Sources Cited in the CB1 & Hair Loss Deep Dive

Organized in order of appearance in the article

Part I: What CB1 Does in the Hair Follicle

[1] Telek et al. (2007) — The foundational CB1 hair follicle study Telek A, Bíró T, Bodó E, Tóth BI, Borbíró I, Kunos G, Paus R. "Inhibition of human hair follicle growth by endo- and exocannabinoids." *The FASEB Journal*, 21(13):3534–3541. https://pubmed.ncbi.nlm.nih.gov/17567570/

The primary study demonstrating that CB1 is expressed in a hair cycle-dependent manner in human scalp follicle epithelium, and that CB1 activation inhibits hair shaft elongation, promotes keratinocyte apoptosis, and induces premature catagen. Effects were blocked by a selective CB1 antagonist.

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[2] Sugawara et al. (2021) — CB1 and human follicle stem cell survival Sugawara K, Zákány N, Tiede S, Purba T, Harries M, Tsuruta D, Bíró T, Paus R. "Human epithelial stem cell survival within their niche requires 'tonic' cannabinoid receptor 1-signalling — lessons from the hair follicle." *PubMed*, 2021. https://pubmed.ncbi.nlm.nih.gov/33523535/

Shows that CB1 promotes apoptosis in differentiated follicle keratinocytes (CK6+) while maintaining stem cells — relevant to miniaturization mechanism.

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[3] Smith & Satino (2022) — Pre-clinical and clinical summary of cannabinoid hair follicle effects Smith GL, Satino J. "A Summary of Pre-Clinical and Clinical Evidence for Cannabinoid Hair Follicle Effects." *Clinical Dermatology Open Access Journal*, 7(4):000290. Medwin Publishers. https://medwinpublishers.com/CDOAJ/a-summary-of-pre-clinical-and-clinical-evidence-for-cannabinoid-hair-follicle-effects.pdf

Summarizes the evidence for THCV, CBDV, and CBD acting as CB1 antagonists for hair growth; describes the TRPV4 overdose paradox.

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[4] Bíró et al. (2009) — The endocannabinoid system of the skin Bíró T, Tóth BI, Haskó

G, Paus R, Pacher P. “The endocannabinoid system of the skin in health and disease: novel perspectives and therapeutic opportunities.” *Trends in Pharmacological Sciences*, 30(8):411–420. https://pubmed.ncbi.nlm.nih.gov/19608284/

Comprehensive review of ECS in skin and appendages, including hair follicles; explains CB1 regulation of follicle cycling.

Part II: Sex Differences in CB1 Activity

[5] Hill et al. (2009) — Estrogenic regulation of limbic cannabinoid receptor binding Hill MN, Karacabeyli ES, Gorzalka BB. “Oestrogen recruits the endocannabinoid system to modulate emotionality.” *Neuropharmacology*, PMC2933663. https://pmc.ncbi.nlm.nih.gov/articles/PMC2933663/

Demonstrates that ovariectomy upregulates CB1 receptor binding site density in the hippocampus, and that estradiol replacement reverses this — confirming estrogen’s inhibitory effect on CB1 density. The mechanistic basis for sex differences in CB1 expression.

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[6] Hill & Gorzalka (2007) — Estrogen recruits the endocannabinoid system to modulate emotionality Hill MN, Gorzalka BB. “Estrogen recruits the endocannabinoid system to modulate emotionality.” *Neuropharmacology*, 2007. https://pubmed.ncbi.nlm.nih.gov/17391861/ https://www.sciencedirect.com/science/article/abs/pii/S0306453007000303

Shows estrogen’s anxiolytic and antidepressant effects are mediated via the endocannabinoid system through FAAH inhibition and resulting anandamide elevation.

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[7] Waleh et al. (2002) / Hill et al. (2007) — FAAH estrogen response element Referenced via: Hill MN, Gorzalka BB (2007) citing Waleh et al. (2002). See also: Spandidos Publications review — “The Complex Interplay between Endocannabinoid System and the Estrogen System.” https://www.spandidos-publications.com/10.3892/ijmm.2016.2779

Documents that the FAAH gene contains an estrogen response element in its promoter sequence, and that estrogen receptor translocation to the nucleus inhibits FAAH transcription — the mechanistic basis for estrogen-mediated anandamide elevation.

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[8] Maccarrone et al. (2003) — Progesterone activates FAAH promoter Maccarrone M et al. "Progesterone Activates Fatty Acid Amide Hydrolase (FAAH) Promoter in Human T Lymphocytes through the Transcription Factor Ikaros." *Journal of Biological Chemistry*, 2003. https://www.sciencedirect.com/science/article/pii/S0021925820838277

Shows progesterone stimulates FAAH activity via an Ikaros binding site, increasing anandamide degradation — explaining the luteal phase decrease in endocannabinoid tone.

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[9] Vallée et al. (2014) — Pregnenolone as CB1 signaling-specific inhibitor Vallée M et al. "Pregnenolone can protect the brain from cannabis intoxication." *Science*, 2014. Referenced in: "The Complex Interplay between Endocannabinoid System and the Estrogen System." *PMC*. https://pmc.ncbi.nlm.nih.gov/articles/PMC7835826/

Demonstrates that pregnenolone acts as a signaling-specific inhibitor of the CB1 receptor, reducing THC toxicity — relevant to endogenous female hormonal protection via CB1 buffering.

Part III: The Population Hierarchy

[10] Cranwell & Sinclair (2016) — Male Androgenetic Alopecia (Endotext/NCBI) Cranwell W, Sinclair R. "Male Androgenetic Alopecia." South Dartmouth (MA): MDText.com, Inc.; 2016. https://www.ncbi.nlm.nih.gov/books/NBK278957/

Authoritative review documenting that AGA is more common in Caucasians than other nationalities; that Japanese men experience AGA onset a decade later than Caucasians; and that Black, Oriental, and Native American men are more likely to preserve their frontal hairlines.

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[11] Ranasinghe et al. / Dermatology Journal (2022) — Racial disparities in AGA clinical trials "Racial and Ethnic Disparities in Androgenetic Alopecia Clinical Trials in the United States." *Dermatology Journal*, 2022. https://www.dermatoljournal.com/articles/racial-and-ethnic-disparities-in-androgenetic-alopecia-clinical-trials-in-the-united-states.html

Documents the AGA prevalence hierarchy: Caucasian highest, followed by African Americans and Asians, with Native Americans and Alaska Natives least commonly affected.

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[12] Characteristics of AGA in Asian populations — PMC Review "Characteristics of

Androgenetic Alopecia in Asian." *PMC*, PMC3412231. https://pmc.ncbi.nlm.nih.gov/articles/PMC3412231/

Reviews the distinct characteristics of AGA in Asian men vs. European descent, including lower prevalence rates and later onset.

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[13] Feng et al. (2013) — CNR1 rs806371 promoter variant reduces gene expression Feng Q et al. "A common functional promoter variant links CNR1 gene expression to HDL cholesterol level." *Nature Communications*, 2013. https://www.nature.com/articles/ncomms2973 https://pmc.ncbi.nlm.nih.gov/articles/PMC3873874/

Identifies rs806371 in the CNR1 promoter as a functional variant that reduces CNR1 gene expression — the specific Caucasian-enriched variant that paradoxically may lower CB1 expression at baseline.

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[14] Mitjans et al. (2013) — CNR1 polymorphisms in European populations and depression Mitjans M et al. "Screening genetic variability at the CNR1 gene in both major depression etiology and clinical response to citalopram treatment." *Psychopharmacology*, 2013. https://link.springer.com/article/10.1007/s00213-013-2995-y

Documents CNR1 haplotype structure in European populations, including the rs806371 and TAG haplotype variants.

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[15] Frontiers in Genetics (2018) — CNR1 variants in African-American population "Detection of Significant Association Between Variants in Cannabinoid Receptor 1 Gene (CNR1) and Personality in African-American Population." *Frontiers in Genetics*, 2018. https://www.frontiersin.org/journals/genetics/articles/10.3389/fgene.2018.00199/full

Documents CNR1 SNP frequencies in African-American populations, including rs9444584 and related variants.

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[16] Genetic Variants and AGA — PMC (2024) "Genetic Variants and Lifestyle Factors in Androgenetic Alopecia Patients: A Case–Control Study." *PMC*, PMC11767835. https://pmc.ncbi.nlm.nih.gov/articles/PMC11767835/

Documents genetic architecture of AGA susceptibility, including the complex multi-gene contribution to phenotype.

[17] Hirvonen et al. (2012) — CB1 receptor downregulation and recovery after cannabis cessation Hirvonen J et al. "Reversible and regionally selective downregulation of brain cannabinoid CB1 receptors in chronic daily cannabis smokers." *Molecular Psychiatry*, 17:642–649. https://pubmed.ncbi.nlm.nih.gov/21747398/ https://pmc.ncbi.nlm.nih.gov/articles/PMC3223558/ https://www.nature.com/articles/mp201182

The primary PET neuroimaging study demonstrating ~20% CB1 receptor downregulation in chronic daily cannabis smokers, with full recovery to normal levels after ~4 weeks of monitored abstinence. Downregulation correlated with years of cannabis smoking.

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[18] D'Souza et al. (2016) — Rapid changes in CB1 receptor availability after cannabis cessation D'Souza DC et al. "Rapid Changes in CB1 Receptor Availability in Cannabis Dependent Males after Abstinence from Cannabis." *PMC*, PMC4742341. https://pmc.ncbi.nlm.nih.gov/articles/PMC4742341/

Documents that CB1 receptor downregulation begins to reverse rapidly upon cessation; provides the timeline for regional recovery (subcortical faster than cortical).

Part IV: The Clinical Evidence — CB1 Antagonists Work

[19] Smith & Satino (2021) — Hair Regrowth with CBD-rich Hemp Extract (original CBD-only study) Smith GL, Satino J. "Hair Regrowth with Cannabidiol (CBD)-rich Hemp Extract – A Case Series." *Cannabis*, 2021. PMC10212262. https://pubmed.ncbi.nlm.nih.gov/37287996/ https://ncbi.nlm.nih.gov/pmc/articles/PMC10212262

The original 6-month CBD-only study showing average 93.5% increase in hair count; showed men responded better than women.

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[20] Smith & Satino (2023) — Hair Regrowth with Novel Hemp Extract (THCV+CBDV+CBD study) Smith GL, Satino J. "Hair Regrowth with Novel Hemp Extract: A Case Series." *PubMed*, 2023. PMC10251293. https://pubmed.ncbi.nlm.nih.gov/37305187/ https://ncbi.nlm.nih.gov/pmc/articles/PMC10251293 https://tricomax.com/pages/hair-regrowth-with-novel-hemp-extract-a-case-series

The follow-up study using THCV+CBDV+CBD (ALPHA VARIN complex) showing average

246% increase in men and 127% in women over 6 months, with all subjects showing some regrowth.

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[21] ALPHA VARIN Complex Trial Summary — TricoMax/BEVARIN "ALPHA VARIN COMPLEX Trial Summary for Hair Growth." NIH-Identified Phase 1 Clinical Trial. *Tricomax.com*. https://tricomax.com/pages/alpha-varin-complex-trial-summary-for-hair-growth ClinicalTrials.gov: NCT04842383 https://clinicaltrials.gov/study/NCT04842383

The 40-person NIH-identified Phase 1 trial showing average 371% increase in new non-vellus hair count over 180 days using the ALPHA VARIN complex.

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[22] CenterWatch — Clinical trial listing NCT04842383 "Androgenetic Alopecia Treatment Using Varin and Cannabidiol Rich Topical Hemp Oil: A Case Series." https://www.centerwatch.com/clinical-trials/listings/NCT04842383/androgenetic-alopecia-treatment-using-varin-and-cannabidiol-rich-topical-hemp-oil-a-case-series

Official clinical trial registration details.

Product Links (Part V)

[23] TricoMax Hair Maximizer — ALPHA VARIN Complex
https://tricomax.com/products/tricomax%E2%84%A2-hair-maximizer-non-tinted *(Also available on Amazon)*

[24] Spectral.CBD by DS Laboratories https://dslaboratories.com/products/spectral-cbd-innovative-hair-loss-treatment-with-nanoxidil-5-cbd

[25] Dakota Hemp Nano CBD+CBG Cream 7500mg
https://dakotahempcbd.com/product/nano-cbd-relief-cream-2oz-7500mg/

Additional Supporting Sources Referenced in Conversation

[26] Tóth et al. (2019) — Cannabinoid signaling in the skin Tóth KF, Ádám D, Bíró T, Oláh A. "Cannabinoid signaling in the skin: therapeutic potential of the 'C(ut)annabinoid' system." *Molecules*, 24(5):918. https://pubs.acs.org/doi/10.1021/cn5000919

Comprehensive review of ECS in skin and hair follicle biology including CB1 and CB2 receptor roles.

[27] Endotext — Male AGA (Androgen Receptor / Native American data) Cranwell W, Sinclair R. *Endotext*, NBK278957. https://www.ncbi.nlm.nih.gov/books/NBK278957/

Documents that Native Americans and Eskimos are least commonly affected by AGA; that Black, Oriental, and Native American men are more likely to have preservation of frontal hairlines and late onset.

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[28] Laprairie et al. (2015) — CBD as negative allosteric modulator of CB1 Laprairie RB, Bagher AM, Kelly ME, Denovan-Wright EM. "Cannabidiol is a negative allosteric modulator of the cannabinoid CB1 receptor." *British Journal of Pharmacology*, 2015. Referenced in Smith & Satino (2021/2022).

Establishes the pharmacological mechanism of CBD as a negative allosteric modulator (partial antagonist) of CB1 — distinct from THCV and CBDV which are full antagonists.

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[29] Rimonabant withdrawal — EMA and clinical trial data Referenced across multiple sources. Rimonabant (Acomplia) was withdrawn from European markets in 2008 by the EMA after Phase 3 trials with 9,000+ patients demonstrated doubling of psychiatric disorder risk and 2.5× increased rate of discontinuation due to depressive mood disorders. Review: https://pubmed.ncbi.nlm.nih.gov/18393062/ (Le Foll et al., 2009)

Note: This reference list covers sources directly cited or discussed in the article and the broader conversation. For a complete academic bibliography, individual claims should be traced to their primary source using the PubMed and PMC links provided above. All PubMed and PMC links are freely accessible.

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