# Sociedad Española de Investigación sobre Cannabinoides

Boletín electrónico (enero-marzo de 2025; nº 81)
Editores: Pablo Peña y Simón Porzio


**Contenido:**



## 1. Saludo de la Presidenta

Querido/as socios/as:
Espero que estéis muy bien y que las lluvias torrenciales de las últimas semanas no hayan impactado negativamente en vuestras actividades profesionales y personales.
Aprovecho estas líneas para daros las gracias por vuestra participación en la encuesta que os enviamos para decidir si la SEIC se adhería a la declaración de ALLEA (All European Academies) sobre las amenazas a la libertad académica y la colaboración internacional en investigación en Estados Unidos. Este ejercicio nos ha servido para hacer una valoración de las ventajas e inconvenientes de esta vía de consulta, asunto que discutiremos en detalle en la asamblea general de 2025 que celebraremos a finales de noviembre en Madrid.
Relacionado con esto último, os informo también de que los preparativos de la Reunión Anual de este año siguen en marcha y que hemos conseguido un par de patrocinadores nuevos (CliniSciences, Dismed y PlantCell), a los que se suma el apoyo un año más de nuestros patrocinadores históricos, Phytoplant y Fundación Canna.

Un abrazo,
Cristina Sánchez

## 2. Effects of cannabis oil on hepatic steatosis, inflammation, and fibrosis through endocannabinoid regulation in NASH model

**Premio a la mejor Comunicación Oral Predoctoral, 24ª Reunión anual de la SEIC, Córdoba (2024)**

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Metabolic dysfunctions like dyslipidemia, hypertension, and obesity have dramatically increased worldwide due to excessive carbohydrate intake (especially sucrose and fructose) and sedentary lifestyles, contributing to non-alcoholic fatty liver disease (NAFLD). This condition progresses from steatosis to non-alcoholic steatohepatitis (NASH), marked by inflammation and fibrosis. Key molecular mechanisms include PPARα, which regulates fatty acid oxidation, and markers such as MPO, TGF-β1, and hydroxyproline, which are involved in inflammation and fibrosis. The endocannabinoid system (ECS), through CB1 and CB2 receptors, plays a pivotal role in NASH, with CB1 linked to steatosis and fibrosis, and CB2 to inflammation. Cannabinoids, especially the most studied CBD and THC, can modulate these receptors promoting or preventing their effects.

This study aims to evaluate whether daily oral administration of 1mg/kg full-spectrum cannabis oil (CBD:THC, 2:1 ratio), over 3 weeks, can prevent NASH development. To achieve that, male Wistar rats were randomly divided and fed for 3 weeks with the following diets: Reference Diet (RD): standard commercial laboratory diet (GEPSA FEED); Sucrose Rich Diet (SRD) and SRD+Cannabis Oil (SRD+Ca): non-invasive oral administration of 1 mg/kg of cannabis oil (CBD:THC, 2:1 ratio). We evaluated in liver: triglyceride (TG) content, histological analysis by optical microscopy and qualitative analysis of ultrastructural images by electron microscopy, NAFLD activity score (NAS), activity of lipogenic, fatty acid oxidation and myeloperoxidase (MPO) enzymes, collagen content, hydroxyproline levels, and expression of TGF-β1 and CB1 and CB2 receptors.

Animals fed with SRD showed a significant increase in TG content as a consequence of enhanced synthesis - and less oxidation - of fatty acids, observing an accumulation of lipid droplets in the liver along with ultrastructural changes and higher NAS score. Additionally, MPO activity, TGF-β1 expression, collagen content and hydroxyproline levels were significantly increased, reflecting inflammation and fibrosis. These alterations were accompanied by a significant increase in the liver expression of CB1 and CB2 receptors. Daily oral administration of cannabis oil for 3 weeks prevented hepatic steatosis, being able to improve TG content, lipid metabolism and lipid droplet accumulation and changes in ultrastructure, with a significantly lower NAS score, while preventing the increase of inflammation and fibrosis markers and the overexpression of the ECS.

Our results reveal the antilipogenic, anti-inflammatory, and antifibrotic properties of cannabis oil and motivates us to continue exploring the molecular pathways behind these effects.

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## 3. The role of PPAR-γ in memory deficits induced by prenatal and lactation alcohol exposure in mice

**Premio a la mejor Publicación Predoctoral, 24ª Reunión anual de la SEIC, Córdoba (2024)**


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Alcohol exposure during gestation and lactation can lead to physical and behavioral impairments collectively known as fetal alcohol spectrum disorder (FASD)[1]. Cognitive dysfunction is one of the most common consequences, significantly affecting patients' quality of life[2]. However, the pathophysiological mechanisms behind these deficits remain unclear. Previous studies have shown that the expanded endocannabinoid system (ECS) is highly sensitive to alcohol exposure during adolescence[3] and adulthood[4]. Yet, its involvement in the cognitive deficits resulting from early alcohol exposure has not been explored.

In this study, we aimed to assess the role of the expanded ECS in the memory impairments caused by prenatal and lactation alcohol exposure (PLAE) in mice. To model binge-like alcohol consumption, C57BL/6 female mice were given limited access to either

water or a 20% v/v alcohol solution during gestation and lactation. Our results showed that PLAE-induced memory deficits were associated with a decrease in N-acylethanolamines (NAEs) and peroxisome proliferator-activated receptor gamma (PPAR-γ) levels in the hippocampus, specifically during a childhood-like period (postnatal day 25, PD25).

To investigate their role in memory deficits, we conducted two pharmacological approaches from PD25 to PD34, followed by memory assessments at PD60: (i) treatment with URB597, a FAAH inhibitor that increases NAEs, combined with GW9662, a PPAR-γ antagonist to block NAEs' effects through PPAR-γ, and (ii) administration of pioglitazone, a PPAR-γ agonist to directly activate this receptor. Our findings revealed that URB597 prevented PLAE-induced memory deficits via a PPAR-γ-dependent mechanism, as its effects were blocked by GW9662. Similarly, direct PPAR-γ activation with pioglitazone also improved memory performance. To further explore the specificity of these effects, we demonstrated that early overexpression of PPAR-γ in hippocampal astrocytes, achieved through a viral vector, mitigated PLAE-induced memory impairments.

Overall, our data suggest that disruptions in PPAR-γ signaling during neurodevelopment contribute to memory deficits caused by early alcohol exposure. Furthermore, PPAR-γ activation during a childhood-like period emerges as a promising therapeutic strategy to counteract these cognitive impairments. These findings offer new insights into the mechanisms underlying memory dysfunction in FASD patients.

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## 4. GPR55-deficient mice are more resistant to the induction of experimental Parkinson's disease

**Premio a la mejor Comunicación Poster Predoctoral, 24ª Reunión anual de la SEIC, Córdoba (2024)**


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Parkinson's disease (PD) is the second most common neurodegenerative disorder. It is primarily characterized by motor symptoms, including bradykinesia, rigidity, and tremor [1]. The disease results from the progressive

degeneration of dopaminergic neurons in the substantia nigra (SN) pars compacta, leading to dopamine denervation and depletion in the striatum [2]. Currently, there is no cure for PD, highlighting the urgent need for novel therapeutic targets. In this context, our study focuses on the G protein-coupled receptor 55 (GPR55), a non-canonical cannabinoid receptor with high affinity for various cannabinoids [3]. GPR55 is abundantly expressed in key basal ganglia structures, such as the striatum and SN [4]. It is also involved in regulating motor activity and providing neuroprotection in neurodegeneration models [5,6], making it a promising candidate for PD research.

To investigate the role of GPR55 in PD, we used GPR55 full knockout (KO) mice of both sexes and induced a PD-like pathology. While GPR55-deficient mice show no significant changes in brain structure or neuronal cytoarchitecture, some motor performance impairments have been detected [5]. Parkinsonian pathology was induced via unilateral inoculation of adeno-associated viruses to locally overexpress the mutated alpha-synucleinA53T in the SN, thereby creating a proteinopathic environment [7]. In this model, the absence of GPR55 attenuated dopaminergic neuron loss caused by the nigral lesion, as evidenced by tyrosine hydroxylase immunostaining in the SN. This neuroprotective effect was more pronounced in females than in males and correlated with motor improvement in the behavioral cylinder rearing test. Additionally, C3 immunoreactivity in GFAP-positive cells, a marker of astrocytic reactivity, revealed that the lesion-induced increase observed in wild-type animals was absent in GPR55-deficient females, whereas no genotype-dependent differences were found in males.

These findings suggest that GPR55 plays a detrimental role in PD progression and that its blockage could represent a promising therapeutic strategy, with a pivotal involvement of glial cells, as previously proposed [8]. Thus, the potential use of GPR55 antagonists to mitigate PD pathology opens new avenues for research that warrants further exploration. Indeed, regarding PD research, one relevant consideration is to include several animal models, as they often recapitulate only limited aspects of human disease. Following this issue, we included a second murine model to confirm our observations. In this model, PD-like pathology was induced by striatal unilateral injection of the neurotoxin 6-hydroxydopamine, leading to mitochondrial dysfunction and oxidative stress [9]. So far, only behavioral tests have been performed, but the results show a consistent trend to the previous outcomes: the absence of GPR55 confers protection against the lesion, particularly in females. Therefore, these findings further support the growing body of evidence suggesting the role of GPR55 in neurodegeneration and its potential as a therapeutic target for PD, although some questions such glial implications and sex differences still need to be fully understood.

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## 5. Agenda

**NEUROFRANCE 2025**
14-16 mayo 2025
Montpellier, Francia
https://www.neurosciences.asso.fr/SN25/

**Fens Regional Meeting (FRM) 2025**
16-19 junio 2025
Oslo, Noruega
https://frm2025oslo.no/

**Annual symposium on the cannabinoids (ICRS)**
6-10 julio 2025
Bloomington, Indiana
https://www.icrs.com/annual-symposium

**5th annual meeting of the IRN iGPCR net**
7-9 julio 2025
Barcelona
http://www.i-gpcrnet.com

**Neuroscience 2025 – Society for Neuroscience Annual Meeting (SENC) 2025**
3-5 septiembre 2025
Las Palmas de Gran Canaria
https://congresosenc.es/

**Society for Neuroscience (SFN) 2025**
15-19 noviembre 2025
San Diego, EEUU
https://www.sfn.org/meetings/neuroscience-2025/

## 6. Últimas publicaciones sobre cannabinoides de investigadores/as españoles (periodo enero-marzo 2025)

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