

RESEARCH MONOGRAPH . COMPOUND PROFILE

Oxandrolone

A 17-alpha-alkylated synthetic anabolic steroid; clinically used in burn recovery and HIV wasting; widely used in non-medical AAS contexts.

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AUTHOR	Kodiac biolabs Research
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RESEARCH USE ONLY

ABSTRACT

Oxandrolone (17-beta-hydroxy-17-alpha-methyl-2-oxa-5-alpha-androstan-3-one; CAS 53-39-4; molecular formula C₁₉H₃₀O₃; molecular weight 306.44) is a 17-alpha-alkylated synthetic anabolic-androgenic steroid (AAS) developed by Searle in 1962 and approved by the FDA (Anavar, Oxandrin) for severe weight loss following surgery, trauma, infection, or chronic illness; for relief of bone pain in osteoporosis; and for compensation of catabolic states. Mechanism: AR agonism (lower than testosterone but with relatively higher anabolic-to-androgenic ratio in the Hersherberger assay). The 2-oxa substitution and 17-alpha alkylation produce oral activity (resistance to first-pass hepatic metabolism) and a more anabolic profile relative to androgenic. Plasma half-life is approximately 9 hours. Schedule III in the US under the CSA. Used in burn recovery (where AAS clinical use is well-established) and in HIV wasting; the most commonly diverted oral AAS for non-medical bodybuilding owing to a perceived favorable side effect profile. Used as a reference 17-alpha-alkylated oral AAS in research.

1. Compound Identification

Oxandrolone is described as 17-alpha-alkylated oral anabolic steroid. A 17-alpha-alkylated synthetic anabolic steroid; clinically used in burn recovery and HIV wasting; widely used in non-medical AAS contexts.

1.1 Class summary

17-alpha-alkylated oral anabolic steroid

1.2 Subtitle

A 17-alpha-alkylated synthetic anabolic steroid; clinically used in burn recovery and HIV wasting; widely used in non-medical AAS contexts.

2. Abstract

Oxandrolone (17-beta-hydroxy-17-alpha-methyl-2-oxa-5-alpha-androstan-3-one; CAS 53-39-4; molecular formula C₁₉H₃₀O₃; molecular weight 306.44) is a 17-alpha-alkylated synthetic anabolic-androgenic steroid (AAS) developed by Searle in 1962 and approved by the FDA (Anavar, Oxandrin) for severe weight loss following surgery, trauma, infection, or chronic illness; for relief of bone pain in osteoporosis; and for compensation of catabolic states. Mechanism: AR agonism (lower than testosterone but with relatively higher anabolic-to-androgenic ratio in the Hersherberger assay). The 2-oxa substitution and 17-alpha alkylation produce oral activity (resistance to first-pass hepatic metabolism) and a more anabolic profile relative to androgenic. Plasma half-life is approximately 9 hours. Schedule III in the US under the CSA. Used in burn recovery (where AAS clinical use is well-established) and in HIV wasting; the most commonly diverted oral AAS for non-medical bodybuilding owing to a perceived favorable side effect profile. Used as a reference 17-alpha-alkylated oral AAS in research.

3. Mechanism of Action

17-alpha-alkylated oral anabolic-androgenic steroid; AR agonism with relatively favorable anabolic-to-androgenic ratio. Hepatotoxic at high doses owing to alkylation.

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.

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. Wolfe RR, et al. Effects of oxandrolone on whole body protein synthesis. J Clin Endocrinol Metab 1995.
2. Berger JR, et al. Oxandrolone in AIDS-wasting myopathy. AIDS 1996.
3. Murad MH, et al. Drug-induced hepatotoxicity. Clin Liver Dis 2011.

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