Special Issue

Regulation of Dietary Glucose Energy Partition

Message from the Guest Editors

High-energy diets induce pathologies (e.g., metabolic syndrome, MS) in humans, and in animals used in experiments. Excess N intake creates certain disposal problems, whereas massive lipid intake (a 'human' discovery) lacks specific mechanisms to prevent this excess energy, resulting in unwanted fat accrual. There is a lack of molecular-based quantitative studies on what causes the MS-driven conversion of dietary glucose to fat but preserving dietary fat for triacylglycerol deposition, both related to altered insulin (and glucocorticoid) function. Testosterone and estradiol effects on the use of glucose for energy may provide additional clues regarding how to counteract inflammation in MS when using high-energy diets (largely fat) destabilizing insulin–glucocorticoid regulation. Nevertheless, it remains critical to quantitatively evaluate how much dietary glucose is needed to maintain energy homeostasis.

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