High-fat diet impairs satiation signaling in obese-prone

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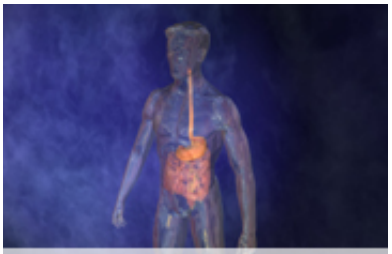

Feeding obese-prone rats a high-fat diet leads to impaired satiation signaling through glucagon-like peptide-1, a gastrointestinal hormone that suppresses food intake and helps regulate energy balance, according to a study published online Feb. 19 in *Diabetes*.

(HealthDay)—Feeding obese-prone rats a high-fat diet leads to impaired satiation signaling through glucagon-like peptide-1 (GLP-1), a gastrointestinal hormone that suppresses food intake and helps regulate energy balance, according to a study published online Feb. 19 in *Diabetes*.

Frank A. Duca, Ph.D., from the Institut National de la Recherche Agronomique in Jouy-en-Josas, France, and colleagues compared food intake in obese-prone and obese-resistant rats fed either normally or a high-energy/high-fat diet, after treatment with the GLP-1 receptor (GLP-1R) agonist exendin-4 or vehicle.

The researchers found that, while exendin-4 suppressed food intake in both types of rats when fed a normal diet, exendin-4 suppressed food intake significantly less in obese-prone rats than obese-resistant rats when fed a high-energy/high-fat diet. Obese-prone rats on a high-energy/high-fat diet had lower GLP-1R expression in the vagal nodose ganglia, less GLP-1 in serum and the intestinal epithelium, and fewer L-cells in the distal ileum.

"These results demonstrate that high-energy/high-fat-feeding coupled with obese-prone phenotype results in reduced endogenous GLP-1 and GLP-1R activation, indicating that impaired GLP-1 signaling during obesity may exacerbate hyperphagia and weight gain," Duca and colleagues conclude.

More information: Abstract
Full Text (subscription or payment may be required)