**DEFICIENCY OF FILAMIN A IN ENDOTHELIAL CELLS IMPAIRS LEFT VENTRICULAR REMODELING AFTER MYOCARDIAL INFARCTION**

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*Objectives and Background*: Actin-binding protein filamin A (FLNA) regulates signal transduction important for cell locomotion, but the role of FLNA after myocardial infarction (MI) has not been explored. The main purpose of this study was to determine the impact of endothelial deletion of FLNA on post-MI remodeling of the left ventricle (LV).

*Methods and Results*: We found that FLNA is expressed in human and mouse endothelial cells during MI. To determine the biological significance of endothelial expression of FLNA, we used mice that are deficient for endothelial FLNA by crossbreeding adult mice expressing floxed Flna (Flnao/fl) with mice expressing Cre under the vascular endothelial-specific cadherin promoter (VECadCre+). Male Flna(o/fl) and Flna(o/fl)/VECadCre+ mice were subjected to permanent coronary artery ligation to induce MI. Flna(o/fl)/VECadCre+ mice that were deficient for endothelial FLNA exhibited larger and thinner LV with impaired cardiac function as well as elevated plasma levels of NT-proBNP and decreased secretion of VEGF-A. The number of capillary structures within the infarcted areas was reduced in Flna(o/fl)/VECadCre+ hearts. Endothelial cells silenced for Flna mRNA expression exhibited impaired tubular formation and migration, secreted less VEGF-A, and produced lower levels of phosphorylated AKT and ERK1/2 as well as active RAC1.

*Conclusions*: Deletion of FLNA in endothelial cells aggravated MI-induced LV dysfunction and cardiac failure as a result of defective endothelial response and increased scar formation by impaired endothelial function and signaling.