

RESEARCH MONOGRAPH . COMPOUND PROFILE

Anandamide (AEA)

The principal endogenous CB1 receptor agonist; a fatty acid amide neurotransmitter implicated in mood, pain, and reward.

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COMPOUND CLASS	Endocannabinoid (N-arachidonylethanolamine)

ABSTRACT

Anandamide (N-arachidonylethanolamine, AEA; CAS 94421-68-8; molecular formula C₂₂H₃₇NO₂; molecular weight 347.53) is the principal endogenous CB1 receptor agonist, isolated by Devane and Mechoulam in 1992 (Nature). The name derives from the Sanskrit ananda (bliss). The compound is synthesized on-demand from membrane phospholipid precursors via NAPE-PLD and degraded primarily by fatty acid amide hydrolase (FAAH); the on-demand synthesis-degradation kinetics produce highly localized signaling without the persistent receptor occupation typical of conventional neurotransmitters. CB1 affinity is approximately 89 nM with full agonist activity; secondary TRPV1 and PPAR-gamma activity. Plasma half-life is on the order of minutes, extremely short owing to rapid FAAH-mediated degradation. Used as the canonical endocannabinoid in academic neuroscience and as a substrate in FAAH inhibitor research. The administration of exogenous anandamide as a drug is impractical owing to the short half-life; FAAH inhibitors (URB597, PF-04457845) are used to elevate endogenous anandamide instead.

1. Compound Identification

Anandamide (AEA) is described as endocannabinoid (n-arachidonylethanolamine). The principal endogenous CB1 receptor agonist; a fatty acid amide neurotransmitter implicated in mood, pain, and reward.

1.1 Class summary

Endocannabinoid (N-arachidonylethanolamine)

1.2 Subtitle

The principal endogenous CB1 receptor agonist; a fatty acid amide neurotransmitter implicated in mood, pain, and reward.

2. Abstract

Anandamide (N-arachidonylethanolamine, AEA; CAS 94421-68-8; molecular formula $C_{22}H_{37}NO_2$; molecular weight 347.53) is the principal endogenous CB1 receptor agonist, isolated by Devane and Mechoulam in 1992 (Nature). The name derives from the Sanskrit ananda (bliss). The compound is synthesized on-demand from membrane phospholipid precursors via NAPE-PLD and degraded primarily by fatty acid amide hydrolase (FAAH); the on-demand synthesis-degradation kinetics produce highly localized signaling without the persistent receptor occupation typical of conventional neurotransmitters. CB1 affinity is approximately 89 nM with full agonist activity; secondary TRPV1 and PPAR-gamma activity. Plasma half-life is on the order of minutes, extremely short owing to rapid FAAH-mediated degradation. Used as the canonical endocannabinoid in academic neuroscience and as a substrate in FAAH inhibitor research. The administration of exogenous anandamide as a drug is impractical owing to the short half-life; FAAH inhibitors (URB597, PF-04457845) are used to elevate endogenous anandamide instead.

3. Mechanism of Action

Endogenous full agonist at CB1 receptors; secondary TRPV1 and PPAR-gamma activity. Rapid on-demand synthesis and FAAH-mediated degradation.

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; in vitro and in vivo experimental concentrations vary.

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. Devane WA, et al. Isolation and structure of a brain constituent that binds to the cannabinoid receptor. Science 1992.
2. Cravatt BF, et al. Molecular characterization of an enzyme that degrades neuromodulatory fatty-acid amides. Nature 1996.
3. Piomelli D. The molecular logic of endocannabinoid signalling. Nat Rev Neurosci 2003.

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