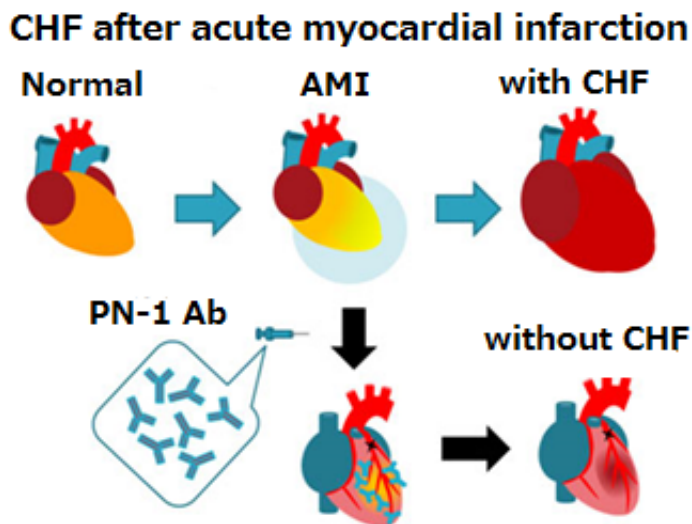


# Towards the prevention of cardiac failure in the chronic phase

January 25 2016

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Congestive heart failure (CHF) after acute myocardial infarction

The onset of cardiac failure after acute myocardial infarction (AMI) is a serious problem throughout the world.

Researchers at Osaka University clarified that the cell adhesion inhibition of periostin1 damages myocardial cells, inducing compromised cardiac myocyte contractile force and myocytes death, leading to the onset of [cardiac failure](#) after AMI through the administration of periostin neutralizing antibodies they had developed on their own.

If the onset of cardiac failure in the chronic phase can be inhibited by this group's research results that will not only improve patients' quality of life, but also reduce high medical costs of cardiac failure treatment as a whole.

Yoshiaki Taniyama, Endowed Chair Associate Professor, Ryuichi Morishita, Endowed Chair Professor, and Fumihiro Sanada, Specially Appointed Assistant Professor at Osaka University focused their attention on periostin, a protein secreted mainly from fibroblasts by mechanical stretch (mechanical stress) after the onset of AMI.

Acute [myocardial infarction](#) generates [mechanical stress](#) on the heart, which causes secretion of periostin from fibroblasts and promotes cardiac failure (Figure 1); however, its mechanism was unclear. There are four splice variants of periostin gene, and Associate Professor Taniyama et al. have reported that periostin1 solely induced cardiac failure. Furthermore, researchers at Harvard University, the University of Cincinnati, and Tokyo Institute of Technology reported that inhibition of all periostins suppressed cardiac failure after AMI, but periostin2 and periostin4 had cardioprotective effects such as myocardial regeneration, angiogenesis, and protection of cardiac rupture after AMI. The suppression of all periostin variants inhibits cardiac failure, but increases death by cardiac rupture in the acute phase after AMI.

Thus, this group considered a therapeutic method in which not periostin2 and periostin4 with cardioprotective effects but periostin1 with cardiac failure -inducing effects was suppressed. This group independently developed periostin1-specific neutralizing antibodies and administered them to patients after the onset of [acute myocardial infarction](#), thereby succeeding in inhibiting the onset of cardiac failure in the chronic phase without the accompaniment of cardiac rupture.

**More information:** Selective Blockade of Periostin Exon 17 Preserves

Cardiac Performance, *Hypertension*, 2016.

Provided by Osaka University

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