Oct1: Role in Thiamine Transport and Liver Steatosis
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Organic cation transporter, OCT1 (SLC22A1), is the major hepatic transporter for the anti-diabetic drug, metformin, and plays a role in the disposition of many other prescription drugs. In this presentation, I will describe how OCT1 plays a major role in hepatic thiamine disposition and metabolic function. First I will show how deletion of Oct1 in mice activates the energy sensor AMP-activated kinase (AMPK), and reduces fatty acid synthesis while increasing fatty acid oxidation. These effects result in reduced hepatic steatosis, and hepatic thiamine deficiency. I will also describe how reduced OCT1 function affects the onset and time course of beriberi in mice fed thiamine deficient diets. Collectively I will describe how OCT1, which has been primarily thought to function as a drug transporter, plays a critical role in thiamine disposition as well as in energy homeostasis. My presentation will suggest that thiamine supplementation in individuals with reduced function polymorphisms of OCT1 may have beneficial effects clinically on carbohydrate and lipid metabolism.