Intake of a high-protein regular snack is caloric compensated in humans

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Snacking is often regarded as one of the causes of overweight. However, the main question is to determine if the consumption of snacks leads to an increase of energy intake or if at the opposite, there is a phenomenon of compensation maintaining constant daily energy intake. Answers to this question depend on numerous factors like the amount of energy or macronutrients in the snack and the concept of conditioning to the snack (learned satiety). The objective of this study is to determine if the repeated consumption of a high-protein snack given as preload alters energy intake at the next meal and throughout the day and if this kind of snack is compensated. In experiment 1, we measured the effect of two HP snacks after a training period (5 days) of consumption. We used a moderate amount of proteins (23 g) and two types of milk proteins were compared. In experiment 2, we determined the importance of conditioning by comparing effect of the consumption of a HP snack on energy intake the first time subjects ingested the snack and after 5 days of repeated consumption of the same snack (training period). The main result of our work is that for both proteins used, a HP snack given 1 h before a meal, leads to partial energy compensation at the following meal and to over-compensation concerning the whole day energy consumption. We did not show a conditioning effect on food intake likely because our snack (cheese portion) was too familiar to volunteers.

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Obesity-prone rats are less sensitive to the satiating effects of exendin-4 than obesity-resistant rats

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Several hormones produced in the gastrointestinal tract lead to decreased food consumption by producing signals which initiate feelings of fullness. Glucagon-like peptide 1 (GLP-1) is a satiety hormone made in the small intestine. Circulating GLP-1 levels are lower after a meal in obese people than lean controls, suggesting decreased feelings of fullness. Osborne-Mendel (OM) and S5B/Pl (S5B) rats were used to investigate the satiating effects of GLP-1. OM rats become obese when eating a high fat diet, whereas S5B rats do not become obese when eating the same diet. Experiment 1 examined the effects of exendin-4 (Ex-4; GLP-1 agonist) administration on OM and S5B rats fed either a high fat (55%) or a low fat (10%) diet. It was hypothesized that S5B rats would be more sensitive to the satiating effects of Ex-4 than OM rats. The data indicated that Ex-4 dose-dependently decreased food intake to a greater extent in S5B rats, compared to OM rats. Intake of the high and low fat diets was differentially regulated by Ex-4 in these strains. Experiment 2 examined proglucagon mRNA expression in the distal intestine of OM and S5B rats. High dietary fat decreased proglucagon mRNA levels in the intestine of OM rats, but not in S5B rats. These data suggest that obesity-prone OM rats are less sensitive to the satiating effects of GLP-1 and produce less proglucagon mRNA when fed a high fat diet. Deficits in the response to and decreased expression of GLP-1 with a high fat diet may be mechanisms through which OM rats overeat and gain weight.

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Conditioned odor preferences mediated by associations with liked tastes

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Almond and banana extracts, which have strong odor components, were mixed with salt and saccharin in conditioned flavor preference learning. The results showed that mixing tastes with extracts increased liking for the extracts (measured by amount consumed in a test following training), but not the tastes. These findings are consistent with findings in humans suggesting that tastes can mediate increased liking to less preferred odors when they are mixed together. It is argued that since subjects typically express many more preferences for foods than aversions, it is particularly important to characterize this learning given its greater influence on diet and intake in the feeding context.

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Addiction-like behaviors in rats previously maintained on a high-fat diet

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The present experiment investigated if addiction-like behaviors previously described by Deroche-Gamonet et al. (2004) are demonstrated in rats exposed to high-fat diets. **Phase I:** Access to fat. In accordance with the methodology of the Corwin lab (Dimitriou et al., 2000), non-food-deprived rats were maintained for 6 weeks on one of four diets: chow only ($n = 9$); ad lib access to fat ($n = 21$); 1 h daily access to fat ($n = 21$); or 1 h access to fat on Monday, Wednesday, and Friday ($n = 21$). **Phase II:** Cocaine self-administration. Rats were then returned to chow only diets, implanted with intrajugular catheters, and trained to self-administer cocaine (0.8 mg/kg) on a fixed ratio (FR) schedule of reinforcement, beginning at FR1 and increasing to FR5, and then FR20. Daily acquisition sessions consisted of three drug periods (40 min each), with successive drug periods separated by a non-drug period (15 min each). **Results:** Rats exposed to fat diets took more infusions than the chow group during FR5 trials and tended to display more goal-directed behavior during FR20 trials. Rats with a history of fat intake also displayed more goal-directed behavior during time out periods following each infusion and during signaled non-availability. These results suggest that a history of fat intake may predispose rats to exhibit more robust "addiction-like" behaviors toward a drug of abuse relative to rats maintained on a diet of standard laboratory chow.

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