

RESEARCH MONOGRAPH . COMPOUND PROFILE

Cannabinol (CBN)

A phytocannabinoid produced by THC oxidation in aged cannabis; weakly intoxicating; widely marketed as a sleep aid despite limited clinical evidence.

DOCUMENT	KDC-MN-322
ISSUED	2026-05-09
REVISION	1.0
AUTHOR	Kodi <i>ac</i> biolabs Research
COMPOUND CLASS	Phytocannabinoid (THC degradation product)

ABSTRACT

Cannabinol (CBN; CAS 521-35-7; molecular formula $C_{21}H_{26}O_2$; molecular weight 310.43) is a phytocannabinoid formed primarily by oxidative degradation of THC; aged cannabis develops higher CBN concentrations. The compound retains weak CB1 partial agonist activity (approximately 10 percent of THC potency at CB1), producing mild psychoactive effects at high doses. CB2 affinity is comparable to or higher than CB1. Pharmacologically distinguished from THC by the aromatized A-ring, which reduces CB1 binding but preserves other interactions. Marketing claims position CBN as a sleep aid; clinical evidence for this is limited (one 1976 trial and small subsequent studies), and the sedation may largely reflect residual THC contamination in early CBN samples. Plasma kinetics parallel other cannabinoids. Used as a CB receptor partial agonist research compound; the sleep marketing claims await rigorous clinical validation.

1. Compound Identification

Cannabinol (CBN) is described as phytocannabinoid (the degradation product). A phytocannabinoid produced by THC oxidation in aged cannabis; weakly intoxicating; widely marketed as a sleep aid despite limited clinical evidence.

1.1 Class summary

Phytocannabinoid (THC degradation product)

1.2 Subtitle

A phytocannabinoid produced by THC oxidation in aged cannabis; weakly intoxicating; widely marketed as a sleep aid despite limited clinical evidence.

2. Abstract

Cannabinol (CBN; CAS 521-35-7; molecular formula $C_{21}H_{26}O_2$; molecular weight 310.43) is a phytocannabinoid formed primarily by oxidative degradation of THC; aged cannabis develops higher CBN concentrations. The compound retains weak CB1 partial agonist activity (approximately 10 percent of THC potency at CB1), producing mild psychoactive effects at high doses. CB2 affinity is comparable to or higher than CB1. Pharmacologically distinguished from THC by the aromatized A-ring, which reduces CB1 binding but preserves other interactions. Marketing claims position CBN as a sleep aid; clinical evidence for this is limited (one 1976 trial and small subsequent studies), and the sedation may largely reflect residual THC contamination in early CBN samples. Plasma kinetics parallel other cannabinoids. Used as a CB receptor partial agonist research compound; the sleep marketing claims await rigorous clinical validation.

3. Mechanism of Action

Oxidation product of THC; weak CB1 partial agonist (~10 percent of THC potency); CB2 activity comparable. Sedative claims poorly substantiated.

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 2.5 to 25 mg per oral administration before sleep; clinical evidence sparse.

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. Karniol IG, et al. Effects of delta-9-tetrahydrocannabinol and cannabinalol in man. Pharmacology 1975.
2. Bonn-Miller MO, et al. Cannabinalol for sleep: an evidence review. Sleep Med Rev 2022.
3. Russo EB. Cannabis pharmacology: the usual suspects and a few promising leads. Adv Pharmacol 2017.

This monograph is provided for research use only. Not for medical, diagnostic, or therapeutic purposes. Not for human consumption. Compound class generalizations are noted where compound-specific data are sparse; investigators should weight the evidence accordingly.