

RESEARCH MONOGRAPH . COMPOUND PROFILE

Triptorelin

A synthetic GnRH agonist decapeptide used for paradoxical HPG suppression in prostate cancer, endometriosis, and precocious puberty.

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COMPOUND CLASS	Synthetic GnRH agonist (decapeptide)

ABSTRACT

Triptorelin (5-oxo-L-prolyl-L-histidyl-L-tryptophyl-L-seryl-L-tyrosyl-D-tryptophyl-L-leucyl-L-arginyl-L-prolylglycinamide; CAS 57773-63-4; molecular formula C₆₄H₈₂N₁₈O₁₃; molecular weight 1311.45) is a synthetic GnRH (LHRH) agonist decapeptide developed in the 1970s and approved by the FDA in 2000 (Trelstar). Mechanism: continuous occupation of pituitary GnRH receptors initially produces a flare in LH, FSH, and consequent sex hormone secretion (1 to 2 weeks); chronic exposure desensitizes and downregulates GnRH receptors, producing a sustained reduction in LH and FSH and, downstream, in testosterone (men) and estradiol (women). Plasma half-life of the parent peptide is approximately 3 hours; depot formulations release peptide over 1, 3, or 6 months for sustained suppression. Approved indications include locally advanced and metastatic prostate cancer, endometriosis, uterine leiomyomata, and central precocious puberty. The flare period in prostate cancer is mitigated by concurrent androgen receptor antagonist (bicalutamide). Used as a reference GnRH agonist in academic endocrine pharmacology.

1. 1. Compound Identification

Triptorelin is described as synthetic gnrh agonist (decapeptide). A synthetic GnRH agonist decapeptide used for paradoxical HPG suppression in prostate cancer, endometriosis, and precocious puberty.

1.1. 1.1 Class summary

Synthetic GnRH agonist (decapeptide)

1.2. 1.2 Subtitle

A synthetic GnRH agonist decapeptide used for paradoxical HPG suppression in prostate cancer, endometriosis, and precocious puberty.

2. 2. Abstract

Triptorelin (5-oxo-L-prolyl-L-histidyl-L-tryptophyl-L-seryl-L-tyrosyl-D-tryptophyl-L-leucyl-L-arginyl-L-prolylglycinamide; CAS 57773-63-4; molecular formula C₆₄H₈₂N₁₈O₁₃; molecular weight 1311.45) is a synthetic GnRH (LHRH) agonist decapeptide developed in the 1970s and approved by the FDA in 2000 (Trelstar). Mechanism: continuous occupation of pituitary GnRH receptors initially produces a flare in LH, FSH, and consequent sex hormone secretion (1 to 2 weeks); chronic exposure desensitizes and downregulates GnRH receptors, producing a sustained reduction in LH and FSH and, downstream, in testosterone (men) and estradiol (women). Plasma half-life of the parent peptide is approximately 3 hours; depot formulations release peptide over 1, 3, or 6 months for sustained suppression. Approved indications include locally advanced and metastatic prostate cancer, endometriosis, uterine leiomyomata, and central precocious puberty. The flare period in prostate cancer is mitigated by concurrent androgen receptor antagonist (bicalutamide). Used as a reference GnRH agonist in academic endocrine pharmacology.

3. Mechanism of Action

GnRH agonist decapeptide; chronic exposure desensitizes pituitary GnRH receptors and suppresses LH/FSH and consequent sex hormones. Initial flare followed by sustained suppression.

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. 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. 5. Reported Research Dose Ranges

Clinical depot 3.75 mg monthly, 11.25 mg three-monthly, or 22.5 mg six-monthly intramuscular. Rodent studies 0.1 to 1 mg/kg.

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. 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. 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. 9. References

1. Plosker GL, Brogden RN. Leuprorelin. A review of its pharmacology and use in prostatic cancer, endometriosis and other sex hormone-related disorders. Drugs Aging 1994.
2. Heyns CF, et al. A randomised comparison of triptorelin pamoate and goserelin acetate. Eur Urol 2003.
3. Eisenberger MA, et al. Bilateral orchiectomy with or without flutamide for metastatic prostate cancer. N Engl J Med 1998.

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