

Oxyntomodulin augments glucose homeostasis

March 21 2018



(HealthDay)—In obese subjects with and without type 2 diabetes



mellitus (T2DM), oxyntomodulin (OXM) significantly augments glucosedependent insulin secretion, according to a study published online March 15 in *Diabetes*.

Sudha S. Shankar, M.D., from Merck & Co. Inc. in Kenilworth, N.J., and colleagues examined the effects of OXM on glucose homeostasis. The effects of an intravenous infusion of native OXM on insulin secretory rates (ISR) and glycemic excursion were assessed in a graded glucose infusion (GGI) procedure in two separate placebo-controlled trials in 12 overweight and obese subjects without <u>diabetes</u> and in 12 obese subjects with T2DM. The glucagon-like peptide-1 (GLP-1) analog liraglutide was a comparator in the T2DM trial.

The researchers found that in the GGI, 3.0 pmol/kg/min of OXM significantly increased ISR in both groups and blunted glycemic excursion relative to placebo. The effects of OXM were comparable to those of liraglutide among individuals with T2DM, including the restoration of beta cell glucose responsiveness to that of non-obese subjects without diabetes.

"Our findings indicate that native OXM significantly augments glucosedependent insulin secretion acutely in obese subjects with and without diabetes, with effects comparable to pharmacologic GLP-1 receptor activation and independent of <u>weight loss</u>," the authors write. "Native OXM has potential to improve hyperglycemia via complementary and independent induction of <u>insulin</u> secretion and weight loss."

Several authors disclosed financial ties to Merck Sharp & Dohme Corp., a subsidiary of Merck & Co. Inc.

More information: <u>Abstract/Full Text (subscription or payment may</u> <u>be required)</u>



Copyright © 2018 <u>HealthDay</u>. All rights reserved.

Citation: Oxyntomodulin augments glucose homeostasis (2018, March 21) retrieved 6 May 2024 from

https://medicalxpress.com/news/2018-03-oxyntomodulin-augments-glucose-homeostasis.html

This document is subject to copyright. Apart from any fair dealing for the purpose of private study or research, no part may be reproduced without the written permission. The content is provided for information purposes only.