

Researchers Enlist Proteins to 'Switch On' Heart Tissue Repair System

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Researchers at the University of Pennsylvania School of Medicine are utilizing a protein to “switch on” the ability to repair damaged heart tissue. By triggering the cell-cycle signal, researchers can manipulate cells in animal models to regenerate damaged heart tissue. If this research is someday successfully translated to humans, it could change the approach to treating heart disease, the nation’s leading killer. The findings, now on-line, are in *Circulation*, the journal of the American Heart Association.

“This is a different concept in terms of how to address heart disease. The classic thinking is to replace a valve, or place a bypass graft. Traditionally, when the heart gets injured, there's dead tissue, and we work our way around it surgically, even replacing the heart with a transplant,” explains principal investigator Joseph Woo, MD, Director of the Minimally Invasive and Robotic Cardiac Surgery Program at Penn and Assistant Professor of Surgery. “So we asked, 'What would be the most ideal, natural way of fixing any sort of problem like this?' If you look at nature, the best way is to simply re-grow the tissue. We know that if we take out a piece of the liver, our body has programming to grow it back to how it was.”

However, unless the body receives some sort of “jump start,” it does not heal dead tissue in the heart. This can have devastating effects. When tissue dies in the heart (for example, due to a heart attack), it is not able to contract and function as effectively to pump oxygenated blood throughout the body, which could ultimately lead to heart failure and

death.

Working to better understand how to reverse this damage in humans, Woo first identified the signals in the rat heart that currently prevent the ability to re-grow damaged heart tissue. The researchers then manipulated those signals so the heart could work to regenerate itself.

Specifically, Woo's team investigated myocardial regeneration by initiating heart cell division and replication. They did this by expressing the cell-cycle regulator, a protein called cyclin A2. It is unique in its control at two major transitions of the cell cycle and is the only cyclin that is completely silenced after birth in mice, rats and humans. This approach -- using cyclin A2 expression via gene transfer -- yielded improved myocardial function.

"Penn is the first to do this kind of research with damaged heart tissue, by ramping up the body's native reparative system," states Woo. "We are examining the potential role of this regenerative strategy as a future therapy for heart failure. Someday, this could lead to less surgery and perhaps even less medicine in treating heart disease." Woo cautions that this research work has not yet been done on humans and that we may still be years away from that accomplishment.

The results of this study are now posted on-line in *Circulation*, the journal of the American Heart Association. The article is titled "Therapeutic Delivery of Cyclin A2 Induces Myocardial Regeneration and Enhances Cardiac Function in Ischemic Heart Failure." Co-authors are Corinna Panlilio; George Liao; Pavan Atluri; Vivian Hsu; and Jeffrey Cohen of Penn; as well as Richard Cheng and Hina Chaudhry of Columbia University.

Source: University of Pennsylvania School of Medicine

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