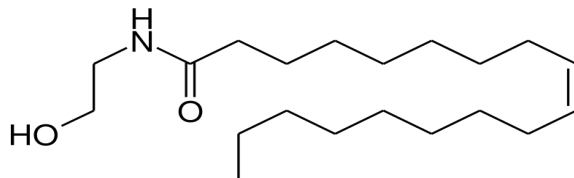# OEA
Oleoylethanolamide:
Organic Molecule Influencing Satiety & Insulin Levels

Bioactive Lipid-derived Factor Inhibiting Food Intake by Activation of PPAR-alpha & GPR119

FOOD INTAKE REGULATES OLEOYLETHANOLAMIDE FORMATION AND DEGRADATION IN THE PROXIMAL SMALL INTESTINE

Oleoylethanolamide (OEA) is a lipid mediator that inhibits food intake by activating the nuclear receptor peroxisome proliferator-activated receptor- α (PPAR-α). In the rodent small intestine, OEA levels decrease during food deprivation and increase upon refeeding, suggesting that endogenous OEA may participate in the regulation of satiety. Here we show that feeding stimulates OEA mobilization in the mucosal layer of rat duodenum and jejunum, but not in the serosal layer from the same intestinal segments, in other sections of the gastrointestinal tract (stomach, ileum, colon), or in a broad series of internal organs and tissues (e.g., liver, brain, heart, plasma). Feeding also increases the levels of other unsaturated fatty-acid ethanolamides (FAEs) (e.g., linoleoylethanolamide) without affecting those of saturated FAEs (e.g., palmitoylethanolamide). Feeding-induced OEA mobilization is accompanied by enhanced accumulation of OEA-generating N-acyl phosphatidylethanolamines (NAPEs), increased activity and expression of the OEA-synthesizing enzyme NAPE-phospholipase D (NAPE-PLD), and decreased activity and expression of the OEA-degrading enzyme fatty-acid amide hydrolase (FAAH). Immunostaining studies revealed that NAPE-PLD and FAAH are expressed in intestinal enterocytes and lamina propria cells. Collectively, these results indicate that nutrient availability controls OEA mobilization in the mucosa of the proximal intestine through a concerted regulation of OEA biosynthesis and degradation.

"OEA, as a GPR119 agonist, could prove useful in the field of diabetes"

GPR119, a novel G protein-coupled receptor target for the treatment of type 2 diabetes and obesity

GPR119 is a G protein-coupled receptor expressed pre-dominantly in the pancreas (beta-cells) and gastrointestinal tract (enteroendocrine cells) in humans. De-orphanization of GPR119 has revealed two classes of possible endogenous ligands, viz., phospholipids and fatty acid amides. Of these, oleoylethanolamide (OEA) is one of the most active ligands tested so far. This fatty acid ethanolamide is of particular interest because of its known effects of reducing food intake and body weight gain when administered to rodents. Agonists at the GPR119 receptor cause an increase in intracellular cAMP levels via G(alphas) coupling to adenylate cyclase. In vitro studies have indicated a role for GPR119 in the modulation of insulin release by pancreatic beta-cells and of GLP-1 secretion by gut enteroendocrine cells. The effects of GPR119 agonists in animal models of diabetes and obesity are reviewed, and the potential value of such compounds in future therapies for these conditions is discussed.
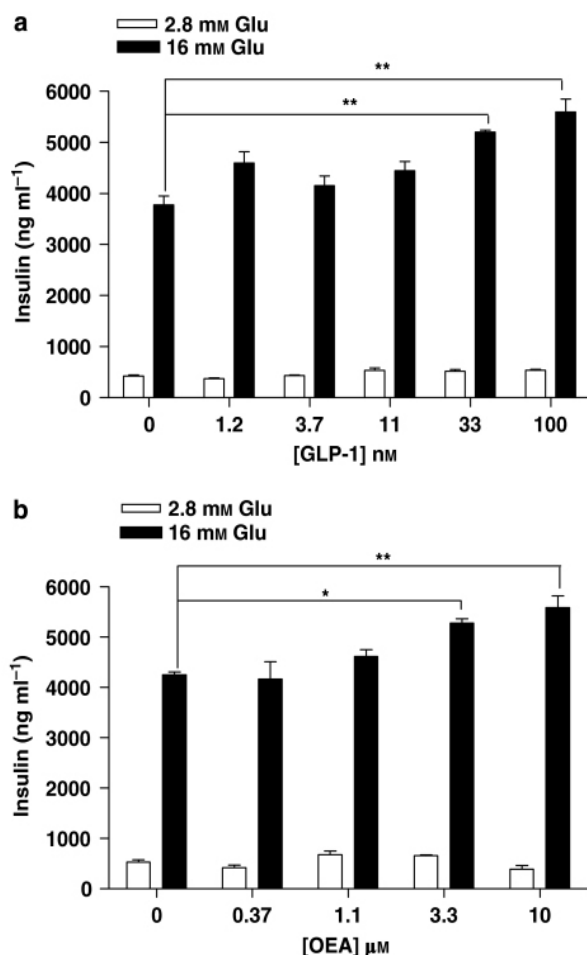
Glucagon-like peptide-1 (GLP-1) and oleoyletha-nolamide (OEA) increase glucose-stimulated insulin secretion.
MIN6c4 cells were stimu-lated with the indicated concentrations of (a) GLP-1, (b) OEA, in the pres-ence of 2.8mM glucose or 16mM glucose.
The insulin levels were measured after 2h incu-bation. Three replicates were measured for every treatment and data pre-sented as ng insulin ml−1 (mean±s.e.).
*P<0.05; **P<0.01 using ANOVA-Bonferroni, com-pared with insulin induced by glucose alone.

Catalog #	Product Name	Standard Size
032-51	OEA (Oleoylethanolamide)	1 mg