Gerald Auer
Public Relations and Event Management
Head of the Organizational Unit

Medical University of Graz
Neue Stiftingtalstraße 6
8010 Graz
gerald.auer@medunigraz.at

Press release
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Fibrosis as a common complication after myocardial infarction
Scientists find a potential way to support recovery

Graz, 22 August 2023: Increased deposition of connective tissue and thus excessive scarring of the damaged myocardium may occur after myocardial infarction. This so-called fibrosis gradually stiffens the myocardium so it is no longer able to work properly, which ultimately results in heart failure. Through international cooperation, Med Uni Graz researchers and their colleagues have now found a way to stop the excessive formation of connective tissue after a heart attack and thus contribute to optimal recovery.

Heart failure: Fibrosis causes the myocardium to stiffen

If interventional reopening of occluded coronary arteries occurs immediately or shortly after the heart attack, they can heal with hardly any consequences. If the heart tissue starts to die due to insufficient blood supply, the result is inflammatory processes in the body. Tissue cells that are already dead are "disposed of" by the immune system, while connective tissue cells migrate to and form scar tissue at these sites. "If this results in excessive formation of scar tissue—called fibrosis—there is a risk of heart failure due to the progressive stiffening of the myocardium and the loss of functioning muscle tissue. Heart failure is a disease associated with great psychological stress and a poor prognosis," says cardiologist Peter Rainer.

A publication that recently appeared in the renowned *Journal of the American College of Cardiology* describes international research that may have found one of the missing links between inflammation and scarring.

Optimal recovery from myocardial infarction as the goal of research

The researchers were able to show that immune cells that migrate to cardiac tissue after a heart attack secrete a protein (extracellular matrix protein 1—or ECM1). "This protein activates connective tissue cells that produce collagens and directly causes scarring," explains Peter Rainer. The scientists then identified a receptor on these cells that is most likely responsible for the observed effects (LRP1 receptor). In ischemic heart disease, ECM1 expression in the patients' hearts was significantly higher. ECM1 thus acts as a mediator between inflammatory and connective tissue cells that regulates wound healing and scarring. "Therapeutic manipulation of ECM1 and the underlying signaling pathway might improve recovery from infarction and impede excessive scarring," hopes Peter Rainer.

Med Uni Graz *Pioneering Minds*

Further information and contact
Peter Rainer
Division of Cardiology
Department of Internal Medicine
Medical University of Graz
Tel.: +43 664 1344513
E-Mail: peter.rainer@medunigraz.at

To the publication
Extracellular Matrix Protein-1 as a Mediator of Inflammation-Induced Fibrosis after Myocardial Infarction – JACC: Basic to Translational Science
https://authors.elsevier.com/sd/article/S2452-302X(23)00231-0