

RESEARCH MONOGRAPH . COMPOUND PROFILE

Levetiracetam

The (S)-enantiomer of etiracetam, FDA-approved as Keppra for partial-onset seizures, characterized by the SV2A synaptic vesicle binding mechanism distinct from classical racetam pharmacology.

DOCUMENT	KDC-MN-033
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COMPOUND CLASS	S-enantiomer pyrrolidinone (SV2A ligand)

ABSTRACT

Levetiracetam (Keppra; (S)- α -ethyl-2-oxo-1-pyrrolidineacetamide; CAS 102767-28-2; molecular formula C₈H₁₄N₂O₂; molecular weight 170.21) is the (S)-enantiomer of etiracetam, developed at UCB Pharma and approved by the FDA in 1999 for partial-onset seizures, with subsequent expansion to primary generalized tonic-clonic seizures and myoclonic seizures. The compound is distinct from other racetams in mechanism: levetiracetam binds with high affinity (K_d approximately 1 μ M) to synaptic vesicle protein 2A (SV2A), a presynaptic vesicle membrane protein involved in neurotransmitter release regulation. SV2A binding modulates calcium-dependent neurotransmitter release and reduces hypersynchronous neuronal firing in epileptic networks. The compound has minimal effect on classical neurotransmitter receptors. Pharmacokinetics: plasma half-life 6 to 8 hours; oral bioavailability essentially complete; renal excretion with minimal hepatic metabolism. Approved doses are 1000 to 3000 mg per day in two divided administrations. The compound is also studied off-label for cognitive enhancement based on hippocampal hyperactivity attenuation, with mixed results in mild cognitive impairment trials. Schedule status varies; the compound is not scheduled federally in the United States but is prescription-only.

1. Compound Identification

Levetiracetam is described as s-enantiomer pyrrolidinone (sv2a ligand). The (S)-enantiomer of etiracetam, FDA-approved as Keppra for partial-onset seizures, characterized by the SV2A synaptic vesicle binding mechanism distinct from classical racetam pharmacology.

1.1 Class summary

S-enantiomer pyrrolidinone (SV2A ligand)

1.2 Subtitle

The (S)-enantiomer of etiracetam, FDA-approved as Keppra for partial-onset seizures, characterized by the SV2A synaptic vesicle binding mechanism distinct from classical racetam pharmacology.

2. Abstract

Levetiracetam (Keppra; (S)- α -ethyl-2-oxo-1-pyrrolidineacetamide; CAS 102767-28-2; molecular formula $C_8H_{14}N_2O_2$; molecular weight 170.21) is the (S)-enantiomer of etiracetam, developed at UCB Pharma and approved by the FDA in 1999 for partial-onset seizures, with subsequent expansion to primary generalized tonic-clonic seizures and myoclonic seizures. The compound is distinct from other racetams in mechanism: levetiracetam binds with high affinity (K_d approximately 1 μ M) to synaptic vesicle protein 2A (SV2A), a presynaptic vesicle membrane protein involved in neurotransmitter release regulation. SV2A binding modulates calcium-dependent neurotransmitter release and reduces hypersynchronous neuronal firing in epileptic networks. The compound has minimal effect on classical neurotransmitter receptors. Pharmacokinetics: plasma half-life 6 to 8 hours; oral bioavailability essentially complete; renal excretion with minimal hepatic metabolism. Approved doses are 1000 to 3000 mg per day in two divided administrations. The compound is also studied off-label for cognitive enhancement based on hippocampal hyperactivity attenuation, with mixed results in mild cognitive impairment trials. Schedule status varies; the compound is not scheduled federally in the United States but is prescription-only.

3. Mechanism of Action

High-affinity binding to synaptic vesicle protein 2A (SV2A); modulates calcium-dependent neurotransmitter release. Distinct from all other racetams in mechanism.

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

1000 to 3000 mg per day in two divided doses (clinical use).

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. Lynch BA, et al. The synaptic vesicle protein SV2A is the binding site for the antiepileptic drug levetiracetam. PNAS 2004.
2. Klitgaard H, et al. Evidence for a unique profile of levetiracetam in rodent models of seizures and epilepsy. Eur J Pharmacol 1998.
3. Patsalos PN. Pharmacokinetic profile of levetiracetam. Clin Pharmacokinet 2004.

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