

RESEARCH MONOGRAPH . COMPOUND PROFILE

Tranylcypromine

*A non-hydrazine cyclopropylamine MAOI
structurally related to amphetamine;
rapid-onset compared to phenelzine.*

DOCUMENT	KDC-MN-227
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COMPOUND CLASS	Irreversible non-selective MAO inhibitor (amphetamine-related)

ABSTRACT

Tranylcypromine ((1S,2R)-2-phenylcyclopropan-1-amine; CAS 155-09-9; molecular formula C₉H₁₁N; molecular weight 133.19) is a non-hydrazine cyclopropylamine MAOI approved by the FDA in 1961 under the trade name Parnate. The cyclopropylamine scaffold is structurally related to amphetamine, producing weak monoamine release in addition to MAO inhibition; the result is faster antidepressant onset (1 to 2 weeks) than phenelzine (3 to 6 weeks) and a mildly activating subjective profile. Inhibition of both MAO-A and MAO-B is irreversible, with similar tyramine cheese-effect risk. Plasma half-life is 1.5 to 3.2 hours, but enzyme inhibition persists for 5 to 10 days. The (-)-trans isomer is the predominant active form; clinical preparations are racemic. Hepatic metabolism is minor; the compound is largely excreted unchanged. Approved for major depressive disorder; used in atypical and treatment-resistant depression. Used as a non-hydrazine MAOI reference compound and as a positive control in dopaminergic and norepinephrine release studies.

1. 1. Compound Identification

Tranylcypromine is described as irreversible non-selective mao inhibitor (amphetamine-related). A non-hydrazine cyclopropylamine MAOI structurally related to amphetamine; rapid-onset compared to phenelzine.

1.1. 1.1 Class summary

Irreversible non-selective MAO inhibitor (amphetamine-related)

1.2. 1.2 Subtitle

A non-hydrazine cyclopropylamine MAOI structurally related to amphetamine; rapid-onset compared to phenelzine.

2. 2. Abstract

Tranlylcypromine ((1S,2R)-2-phenylcyclopropan-1-amine; CAS 155-09-9; molecular formula C₉H₁₁N; molecular weight 133.19) is a non-hydrazine cyclopropylamine MAOI approved by the FDA in 1961 under the trade name Parnate. The cyclopropylamine scaffold is structurally related to amphetamine, producing weak monoamine release in addition to MAO inhibition; the result is faster antidepressant onset (1 to 2 weeks) than phenelzine (3 to 6 weeks) and a mildly activating subjective profile. Inhibition of both MAO-A and MAO-B is irreversible, with similar tyramine cheese-effect risk. Plasma half-life is 1.5 to 3.2 hours, but enzyme inhibition persists for 5 to 10 days. The (-)-trans isomer is the predominant active form; clinical preparations are racemic. Hepatic metabolism is minor; the compound is largely excreted unchanged. Approved for major depressive disorder; used in atypical and treatment-resistant depression. Used as a non-hydrazine MAOI reference compound and as a positive control in dopaminergic and norepinephrine release studies.

3. Mechanism of Action

Irreversible non-selective MAO inhibition with weak monoamine release from amphetamine-like scaffold. Activating subjective profile.

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 30 to 60 mg per oral administration daily, in divided doses. Rodent studies 1 to 10 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. Frieling H, Bleich S. Tranylcypromine: new perspectives on an old drug. Eur Arch Psychiatry Clin Neurosci 2006.
2. Heinze G, et al. Tranylcypromine vs SSRIs in treatment-resistant depression. Pharmacopsychiatry 2018.
3. Riederer P, et al. The pharmacology of MAO inhibitors. Neurology 2004.

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