

RESEARCH MONOGRAPH . COMPOUND PROFILE

Topiramate

A sulfamate-substituted monosaccharide anticonvulsant with broad mechanism (sodium channels, GABA-A potentiation, AMPA antagonism, carbonic anhydrase inhibition); used for migraine prophylaxis and weight loss.

DOCUMENT	KDC-MN-245
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COMPOUND CLASS	Sulfamate-substituted monosaccharide / multifactorial anticonvulsant

ABSTRACT

Topiramate (2,3:4,5-bis-O-(1-methylethylidene)-beta-D-fructopyranose sulfamate; CAS 97240-79-4; molecular formula C₁₂H₂₁NO₈S; molecular weight 339.36) is a sulfamate-substituted monosaccharide anticonvulsant developed at Johnson and Johnson and approved by the FDA in 1996 under the trade name Topamax. Mechanism is multifactorial: state-dependent sodium channel inhibition; GABA-A receptor potentiation at a non-benzodiazepine site; AMPA/kainate glutamate receptor antagonism; carbonic anhydrase inhibition (isozymes II and IV); high-voltage calcium channel inhibition. The carbonic anhydrase activity contributes to the characteristic adverse events: paresthesias (40 percent of patients), nephrolithiasis (1.5 percent), metabolic acidosis. Cognitive side effects are pronounced: word-finding difficulty, slowed mentation. Plasma half-life is 21 hours; metabolism is approximately 70 percent renal excretion unchanged. Approved for partial-onset and generalized tonic-clonic seizures, Lennox-Gastaut syndrome, migraine prophylaxis, and (with phentermine, as Qsymia) chronic weight management. Used as the canonical multifactorial anticonvulsant and as a reference carbonic anhydrase inhibitor in CNS pharmacology.

1. 1. Compound Identification

Topiramate is described as sulfamate-substituted monosaccharide / multifactorial anticonvulsant. A sulfamate-substituted monosaccharide anticonvulsant with broad mechanism (sodium channels, GABA-A potentiation, AMPA antagonism, carbonic anhydrase inhibition); used for migraine prophylaxis and weight loss.

1.1. 1.1 Class summary

Sulfamate-substituted monosaccharide / multifactorial anticonvulsant

1.2. 1.2 Subtitle

A sulfamate-substituted monosaccharide anticonvulsant with broad mechanism (sodium channels, GABA-A potentiation, AMPA antagonism, carbonic anhydrase inhibition); used for migraine prophylaxis and weight loss.

2. 2. Abstract

Topiramate (2,3:4,5-bis-O-(1-methylethylidene)-beta-D-fructopyranose sulfamate; CAS 97240-79-4; molecular formula C₁₂H₂₁NO₈S; molecular weight 339.36) is a sulfamate-substituted monosaccharide anticonvulsant developed at Johnson and Johnson and approved by the FDA in 1996 under the trade name Topamax. Mechanism is multifactorial: state-dependent sodium channel inhibition; GABA-A receptor potentiation at a non-benzodiazepine site; AMPA/kainate glutamate receptor antagonism; carbonic anhydrase inhibition (isozymes II and IV); high-voltage calcium channel inhibition. The carbonic anhydrase activity contributes to the characteristic adverse events: paresthesias (40 percent of patients), nephrolithiasis (1.5 percent), metabolic acidosis. Cognitive side effects are pronounced: word-finding difficulty, slowed mentation. Plasma half-life is 21 hours; metabolism is approximately 70 percent renal excretion unchanged. Approved for partial-onset and generalized tonic-clonic seizures, Lennox-Gastaut syndrome, migraine prophylaxis, and (with phentermine, as Qsymia) chronic weight management. Used as the canonical multifactorial anticonvulsant and as a reference carbonic anhydrase inhibitor in CNS pharmacology.

3. Mechanism of Action

Multifactorial: sodium channel inhibition, GABA-A potentiation, AMPA antagonism, carbonic anhydrase inhibition. Used in epilepsy, migraine prophylaxis, and weight management.

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

Clinical 50 to 400 mg per oral administration daily. Rodent studies 10 to 100 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. 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. Shank RP, et al. An overview of the preclinical aspects of topiramate. *Epilepsia* 2000.
2. Silberstein SD, et al. Topiramate in migraine prevention. *Headache* 2007.
3. Rosenfeld WE. Topiramate: a review of preclinical, pharmacokinetic, and clinical data. *Clin Ther* 1997.

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.