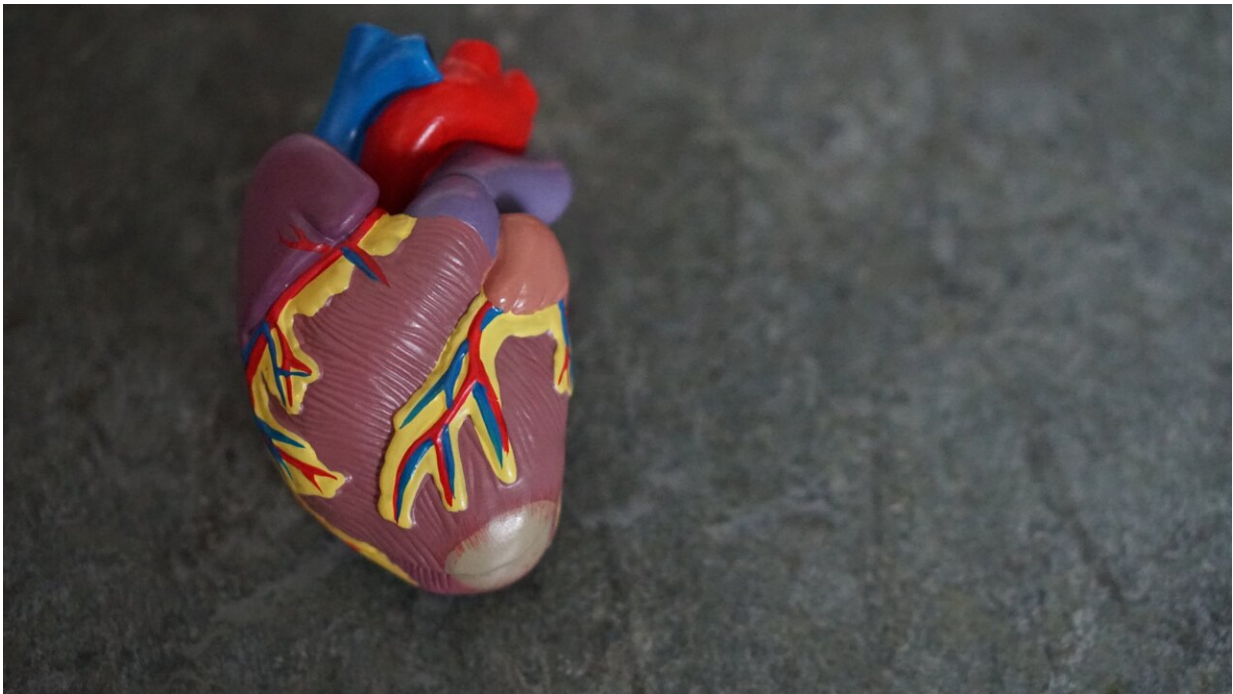


Exploring the recovery process after a heart attack

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A recent study published in the journal *Molecular Therapy - Nucleic Acids* on the harmful effects of acute myocardial infarction opens the door to the administration of a molecule (urocortin-2) as a potential treatment to regulate recovery processes after the ischemic event. The study has been led by Dr. Tarik Smani, the researcher in charge of the "Cardiovascular Physiopathology" group at IBiS and professor in the

Department of Medical Physiology and Biophysics at the University of Seville.

The project was carried out at the Institute of Biomedicine of Seville using heart samples from an [animal model](#), which simulates myocardial infarction, and biopsies from patients with [heart failure](#) in order to study the cardioprotective effect of the molecule urocortin-2 after acute [myocardial infarction](#).

After combining functional, biochemical and molecular techniques, the conclusions obtained point to urocortin-2 as a potential treatment to modulate miR-29a, a molecule that regulates different cellular functions, the expression of which increases significantly during infarction. Treatment with urocortin-2 could therefore favor patient recovery. The study demonstrates that urocortin-2, through miR-29a, regulates the expression of genes related to a type of cell death called apoptosis, suggesting that miR29a participates in the progressive adaptation of the heart to post-infarction stress.

MicroRNAs are already considered promising drug targets for disorders associated with coronary heart disease. The results obtained by the group of Dr. Smani open the door to devising new methods to treat and improving the well-being of heart attack patients.

More information: Isabel Mayoral-González et al, Cardiac protection induced by urocortin-2 enables the regulation of apoptosis and fibrosis after ischemia and reperfusion involving miR-29a modulation, *Molecular Therapy - Nucleic Acids* (2022). DOI: [10.1016/j.omtn.2022.01.003](https://doi.org/10.1016/j.omtn.2022.01.003)

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