

RESEARCH MONOGRAPH . COMPOUND PROFILE

Rivastigmine

A pseudo-irreversible carbamate dual cholinesterase inhibitor approved for Alzheimer disease and Parkinson disease dementia, available as oral and transdermal formulations.

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COMPOUND CLASS	Acetylcholinesterase + butyrylcholinesterase inhibitor

ABSTRACT

Rivastigmine (Exelon; CAS 123441-03-2; molecular formula $C_{14}H_{22}N_2O_2$; molecular weight 250.34) is a pseudo-irreversible carbamate cholinesterase inhibitor approved by the FDA in 2000 for mild-to-moderate Alzheimer disease and subsequently expanded to Parkinson disease dementia. The compound is distinctive among the marketed AChE inhibitors in showing dual inhibition of acetylcholinesterase (AChE) and butyrylcholinesterase (BChE); BChE may play a more prominent role in advanced Alzheimer disease as AChE expression declines, providing a theoretical advantage for rivastigmine in later disease stages. The pseudo-irreversible mechanism involves carbamoylation of the AChE serine residue, with slow regeneration over hours. Pharmacokinetics: short plasma half-life of the parent compound (1 to 2 hours) but the carbamoylation produces extended pharmacological effect (8 to 10 hours functional duration). Available as oral capsules and a transdermal patch (Exelon Patch); the patch produces smoother plasma exposure with reduced gastrointestinal adverse events. Approved doses are 6 to 12 mg per day oral or 4.6 to 13.3 mg/24 hr patch.

1. Compound Identification

Rivastigmine is described as acetylcholinesterase + butyrylcholinesterase inhibitor. A pseudo-irreversible carbamate dual cholinesterase inhibitor approved for Alzheimer disease and Parkinson disease dementia, available as oral and transdermal formulations.

1.1 Class summary

Acetylcholinesterase + butyrylcholinesterase inhibitor

1.2 Subtitle

A pseudo-irreversible carbamate dual cholinesterase inhibitor approved for Alzheimer disease and Parkinson disease dementia, available as oral and transdermal formulations.

2. Abstract

Rivastigmine (Exelon; CAS 123441-03-2; molecular formula $C_{14}H_{22}N_2O_2$; molecular weight 250.34) is a pseudo-irreversible carbamate cholinesterase inhibitor approved by the FDA in 2000 for mild-to-moderate Alzheimer disease and subsequently expanded to Parkinson disease dementia. The compound is distinctive among the marketed AChE inhibitors in showing dual inhibition of acetylcholinesterase (AChE) and butyrylcholinesterase (BChE); BChE may play a more prominent role in advanced Alzheimer disease as AChE expression declines, providing a theoretical advantage for rivastigmine in later disease stages. The pseudo-irreversible mechanism involves carbamoylation of the AChE serine residue, with slow regeneration over hours. Pharmacokinetics: short plasma half-life of the parent compound (1 to 2 hours) but the carbamoylation produces extended pharmacological effect (8 to 10 hours functional duration). Available as oral capsules and a transdermal patch (Exelon Patch); the patch produces smoother plasma exposure with reduced gastrointestinal adverse events. Approved doses are 6 to 12 mg per day oral or 4.6 to 13.3 mg/24 hr patch.

3. Mechanism of Action

Pseudo-irreversible carbamate inhibitor of both AChE and BChE; carbamoylation produces extended functional effect.

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

6 to 12 mg per day oral or 4.6 to 13.3 mg/24 hr transdermal.

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. Polinsky RJ. Clinical pharmacology of rivastigmine: a new generation acetylcholinesterase inhibitor for the treatment of Alzheimer's disease. Clin Ther 1998.
2. Birks J, Iakovidou V, Tsolaki M, Holt FE. Rivastigmine for Alzheimer's disease. Cochrane Database Syst Rev 2015.

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