

# Research reveals new insights into why the heart does not repair itself

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Human heart. Credit: copyright American Heart Association

Heart muscle is one of the least renewable tissues in the body, which is one of the reasons that heart disease is the leading cause of death for both men and women in the United States, according to the Centers for Disease Control and Prevention. Inspired by the idea of helping the heart

repair itself, researchers at Baylor College of Medicine and the Texas Heart Institute have studied pathways known to be involved in heart cell functions and discovered a previously unknown connection between processes that keep the heart from repairing itself. This finding, published in the journal *Nature*, opens the possibility of developing strategies that will promote heart cell renewal in the future.

"We are investigating the question of why the [heart muscle](#) doesn't renew," said senior author Dr. James Martin, professor and Vivian L. Smith Chair in Regenerative Medicine at Baylor College of Medicine. "In this study, we focused on two pathways of cardiomyocytes or heart cells; the Hippo pathway, which is involved in stopping renewal of adult cardiomyocytes, and the dystrophin glycoprotein complex (DGC) pathway, essential for cardiomyocyte normal functions.

We are also interested in studying mutations in DGC components because patients with these mutations have a [muscle wasting disease](#) called muscular dystrophy.

Previous work had hinted that components of the DGC pathway may somehow interact with members of the Hippo pathway. In this study, Martin and colleagues studied the consequences of this interaction in animal models. The researchers genetically engineered mice to lack genes involved in one or both pathways, and then determined the ability of the heart to repair an injury. These studies showed for the first time that dystroglycan 1, a component of the DGC pathway, directly binds to Yap, a part of the Hippo pathway, and that this interaction inhibited cardiomyocyte proliferation.

"The discovery that the Hippo and the DGC pathways connect in the cardiomyocyte and that together they act as 'brakes' or stop signals to cell proliferation opens the possibility that by disrupting this interaction one day it might be possible to help adult cardiomyocytes proliferate and

heal injuries caused by a [heart](#) attack, for example," Martin said.

Another long-term application of this discovery could be to improve cardiac function in children with muscular dystrophy.

"Patients with muscular dystrophy can have severe reduction in cardiac function," Martin said. "Our findings may help to design medicines to slow down cardiac decline in [muscular dystrophy](#) by stimulating cardiomyocyte proliferation. In order to do that, we need more research to understand cardiomyocyte growth control pathways in greater detail."

**More information:** Yuka Morikawa et al, Dystrophin glycoprotein complex sequesters Yap to inhibit cardiomyocyte proliferation, *Nature* (2017). [DOI: 10.1038/nature22979](https://doi.org/10.1038/nature22979)

Provided by Baylor College of Medicine

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