Looking deep inside the heart

The hereditary disease cardiomyopathy, which makes it difficult for the heart to pump enough blood to meet the body's needs, sometimes leads to heart failure. Drugs like beta blockers control the brain's signals to the heart requesting more blood for the body. Other drugs control the amount of blood entering or leaving the heart, diminishing the heart's workload. None of these therapies, however, act *within* the heart cells.

Researcher Jose Pinto is looking into a treatment that does just that. Using a recent two-year grant from the American Heart Association, first he must demonstrate that mutations in a certain gene – Troponin C – cause the disease.

"Troponin C is the calcium sensor in the heart," said the Department of Biomedical Sciences assistant professor. "In cardiac cells, when calcium binds to this protein, it triggers contraction. That's how the heart muscle initiates pumping blood."

If Troponin C isn't working properly, the heart can't pump blood at a healthy pace. The result is either hypertrophic cardiomyopathy, where the heart becomes abnormally thick, or dilated cardiomyopathy, where the pumping chamber stretches and thins the walls.

"Animal models will test the hypothesis that Troponin C can be categorized as a pathogenic gene and that changes in its calcium affinity are responsible for the development of those diseases," said Pinto.

After testing this hypothesis, Pinto's team will focus on a possible treatment of hypertrophic cardiomyopathy using an enzyme inside heart cells.

"The nice thing about the enzyme we are targeting is that it has been shown to be only present in the heart," said Pinto. "The main problem with most drugs developed to target enzymes is that they affect other cells, healthy cells, and the patient experiences unwanted side effects."

Pinto wants to target hypertrophic cardiomyopathy from within the heart cells, preventing those side effects: "There are no drugs available that defeat abnormal muscle contraction caused by altered Troponin C function *inside* the cell. That's the approach that we are taking."

He and his lab will breed mice that develop hypertrophic cardiomyopathy — and then will inject a drug and measure its effect.

"We are trying to reduce the levels of this enzyme-like protein in hypertrophic hearts to see if we can counter defects in the calcium sensor, correcting the heart-contraction function and potentially reversing hypertrophic cardiomyopathy," said Pinto. "This work can serve as a proof-of-concept for the development of new therapeutic strategies to treat heart disease."

Jose Pinto

COLIN HACKLEY

Soiree for science

Every February, FSU alumni Erwin and Stefanie Jackson sponsor a Valentine's Day performance to support research for a debilitating disease that has impacted their family and is the subject of College of Medicine research.

The Valentine's Day Soiree provides funding for The Brian Jackson Dystonia Research and Discovery Program. Proceeds support a collaborative effort between the College of Medicine and Tallahassee Memorial HealthCare to better understand the neurological movement disorder first diagnosed in the Jacksons' son Brian when he was 15.

The event (6 p.m. Feb. 13 at the University Center Club in Tallahassee) features nationally acclaimed performer and FSU School of Theatre graduate Davis Gaines in "Broadway and Beyond." Gaines is best known for more than 2,000 performances on Broadway as the phantom in "Phantom of the Opera."

For event details and ticket or sponsorship information, visit ValentinesDaySoiree.com.

VALENTINE'S DAY SOIRÉE