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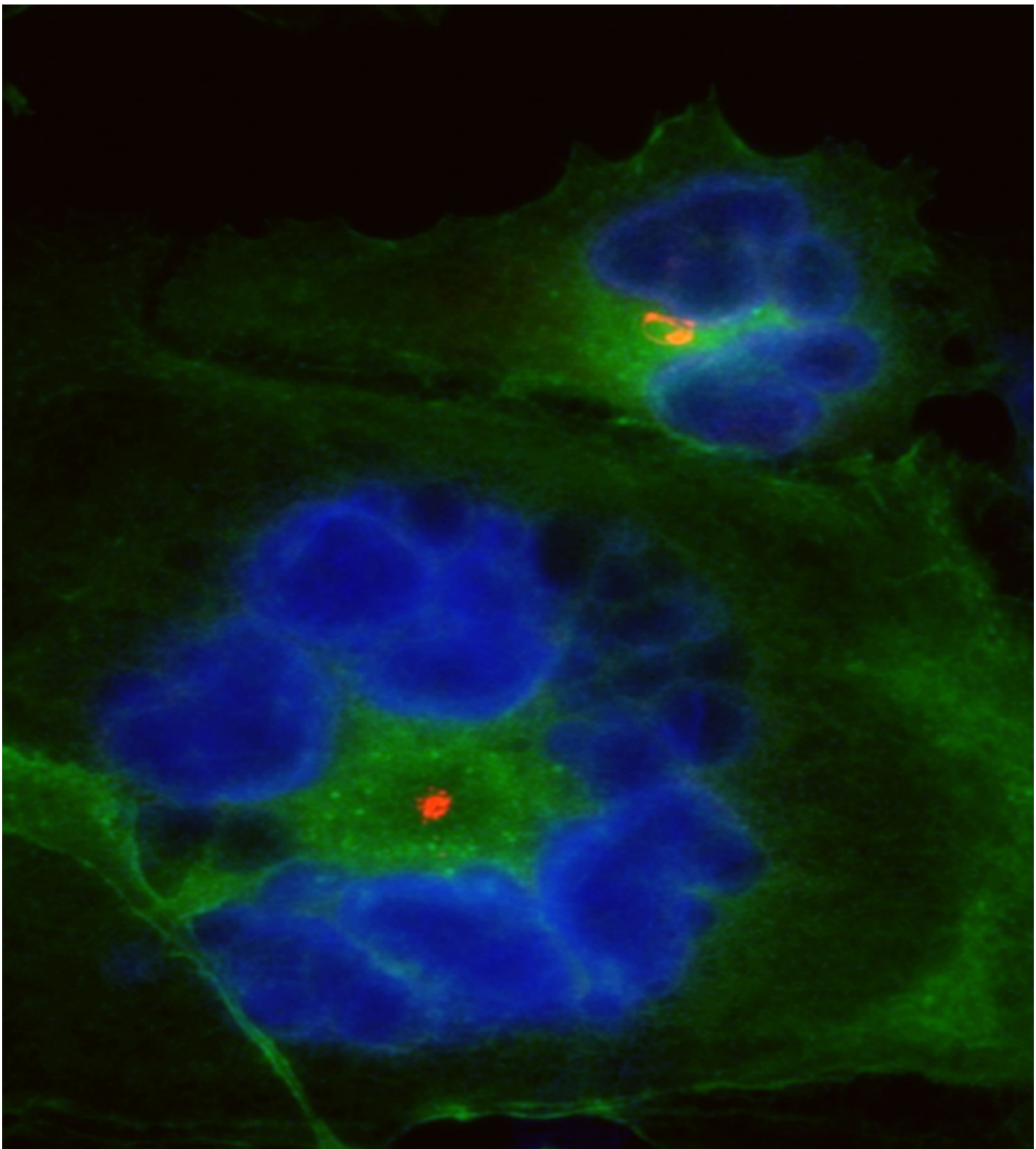

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PABMB EXECUTIVE COMMITTEE

10:05-10:17
BT-C01
IDENTIFICATION AND CHARACTERIZATION OF NOVEL B-GALACTOSIDASES FROM A SEQUENCE-BASED METAGENOME ANALYSIS OF STABILIZATION PONDS
Eberhardt, MF, Irazoqui, JM, Amadio, A. INTA EEA-Rafaela – CONICET.

10:18-10:30
CB-C06
CHMP4B IS REQUIRED FOR THE EFFICIENT REPLICATION OF *TOXOPLASMA GONDII* IN DENDRITIC CELLS
Croce C[1] , Mayorga LS[1] , Blanchard N[2] , Cebrián I[1]. [1]IHEM-CONICET, Facultad de Ciencias Médicas, UNCuyo, Mendoza, ARGENTINA. [2]CNRS-INSERM-Universitéde Toulouse-UPS, CPTP, Toulouse, FRANCIA.E-mail: croce.cristina@gmail.com

ROOM LAPACHO
Chairpersons: Cecilia Casali and Andrea Rópolo

9:00-9:12
LI-C01
α-SYNUCLEIN AND LIPID METABOLISM: INTERSECTING PATHWAYS
Alza, NP[1,2], Conde, MA[1,3], Scodelaro Bilbao PG[3,4], González Pardo V[2], Salvador GA[1,3]. [1]INIBIBB-CONICET, [2]DQ-UNS, [3]DBByF -UNS, [4]CERZOS-CONICET, Bahía Blanca, Argentina.

9:13-9:25
LI-C02
LIPID DROPLETS POPULATIONS IN THE INSECT VECTOR OF CHAGAS DISEASE (*TRIATOMA INFESTANS*)
Girotti JR[1], Borús DL[1], Scelsio NS[1], Favale NO[2,3], Ves-Losada A[1,4]. [1]INIBIOLP-CCT-La Plata-CONICET-UNLP, [2]Cat Biol Cel Mol, FFB, UBA, [3]IQUIFIB-CONICET, [4]Dep. Cs Biol. FCE, UNLP, Argentina.

9:26-9:38
LI-C03
THE REGULATION OF PROTEINS 14-3-3 AND THE HIPPO VIA AFFECT THE ADIPOGENESIS OF 3T3-L1
Del Veliz S[1,3], Uhart M[1], Lim Gareth E[3], Bustos Diego M[1,2], IHEM[1] (CONICET-UNCuyo), FECEN[2], UNCuyo, Argentina. CRCHUM[3], Canada.

9:39-9:51
LI-C05
SPHINGOSINE KINASE 2 AS REGULATOR OF LIPID DROPLETS BIOGENESIS
Santacreu BJ, Romero, DJ, Tarallo E, Otero D, Sterin de Speziale NB; Favale NO. Facultad de Farmacia y Bioquímica, Cátedra de Biología Celular y Molecular, UBA, Argentina. IQUIFIB-CONICET Buenos Aires, Argentina.

Veuthey T, Giunti S, De Rosa MJ, Rayes D.
Instituto de Investigaciones Bioquímicas de Bahía Blanca (INIBIBB) (CONICET–UNS)/ DBByF-UNS. E-mail: tveuthey@uns.edu.ar

The perpetuation of the flight response inhibits defensive cytoprotective mechanisms, leading to reduced resistance to environmental stressors, early onset of age-related disorders and shorter lifespan from invertebrates to mammals. We have recently shown that, in *Caenorhabditis elegans*, the flight response induces the neuronal release of Tyramine (TA, the invertebrate analog of adrenaline), which stimulates the adrenergic-like receptor TYRA-3 in the intestine. This leads to the activation of the DAF-2/Insulin/IGF-1 pathway and the inhibition of cytoprotective mechanisms, such as translocation of DAF-16/FOXO or HSF-1, not only in the intestine but also in other tissues. However, the signals that bridge the stimulation of TYRA-3 in the intestine with the activation of the DAF-2 insulin receptor in other tissues remain unknown. *C. elegans* genome encodes 40 Insulin-like peptides (ILPs), which in principle could bind to DAF-2, and many of them are expressed in the intestine. We, therefore, used RNAi to individually silence intestinal ILPs and test the resistance to environmental stressors such as oxidative and thermal stress. We found that the silencing of one of those ILPS, *ins-3*, improves the resistance to environmental stressors. In contrast to control, the addition of exogenous TA does not impair the oxidative or thermal stress resistance in *ins-3*-silenced animals. Moreover, we generated double null mutants of *ins-3* and TA-deficient mutants and found that this double mutant is as resistant to environmental stress as single mutants. This suggests that tyramine and INS-3 act in the same pathway to control stress resistance. Since *ins-3* is also expressed in neurons, we injected *ins-3* cDNA driven by intestinal and neuronal promoters to *ins-3* null mutant animals, to assess the tissue where the expression of *ins-3* is relevant for controlling stress resistance. We found that only intestinal expression of *ins-3* restores the resistance to wild-type levels. Moreover, we found that the stress resistance of *ins-3 null mutants* is mediated, at least partially, by DAF-16/FOXO. We, therefore, propose that the activation of the intestinal GPCR TYRA-3 by the escape neurohormone TA leads to the release of INS-3 which acts as endocrine, autocrine and/or paracrine signal to activate the insulin receptor DAF-2 not only in the intestine but also in distal tissues. Given the high degree of conservation of fundamental mechanisms among species, this study can contribute to understanding molecular pathways and cellular communication involved in neural regulation of stress response in multicellular organisms.

LIPIDS

LI-C01
α-SYNUCLEIN AND LIPID METABOLISM: INTERSECTING PATHWAYS

Alza, NP[1,2], Conde, MA[1,3], Scodelaro Bilbao PG[3,4], González Pardo V[2], Salvador GA[1,3].
[1]INIBIBB-CONICET, [2]DQ-UNS, [3]DBByF -UNS, [4]CERZOS-CONICET, Bahía Blanca, Argentina. E-mail: natalia.alza@uns.edu.ar

α-synuclein (α-syn) aggregation and fibrillation is a hallmark of a class of neurodegenerative disorders known as synucleinopathies. An intriguing and not completely clarified feature of α-syn is the many ways in which it interacts with lipids. In the present study, we aimed to investigate the effect of α-syn overexpression on neuronal lipid metabolism. For this purpose, human IMR-32 neuroblastoma cells stably transfected with either pcDNA3 vector (control) or pcDNA3-WT-α-syn (WT α-syn) were used. We observed that α-syn overexpression induced the accumulation of cytosolic lipid droplets (LD) and cholesterol (Chol) in lysosomes. LD increase was coincident with a rise in triacylglycerol (TAG) and Chol esters content. To ascertain the mechanism involved in LD accumulation, pharmacological inhibitors of proteasomal degradation and autophagy were used. Whereas autophagy inhibition did not affect neutral lipids content, the blockage of proteasomal degradation was able to increase LD accumulation in WT α-syn cells. *In silico* analysis performed with MyProteinNet server (Yeger-Lotem lab) postulates a positive correlation between α-syn and sterol regulatory element-binding gen (SREBF-2). To corroborate these data in our experimental model, we evaluated the status of the transcription factors SREBP-1 and SREBP-2. SREBP-1 nuclear localization was slightly diminished by α-syn overexpression with decreased levels of fatty acid synthase protein expression. In contrast, α-syn overexpression promoted SREBP-2 nuclear translocation, with no increment in the expression levels of the downstream genes related to Chol synthesis. Intriguingly, fatty acid Coenzyme A esterification and acylation into Chol and diacylglycerides were increased in WT α-syn cells. To elucidate the source of fatty acids availability, we measured phospholipid content and TAG hydrolysis. WT α-syn cells displayed diminished levels of cardiolipin and phosphatidic acid with no changes in TAG hydrolysis. Our results allow us to conclude that: α-syn overexpression induces a metabolic switch that triggers the neuronal accumulation of neutral lipids by activating several mechanisms: (i) increased phospholipid hydrolysis, (ii) a rise in fatty acids esterification into Chol and diacylglycerols, and (iii) Chol accumulation in lysosomes probably due to an increment in its uptake. *Funding: ANPCyT, CONICET, and UNS.*

LI-C02
LIPID DROPLETS POPULATIONS IN THE INSECT VECTOR OF CHAGAS DISEASE (*TRIATOMA INFESTANS*)