

RESEARCH MONOGRAPH . COMPOUND PROFILE

Estradiol (Research)

*17-beta-estradiol; the principal
endogenous estrogen and the foundational
compound of estrogen pharmacology.*

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COMPOUND CLASS	Endogenous estrogen / ER-alpha and ER-beta agonist

RESEARCH USE ONLY

ABSTRACT

Estradiol (17-beta-estradiol, E2; CAS 50-28-2; molecular formula C₁₈H₂₄O₂; molecular weight 272.39) is the principal endogenous estrogen, biosynthesized from testosterone (and androstenedione) by aromatase in ovary, adipose tissue, brain, and bone. The compound is a high-affinity full agonist at estrogen receptors ER-alpha and ER-beta (K_i approximately 0.1 nM at both), with downstream genomic effects on estrogen-responsive gene transcription and rapid non-genomic effects through membrane-associated signaling. Approved indications include menopausal hormone therapy (multiple oral, transdermal, vaginal, and parenteral formulations), prevention of postmenopausal osteoporosis, hypoestrogenism (Turner syndrome), and as part of gender-affirming feminizing hormone therapy. Pharmacokinetics depend strongly on formulation: oral micronized estradiol undergoes substantial first-pass metabolism; transdermal forms (patches, gels, sprays) bypass first-pass and produce more physiological plasma levels. Schedule N (non-controlled). Used as the canonical estrogen receptor agonist in research.

1. Compound Identification

Estradiol (Research) is described as endogenous estrogen / er-alpha and er-beta agonist. 17-beta-estradiol; the principal endogenous estrogen and the foundational compound of estrogen pharmacology.

1.1 Class summary

Endogenous estrogen / ER-alpha and ER-beta agonist

1.2 Subtitle

17-beta-estradiol; the principal endogenous estrogen and the foundational compound of estrogen pharmacology.

2. Abstract

Estradiol (17-beta-estradiol, E2; CAS 50-28-2; molecular formula C₁₈H₂₄O₂; molecular weight 272.39) is the principal endogenous estrogen, biosynthesized from testosterone (and androstenedione) by aromatase in ovary, adipose tissue, brain, and bone. The compound is a high-affinity full agonist at estrogen receptors ER-alpha and ER-beta (K_i approximately 0.1 nM at both), with downstream genomic effects on estrogen-responsive gene transcription and rapid non-genomic effects through membrane-associated signaling. Approved indications include menopausal hormone therapy (multiple oral, transdermal, vaginal, and parenteral formulations), prevention of postmenopausal osteoporosis, hypoestrogenism (Turner syndrome), and as part of gender-affirming feminizing hormone therapy. Pharmacokinetics depend strongly on formulation: oral micronized estradiol undergoes substantial first-pass metabolism; transdermal forms (patches, gels, sprays) bypass first-pass and produce more physiological plasma levels. Schedule N (non-controlled). Used as the canonical estrogen receptor agonist in research.

3. Mechanism of Action

High-affinity ER-alpha and ER-beta full agonist; genomic transactivation of estrogen-responsive genes plus rapid non-genomic membrane signaling.

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

Menopausal HRT 1 to 2 mg PO daily or 0.025 to 0.1 mg/24 h transdermal patch; gender-affirming HRT 2 to 8 mg PO daily.

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. Mendelsohn ME, Karas RH. The protective effects of estrogen on the cardiovascular system. N Engl J Med 1999.
2. Stuenkel CA, et al. Treatment of symptoms of the menopause: an Endocrine Society clinical practice guideline. J Clin Endocrinol Metab 2015.
3. Hembree WC, et al. Endocrine treatment of gender-dysphoric/gender-incongruent persons: an Endocrine Society clinical practice guideline. J Clin Endocrinol Metab 2017.

This monograph is provided for research use only. Not for medical, diagnostic, or therapeutic purposes. Not for human consumption. Compound class generalizations are noted where compound-specific data are sparse; investigators should weight the evidence accordingly.