

XXXIII
CONGRESO ANUAL
SAN 2018
CORDOBA –
ARGENTINA
24 AL 26 DE OCTUBRE

PRE-CONGRESS COURSE “NEUROBIOLOGY OF DRUG ADDICTION”

SAN IBRO LARC Course and ISN Small Conference (ISN-CC) Associated to the XXXIII SAN 2018 Meeting

October 22nd -23rd, 2018

Ciudad Universitaria, Córdoba, Argentina

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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:

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XXXII Congress of the Argentine Society for Research in Neuroscience

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P105.- α -synuclein overexpression triggers a lipid metabolic switch: lipid droplets as an early marker of neurodegeneration

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Pathological accumulation of α -synuclein (α -syn) is a hallmark of Parkinson 's disease. α -syn is highly expressed in the brain and has the intriguing characteristic of interacting with lipids. However, little is known about its biological role. We demonstrated that α -syn overexpression downregulates neurofilament expression (NF) through the modulation of phosphatidic acid signaling (Conde et al, 2018). Here, we studied lipid metabolism in neuroblastoma cells either stably transfected with pcDNA3 vector (as a transfection control) or pcDNA-WT- α -syn (WT α -syn). WT α -syn neurons displayed an increase in triacylglycerides (TAG) and cholesterol content consequently with lipid droplet (LD) accumulation. α -syn overexpression also triggered SREBP-2 nuclear translocation coincidentally with this lipid metabolic switch. Enhancers of α -syn aggregation (iron, manganese and bortezomib) increased LD content. WT α -syn overexpression also induced Acyl-CoA synthetase activation which explained, at least in part, the increase in TAG, a rather unusual occurrence in healthy neurons. Pharmacological inhibition of TAG synthesis turned the neurons more vulnerable to the presence of WT α -syn. Additionally, NF recovery increased the expression of cleaved caspase 3. In conclusion, α -syn modulates neuronal lipid biology together with the loss of NF as part of a neuroprotective strategy.