# Hyper-Sedentary Behavior, Energy Balance, Adipogenic Nutrient Partitioning and the Etiology of Obesity

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## ABSTRACT

**BACKGROUND:** The increased prevalence of obesity over the past decades is not adequately explained by an increase in positive energy balance. The current paradigm of 'move more and eat less' to combat obesity is derived from deterministic theories in which positive energy balance leads to an obligate increase in BMI. The ineffectiveness of interventions which focus on energy expenditure suggests that other causal pathways be investigated. The role of **adipogenic nutrient partitioning (ANP)** (i.e., the storage vs. oxidation of metabolic substrates) induced via **hyper-sedentary behavior (HSB)** (i.e., chronic extended unloading of postural musculature) has not been examined.

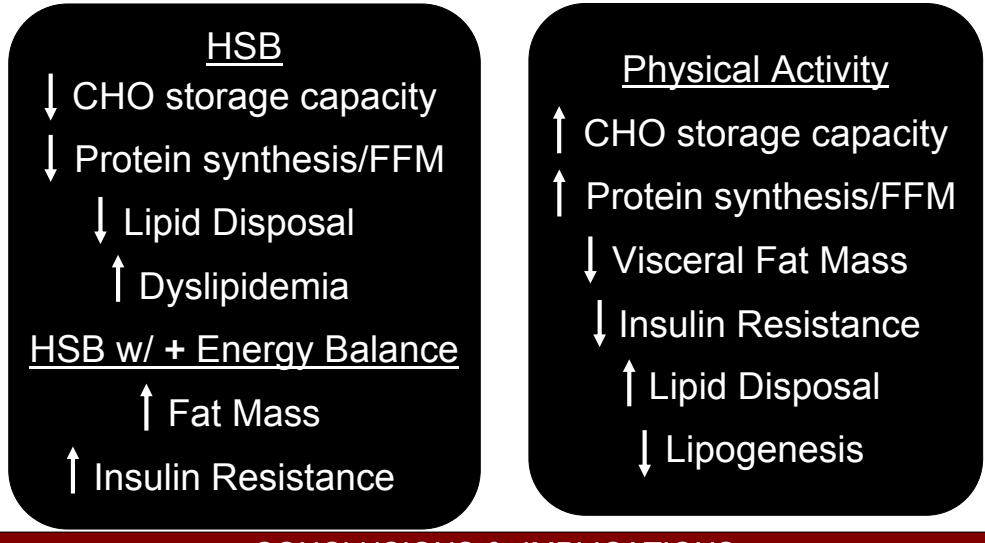
**PURPOSE: 1)** To explore a novel perspective on the obesity epidemic in which energy imbalances play neither a decisive nor predominant role. **2)** To examine the role of HSB and ANP in the etiology of obesity.

**METHOD:** A systematic search and review of scientific literature was conducted via electronic databases (i.e., PubMed, Medline) using the terms energy balance, nutrient partitioning, adipogenesis, obesity, physical activity and sedentary behavior. The review inclusion criteria required 1) a causal mechanism for obesity, 2) a role for physical activity, 3) a high citation rate. Both animal and human models of obesity were analyzed.

**RESULTS:** The search resulted in 149 papers that met the review criteria. The synthesis of results led to the development of a conceptual model in which HSB engenders ANP and potentiates obesity: **HSB ➡ ANP ➡ Ob**. This model suggests that the dominant agents in the obesity epidemic are alterations in the neural, endocrine and biochemical mechanisms which determine nutrient partitioning. Decrements in lipid and glucose metabolism and a reduction in β-adrenergic support of metabolic rate generate a positive feedback loop in which hyperglycemia, hyperlipedemia and insulin resistance result in hypertrophic and hyperplastic obesity.

**CONCLUSION:** These data indicate causal roles for HSB and ANP in the etiology of obesity. The results suggest that the population-wide increase in Ob is indicative of pathological patterns of nutrient partitioning rather than an excess of calories and a dearth of exercise. As such, future research may be best served by investigations that target the primary causal agents in the obesity epidemic: HSB and ANP.

## RESULTS




## CONCLUSIONS & IMPLICATIONS

Extended periods of physical inactivity **(HSB)**, independent of the effects on energy balance, impact energy substrate partitioning and obesity.

**Hyper-sedentary behavior** induces a shift in substrate utilization that diminishes the capacity to store carbohydrate (CHO) as glycogen and increases both hepatic lipogenesis and the storage of saturated fatty acids in adipose tissue. This pathological pattern of nutrient partitioning results in a number of morbidities including obesity, hyperglycemia, hyperlipidemia and insulin resistance.

Public Health Interventions should focus on the determinants of **adipogenic nutrient partitioning (ANP)** (e.g., **HSB**) rather than alterations in energy balance.