

RESEARCH MONOGRAPH . COMPOUND PROFILE

Sodium Valproate

A simple branched-chain fatty acid with broad anticonvulsant activity and mood-stabilizing efficacy in bipolar disorder.

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ABSTRACT

Sodium valproate (sodium 2-propylpentanoate; CAS 1069-66-5; molecular formula $C_8H_{15}NaO_2$; molecular weight 166.20) is a branched-chain fatty acid anticonvulsant first characterized as bioactive in 1962 (incidentally, while serving as a solvent in pharmacological screening at Lyon). Approved by the FDA in 1978 (Depakote, the divalproex sodium delayed-release form). Mechanism is multifactorial: GABA transaminase inhibition increases brain GABA; sodium channel blockade reduces neuronal hyperexcitability; T-type calcium channel inhibition supports anti-absence activity; histone deacetylase (HDAC) inhibition modifies gene expression with neuroprotective and possibly disease-modifying implications in bipolar disorder. Plasma half-life is 9 to 16 hours; metabolism is via beta-oxidation, omega-oxidation, and glucuronidation. Approved indications include all major seizure types (partial, generalized tonic-clonic, absence, myoclonic), bipolar I disorder mania, and migraine prophylaxis. The principal limitation is teratogenicity (neural tube defects, cognitive deficits in offspring) requiring strict avoidance in pregnancy and women of childbearing age. Hepatotoxicity, pancreatitis, and hyperammonemia are recognized adverse events. Used as the canonical broad-spectrum anticonvulsant and HDAC inhibitor reference.

1. Compound Identification

Sodium Valproate is described as branched-chain fatty acid anticonvulsant / mood stabilizer. A simple branched-chain fatty acid with broad anticonvulsant activity and mood-stabilizing efficacy in bipolar disorder.

1.1 Class summary

Branched-chain fatty acid anticonvulsant / mood stabilizer

1.2 Subtitle

A simple branched-chain fatty acid with broad anticonvulsant activity and mood-stabilizing efficacy in bipolar disorder.

2. Abstract

Sodium valproate (sodium 2-propylpentanoate; CAS 1069-66-5; molecular formula $C_8H_{15}NaO_2$; molecular weight 166.20) is a branched-chain fatty acid anticonvulsant first characterized as bioactive in 1962 (incidentally, while serving as a solvent in pharmacological screening at Lyon). Approved by the FDA in 1978 (Depakote, the divalproex sodium delayed-release form). Mechanism is multifactorial: GABA transaminase inhibition increases brain GABA; sodium channel blockade reduces neuronal hyperexcitability; T-type calcium channel inhibition supports anti-absence activity; histone deacetylase (HDAC) inhibition modifies gene expression with neuroprotective and possibly disease-modifying implications in bipolar disorder. Plasma half-life is 9 to 16 hours; metabolism is via beta-oxidation, omega-oxidation, and glucuronidation. Approved indications include all major seizure types (partial, generalized tonic-clonic, absence, myoclonic), bipolar I disorder mania, and migraine prophylaxis. The principal limitation is teratogenicity (neural tube defects, cognitive deficits in offspring) requiring strict avoidance in pregnancy and women of childbearing age. Hepatotoxicity, pancreatitis, and hyperammonemia are recognized adverse events. Used as the canonical broad-spectrum anticonvulsant and HDAC inhibitor reference.

3. Mechanism of Action

Multifactorial: GABA transaminase inhibition, sodium and T-type calcium channel block, HDAC inhibition. Broad anticonvulsant activity and mood stabilization.

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

Clinical 750 to 3000 mg per oral administration daily. Rodent studies 100 to 600 mg/kg/day.

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. Loscher W. Basic pharmacology of valproate: a review after 35 years of clinical use for the treatment of epilepsy. CNS Drugs 2002.
2. Bowden CL, et al. Efficacy of divalproex vs lithium and placebo in the treatment of mania. JAMA 1994.
3. Phiel CJ, et al. Histone deacetylase is a direct target of valproic acid, a potent anticonvulsant, mood stabilizer, and teratogen. J Biol Chem 2001.

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.