Olive oil ingredient ups the time between meals

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A fatty acid found in abundance in olive oil and other "healthy" unsaturated fats has yet another benefit: it helps keep the body satisfied to prolong the time between meals.

A new study in the October *Cell Metabolism*, a publication of Cell Press, reveals that once this type of fat, known as oleic acid, reaches the intestine, it is converted into a lipid hormone (oleoylethanolamide, or OEA) that wards off the next round of hunger pangs. The researchers said it may be the first description of an ingredient in food that directly provides the raw materials for a hormone's production.

The findings in rats may yield insight into the precise dietary makeup of fat and protein for optimal hunger control, the researchers said. (Protein plays in important role in limiting hunger as well, but by different means.) The newly discovered signaling pathway might also be tapped into with drugs designed to control appetite by supplementing OEA levels or blocking its breakdown. Similarly, in conditions where people don't eat enough, the researchers speculate that treatments targeting this system might improve the appetite.

Importantly, diets high in processed foods that are riddled with saturated fats might throw a wrench into this system of metabolic control, the researchers said.

" Eating is one of the most important things animals do," said Daniele Piomelli of the University of California, Irvine. "This is just one of

many things that control it. That said, a system like this could be forced to inactivation by inappropriate feeding," he said, noting that saturated fats generally lack in oleic acid.

While such diets may lead people to overeat, Piomelli said it will also be of interest to see if this mechanism may be defective in some who tend to eat in excess.

Previous studies had shown that feeding stimulates cells in the intestinal lining to produce OEA, which, when administered as a drug, decreases meal frequency by engaging receptors called peroxisome proliferator-activated receptors a (PPARa).

Piomelli's team now reports that infusion of fat into the small intestine stimulates the release of OEA, whereas infusion of protein or carbohydrate does not. They also demonstrate that OEA production uses dietary oleic acid and is disrupted in mutant mice lacking the membrane fatty-acid transporter CD36. Treatments that disrupt CD36 or PPARa undermine the hunger control otherwise driven by fat.

Overall, the results suggest that activation of small-intestinal OEA release, enabled by CD36-mediated uptake of oleic acid from the diet, serves as a molecular sensor linking fat consumption to satiety. (Piomelli said satiety is perhaps best described as the opposite of hunger.)

"In conclusion," the researchers wrote, "our studies identify OEA as a key physiological signal that specifically links dietary fat ingestion to across-meal satiety. Nutritional and pharmacological strategies aimed at magnifying this lipid-sensing mechanism, such as inhibitors of OEA degradation, might be useful in the treatment of obesity and other eating disorders."

Source: Cell Press