

# The memory of a heart attack is stored in our genes

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Credit: NIH

Both heredity and environmental factors influence our risk of cardiovascular disease. A new study, by researchers at Uppsala University, shows now that the memory of a heart attack can be stored in our genes through epigenetic changes. The results have been published in the journal *Human Molecular Genetics*.

We inherit our [genes](#) from our parents at birth. During our lifetime, chemical modifications of DNA that activate or deactivate genes, so-called [epigenetic changes](#), occur. These changes can lead to the



development of various diseases. In the current study, the researchers examined epigenetic changes in people who have had a previous heart attack.

'During a heart attack, the body responds by activating certain genes. This mechanism protects the tissue during the acute phase of the disease and restores the body after the heart attack. It is therefore likely that epigenetic changes associated with heart attack also occur,' says Åsa Johansson, a researcher at the Department of Immunology, Genetics and Pathology, who led the study.

The results of the study showed that there are many epigenetic changes in individuals who experienced a heart attack. Several of these changes are in genes that are linked to [cardiovascular disease](#). However, it was not possible to determine whether these differences contributed to the development of the disease, or if they live on as a memory of gene activation associated with the heart attack.

'We hope that our new results should contribute to increasing the knowledge of the importance of epigenetic in the clinical picture of a [heart attack](#), which in the long run, could lead to better drugs and treatments,' says Åsa Johansson.

**More information:** Mathias Rask-Andersen et al. Epigenome Wide Association Study Reveals Differential DNA Methylation in Individuals With a History of Myocardial Infarction, *Human Molecular Genetics* (2016). [DOI: 10.1093/hmg/ddw302](https://doi.org/10.1093/hmg/ddw302)

Provided by Uppsala University

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