Cardiac and Skeletal Muscle Lipotoxicity in a Rat Model of Pulmonary Arterial Hypertension

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Patients with Pulmonary Arterial Hypertension (PAH)experience shift from aerobic to anaerobic respiration in cardiac and skeletal muscle myocytes. This shift can be identified histologically by an increased presence of the glucose transporter Glut 1 in the tissue, indicating increased reliance on cytoplasmic glycolysis. Previous studies have demonstrated that in patients with diabetes, an increase in Glut 1 is accompanied by an increase in fat storage in the cell. Excessive myocyte fat storage may contribute to tissue and systemic inflammation and has therefore been termed ‘lipotoxicity’. This study tested the hypothesis in a PAH rat model that an increase in cardiac and skeletal muscle Glut 1 abundance would be associated with an increase in fat storage in the cell. Oil Red O staining was performed to assay for lipotoxicity in cryosections of right ventricle and soleus muscle tissue, imaged using brightfield microscopy. The experiment was conducted using tissue from a moderately severe PAH phenotype produced by monocrotaline(60 mg/kg) injection, as well as from saline injected control animals. Lipids have been observed in the first few samples tested with Oil Red O staining, and results are still pending as a larger sample size is currently being collected.

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