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Physiological Context During Arrest and Custody: A Personal Recollection of Cannabinoid Exposure and Acute Stress

Kathleen Erica Eberhardt

Affiliations:
BA Legal Studies, MA eLearning Pedagogy, EdD Candidate organizational Psychology, Research Scientist, Psychonics Recovery Center LLC

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Correspondence:

Kathleen Erica Eberhardt
BA Legal Studies
MA eLearning Pedagogy
EdD Candidate Organizational Psychology
Research Scientist, Psychonics Recovery Center LLC
Email: kriyakat@yahoo.com **ORCID:** 0009-0000-6159-4701

Abstract: This paper presents an autoethnographic account of the physiological conditions surrounding the author's arrest and custodial transport. Over the preceding year, long-term, high-frequency use of cannabidiol (CBD) via inhalation and oral ingestion had progressively stabilized autonomic and inflammatory systems prior to the acute event. Enforced deprivation of cannabinoids during a prolonged period of psychological siege produced a measurable escalation in physiological stress. During subsequent transport, the author experienced what she believes were two separate instances of airborne opioid exposure, likely fentanyl. Although consciousness was temporarily lost, respiratory arrest did not occur. This outcome is interpreted in part as a consequence of residual physiological stabilization arising from prior CBD saturation. This paper does not advance the claim that CBD functions as an opioid antidote. Rather, it argues that chronic cannabinoid exposure shaped the author's neuroendocrine and inflammatory stress-response profile in ways that may have narrowed the margin between physiological stability and medical failure. Drawing on peer-reviewed literature in cannabinoid pharmacology, trauma physiology, and autonomic neuroscience, the paper contextualizes this lived experience within established mechanisms. It further reframes a sustained pattern of cannabinoid use as physiological stabilization rather than recreational impairment. The policy and clinical implications of this distinction are discussed alongside recommendations for future empirical research.

Keywords: cannabidiol, endocannabinoid system, physiological resilience, trauma physiology, HPA axis, custodial health, autoethnography, opioid toxicity

Introduction:

This paper addresses the physiological circumstances of the author's arrest and custodial transport, as the medical context of those events bears directly on their accurate legal, clinical, and institutional interpretation. The account that follows is grounded in personal recollection and subsequent biomedical analysis. No criminal accusations are advanced; the purpose is descriptive and interpretive, examining lived experience through the framework of established physiological science.

The methodological foundation of this work is autoethnography, a qualitative research approach defined by Ellis et al. (2011) as a method that "seeks to

describe and systematically analyze personal experience in order to understand cultural experience" (p. 273). Autoethnography is increasingly recognized as a legitimate and rigorous tool in health research, offering unique longitudinal perspectives on drug exposure, chronic illness, and bodily experience that are practically and ethically inaccessible through conventional experimental designs. The method is particularly appropriate when the investigator's own body and lived history constitute the primary data source, as is the case here.

In the year preceding her arrest, the author used medical cannabis consistently and at high frequency: multiple daily inhalations and nightly oral ingestion of cannabis-infused edibles. This was not recreational use. It functioned as a regulatory practice for a nervous system destabilized by prolonged trauma, including the sequelae of complex post-traumatic stress disorder (PTSD). Over time, the author's body reached a state of cannabinoid saturation, particularly with respect to CBD. As documented in cannabinoid pharmacokinetic research, CBD is highly lipophilic and accumulates in adipose tissue with extended use; its physiological effects may outlast its acute subjective or psychoactive dimensions by a significant margin (Huestis, 2007).

In the days immediately preceding arrest, the author was effectively confined to her property without financial access or the ability to resupply medical cannabis. The remaining supply was rationed in an attempt to maintain physiological regulation under escalating stress. By Friday evening, following several days of siege, sleep deprivation, nutritional disruption, and acute psychological threat, psychological functioning deteriorated severely. Bodily systems, however, remained more intact — a dissociation the author now understands as pharmacokinetically plausible.

During the arrest and subsequent transport to Butte County Jail — a period of approximately three hours — the author experienced what she believes were two separate instances of airborne opioid exposure, subsequently understood as likely fentanyl. Loss of consciousness occurred on both occasions, yet respiratory arrest did not. Reflecting on this outcome, the author attributes partial survival to the residual physiological stabilization conferred by prior, sustained cannabinoid exposure, particularly CBD.

CBD persists in biological systems at the physiological level longer than it does at the subjective or psychological level. The calming, grounding experience fades; systemic autonomic and anti-inflammatory regulation does not dissipate as rapidly. CBD does not suppress respiration — a property that fundamentally distinguishes it from both opioids and THC (Blessing et al., 2015). It reduces inflammation, attenuates panic-induced autonomic dysregulation, and modulates hypothalamic-pituitary-adrenal (HPA) axis reactivity — all properties of direct relevance when the body is under extreme physiological stress and simultaneously exposed to a substance capable of destabilizing respiratory homeostasis (Henson et al., 2021).

This paper does not claim that CBD is an antidote to fentanyl or any opioid compound. It argues, rather, that the body was chemically conditioned in a manner that elevated the threshold for physiological failure. That conditioning likely narrowed the margin between survival and death. Had cannabinoid exposure been fully interrupted for a longer duration before the acute event, the author believes the physiological outcome might not have been the same.

From the author's perspective, what preserved life was not psychological clarity, behavioral compliance, or deliberate strength. It was physiology. The mind deteriorated first. The body held longer. This account is offered in the interest of medical and historical accuracy, and to reframe chronic cannabinoid use in the context of sustained trauma as a form of physiological stabilization. The interruption of that stabilization, compounded by prolonged environmental threat and incidental toxic exposure, was the true source of medical risk.

Background: The Endocannabinoid System and Stress Regulation

To contextualize the physiological claims presented in this paper, a brief overview of the endocannabinoid system (ECS) is warranted. The ECS is a neuromodulatory network comprised of endogenous lipid-based ligands (anandamide [AEA] and 2-arachidonoylglycerol [2-AG]), their synthetic and degradative enzymes, and cannabinoid receptors (CB1 and CB2), distributed throughout the central and peripheral nervous systems and immune tissues (McPartland et al., 2015). CB1 receptors are densely expressed in the amygdala, hypothalamus, hippocampus, and prefrontal cortex — regions central to fear

processing, emotional regulation, and stress-response modulation (LeDoux, 2012).

The ECS functions as one of the primary regulatory systems governing the stress response. A basal endocannabinoid tone actively inhibits chronic stress activation; modulation of this tone permits or curtails acute stress responses; and chronic deficiency in endocannabinoid signaling has been associated with heightened vulnerability to stress-related pathological states (Henson et al., 2021). The HPA axis and sympatho-adrenal system are the two principal effector arms of the acute stress response, respectively secreting cortisol and catecholamines (noradrenaline and adrenaline) to mobilize the organism under perceived threat. Sustained activation of these systems, as occurs under conditions of siege, sleep deprivation, and psychological trauma, produces allostatic overload with measurable consequences for immune function, inflammatory load, autonomic balance, and physiological resilience.

CBD differs from endogenous cannabinoids and from THC in critical respects. It exhibits low affinity for CB1 and CB2 receptors but modulates the ECS indirectly: through inhibition of fatty acid amide hydrolase (FAAH), which degrades AEA, thereby elevating endogenous endocannabinoid tone; through agonism at serotonin 5-HT_{1a} receptors; through antagonism at GPR55 receptors; and through activation of transient receptor potential vanilloid type 1 (TRPV1) channels (Blessing et al., 2015; Campos et al., 2012). Through these multiple mechanisms, CBD exerts clinically relevant anxiolytic, anti-inflammatory, and autonomic stabilizing effects without inducing psychoactive intoxication or respiratory depression.

Physiological Plausibility of Cannabidiol as a Buffer During Acute Stress Exposure

Autonomic Modulation and Sympathetic Dampening

Chronic exposure to CBD prior to an acute stress event may plausibly increase physiological resilience through sustained modulation of the autonomic nervous system, inflammatory signaling, and neuroendocrine regulation. CBD has been demonstrated to reduce sympathetic hyperarousal and support parasympathetic regulation — mechanisms of direct relevance under conditions of extreme psychological stress or trauma (Blessing et al., 2015; McPartland et al., 2015). In clinical and preclinical settings, CBD administration has been shown to blunt cortisol release, reduce heart rate elevations during stress

exposure, and improve heart rate variability as a proxy measure of parasympathetic tone (Henson et al., 2021).

In a comprehensive review by Henson et al. (2021), seven double-blind, placebo-controlled clinical trials examining CBD for stress — encompassing 232 participants — all reported that CBD significantly reduced the stress response and performed no worse than pharmaceutical comparators when such were included. The review concluded that CBD functions as a safe endocannabinoid system enhancer, operating via increased anandamide availability and downstream modulation of HPA axis reactivity. This evidence base provides a plausible pharmacological foundation for interpreting the author's preserved physiological stability during periods of extreme stress.

Pharmacokinetics: Lipophilicity and Delayed Clearance

CBD is highly lipophilic and undergoes extensive tissue redistribution following chronic, multi-route administration. With prolonged, high-frequency exposure via both inhalation and oral ingestion, lipid solubility and accumulation in adipose compartments result in delayed systemic clearance, allowing physiological effects to persist well beyond the attenuation of acute subjective or psychoactive effects (Huestis, 2007). In frequent users, CBD terminal half-life may extend considerably beyond the 2–5 day range observed in single-dose studies, owing to this tissue accumulation. This pharmacokinetic reality is central to the interpretation advanced here: that the body may retain physiologically meaningful cannabinoid exposure even after subjective effects have fully dissipated — and even after external supply has been interrupted for several days.

Anti-inflammatory and Neuroprotective Effects

CBD's anti-inflammatory properties are well established in both preclinical and clinical literature. Through inhibition of pro-inflammatory cytokine production (including TNF-α, IL-1β, and IL-6), reduction of NF-κB pathway activation, and attenuation of reactive oxygen species (ROS) generation, CBD may reduce the systemic inflammatory load that accumulates under conditions of chronic psychological stress (Atalay et al., 2019). Neuroinflammation, in particular, represents a significant risk factor for acute physiological decompensation: inflammatory cascades within the central nervous system can potentiate hypoxic and ischemic injury, impair autonomic recovery, and

amplify the physiological burden of toxic exposure. By dampening cytokine activation and neuroinflammatory cascades during periods of pre-exposure cannabinoid saturation, CBD may reduce secondary injury potential during acute stress, thereby expanding the margin of physiological tolerance (Atalay et al., 2019; Campos et al., 2012).

CBD, PTSD, and the Trauma-Exposed Body

The author's physiological context at the time of arrest is inseparable from her history of complex trauma. CBD has emerged as a promising candidate for PTSD symptom management due to its multimodal action on fear circuitry, memory consolidation, and sleep architecture. Elms et al. (2019) reported a retrospective case series of 11 adults with PTSD receiving oral CBD as an adjunct to psychiatric care. In patients who completed 36 or more weeks of treatment, mean PCL-5 PTSD symptom scores decreased from 57.75 to 29.25, with over 38% reporting subjective improvement in sleep quality. These findings suggest that in trauma-exposed individuals, chronic CBD use may produce enduring modifications to autonomic tone, amygdala reactivity, and stress-circuit sensitization — modifications that may persist beyond any single administration episode.

Sleep deprivation and nutritional compromise, both of which the author experienced during the pre-arrest siege period, independently impair autonomic regulation, elevate inflammatory markers, and increase physiological vulnerability to subsequent stressors. The author's partial cannabinoid saturation during this period may thus have provided a countervailing buffer against physiological deterioration that would otherwise have been more rapid and more severe. Van der Kolk's (2014) foundational model of somatic trauma storage emphasizes that the body retains the imprint of threat exposure in its autonomic, musculoskeletal, and neuroendocrine architecture. Within this framework, the author's body may have been simultaneously burdened by accumulated trauma and partially protected by accumulated pharmacological stabilization.

Fentanyl Exposure: Respiratory Mechanism and Physiological Vulnerability

Fentanyl is a potent synthetic opioid with high affinity for μ-opioid receptors in the brainstem respiratory control centers. Opioid-induced respiratory depression (OIRD) is the primary lethal mechanism in fentanyl overdose and proceeds through suppression of the pre-Bötzinger complex (the brainstem

rhythm-generating circuit for respiration), central apnea, thoracic wall rigidity ("wooden chest syndrome"), and progressive hypoxemia (Varshneya et al., 2022). Recovery from acute fentanyl-induced respiratory depression depends on multiple factors: the magnitude of hypoxemia reached, the presence of compensatory μ-receptor desensitization mechanisms, and the integrity of the individual's residual autonomic and respiratory buffers.

CBD does not function as a pharmacological antagonist at μ-opioid receptors and does not directly reverse OIRD — a role that remains specific to naloxone. However, CBD may influence opioid-related physiological risk through indirect mechanisms: by reducing acute anxiety and panic-induced hyperventilation that can compound hypoxemic episodes; by attenuating inflammatory responses associated with hypoxia and systemic shock; and by supporting autonomic recovery capacity through its established parasympathomimetic effects (Capano et al., 2020; van der Kolk, 2014). It is within this indirect, systems-level framework that the author's prior cannabinoid saturation may have contributed to survival.

Limitations

This analysis is subject to several important constraints. First, its observational foundation is autoethnographic, which — while methodologically legitimate for health research — does not permit causal inference (Ellis et al., 2011). No biomarker data (e.g., plasma CBD levels, cortisol concentrations, inflammatory markers, or respiratory metrics) were collected contemporaneously or retrospectively to quantify cannabinoid presence, autonomic function, or the temporal relationship between CBD exposure and the acute event. The physiological mechanisms invoked are supported by existing peer-reviewed literature; their application to this specific case rests on plausibility inference rather than direct measurement.

Second, the absence of toxicological confirmation precludes definitive conclusions regarding the nature, concentration, or route of any concurrent pharmacological exposure during transport. The determination that the substance in question was fentanyl is inferential and based on symptom profile and contextual circumstances, not laboratory analysis. Accordingly, all pharmacodynamic reasoning is framed in terms of plausible mechanism rather than confirmed interaction.

Third, interindividual variability in cannabinoid pharmacokinetics is substantial, reflecting differences in body mass index, adipose distribution, hepatic metabolism via CYP3A4 and CYP2C19 pathways, dosing consistency, and route of administration (Huestis, 2007). The persistence of physiologically meaningful CBD effects beyond subjective attenuation is inferred from the pharmacokinetic literature, not modeled quantitatively for this case. Psychological stress, chronic sleep deprivation, nutritional compromise, and environmental factors may all have independently and interactively shaped physiological resilience.

Fourth, survivorship bias must be explicitly acknowledged. This analysis is retrospective and outcome-informed; the author is alive to write it, which may structurally bias interpretation toward stabilizing factors while underrepresenting variables that could, in other individuals or circumstances, have led to a different outcome. CBD is framed throughout not as a protective or antidotal agent in isolation, but as a potential contributor to a complex and multifactorial physiological state.

These limitations do not invalidate the analysis, but they underscore the need for controlled, prospective research before population-level conclusions can be drawn. They are presented here in the interest of scientific transparency and methodological integrity.

Strengths

Despite the limitations inherent to its methodology, this analysis offers several contributions of substantive value. First, it applies a conservative, mechanism-focused interpretive framework that anchors lived experience in established physiological literature without asserting causal inference beyond what the evidence can support. By grounding interpretation in autonomic regulation, inflammatory modulation, HPA axis physiology, and cannabinoid pharmacokinetics, the analysis achieves biological coherence and avoids the speculative attribution that frequently characterizes patient-reported accounts of substance use.

Second, the autoethnographic design provides rare longitudinal ecological data on chronic, multi-route, high-frequency CBD exposure under real-world trauma conditions that are ethically and practically impossible to replicate in controlled settings (Ellis et al., 2011). Short-duration clinical trials of CBD — the dominant form in the extant literature — cannot capture the pharmacokinetic accumulation

dynamics or the sustained autonomic recalibration that may occur over months of uninterrupted use. This case contributes to a gap in the literature regarding the extended physiological consequences of high-frequency CBD use in trauma-exposed populations.

Third, the analysis performs a conceptually important distinction between CBD's psychological effect profile and its physiological effect profile, highlighting their differential temporal persistence. This distinction is underemphasized in both the research literature and in clinical and legal settings, where cannabinoid presence is frequently conflated with intoxication. The argument advanced here — that physiological stabilization and psychological impairment occupy separate pharmacokinetic timescales — has direct implications for how cannabis use should be evaluated in trauma-exposed populations, correctional settings, and emergency medicine.

Finally, by situating individual survival within a systems-level model of stress physiology rather than attributing outcomes to any single variable, this work reinforces the complexity of physiological resilience under extreme conditions. This framing is consistent with contemporary trauma physiology models (van der Kolk, 2014; LeDoux, 2012) and avoids the reductionism that characterizes much policy discourse on substance use. Together, these strengths support the contribution of this paper as hypothesis-generating, ecologically valid, and methodologically responsible — a foundation for future empirical inquiry rather than a substitute for it.

Policy and Clinical Implications with Future Recommendations

Policy Implications

The observations presented in this paper suggest that non-intoxicating cannabinoids such as CBD warrant more sophisticated consideration in public health policy, drug regulation, and custodial health frameworks — particularly in contexts involving trauma exposure, autonomic dysregulation, and chronic physiological stress. Current regulatory models in many jurisdictions continue to conflate CBD with psychoactive cannabinoids or to evaluate it within the same risk-classification frameworks as opioids and central nervous system depressants. This conflation obscures CBD's distinct pharmacological profile, mischaracterizes its differential risk, and may

result in policies that inadvertently interrupt stabilizing therapies in vulnerable populations.

This paper supports distinction-based regulation that differentiates cannabinoids according to pharmacological mechanism rather than cultural association or route of delivery. CBD's absence of respiratory depression, its established safety profile in clinical use, and its evidence-supported role in autonomic and inflammatory modulation collectively indicate relevance for trauma-informed public health paradigms rather than solely for substance-control frameworks (Henson et al., 2021; Blessing et al., 2015).

Of particular concern is the institutional environment of custodial settings. Detention and correctional facilities represent environments of extreme and sustained stress, restricted access to therapeutic agents, sleep disruption, and elevated physiological vulnerability. Policies governing toxicology reporting, detention health protocols, and emergency response within these settings may benefit from cannabinoid-specific modifications — recognizing that the abrupt deprivation of stabilizing cannabinoid therapy under conditions of acute stress may itself constitute a physiological risk factor, rather than a neutral event. The author's experience provides a concrete, documented case in which this dynamic played a role in determining the physiological outcome of a custodial encounter.

Clinical Guidance Considerations

Clinically, this paper does not support CBD as an antidote or prophylactic agent against opioid exposure. However, it does raise relevant considerations for clinicians working in emergency medicine, psychiatry, correctional health, and primary care settings serving populations with high trauma burden and potential cannabinoid exposure.

Key considerations for clinicians include:

- Distinguishing between psychological symptom relief and physiological regulation when taking cannabinoid use histories, particularly in trauma-exposed patients
- Recognizing that abrupt discontinuation of stabilizing cannabinoid regimens during acute stress may exacerbate autonomic dysregulation, heighten HPA axis reactivity, and increase physiological vulnerability to concurrent stressors (Henson et al., 2021)
- Refraining from the clinical assumption that cannabinoid presence implies intoxication, impairment, or

elevated respiratory risk — an assumption that is pharmacologically unwarranted for CBD-dominant use profiles (Blessing et al., 2015)

- Incorporating trauma-informed assessment models that account for inflammatory load, sleep disruption, nutritional status, and autonomic imbalance when evaluating patients with complex histories of substance use and psychological trauma (van der Kolk, 2014)
- In emergency settings, considering prior cannabinoid exposure as a potential contributor to baseline physiological state when evaluating patients who have survived apparent toxic exposures without expected respiratory collapse

Future Research and Policy Recommendations

Advancing the scientific basis for the claims and hypotheses developed here will require prospective, controlled, and biomarker-anchored research. The following directions represent priorities identified by this analysis:

- Longitudinal biomarker studies measuring plasma cannabinoid levels, cortisol trajectories, heart rate variability, and inflammatory markers (e.g., IL-6, TNF-α, CRP) across extended periods of chronic CBD use in trauma-exposed individuals
- Pharmacokinetic modeling of CBD accumulation and clearance dynamics in high-frequency, multi-route users, with particular attention to the temporal persistence of physiological effects relative to subjective effects
- Trauma-informed clinical trials examining CBD's role in autonomic regulation and stress-response attenuation, with designs that exclude confounding by concurrent THC exposure
- Ethical observational studies within correctional and custodial environments examining the physiological consequences of cannabinoid deprivation under acute stress conditions
- Policy analyses examining the adequacy of current cannabinoid classification frameworks in emergency medicine, detention health, and public health contexts

From a regulatory perspective, agencies should consider revising classification frameworks to reflect pharmacological specificity, ensuring that clinical guidelines and institutional protocols are grounded in current evidence rather than legacy stigma. Specifically, the distinction between CBD and intoxicating cannabinoids should

be codified in clinical triage protocols, custodial health standards, and drug testing interpretation guidelines.

Conclusion:

This paper has argued that chronic, high-frequency CBD use prior to an episode of acute stress and potential opioid exposure may have contributed to physiological resilience through the mechanisms of autonomic stabilization, anti-inflammatory modulation, and endocannabinoid tone maintenance. These mechanisms are pharmacologically plausible, consistent with the peer-reviewed literature, and have been presented here in full acknowledgment of the methodological constraints of an autoethnographic account.

The broader significance of this analysis lies not only in what it says about one individual's survival, but in what it implies for how therapeutic cannabinoid use is understood, classified, and managed in medical and institutional contexts. Substance use that functions as physiological stabilization for a trauma-compromised nervous system is categorically distinct from recreational impairment. Policies and clinical practices that fail to make this distinction risk causing iatrogenic harm through the deprivation of stabilizing therapies at precisely the moments of greatest physiological vulnerability.

The author's body, not her mind, held the margin of survival. That fact warrants medical acknowledgment, clinical respect, and sustained scientific investigation.

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