

# **COLLOQUIA IN CELLULAR SIGNALLING**

Venue: Medical University Vienna, Center for Biomolecular Medicine and Pharmacology,  
Institute of Physiology, Schwarzspanierstraße 17, 1090 Vienna,

## **"Big Lecture Hall Physiology"**

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Friday 14.12.2012 11:00 c.t. **Michael Roden** (host: Christian Nanoff)  
Director German Diabetes Center  
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### ***"Mechanisms of altered energy metabolism in human insulin resistance and type 2 diabetes"***

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#### Abstract:

Insulin resistance in skeletal muscle and liver is a central feature of humans at risk of or with overt type 2 diabetes. Decreased systemic glucose disposal and elevated endogenous glucose production during insulin stimulation characterize insulin resistance. These alterations occur in parallel with augmented lipid storage in both liver and skeletal muscle suggesting impaired lipid oxidation capacity. Indeed, patients with obesity, type 2 diabetes and their nondiabetic first-degree relatives can exhibit reductions of mitochondrial oxidative phosphorylation capacity, submaximal ADP-stimulated oxidative phosphorylation, plasticity of mitochondria and/or lower mitochondrial content in skeletal muscle and liver. Inherited insulin resistance is frequently associated with reduced mitochondrial activity at rest, probably due to diminished mitochondrial content. Acquired insulin resistance can be associated with reduced insulin-stimulated mitochondrial activity as the result of blunted mitochondrial plasticity, but may also occur independently of changes in mitochondrial function. In particular, augmented lipid availability can rapidly induced insulin resistance even in healthy humans without risk of type 2 diabetes. The associations between energy metabolism and insulin sensitivity will be discussed with a focus on studies in humans.