

Blood cancer gene could be key to preventing heart failure

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Credit: University of Glasgow

A new study, published today in *Circulation*, shows that the gene Runx1 increases in damaged heart muscle after a heart attack. An international collaboration led by researchers from the University of Glasgow, found that mice with a limited capacity to increase Runx1 gene activation were protected against the adverse changes that lead to heart failure.

Coronary heart disease is the leading cause of death worldwide and, according to figures from the British Heart Foundation, is responsible for nearly 70,000 deaths in the UK each year. Most of these deaths are caused by a [heart attack](#) (myocardial infarction) where the blood flow to the heart is acutely blocked causing irreversible damage to the [heart muscle](#).

Over the last few decades, research-led improvements in healthcare mean that more people survive a heart attack. However, the damage that has occurred to the heart muscle can predispose these patients to go on to develop [heart failure](#) – a debilitating condition in which the heart cannot pump blood around the body as well

as it should.

The Runx1 gene has been extensively studied in the context of its role in leukaemia and normal blood cell development, however until now its role in the heart was unknown.

Now researchers believe that the increased expression of the Runx1 gene, which happens after a heart attack, contributes to adverse changes in the shape and pumping action of the heart.

Lead author of the study, Dr Christopher Loughrey, from the University of Glasgow's Institute of Cardiovascular and Medical Sciences, said: "We are very excited by our findings related to the Runx1 gene and myocardial infarction. Our study shows that this gene, which is well-known in relation to blood cancers, has a key part to play in [heart muscle damage](#) after a heart attack.

Currently, more than 500,000 people across the UK have been diagnosed with heart failure and despite healthcare advances, mortality rates continue to be high. New therapeutic options to limit the development of heart failure in patients who have had a heart attack are urgently required.

Dr Loughrey said: "The study not only describes a novel role for Runx1 in the heart but also provides us with a new therapeutic strategy with great potential to improve the ability of the heart to pump and thereby limit the development of heart failure in patients who have survived a [heart](#) attack."

The paper, "Runx1 deficiency protects against adverse cardiac remodelling following [myocardial infarction](#)," is published in *Circulation*.

Provided by University of Glasgow

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