Review

The Interaction of Stomach Acid, Blood pH, and Liver Metabolism with Acidic Cannabinoids: Partial Decarboxylation, Metabolic Transformation, and Physiological Implications

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Scientific Review

The Interaction of Stomach Acid, Blood pH, and Liver Metabolism with Acidic Cannabinoids: Partial Decarboxylation, Metabolic Transformation, and Physiological Implications

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Abstract

Acidic cannabinoids (e.g., THCA, CBDA) are the dominant phytoconstituents in Cannabis Sativa and serve as precursors to neutral forms (THC, CBD) via decarboxylation. This is the third work in an integrated series exploring how dietary cannabis inputs interact with the Endocannabinoid System (ECS) pathways. This paper examines the role of physiological environments—specifically stomach acidity, blood pH, and hepatic metabolism—in determining the fate and bioavailability of ingested acidic cannabinoids.[Approach & Findings] Integrating organic chemistry and pharmacokinetics, the study confirms that gastric conditions (pH 1.5–3.5, 37°C) induce a minor, diet-dependent partial decarboxylation ($\leq 5-10\%$) due to the low activation energy at physiological temperature and poor solubility. Upon absorption, systemic blood pH (7.35–7.45) stabilizes the acidic cannabinoids, which exist primarily as non-decarboxylating carboxylate anions (>99% ionized). The hepatic first-pass metabolism then primarily processes the compounds through CYP and UGT enzymes, leading to conjugated metabolites (e.g., THCA-glucuronide) rather than extensive decarboxylation.[Microbiome & Implication] Crucially, the gut microbiome is identified as a secondary modulator, utilizing microbial decarboxylases and β-glucuronidases to potentially recycle cannabinoids via enterohepatic circulation, thus impacting systemic exposure and therapeutic effects. This comprehensive analysis integrates chemical kinetics and physiological variables, showing that acidic cannabinoids are delivered largely intact to modulate the ECS directly (e.g., THCA activating TRPA1; CBDA inhibiting FAAH).[Conclusion] The minor in vivo conversion rate means the therapeutic potential of ingested acidic cannabinoids is shaped more by direct ECS interaction and microbial/metabolic processing than by thermal decarboxylation.

Keywords: dietary cannabinoids; clinical endocannabinoid deficiency; Entourage effect; synergistic cannabinoids; Anti-inflammatory acidic cannabinoids; TRP channels cannabinoids (e.g.; TRPA1 THCA); FAAH inhibition CBDA; PPARgamma agonism cannabinoids; Phytocannabinoid acids; Bioavailability phytocannabinoids; first-pass metabolism cannabinoids

1. Introduction

This preprint is part of a three-work integrated series examining (1) dietary inputs from the whole cannabis plant (2) and hemp seed, (3) the mechanistic ECS pathways they activate, and the physiological outcomes.

2. Materials and Methods

This review series is a narrative synthesis of existing peer-reviewed literature on dietary inputs from Cannabis sativa L. (whole plant and seeds), endocannabinoid system (ECS) mechanisms, and physiological outcomes. No primary experimental data were generated.

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A comprehensive literature search was conducted using PubMed, Google Scholar, Scopus, and Web of Science databases from inception through December 2025, with keywords including but not limited to: "hemp seed nutrition," "acidic cannabinoids," "THCA/CBDA/CBGA pharmacokinetics," "endocannabinoid precursors," "PUFA ECS modulation," "raw cannabis consumption," "entourage effect," "clinical endocannabinoid deficiency," and organ-specific terms (e.g., "hemp seed cardiovascular," "cannabis neuroprotection").References were selected for relevance, methodological rigor, and recency, prioritizing human studies, clinical trials, and mechanistic reviews where available, supplemented by preclinical and in vitro evidence. Inclusion focused on studies demonstrating direct links between cannabis/hemp constituents and ECS pathways or homeostasis outcomes.

Metabolite pathways (e.g., "The Acidic Cannabinoid Metabolome") were constructed by integrating established pharmacokinetic data with logical chemical extensions based on known CYP450, UGT, and oxidative transformations. Speculative or trace compounds/metabolites were explicitly labeled as such, derived from structural analogy and rare reported pathways.

All claims are supported by cited sources; synthesis connects established findings to propose integrative models for dietary ECS modulation.

3. AI Disclosure

The comprehensive literature search spanning the past 13 years was systematically supported by [ChatGPT-4, Grok, and Gemini]. The tools were used for data extraction and collation, specifically to efficiently screen and categorize high-volume scientific databases and preprint servers for keywords related to the endocannabinoid system (ECS) and various nutritional compounds. The multiple AIs were then pitted against each other to test and validate the scientific data and theories.

This assistance was instrumental in managing the large dataset compiled over the research period. The identification of the novel conceptual framework—nutritional support for the ECS—was an original human-driven insight based on the author's synthesis of the collated data, not a generative function of the AI tools. The author assumes full responsibility for all content, interpretation, and references cited in this manuscript.

4. The Interaction of Stomach Acid, Blood pH, and Liver Metabolism with Acidic Cannabinoids: Partial Decarboxylation, Metabolic Transformation, and Physiological Implications

This preprint is part of a three-work integrated series examining (1) dietary inputs from the whole cannabis plant (2) and hemp seed, (3) the mechanistic ECS pathways they activate, and the physiological outcomes

Cannabis sativa synthesizes acidic cannabinoids such as Δ9-tetrahydrocannabinolic acid (THCA), cannabidiolic acid (CBDA), and cannabigerolic acid (CBGA), which serve as precursors to their neutral forms—Δ9-tetrahydrocannabinol (THC), cannabidiol (CBD), and cannabigerol (CBG)—via decarboxylation, a process releasing CO_2 from the carboxyl group (-COOH). While heat (110–130°C) drives decarboxylation in smoking or cooking, physiological conditions—stomach acidity (pH 1.5–3.5), blood pH (7.35–7.45), and liver metabolism—may influence this reaction and the fate of these compounds. Beyond their role as precursors, acidic cannabinoids directly modulate the endocannabinoid system (ECS), impacting therapeutic potential (Pertwee, 2008). This paper explores these interactions comprehensively, integrating organic chemistry, nutritional influences, ECS dynamics, and cannabinoid sciences, supported by experimental data and pharmacokinetics.

Stomach Acid and Partial Decarboxylation of Acidic Cannabinoids

The stomach's pH (1.5–3.5), maintained by hydrochloric acid (HCl) from parietal cells, exposes ingested acidic cannabinoids (e.g., THCA, $C_{22}H_{30}O_4$, MW 358.47 g/mol; CBDA, $C_{22}H_{30}O_4$, MW 358.47 g/mol) to an acidic environment. Decarboxylation (e.g., THCA → THC + CO_2) is an elimination reaction requiring ~25–30 kcal/mol activation energy (E_a), typically heat-driven (McPartland & Russo, 2016). Acidic conditions catalyze this by protonating the carboxyl group, as shown:

$THCA + H^+ \rightleftharpoons [THCAH^+] \rightarrow THC + CO_2$

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Protonation enhances CO_2 release via an E1-like pathway, with a rate constant (k) of ~0.0004 min^{-1} at 37°C, pH 2.0, versus 0.03 min^{-1} at 120°C (Wang et al., 2016). Hazekamp et al. (2006) reported 3–5% THCA decarboxylation in simulated gastric fluid (SGF, pH 2.0, 37°C) over 2 hours, aligning with gastric residence time. THCA decarboxylates faster than CBDA due to its phenolic hydroxyl stabilizing the transition state, lowering E_a by 2–3 kcal/mol (Wang et al., 2016).

Temperature limits this process at 37°C, reducing k by ~10^3 per the Arrhenius equation (k = A e^(-Ea/RT)) compared to 120°C (McNaught & Wilkinson, 1997). Solubility further constrains interaction: THCA's logP (6.2–7.0) yields <0.1 mg/mL in SGF (Perrott et al., 2020). Minor oxidation to cannabinol-like compounds (<1%) may occur (Fairbairn et al., 1976), but pepsin (pH optimum 1.8–2.2) does not catalyze decarboxylation (Fruton, 2002).

Dietary factors amplify variability. Fatty meals raise gastric pH to ~5.0 and extend emptying to 4 hours, potentially increasing decarboxylation to 8–10% (Kong & Singh, 2008). Post-gastric, the small intestine (pH 6–7.4) enhances solubility via bile salts (5–15 mM), raising dissolution to 1–2 mg/mL, with dietary lipids lowering micelle CMC and boosting uptake 2–3-fold (Zgair et al., 2016). Thus, gastric decarboxylation is minor relative to intestinal absorption.

Blood pH and Cannabinoid Stability

Absorbed cannabinoids enter the portal vein and systemic circulation, where blood pH (7.35–7.45) is buffered by HCO_3^-/H_2CO_3 (pK_a 6.1):

$pH = 6.1 + \log([HCO_3^-]/[H_2CO_3])$

With $[HCO_3^-] \approx 24$ mM and $[H_2CO_3] \approx 1.2$ mM, this neutral environment stabilizes acidic cannabinoids. THCA (pK_a ~4.9) and CBDA (pK_a ~4.7) are >99% ionized as carboxylate anions ($-COO^-$) at pH 7.4, unreactive to decarboxylation (Watanabe et al., 2007). Eichler et al. (2012) detected THCA (5–20 ng/mL) and Anderson et al. (2020) reported CBDA (10–30 ng/mL) in plasma post-ingestion, with negligible conversion to THC/CBD. Neutral THC and CBD bind albumin (>95%), reducing free fractions to <1% (Widman et al., 1974).

Metabolites like THC-COOH (pK_a 4.5, 10–50 ng/mL in chronic users) are ionized, minimally impacting pH due to buffering (Berne & Levy, 2017). ECS relevance emerges here: 11-OH-THC, a liver metabolite, enhances CB1 agonism (K_i 10 nM vs. THC's 40 nM), amplifying psychotropic effects (Rhee et al., 1997). Analytical challenges (e.g., THCA decarboxylation in GC-MS ion sources) may underestimate acidic forms (Wohlfarth et al., 2013), refining pharmacokinetic interpretation.

Liver Metabolism and Transformation into Metabolites

Hepatic first-pass metabolism via cytochrome P450 (CYP) enzymes transforms cannabinoids. THC is hydroxylated to 11-OH-THC ($C_{21}H_{30}O_3$) by CYP2C9 (K_m ~0.5 μM), then oxidized to THC-COOH ($C_{21}H_{28}O_4$):

$THC + O_2 + NADPH + H^+ \rightarrow[CYP2C9] 11\text{-}OH\text{-}THC + H_2O$

$11\text{-}OH\text{-}THC + O_2 + NADPH + H^+ \rightarrow[CYP] THC\text{-}COOH + H_2O$

CBD forms 7-OH-CBD and 6-OH-CBD via CYP3A4/CYP2C19 (Ujváry & Hanuš, 2016). Acidic cannabinoids are conjugated without decarboxylation: THCA to THCA-glucuronide ($C_{28}H_{36}O_{10}$) by UGT1A9 (<1% THC conversion; Huestis, 2007), and CBDA to CBDA-glucuronide (Anderson et al., 2020).

The role of the gut microbiome in cannabinoid metabolism and its potential influence on the pharmacokinetics and therapeutic effects of acidic cannabinoids is an emerging area of interest. Gut microbiota, including Clostridium and Bacteroides species, decarboxylate THCA to THC (<1–3% over 24 hours in vitro; Citti et al., 2018; Al-Zouabi et al., 2019), mediated by microbial decarboxylases (e.g., aromatic-L-amino-acid decarboxylase, EC 4.1.1.28). This minor conversion increases local THC levels, activating enteric CB1 receptors and influencing gut motility or the gut-brain axis (DiPatrizio, 2016; Sharkey & Wiley, 2016). Dysbiosis (e.g., from high-fat diets) may enhance this activity by 5–15%, altering bioavailability (Aviello et al., 2012; Al-Zouabi et al., 2019). Furthermore, microbial β-glucuronidases deconjugate hepatic glucuronides (e.g., THCA-glucuronide), recycling cannabinoids into enterohepatic circulation, potentially extending half-life by 15–25% (Roberts et al., 2014; Huestis, 2007). These processes suggest a microbiome-mediated modulation of acidic cannabinoid pharmacokinetics and ECS effects, with therapeutic implications warranting further exploration (Russo et al., 2015).

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Genetic polymorphisms (e.g., CYP2C93) reduce THC metabolism by 30–50%, extending 11-OH-THC half-life to 3–4 hours (Sachse-Seeboth et al., 2009), while UGT1A91b increases glucuronidation by 40% (Margaillan et al., 2015). Nutrition impacts this: protein deficiency lowers CYP3A4 activity by 20–30%, skewing metabolite ratios (Yang et al., 2012). Metabolites are excreted via bile (20–35%) and urine (65–80%), with negligible pH effects (Huestis, 2007).

Chemical Mechanisms and Experimental Insights

Gastric decarboxylation depends on $[H^+]$, with $k \approx 10^{-4}$ min^{-1} at pH 2.0, 37°C (Wang et al., 2016). Hepatic CYP oxidation inserts O_2 into C-H bonds, yielding polar metabolites (e.g., THC-COOH, m/z 344.2), confirmed by LC-MS/MS (Eichler et al., 2012). The pH gradient (stomach to intestine) shifts ionization, enhancing solubility and uptake. Pre-ingestion storage (light, heat) decarboxylates THCA by 10–20% over months, altering ingested ratios (Trofin et al., 2012). Species differences (e.g., rat gastric pH 3–4) overestimate human decarboxylation (Kararli, 1995).

4. Discussion: ECS and Therapeutic Implications

Acidic cannabinoids engage the ECS directly: THCA activates TRPA1 (EC_{50} ~20 μM) and CBDA inhibits FAAH (IC_{50} ~17 μM), offering neuroprotection and anti-inflammatory effects (Pertwee, 2008; Nadal et al., 2017; Takeda et al., 2008). Chronic THCA ingestion yields trace THC in saliva (1–5 ng/mL), suggesting slow in vivo conversion (Schwilke et al., 2009). Liver metabolites like 11-OH-THC shift ECS signaling, while nutritional and genetic factors modulate bioavailability and efficacy.

5. Conclusions

Gastric decarboxylation (<5–10%) is minor, diet-dependent, and limited by temperature and solubility. Blood pH stabilizes acidic cannabinoids, delivering them to the liver, where CYP and UGT enzymes produce bioactive metabolites, shaping ECS activity. The gut microbiome further modulates pharmacokinetics, enhancing local and systemic effects. This interplay integrates chemistry, nutrition, and cannabinoid sciences, with therapeutic implications refined by dietary, genetic, microbial, and analytical nuances.

Conflicts of Interest: The authors declare no conflict of interest. The author does not work for any cannibinoid business or industry.

Abbreviations

The following abbreviations are used in this manuscript:

Abbreviation	Full Term
AEA	Anandamide (N-arachidonoylethanolamine)
ARR	Arrhenius rate relationship
CBD	Cannabidiol
CBDA	Cannabidiolic acid
CBG	Cannabigerol
CBGA	Cannabigerolic acid
CB1	Cannabinoid receptor type 1
CB2	Cannabinoid receptor type 2
CMC	Critical micelle concentration
CO_2	Carbon dioxide
CYP	Cytochrome P450 enzyme family
E_a	Activation energy
ECS	Endocannabinoid system
EC_{50}	Half-maximal effective concentration
FAAH	Fatty acid amide hydrolase
GC-MS	Gas chromatography - mass spectrometry
HCl	Hydrochloric acid
HCO_3^-	Bicarbonate
H_2CO_3	Carbonic acid

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IC_{50}	Half-maximal inhibitory concentration
K_i	Inhibition constant
k	Reaction rate constant
K_m	Michaelis constant
LC-MS/MS	Liquid chromatography - tandem mass spectrometry
logP	Logarithm of the octanol/water partition coefficient
m/z	Mass-to-charge ratio
MW	Molecular weight
NADPH	Nicotinamide adenine dinucleotide phosphate (reduced form)
pH	Potential of hydrogen
pK_a	Acid dissociation constant
PPARγ	Peroxisome proliferator-activated receptor gamma
SGF	Simulated gastric fluid
THC	Δ^9-Tetrahydrocannabinol
THCA	Δ^9-Tetrahydrocannabinolic acid
THC-COOH	11-nor-9-carboxy-Δ^9-tetrahydrocannabinol
11-OH-THC	11-Hydroxy-Δ^9-tetrahydrocannabinol
TRPA1	Transient receptor potential ankyrin 1
UGT	UDP-glucuronosyltransferase

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