

RESEARCH MONOGRAPH . COMPOUND PROFILE

Cannabidiol (CBD)

A non-psychotomimetic phytocannabinoid from Cannabis; FDA-approved for refractory pediatric epilepsy syndromes; widely used for anxiety, sleep, and inflammation.

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COMPOUND CLASS	Phytocannabinoid (non-intoxicating)

RESEARCH USE ONLY

ABSTRACT

Cannabidiol (CBD; CAS 13956-29-1; molecular formula $C_{21}H_{30}O_2$; molecular weight 314.46) is the principal non-intoxicating phytocannabinoid of *Cannabis sativa*, approved by the FDA in 2018 as Epidiolex for Dravet syndrome, Lennox-Gastaut syndrome, and tuberous sclerosis complex-associated seizures. Mechanism is multifactorial and incompletely characterized: minimal CB1 affinity (avoiding the THC-like psychoactivity), CB2 partial agonism, allosteric CB1 negative modulation (potentially attenuating THC effects), TRPV1 agonism, GPR55 antagonism, 5-HT1A partial agonism, and inhibition of fatty acid amide hydrolase (raising endogenous anandamide). Plasma half-life is 18 to 32 hours; metabolism is via CYP3A4 and CYP2C19, with CBD itself being a moderate inhibitor of CYP3A4 and CYP2D6 producing clinically relevant drug-drug interactions. Approved indications: refractory pediatric epilepsy. Off-label use is extensive (anxiety, sleep, pain, inflammation) with mixed clinical evidence and substantial product variability in the consumer market. Used as the canonical non-intoxicating phytocannabinoid in research.

1. Compound Identification

Cannabidiol (CBD) is described as phytocannabinoid (non-intoxicating). A non-psychotomimetic phytocannabinoid from Cannabis; FDA-approved for refractory pediatric epilepsy syndromes; widely used for anxiety, sleep, and inflammation.

1.1 Class summary

Phytocannabinoid (non-intoxicating)

1.2 Subtitle

A non-psychotomimetic phytocannabinoid from Cannabis; FDA-approved for refractory pediatric epilepsy syndromes; widely used for anxiety, sleep, and inflammation.

2. Abstract

Cannabidiol (CBD; CAS 13956-29-1; molecular formula C₂₁H₃₀O₂; molecular weight 314.46) is the principal non-intoxicating phytocannabinoid of *Cannabis sativa*, approved by the FDA in 2018 as Epidiolex for Dravet syndrome, Lennox-Gastaut syndrome, and tuberous sclerosis complex-associated seizures. Mechanism is multifactorial and incompletely characterized: minimal CB1 affinity (avoiding the THC-like psychoactivity), CB2 partial agonism, allosteric CB1 negative modulation (potentially attenuating THC effects), TRPV1 agonism, GPR55 antagonism, 5-HT_{1A} partial agonism, and inhibition of fatty acid amide hydrolase (raising endogenous anandamide). Plasma half-life is 18 to 32 hours; metabolism is via CYP3A4 and CYP2C19, with CBD itself being a moderate inhibitor of CYP3A4 and CYP2D6 producing clinically relevant drug-drug interactions. Approved indications: refractory pediatric epilepsy. Off-label use is extensive (anxiety, sleep, pain, inflammation) with mixed clinical evidence and substantial product variability in the consumer market. Used as the canonical non-intoxicating phytocannabinoid in research.

3. Mechanism of Action

Multifactorial: weak CB1, CB2 partial agonism, allosteric CB1 modulation, TRPV1, GPR55, 5-HT1A, FAAH inhibition. CYP3A4 and CYP2D6 inhibition.

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 25 mg/kg/day per oral administration (Epidiolex). Supplement use 25 to 1000 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. Devinsky O, et al. Trial of cannabidiol for drug-resistant seizures in the Dravet syndrome. N Engl J Med 2017.
2. Ibeas Bih C, et al. Molecular targets of cannabidiol in neurological disorders. Neurotherapeutics 2015.
3. Bonn-Miller MO, et al. Labeling accuracy of cannabidiol extracts sold online. JAMA 2017.

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