Sociedad Española de Investigación sobre Cannabinoides

Boletín electrónico (octubre-diciembre 2013; nº 41)

Contenido:

1. Saludo del Presidente

Estimados socios:

Cerramos el año 2013 con la celebración en Barcelona de la 14ª reunión científica anual de nuestra Sociedad (muchas gracias de nuevo, Ester, Andrés, Rafael y Fernando, por vuestro excelente trabajo en la organización del evento) y comenzamos ahora un año 2014 que espero y deseo sea muy feliz personal y profesionalmente hablando para todos vosotros.

Una vez más, entre las acciones generales a proseguir por la SEIC destacaría, en primer lugar, el apoyar en la mayor medida posible a sus investigadores jóvenes mediante, por ejemplo, el manteniendo de cuotas muy asequibles para la 15ª reunión nacional de Cuenca (muchas gracias de nuevo, Pepe, por tu entusiasta ofrecimiento a hacernos de cicerone) y la convocatoria de ayudas de viaje tanto para dicha reunión como para la 24ª reunión anual de la ICRS de Baveno, Italia (convocatoria que, como recordaréis, aprobamos en nuestra última Asamblea General). Para ello, y como prioridad asociada, deberemos mantener saneadas las arcas de la SEIC, lo cual dependerá, entre otros factores, del ingreso de ayudas de patrocinadores externos (responsabilidad mayoritariamente de la Junta Directiva) y del pago puntual de las cuotas de socio (responsabilidad de todos y cada uno de nosotros). Nos gustaría también continuar realizando divulgación científica de interés para colectivos como adolescentes, educadores, profesionales sanitarios, pacientes y consumidores, y seguir contribuyendo a apoyar iniciativas encaminadas a evitar recortes al sistema de I+D+i de nuestro entorno.

Como comentamos en la última Asamblea General, creo con franqueza que todos podemos estar contentos de que la SEIC haya conseguido llegar a ser lo que actualmente es. No obstante, esta situación es en cierta manera "asintótica" en cuanto al techo que parecemos haber alcanzado en, por ejemplo, número de socios, número de asistentes a las reuniones anuales y presupuesto disponible para apoyar subvenciones como las mencionadas en el párrafo anterior. Por ello, una cuestión que en la Junta Directiva venimos considerando desde hace años es cómo tratar de ampliar nuestras miras y dotar a la Sociedad de una mayor visibilidad y enjundia. En este sentido han ido dirigidas algunas actuaciones de los últimos años, tales como la asociación a la COSCE y la celebración de nuestra 13ª reunión anual junto con los colegas cannabinólogos italianos (Madrid, 2012), e iría asimismo dirigida la posibilidad discutida en la última Asamblea General de realizar nuestra 16ª reunión anual de 2015 en el marco del congreso bianual de la SENC. Quería informaros que dichas negociaciones con la SENC ya han comenzado y que la Junta Directiva de la SENC ha visto con muy buenos ojos nuestra propuesta, que se basa, por supuesto, en preservar el apoyo incondicional a los investigadores jóvenes de la SEIC (en otras palabras, en mantener para nuestra reunión su formato científico y cuotas de inscripción habituales). El evento se celebraría en Granada o Alicante, dos buenas localizacio-

nes en las cuales nunca hemos organizado, ni creo que vayamos a hacerlo a corto plazo, una reunión de la SEIC, y el acuerdo se limitaría puntualmente al congreso del año 2015.

Para nuestra Junta Directiva, dichas condiciones serían bastante positivas para la SEIC, no solo en lo que respecta a la visibilidad general como Sociedad sino también en lo relativo a todos nuestros socios que deseen asistir a ambos congresos: pago de un solo desplazamiento, mejor aprovechamiento de fechas, posible rebaja en las cuotas de inscripción al congreso de la SENC (aspecto que también estamos negociando), etc. En la pasada Asamblea General, cuando comentamos el asunto, creo que se manifestó (explícita o implícitamente) una opinión favorable a la realización del evento conjunto siempre y cuando se cumplieran las condiciones que os he mencionado antes. No obstante, no se me olvida que Paz, Fran y otros realizasteis algunas acertadas matizaciones que obviamente trataremos de tener en cuenta.

En conjunto, me gustaría pediros (i) vuestra confianza para que desde la Junta Directiva llevemos a cabo las negociaciones con la SENC y (ii) que nos manifestéis por correo electrónico cualquier sugerencia que tengáis con respecto a ese posible evento conjunto de 2015. Creo que huelga decir que los miembros de la Junta Directiva somos los primeros interesados en no malograr lo conseguido hasta ahora en las reuniones de nuestra Sociedad.

Saludos cordiales y (de nuevo) muy feliz año nuevo.

Manuel

2. Premio de la 13ª Reunión anual de la SEIC, Madrid 2012

TARGETED LIPIDOMICS PROFILING OF INJURED RAT BRAIN: *N*-OLEOYL-GLYCINE AND ITS POSSIBLE MECHANISM OF ACTION

Fabiana Piscitelli, Institute of Biomolecular Chemistry, Consiglio Nazionale delle Ricerche, Pozzuoli (NA), Italy

Traumatic brain injury (TBI) is a major cause of mortality and morbidity in the young age people (<40). At present, there are no effective drugs to treat brain trauma, although it is well established that the injury triggers both the accumulation of harmful mediators that lead to secondary damage and the initiation of neuroprotective processes (Mechoulam and Shohami 2007). The secondary damage occurs in the area surrounding the trauma and triggers a cascade of events that lead to the impairment of brain ionic homeostasis and release of glutamate, ROS and inflammatory cytokines (Beit-Yannai, Kohen et al. 1997). There is a large body of evidence showing that the endocannabinoid system is activated in response to pathogenic events, such as brain trauma, suggesting that it participates to compensatory mechanisms of the brain mediated by CB_1 or CB_2 receptors (Mechoulam and Shohami 2007). It was reported that cannabinoid receptor agonists protect cultured rat hippocampal neurons from neurotoxicity, and several groups reported enhanced levels of AEA after acute injury (Mechoulam, Panikashvili et al. 2002). Moreover, in response to TBI, in the closed head injury model (CHI), there is local and transient accumulation of 2-AG at the site of injury, peaking at 4 h and sustained up to at least 24 h (Shohami, Cohen-Yeshurun et al. 2011). More recently, a study from Naqvi and co-workers (Naqvi, Rudrauf et al. 2007) reported that cigarette smokers with brain damage involving the insula, presented a cessation of smoking addiction after TBI. The insula is a brain region involved in conscious urges and several studies showed that exposure to drug-associated cues activate cortical regions, such as the anterior cingulated cortex, the orbitofrontal cortex, and the insula (Naqvi, Rudrauf et al. 2007). Other evidence showed that the right side of the insula is associated with relapse to drug use (Paulus, Tapert et al. 2005). Therefore, giving the role of the endocannabinoid system in controlling the motivational properties and reinforcing effects of nicotine (Scherma, Fadda et al. 2008), this study aims to show that TBI is possibly accompanied by reduced levels of endocannabinoids in the insular cortex. We aim also to discover new endocannabinoid-like molecules associated to TBI, such as fatty acid amides of amino acids (FAAAs). In fact, a study from

Cohen-Yeshurun and co-workers reported the role of *N*-arachidonoyl-L-serine (*N*-AA-Ser) as a new neuroprotective lipid mediator after TBI (Cohen-Yeshurun, Trembovler et al. 2011). It is also possible that this or other endocannabinoid-like mediators might be produced during TBI and act as functional CB_1 antagonists. Therefore, we monitored the levels of endocannabinoids and other endocannabinoid-like molecules in brain areas involved in TBI using the lateral fluid percussion model (LFP).

We showed that comparing the levels of the two endocannabinoids in injured (i.e. ipsilateral) areas only to the corresponding values in sham rats and to those in the contralateral sides of injured rats, and considering the reinforcing role of CB_1 in nicotine addiction, it can be concluded that endocannabinoid tone is clearly decreased in the ipsilateral rat hippocampus and insula following TBI, thus suggesting that this decrease might underlie the decreased nicotine self-administration observed in TBI subjects. Finally, we developed a very sensitive and high resolutive method using the LC-ESI-IT-TOF technique to identify and quantify new endocannabinoid-like molecules involved in this model of TBI (*N*-acyl-serines, *N*-acyl-dopamines and *N*-acyl-glycines). Intriguingly, we reported for the first time that in the prefrontal cortex and hippocampus of the injured hemisphere, trauma is accompanied by a strong elevation of *N*-oleoylglycine (OlGly). The role of this lipid mediator is not known and whether its levels are elevated to afford neuroprotection or to participate in trauma-induced reduction of nicotine craving is currently being investigated. Previously, it has been reported that the arachidonate homolog of OlGly, *N*-arachidonoyl-glycine (NAGly), strongly inhibits recombinant and native T-current in sensory neurons (Barbara, Alloui et al. 2009) and another study showed that T-type calcium channel antagonists have potential for alleviating nicotine addiction by selectively decreasing the incentive motivational properties of nicotine (Uslaner, Vardigan et al. 2010), thus suggesting the opportunity to investigate the effects of other *N*-acylglycines on these channels.

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3. Premio de la 13ª Reunión anual de la SEIC, Madrid 2012

THE ENDOCANNABINOID SYSTEM CONTROLS SKELETAL MUSCLE CELL DIFFERENTIATION VIA CB1 RECEPTOR ACTIVATION

Fabio Iannotti, Institute of Biomolecular Chemistry, Consiglio Nazionale delle Ricerche, Pozzuoli (NA), Italy

In mammals, the ECS regulates a large number of physiological aspects; alterations in its activity are in fact responsible for the onset or progression of many types of disorders affecting both the central and peripheral nervous system as well as other organs (1, 2, 3, 4). To date, few studies have reported that CB1 receptor activity also controls key skeletal muscle metabolic processes such as insulin signalling, glucose uptake and fatty acid oxidation (5, 6). However, little is known about the expression profile and the functional role played by the ECS during skeletal muscle development.

Skeletal myogenesis, is a tightly regulated process that requires coordinated changes of a large number of genes allowing proliferating myoblasts to withdraw from the cell cycle and fuse to form large multinucleated myotubes (7). Using murine C2C12 cells and human primary myoblasts as a experimental paradigms of *in vitro* myogenesis our research group has recently found that the expression of genes involved in the metabolism of the endocannabinoid 2-AG changes during myotube formation, correspondingly 2-AG levels are also decreased. 2-AG, as well as the selective CB1 agonist, ACEA, stimulated myoblast proliferation and prevented myotube formation in a manner CB1-depedend as demonstrated by CB1 knock-down and CB1-selective antagonists, the latter of which instead stimulated differentiation.

We then went on to elucidate the molecular mechanism(s) through which CB1 controls myoblast proliferation and differentiation. CB1 belongs to the metabotropic class of receptors and, although prevalently coupled to $G_{i/o}$ proteins, it can also trigger $G_{q/11}$ activation and subsequent PLC activation (8, 9, 10). Indeed, we found that in C_2C_{12} cells, ACEA reduces the endogenous levels of the PLC substrate, PIP2. Accordingly, we showed that the pro-proliferative effect produced by ACEA on C_2C_{12} myoblasts are abolished by the PLC inhibitor D609, but not by the $G_{i/o}$ selective blocker PTX.

In conclusion, the present study suggests that 2-AG, via CB1 activation, plays a crucial role in the control of myotube formation whilst inducing and/or maintaining myoblast proliferation. This discovery of a novel molecular mechanism by which CB1 regulates skeletal muscle development opens new avenues for endocannabinoid-based therapies against skeletal muscle diseases characterized by abnormal repair and differentiation.

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4. Agenda

ICRS 2014 Symposium
28 de junio-3 de julio de 2014
Baveno, Italia
Más información: http://www.icrs2014.org/

Neuroscience 2014
15-19 de noviembre de 2014
Washington DC, EEUU
Más información: http://www.sfn.org/annual-meeting/neuroscience-2014

15ª Reunión Anual SEIC
27-29 de noviembre de 2014
Cuenca
Más información: http://www.seic.es/

5. Últimas publicaciones sobre cannabinoides de investigadores españoles

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Composición de la Junta Directiva de la SEIC

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Vicepresidente: Julián Romero (Fundación Hospital Alcorcón, Madrid)
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Susana Mato (Universidad del País Vasco)
Juan Suárez (Hospital Carlos Haya, Málaga)
Secretaria: Cristina Sánchez (Universidad Complutense de Madrid)

Dirección de contacto de la SEIC

Sociedad Española de Investigación sobre Cannabinoides (SEIC)
Departamento de Bioquímica y Biología Molecular III
Facultad de Medicina, Universidad Complutense
Ciudad Universitaria, s/n, 28040 Madrid
Teléfonos: 913941450/913941454; fax: 913941691; e-mail: info@seic.es
Dirección Web: http://www.seic.es
Facebook: Sociedad Española de Investigación sobre Cannabinoides-SEIC
Twitter: @SEICannabinoide