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Throwing the kitchen sink at heart metabolism

Matthew E Merritt
University of Florida, USA

The heart is an omnivorous organ, able to metabolize fatty acids (FAs), carbohydrates and ketones to acetyl-CoA depending on their availability in the bloodstream. When the heart is subjected to overpressure due to stenosis or high blood pressure, it can hypertrophy and ultimately fail. This physical phenomenon is intimately connected with a change in the substrate preference for the heart, where carbohydrate oxidation is emphasized over FA oxidation. Measuring substrate preference in the heart is tractable when nutrients positionally enriched in ^{13}C can be supplied. Here, we use a standard model of myocardial metabolism, the perfused mouse heart to study substrate selection and the impact of providing propionate, an odd-chain fatty acid on overall metabolism. We have found that propionate activates carbohydrate oxidation leading to avid utilization of a hyperpolarized pyruvate tracer. Additionally, the propionate can cause a large change in pool sizes for the Krebs cycle intermediates without enforcing a change in flux as measured by O_2 consumption. The propionate perfused heart should serve as an excellent model for validation of new isotopomer based methods for measuring metabolic flux.

Biography

Matthew E Merritt has completed his PhD in Physical Chemistry at Washington University in St. Louis in 1996. He has completed his Post-doctoral training with Dr. Gary Drobny at University of Washington in 2000 and joined the Staff at UT Southwestern Medical Center. In 2008, he became an Assistant Professor (tenure track) and achieved the rank of Associate Professor in 2015. In August 2015, he joined the Faculty of University of Florida, Department of Biochemistry and Molecular Biology.

matthewmerritt@ufl.edu

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