

Targeting taste receptors in the gut may help fight obesity

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Despite more than 25 years of research on antiobesity drugs, few medications have shown long-term success. Now researchers reporting online on December 21 in the Cell Press journal *Trends in Endocrinology & Metabolism* say that targeting taste sensors in the gut may be a promising new strategy.

The gut "tastes" what we eat—bitter, sweet, fat, and savory—in much the same way as the tongue and through the use of similar signaling mechanisms. The result is the release of hormones to control satiety and blood sugar levels when food reaches the gut. The sensors, or receptors, in the stomach respond to excess food intake, and their malfunction may play a role in the development of obesity, diabetes, and related metabolic conditions.

Drs. Sara Janssen and Inge Depoortere, of the Translational Research Center for Gastrointestinal Disorders at the Catholic University of Leuven, Belgium, examine this possibility, offering insights into the latest research on the topic. They say growing evidence suggests that obesity and related conditions might be prevented or treated by selective targeting of taste receptors on cells in the gut to release hormones that signal a feeling of fullness, thereby mimicking the physiological effects of a meal and fooling the body into thinking that it has eaten.

"The effectiveness of bariatric surgery to cause profound weight loss and a decrease in the prevalence of diabetes and other obesity-related conditions is not completely understood, but it may involve changes in



the release of gut hormones," says Dr. Depoortere. "Targeting extraoral taste receptors that affect the release of hormones that control food intake may offer a new road to mimic these effects in a nonsurgical manner."

Additional studies are needed to show which gut <u>taste receptors</u> might be effective <u>drug</u> targets for the prevention and treatment of <u>obesity</u> and diabetes.

More information: Janssen et al.: "Nutrient sensing in the gut: new roads to therapeutics?" <u>dx.doi.org/10.1016/j.tem.2012.11.006</u>

Provided by Cell Press

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