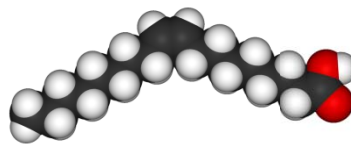


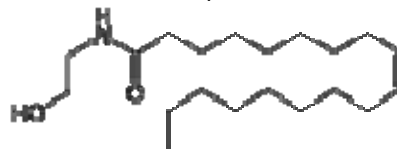
Proposed Mechanism of Action (Hypothetical)



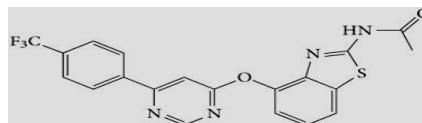
Achyranthes Aspera



Oleic Acid



Oleoylethanolide (Peroxisom Activated receptor)



Transient Receptor Potential Vanilloid Type 1 (TRPV1)

Duel Effects

Vagas Nerve Stimulation

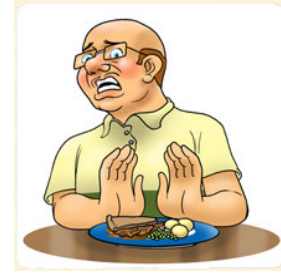
Lypolysis Stimulation



(A) Satiety Effect



(B) Fat reduction



(A) Satiety effect by Vagus Nerve Stimulation :

Achyranthes seeds have Protein, Fatty acids like Oleic acid and Linoleic acid and Saponin Glycosides.

Dietary Oleic acid can convert into a fat messenger called oleoylethanolamide (OEA).

Oleoylethanolamide (OEA), a lipid synthesized in the intestine, reduces food intake and stimulates lipolysis through **peroxisome** proliferator-activated receptor- α . OEA also activates transient receptor potential vanilloid type 1 (**TRPV1**) in vitro.

Oleic acid, a major digestive product of dietary fat, is transported into small-intestinal enterocytes by the fatty acid translocase CD36. There a fraction of Oleic acid is converted to OEA.

OEA suppresses feeding without causing visceral illness and that neither ghrelin, peptide YY, glucagon-like peptide 1, apolipoprotein A-IV, nor CCK plays a critical role in this effect¹. Giving the body time to handle the metabolic challenge presented by a fatty meal, OEA affects sensory vagus nerve activity through PPAR- α to induce satiety and decrease meal frequency.

It does not impact upon the size of a meal: satiation prompts meal termination and is mediated by gut peptides, whereas satiety is a separate response.

This acts as a sensor for ingestion of fat, activating PPAR- α to enhance lipid absorption (including by increased expression of CD36). OEA is disrupted in mutant mice lacking the membrane fatty-acid transporter CD36. Targeted disruption of CD36 or PPAR- α abrogates the satiety response induced by fat².

OEA decreases meal frequency by engaging peroxisome proliferator-activated receptors- α (PPAR- α). The activation of small-intestinal OEA mobilization, enabled by CD36-mediated uptake of dietary oleic acid, serves as a molecular sensor linking fat ingestion to satiety².



(B) Fat Reduction Action by stimulation of Lipolysis :

OEA acts independently of the cannabinoid pathway and regulates PPAR- α activity to stimulate lipolysis (the breakdown of fat stored in fat cells).^[3]

In adipocytes and hepatocytes, OEA inhibits mitogenic and metabolic signaling by the insulin receptor and produces glucose intolerance⁴.

Activation of the TRPV1 channel leads to an increase in cytosolic calcium and that lead to prevention of adipogenesis⁵.

Activation of TRPV1 channel, is necessary to prevent preadipocyte-to-adipocyte differentiation. TRPV1 activation may ultimately reduce the number and size of fat cells, and therefore reduce the tendency for fat to develop⁶.

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