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Effect of Cardio-Specific Overexpression of MUL1, a Mitochondrial Protein on Myocardial Function

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Abstract : MUL1, a mitochondrial E3 ubiquitin ligase anchored to the outer mitochondrial membrane, is highly expressed in the heart. MUL1 is involved in multiple biological pathways associated with mitochondrial dynamics. Increased MUL1 affects the balance between fission and fusion, affecting mitochondrial function, which plays a crucial role in myocardial function. Therefore, it is interesting to evaluate the effect of cardiac-specific overexpression of MUL1 on myocardial function. Aim: To determine heart functionality in a mouse model with cardio-specific overexpression MUL1 protein. Methods and Results: Male C57BL/Tg transgenic mice with cardiomyocyte-specific overexpression of MUL1 (n=10) and control (n=4) were evaluated at 12, 27, and 35 weeks of age. Glucose tolerance curve determination was performed after a 6-hours fast to assess metabolic capacity, treadmill test, and systolic, and diastolic pressure was evaluated by the mouse tail-cuff blood pressure system equipment. The result showed no glucose tolerance curve, and the treadmill test demonstrated no significant changes between groups. However, substantial changes in diastolic function were observed by ultrasound and determination of cardiac hypertrophy proteins by western blot. Conclusions: Cardio-specific overexpression of MUL1 in mice without any treatment affects diastolic cardiac function, thus showing the important role contributed by MUL1 in the heart. Future research should evaluate the effect of cardiomyocyte-specific overexpression of MUL1 in pathological conditions such as a high-fat diet is one of the main risk factors for cardiovascular disease.

Keywords : diastolic dysfunction, hypertrophy cardiac, mitochondrial E3 ubiquitin ligase 1, MUL1
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