Novel protein in heart muscle linked to cardiac short-circuiting and sudden cardiac deaths

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Cardiovascular scientists at NYU Langone Medical Center have identified in mouse models a protein known as Pcp4 as a regulator of the heart's rhythm. Additionally, when the Pcp4 gene is disrupted, it can cause ventricular arrhythmias.

Results from this animal study were released online Oct. 8 in the peer-reviewed publication, *The Journal of Clinical Investigation*.

"This study demonstrates that Purkinje cell protein-4 (Pcp4) is not only important in maintaining the heart's normal rhythmic behavior, but that when Pcp4 expression is reduced, it short-circuits electrical activity in a small but critical population of cells in the heart muscle, leading to cardiac arrhythmias," said Glenn I. Fishman, MD, William Goldring Professor of Medicine and Director of the Leon H. Charney Division of Cardiology at NYU Langone, and the study's senior author. "We see increased morbidity and mortality when Pcp4 expression is abnormal in our animal models, including ventricular arrhythmias and sudden cardiac death."

Using mouse models of cardiomyopathy and fluorescent tags, the research team was able to isolate cardiac Purkinje cells and show that Pcp4 expression was down-regulated in the diseased hearts, producing electrical abnormalities that increased their susceptibility to arrhythmias. Investigators also found Pcp4 in cardiac ganglia, where it also influences

the heart's rhythm and modulates heart rate control. "Now that we know that Pcp4 is an important regulator of the heart's rhythm, it could serve as an important drug target for treating arrhythmias," added Dr. Fishman. "Although much work remains to be done, our data suggest that drugs that mimic Pcp4's action in the heart could potentially stabilize the heart's rhythm."

According to the American Heart Association, an estimated 2.7 million Americans are living with arrhythmias. People with arrhythmias can be treated with a surgical procedure, such as getting a pacemaker, implantable defibrillator or a cardiac ablation, or by delivering a shock with external defibrillators. Ablations are effective but limited to specific types of rhythm abnormalities. Drug therapies have fallen out of favor because of side effects. The NYU Langone research team believes that with better understanding of the molecular behavior underpinning arrhythmias, more targeted drugs are on the horizon.

Provided by New York University School of Medicine

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