

Cannabinol and Sleep: Separating Fact from Fiction

Jamie Corroon^{1,2,*i}

Abstract

In recent years, marketers of cannabis (i.e., marijuana) products have claimed that cannabinol (CBN) has unique sleep-promoting effects. Despite a plausible mechanism, it is possible that such claims are merely rooted in cannabis lore. The aim of this narrative review was to answer the question: “Is there sufficient clinical evidence to support claims that CBN has sleep-promoting effects?” A systematic search of PubMed/MEDLINE was performed to evaluate the published evidence. The abstracts of 99 human studies were screened for relevance by the author and reviewed for compliance with the inclusion criteria. The characteristics and principal findings were extracted from eight full-text articles that met inclusion criteria for detailed review. Pre-clinical and clinical research investigating the effects of CBN is dated and limited, with the preponderance of human studies occurring in the 1970–1980s with small sample sizes lacking diversity in sociodemographic characteristics. Studies specifically assessing subjective effects associated with sleep, such as sedation or fatigue, are rare. Most importantly, published clinical trials investigating associations between CBN and validated sleep questionnaires and/or formal polysomnography were not identified in this review. In addition, evidence demonstrating that CBN itself elicits cannabis-like effects in humans is mixed, with the majority of available evidence demonstrating a lack of such an effect. Consequently, there is insufficient published evidence to support sleep-related claims. Randomized controlled trials are needed to substantiate claims made by manufacturers of cannabis products containing CBN. These studies should specifically evaluate its effects on sleep through polysomnography, or at minimum, through validated sleep questionnaires, and use dosages significantly higher than those found in currently available cannabis products marketed for sleep (typically ≤ 5 mg). Individuals seeking cannabis-derived sleep aids should be skeptical of manufacturers’ claims of sleep-promoting effects.

Keywords: cannabinoids; cannabis; CBN; marijuana; sleep; THC

Introduction

In recent years, marketers of cannabis (i.e., marijuana) products have claimed on websites, marketing materials, and product labels that cannabinol (CBN) has unique sleep-promoting effects (e.g., “CBN’s sedative properties are up to 10 times stronger than those of prescription and over-the-counter sleeping drugs”).^{1,2} It is possible such claims are rooted in cannabis lore. Namely, that “old” cannabis makes users sleepy.³ In this historical context, and given a plausible mecha-

nism of action, the aim of this narrative review was to answer the question: “Is there sufficient clinical evidence to support claims that CBN has sleep-promoting effects?”

In the United States, as a constituent in cannabis, CBN is deemed a Schedule I controlled substance, per the definition of marijuana in the Controlled Substances Act of 1970.⁴ As a constituent in hemp, it is not a controlled substance at all, per the Agricultural Improvement Act of 2018 (aka the 2018 Farm Bill).⁵

¹The Center for Medical Cannabis Education, Del Mar, California, USA.

²National University of Natural Medicine, Helfgott Research Institute, Portland, Oregon, USA.

³ORCID ID (<https://orcid.org/0000-0003-3930-6093>).

*Address correspondence to: Jamie Corroon, ND, MPH, The Center for Medical Cannabis Education, 1155 Camino del Mar, #187, Del Mar, CA 92014, USA, E-mail: jamie@corroon.com

As such, the regulatory status of CBN is similar to that of cannabidiol (CBD), or any other phytocannabinoid derived from *Cannabis* spp., with the exception of THC in hemp, which is not a controlled substance in concentrations up to, and including, 0.3% on a dry weight basis.⁶

Cannabis products containing CBN are only available in US states with regulated medical and/or recreational cannabis programs. Despite the absence of restrictive federal regulations, hemp-derived CBN products are a rarity compared with their CBD-containing counterparts. Health-related claims for cannabis products are regulated on a state-by-state basis, whereas claims for hemp products are regulated by the U.S. Food and Drug Administration and Federal Trade Commission.⁷

Internationally, the United Nations Single Convention of Narcotic Drugs in 1961 (Single Convention) does not discriminate between cannabis and hemp. According to the Single Convention, compounds derived from “any plant of the genus *Cannabis*” are deemed narcotics.⁸ Individual countries and regions (e.g., European Union) may have their own regulations, but in their absence the Single Convention will prevail unless and until the United Nations votes to change it.

Phytochemistry and pharmacognosy

CBN was the first phytocannabinoid to be identified in the *Cannabis sativa* L. plant by scientists in the 1930s.⁹ Unlike other phytocannabinoids, CBN is not biosynthesized in an acid form by the plant. Rather, is a degradative product of delta-9-tetrahydrocannabinol (delta-9-THC or THC). As such, CBN concentrations in plant material and extracts are low, but increase over time as THC is exposed to light, oxygen, and heat.¹⁰

CBN is a partial agonist of the CB1 receptor with a lesser affinity than THC (CBN: K_i at CB1 = 211.2 nM vs. THC K_i at CB1 = 21 nM).^{11,12} This receptor is purportedly responsible for the “psychoactive” effects of *Cannabis* spp., including sleep-related effects such as sedation (e.g., hypolocomotion and catalepsy).¹³

Pre-clinical research

Pre-clinical research investigating *in vivo* effects of CBN dates back to the 1940s. A 1945 pre-clinical study by Loewe, entitled “Marihuana Activity of Cannabinol,” found that 12 mg/kg of CBN administered intravenously produced clear signs of

ataxia in dogs.¹⁴ The author concluded, “cannabinol must be included among compounds having marihuana activity.”

Work by Mechoulam et al.¹⁵ in 1970 and Frankenheim et al.¹⁶ in 1971, however, reported no effect of CBN on monkeys or pigeons, respectively. Yet in 1974, Karniol et al. demonstrated that CBN prolonged barbiturate-induced sleeping time in mice,¹⁷ a finding that was replicated in 1987 by Yamamoto et al. with additional effects on catalepsy and hypothermia.¹⁸

Methods

To evaluate the published evidence regarding CBN and sleep, a narrative review was prepared after a systematic search of PubMed/MEDLINE for original research articles using the following keywords: “Cannabinol”[Mesh] (242 studies published between 1945 and January 2021) and “Cannabinol”[Mesh] AND “Humans”[Mesh] (99 studies published between 1945 and January 2021). Inclusion criteria were twofold: (1) administration of CBN to human participants and (2) measurement of subjective and/or objective outcomes related to sleep (fatigue, tiredness, etc.) and/or THC-like effects. The abstracts of 99 human studies were screened for relevance by the author and reviewed for compliance with the inclusion criteria. The characteristics and principal findings were extracted from eight full-text articles that met inclusion criteria for detailed review.

Results

Clinical research

In 1973, Perez-Reyes et al. compared the pharmacological activity of various phytocannabinoids with placebo (See Table 1). Six healthy male volunteers were administered an intravenous (IV) infusion of CBN at a rate of ~1.2 mg/min in a hospital setting.¹⁹ A dose of 200 μ g/kg was required for volunteers to demonstrate a 25% increase in heart rate (a physiological effect consistent with CB1 mediation of the central nervous system) and report any subjective effects of a cannabis-like “high.” The authors concluded, “we have found that cannabinol is capable of producing a marihuana-like ‘high’ although the doses necessary for it are several orders of magnitude larger than those of delta-9-THC.” Neither fatigue, as an adverse effect, nor sleep were specifically assessed.

In the same year, Hollister orally administered CBN ($n=6$) and CBD ($n=5$) separately to healthy male

Table 1. Clinical Studies Included in This Review

Participant characteristics				Study characteristics			CBN-specific result
Author	Health status	Age (Years)	Sample size	Design	Study drug(s)	Dose, route of administration	
Perez-Reyes et al. ¹⁹	Healthy male volunteers	NA	n = 32	Single-blind placebo-controlled trial	CBN, THC, CBD	Intravenous administration (CBN: 1.2 mg/min; THC: 0.2 mg/min; CBD: 1.78 mg/min)	Cannabis-like activity demonstrated "we have found that cannabidiol is capable of producing a marihuana-like 'high' although the doses necessary for it are several orders of magnitude larger than those of delta-9-THC." "At no oral dose level were any of the characteristic mental or physical effects of THC observed."
Hollister ²⁰	Healthy male volunteers	30–50	n = 15	Clinical trial	CBN, CBD	Oral administration (CBN: 20 to 400 mg; CBD: 20 to 100 mg)	"...subjects reported that they felt drowsy under the influence of delta-9-THC, but not under the influence of CBN."
Karniol et al. ²³	Male volunteers	25–29	n = 5	Double-blind placebo-controlled drug interaction trial	CBN, THC, and different combinations of both	Oral administration (placebo; CBN: 50 mg; THC: 25 mg; CBN: 12.5 mg + THC: 25 mg; CBN: 25 mg + THC: 25 mg; CBN: 50 mg + THC: 25 mg)	"...subjects reported that they felt drowsy under the influence of delta-9-THC, but not under the influence of CBN."
Hollister and Gillespie ²⁴	Healthy male volunteers	> 18	n = 15	Double-blind placebo-controlled trial	THC combined with placebo, CBN, or CBD	Oral administration (placebo; THC: 20 mg + placebo; THC: 20 mg + CBN: 40 mg; THC: 20 mg + CBD: 40 mg)	"No quantitative or temporal difference was observed between THC-placebo and THC-CBN in terms of clinical effects. Qualitatively, each treatment produced identical effects."
Bird et al. ²⁵	Healthy volunteers	18–36	n = 161	Double-blind placebo-controlled trial	THC, CBD, and CBN alone and in all possible combinations	Oral administration (placebo; THC: 215 µg/kg; CBD: 320 µg/kg; CBN: 320 µg/kg; ethanol: 0.54 g/kg)	"There was no suggestion of systematic effects involving CBD or CBN, either alone or in combination with other drugs."
Agurell et al. ²⁶	Healthy male volunteers	18–40	n = 12	Cross-over pharmacokinetic drug interaction trial	THC alone and with CBD and CBN	Oral administration (THC: 20 mg; THC: 20 mg + placebo; THC: 20 mg + 40 mg CBN; THC: 20 mg + 40 mg CBD)	Concurrent administration of CBN and THC resulted in a blood profile similar to that of THC itself (i.e., absence of a drug-drug interaction).
Gong et al. ²⁷	Experienced cannabis smokers	21–32	n = 59	Three double-blind clinical trials (i.e., dose-response study, interaction study, subacute study)	THC, CBN, and CBD alone and in various combinations	Oral administration. Dose-response study: delta-9-THC: 20 mg; delta-8-THC: 50 mg, 75 mg; CBN: 100 mg, 600 mg, 1200 mg; CBD: 100 mg, 600 mg, 1200 mg; diazepam 5 mg). Interaction study: (placebo; THC 10 mg; CBN 600 mg; CBD 1200 mg; THC 5 mg + CBN 400 mg; THC 5 mg + CBD 400 mg)	"Only delta-9-THC and both doses of delta-8-THC induced higher mean peak highs than placebo."
Johansson et al. ²⁸	Healthy male volunteers	19–31	n = 6	Cross-over pharmacokinetic trial comparing IV administration with inhalation	CBN	IV administration: CBN: 20 mg; inhalation: CBN: 20 mg. THC: 0 mg	"no psychoactive effects noted."

CBD, cannabidiol; CBN, cannabiniol; IV, intravenous; THC, delta-9-tetrahydrocannabinol.

volunteers in doses of CBN ranging from 20 to 400 mg.²⁰ The authors stated, “At no oral dose level were any of the characteristic mental or physical effects of THC observed.”

The contradictory conclusions from Perez-Reyes et al. and Hollister et al. in 1973 are not easily reconciled by differences in dosage and route of administration. In the former study, CBN was administered intravenously in milligram doses (up to ~12.4 mg before demonstrating a 25% increase in heart rate (estimated using a global average body mass of 62 kg)²¹). In the latter, it was administered orally in milligram doses. Oral bioavailability of phytocannabinoids is low due to poor aqueous solubility and extensive first-pass metabolism.²² Like THC, biotransformation of CBN by cytochrome P450 enzymes at the 11th carbon yields a more active metabolite (i.e., 11-hydroxy-CBN); twice as active in at least three pharmacological indices.¹⁸ The highest orally administered dose (i.e., 400 mg) in the Hollister et al. study would likely yield an estimated blood level of CBN, and its metabolites, above that of the intravenously administered dose. Thus, an explanation of the contradictory outcomes is elusive.

In a 1975 study, Karniol et al. investigated the effects of, and interactions between, various doses and combinations of THC and CBN. Five male volunteers were orally administered six study drugs (placebo, CBN 50 mg, THC 25 mg, CBN 12.5 mg + THC 25 mg, CBN 25 mg + THC 25 mg, and CBN 50 mg + THC 25 mg) in a double-blind design.²³ A number of physiological and psychophysical outcomes were measured, including a 66-item Drug Reaction Scale. Volunteers reported feeling more “drugged, drunk, dizzy, and drowsy” when using some of the combined drug treatments (CBN and THC), but not when using CBN alone or placebo.

In addition, only some of the three CBN and THC combinations led to differences in subjective reports of feeling “drugged, drunk, dizzy, and drowsy” when compared with THC alone (for drugged: all combinations > THC 25 mg; for drowsy: CBN 25 mg + THC 25 mg and CBN 50 mg + THC 25 mg > THC 25 mg; for drunk: all combinations > THC 25 mg; dizzy: CBN 12.5 mg + THC 25 mg > THC 25 mg). No dose-response relationship was observed. More importantly, for CBN itself, the mean change from predrug state was not statistically significantly different from placebo for all of the four subjectively reported categories. The authors concluded, “subjects reported that they felt

drowsy under the influence of delta-9-THC, but not under the influence of CBN.” A potentially significant limitation of this study, however, is the small sample size that consisted of five males in their mid-to-late 20s, four of whom were residents at a psychiatric facility and were likely taking other psychotropic medications, which may have confounded the results and limits general conclusions.

In the same year, Hollister et al. administered oral THC (20 mg) combined with either placebo, CBN (40 mg), or CBD (40 mg) to 15 healthy male volunteers in random order on separate occasions.²⁴ Unlike the Karniol et al. study, CBN did not affect heart rate or subjective drug effects when combined with THC. The investigators concluded, “No quantitative or temporal difference was observed between THC-placebo and THC-CBN in terms of clinical effects. Qualitatively, each treatment produced identical effects.” The larger sample size in this later study, combined with the absence of a difference between CBN and placebo in either study, lends greater credibility to the conclusion that there is no difference in subjective effects between THC and the same dose of THC combined with CBN.

In 1980, Bird et al. compared the effects of orally administered doses of THC, CBD, and CBN (320 μ g/kg, ~20 mg, using a global average body mass of 62 kg), alone and in all possible combinations, in human volunteers across a series of perceptual, cognitive, and motor function tests.²⁵ The authors concluded, “There was no suggestion of systemic effects involving CBD or CBN, either alone or in combination with other drugs.” In terms of potentiation of THC effects, these findings agree with the findings of Hollister et al. in 1975 but disagree with the findings of Karniol et al. in the same year, where CBN seemed to potentiate some effects of THC (i.e., “drugged, drunk, dizzy, and drowsy”).

In a pharmacokinetic study conducted in 1981, Agurell et al. studied blood concentrations of select phytocannabinoids after oral administration of 20 mg THC combined with placebo, 40 mg CBN or 40 mg CBD in a cross-over sequence.²⁶ The concurrent administration of CBN and THC resulted in a similar blood profile to that of THC itself. This finding suggests that any systemic effects, if observed, would not be due to elevated blood levels of THC resulting from a drug-drug interaction with CBN.

In 1984, Gong et al. evaluated bronchodilating activity of orally administered cannabinoids in 59

experienced male cannabis smokers in three distinct studies.²⁷ In a dose–response study, CBN was administered in doses of 100 mg ($n=6$), 600 mg, and 1200 mg ($n=14$ for each) and showed no dose-related bronchodilating effects. On a subjectively reported 7-point intoxication scale, mean peak “high” scores were <1 for all doses of CBN with no indication of a linear dose–response relationship. According to the authors, “Only delta-9-THC and both doses of delta-8-THC induced higher mean peak highs than placebo.”

Twelve of the participants were enrolled in a randomized double-blind crossover drug interaction study and received THC, CBN, and CBD alone and in various combinations (placebo, THC 10 mg, CBN 600 mg, CBD 1200 mg, THC 5 mg+CBN 400 mg, THC 5 mg+CBD 400 mg). The authors reported that CBN and CBD had “no demonstrable effect on heart rate or ‘high.’” No tolerance was noted after 20-day administration of 600 mg of CBN daily.

In 1987, Johansson et al. measured blood levels of CBN in six healthy male volunteers after IV infusion of 20 mg of CBN, and again, a week later, after inhalation of a *Cannabis* spp. cigarette with 20 mg of CBN and no THC.²⁸ Subjective effects such as fatigue and sedation were not formally assessed, but the authors concluded there were “no psychoactive effects noted” for either route of administration.

This study was overseen by Hollister who reported an absence of THC-like effects after orally administering CBN to 6 healthy male volunteers in 1973 and coadministering CBN and THC to 15 healthy male volunteers in 1975. These studies,^{20,24,25,27,28} in combination with Karniol et al.’s findings of no difference between CBN and placebo in 1975, demonstrate a consistent absence of cannabis-like activity for CBN.

Safety and tolerability

Other than expected THC-induced deficits in cognitive, perceptual, and motor functions, which correlated with self-reported intoxication ratings (when reported), tolerability and safety measures were noticeably absent from the studies included in this review. Only Bird et al. reported adverse reactions in the article. Five of the subjects experienced adverse reactions when receiving THC with at least one other cannabinoid. These adverse events were described as “mild vagal reactions which were accompanied by a degree of anxiety.” It is unlikely that these adverse effects were due to CBN, given that the authors reported “no suggestion of systematic effects involving CBD or

CBN.” Specifically with regard to CBN, Perez-Reyes et al. reported that the volunteers were encouraged to receive the largest amount of CBN they could “comfortably tolerate.” None of the subjects asked for the infusion to be terminated and the experience was “mild and enjoyable” per their report.

Discussion

Pre-clinical and clinical research investigating the effects of CBN is dated and limited, with the preponderance of human studies occurring in the 1970–1980s with small sample sizes lacking diversity in sociodemographic characteristics. Studies specifically assessing subjective effects associated with sleep, such as sedation or fatigue, are rare. Most importantly, published clinical trials investigating associations between CBN and validated sleep questionnaires and/or formal polysomnography were not identified in this review. Evidence demonstrating that CBN itself elicits cannabis-like effects in humans is mixed, with the majority of available evidence demonstrating a lack of such an effect. The same is true for CBN-induced potentiation of THC effects.

From a mechanistic perspective, a low-affinity partial agonist of the CB1 receptor, like CBN, could cause sedation, as evidenced by experimental models investigating the effects of THC, another low-affinity partial agonist.⁹ As reviewed, this effect has only been demonstrated in some, not all, pre-clinical studies, and in only one clinical study. Consequently, there is insufficient published evidence to support a health claim related to sleep.

Given the timing, methodological limitations, and scarcity of the evidence base, it is possible that sleep-related effects have simply not yet been elucidated in an appropriately designed clinical trial. If pursued, future trials could focus on CBN derived from hemp, and/or non-narcotic sources (e.g., synthetic or semisynthetic) as a way of bypassing cumbersome regulations related to researching cannabis products.

The signal is weak in the current evidence base, and no clinical trials investigating CBN for sleep, or a related effect, are currently registered at ClinicalTrials.gov (positive results of a phase 1b/2a trial investigating an extract containing THC, CBD, and CBN among chronic insomnia patients were reported in February of 2020).²⁹ Future research investigating CBN should specifically evaluate its effects on sleep through polysomnography, or at minimum,

through validated sleep questionnaires, and use dosages significantly higher than those found in currently available cannabis products marketed for sleep (typically ≤ 5 mg).

Individuals seeking cannabis-derived sleep aids should be skeptical of manufacturers' claims of sleep-promoting effects.

Acknowledgments

The author thanks Michelle Sexton, ND, Ryan Bradley ND, MPH, and Rod Kight Esq. for their help with preparation of the article.

Author Disclosure Statement

J.C. is the medical director at the Center for Medical Cannabis Education, a for-profit clinical and consulting entity.

Funding Information

No funding was received for this article.

References

- Garber-Paul Elisabeth. Why CBN is a psychoactive sleep aid alternative. Rolling Stone. 2020. Updated July 21, 2020. <https://www.rollingstone.com/culture/culture-news/cbn-sleep-aid-psychoactive-1031811> Accessed July 21, 2020.
- Is CBN Oil Legal? 2021. <https://cbnoilforsleep.com/is-cbn-oil-legal> Accessed April 12, 2020.
- Peter Grinspoon MD. Beyond CBD: here come the other cannabinoids, but where's the evidence? Harvard Health Blog. 2021.
- Section 802—Title 21 United States Code (USC) Controlled Substances Act. 2018. <https://www.deadiversion.usdoj.gov/21cfr/21usc/802.htm> Accessed April 12, 2020.
- McConnell Mitch. Text—S.2667—115th Congress (2017–2018): Hemp Farming Act of 2018. 2018.
- Corroon J, Kight R. Regulatory status of cannabidiol in the United States: a perspective. Cannabis Cannabinoid Res. 2018;3:190–194.
- Corroon J, MacKay D, Dolphin W. Labeling of cannabidiol products: a public health perspective. Cannabis Cannabinoid Res. 2020;5:274–278.
- Crime United Nations Office on Drugs and. The International Drug Control Conventions. United Nations. 2021. www.unodc.org/unodc/en/commissions/CND/conventions.html Accessed May 12, 2020.
- Pertwee RG. Cannabinoid pharmacology: the first 66 years. Br J Pharmacol. 2006;147 Suppl 1:S163–S171.
- Kendall D, Alexander S. Cannabinoid pharmacology, vol. 80, 1st ed. Academic press: Cambridge, MA, 2020.
- Rhee MH, Vogel Z, Barg J, et al. Cannabinol derivatives: binding to cannabinoid receptors and inhibition of adenylyl cyclase. J Med Chem. 1997; 40:3228–3233.
- Showalter VM, Compton DR, Martin BR, et al. Evaluation of binding in a transfected cell line expressing a peripheral cannabinoid receptor (CB2): identification of cannabinoid receptor subtype selective ligands. J Pharmacol Exp Ther. 1996;278:989–999.
- Loewe S. Studies on the pharmacology and acute toxicity of compounds with marihuana activity. J Pharmacol Exp Ther. 1946;88:154–161.
- Loewe S. Marihuana activity of cannabiol. Science: New York, NY, 1945:102.
- Mechoulam R, Shani A, Ederly H, et al. Chemical basis of hashish activity. Science. 1970;169:611–612.
- Frankenheim JM, McMillan DE, Harris LS. Effects of I- Δ 9- and I- Δ 8-tetrahydrocannabinol and cannabiol on schedule-controlled behavior of pigeons and rats. J Pharmacol Exp Ther. 1971;178:241–252.
- Karniol IG, Takahashi RN, Musty RE. Effects of delta9-tetrahydrocannabinol and cannabiol on operant performance in rats. Arch Int Pharmacodyn Ther. 1974;212.
- Yamamoto I, Watanabe K, Kuzuoka K, et al. The pharmacological activity of cannabiol and its major metabolite, 11-hydroxycannabiol. Chem Pharm Bull. 1987;35.
- Perez-Reyes M, Timmons MC, Davis KH, et al. A comparison of the pharmacological activity in man of intravenously administered delta9-tetrahydrocannabinol, cannabiol, and cannabidiol. Experientia. 1973;29: 1368–1369.
- Hollister LE. Cannabidiol and cannabiol in man. Experientia. 1973;29: 825–826.
- Walpole Sarah Catherine, Prieto-Merino David, Edwards Phil, et al. The weight of nations: an estimation of adult human biomass. BMC Public Health. 2012;12:1–6.
- Huestis MA. Human cannabinoid pharmacokinetics. Chem Biodivers. 2007;4:1770–1804.
- Karniol IG, Shirakawa I, Takahashi RN, et al. Effects of delta9-tetrahydrocannabinol and cannabiol in man. Pharmacology. 1975;13: 502–512.
- Hollister LE, Gillespie H. Interactions in man of delta-9-tetrahydrocannabinol. II. Cannabiol and cannabidiol. Clin Pharmacol Ther. 1975;18.
- Bird KD, Boleyn T, Chesher GB, et al. Intercannabinoid and cannabinoid-ethanol interactions on human performance. Psychopharmacology. 1980;71:181–188.
- Agurell S, Carlsson S, Lindgren JE, et al. Interactions of delta 1-tetrahydrocannabinol with cannabiol and cannabidiol following oral administration in man. Assay of cannabiol and cannabidiol by mass fragmentography. Experientia. 1981;37:1090–1092.
- Gong H, Tashkin DP, Simmons MS, et al. Acute and subacute bronchial effects of oral cannabinoids. Clin Pharmacol Ther. 1984;35: 26–32.
- Johansson E, Ohlsson A, Lindgren JE, et al. Single-dose kinetics of deuterium-labelled cannabiol in man after intravenous administration and smoking. Biomed Environ Mass Spectrom. 1987;14:495–499.
- Search of: cannabiol—List Results—ClinicalTrials.gov. 2021. <https://www.clinicaltrials.gov/ct2/results?cond=&term=cannabiol&cntry=&state=&city=&dist=> Accessed May 19, 2020.

Cite this article as: Corroon J (2021) Cannabiol and sleep: separating fact from fiction, *Cannabis and Cannabinoid Research* X:X, 1–6, DOI: 10.1089/can.2021.0006.

Abbreviations Used

CBD = cannabidiol
 CBN = cannabiol
 IV = intravenous
 THC = delta-9-tetrahydrocannabinol