Treating Heart Failure by Inhibiting Myosin Interaction with a Regulatory Myosin Binding Protein

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The Wisconsin Alumni Research Foundation (WARF) is seeking commercial partners interested in developing and using peptides that promote cardiac muscle contraction by disrupting the binding of MyBP-C to myosin.

OVERVIEW

In the United States, one in every four deaths is attributed to heart disease. That amounts to about 600,000 people every year. Heart failure can arise due to many causes, such as heart attack and coronary artery disease, and generally is characterized by deficient blood pumping. Diagnosing and supporting patients suffering from these diseases costs hundreds of billions of dollars.

Current medications, like diuretics and beta adrenergic receptor blockers, can have deleterious side effects and do not improve prognosis. Many patients would benefit from new, safe and effective treatment options. One promising approach looks to better understand and harness the molecular mechanisms of heart function.

THE INVENTION

A UW–Madison researcher in collaboration with others has developed peptides for treating and slowing the progression of heart failure. The peptides are designed to disrupt a key interaction involving myosin and Myosin Binding Protein C (MyBP-C).

In healthy hearts, myosin is responsible for generating the force that drives normal cardiac function. It works by continually binding and releasing a protein called actin, in a process that powers heart muscle contractions. In healthy hearts, the process is slowed when myosin is also bound to a regulatory protein, MyBP-C. In compromised hearts, the result of this interaction is to further slow and weaken muscle contractions.

The new peptides target a specific binding site of MyBP-C, thereby blocking attachment to myosin. The peptides could be administered as small-molecule pharmaceuticals in conjunction with other therapies like beta blockers and diuretics.
APPLICATIONS

• Treating and slowing the progression of heart failure

KEY BENEFITS

• May dramatically improve speed and strength of heart muscle contractions
• First potential drug of its kind
• Does not stress or require more energy from cardiac cells
• Could support longer, healthier lives for heart failure patients

STAGE OF DEVELOPMENT

Preliminary in vitro data suggests that cardiac muscle cells treated with the peptides recovered 40 percent of their contractile force.

ADDITIONAL INFORMATION

Related Technologies
WARF reference number P03166US describes an improved transgenic mouse model of myocardial function.
WARF reference number P01031US describes an open-chested animal teaching video of myocardial infarction.

Publications

Tech Fields
Pharmaceuticals & Vitamin D - Cardiovascular

CONTACT INFORMATION

For current licensing status, please contact John Nagel at inagel@warf.org or 608-960-9848.