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Editores

Esther Montoliu y Simón Porzio

Contenido

1. Saludo de la Presidenta

Queridos/as socios/as:

Espero que estéis todos/as muy bien y disfrutando de esta primavera incipiente que ha llegado con fuerza.

Como os informamos hace un par de semanas, y tal y como decidimos en nuestra última asamblea, le hemos dado un lavado de cara al logotipo y a la página web de nuestra sociedad. Creemos que esta nueva imagen de la SEIC es más moderna, fresca y profesional, y esperamos que sea de vuestro agrado. Os animamos a que, si no lo habéis hecho ya, le echéis un vistazo y nos hagáis llegar las sugerencias que consideréis oportunas.

Relacionado con este cambio, hemos tenido algún desafío técnico relacionado con el envío de correos electrónicos a los/as socios/as que ya ha sido solventado.

Quiero aprovechar este hueco para dar las gracias a las personas que se han encargado de esta tarea, muy especialmente a Leyre Urigüen y Onintza Sagredo.

Un abrazo,

Cristina

2. Premio a la mejor publicación postdoctoral (25ª Reunión anual de la SEIC, Madrid 2025)

Activation of central cannabinoid type 2 receptors, but not on peripheral immune cells, is required for endocannabinoid-mediated neuroprotection in Parkinson's disease

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Neuroinflammation is a key feature of Parkinson's disease (PD). The cannabinoid receptor type 2 (CB2R) is expressed by cells of the innate and adaptive immune systems. Inhibition of monoacylglycerol lipase (MAGL) with JZL184 increases the levels of the endocannabinoid 2-arachidonoylglycerol (2-AG), which is neuroprotective for dopaminergic neurons. The aim of this study was to determine whether the neuroprotective effect of MAGL inhibition is mediated by CB2R activation on specific immune cell populations.

Experimental parkinsonism was induced by chronic administration of MPTP and probenecid (MPTPp). To generate the experimental model, 4-month-old mice received 10 i.p. injections of MPTP plus probenecid to delay renal MPTP clearance. The compounds were co-administered in two consecutive injections twice weekly for 5 weeks.

We prepared cell suspensions from the striatum and the ventral midbrain to analyze immune cells by flow cytometry. Myeloid cells were identified by the expression of CD11b and CD45, the CD11b+CD45low gate was assigned to resident microglia and the CD11b+CD45high gate to myeloid infiltrating cells and resident activated microglia. CD4+ and CD8+ T cells were selected from the CD11b-CD45high population. A specific increase in CD4+ T cell infiltration was observed in the ventral midbrain of MPTPp mice, but not in JZL184-treated animals. These results suggest an association between CD4+ T cell infiltration in the ventral midbrain and dopaminergic neuronal cell death.

We next asked whether immune cells were directly involved in the neuroprotective effect of JZL184 or were indirectly affected by the improvement in neuronal survival. In the brain, CB2R expression was restricted to myeloid cells and lymphocytes, and increased in microglia under parkinsonian conditions. Constitutive CB2R KO mice were intoxicated with MPTPp and treated with JZL184. The drug had no effect in CB2R KO mice, suggesting that CB2R is required for neuroprotection.

To validate the neuroprotective effect of CB2R activation, MPTPp mice were treated with a central CB2R agonist, JWH133. The drug exerted a beneficial effect similar to that of JZL184, whereas the peripheral agonist RO304 lacked neuroprotective activity.

These results were confirmed using bone marrow chimeric mice. Irradiated WT mice were transplanted with HSCs from WT (Chi-WT) or CB2R KO (Chi-CB2R KO) donor mice. MPTPp-intoxicated chimeric mice treated with JZL184 showed a neuroprotective effect independently of the transplant. These results suggest that CB2R activation in the brain, probably in microglial cells, but not in infiltrating peripheral immune cells is necessary for neuroprotection.

In silico analysis, showed that transcripts related to 2-AG biosynthesis were downregulated in the midbrain microglia from PD patients.

In summary, our results show that activation of CB2R in the brain prevents nigrostriatal degeneration, CD4+ T cell infiltration and TNFα production in the midbrain of parkinsonian mice. The reduced 2-AG signaling in microglia from PD patients suggests that activation of microglial CB2R may be an interesting strategy for the treatment of PD.

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3. Premio a la mejor publicación postdoctoral (25ª Reunión anual de la SEIC, Madrid 2025)

Premio a la mejor Comunicación Oral Predoctoral, 25ª Reunión anual de la SEIC, Madrid (2025)

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Dravet Syndrome (DS) is a rare and severe form of childhood epilepsy caused by mutations in the SCN1A gene, leading to abnormal neuronal hyperexcitability through Nav1.1 sodium channels, resulting in severe epileptic seizures and various associated comorbidities. This disease is refractory to current pharmacological treatments, highlighting the urgent need for the identification and validation of novel therapeutic targets. A few years ago, Epidiolex, an oral formulation of cannabidiol (CBD), and fenfluramine (FFA), which acts on the serotonergic system, were approved in both the United States and Europe for the reduction of epileptic seizures in patients with Dravet Syndrome (DS).

The aim of this study is to evaluate the disease-modifying effects of compounds targeting the endocannabinoid and serotonergic systems. To this end, we employed RO-6866945 (RO-686), a CB2 receptor agonist, and FFA, which modulates the serotonergic system. Furthermore, we evaluated whether the combined administration of FFA and RO-686 enhanced the effects observed with each drug when administered individually.

Conditional knock-in mutant mice (Syn1-Cre/Scn1a$^{WT/A1783V}$) generated by Cre-loxP technology were used. Treatments were administered intraperitoneally every two days between PND10 and PND24, and motor reflexes and body weight were recorded on alternate days. Behavioural tests were conducted on PND23 and PND24 to evaluate motor deficits, cognitive impairment, and autistic traits. Finally, on PND25, the animals were euthanized to analyse glial reactivity using immunohistochemical techniques.

Treatment with RO-686, as well as with FFA, improved survival, the acquisition of motor reflexes, and the performance in locomotion and social interaction tests. Additionally, a reduction in astrogliosis (GFAP) in the hippocampus and prefrontal cortex was observed. However, the combined administration of both compounds did not provide additional benefits beyond those obtained with the individual administration of each drug.

In conclusion, both FFA and Ro686 exert beneficial effects on several neurological and behavioural outcomes in the DS model. Although their combined administration did not enhance the individual effects, these findings indicate that independent modulation of either endocannabinoid or serotonergic systems may represent a promising strategy to alleviate some DS-associated comorbidities. Moreover, they underscore the importance of further investigating the cellular and molecular mechanisms underlying these neuroprotective effects.

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4. Agenda

Cannabis Europa 2026 – London
26-27 mayo 2026
Londres, Reino Unido
https://cannabis-europa.com

Annual Symposium on the Cannabinoids (ICRS) 2026
28 junio – 2 julio 2026
Dijon, Francia
https://www.icrs.com

14th International Conference on Cannabis & Medicinal Research
23-24 julio 2026
Ámsterdam, Países Bajos
https://cannabis-marijuana.neurologyconference.com

Cannabis Research Conference 2026
19-21 octubre 2026
San Diego, California, EE. UU.
https://www.instituteofcannabisresearchcolorado.org/conference/

5. Últimas publicaciones sobre cannabinoides de investigadores españoles (enero-marzo 2026)

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