

had unlimited access to food, whereas those in the fasted group were deprived of food for 18 h prior to the start of the experiment. The myocardial infarction experiment in isolated hearts from fed (n=10) and fasted (n=10) rats was performed. To determine the accumulation of glucose and palmitate metabolites in mitochondria, hearts were perfused with radiolabelled glucose and palmitate. The preference of substrates in isolated cardiac mitochondria was determined using radiolabelled substrates. Lactate and pyruvate oxidation rates were measured in the presence or absence of different concentrations of palmitoyl-carnitine, while palmitate oxidation rate was measured in the presence or absence of lactate or pyruvate. Permeabilized cardiac fibers were used to measure mitochondrial respiration on pyruvate+malate and palmitoyl-CoA.

Infarct size in the fed state was 2.7-fold smaller than in the fasted state. Mitochondrial respiration was increased by 20% in the fed state when pyruvate+malate were used as substrates, but no difference was observed when palmitoyl-CoA was used as a respiratory substrate. The content of FA metabolites in mitochondria in the fasted state was 3.5-fold higher than in the fed state. Meanwhile no changes in glucose metabolites were observed in fed and fasted states. Measurements of pyruvate, lactate and palmitate oxidation in mitochondria showed that the oxidation rates are highly dependent on the concentration for these three substrates. Palmitoyl-carnitine effectively reduced the rates of pyruvate and lactate oxidation in mitochondria in a dose-dependent manner. In contrast, only pyruvate, but not lactate, significantly reduced the palmitate oxidation rate in mitochondria.

Our results demonstrate that cardiac recovery from ischemia-reperfusion injury is improved in the fed state due to enhanced lactate and pyruvate (glucose) metabolism. Altogether our results suggest that the activated long chain FAs content within the mitochondria determines the rates of pyruvate and lactate metabolism, particularly in the fasted state.

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**THE SEVERITY OF ISCHEMIA REPERFUSION DAMAGE IS DETERMINED BY THE CHANGES IN METABOLISM PATTERN OF ENERGY SUBSTRATES IN MITOCHONDRIA**

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doi:10.1136/heartjnl-2013-304019.258

Healthy heart is able to switch appropriately between glucose and fatty acids (FAs) to meet the energy requirements. Due to rapid energy turnover and low energy storage capacity, myocardial functioning relies on energy substrate levels in blood plasma. Availability and utilization of energy substrates can determine the outcome of cardiovascular events, particularly ischemia related, where stimulated glucose metabolism could be preferable. We hypothesize that changes in metabolism pattern of glucose and FAs in mitochondria determine the outcome of ischemia-reperfusion injury in the fed and fasted states.

Male Wistar rats were randomly separated into two experimental groups, fed (n=18) and fasted (n=18). Rats in the fed group