

Genetic therapy heals damage caused by heart attack

May 8 2019



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Researchers from King's College London have found that therapy that

can induce heart cells to regenerate after a heart attack.

Myocardial infarction, more commonly known as a heart attack, caused by the sudden blocking of one of the cardiac coronary arteries, is the main cause of [heart failure](#), a condition that now affects over 23 million population in the world, according to the World Health Organisation.

At present, when a patient survives a [heart attack](#), they are left with permanent structural damage to their heart through the formation of a scar, which can lead to heart failure in the future. In contrast to fish and salamander, which can regenerate the heart throughout life.

In this study, published today in *Nature*, the team of investigators delivered a small piece of genetic material, called microRNA-199, to the heart of pigs, after a [myocardial infarction](#) which resulted in the almost complete recovery of cardiac function at one month later.

Lead author Professor Mauro Giacca, from King's College London said: "It is a very exciting moment for the field. After so many unsuccessful attempts at regenerating the heart using stem cells, which all have failed so far, for the first time we see real cardiac repair in a large animal."

This is the first demonstration that cardiac regeneration can be achieved by administering an effective genetic drug that stimulates cardiac regeneration in a large animal, with heart anatomy and physiology like that of humans.

"It will take some time before we can proceed to [clinical trials](#)" explained Professor Giacca.

"We still need to learn how to administer the RNA as a synthetic molecule in large animals and then in patients, but we already know this works well in mice".

More information: MicroRNA therapy stimulates uncontrolled cardiac repair after myocardial infarction in pigs, *Nature* (2019). [DOI: 10.1038/s41586-019-1191-6](https://doi.org/10.1038/s41586-019-1191-6) , www.nature.com/articles/s41586-019-1191-6

Provided by King's College London

Citation: Genetic therapy heals damage caused by heart attack (2019, May 8) retrieved 19 April 2024 from <https://medicalxpress.com/news/2019-05-genetic-therapy-heart.html>

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