

RESEARCH MONOGRAPH . COMPOUND PROFILE

Carbamazepine

*A first-generation iminostilbene
anticonvulsant and mood stabilizer with
potent CYP3A4 induction.*

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ABSTRACT

Carbamazepine (5H-dibenzo[b,f]azepine-5-carboxamide; CAS 298-46-4; molecular formula C₁₅H₁₂N₂O; molecular weight 236.27) is an iminostilbene anticonvulsant developed by Walter Schindler at Geigy in 1953 and approved by the FDA in 1968 under the trade name Tegretol. Mechanism: state-dependent voltage-gated sodium channel inhibition (similar to lamotrigine), producing reduced neuronal hyperexcitability in seizure foci. Active metabolite carbamazepine-10,11-epoxide retains anticonvulsant activity. Plasma half-life is 25 to 65 hours initially but auto-induces own metabolism, dropping to 12 to 17 hours at steady state. Strong CYP3A4 induction produces clinically significant interactions with hormonal contraception, immunosuppressants, anticoagulants, antiretrovirals, and many other drugs. Approved for partial-onset and generalized tonic-clonic seizures, trigeminal neuralgia, and bipolar I mania (in combination products). The principal safety concerns are aplastic anemia and agranulocytosis (rare but serious; HLA-B*1502 association predicts SJS/TEN risk in Asian populations and is screened pretreatment). Used as the reference iminostilbene voltage-gated sodium channel blocker.

1. Compound Identification

Carbamazepine is described as iminostilbene voltage-gated sodium channel blocker. A first-generation iminostilbene anticonvulsant and mood stabilizer with potent CYP3A4 induction.

1.1 Class summary

Iminostilbene voltage-gated sodium channel blocker

1.2 Subtitle

A first-generation iminostilbene anticonvulsant and mood stabilizer with potent CYP3A4 induction.

2. Abstract

Carbamazepine (5H-dibenzo[b,f]azepine-5-carboxamide; CAS 298-46-4; molecular formula C₁₅H₁₂N₂O; molecular weight 236.27) is an iminostilbene anticonvulsant developed by Walter Schindler at Geigy in 1953 and approved by the FDA in 1968 under the trade name Tegretol. Mechanism: state-dependent voltage-gated sodium channel inhibition (similar to lamotrigine), producing reduced neuronal hyperexcitability in seizure foci. Active metabolite carbamazepine-10,11-epoxide retains anticonvulsant activity. Plasma half-life is 25 to 65 hours initially but auto-induces own metabolism, dropping to 12 to 17 hours at steady state. Strong CYP3A4 induction produces clinically significant interactions with hormonal contraception, immunosuppressants, anticoagulants, antiretrovirals, and many other drugs. Approved for partial-onset and generalized tonic-clonic seizures, trigeminal neuralgia, and bipolar I mania (in combination products). The principal safety concerns are aplastic anemia and agranulocytosis (rare but serious; HLA-B*1502 association predicts SJS/TEN risk in Asian populations and is screened pretreatment). Used as the reference iminostilbene voltage-gated sodium channel blocker.

3. Mechanism of Action

State-dependent voltage-gated sodium channel inhibition. Active metabolite carbamazepine-10,11-epoxide. Strong CYP3A4 induction.

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 400 to 1200 mg per oral administration daily, in divided doses. Rodent studies 25 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. 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. Macdonald RL, Kelly KM. Antiepileptic drug mechanisms of action. *Epilepsia* 1995.
2. Post RM, et al. Carbamazepine in psychiatric illness. *CNS Drugs* 1995.
3. Chung WH, et al. Medical genetics: a marker for Stevens-Johnson syndrome. *Nature* 2004.

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