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## **A new monoclonal antibody for heart attack and ischemic heart disease therapy**

Cardiovascular disorders are the leading cause of death and the greatest healthcare cost worldwide. In Italy, over 20 billion euros a year are spent on treating these disorders. And the numbers are set to grow over the coming years.

A new study published in *Nature Communications* [1] has shown the efficacy of a new biological drug, a monoclonal antibody capable of blocking fibrosis and protecting the heart muscle after myocardial infarction. The study showed that this antibody has a beneficial effect by means of a double mechanism: on the one hand, it reduces the deposition of fibrous tissue which limits the heart's pump function, and on the other, it promotes the survival of heart muscle cells.

The study, led by Serena Zacchigna, professor of Molecular Biology at the University of Trieste and head of the Cardiovascular Biology lab of the International Centre for Genetic Engineering and Biotechnology (ICGEB) in Trieste, represents a turning point in the sector of innovative cardiovascular therapies.

"In contrast to the great social and healthcare pressure exerted by these diseases, the drugs we use to treat patients with heart disease are rather dated. New biological therapies" - explains Zacchigna - "are transforming oncological or hereditary disease treatments, while there are very few biological drugs for the treatment of cardiovascular disorders. The vast majority of therapies approved to date are small chemical molecules that generally have a single target, blocking the action of an enzyme or receptor, for example. By contrast, biological drugs (recombinant proteins, gene therapy products and cell therapy) reproduce elements that normally exist in our tissues and therefore, have the potential to interfere with complex therapy mechanisms. However, they are more difficult to prepare and use, as well as being more expensive, making them complicated to translate from experimental studies to patients".

This study, which is the result of a long collaboration between the Trieste institutes (ICGEB and University of Trieste) and the University of Zagreb, Croatia, reveals the fundamental role of a family of proteins, called Bone Morphogenetic Proteins (BMPs), in the evolution of cardiac fibrosis after an ischemic event.

For years, the Croatian team has been a centre of excellence for the study of BMPs which, as the name implies, play a key role in bone formation, but which have recently also been implicated in other processes, such as fibrosis.

"Having been able to collaborate with our Croatian colleagues" - says Andrea Colliva, first author of the study and UniTS researcher working at ICGEB - "has allowed us to test the effectiveness of a monoclonal antibody which blocks a particular version of BMP protein (BMP1.3), whose levels are particularly high in patients brought to the emergency room for a myocardial infarction".

In the last phase of the project, a group of cardiac surgeons from Innsbruck joined the Trieste-Zagreb axis and contributed their experiences and skills to the mechanisms underlying ischemic damage and the development of innovative therapies. This was made possible by the INCardio project - Innovative Therapies for Cardiovascular Disorders, led by ICGEB and financed by the European Regional Development Fund and by Interreg V-A Italy-Austria 2014-2020. "The collaboration with the Austrian colleagues", continues Colliva,

“will be fundamental to validate the results of this study in other contexts of ischemia and fibrosis and to pave the road towards a possible clinical application of these results”.

Promoting cross-border innovation in the treatment of cardiovascular disorders is precisely the main objective of the INCardio cross-border project, which has consolidated the project and unites about thirty researchers working in the sector. "We are confident that this work will open the door to other biological drugs in the cardiovascular sector" concludes Zacchigna, "as we need cooperation and collaboration among different skills to ensure that the study results can reach patients and that this can also be applied in Italy".

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**References:**

[1] Bone morphogenetic protein 1.3 inhibition decreases scar formation and supports cardiomyocyte survival after myocardial infarction

<http://www.nature.com/ncomms>

Further information on the INCardio project: [www.incardio.eu](http://www.incardio.eu)