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Protein could be new target to reduce damage after heart attack

February 24 2011

Scientists have identified a protein that plays a key role in debilitating changes that occur in the heart after a heart attack, according to research reported in *Circulation Research: Journal of the American Heart Association*.

These changes, or "remodeling" of the heart, often lead to fatal heart failure, which kills nearly 60,000 Americans each year. The findings suggest a possible future therapy for preventing or reducing heart muscle damage after a heart attack.

Researchers compared the effect of heart attacks in two groups of mice. One group was genetically engineered to lack fibronectin-EDA (FN-EDA), a protein that exists in the space surrounding cells and is important for processes such as cell migration and wound healing. The other mice were genetically normal.

After inducing a heart attack in the left coronary artery of each mouse, the team found that the hearts of mice lacking FN-EDA had less enlargement in the left ventricle, better pumping ability and less thickening of the heart muscle) compared to the control mice.

At the tissue level, the genetically engineered mice also had less inflammation; diminished activity of the enzymes metalloproteinase 2 and 9, which are involved in heart remodeling; and reduced myofibroblast transdifferentiation (a process in which cells near an injury site transform into myofibroblasts, which are cells that help heal

injured tissue).

Bone marrow transplantation experiments revealed that the FN-EDA involved in the remodeling process came from the heart and not from cells circulating in the bloodstream.

Provided by American Heart Association