

RESEARCH MONOGRAPH . COMPOUND PROFILE

Rasagiline

A second-generation propargyl MAO-B inhibitor lacking the amphetamine metabolites of selegiline.

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COMPOUND CLASS	Selective irreversible MAO-B inhibitor (second-generation)

ABSTRACT

Rasagiline ((R)-N-(prop-2-yn-1-yl)-2,3-dihydro-1H-inden-1-amine; CAS 136236-51-6; molecular formula C₁₂H₁₃N; molecular weight 171.24) is a second-generation irreversible MAO-B inhibitor developed at Teva and approved by the FDA in 2006 under the trade name Azilect. Structurally distinct from selegiline by the indane scaffold replacing the phenethylamine, rasagiline does not yield amphetamine-class metabolites, simplifying its drug screening profile and avoiding the subjective stimulation associated with selegiline. MAO-B selectivity is approximately 14:1 over MAO-A at clinical dose (1 mg daily); the inhibition is irreversible and recovery requires de novo enzyme synthesis (approximately 14-day washout). Approved as monotherapy in early Parkinson disease and as adjunct to levodopa in advanced disease. The ADAGIO trial suggested possible disease-modifying effect for the 1 mg dose, though this remains debated. Tyramine restriction is not required at 1 mg daily; at higher experimental doses (2 mg) the cheese-effect risk emerges. Plasma half-life is approximately 3 hours, but the irreversible binding produces effective enzyme inhibition for weeks. Used as a reference second-generation MAO-B inhibitor in Parkinson research.

1. Compound Identification

Rasagiline is described as selective irreversible mao-b inhibitor (second-generation). A second-generation propargyl MAO-B inhibitor lacking the amphetamine metabolites of selegiline.

1.1 Class summary

Selective irreversible MAO-B inhibitor (second-generation)

1.2 Subtitle

A second-generation propargyl MAO-B inhibitor lacking the amphetamine metabolites of selegiline.

2. Abstract

Rasagiline ((R)-N-(prop-2-yn-1-yl)-2,3-dihydro-1H-inden-1-amine; CAS 136236-51-6; molecular formula C₁₂H₁₃N; molecular weight 171.24) is a second-generation irreversible MAO-B inhibitor developed at Teva and approved by the FDA in 2006 under the trade name Azilect. Structurally distinct from selegiline by the indane scaffold replacing the phenethylamine, rasagiline does not yield amphetamine-class metabolites, simplifying its drug screening profile and avoiding the subjective stimulation associated with selegiline. MAO-B selectivity is approximately 14:1 over MAO-A at clinical dose (1 mg daily); the inhibition is irreversible and recovery requires de novo enzyme synthesis (approximately 14-day washout). Approved as monotherapy in early Parkinson disease and as adjunct to levodopa in advanced disease. The ADAGIO trial suggested possible disease-modifying effect for the 1 mg dose, though this remains debated. Tyramine restriction is not required at 1 mg daily; at higher experimental doses (2 mg) the cheese-effect risk emerges. Plasma half-life is approximately 3 hours, but the irreversible binding produces effective enzyme inhibition for weeks. Used as a reference second-generation MAO-B inhibitor in Parkinson research.

3. Mechanism of Action

Irreversible MAO-B inhibition (selective at 1 mg). Indane scaffold avoids amphetamine metabolites of selegiline. Effective enzyme inhibition persists for weeks owing to irreversible binding.

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 0.5 to 1 mg per oral administration daily. Rodent studies 0.05 to 1 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. Olanow CW, et al. A double-blind, delayed-start trial of rasagiline in Parkinson's disease. N Engl J Med 2009.
2. Chen JJ, Swope DM. Pharmacotherapy for Parkinson's disease. Pharmacotherapy 2007.
3. Lecht S, et al. Rasagiline - a novel MAO B inhibitor. CNS Drug Rev 2007.

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