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2<sup>nd</sup> International Conference and Expo on

# **Lipids: Metabolism, Nutrition & Health**

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### Sphingosine-1-phosphate signaling regulates key metabolic outcomes of obesity

Sphingosine-1-phosphate is a lysophospholipid signaling molecule that works through both receptors dependent and independent mechanisms. Sphingosine-1-phosphate is generated via phosphorylation of sphingosine by sphingosine kinases. We discovered that sphingosine kinase 1 is upregulated by saturated fatty acids, a finding which placed this pathway in the context of metabolic homeostasis. Since those initial studies, our group and others have demonstrated roles for sphingosine kinase 1 in pathophysiological processes in numerous organs including pancreas, skeletal muscle, adipose tissue and liver. Moreover, ablation of this pathway in mice conferred resistance to obesity-induced inflammation and insulin resistance. Together, these data have supported the general idea that sphingosine kinase 1 mediates obesity-induced disease. To gain more mechanistic insights, we recently generated mice with an adipocyte-specific sphingosine kinase 1 deletion. Data from these animals has revealed novel and surprising roles for this pathway in maintaining metabolic homeostasis. For example, in contrast to the whole-body deletion, which exhibits protection from obesity-induced insulin resistance, the adipocyte-specific sphingosine kinase 1 deletion mouse exhibits basal insulin resistance. These and other data have revealed that this pathway serves both protective and deleterious roles depending on cell type and disease context. Ongoing work in the laboratory is addressing the specific roles for sphingosine-1-phosphate signaling in regulating crucial adipocyte functions including lipogenesis, lipolysis, proliferation and differentiation. These pathways might eventually be pharmacologically targeted for treatment of obesity-induced disease.

#### **Biography**

L Ashley Cowart has obtained her PhD in 2001 from Vanderbilt University and has done her Post-doctoral studies at the Medical University of South Carolina, where she is now an Associate Professor in Biochemistry. She has participated in developing the strong research programs in Lipid Signaling and Lipidomics at MUSC. She has published over 40 manuscripts and authored numerous reviews in Sphingolipid Metabolism and Signaling in Diabetes.

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