

RESEARCH MONOGRAPH . COMPOUND PROFILE

URB597

A carbamate inhibitor of fatty acid amide hydrolase (FAAH); the canonical research probe for elevating endogenous anandamide.

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COMPOUND CLASS	Selective FAAH inhibitor (research)

ABSTRACT

URB597 (KDS-4103, cyclohexyl carbamic acid 3'-carbamoyl-biphenyl-3-yl ester; CAS 546141-08-6; molecular formula C₂₀H₂₂N₂O₃; molecular weight 338.40) is a covalent carbamate inhibitor of fatty acid amide hydrolase (FAAH) developed by Piomelli and colleagues at UC Irvine. The compound carbamoylates the active-site serine of FAAH, producing irreversible enzyme inactivation. Selective for FAAH over MAGL and other lipases. Pharmacological consequence: elevation of endogenous anandamide and other N-acylethanolamides without direct CB receptor agonism. The behavioral profile differs from direct CB1 agonists like THC: anxiolytic and analgesic effects without the catalepsy, hypothermia, and intoxication characteristic of CB1 full agonism. Phase 2 clinical trials in major depressive disorder did not demonstrate efficacy; the compound did not advance to phase 3. Plasma half-life is approximately 6 hours; the irreversible mechanism produces effective FAAH inhibition for 24-48 hours after a single dose. Used as the canonical FAAH inhibitor in academic neuroscience.

1. 1. Compound Identification

URB597 is described as selective faah inhibitor (research). A carbamate inhibitor of fatty acid amide hydrolase (FAAH); the canonical research probe for elevating endogenous anandamide.

1.1. 1.1 Class summary

Selective FAAH inhibitor (research)

1.2. 1.2 Subtitle

A carbamate inhibitor of fatty acid amide hydrolase (FAAH); the canonical research probe for elevating endogenous anandamide.

2. Abstract

URB597 (KDS-4103, cyclohexyl carbamic acid 3'-carbamoyl-biphenyl-3-yl ester; CAS 546141-08-6; molecular formula C₂₀H₂₂N₂O₃; molecular weight 338.40) is a covalent carbamate inhibitor of fatty acid amide hydrolase (FAAH) developed by Piomelli and colleagues at UC Irvine. The compound carbamoylates the active-site serine of FAAH, producing irreversible enzyme inactivation. Selective for FAAH over MAGL and other lipases. Pharmacological consequence: elevation of endogenous anandamide and other N-acylethanolamides without direct CB receptor agonism. The behavioral profile differs from direct CB1 agonists like THC: anxiolytic and analgesic effects without the catalepsy, hypothermia, and intoxication characteristic of CB1 full agonism. Phase 2 clinical trials in major depressive disorder did not demonstrate efficacy; the compound did not advance to phase 3. Plasma half-life is approximately 6 hours; the irreversible mechanism produces effective FAAH inhibition for 24-48 hours after a single dose. Used as the canonical FAAH inhibitor in academic neuroscience.

3. Mechanism of Action

Covalent carbamate inhibitor of FAAH; irreversible carbamoylation of active-site serine. Selective for FAAH over other lipases; elevates endogenous anandamide without direct CB receptor agonism.

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. 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. 5. Reported Research Dose Ranges

Rodent research 0.1 to 1 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. 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. 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. 9. References

1. Kathuria S, et al. Modulation of anxiety through blockade of anandamide hydrolysis. Nat Med 2003.
2. Piomelli D, et al. Pharmacological profile of the selective FAAH inhibitor KDS-4103 (URB597). CNS Drug Rev 2006.
3. Boger DL, et al. Discovery of a potent, selective, and efficacious class of reversible alpha-ketoheterocycle inhibitors of fatty acid amide hydrolase. J Med Chem 2005.

This monograph is provided for research use only. Not for medical, diagnostic, or therapeutic purposes. Not for human consumption. Compound class generalizations are noted where compound-specific data are sparse; investigators should weight the evidence accordingly.