Abstract:

Evolution has led to metabolic thrift in humans - a genetic heritage that, when exposed to the modern "obesogenic" milieu with energy dense food and a sedentary lifestyle, predispose to obesity. The current paradigm that overeating of easy digestible carbohydrates and the resulting imbalance between energy in and out as the cause of overweight has recently been challenged. Indeed, studies suggest that the host response to various nutrients contribute to overeating and fat accumulation. Alterations in neurotransmitter functions, changes in the epigenome, dysbiosis of gut microbiota and effects of specific nutrients (or lack of such nutrients) on mitochondrial function and signaling pathways, may promote fat accumulation independent of calories. Whereas nutrients that stimulate generation of uric acid (such as fructose and purine rich food) cause insulin resistance and fat accumulation, other nutrients (such as anti-oxidants, plant food, probiotics, nuts, soy, and omega-3) counteract the negative effects of a calorie-rich diet by effects on mitochondrial biogenesis. Thus, the specific metabolic effects of different nutrients may be more important than the total energy content. By studying the impact of nutrients on mitochondrial health, as well as the trans-generational impact of nutrients during fetal life, and how specific bacterial species correlate with fat mass accumulation, new dietary targets for obesity management may emerge. Overeating and overshooting of calories could to a large extent represent a symptom rather than a cause of obesity; therefore, hypocaloric diets should probably not be the main, and certainly not the only, focus for treatment.

Key words: obesity, insulin resistance, epigenome, mitochondra, gut microbiota, anti-oxidants