Investigating the Impact of Pyruvate Kinase Muscle Isoform 2 (PKM2) Knockout on Cardiac Murine Cellular Hypertrophy

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Myocardial infarction (MI), or heart attack, is the death of cardiac tissue to improper blood and oxygen flow. MI is a form of cardiovascular disease, the current leading cause of death, according to the World Health Organization. Post-MI, surviving cardiomyocytes must depend on the less efficient anaerobic process of glycolysis for energy. Pyruvate kinase is the final enzyme of glycolysis, which starts a positive feedback mechanism loop to harness as much energy as possible in cells. It was found that three days post-MI, while pyruvate kinase levels decrease, the typically less active and effective isoform of pyruvate kinase, pyruvate kinase muscle isoform 2 (PKM2), is upregulated, while pyruvate kinase muscle isoform 1 (PKM1), the more active and effective isoform is downregulated. To confirm the role that PKM2 has on heart physiology three days post-MI, average cardiomyocyte area amongst PKM2 flox/flox and PKM2 knockout mice will be analyzed. It was identified that the knockout of PKM2 does not cause any physiological changes three days post-MI. With a validated understanding of the immediate impact of PKM2, scientists are redirected towards another approach in post-MI recovery research.

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