# Changes in HSPA9 in the failing heart following myocardial infarction

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In the failing heart following myocardial infarction (MI), mitochondrial dysfunction induces myocardial apoptosis and cardiac pump failure. Heat shock protein (HSP) A9 contributes to mitochondrial homeostasis. However, pathophysiological roles of HSPA9 during the development of heart failure following MI remain unclear. In this study, we assessed changes in HSPA9 in the left ventricle (LV) during the development of heart failure following MI. MI was induced by the left coronary artery ligation (CAL). At 8th week after CAL (8W-CAL), cardiac output index and mitochondrial activity were reduced. HSPA9 in the mitochondria-enriched fraction of the 8W-CAL animals were determined. In the cytosolic and nuclei-enriched fractions, HSPA9 of the 8W-CAL animals were increased. In the co-immunoprecipitation of the LV, the apoptotic signaling protein p53 in the HSPA9 immunocomplex of the 8W-CAL animal was increased. These findings suggest that HSPA9 accumulation in the mitochondria contributes to keep the LV function after MI. In contrast, the accumulation of the complex of HSPA9 and p53 in cytosolic and nucleic fractions is a possible mechanism to induce apoptosis of cardiomyocytes during the development of heart failure following MI.