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Topic Category: 4092-ASIP Metabolic disorders

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Presentation Preference: Oral

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Keywords: 1. Ketones 2. Insulin 3. Mitochondria

Insulin and ketones elicit disparate effects on mitochondrial uncoupling in adipose tissue

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Objective. Insulin is a powerful regulator of metabolic function, promoting a general storage of nutrients. In contrast, ketones, inversely regulated by insulin, lead to enhanced energy use and wasting. The purpose of this study was to explore the effects of insulin and ketones on mitochondrial coupling status in adipose tissue.

Methods. We used cell, rodent, and human models, to varying degrees. 3T3-L1 adipocytes were treated at various times with insulin or ketones. Similarly, rodents were chronically injected with insulin or placed on ketogenic diets. Finally, human fat biopsies were obtained from in states of ketosis. In all models, mitochondrial respiration was determined and, where available, uncoupling protein (UCP) 1 was measured.

Results. In cells and rodents, insulin reduced mitochondrial respiration and, in rodents, reduced indirect calorimetry. In all models, including human adipose, ketones increased respiration rates and UCP1 levels.

Conclusions. Insulin and ketones elicit disparate effects on mitochondrial uncoupling in adipose tissue. Whereas insulin reduces uncoupling, resulting in more tightly coupled electron transport, ketones enhanced uncoupling, resulting in energy wasting.