

RESEARCH MONOGRAPH . COMPOUND PROFILE

# Cardarine (GW-501516)

*A PPAR-delta agonist developed for dyslipidemia; widely used recreationally as an endurance enhancer despite cancer findings in long-term rodent studies.*

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## ABSTRACT

Cardarine (GW-501516, Endurobol; CAS 317318-70-0; molecular formula  $C_{21}H_{18}F_3NO_3S_2$ ; molecular weight 453.50) is a peroxisome proliferator-activated receptor delta (PPAR-delta) agonist developed at GlaxoSmithKline for dyslipidemia. The compound is not a SARM; it is grouped with SARMs in popular discourse owing to similar recreational use patterns. PPAR-delta activation in skeletal muscle increases fatty acid oxidation, mitochondrial biogenesis, and endurance capacity. Phase 2 trials in dyslipidemia demonstrated favorable lipid profile changes (HDL increase, triglyceride decrease). Development was halted by GSK in 2007 after long-term carcinogenicity studies in rats and mice showed dose-dependent tumor formation in multiple organs at all dose levels tested. The compound retains research utility as the canonical PPAR-delta agonist for academic metabolic research and is widely used recreationally despite the carcinogenicity signal; banned by WADA. Plasma half-life is approximately 24 hours.

# 1. Compound Identification

Cardarine (GW-501516) is described as ppar-delta agonist (not a sarm). A PPAR-delta agonist developed for dyslipidemia; widely used recreationally as an endurance enhancer despite cancer findings in long-term rodent studies.

## 1.1 Class summary

PPAR-delta agonist (not a SARM)

## 1.2 Subtitle

A PPAR-delta agonist developed for dyslipidemia; widely used recreationally as an endurance enhancer despite cancer findings in long-term rodent studies.

## 2. Abstract

Cardarine (GW-501516, Endurobol; CAS 317318-70-0; molecular formula C<sub>21</sub>H<sub>18</sub>F<sub>3</sub>NO<sub>3</sub>S<sub>2</sub>; molecular weight 453.50) is a peroxisome proliferator-activated receptor delta (PPAR-delta) agonist developed at GlaxoSmithKline for dyslipidemia. The compound is not a SARM; it is grouped with SARMs in popular discourse owing to similar recreational use patterns. PPAR-delta activation in skeletal muscle increases fatty acid oxidation, mitochondrial biogenesis, and endurance capacity. Phase 2 trials in dyslipidemia demonstrated favorable lipid profile changes (HDL increase, triglyceride decrease). Development was halted by GSK in 2007 after long-term carcinogenicity studies in rats and mice showed dose-dependent tumor formation in multiple organs at all dose levels tested. The compound retains research utility as the canonical PPAR-delta agonist for academic metabolic research and is widely used recreationally despite the carcinogenicity signal; banned by WADA. Plasma half-life is approximately 24 hours.

### 3. Mechanism of Action

PPAR-delta agonist; activates fatty acid oxidation, mitochondrial biogenesis, and endurance capacity in skeletal muscle. Not a SARM despite popular grouping.

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

Trial doses 2.5 to 10 mg per oral administration daily; recreational use commonly 10 to 20 mg 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. Sprecher DL, et al. Triglyceride: high-density lipoprotein cholesterol effects in healthy subjects administered a peroxisome proliferator activated receptor delta agonist. *Arterioscler Thromb Vasc Biol* 2007.
2. Wang YX, et al. Regulation of muscle fiber type and running endurance by PPAR-delta. *PLoS Biol* 2004.
3. Geiger LE, et al. Rat carcinogenicity study with GW501516, a PPAR delta agonist. *The Toxicologist* 2009.

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