To meet this demand, cardiomyocytes are loaded with mitochondria, organelles that generate the majority of energy for cells. These mitochondria are dynamically regulated to ensure that damaged mitochondria are removed and replaced by healthy mitochondria.

In this month's issue of *JCI Insight*, James George, Anupam Agarwal, and colleagues at the University of Alabama at Birmingham examined the role of the inducible stress response gene heme oxygenase-1 in mediating mitochondrial quality control in the heart.

The research team found that overexpression of human heme oxygenase-1 in mice protected animals from dilated cardiomyopathy induced by the mitochondrial toxin doxorubicin.

Additionally, heme oxygenase-1 overexpression reduced [mitochondrial fragmentation](#) and promoted the generation of new [mitochondria](#). Cumulatively, this study demonstrates the importance of heme oxygenase-1 in controlling [mitochondrial dynamics](#) in the heart.

More information: Travis D. Hull et al. Heme oxygenase-1 regulates mitochondrial quality control in the heart, *JCI Insight* (2016). [DOI: 10.1172/jci.insight.85817](https://doi.org/10.1172/jci.insight.85817)

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