

RESEARCH MONOGRAPH . COMPOUND PROFILE

Cannabigerol (CBG)

A non-intoxicating phytocannabinoid; the metabolic precursor of THC and CBD; investigated for inflammation, neuroprotection, and inflammatory bowel disease.

DOCUMENT	KDC-MN-321
ISSUED	2026-05-09
REVISION	1.0
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COMPOUND CLASS	Phytocannabinoid (CB receptor weak agonist)

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ABSTRACT

Cannabigerol (CBG; CAS 25654-31-3; molecular formula $C_{21}H_{32}O_2$; molecular weight 316.48) is a non-intoxicating phytocannabinoid from *Cannabis sativa*. The compound is the biosynthetic precursor of THC, CBD, and CBC in plants; mature plants typically contain less than 1 percent CBG owing to enzymatic conversion to these downstream cannabinoids. Mechanism: CB1 and CB2 partial agonism (weaker than THC), α -2 adrenergic agonism, 5-HT1A antagonism (opposite of CBD), and PPAR-gamma agonism. The mechanism profile is distinct from both THC and CBD. Preclinical evidence supports anti-inflammatory effects in colitis models, neuroprotection in Huntington and Parkinson models, and antibiotic activity against MRSA. Human clinical trial data are sparse; the compound is sold as a supplement with limited regulatory guidance. Plasma pharmacokinetics are similar to other phytocannabinoids: high lipophilicity, hepatic metabolism via CYP enzymes, long terminal half-life. Used as a non-intoxicating phytocannabinoid in inflammation and neuroprotection research.

1. Compound Identification

Cannabigerol (CBG) is described as phytocannabinoid (cb receptor weak agonist). A non-intoxicating phytocannabinoid; the metabolic precursor of THC and CBD; investigated for inflammation, neuroprotection, and inflammatory bowel disease.

1.1 Class summary

Phytocannabinoid (CB receptor weak agonist)

1.2 Subtitle

A non-intoxicating phytocannabinoid; the metabolic precursor of THC and CBD; investigated for inflammation, neuroprotection, and inflammatory bowel disease.

2. Abstract

Cannabigerol (CBG; CAS 25654-31-3; molecular formula C₂₁H₃₂O₂; molecular weight 316.48) is a non-intoxicating phytocannabinoid from *Cannabis sativa*. The compound is the biosynthetic precursor of THC, CBD, and CBC in plants; mature plants typically contain less than 1 percent CBG owing to enzymatic conversion to these downstream cannabinoids. Mechanism: CB1 and CB2 partial agonism (weaker than THC), alpha-2 adrenergic agonism, 5-HT_{1A} antagonism (opposite of CBD), and PPAR-gamma agonism. The mechanism profile is distinct from both THC and CBD. Preclinical evidence supports anti-inflammatory effects in colitis models, neuroprotection in Huntington and Parkinson models, and antibiotic activity against MRSA. Human clinical trial data are sparse; the compound is sold as a supplement with limited regulatory guidance. Plasma pharmacokinetics are similar to other phytocannabinoids: high lipophilicity, hepatic metabolism via CYP enzymes, long terminal half-life. Used as a non-intoxicating phytocannabinoid in inflammation and neuroprotection research.

3. Mechanism of Action

CB1/CB2 partial agonism (weaker than THC); alpha-2 adrenergic agonism; 5-HT1A antagonism; PPAR-gamma agonism.

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

Supplement use 5 to 50 mg per oral administration daily; clinical trial doses up to 600 mg/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. Borrelli F, et al. Beneficial effect of the non-psychotropic plant cannabinoid cannabigerol on experimental inflammatory bowel disease. *Biochem Pharmacol* 2013.
2. Valdeolivas S, et al. Neuroprotective properties of cannabigerol in Huntington's disease. *Neurotherapeutics* 2015.
3. Appendino G, et al. Antibacterial cannabinoids from *Cannabis sativa*. *J Nat Prod* 2008.

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