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Strubbe, J.H.; Heinsbroek, A.C.M.; Alessio, D. D'; Dijk, G. van

Published in:
Appetite

DOI:
[10.1016/j.appet.2008.04.234](https://doi.org/10.1016/j.appet.2008.04.234)

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Document Version
Publisher's PDF, also known as Version of record

Publication date:
2008

[Link to publication in University of Groningen/UMCG research database](#)

Citation for published version (APA):

Strubbe, J. H., Heinsbroek, A. C. M., Alessio, D. D., & Dijk, G. V. (2008). Involvement of peripheral GLP-1 in satiety, glycemic control and gastric motility. *Appetite*, 51(2), 403-403.
<https://doi.org/10.1016/j.appet.2008.04.234>

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Obese individuals show blunted striatal response to food: Relation moderated by DRD2 gene

E. STICE^{1,*}, S. SPOOR², C. BOHON³, J. NG³, D. SMALL⁴. ¹ Oregon Research Institute, Eugene, USA ² University of Texas at Austin, Austin, USA ³ University of Oregon, Eugene, USA ⁴ Peirce Laboratory, Yale University, New Haven, USA

As dopamine D2 receptor density in the striatum is inversely related to BMI among obese humans and obese rats have lower basal dopamine levels and reduced D2 receptor expression, it has been proposed that people with hypofunctioning reward circuitry overeat to boost a sluggish reward system. We conducted four fMRI studies that tested whether obese humans would show blunted activation of the striatum in response to food. In all studies, obese humans showed reduced activation of the striatum in response to receipt of chocolate milkshake and BMI was inversely related to activation in the striatum. As humans with an A1 allele of the Taq1 A1 DRD2 gene have 30–40% fewer D2 receptors than those without an A1 allele, we next tested whether the relation between obesity and blunted striatal response would be amplified in those with the A1 allele because they have reduced dopamine signaling capacity: the inverse relation of BMI to blunted striatal response to food receipt was amplified in those with the A1 allele. Finally, we tested whether humans with a blunted striatal response to food intake are at increased risk for future weight gain and whether the presence of the A1 allele of the DRD2 gene amplified this relation: the blunted striatal response predict future weight gain over a 1-year follow-up period, but only for those with the A1 allele. Findings collectively suggest that hypofunctioning food reward circuitry increases risk for obesity.

doi:10.1016/j.appet.2008.04.233

Involvement of peripheral GLP-1 in satiety, glycemic control and gastric motility

J.H. STRUBBE^{1,*}, A.C.M. HEINSBROEK¹, D.D'. ALESSIO², G. VAN DIJK¹. ¹ Center for Behavior and Neurosciences, University of Groningen, Haren, Netherlands ² Department of Medicine, University of Cincinnati, Cincinnati, USA

Glucagon-like peptide-1 amide (GLP-1) is secreted from ileal L-cells, pancreatic A-cells and from nerve terminals in the CNS. In the periphery, GLP-1 is secreted in response to ingested nutrients and is thought to ameliorate fuel excursions associated with ingested food. In the CNS, GLP-1 participates in regulation of ingestion and neuroendocrine outflow. This study investigated whether peripherally produced GLP-1 is able to affect ingestive behavior, gastrointestinal motility, and glucose homeostasis in male Wistar rats. Elevated plasma GLP-1 concentration was reached by intra-gastric infusion of sucrose (S) and acarbose. Acarbose (A) causes dumping of S into the lower intestine (because of inhibiting the luminal brush border enzyme α -glucosidase) which subsequently increases GLP-1 release into the blood stream. Relative to A alone, S+A had a more profound anorexigenic efficacy than S (without taste aversions), and this was blocked by prior peripheral immunoneutralization of GLP-1 (by i.v. infusion of an antibody raised against GLP-1). While GLP-1-immunoneutralization greatly augmented plasma insulin levels following S but not following S+A, the effect of S+A to inhibit gastric motility was not blocked by GLP-1 immuno-neutralization. We conclude that circulating GLP-1 is involved in satiety and glucose disposal, but not in the ileal brake mechanism.

doi:10.1016/j.appet.2008.04.234

Persistent effects of high-intensity sweeteners on body weight gain in rats

S.E. SWITHERS*, C.R. BAKER, M. MCCURLEY, T.L. DAVIDSON. Purdue University, West Lafayette, USA

Saccharin-sweetened diets have been shown to increase body weight gain, food intake and adiposity in rats. The present experiments examined the persistence of these effects, and whether AceK produced similar effects. Adult male rats were assigned to one of three groups and all received daily access to sweetened or unsweetened yogurt along with chow for 12 days. Yogurt and chow were available for 23 h per day in the first experiment; in the second experiment yogurt was available for 1 h per day while chow was unavailable; chow was available for the remaining 23 h. Each group received 6 days on which the yogurt was unsweetened, and 6 days on which the yogurt was sweetened. For one group, yogurt was sweetened with 20% glucose, for a second group, 0.3% AceK and for the third group, 0.3% saccharin. In both experiments, animals given the AceK and saccharin sweetened yogurt gained significantly more weight than the animals given the glucose-sweetened yogurt. In the final experiment, naive male rats were assigned to glucose or saccharin groups, and given access to 30 g yogurt for 2 weeks as in the first experiment. Yogurt exposure was then discontinued and body weight was monitored for an additional 2 weeks. Saccharin-exposed animals gained more weight than glucose-exposed animals, and these differences in body weight gain persisted for 2 weeks following termination of yogurt consumption. These data suggest that the effects of consuming artificially sweetened diets on body weight gain are not specific to saccharin and persist after the discontinuation of the non-predictive sweet taste—calorie experience.

doi:10.1016/j.appet.2008.04.235

Effect of pace of eating on within-session decreases in human eating behavior by instructing participants to chew each mouthful over 30 times

Y. TAKAKI^{1,*}, K. AOYAMA². ¹ Department of Psychology, Graduate School of Letters, Doshisha University, Kyoto, Japan ² Department of Psychology, Faculty of Letters, Doshisha University, Kyoto, Japan

Eating slowly has been believed to reduce food intake in behavior therapy of obesity (Stuart, 1967). Therefore, many experimental studies have been done in recent years to validate whether eating slowly actually reduced meal intake. In experimental studies, eating rate was manipulated by a variety of techniques. For example, Spiegel et al. (1993) manipulated bite size of sandwiches in three conditions (5, 10 and 15 g). However, few studies directly manipulated chewing number of times by instruction. This study examined effects of pace of eating on within-session decreases (transition of pace of eating in eating session) in eating behavior with participants instructed to chew each mouthful over 30 times. All participants were healthy men and session lengths were 20 min. The test foods were potato chips. Participants in a slow group ate one potato chip at a time, chewing over 30 times, while participants in a control group were not given an instruction about number of chews. Participants could eat freely and stop eating when they felt full. As a result, the control group ate more at the beginning of the session. However, within-session decreases were steeper in control groups than slow groups. In addition, median of total amount of time was not significantly different in both groups, but total amount of intake was significantly smaller in the slow group than the control group.

doi:10.1016/j.appet.2008.04.236