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I-1c gene therapy: Not such a good idea in heart failure?

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Several lines of evidence, including the observation that the protein I-1 is downregulated in human failing hearts, have led to the suggestion that gene therapy to express a constitutively active form of the protein (I-1c) might provide a new approach to treating heart failure.

However, Ali El-Armouche, Thomas Eschenhagen, and colleagues, at University Medical Center Hamburg-Eppendorf, Germany, have now generated data in mice indicating that I-1c might have deleterious effects on the heart under certain circumstances, leading them to suggest that the benefit/risk ratio of I-1c gene therapy should be reevaluated.

In the study, I-1-deficient mice were engineered to express I-1c in heart muscle cells (dTGI-1c mice). The hearts of young, resting dTGI-1c mice showed enhanced contractile function. However, when the mice were infused with catecholamine, a hormone released by the body in response to stress, they developed abnormal heartbeats and were susceptible to sudden death.

Furthermore, the hearts of aged dTGI-1c mice were found to spontaneously develop the characteristic features of heart failure. As heart failure tends to be a disease of the elderly, the authors suggest that their data need to be considered by those developing I-1c gene therapy for the treatment of heart failure.

More information: Constitutively active phosphatase inhibitor-1 improves cardiac contractility in young mice but is deleterious after

catecholaminergic stress and with aging. View this article at:
www.jci.org/articles/view/4054 … 6aaaec79fcaa30d87e28

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