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Tel. 5287-3827 Int. 73919 y 4523-6619
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**Diagramación y Diseño:** Andrés Esteban Zapata - aez.sgi@gmail.com

*Química General e Inorgánica. Buenos Aires, Argentina.*
*[2]Universidad de Buenos Aires. Facultad de Farmacia y Bioquímica. Departamento de Ciencias Químicas. Cátedra de Fisicoquímica. Buenos Aires, Argentina.*
*[3] CONICET- Universidad de Buenos Aires. Instituto de Bioquímica y Medicina Molecular (IBIMOL), Buenos Aires, Argentina.*
*[4] CONICET- Universidad de Buenos Aires. Instituto de Biología Celular y Neurociencia "Prof. E. De Robertis", Buenos Aires, Argentina.*

Glaucoma is the first irreversible cause of blindness worldwide and affects eye structures and brain areas related to the visual system. Oxidative stress plays a key role in the development and progression of the disease. The aim was to evaluate the mitochondrial dynamics in the primary visual cortex in a glaucoma model. Three-month Wistar rats were operated by cauterizing two of the episcleral veins in the left eye: glaucoma group (G n=8); the control group (n=8) received a sham procedure. Seven days after surgery rats were euthanized, and the primary visual cortex was dissected. We separated both hemispheres in G, the ipsilateral (GI) and contralateral (GC) (CICUAL FFyB n° 3314). We evaluated OPA-1 and DRP-1 expression in both mitochondrial and cytosolic fractions, and PGC-1α expression in primary visual cortex homogenates. Mitochondrial ultrastructure was studied by transmission electron microscopy (TEM). When compared to control, GC and GI showed an increase of 47% and 58%, respectively, in OPA-1 expression in the mitochondrial fraction ($p<0.01$). However, only GI showed an increase of 41% in OPA-1 expression in cytosolic fraction ($p<0.05$). Regarding DRP-1 expression, only GI cytosolic fraction showed an increase of 22% ($p<0.01$), with no changes in GC and mitochondrial fraction. There were no changes in PGC-1α expression in GC and GI compared to control group. Finally, TEM images showed a slight clarification, swelling and disruption of mitochondrial internal structure in GC and GI compared with control. These results suggest that glaucoma alters mitochondrial dynamics, showing an increase in the fusion process, with no changes in fission process or biogenesis. In addition, mitochondrial ultrastructure is altered in the primary visual cortex. Understanding the key drivers of mitochondrial impairment in glaucoma are crucial to identify new therapeutic targets that would halt disease progression.

**419.** (197) **CORTICOSPINAL AXONS RECONNECTION PROMOTES THE RECOVERY OF VOLUNTARY LOCOMOTION AFTER ACUTE SPINAL CORD INJURY**

Julieta Schmidt[1], Ramiro Quintá[2,3] [1] Facultad de Medicina - UBA.
*[2]Consejo Nacional de Investigaciones Científicas y Técnicas –CONICET; y [3]Laboratorio de Medicina Experimental, Hospital Alemán.*

In traumatic spinal cord injury (SCI), the flux of information along the shaft of long motor and sensory spinal axons is interrupted. The goal is to overcome the deleterious effect produced by SCI promoting re-growth of the damage axons as well as a functional re- connection of its with the lower neuronal targets to achieve a recovery of locomotor functions. According to this, the aim of this research is focused in the design of a therapeutic approach in SCI. Netrin-1, a chemoattractant protein, is involved in axonal growth during the embryonic development. Netrin-1 drives the corticospinal axons growth and its navigation across the pyramidal decussation to the white matter spinal cord by a haptotaxis phenomenon. As I previously described, Netrin-1 promotes a significant recovery of locomotor activity in rats with a complete SCI at Th10 level, assessed by BBB score. Furthermore, this result correlates with a significant improvement in the control of voluntary locomotion, assessed by ladder rung test. In line with this, a stereotaxic surgery was carried out to trace the corticospinal axons. Using the clearing technique it was observed a significant regeneration of corticospinal axons at the lesion site only in Netrin-1 treated rats, as well as a significant preservation in the number of synaptic contacts downstream of the lesion. Besides, an in-vivo trans-synaptic interaction was revealed only in treated rats. Finally, using a 3-Tesla MRI, a preservation of myelinated spinal tissue was shown in Netrin-1 treated rats after SCI. In conclusion, the administration of Netrin-1 in acute SCI promotes regeneration of corticospinal axons, prevents axonal dying back, stimulates neo-formation and re-arrangement of synaptic contacts and preserves the myelinated spinal tissue. All of these cellular processes could partially explain the pathway by which Netrin-1 addition produces a significant recovery of locomotor function after injury.

**420.** (206) **2-ARACHIDONOYLGLYCEROL METABOLISM IS MODULATED BY CANNABINOID RECEPTOR LIGANDS AND BY PHOTOTRANSDUCTION RELATED PROTEINS IN RETINAL ROD OUTER SEGMENTS**

Estefania Chamorro Aguirre, Virginia Lucía Gaveglio, Ana Clara Pascual, Susana Juana Pasquaré
*Laboratorio de Cannabinología, INIBIBB-UNS-CONICET. Dpto. Biología, Bioquímica y Farmacia, UNS, Bahía Blanca, Buenos Aires, Argentina.*

The endocannabinoid 2-arachidonoylglycerol (2-AG) level in the central nervous system is regulated mainly by diacylglycerol- and monoacylglycerol-lipase (DAGL and MAGL) activities. Lysophosphatidate phosphohydrolase (LPAP) could also generate 2-AG from 2-arachinodonoyl lysophosphatidate. Previous studies demonstrated that MAGL activity is modified in rod outer segment (ROS) membranes treated with buffers of low (5 mM Tris-HCl) or moderate (100 mM Tris-HCl) ionic strength, which mostly extract rhodopsin kinase (RK) and arrestin (Arr), both being proteins related to the phototransduction process. One aim of this work was to analyze if cannabinoid receptor ligands WIN55212-2 (WIN), JWH-133 (JWH), SR141716 (SR1) and SR144528 (SR2) modulate these enzymatic activities in ROS isolated from dark-adapted bovine retinas and exposed to light (3000 lux, ROS B) or kept under darkness (ROS O) for 30 min at 37 °C. Thus, ROS O/B were incubated with cannabinoid ligands for 10 min before adding the radiolabeled substrate. It was observed that WIN and JWH agonists (5 $\mu$M) diminished MAG production from LPA by 32% and 60% in ROS O and ROS B, respectively. SR1 and SR2 antagonists (1 $\mu$M) also generated a MAG diminution of 71% and 51% in ROS O, respectively, while both antagonists diminished it by 48% in ROS B (n=5, $p<0.05$). On the other hand, DAGL activity was evaluated in ROS membranes treated at 5 mM or 100 mM in dark or light (3000 lux) for 30 min using radiolabeled diacylglycerol as substrate. DAGL activity diminished by 25% and 55% at low and moderate ionic strength in ROS O, respectively. A diminution of 60% in DAGL activity under both treatments was observed in ROS B (n=4, $p<0.05$). This suggests that RK or Arr modulate DAGL activity. Taken together, these results indicate that 2-AG metabolism is regulated by cannabinoid receptor ligands as well as by the phototransduction process, suggesting an important role of the endocannabinoid system in the visual cycle.

**421.** (325) **THE HYPOTHERMIA MIMETIC SYNTHETIC MOLECULE ZR17-2 PREVENTS RETINAL DAMAGE CAUSED BY PERINATAL ASPHYXIA IN THE RAT**

Ronan Nakamura [1], Manuel Rey-Funes [1], Juan Carlos Fernández[1] Rafael Peláez [2,] Manuel Soliño [1], Daniela Contartese [1], Nicolas Sebastián Ciranna [1], Verónica Berta Dorfman [3], José María Zapico [4], Ana Ramos [4], Beatriz de Pascual-Teresa [4], Juan José López-Costa [1], Ignacio Larrayoz [2], Alfredo Martínez [2,¶] and Cesar Fabián Loidl [1,¶]
*[1]Instituto de Biología Celular y Neurociencia "Prof. E. de Robertis", Facultad de Medicina, Universidad de Buenos Aires, Argentina; [2]Center for Biomedical Research of La Rioja (CIBIR), Logroño, Spain; [3]Centro de Estudios Biomédicos, Biotecnológicos, Ambientales y Diagnóstico (CEBBAD), Universidad Maimónides, Buenos Aires, Argentina; [4]Department of Chemistry and Biochemistry, Facultad de Farmacia, Universidad San Pablo-CEU, CEU Universities, Madrid, Spain.*
*[¶]These authors contributed equally to this study and should be considered co-last authors.*

Introduction. Perinatal asphyxia (PA) is responsible for a large proportion of neonatal deaths and numerous neurological sequelae, including visual dysfunction and blindness. During PA, the retina