

RESEARCH MONOGRAPH . COMPOUND PROFILE

Beta- Phenylethylamine (PEA)

An endogenous trace amine; a TAAR1 agonist found at low concentrations in mammalian brain; structurally the parent of amphetamine.

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COMPOUND CLASS	Endogenous trace amine / TAAR1 agonist

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ABSTRACT

Beta-phenylethylamine (PEA; 2-phenylethylamine; CAS 64-04-0; molecular formula $C_8H_{11}N$; molecular weight 121.18) is an endogenous trace amine produced by decarboxylation of L-phenylalanine. The compound is found at low concentrations in mammalian brain (nM range) and in dietary sources including chocolate, cheese, and certain fermented foods. PEA is the prototype TAAR1 agonist (K_i approximately 700 nM at human TAAR1) with secondary effects on monoamine release and inhibition of MAO-B. Plasma half-life is extremely short (approximately 5 to 10 minutes) owing to rapid MAO-B-mediated degradation; exogenous oral PEA is therefore largely inactive without MAO-B inhibition (selegiline combination is used to extend duration). Behavioral and physiological effects parallel amphetamine but with shorter duration and lower potency. Used as the canonical endogenous TAAR1 agonist in trace amine research.

1. Compound Identification

Beta-Phenylethylamine (PEA) is described as endogenous trace amine / taar1 agonist. An endogenous trace amine; a TAAR1 agonist found at low concentrations in mammalian brain; structurally the parent of amphetamine.

1.1 Class summary

Endogenous trace amine / TAAR1 agonist

1.2 Subtitle

An endogenous trace amine; a TAAR1 agonist found at low concentrations in mammalian brain; structurally the parent of amphetamine.

2. Abstract

Beta-phenylethylamine (PEA; 2-phenylethylamine; CAS 64-04-0; molecular formula $C_8H_{11}N$; molecular weight 121.18) is an endogenous trace amine produced by decarboxylation of L-phenylalanine. The compound is found at low concentrations in mammalian brain (nM range) and in dietary sources including chocolate, cheese, and certain fermented foods. PEA is the prototype TAAR1 agonist (K_i approximately 700 nM at human TAAR1) with secondary effects on monoamine release and inhibition of MAO-B. Plasma half-life is extremely short (approximately 5 to 10 minutes) owing to rapid MAO-B-mediated degradation; exogenous oral PEA is therefore largely inactive without MAO-B inhibition (selegiline combination is used to extend duration). Behavioral and physiological effects parallel amphetamine but with shorter duration and lower potency. Used as the canonical endogenous TAAR1 agonist in trace amine research.

3. Mechanism of Action

Endogenous trace amine TAAR1 agonist; secondary monoamine release. Rapidly degraded by MAO-B, severely limiting oral activity without MAO inhibition.

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

Supplement 100 to 500 mg per oral administration (often combined with MAO-B inhibitor for efficacy).

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. Borowsky B, et al. Trace amines: identification of a family of mammalian G protein-coupled receptors. Proc Natl Acad Sci 2001.
2. Berry MD. Mammalian central nervous system trace amines. Pharmacologic amphetamines, physiologic neuromodulators. J Neurochem 2004.
3. Janssen PA, et al. Does phenylethylamine act as an endogenous amphetamine? Eur J Pharmacol 1999.

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