

RESEARCH MONOGRAPH . COMPOUND PROFILE

Escitalopram

The (S)-enantiomer of citalopram; the active component of the racemate marketed as a separate antidepressant with improved tolerability.

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COMPOUND CLASS	S-enantiomer SSRI

ABSTRACT

Escitalopram (S-(+)-1-[3-(dimethylamino)propyl]-1-(4-fluorophenyl)-1,3-dihydro-2-benzofuran-5-carbonitrile; CAS 128196-01-0; molecular formula C₂₀H₂₁FN₂O; molecular weight 324.39) is the (S)-enantiomer of citalopram, marketed as Lexapro by H. Lundbeck and Forest Pharmaceuticals after FDA approval in 2002. SERT affinity is approximately 1 nM; selectivity for SERT over NET and DAT is the highest of any clinical SSRI. The active enantiomer is responsible for essentially all SERT inhibitory activity in racemic citalopram; the (R)-enantiomer attenuates (S) binding through an allosteric SERT interaction, so removing it produces a small but measurable increase in serotonergic efficacy per milligram. Plasma half-life is approximately 27 to 32 hours, slightly shorter than racemic citalopram. Hepatic metabolism via CYP2C19 with secondary CYP3A4 and CYP2D6 contribution. The compound exhibits the QTc prolongation profile of racemic citalopram but with a cleaner adverse event signature owing to the absence of the (R)-enantiomer. Approved for major depressive disorder and generalized anxiety disorder. Used as a high-purity SERT-selective reference compound in mechanism studies.

1. Compound Identification

Escitalopram is described as s-enantiomer ssri. The (S)-enantiomer of citalopram; the active component of the racemate marketed as a separate antidepressant with improved tolerability.

1.1 Class summary

S-enantiomer SSRI

1.2 Subtitle

The (S)-enantiomer of citalopram; the active component of the racemate marketed as a separate antidepressant with improved tolerability.

2. Abstract

Escitalopram (S-(+)-1-[3-(dimethylamino)propyl]-1-(4-fluorophenyl)-1,3-dihydro-2-benzofuran-5-carbonitrile; CAS 128196-01-0; molecular formula C₂₀H₂₁FN₂O; molecular weight 324.39) is the (S)-enantiomer of citalopram, marketed as Lexapro by H. Lundbeck and Forest Pharmaceuticals after FDA approval in 2002. SERT affinity is approximately 1 nM; selectivity for SERT over NET and DAT is the highest of any clinical SSRI. The active enantiomer is responsible for essentially all SERT inhibitory activity in racemic citalopram; the (R)-enantiomer attenuates (S) binding through an allosteric SERT interaction, so removing it produces a small but measurable increase in serotonergic efficacy per milligram. Plasma half-life is approximately 27 to 32 hours, slightly shorter than racemic citalopram. Hepatic metabolism via CYP2C19 with secondary CYP3A4 and CYP2D6 contribution. The compound exhibits the QTc prolongation profile of racemic citalopram but with a cleaner adverse event signature owing to the absence of the (R)-enantiomer. Approved for major depressive disorder and generalized anxiety disorder. Used as a high-purity SERT-selective reference compound in mechanism studies.

3. Mechanism of Action

Same SERT inhibition as racemic citalopram, attributable entirely to the (S)-enantiomer. Highest SERT selectivity among clinical SSRIs.

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 5 to 20 mg per oral administration daily. Rodent studies 0.5 to 5 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. Sanchez C, et al. Escitalopram, the S-(+)-enantiomer of citalopram, is a selective serotonin reuptake inhibitor with potent effects in animal models predictive of antidepressant and anxiolytic activities. Psychopharmacology 2003.
2. Burke WJ. Escitalopram. Expert Opin Investig Drugs 2002.
3. Owens MJ, et al. Second-generation SSRIs: human monoamine transporter binding profile of escitalopram and R-fluoxetine. Biol Psychiatry 2001.

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