

RESEARCH MONOGRAPH . COMPOUND PROFILE

HU-308

A synthetic CB2-selective cannabinoid agonist; a research tool for CB2 pharmacology without psychoactive CB1 activity.

DOCUMENT	KDC-MN-327
ISSUED	2026-05-09
REVISION	1.0
AUTHOR	Kodiac bioLabs Research
COMPOUND CLASS	Synthetic CB2-selective cannabinoid

RESEARCH USE ONLY

ABSTRACT

HU-308 ([4-[4-(1,1-dimethylheptyl)-2,6-dimethoxyphenyl]-6,6-dimethyl-bicyclo[3.1.1]hept-2-en-2-yl]methanol; CAS 256934-39-1; molecular formula C₂₇H₄₂O₄; molecular weight 430.62) is a synthetic CB₂-selective cannabinoid agonist developed by Mechoulam and colleagues at Hebrew University. The compound is a pinene-scaffold cannabinoid with high CB₂ affinity (K_i approximately 23 nM) and minimal CB₁ binding (greater than 440-fold selectivity), eliminating the psychoactivity associated with CB₁-mediated cannabinoid effects. CB₂ is expressed predominantly in peripheral immune cells (macrophages, B cells, T cells, microglia in CNS); selective activation produces anti-inflammatory and analgesic effects without psychotropic side effects. Preclinical studies demonstrate efficacy in inflammatory bowel disease, neuropathic pain, hepatic ischemia, and atherosclerosis models. Plasma half-life is approximately 2 to 3 hours. Used as the canonical CB₂-selective research probe in academic immunology and pain research.

1. Compound Identification

HU-308 is described as synthetic cb2-selective cannabinoid. A synthetic CB2-selective cannabinoid agonist; a research tool for CB2 pharmacology without psychoactive CB1 activity.

1.1 Class summary

Synthetic CB2-selective cannabinoid

1.2 Subtitle

A synthetic CB2-selective cannabinoid agonist; a research tool for CB2 pharmacology without psychoactive CB1 activity.

2. Abstract

HU-308 ([4-[4-(1,1-dimethylheptyl)-2,6-dimethoxyphenyl]-6,6-dimethylbicyclo[3.1.1]hept-2-en-2-yl]methanol; CAS 256934-39-1; molecular formula C₂₇H₄₂O₄; molecular weight 430.62) is a synthetic CB₂-selective cannabinoid agonist developed by Mechoulam and colleagues at Hebrew University. The compound is a pinene-scaffold cannabinoid with high CB₂ affinity (K_i approximately 23 nM) and minimal CB₁ binding (greater than 440-fold selectivity), eliminating the psychoactivity associated with CB₁-mediated cannabinoid effects. CB₂ is expressed predominantly in peripheral immune cells (macrophages, B cells, T cells, microglia in CNS); selective activation produces anti-inflammatory and analgesic effects without psychotropic side effects. Preclinical studies demonstrate efficacy in inflammatory bowel disease, neuropathic pain, hepatic ischemia, and atherosclerosis models. Plasma half-life is approximately 2 to 3 hours. Used as the canonical CB₂-selective research probe in academic immunology and pain research.

3. Mechanism of Action

CB2-selective cannabinoid agonist (~440-fold over CB1); peripheral immune cell activation without psychotropic effects.

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

Rodent research 1 to 30 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. Hanus L, et al. HU-308: a specific agonist for CB2, a peripheral cannabinoid receptor. Proc Natl Acad Sci 1999.
2. Pacher P, Mechoulam R. Is lipid signaling through cannabinoid 2 receptors part of a protective system? Prog Lipid Res 2011.
3. Whiteside GT, et al. The role of the cannabinoid CB2 receptor in pain transmission and therapeutic potential of small molecule CB2 receptor agonists. Curr Med Chem 2007.

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.