

# Healing through homeostasis: the therapeutic potential of *Cannabis sativa* and the endocannabinoid system

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Patricia Beck Eichler-Barker

EcoLogicProject, Highway 9, Boulder Creek, 95006, California, USA

**Correspondence:** Patricia Beck Eichler-Barker, EcoLogicProject, Highway 9, Boulder Creek, 95006, California, USA, Tel 5548999590528**Received:** February 25, 2026 | **Published:** March 6, 2026

## Editorial

Chronic conditions such as persistent pain, inflammation, neurological dysfunction, and spasticity continue to present significant therapeutic challenges, particularly in patients who exhibit limited response to conventional pharmacological interventions. In recent decades, *Cannabis sativa* has re-emerged as a subject of rigorous biomedical investigation, driven by expanding evidence regarding its pharmacologically active constituents and their interaction with endogenous regulatory systems.<sup>1</sup> Accumulating data suggest that cannabinoid-based therapies may offer clinically relevant benefits in selected chronic disorders, particularly where standard treatments demonstrate limited efficacy or unfavorable safety profiles. The therapeutic rationale for *Cannabis*-based interventions is grounded in its interaction with the endocannabinoid system (ECS), a highly conserved neuro modulatory and immunoregulatory network present in humans and other mammals.<sup>2,3</sup> The ECS comprises cannabinoid receptors (CB1 and CB2), endogenous ligands—primarily anandamide (AEA) and 2-arachidonoylglycerol (2-AG)—and the enzymatic machinery responsible for their synthesis and degradation. CB1 receptors are predominantly expressed in the central nervous system and are involved in the modulation of nociception, emotional processing, memory, and motor coordination, whereas CB2 receptors are largely distributed in immune and peripheral tissues, where they regulate inflammatory responses.<sup>3</sup>

Endocannabinoids are synthesized “on demand” and rapidly degraded by enzymes such as fatty acid amide hydrolase (FAAH) and monoacylglycerol lipase (MAGL), enabling fine-tuned physiological control.<sup>4</sup> Through these coordinated mechanisms, the ECS contributes to systemic homeostasis across multiple biological domains, including pain modulation, immune function, stress adaptation, appetite regulation, sleep architecture, and affective balance. Anandamide primarily interacts with CB1 receptors and plays a central role in reward circuitry and emotional modulation, acting as a transient neuromodulator due to its short half-life.<sup>4</sup> In contrast, 2-AG is typically more abundant within the nervous system and binds both CB1 and CB2 receptors, contributing to synaptic plasticity and immune regulation.<sup>3</sup> Dysregulation of ECS signaling has been hypothesized to underlie certain chronic pain syndromes, including migraine and fibromyalgia, a theoretical construct described as clinical endocannabinoid deficiency, although further empirical validation is required. Phyto cannabinoids derived from *Cannabis sativa*, particularly  $\Delta^9$ -tetrahydrocannabinol (THC) and cannabidiol (CBD), interact with this endogenous system through distinct pharmacodynamic mechanisms.

THC functions as a partial agonist at CB1 and CB2 receptors, exerting analgesic and antispastic effects but also producing dose-

dependent psychoactive outcomes.<sup>5</sup> CBD demonstrates low direct affinity for cannabinoid receptors yet modulates ECS activity indirectly, including inhibition of FAAH, thereby potentially increasing anandamide bioavailability.<sup>7</sup> Through these mechanisms, phytocannabinoids may attenuate nociceptive transmission and inflammatory cascades, supporting their investigation in chronic inflammatory and neuropathic conditions.<sup>6</sup> A comprehensive review by the National Academies of Sciences, Engineering, and Medicine concluded that substantial evidence supports the efficacy of *Cannabis* or cannabinoids in the management of chronic pain in adults, chemotherapy-induced nausea and vomiting, and spasticity associated with multiple sclerosis.<sup>7</sup> Additional evidence supports their use in treatment-resistant epilepsy syndromes and neuropathic pain states.<sup>8</sup> Compared with opioids, cannabinoids demonstrate a markedly lower risk of fatal toxicity, although adverse effects and long-term safety considerations remain clinically relevant.<sup>5</sup>

Emerging research has also explored the relationship between the ECS and menopause. The menopausal transition involves declining estrogen and progesterone levels, leading to neuroendocrine alterations affecting thermoregulation, mood stability, sleep, and genitourinary health. Estrogen is known to influence endocannabinoid tone and receptor expression; thus, hormonal fluctuations may alter ECS signaling and contribute to vasomotor symptoms and mood disturbances. A case study conducted by Eichler-Barker (2024) evaluated a vaginal formulation containing shea butter, medium-chain triglyceride (MCT) oil, and full-spectrum *Cannabis sativa* extract (5% THC and 2.5% CBD) in ten menopausal women.<sup>9</sup> After four weeks of application, participants reported improvements in hot flashes, vaginal dryness, mood stability, and sleep quality. Although limited by small sample size and absence of a control group, the findings suggest that localized cannabinoid delivery may modulate ECS activity in reproductive and neural tissues, warranting further controlled investigation.<sup>9</sup> Despite promising findings, significant challenges remain, including variability in *Cannabis* preparations, heterogeneity in dosing regimens, and regulatory barriers that complicate large-scale randomized controlled trials.<sup>5</sup> Rigorous, well-designed clinical studies are necessary to clarify therapeutic indications, long-term safety, and pharmacokinetic parameters across diverse populations.

## Conclusion

The endocannabinoid system represents a central integrative network responsible for maintaining physiological equilibrium. Through pharmacological interaction with this system, *Cannabis sativa* and its phytocannabinoids demonstrate therapeutic potential in chronic pain, inflammatory disorders, neurological conditions, and possibly menopausal symptom management. Continued translational and clinical research will be essential to define their role within evidence-based medical practice.

## Acknowledgments

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## Conflicts of interest

None.

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