

RESEARCH MONOGRAPH . COMPOUND PROFILE

Oleoylethanolamide (OEA)

*An endogenous N-acylethanolamide; a
PPAR-alpha agonist that signals satiety
from the small intestine after fatty meals.*

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COMPOUND CLASS	Endogenous N-acylethanolamide (PPAR-alpha satiety signal)

ABSTRACT

Oleoylethanolamide (OEA; CAS 111-58-0; molecular formula $C_{20}H_{39}NO_2$; molecular weight 325.53) is an endogenous N-acylethanolamide synthesized in the small intestine in response to dietary fat. The compound binds PPAR- α (K_i approximately 40 nM) with full agonist activity and signals satiety to the brain via vagal afferents and through direct CNS PPAR- α activation. The mechanism is distinct from leptin and ghrelin and represents a separate satiety pathway tied directly to fat intake. Pharmacological administration in rodents reduces food intake and body weight; clinical translation has been complicated by the short half-life (FAAH-mediated degradation). Used as the canonical PPAR- α satiety signal in metabolic and feeding research.

1. Compound Identification

Oleoylethanolamide (OEA) is described as endogenous n-acylethanolamide (ppar-alpha satiety signal). An endogenous N-acylethanolamide; a PPAR-alpha agonist that signals satiety from the small intestine after fatty meals.

1.1 Class summary

Endogenous N-acylethanolamide (PPAR-alpha satiety signal)

1.2 Subtitle

An endogenous N-acylethanolamide; a PPAR-alpha agonist that signals satiety from the small intestine after fatty meals.

2. Abstract

Oleoylethanolamide (OEA; CAS 111-58-0; molecular formula $C_{20}H_{39}NO_2$; molecular weight 325.53) is an endogenous N-acylethanolamide synthesized in the small intestine in response to dietary fat. The compound binds PPAR-alpha (K_i approximately 40 nM) with full agonist activity and signals satiety to the brain via vagal afferents and through direct CNS PPAR-alpha activation. The mechanism is distinct from leptin and ghrelin and represents a separate satiety pathway tied directly to fat intake. Pharmacological administration in rodents reduces food intake and body weight; clinical translation has been complicated by the short half-life (FAAH-mediated degradation). Used as the canonical PPAR-alpha satiety signal in metabolic and feeding research.

3. Mechanism of Action

Endogenous PPAR-alpha agonist released postprandially from intestine; signals satiety via vagal afferents and central PPAR-alpha activation. Distinct from leptin and ghrelin pathways.

The mechanistic profile described above represents the consensus position from peer-reviewed literature where available; for compounds with sparse formal characterization, this section reflects vendor-grade reports and structurally-related class pharmacology and should be interpreted with appropriate caution.

4. Pharmacokinetics and Routes of Administration

Pharmacokinetic data are summarized within the abstract for compounds with formal Phase 1 records. Compounds without published human pharmacokinetic studies are noted as such; investigators should treat the absence of data as a research limitation.

5. Reported Research Dose Ranges

Research substrate; rodent studies 1 to 20 mg/kg.

Doses are scaled from clinical use where the compound is approved as a medicine, from Phase 2/3 trials where development advanced, or from rodent dose-response data scaled by allometric assumptions where the compound has no human clinical record. Investigators should treat dose ranges from research-chemical-grade vendors as starting points for their own pharmacokinetic and tolerability characterization rather than validated dosing recommendations.

6. Sourcing and Quality Verification

Investigators should obtain analytical confirmation of identity (mass spectrometry; for peptides, additionally amino acid analysis or N-terminal sequencing) and purity (HPLC area-percent, ideally greater than 95 percent) on every lot. Vendor certificates of analysis vary in quality; third-party analytical confirmation through independent laboratories (e.g., Janoshik Analytical, Anabolic Lab) is the gold standard for research-grade peptides and small molecules.

For compounds with established pharmaceutical-grade marketing in any jurisdiction, sourcing from the marketed product (where legal and accessible) provides the highest quality assurance.

7. Reconstitution and Handling

Peptides should be reconstituted in bacteriostatic water or sterile water for injection in the volume appropriate to the desired concentration; lyophilized peptides should be allowed to come to room temperature before reconstitution to minimize precipitation. Reconstituted peptides should be stored refrigerated and used within the timeframe specified by the manufacturer (typically 14 to 30 days for bacteriostatic water reconstitutions, shorter for sterile water).

Small molecules: storage conditions vary by compound; consult the certificate of analysis for the specific product. Most are stable at room temperature in dry, dark conditions; oxidation-sensitive compounds (e.g., catecholamine precursors) require cool storage and protection from light.

8. Stack Interaction Considerations

Investigators combining this compound with other research substances should consider receptor-level interactions (additive effects at shared targets), enzyme-level interactions (CYP-mediated drug-drug interactions where applicable), and pharmacodynamic interactions (additive sedation, additive serotonergic activity, additive hemodynamic effects).

The stack interaction section in published monographs of well-characterized compounds is necessarily incomplete for any specific combination; investigators should treat novel combinations with elevated caution and titrate gradually.

9. References

1. Rodriguez de Fonseca F, et al. An anorexic lipid mediator regulated by feeding. Nature 2001.
2. Fu J, et al. Oleyethanolamide regulates feeding and body weight through activation of the nuclear receptor PPAR-alpha. Nature 2003.
3. Piomelli D. A fatty gut feeling. Trends Endocrinol Metab 2013.

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