

RESEARCH MONOGRAPH . COMPOUND PROFILE

Tamoxifen

A triphenylethylene SERM; the foundational antiestrogen for ER-positive breast cancer and an off-label tool for SARM/AAS post-cycle therapy.

DOCUMENT	KDC-MN-271
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COMPOUND CLASS	Selective estrogen receptor modulator (triphenylethylene)

ABSTRACT

Tamoxifen ((Z)-2-[4-(1,2-diphenyl-1-butenyl)phenoxy]-N,N-dimethylethanamine; CAS 10540-29-1; molecular formula C₂₆H₂₉NO; molecular weight 371.51) is a triphenylethylene SERM developed at ICI Pharmaceuticals (now AstraZeneca) and approved by the FDA in 1977 under the trade name Nolvadex. The compound is a tissue-selective estrogen receptor modulator: antagonist in breast tissue (the basis for breast cancer indications) and partial agonist in bone, liver, and uterus. The active metabolite endoxifen (4-hydroxy-N-desmethyldoxifen) generated via CYP2D6 is approximately 100-fold more potent than tamoxifen at ER binding and contributes substantially to clinical activity; CYP2D6 polymorphism affects clinical response. Plasma half-life is approximately 5 to 7 days for parent compound. Approved indications include adjuvant treatment of ER-positive breast cancer (premenopausal and postmenopausal), metastatic breast cancer, and breast cancer prevention in high-risk patients. Off-label use in male hypogonadism, gynecomastia treatment, and post-cycle therapy following SARM or anabolic steroid use is common. Adverse events include endometrial cancer (rare), thromboembolism, and hot flashes. Used as the canonical SERM in mechanism studies.

1. Compound Identification

Tamoxifen is described as selective estrogen receptor modulator (triphenylethylene). A triphenylethylene SERM; the foundational antiestrogen for ER-positive breast cancer and an off-label tool for SARM/AAS post-cycle therapy.

1.1 Class summary

Selective estrogen receptor modulator (triphenylethylene)

1.2 Subtitle

A triphenylethylene SERM; the foundational antiestrogen for ER-positive breast cancer and an off-label tool for SARM/AAS post-cycle therapy.

2. Abstract

Tamoxifen ((Z)-2-[4-(1,2-diphenyl-1-butenyl)phenoxy]-N,N-dimethylethanamine; CAS 10540-29-1; molecular formula C₂₆H₂₉NO; molecular weight 371.51) is a triphenylethylene SERM developed at ICI Pharmaceuticals (now AstraZeneca) and approved by the FDA in 1977 under the trade name Nolvadex. The compound is a tissue-selective estrogen receptor modulator: antagonist in breast tissue (the basis for breast cancer indications) and partial agonist in bone, liver, and uterus. The active metabolite endoxifen (4-hydroxy-N-desmethyltamoxifen) generated via CYP2D6 is approximately 100-fold more potent than tamoxifen at ER binding and contributes substantially to clinical activity; CYP2D6 polymorphism affects clinical response. Plasma half-life is approximately 5 to 7 days for parent compound. Approved indications include adjuvant treatment of ER-positive breast cancer (premenopausal and postmenopausal), metastatic breast cancer, and breast cancer prevention in high-risk patients. Off-label use in male hypogonadism, gynecomastia treatment, and post-cycle therapy following SARM or anabolic steroid use is common. Adverse events include endometrial cancer (rare), thromboembolism, and hot flashes. Used as the canonical SERM in mechanism studies.

3. Mechanism of Action

Triphenylethylene SERM with tissue-selective ER modulation: antagonist in breast, partial agonist in bone/uterus. Active metabolite endoxifen drives clinical activity. CYP2D6-dependent metabolism.

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 10 to 40 mg per oral administration daily. PCT use commonly 20 to 40 mg/day. 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. 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. Jordan VC. Tamoxifen: a most unlikely pioneering medicine. Nat Rev Drug Discov 2003.
2. Goetz MP, et al. The impact of cytochrome P450 2D6 metabolism in women receiving adjuvant tamoxifen. Breast Cancer Res Treat 2007.
3. Visvanathan K, et al. Use of pharmacologic interventions for breast cancer risk reduction. ASCO Clinical Practice Guideline 2019.

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.