

WHITE PAPER

Tetrahydrocannabivarin (THCV)

History, chemistry & emerging therapeutic science

White paper | September 2026

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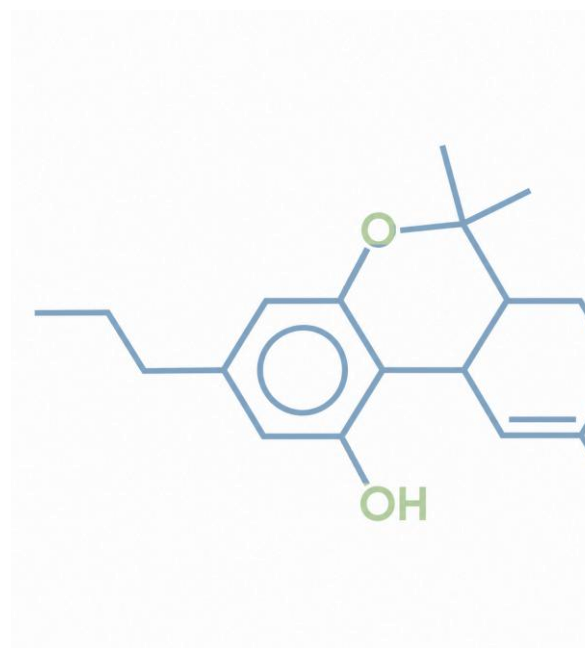


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Executive Summary

Tetrahydrocannabivarin (THCV) is a naturally occurring phytocannabinoid structurally related to Δ^9 -tetrahydrocannabinol (Δ^9 -THC), but it follows a different biosynthetic branch and has a materially different pharmacological profile. THCV carries a three-carbon propyl side chain, whereas Δ^9 -THC carries a five-carbon pentyl side chain. This apparently small structural change is associated with different receptor behaviour and requires a varin-capable chemotype in the plant.

THCV also has a direct historical connection to the last era of legal prescription cannabis in the UK. Tincture of Cannabis remained legally prescribable under a “licence of right” until 1973, when that licence was not renewed and medical use was prohibited. The commercial B.P.C. tincture studied by Gill, Paton and Pertwee was prepared from Cannabis sativa grown in Pakistan and imported into Britain under licence; they isolated a propyl homologue of THC, now recognised as THCV, from that material.¹

At lower concentrations, THCV is generally described as a CB1 neutral antagonist, whereas at higher concentrations weak partial agonist activity has been reported. This is an important distinction from rimonabant, which was developed as a CB1 inverse agonist and whose European marketing authorisation was suspended in 2008 because the balance of benefit and psychiatric risk was considered unfavourable.²

Within this context, THCV Harmony® is being developed by Glass Pharms® as a flower-format chemotype containing 10% THC and 7% THCV. Its potential lies in providing a repeatable, differentiated cannabinoid profile for future research and product development. This is a scientific development rationale, not a claim of established clinical efficacy.

¹ British Pharmaceutical Codex 1949. London: Pharmaceutical Press. Monographs for Cannabis, Extractum Cannabis and Tinctura Cannabis.

² McPartland JM et al. Are cannabidiol and Δ^9 -tetrahydrocannabivarin negative modulators of the endocannabinoid system? Br J Pharmacol. 2015;172:737–753. doi:10.1111/bph.12944; European Medicines Agency. Acomplia (rimonabant): EPAR and 2008 suspension/withdrawal information.

1. Historical Context

Cannabis tincture had been used medicinally in Britain for more than a century before its final withdrawal. The Medicines Act 1968 gave existing preparations a “licence of right”, which meant that doctors could continue to prescribe Tincture of Cannabis. In 1973 those licences were reviewed; the cannabis licence was not renewed and the first Misuse of Drugs Regulations placed cannabis and its derivatives in the category that prohibited medical use.³

The B.P.C. 1949 tincture

The British Pharmaceutical Codex (B.P.C.) 1949 retained standards for cannabis, cannabis extract and tincture after those preparations had been removed from the British Pharmacopoeia in 1932. William Ransom & Son Ltd of Hitchin, Hertfordshire manufactured a commercial tincture under this standard; see Figure 1. An Oxford pharmacology group led by Pertwee analysed the tincture and recorded that the material was prepared from Cannabis sativa grown in Pakistan and imported into Britain under licence.⁴

Formulation and cannabinoid profile

The B.P.C. 1949 standard defined both the extract and the tincture. Extractum Cannabis was prepared by percolating 1,000 g of coarsely powdered cannabis with 90% alcohol and evaporating the percolate to a soft extract. Tinctura Cannabis was then prepared by dissolving 50 g of that extract in 90% alcohol to a final volume of 1,000 mL. The finished tincture was specified at 83–87% v/v alcohol, with a stated dose of 0.3–1 mL.⁵

The commercial William Ransom & Son tincture later studied at Oxford was prepared from leaves and flowers of Pakistani-origin Cannabis sativa using cold ethanol extraction, solvent evaporation, and redissolution of the resinous residue in fresh ethanol. Analysis of that residue identified 6.4% by weight of Δ^1 -THC (the older numbering convention for modern Δ^9 -THC), 3.4% of its n-propyl homologue (now Δ^9 -THCV) and 3.6% cannabidiol (CBD). The medicine was therefore a mixed-cannabinoid preparation containing THC, THCV and CBD, rather than a single-cannabinoid product.⁶

B.P.C. 1949 / analysed material	Specification / finding
Extractum Cannabis	1,000 g coarsely powdered cannabis; 90% alcohol q.s.; percolate and evaporate to a soft extract.
Tinctura Cannabis	50 g cannabis extract; 90% alcohol to 1,000 mL; final alcohol 83–87% v/v; stated dose 0.3–1 mL.
Commercial tincture resin analysed at Oxford	6.4% Δ^1 -THC (modern Δ^9 -THC), 3.4% n-propyl Δ^1 -THC homologue (modern Δ^9 -THCV), and 3.6% CBD by weight of the resinous residue.

³ House of Lords Select Committee on Science and Technology, Cannabis: The Scientific and Medical Evidence, Ninth Report (1998), paras 2.7–2.10. The report records that the tincture received a licence of right under the Medicines Act 1968 and that the licence was not renewed in 1973.

⁴ Pertwee RG. Cannabinoid pharmacology: the first 66 years. Br J Pharmacol. 2006;147(S1):S163–S171. The review states that the commercial Tincture of Cannabis used by Gill et al. was prepared from Cannabis sativa grown in Pakistan and imported into Britain under licence.

⁵ British Pharmaceutical Codex 1949. London: Pharmaceutical Press. Extractum Cannabis and Tinctura Cannabis monographs: extract prepared from 1,000 g cannabis with 90% alcohol; tincture prepared from 50 g extract to 1,000 mL with 90% alcohol; final alcohol 83–87% v/v; stated tincture dose 0.3–1 mL.

⁶ Paton WDM, Pertwee RG. Effect of cannabis and certain of its constituents on pentobarbitone sleeping time and phenazone metabolism. Br J Pharmacol. 1972;44(2):250–261. doi:10.1111/j.1476-5381.1972.tb07261.x. The commercial tincture resin was reported to contain 6.4% Δ^1 -THC, 3.4% of the n-propyl Δ^1 -THC homologue and 3.6% CBD by weight.

Table 1. British Pharmaceutical Codex 1949 cannabis formulation and reported cannabinoid composition of the commercial tincture residue.

In 1970 Gill, Paton and Pertwee reported a tetrahydrocannabinol homologue containing a three-carbon side chain in this Tincture of Cannabis. Merkus subsequently identified the compound as tetrahydrocannabivarin (THCV). The historical product can therefore reasonably be described as THCV-bearing and evidently rich enough in the varin series for THCV to be isolated using the analytical techniques then available. Historical sources do not, however, provide a modern quantitative THCV assay, so a precise percentage should not be inferred.⁷

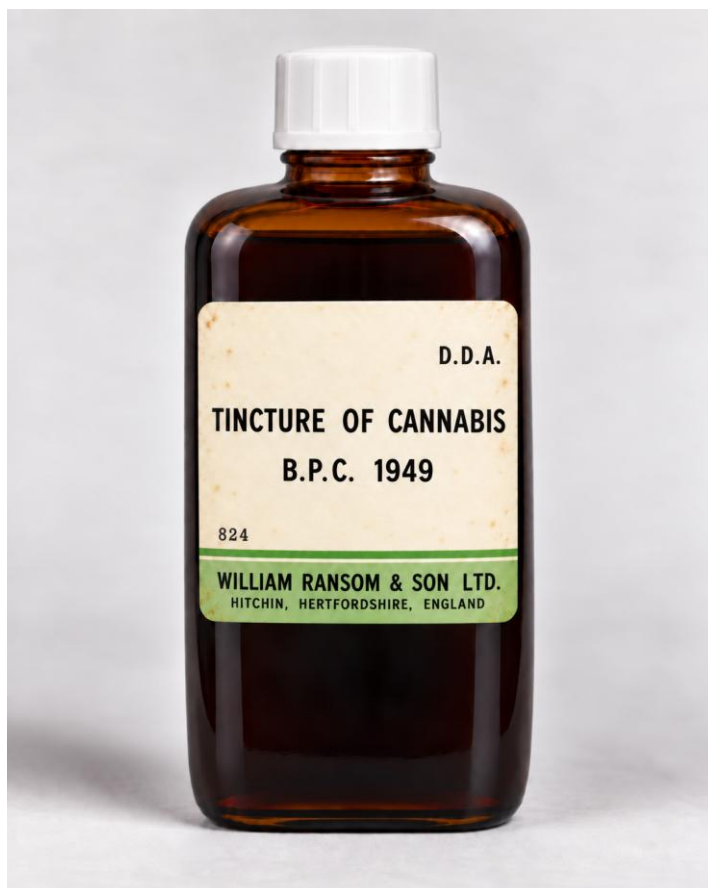


Figure 1. Artist's recreation of a William Ransom & Son Ltd Tincture of Cannabis B.P.C. 1949 bottle. The image is a modern reconstruction for illustrative purposes.

⁷ Gill EW, Paton WDM, Pertwee RG. Preliminary experiments on the chemistry and pharmacology of cannabis. *Nature*. 1970;228:134–136. doi:10.1038/228134a0; Merkus FWHM. Cannabivarin and tetrahydrocannabivarin, two new constituents of hashish. *Nature*. 1971;232:579–580. doi:10.1038/232579a0.

2. Δ^9 -THCV Chemistry

Δ^9 -THCV and Δ^9 -THC share the same tricyclic cannabinoid scaffold, but differ in the length of the alkyl side chain attached to the aromatic ring. Δ^9 -THC is a C5 cannabinoid with a five-carbon pentyl side chain; Δ^9 -THCV is the corresponding C3 varin homologue with a three-carbon propyl side chain. The molecular formulae are $C_{21}H_{30}O_2$ for Δ^9 -THC and $C_{19}H_{26}O_2$ for Δ^9 -THCV.

The “varin” suffix is sometimes assumed to refer to a cannabis variety. It does not. In cannabinoid nomenclature, varin identifies the propyl (C3) homologues and derives from divarinol, the C3 resorcinol associated with this chemical series. In the plant, the corresponding biosynthetic branch begins with divarinolic acid rather than the olivetolic acid used by the conventional pentyl cannabinoids. The name therefore points directly to biosynthetic origin — the subject of the next chapter.⁸

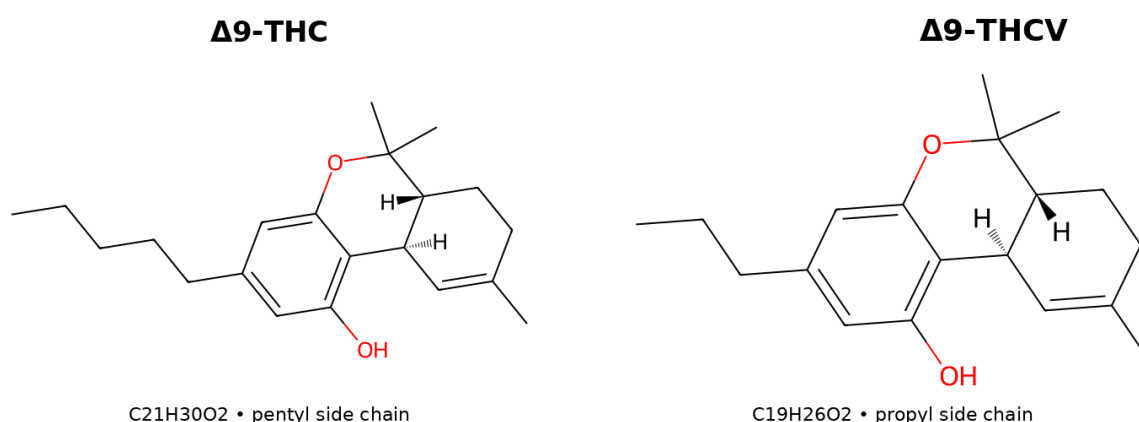


Figure 2. Structural comparison of Δ^9 -THC and Δ^9 -THCV. Δ^9 -THCV retains the cannabinoid core but has a three-carbon propyl side chain instead of the five-carbon pentyl side chain of Δ^9 -THC.

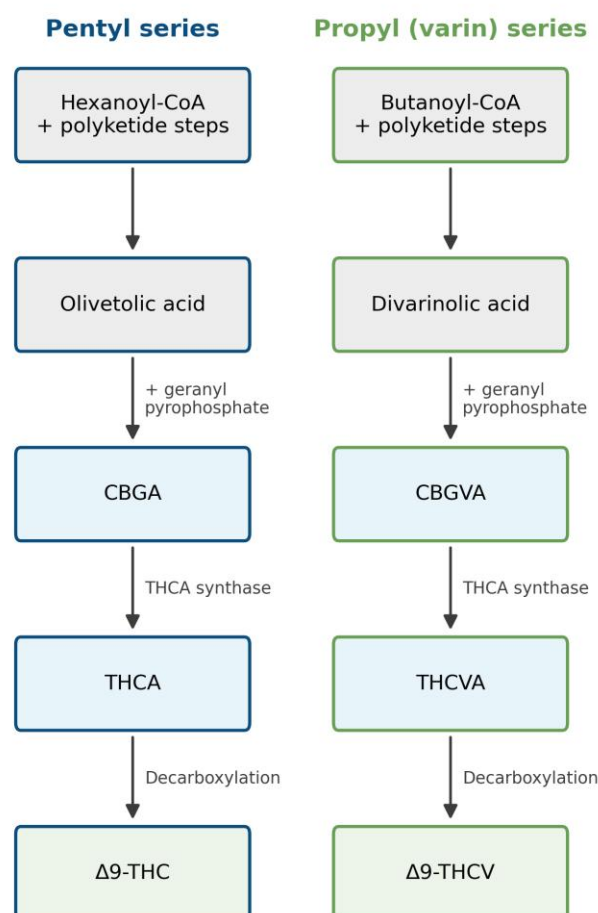
⁸Cerrato A, Cannazza G, Citti C, Laganà A, Paris R, Capriotti AL. Kendrick Mass Defect Filtering Enables High-Throughput Untargeted Annotation of Minor Phytocannabinoids: Toward Streamlined Phytocannabinomics. ACS Meas Sci Au. 2025;5(6):912–922. doi:10.1021/acsmesuresciau.5c00106. Propyl homologues are termed varinoids because the m-propyl resorcinol is divarinol.

3. Biosynthetic Pathway

Δ^9 -THCV is not a degradation product of Δ^9 -THC and cannot be created simply by processing an ordinary THC-dominant cultivar. The divergence occurs upstream in the plant. Conventional C5 cannabinoids originate from olivetolic acid, whereas C3 varin cannabinoids originate from divarinolic acid. Divarinolic acid is prenylated to form cannabigerovarinic acid (CBGVA), which is converted to tetrahydrocannabivarinic acid (THCVA) and subsequently decarboxylated to Δ^9 -THCV.

The practical implication is genetic: a plant must contain and express the varin pathway if it is to produce Δ^9 -THCV at meaningful concentrations. THC-rich flower is therefore a distinct chemotype rather than a conventional high-THC product with a different post-harvest treatment.

Simplified biosynthetic routes to Δ^9 -THC and Δ^9 -THCV



The “varin” suffix identifies the propyl-series cannabinoid family; it derives from divarinol/divarinolic acid, not from “variety”. The different side-chain precursor is established before CBGA/CBGVA formation.

Key distinction: Δ^9 -THCV is not produced by conversion or degradation of Δ^9 -THC.

Figure 3. Simplified biosynthetic comparison. Δ^9 -THCV arises through the divarinolic acid $\rightarrow$ CBGVA $\rightarrow$ THCVA branch rather than through the olivetolic acid $\rightarrow$ CBGA $\rightarrow$ THCA pathway that produces Δ^9 -THC.

4. Believed Mode of Action

THCV interacts with the endocannabinoid system, but its CB1 pharmacology differs from that of Δ^9 -THC. The literature most commonly describes THCV as a CB1 neutral antagonist at lower concentrations: it can occupy the receptor and oppose agonist signalling without necessarily reducing constitutive receptor activity. At higher concentrations, some experimental systems show weak partial agonist behaviour.⁹

THCV also interacts with CB2 and has been studied at other molecular targets. The overall pharmacology is therefore context- and concentration-dependent. This makes simplified descriptions such as “non-intoxicating THC” or “an appetite-blocking cannabinoid” scientifically inadequate.

Human evidence remains preliminary

A randomised, placebo-controlled 13-week pilot study in people with type 2 diabetes investigated purified THCV and CBD across glycaemic and lipid endpoints. THCV was well tolerated and, compared with placebo, was associated with a reduction in fasting plasma glucose and improvements in HOMA2 measures of pancreatic β -cell function. The study was small and was not designed to establish broad therapeutic efficacy.¹⁰

A separate 90-day placebo-controlled study of 44 participants evaluated mucoadhesive strips containing combined THCV and CBD at two dose levels. It reported changes in weight and metabolic markers, but because the intervention combined two cannabinoids and involved a small sample, it cannot establish that any observed effect was caused by THCV alone.¹¹

Controlled human studies have not demonstrated these effects with Δ^9 -THC; acute studies of Δ^9 -THC have instead reported either no material improvement in glucose regulation or impaired glucose tolerance¹².

⁹ Pertwee RG. The diverse CB1 and CB2 receptor pharmacology of three plant cannabinoids: Δ^9 -THC, cannabidiol and Δ^9 -THCV. *Br J Pharmacol*. 2008;153:199–215. doi:10.1038/sj.bjp.0707442; McPartland JM et al. *Br J Pharmacol*. 2015;172:737–753. doi:10.1111/bph.12944.

¹⁰ Jadoon KA et al. Efficacy and Safety of Cannabidiol and Tetrahydrocannabivarin on Glycemic and Lipid Parameters in Patients With Type 2 Diabetes. *Diabetes Care*. 2016;39(10):1777–1786. doi:10.2337/dc16-0650.

¹¹ Smith GL. Weight Loss and Therapeutic Metabolic Effects of Tetrahydrocannabivarin (THCV)-Infused Mucoadhesive Strips. *Cannabis*. 2025;8(1):109–120. doi:10.26828/cannabis/2024/000206. The intervention combined THCV and CBD.

¹² Hollister LE, Reaven GM. “Delta-9-tetrahydrocannabinol and glucose tolerance.” *Clinical Pharmacology & Therapeutics*. 1974;16(2):297–302. DOI: 10.1002/cpt1974162297.

5. Why THCV is not Rimonabant

Rimonabant provides an important historical caution when discussing CB1 modulation. It was developed as a synthetic CB1 inverse agonist for weight management. An inverse agonist does more than block an agonist: it reduces the receptor's constitutive background signalling. The European Medicines Agency suspended Acomplia (rimonabant) in 2008 after concluding that its benefits no longer outweighed psychiatric risks, including depression, anxiety and reported suicides.¹³

THCV is generally characterised differently. At lower concentrations, it is more commonly described as a neutral antagonist at CB1; neutral antagonism blocks agonist action without the same theoretical reduction in basal receptor activity. At higher concentrations, THCV may show partial agonism. These distinctions mean that THCV should not simply be described as a botanical version of rimonabant.¹⁴

The mechanistic distinction is important but should not be converted into a safety or efficacy claim. It does not prove that THCV will avoid every risk associated with CB1 modulation, nor does it prove effectiveness for weight management. Those questions require adequately powered and appropriately controlled clinical studies.

¹³ European Medicines Agency. Acomplia (rimonabant). EPAR. Marketing suspended in EU Member States in November 2008; marketing authorisation withdrawn in January 2009 following psychiatric safety concerns.

¹⁴ McPartland JM et al. Br J Pharmacol. 2015;172:737–753; Pertwee RG. Br J Pharmacol. 2008;153:199–215. These sources discuss THCV antagonism/neutral antagonism and concentration-dependent agonist behaviour.

6. THCV Harmony®

Defined flower chemotype

THCV Harmony® is being developed as dried flower with a target cannabinoid profile of 10% THC and 7% THCV. This is unusual because most commercial cultivars contain only trace THCV. The product concept is therefore based on making THCV a major, repeatable component of the flower rather than an incidental minor constituent.

Attribute	THCV Harmony®	Development relevance
Format	Dried flower	Preserves a whole-flower cannabinoid and terpene matrix
Target THC	10%	Moderate THC background rather than a high-THC-only proposition
Target THCV	7%	Makes THCV a major constituent rather than a trace cannabinoid
THC:THCV relationship	Approximately 1.4:1 by stated percentages	Distinctive chemotype suitable for comparative research
Cultivation strategy	Clonal genetics and controlled environment	Supports batch-to-batch reproducibility

Table 2. Proposed THCV Harmony® flower profile and its relevance to product development.

Why it may be a promising therapeutic development option

The scientific case for THCV Harmony® is not that therapeutic benefit has already been proven. The opportunity is that a stable flower containing 7% THCV alongside 10% THC provides a defined and differentiated material for research. It enables investigators to study a THCV-rich whole-flower matrix rather than extrapolating from isolated THCV or from conventional cannabis containing only trace THCV.

For Glass Pharms®, the development strategy also aligns with cannabinoid precision. Tissue-culture clones provide a common genetic starting point, while controlled cultivation and analytical testing can be used to reduce batch-to-batch variability. That combination may make THCV Harmony® useful for future translational and clinical work where chemical repeatability is essential.¹⁵

Potential areas of scientific interest include metabolic endpoints, appetite and reward signalling, and the interaction between THCV and THC. These are research directions rather than product claims. Any indication-specific positioning would require evidence appropriate to the proposed use and regulatory pathway.

¹⁵ Glass Pharms. The Science of THCV, internal briefing draft, 2026. The briefing describes the use of selected tissue-culture clones, controlled growing conditions and analytical testing to support repeatable cannabinoid profiles.

7. Conclusion

THCV is both historically established and scientifically underexplored. A THCV-bearing cannabis tincture remained part of the final era of prescription cannabis in Britain before prohibition took full effect in 1973. Modern cannabinoid chemistry has since shown that THCV is not merely a variant produced from THC, but the product of a distinct C3 varin biosynthetic pathway.

Its receptor pharmacology also differs materially from Δ^9 -THC and from the inverse-agonist profile of rimonabant. Human research is still limited, but the emerging evidence is sufficient to justify careful investigation without overstating what is known.

THCV Harmony[®] is intended to bring that investigation into a reproducible flower format: 10% THC and 7% THCV, produced from controlled genetics. Its promise lies in the precision of the chemotype and the quality of the questions it allows clinicians to explore, rather than in claims that have yet to be established in formal randomised control trials.

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8. References

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