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Preventing a broken heart: Research aims to reduce scarring from heart attacks

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A heart damaged by heart attack is usually broken, at least partially, for good. The injury causes excessive scar tissue to form, and this plays a role in permanently keeping heart muscle from working at full capacity.

Now researchers have identified a key molecule involved in controlling excessive scar tissue formation in mice following a heart attack. When they stopped the scarring from occurring, the scientists found that the animals' heart function greatly improved following the injury.

The study, by scientists at the University of Wisconsin-Madison and Cornell University, appears in *Nature Cell Biology* online Dec. 14, 2008.

The findings offer heartening news for the millions who have heart attacks each year and suffer from the resulting poor heart function. The study raises the hope that the outlook for people with this major disability might be markedly improved.

The scientists studied a protein, sFRP2, which they unexpectedly found to be involved in the formation of collagen, the main component of scar tissue.

"With many injuries and diseases, large amounts of collagen are formed and deposited in tissues, leading to scarring and a condition called fibrosis," explains co-author Daniel S. Greenspan, professor of pathology and laboratory medicine at the UW School of Medicine and Public Health. "Fibrosis can seriously affect the functioning of heart,

lung, liver and other tissues."

Greenspan, an expert on collagen, joined with Thomas Sato of Weill Cornell Medical College to study mice that don't produce sFRP2 to understand how the protein works. When the scientists restricted blood flow to the animals' hearts, mimicking a heart attack, they found that scarring was significantly reduced in these sFRP2-free animals.

"Importantly, we found that when we reduced the level of fibrosis, heart function significantly improved in the mice," says Greenspan, also a professor of pharmacology at UW-Madison.

Identifying agents that specifically target sFRP2 and halt its activity will be a promising approach to controlling heart attack-induced scarring and impaired heart function, says Greenspan, and his lab has begun the search. The UW scientists also hope to study how sFRP2 and other proteins that enhance collagen formation may interact.

The protein may also be important in treating other diseases resulting in severe fibrosis, adds Greenspan, including liver cirrhosis and interstitial lung disease.

Source: University of Wisconsin-Madison