

Metabolic Flexibility, Energy Availability, and Substrate Utilisation: Extending the EPK Framework Beyond Static Calorie Models

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Abstract

Human fat gain obeys energy balance, but carbohydrate and dietary fat are not metabolically identical. Under ordinary mixed-diet conditions, net de novo lipogenesis from carbohydrate is generally limited, whereas dietary fat can be stored efficiently during energy excess. Carbohydrate can still promote fat storage indirectly by suppressing fat oxidation. This paper asks whether chronic low-energy physiology, reduced metabolic throughput and a high dietary-fat proportion may combine to impair metabolic flexibility.^{1,2,3,4,5,6}

De Novo Lipogenesis and Fat Storage

Classic overfeeding studies show that carbohydrate is preferentially oxidised and stored as glycogen before large-scale de novo lipogenesis becomes quantitatively important. Dietary fat can enter adipose storage with relatively little conversion. The implication is not that carbohydrate cannot contribute to fat gain; during energy surplus it can suppress fat oxidation and thereby spare dietary fat for storage.^{1,2,3}

Low-Energy Physiology and Substrate Competition

Restriction can lower resting expenditure, spontaneous movement and thermogenesis. If a low-calorie diet remains high in fat percentage, carbohydrate availability may become low at the same time as total physiological output is falling. The Randle cycle provides a mechanism through which high fatty-acid availability can suppress glucose oxidation. This is normal physiology, but chronic inflexibility may become relevant when low movement, high fatty-acid exposure and poor glucose utilisation coexist.^{7,8,4,5,6}

PUFA and Oxidative Stress

Highly unsaturated fatty acids are susceptible to lipid peroxidation, and oxidised lipid products can affect membranes, signalling and mitochondria. These mechanisms justify research into

fatty-acid composition in aging and metabolic dysfunction. They do not justify the stronger claim that PUFA simply blocks cellular respiration or directly causes obesity.⁹

EPK Strategy and Conclusion

The EPK model seeks to restore sufficient energy relative to mass and demand while providing enough carbohydrate to support glucose oxidation, glycogen restoration, movement and recovery. Stored fat remains an important fuel during lower-demand and fasting periods, including overnight. The hypothesis is that a well-powered, metabolically flexible system can oxidise carbohydrate when appropriate and draw on stored fat when appropriate rather than continually supplying large amounts of dietary fat in a low-throughput state.^{4,5,6}

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Publication Note

This manuscript is part of the EPK (Energy Per Kilogram) paper series developed by Billy Craig, MSc. The paper should be cited as a working/preprint paper until peer-reviewed publication.

EPK-specific propositions are presented as hypotheses where they extend beyond the cited evidence.