LVIII Annual Meeting of the Argentine Society for Biochemistry and Molecular Biology Research

(SAIB)

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Cell Biology Pablo Aguilar CONICET – Universidad de Buenos Aires

> *Lipids* Martín Oresti CONICET - Universidad del Sur

> > Microbiology Hebe Dionisi CONICET

Plants Elina Welchen CONICET – Universidad Nacional del Litoral

> Signal Transduction Graciela Boccaccio CONICET

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PROGRAM AT A GLANCE

	Tuesday 8 th	Wednesday 9 th	Thursday 10 th	Friday 11 th
8:30 - 10:30		Oral Communications "Sala Magna" Microbiology "Sala Plumerillo" Plants "Sala Horcones" Lipids - Neurosciences	Oral Communications "Sala Magna" Plants "Sala Plumerillo" Microbiology "Sala Horcones" Cell Biology	Oral Communications "Sala Magna" Cell Biology "Sala Plumerillo" Plants - Biotechnology "Sala Horcones" Signal Transduction – Enzymes
10:30 – 11:00		<i>"Sala Magna"</i> Round Table Surf your career Coffee break	Coffee break	Coffee break
11:00 - 12:30		<i>"Sala Magna"</i> Plenary Lecture Dr. Ernesto Podesta	<i>"Sala Magna"</i> Plenary Lecture Dr. Mario Feldman	<i>"Sala Magna"</i> Plenary Lecture "Cono Sur" Dr. Rodrigo Gutierrez
12:30 - 14:30		Free time for lunch	Free time for lunch	Free time for lunch
14:30 - 16:30	Registration	Symposium "Sala Magna" Signal Transduction "Sala Plumerillo" Plants "Sala Horcones" Young Investigators	Symposium "Sala Magna" Lipids "Sala Plumerillo" Microbiology "Sala Horcones" Young Investigators	Symposium "Sala Magna" Cell Biology "Sala Plumerillo" Plants "Sala Horcones" Short Plenary Lectures
16:30 – 17:00		Coffee break	Coffee break	Coffee break
17:00 – 19:00	"Sala Magna" Opening Ceremony Plenary Lecture Alberto Sols. Dra. Isabel Varela Nieto	POSTERS (Central Hall)	POSTERS (Central Hall)	POSTERS (Central Hall)
19:00 - 20:30	"Sala Magna" Plenary Lecture Dr. Craig Roy	"Sala Magna" Plenary Lecture Hector Torres Dra. Ana Belén Elgoyhen	"Sala Magna" Plenary Lecture Ranwel Caputto Dra. Alejandra del Carmen Alonso	"Sala Magna" Plenary Lecture Dr Maximiliano Gutierrez
20:30 22:00	Welcome Cocktail Central Hall		SAIB Society Annual Meeting	Awards presentation and Closing Ceremony

of an iron-sulfur cluster-binding domain (2Fe-2S) in the sequence of DGAT3 indicates that this protein could accept electrons, directly or indirectly, from the photosynthetic machinery. The aim of this study was to investigate the relationship between DGAT3 expression and photosynthetic electron transport. With that purpose, we incubated *C. reinhardtii* wild type cc-125 cells with two photosystem II (PSII) inhibitors, 3-(3,4-dichlorophenyl)-1,1-dimethylurea (DCMU) and 2,5-dibromo-6-isopropyl-3-methyl-1,4-benzoquinone (DBMIB), and one photosystem I (PSI) inhibitor, N,N'-dimethyl-4 4'-bipyridinium dichloride (paraquat). Both *Dgat3* mRNAs and TAGs increased in DCMU and DBMIB-treated cells as early as 15 minutes after initiating the experiment, whereas no significant variations were observed in paraquat-treated cells in the course of a 3-h incubation period. Our results suggest that DGAT3 expression and TAG biosynthesis increase when PSII is over-reduced in order to avoid photodamage, as TAG is an adequate molecule to store excess electrons. PSII over-reduction occurs naturally in situations in which light absorption is higher than the rate of photosynthesis (e.g. during illumination at high light intensity) or artificially in chemically-altered PSII centers. Currently, this hypothesis is being evaluated in mutants that have deficiencies in the function of PSI, PSII or the cytb6f complex.

LI-14

NUCLEAR LIPID DROPLETS IN OENOCYTES CELLS FROM TRIATOMA INFESTANS INSECTS ARE A DYNAMIC NUCLEAR ORGANOID

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The objective of this work was to characterize nuclear populations of Lipid Droplets (nLD) in Triatoma infestans (vinchuca) under different development conditions. This hematophage insect is one of the main vectors of the parasite Trypanosoma cruzi, the causative agent of Chagas disease in Argentina and the Americas. The cuticle (C) is the insect most external structure, which protects against physical, chemical (dehydration, etc.) and biological (infections, etc.) external factors. Oenocyte cells (OE) are involved in the anabolism of C hydrophobic molecules (hydrocarbons, alcohols, waxes, glycerides, fatty acids, etc). The fat body (FB) is the organ that regulates the entire insect metabolism. The information on the lipid metabolism of the insect will allow the acquisition of new tools to control the vector. Taking into account the scarce information on OE from C in Triatoma infestans, the aim of the work was to characterized the LD populations in these cells as organoids involved in the genesis of the cuticle. Previously, we demonstrated that in liver, LD populations are dynamic organelle where neutral lipids are stored, mainly located in the cytosol (cLD) and in a small proportion in the nucleus (nLD). For this purpose, protocols were developed and optimized to identify and characterize LD populations in the different cells beneath the cuticle. We examined and characterized the LD populations of OE cells from fifth instar nymphs of the insect that were feed or starved for 1 month. Light field microscopy and fluorescence (epifluorescence and confocal) and hematoxylin / Oil Red and DAPI / BODIPY stains were used, respectively. In OE cells the main LD population is located in the cytosol and a small population within the cell nucleus (nLD) in both conditions, feed and starved insects. These results would confirm the role assigned to OE to actively participate in the anabolism of the cuticle components.

In conclusion, *Triatoma infestans* cuticular oenocytes were characterized as cells that have a very varied morphology, depending on the development state of the insect, and are larger than the surrounding epithelial cells. The OEs have two LD populations in both conditions tested, a main cytosolic and a nuclear one. These are the first results where nLDs are described in insects.

LI-15

PROGRESSIVE ACCUMULATION OF N-9 PUFAS IN TESTICULAR LIPIDS DURING EX VIVO TISSUE MAINTENANCE

Santiago Valtierra, Florencia Ximena; Luquez, Jessica Mariela; Tajes Ardanaz, Oliverio Julián, Torlaschi, Camila; Oresti, Gerardo Martín.

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Spermatogenesis has been achieved in vitro using a gas-liquid interphase culture system. In this setting, it is possible to follow lipid metabolism to know its role during the spermatogenic process, thus gathering potentially useful information for ex vivo spermatogenesis biotechnology. We observed a relationship between the progression of spermatogenesis in both, in vivo and *ex vivo*, at cytological and histological level and the gene expression of enzymes involved in fatty

acid desaturation, elongation and transport. The aim of this study was to examine and extend whether developmental changes that occur in vivo in testicular lipids with long-chain (Cl8-C22) and very long-chain (\geq C24) polyunsaturated fatty acids (PUFA) also occur *ex vivo* in explants of neonatal testes in culture. Explants from 6-day old mice cultured for 22 days showed that differentiation proceed from spermatogonial stem cells up to haploid round spermatids. Notably, after 44 days in culture we were able to detect some spermatozoa, although still scarce. Interestingly, like *in vivo*, total lipids from explants increased their proportion of PUFA during the period in culture. In addition to the common n-6 and n-3 PUFAs (22:5n-6, 22:6n-3, 24:4 and 24:5) we observed that unusual n-9 PUFAs (20:3n-9 and 22:4n-9) were accumulated in the explants. The 22:5n-6/20:4n-6 ratio in the glycerophospholipids was increased during the first 22 days associated to the appearance of haploid spermatids and then it remains unchanged until day 44. In addition, we noted that in culture the testicular tissue accumulated neutral lipids, mainly, triacylglycerides (TAG) and cholesterol esters (CE) that contained a high proportion of n-9 PUFAs. *In vivo*, the increase of neutral lipids occurs in testicular tissue associated with spermatogenesis impairment. The presence of the unusual fatty acids of the n-9 serie in the explants suggests that the culture system does not ensure the provision of essential fatty acids to the tissue. Addressing this point could improve the rate of gamete production in this system. Supported by SGCyT UNS-PGI-UNS [24/B272 to GMO], FONCyT, [PICT2017-2535, PICT2020-02056 to GMO].

LI-16

HDAC2 MEDIATED REPRESSION OF BDNF IN OLD HIPPOCAMPUS TRIGGERED BY CHOLESTEROL LOSS

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Aging is associated to epigenetic alterations which lead to diminished expression of memory-related genes. One of the main alterations in the aging brain is cholesterol loss. This lipid is capable of interacting both with sphingolipids and proteins and thus play a key role in Membrane Lipid Rafts formation (MLR), key structures for proper function of some receptors such as the NMDAR. The cholesterol loss during aging reduces MLR formation and impairs proper NMDAR activation and synaptic-activity dependent transcription of memory-genes. Contributing to this process, a decrease in histone residues acetylation due to Histone Deacetylase 2 (HDAC2) accumulation has been observed. This particular event is determinant for memory loss during aging. In this work, we found that aging triggers the accumulation of HDAC2 in promoters II and VI of the BDNF gene, a key transcription factor for synaptic plasticity, learning and memory formation. We found that the transcriptional co-repressor Chromodomain Y like protein (CDYL), which interacts with HDAC2 in hippocampal extracts, is accumulated in the nucleus of old neurons. In addition, the co-accumulation of CDYL and HDAC2 was observed in neurons of transgenic Thy-1(GFP) mice brain slices and after cholesterol oxidase treatment in 14-DIV rat hippocampal neurons. Taking into account that has been reported that CDYL degradation is triggered by synaptic activity, and we observe a decrease in CDYL mean fluorescence intensity after NDMA stimulation, we propose that CDYL accumulation can occur as a consequence of impaired NMDA receptor activation due to reduced MLR formation. The findings of this work contribute to the understanding of the epigenetic mechanisms underlying synaptic impairment during aging.

Signal Transduction

ST-05

STUDY OF PDK1 REGULATION BY METABOLITES AND INTERACTION WITH KINASE SUBSTRATES

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