Abstract
Understanding the Impact of UCP2 on Metabolism using Metabolomic Approaches
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Uncoupling protein 2 (UCP2) belongs to a family of mitochondrial inner membrane proteins and has been reported to play a protective role in acetaminophen-induced hepatotoxicity and atherosclerosis. This protein has also been reported to play a role in neurological disorders, obesity and nonalcoholic fatty liver disease. While the role of UCP2 in disease pathogenesis has been extensively studied, its role under normal physiological conditions, particularly in the liver, has not been characterized. It is demonstrated that UCP2 function, increased in the presence of fatty acids suggesting a role in fatty acid catabolism, decreases reactive oxygen species (ROS) production in a variety of tissues. ROS present at the mitochondrial membrane can cause lipid peroxidation and result in reactive alkenals. Our goal is to use metabolomic approaches in cell and mouse models to determine whether UCP2 activity can reduce lipid peroxide levels and impact fatty acid catabolism. Gas chromatography coupled with mass spectrometry will be used to assess lipid peroxidation levels while ^{1}H nuclear magnetic resonance will be used to examine the global effects of UCP2 activity on metabolism. We hypothesize that UCP2 reduces reactive alkenals resulting from lipid peroxidation and increases fatty acid catabolism in the liver.