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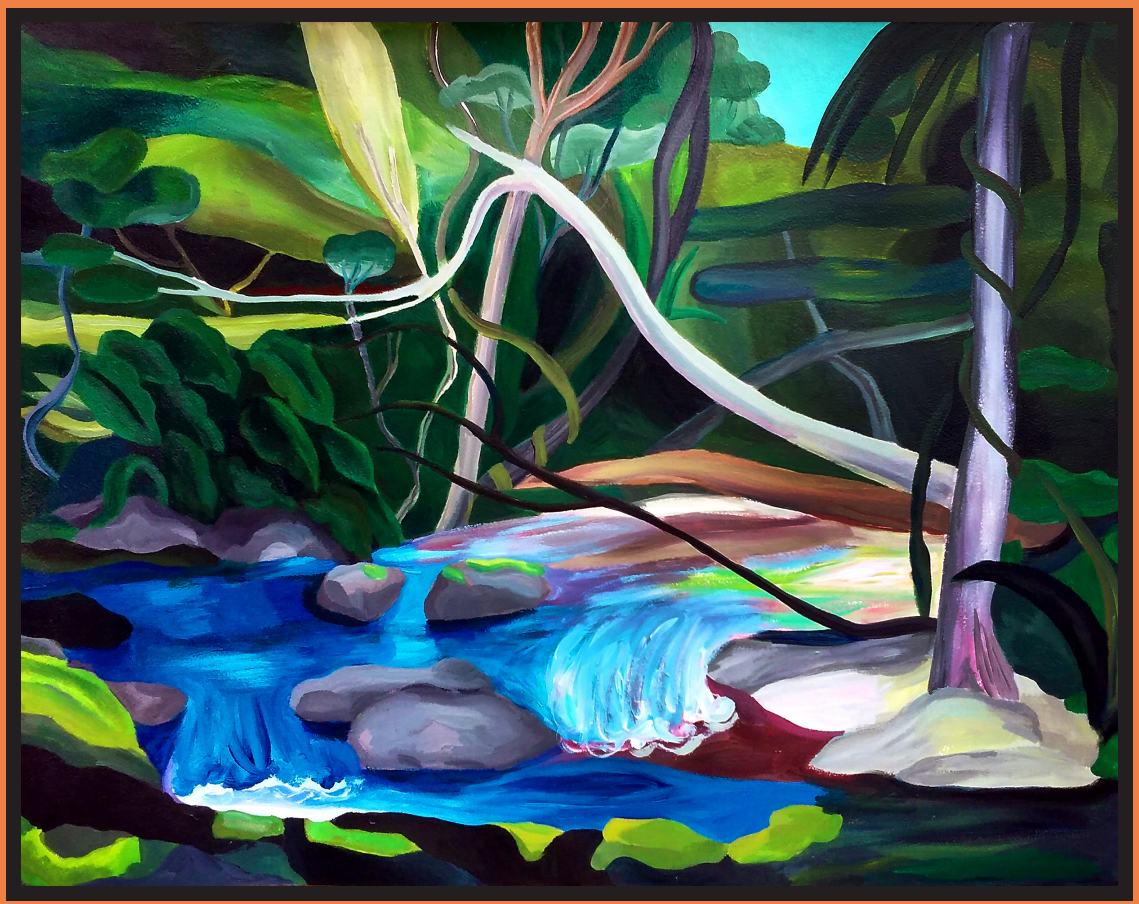

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SOCIEDAD ARGENTINA DE INVESTIGACIÓN CLÍNICA
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XXV JORNADAS ANUALES DE LA SOCIEDAD
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15-17 de noviembre de 2023
Hotel 13 de Julio – Mar del Plata

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JOINT MEETING SAIC SAB AAFE AACYTAL 2023

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HORMESIS MEDIATED BY IL-1b PROTECTS PANCREATIC b-CELLS FROM DYSFUNCTION AND DEATH INDUCED BY INFLAMMATORY CYTOKINES

Carolina Sétula[1,2], Miranda Sol Orellano[1,2], Milagros Argañaras[1], Luz Andreone[1,2], Marcelo Javier Perone[1,2]
1 Laboratorio de Inmuno-Endocrinología, Diabetes y Metabolismo, Instituto de Investigaciones en Medicina Traslacional (IIMT-CONICET-Univ. Austral), Pilar, Argentina. 2 Facultad de Ciencias Biomédicas, Universidad Austral, Pilar, Argentina.

Both type 1 and type 2 diabetes share pancreatic islet inflammation. Hormesis is a phenomenon by which a harmful substance administered to an organism in small doses provides resistance to subsequent contacts with higher doses. We aimed to assess if physiological concentrations of IL-1β induce hormesis leading to adaptive mechanisms, safeguarding β-cells against the characteristic inflammatory environment of diabetes. We used INS-1E rat cells and mouse pancreatic islets and measured NO by Griess, viability (MTT), death (Hoechst/PI, microscopic fluorescence; Annexin V-PE/7-AAD, flow cytometry), mRNA by RT-qPCR, NF-κB (immunofluorescence) and insulin (ELISA). GSIS (glucose-stimulated insulin secretion) index was calculated as the ratio of insulin released during 1h under stimuli of 20mM/2mM glucose. Hormesis was induced by incubation with 10 pg/ml IL-1β for 72h (IL-1β^{low}). Cytokine-induced damage was triggered by 100 pg/ml IL-1β + 5 ng/ml IFNγ for 16h (CYT). Hormesis induced by IL-1β^{low} protects INS-1E from the decrease in mitochondrial reduction potential triggered by CYT, diminishes cell death ($p<0.05$ vs CYT) and improves GSIS ($p<0.05$ vs CYT), the latter in pancreatic islets as well ($p<0.05$ vs CYT). IL-1β^{low} reduces NO production ($p<0.001$ vs CYT) through a decrease in *iNOS* mRNA ($p<0.01$ vs CYT) and its protein expression ($p<0.01$ vs CYT). IL-1β^{low} reduces CYT-triggered NF-κB nuclear translocation ($p<0.05$ vs CYT). IL-1β^{low} hampers the increase in CHOP expression ($p<0.001$ vs CYT), decreases the mRNA expression of *DP5* ($p<0.05$ vs CYT), *PUMA* ($p<0.05$ vs CYT), *Bax/Bcl-2* ratio ($p<0.01$ vs CYT) and caspase-3 ($p<0.001$ vs CYT; by WB). We demonstrate, for the first time, that β-cells are capable of exhibiting IL-1β-mediated hormesis. Interventions aimed at enhancing the hormetic response present a novel therapeutic avenue to strengthen β-cell functionality and viability, thereby mitigating the detrimental inflammatory conditions linked to metabolic disorders such as diabetes.

SAIC AWARD - Eugenia Sacerdote de Lustig – mAbxience - Neurosciences.
Friday 17th November 16:00-18:30
Juries: Diego Gelman; Laura Morelli; Carina Ferrari

LIPID DROPLETS AS YING-YANG MARKERS OF NEURODEGENERATION IN MODELS OF SYNUCLEINOPATHIES

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Alpha-synuclein (aSyn) pathology is a hallmark in the onset and progression of several synucleinopathies, including Parkinson's disease. It has been demonstrated that lipid disturbances are associated with aSyn pathology. Our aim was to study neutral lipid metabolism in several *in vivo* and *in vitro* models of aSyn accumulation. To this end, different forms of aSyn overexpressed in neurons and toxicant-induced animal models related with synucleinopathies were used. In neuronal cultures, we demonstrated that the overexpression of aSyn (WT and mutant A53T) induced the accumulation of lipid droplets (LD) and free cholesterol ($p<0.01$). We found that LD biogenesis is a "hormesis mechanism" for preventing neuronal death induced by proteostasis impairment. Neuron-glia crosstalk was evaluated using neuronal secretomes, demonstrating that WT and A53T neurons exacerbated LD accumulation in glial cells. Moreover, the pesticide maneb triggered ferroptosis associated with aSyn overexpression in neurons with a rise in neutral lipid content ($p<0.05$). These results suggest that altered lipidostasis could be a hallmark of early aSyn-induced neurodegeneration. Mice exposed to neurotoxicants, as maneb and iron overload, showed aSyn upregulation ($p<0.05$) in whole brain and midbrain associated with motor impairment. In addition, the loss of tyrosine hydroxylase neurons in midbrain ($p<0.001$) was related to ferroptosis markers. Midbrain lipid profiles revealed that injured mice presented lipolysis as a consequence of diminished neutral lipid acylation ($p<0.01$) and lipogenesis, and higher cholesteryl ester deacylation rendering cholesterol accumulation ($p<0.05$). Neuronal death and movement disorders are linked with active lipolysis in *in vivo* models. Thus, indicating that marked injury in synucleinopathies is accompanied by impaired LD formation with cholesterol accumulation. Taken together, our results postulate LD as ying/yang markers of different stages of aSyn-induced neurodegeneration.