

RESEARCH MONOGRAPH . COMPOUND PROFILE

# Testosterone (Research)

*The principal endogenous androgen; the foundational compound of androgen pharmacology and the active component of TRT and many AAS regimens.*

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| DOCUMENT       | KDC-MN-386                                        |
| ISSUED         | 2026-05-09                                        |
| REVISION       | 1.0                                               |
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| COMPOUND CLASS | Endogenous androgen / anabolic-androgenic steroid |

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## ABSTRACT

Testosterone (17-beta-hydroxyandrost-4-en-3-one; CAS 58-22-0; molecular formula C<sub>19</sub>H<sub>28</sub>O<sub>2</sub>; molecular weight 288.42) is the principal endogenous androgen, biosynthesized from cholesterol via pregnenolone and progesterone in Leydig cells of the testis (males) and theca cells, ovary, and adrenal cortex (females). The compound is the prototype androgen receptor agonist and the foundation of androgen pharmacology. Pharmacologically, testosterone binds AR (K<sub>i</sub> approximately 0.5 nM) and is converted to dihydrotestosterone (DHT) by 5-alpha-reductase (more potent AR agonist) and to estradiol by aromatase (estrogen receptor agonist). Approved as testosterone replacement therapy in male hypogonadism via various ester formulations: cypionate (weekly), enanthate (weekly), propionate (every 2 to 3 days), undecanoate (oral and depot), gels and patches (daily). Schedule III in the US under the CSA. The compound is the foundation of most anabolic-androgenic steroid (AAS) regimens used non-medically for muscle building. Plasma half-life of native testosterone is short (1 to 2 hours); ester formulations release the parent compound over days. Used as the canonical AR agonist in research.

# 1. 1. Compound Identification

**Testosterone (Research)** is described as endogenous androgen / anabolic-androgenic steroid. The principal endogenous androgen; the foundational compound of androgen pharmacology and the active component of TRT and many AAS regimens.

## 1.1. 1.1 Class summary

Endogenous androgen / anabolic-androgenic steroid

## 1.2. 1.2 Subtitle

The principal endogenous androgen; the foundational compound of androgen pharmacology and the active component of TRT and many AAS regimens.

## 2. 2. Abstract

Testosterone (17-beta-hydroxyandrost-4-en-3-one; CAS 58-22-0; molecular formula C<sub>19</sub>H<sub>28</sub>O<sub>2</sub>; molecular weight 288.42) is the principal endogenous androgen, biosynthesized from cholesterol via pregnenolone and progesterone in Leydig cells of the testis (males) and theca cells, ovary, and adrenal cortex (females). The compound is the prototype androgen receptor agonist and the foundation of androgen pharmacology. Pharmacologically, testosterone binds AR (K<sub>i</sub> approximately 0.5 nM) and is converted to dihydrotestosterone (DHT) by 5-alpha-reductase (more potent AR agonist) and to estradiol by aromatase (estrogen receptor agonist). Approved as testosterone replacement therapy in male hypogonadism via various ester formulations: cypionate (weekly), enanthate (weekly), propionate (every 2 to 3 days), undecanoate (oral and depot), gels and patches (daily). Schedule III in the US under the CSA. The compound is the foundation of most anabolic-androgenic steroid (AAS) regimens used non-medically for muscle building. Plasma half-life of native testosterone is short (1 to 2 hours); ester formulations release the parent compound over days. Used as the canonical AR agonist in research.

### 3. Mechanism of Action

Androgen receptor agonist; converted to DHT (5-alpha-reductase) and estradiol (aromatase) for tissue-specific effects. Ester formulations extend release.

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

TRT 50 to 200 mg cypionate IM weekly; non-medical AAS use is supratherapeutic.

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. Bhasin S, et al. Testosterone therapy in men with hypogonadism: an Endocrine Society clinical practice guideline. J Clin Endocrinol Metab 2018.
2. Kicman AT. Pharmacology of anabolic steroids. Br J Pharmacol 2008.
3. Mooradian AD, et al. Biological actions of androgens. Endocr Rev 1987.

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