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PRE-CONGRESS COURSE "NEUROBIOLOGY OF DRUG ADDICTION"

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October 22nd -23rd, 2018

Ciudad Universitaria, Córdoba, Argentina

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-Salón Auditorio, Edificio Integrador, Facultad de Ciencias Químicas, Universidad Nacional de Córdoba.
-Salón de Actos Pabellón Argentina, Ciudad Universitaria, Córdoba, Argentina

WORKSHOP *Homage to Ricardo Miledi*
"Workshop: Past, Present and Beyond of Synaptic Transmission"

Previous and satellite activity of the XXXIII Annual Congress of the Argentine Society of Neuroscience Research – SAN

October 22th – 23th, 2018 – Instituto Martín y Mercedes Ferreyra, Córdoba

LOCATION:
Instituto de Investigaciones Médicas
Mercedes y Martín Ferreyra (INIMEC)
Ciudad de Córdoba, República Argentina

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P101.-Ceramide induces the death of retina photoreceptors through activation of parthanatos

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Ceramide (Cer) has been proposed as a messenger in photoreceptor cell death in the retina. Here we explored the pathways induced by C2-acetylsphingosine (C2-Cer), a cell permeable Cer, to elicit photoreceptor death. Treating pure retina neuronal cultures with 10 µM C2-Cer for 6 h selectively induced photoreceptor death, decreasing mitochondrial membrane potential and increasing the formation of reactive oxygen species. Noteworthy, the amount of TUNEL-labeled cells and photoreceptors expressing cleaved-caspase 3 remained constant and pretreatment with a pan-caspase inhibitor did not prevent C2-Cer-induced death. C2-Cer provoked polyADP ribosyl polymerase-1 (PARP-1) overactivation. increased polyADP ribose polymer (PAR) levels and induced the nuclear translocation of apoptosis inducing factor (AIF). Inhibiting PARP-1 decreased C2-Cer induced photoreceptor death and prevented AIF translocation. A calpain inhibitor reduced photoreceptor death whereas selective cathepsin inhibitors granted no protection. Combined pretreatment with a PARP-1 and a calpain inhibitor evidenced the same protection as each inhibitor by itself. Neither autophagy nor necroptosis were involved in C2-Cer-elicited death. These results suggest that C2-Cer induced photoreceptor death by a novel, caspase independent mechanism, involving activation of PARP-1, decline of mitochondrial membrane potential, calpain activation and AIF translocation, which are all biochemical features of parthanatos.