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Protein leverage and energy intake

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Increased energy intakes are contributing to overweight and obesity [1,2]. Growing evidence supports the role of protein appetite in driving excess intake when dietary protein is diluted (the protein leverage hypothesis) [3—8]. Understanding the interactions between dietary macronutrient balance and nutrient-specific appetite systems will be required for designing dietary interventions that work with, rather than against, basic regulatory physiology. Data were collected from 26 published experimental trials measuring ad libitum intake in subjects confined to menus differing in macronutrient composition. Collectively, these trials provided variation in percent protein (spanning 8—54% of total energy), carbohydrate (1.6—72%) and fat (11—66%). These data provided an opportunity to describe the individual and interactive effects of dietary protein, carbohydrate and fat on the control of total energy intake. Percent dietary protein was negatively associated with total energy intake ($F = 13.4$, $p < 0.001$) irrespective of whether carbohydrate ($F = 0.0001$, $p = 0.27$) or fat ($F = 0.00$, $p = 0.58$) were the diluents of protein. The analysis strongly supports a role for protein leverage in lean, overweight and obese humans. A better appreciation of the targets and regulatory priorities for protein, carbohydrate and fat intake will inform the design of effective and health-promoting weight loss diets, food labeling policies, food production systems, and regulatory frameworks.

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Comparison of the behavioral and metabolic effects of chronic 10% sucrose drink consumption in Albino and Hooded Wistar rats

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The metabolic consequences of providing rats with extended access to sugar solutions have varied across studies. The first stage of this study sought to determine the extent to which strain differences are responsible for such variation. Exposure to sugar solutions can also have behavioral consequences, some similar to those produced by drugs of abuse. Thus, the aim of the second stage was to test whether sucrose consumption would impact on reward-oriented behavior. In Stage 1 (8 weeks) Albino and Hooded Wistar rats had 24-h access to 10% sucrose solution in addition to their normal diet and water; controls were not given sucrose. The sucrose treatment elevated fasting blood glucose and resistance to insulin in both strains, but only increased weight gain in Albino rats. In Stage 2 (6 weeks) access to sucrose was ended and access to food was restricted in order to train lever-pressing prior to an outcome devaluation (by specific satiety) test of habit formation. Treatment in Stage 1 had no effect on habit formation, but responding by sucrose-fed Albino rats was the least affected by pre-feeding. Furthermore, despite 6 weeks of abstinence from sucrose, sucrose-induced obesity in Albino rats persisted throughout behavioral testing, while disturbances to blood glucose and insulin resolved to control levels 1 and 3 weeks after the removal of sucrose. At cull retroperitoneal fat