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Fatty Acid Metabolism in Hypertension

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Abstract : Cardiac metabolism is essential in myocardial contraction. In addition to glucose, fatty acids (FA) are essential in producing energy in the myocardium since FA-dependent beta-oxidation accounts for > 70-90% of cellular ATP under resting conditions. However, metabolism shifts from FAs to glucose utilization during disease progression (e.g. hypertrophy and ischemic myocardium), where glucose oxidation and glycolysis become the predominant sources of cellular ATP. At advanced failing stage, both glycolysis and beta-oxidation are dysregulated, result in insufficient supply of intracellular ATP and weakened myocardial contractility. Undeniably, our understandings of myocyte function in healthy and diseased hearts are based on glucose (10 mM)-dependent metabolism because glucose is the “sole” metabolic substrate in most of the physiological experiments. In view of the importance of FAs in cardiovascular health and diseases, we aimed to elucidate the impacts of FA supplementation on myocyte contractility and evaluate cellular mechanisms those mediate the functions in normal heart and with pathological stress. In particular, we have investigated cardiac excitation-contraction (E-C) coupling in the presence and absence of FAs in normal and hypertensive rat left ventricular (LV) myocytes. Our results reveal that FAs increase mitochondrial activity, intracellular $[Ca^{2+}]i$, and LV myocyte contraction in healthy LV myocytes, whereas FA-dependent cardiac inotropyis attenuated in hypertension. FA-dependent myofilament Ca^{2+} desensitization could be fundamental in regulating $[Ca^{2+}]i$. Collectively, FAs supplementation resets cardiac E-C coupling scheme in healthy and diseased hearts.

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