

Brain molecule reduces food intake

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Researchers at Imperial College London have identified a new appetite suppressant for promoting weight loss that they say works in rodents and may one day be used to develop an effective anti-obesity treatment.

The experimental treatment, prokineticin 2, is a recently discovered signaling molecule that occurs naturally in the part of the brain that helps control hunger. Both lean and obese mice treated with PK2 for 5 days lost almost 5 percent of their body weight, the authors reported.

"This is a greater weight loss than people achieve with current nonsurgical weight loss therapies," said study co-author Waljit Dhillo, a clinical senior lecturer at Imperial College London.

The researchers first dissolved a commercially available form of PK2 (from PeproTech Ltd.) in saline and injected it into the brain of 12 rats, which were allowed to eat as much as they wanted for 24 hours. Compared with 12 control rats that received only saline injections, the treated rats ate much less food; in the first hour alone, their <u>food intake</u> was 86 percent less.

PK2 did not affect movement, behavior or the ability to burn off calories, the authors reported.

To establish whether PK2 could be a potential anti-obesity treatment, the investigators changed the route of drug administration to be similar to the way patients with diabetes inject insulin—into the stomach. For 5 days, 10 lean mice and 10 obese mice received two PK2 injections a day



into the abdomen. Again, the treated mice ate much less than did the control mice, which resulted in <u>weight loss</u> of almost 5 percent of their body weight.

"Our research shows that PK2 reduces hunger," Dhillo said. "The results hold the promise that PK2 may be developed as a drug to treat obesity, which could help the millions of people suffering from obesity and its consequences."

Dhillo and co-workers plan to conduct longer-term studies of PK2 in animals before proceeding to human studies.

Source: The Endocrine Society (<u>news</u>: <u>web</u>)

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