

# **LX Annual Meeting** of the Argentine Society for Biochemistry and Molecular Biology Research (SAIB)

Del 5 al 8 de noviembre de 2024  
Pabellón Argentina de la UNC,  
Ciudad de Córdoba, Argentina.



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**Dr. Andrés Dekanty**

*Signal Transduction*

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## PROGRAM AT A GLANCE

### TUESDAY 5/11

**14.30 h - 16.30 h. Accreditation**

**16.30 h - 16.45 h. Opening Ceremony** (Sala de las Américas)

**16.45 h - 18.00 h. "IUBMB" Plenary Lecture**

Dr. Raphael Valdivia (Sala de las Américas)

**18.00 h - 19.15 h. "Alberto Sols" Plenary Lecture**

Dr. Juan Hermoso (Sala de las Américas)

**19:15 h - 19:45 h. Celebration 60th Anniversary of SAIB Congress**

(Sala de las Américas)

**20.00 h - 22.00 h. Welcome Cocktail** (Hall Central)

### WEDNESDAY 6/11

**08.30 h - 10.30 h. Oral Communications**

**Microbiology/Cell Biology** (Sala de las Américas)

**Lipids** (Salón de Actos)

SSOR

**10.30 h - 11.00 h. Coffee-Break**

**11.00 h - 12.15 h. Plenary Lecture**

Dr. George O'Toole (Sala de las Américas)

**12.15 h - 14.30 h. Lunch Time**

**14.30 h - 16.30 h. Symposia**

**Microbiology** (Sala de las Américas)

**Lipids** (Salón de Actos)

**Methodological advances** (Auditorio Rébora. Facultad de Arquitectura)

**16.30 h - 17.00 h. Coffee-Break**

**17.00 h - 18.15 h. Plenary Lecture**

Dr. Gabriela Salvador (Sala de las Américas)

**18.15 h – 20.00 h. POSTERS Microbiology (MI), Lipids (LI), Biotechnology (BT), Enzymology (EN).**

**19.00 h – 22.00 h. SAIB Assembly** (Sala de las Américas)

## THURSDAY 7/11

**08.30 h – 10.30 h. Oral Communications**

**Plants** (Sala de las Américas)

**Signal Transduction** (Salón de Actos)

SSOR

**10.30 h – 11.00 h. Coffee-Break**

**11.00 h – 12.15 h. “Ranwel Caputto” Plenary Lecture**

Dr. María Fabiana Drincovich (Sala de las Américas)

**12.15 h – 14.30 h. Lunch Time**

**14.30 h – 16.30 h. Symposia**

**Plants** (Sala de las Américas)

**Signal Transduction** (Salón de Actos)

**Young Investigators I** (Auditorio Rébora. Facultad de Arquitectura)

**16.30 h – 17.00 h. Coffee-Break**

**17.00 h – 18.15 h. “PABMB” Plenary Lecture**

Dr. Hailing Jin (Sala de las Américas)

**18.15 h – 20.00 h. POSTERS Plants (PL), Signal Transduction (ST), Structural Biology (SB).**

## FRIDAY 8/11

**08.30 h – 10.30 h. Oral Communications**

**Cell Biology** (Sala de las Américas)

**Plants/Biotechnology/Structural Biology/Neurosciences** (Salón de Actos)

**10.30 h – 11.00 h. Coffee Break**

**11:00–12:15. Plenary Lecture**

**Plenary Lecture**

Dr. María Castro (Sala de las Américas)

**12.15 h – 14.30 h. Lunch Time**

**14.30 h – 16.30 h. Symposia**

**Cell Biology** (Sala de las Américas)

**Molecular Biology of Cancer** (Salón de Actos)

**Young Investigators II** (Auditorio Rébora. Facultad de Arquitectura)

**16.30 h – 17.00 h. Coffee-Break**

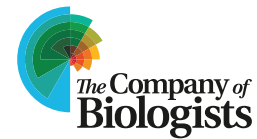
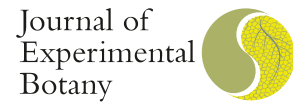
**17.00 h – 18.45 h. POSTERS Cell Biology (CB), Neurosciences (NS).**

**19.00 h – 20.15 h. “Héctor Torres” Plenary Lecture**

Dr. Juan Bonifacino (Sala de las Américas)

**20.15 h–21.00 h. Awards and Closing Ceremony Room** (Sala de las Américas)

This meeting was supported by:



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secretome from astrocytes exposed to the pesticide downregulated *Pla2g2d* expression, indicating that when neurons received DHA exogenously, the enzyme was downregulated. In addition, the inhibition of secretory PLA2 prevented the astrocytic proliferative effect elicited by neurons. Our results argue in favor of the relevance of resolution pathways involving sPLA2-IIID acting as a modulator of DHA levels in the glial response against the pesticide.

## LI-2

### UNSATURATED FATTY ACIDS ARE KEY MOLECULES TO ENSURE REPRODUCTION IN *CAENORHABDITIS ELEGANS*

*Battista B*<sup>1,2</sup>, *Hernández-Craverro B*<sup>1</sup>, *Láscarez Lagunas L*<sup>5</sup>, *Cochella L*<sup>4</sup>, *Binolfi A*<sup>1,3</sup>, *Colaiácovo M*<sup>5</sup> and *de Mendoza D*<sup>1</sup>

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Lipids perform a wide variety of functions in the cell, from optimal membrane fluidity to energy homeostasis as a result of their highly reduced state<sup>1</sup>. Although all living organisms must produce thousands of distinct lipids, a unique aspect of *Caenorhabditis elegans* metabolism is the ability to synthesize a wide range of polyunsaturated fatty acids (PUFAs). The pathway for unsaturated fatty acids (UFAs) synthesis in *C. elegans* begins with the desaturation of palmitic acid (16:0) to palmitoleic acid (16:1) or the elongation of 16:0 to stearic acid (18:0). The last one is further desaturated to oleic acid (18:1), that in the nematode is further used to form all the PUFAs. The biosynthesis of 16:1 and 18:1 requires three  $\Delta^9$  desaturases named FAT-5, FAT-6 and FAT-7. Given the essentiality of UFAs, the *fat-6, fat-5, fat-7* triple mutant strain is lethal. Thus, using CRISPR-Cas9 technology we have constructed a conditional *fat-7* mutant in a *fat-6, fat-5* background. This strain allows the inducible suppression of FAT-7 activity in living worms. By applying a combination of GC-MS and NMR we have demonstrated the specificity of the degradation system and corroborated that the reduction in total UFA content is due to a decrease in PUFA levels rather than MUFA content. We have characterized the phenotypical consequences of total UFAs deficiency in nematode's growth and fertility. We also explored the potential causes behind the reduced fertility employing DNA staining, immunohistochemistry, phenotype characterization and super resolution microscopy. We have noted that the reduction in UFA content induces gonad shrinkage and disorganization of germline cells within the gonad arm. In addition, we have also found that suppression of FAT-7 activity leads to mitotic quiescence and impairs DNA synthesis. By scoring specific meiosis markers, we have demonstrated that UFA deficiency also alters the normal meiotic progression of the germline. However, this alteration of lipid metabolism does not induce higher apoptotic rates. These results suggest the fertility defects exhibited are caused by a reduction in the germ cell production rather than an increased cellular death. The findings presented here are expected to contribute to a better understanding of the role of UFAs in complex processes such as reproduction.

## LI-3

### ALPHA-SYNUCLEIN AND SCHWANN CELL DIFFERENTIATION: A POTENTIAL CROSSTALK WITH LIPID METABOLISM?

*Conde MA*<sup>\*1,2</sup>, *Aparicio GI*<sup>\*3</sup>, *Andersen N*<sup>1,2</sup>, *Quintero J*<sup>3</sup>, *Gerhardt G*, *van Horne C*<sup>3</sup>, *Monje P*<sup>#3</sup>, *Salvador GA*<sup>#1,2</sup>

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Alpha-synuclein (aS) is a moonlighting protein involved in multiple cellular events. This protein has been intensively studied in central nervous system (CNS) neurons in relationship to the pathogenicity of Parkinson's disease (PD). Curiously, aS is also expressed in the peripheral nervous system (PNS) specifically in the cytoplasm of myelinating glial cells or Schwann cells (SCs). In fact, the localization of the aS protein in the normal and parkinsonian PNS follows a pattern similar to the one of S100 $\beta$ , a ubiquitous mature SC-specific marker. However, the role of aS in SCs is not known. Therefore, we took advantage of available RNAseq datasets from human nerve tissues and cultured purified SCs to investigate the relationship between aS expression and SC differentiation. Intriguingly, purified human and rat SCs induced to differentiate in vitro showed a rapid increase in aS gene expression (*SNCA*) concomitantly with the upregulation of myelination-associated genes such as *MPZ* and *MBP*. Transcriptomics and immunostaining analysis of donor-matched transected sural nerve from participants of a nerve transplantation trial (NCT02369003) showed that the levels of aS mRNA and protein were reduced in dedifferentiating (injury-induced) SCs similar to myelin-specific genes. We previously described an unexpected role for aS overexpression as a modulator of neuronal lipid metabolism. aS overexpression in neurons triggered lipid droplets and free cholesterol accumulation. Because myelin is mostly composed of lipids (40 % cholesterol and 40 % phospholipids), we then focused on the identification of lipid metabolism-associated genes. As expected, SC differentiation in vitro was accompanied by the increased expression of

the transcription factor *MAFI*, which is necessary for cholesterol synthesis. Similar to aS, different isoforms of the cholesterol transporter *ABCA* and the Acetyl-CoA carboxylase gene (*ACACB*), the rate-limiting step for fatty acid synthesis, were upregulated in differentiated SCs once again highlighting the close relationship between aS and myelin gene expression. Concomitantly, *ABCA* transporters and *LPINI*, the rate-limiting step enzyme of the Kennedy pathway, were reduced in injured nerves. The transcriptomics and immunochemical data analyzed here highlighted the importance of aS as a novel biomarker of the differentiated SC phenotype while raising the interesting hypothesis that its action may be linked to lipid metabolism, as shown previously in the CNS. Further biochemical and cell biology studies are necessary to challenge this hypothesis and explore the biological role of aS in the SC's lipidome.

#### LI-4

### **SILENCING OF CYP4G GENE AND ITS EFFECT ON SURVIVAL, REPRODUCTION AND CUTICULAR RESISTANCE TO INSECTICIDES IN *TRITOMA INFESTANS*.**

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The lipids that cover the insect cuticular surface play an essential role on their physiology and survival. They comprise a complex mixture of non-polar long and very long chain compounds, where hydrocarbons (HC), fatty alcohols, waxes, glycerides, and free fatty acids usually prevail. The large and diverse cytochrome P450 (CYP) gene superfamily encodes enzymes with a wide range of activities. The CYP enzymes from the 4G subfamily (CYP4G) catalyze the final step of hydrocarbon synthesis in insects. HC have multiple functions, being involved in desiccation resistance, chemical communication, a reduced penetration of insecticides and pathogenic microorganisms and other essential roles for insect survival. To date, at least one CYP4G gene has been found in all insect genomes, and two CYP4G genes (CYP4G106 and CYP4G107) have been described in the two major vectors of Chagas Disease, *Triatoma infestans* and *Rhodnius prolixus*. In the latter, it was demonstrated that the CYP4G106 gene is mostly involved in the formation of linear HC, while the CYP4G107 gene in that of methyl-branched HC. In order to expand the knowledge about the many roles that CYP4G genes may have in the physiology of *T. infestans*, their CYP4G106 and CYP4G107 genes were silenced by RNA interference and the effect on the cuticle hydrocarbon content, molting, survival, mating behavior, reproductive parameters and insecticide resistance was subsequently analyzed. The hydrocarbon profile showed a significant decrease in the content of both linear and methyl-branched HC in the CYP4G106+CYP4G107-silenced insects. However, that decrease was higher for linear HC than for methyl-branched as the silencing of CYP4G106 led to an unexplained overexpression of CYP4G107 gene, similarly to that previously shown in *R. prolixus*. Silencing of the CYP4G genes reduced significantly the proportion of fifth-stage nymphs that underwent metamorphosis but did not affect the molting of fourth-stage nymphs to the next stage. CYP4G silencing showed varying results on insect survival; it reduced significantly the survival of CYP4G106+CYP4G107-silenced adults and that of CYP4G107-silenced fifth stage nymphs but not that of CYP4G106-silenced ones. In contrast, it did not have any significantly effect in the molting time, the mating attempts between CYP4G-silenced females and control males, or the female/male ratio. Reproduction was strongly affected as CYP4G106+CYP4G107-silenced females laid significantly fewer eggs than control. Regarding insecticide resistance, CYP4G106-silenced fifth-stage nymphs showed to be significantly more susceptible to deltamethrin than control nymphs. Overall, our results highlight the relevant role that HC play in egg viability and in the formation of a cuticle barrier that avoids water loss, thus increasing insect survival, and diminish the penetration of insecticides in *T. infestans*.

#### LI-5

### **THE PALMITOYLPROTEOME AND THE PROXITOME OF PALMITOYLTRANSFERASES IN *SACCHAROMYCES CEREVISIAE***

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Protein S-acylation, commonly known as palmitoylation, involves the covalent attachment of long-chain fatty acids, typically palmitate, to cysteine residues in proteins. It is one of the most frequent protein post-translational modifications in eukaryotes and influences subcellular localization, trafficking, and molecular interactions of its substrates. Mutations in the enzymes catalyzing this reaction, named palmitoyltransferases (PATs), have been linked to numerous pathologies such as intellectual disability, schizophrenia, and various types of cancer. Despite its significance, many unresolved questions remain. Notably, the palmitoylproteome appears incomplete, and the mechanisms regulating PAT trafficking, substrate specificity, and activity are not fully characterized. To address these