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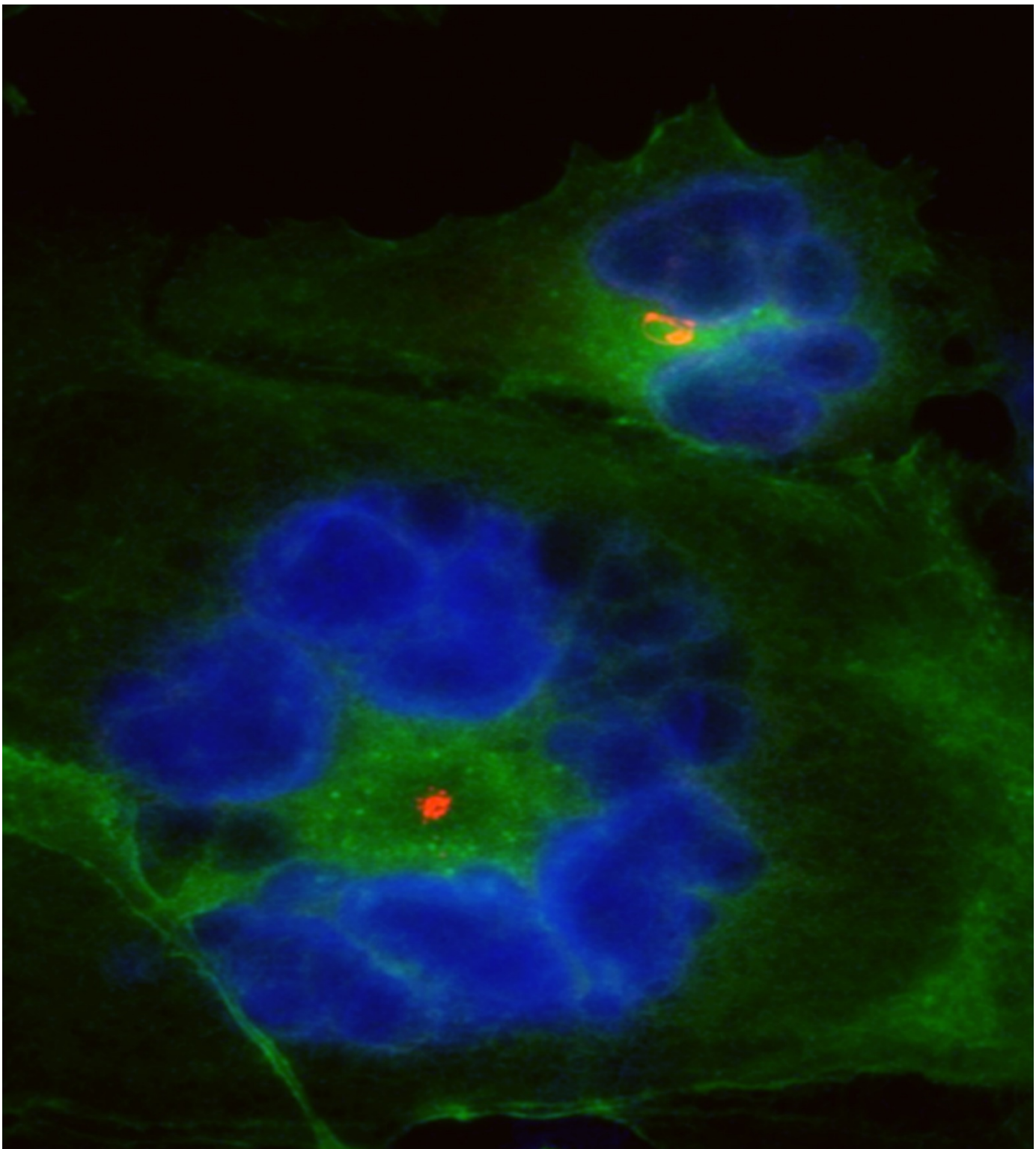

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PABMB EXECUTIVE COMMITTEE

Schedule	Tuesday November 5			Wednesday November 6	Thursday November 7	Friday November 8
8:30-9:00	**WORKSHOPS** **Workshop Accreditation**					
9:00-11:00	**Biochemistry Education Workshop**	**1st Workshop On Drug Discovery**		**Oral Communications** **Room Jacaranda** PL-C01, PL-C02, PL-C04 to PL-C06, PL-C09, PL-C12, ST-02, BT-C02 **Room Los Ceibos** CB-C01 to CB-C04, CB-C07 to CB-C10, ST-C01	**Conferences** ***Ro*om Lapacho** *Robert Gennis* *Francisco Barrantes*	**Oral Communications** **Room Jacaranda** PL-C03, PL-C07, PL-C08, PL-C10, PL-C11, PL-C13, PL-C14, PL-C15 **Room Los Ceibos** MI-C01 a MI-C06, BT-C01, CB-C06 **Room Lapacho** LI-C01 to LI-C05, ST-C03, CB-C11, CB-C05
11:00-11:30				**COFFEE-BREAK**		
11:30-12:30				**Plenary lecture** *Bruno Amati* **Room Lapacho**	**IUBMB Jubilee Lecture** *Philip D. Stahl* **Room Lapacho**	**"Hector Torres" Plenary Lecture** *Alejandro Colman Lerner* **Room Lapacho**
12:30-14:30				**LUNCH TIME**		
14:30-16:30			**Congress Accreditation**	**Symposia** *Lipids* **Room Jacaranda** *Plants* **Room Los Ceibos** *Signal Transduction* **Room Lapacho**	**Symposia** *Cell Biology* **Room Jacaranda** *RNA* **Room Los Ceibos**	**Symposia** *Microbiology* **Room Los Ceibos** *PABMB Young Investigators* **Room Jacaramda**
16:30-17:00				**COFFEE –BREAK**		
16:30-18:30			**Opening Ceremony** **Room Lapacho** ***In memoriam* of** Horacio Pontis **Room Lapacho**	**POSTERS** BT-P01 to BT-P06 CB-P01 to CB-P15 MI-P01 to MI-P18 PL-P01 to PL-P15 ST-P01 to ST-P13	**POSTERS** BT-P07 to BT-P12 CB-P16 to CB-P31 EN-P01 to EN-P11 MI-P19 to MI-P37 PL-P16 to PL-P32	**POSTERS** BT-P13 to BT-P19 CB-P32 to CB-P47 LI-P01 to LI-P15 MI-P38 to MI-P49 PL-P33 to PL-P48
18:30-19:30			**"Alberto Sols" Plenary Lecture** **Room Lapacho** *Encarnación Martinez Salas*	**EMBO Keynote Lecture** *F. Gisou van der Goot* **Room Lapacho**	**"Ranwel Caputto" Plenary Lecture** *Maria Elena Alvarez* **Room Lapacho**	**Closing Ceremony** **Room Lapacho**
			Cocktail 20:00 hs		**SAIB Assembly 19:45 hs**	**Dinner 20:00 hs**

this work, our aim was to study the involvement of PLA2s in the regulation of α-syn biology and the neuronal redox response to Fe overload. We also investigated the role of glia-secreted factors in the neuronal outcome. For this purpose, we exposed human neuroblastoma cells (IMR-32) to different ferric ammonium citrate concentrations (300–1000 μM) or vehicle for different incubation times (24–72 h). Using these experimental conditions, redox status, α-syn expression and phosphorylation, and the participation of calcium-independent and calcium-dependent PLA2 isoforms (iPLA2 and cPLA2, respectively) in the regulation of these events were studied. IMR-32 neurons exposed to Fe overload showed increased expression levels of iPLA2, concomitantly with an increase in lipid peroxides and reactive oxygen species. The pharmacological blockage of iPLA2 activity increased, even more, the levels of lipid peroxides and the content of reactive oxygen species. On the contrary, the inhibition of cPLA2 showed the opposite effect by promoting a decrease in oxidative stress markers associated with increased neuronal viability. Fe-challenged neurons also displayed increased α-syn expression and phosphorylation. The phosphorylation of α-syn was blocked by the inhibition of iPLA2 activity. To study the role of glia in the neuronal response to Fe, C6 astroglioma cells were challenged with ferric ammonium citrate or vehicle, and the astrocyte-derived media were added to neuronal cultures. Astrocytes exposed to Fe showed an increase in the glial marker S100B and lipid peroxidation levels, thus indicating reactivity to oxidative stress. Neurons incubated with the mentioned astrocyte-derived media displayed lower levels of oxidative injury than neurons only exposed to Fe. Astrocytes were positive for the rate-limiting step enzyme for glutathione biosynthesis. Altogether, our results show specific roles for the different PLA2 isoforms in the neuronal response to Fe-induced injury: whereas iPLA2 showed to be neuroprotective and also to be involved in the regulation of α-syn phosphorylation, cPLA2 appeared to act as a damage promoter. To ascertain the nature of the effect exerted by astrocytes on the neuronal response to oxidative stress, we are currently studying glutathione synthesis and how the isoform-specific PLA2-inhibition could be involved. *Sponsored by FONCyT, CONICET, UNS.*

LI-P04
SUPPLEMENTATION OF *EX VIVO* MOUSE TESTES EXPLANTS WITH PUFA-RICH LIPIDS STIMULATES SPERMATOGENESIS

Luquez, JM[1], Santiago Valtierra FX[1], Isoler-Alcaraz J[2], Aveldaño MI[1], del Mazo J[2], Oresti GM[1]
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Using a gas-liquid interphase culture system from neonatal mouse testes, we previously observed in *ex vivo* explants a relationship between the progression of spermatogenesis at both cytological and histological levels and the gene expression of some of the enzymes involved in lipid metabolism. Here, we examined by RT-qPCR the expression of two PUFA elongases (*Elovl2* and *Elovl4*), Δ6-desaturase (*Fads2*), fatty acid 2-hydroxylase (*Fa2h*), two fatty-acid-binding proteins (*Fabp3* and *Fabp9*) and a diacylglycerol acyltransferase (*Dgat2).* Testis explants from 6 days old mice cultured for 22 days evidenced progress in spermatogenesis beyond the meiotic phase in some of the tubules. Although delayed *in vitro* in comparison with the *in vivo* development, in both cases the appearance of haploid germ cells occurred concomitantly with an increase in the expression of *Fabp9, Dgat2*, and *Fa2h*. Interestingly, the genes involved in PUFA synthesis (*Elovl2*, *Elovl4*, *Fads2*) and transport (*Fabp3*) were up-regulated in the testicular explants in comparison with the *in vivo* situation. This suggested, as a possible cause, partial insufficiency in the culture system of the C20-C22 PUFA required as substrates by these biosynthetic enzymes. This proved to be the case, as this medium contained low proportions (less than 4%) of these fatty acids. Supplementation of *ex vivo* explants with a PUFA-rich total lipid extract (TL) from adult mouse testis allowed progression into meiosis at the times in culture examined. Moreover, after 22 days in culture, the TL-supplemented explants contained more tubules with spermatogenic cells that had succeeded to reach the spermatid stage. Thus, in addition to growth factors and hormones, influences that promote the biosynthesis of PUFA-containing lipids are among the factors required to optimize spermatogenesis in *ex vivo* tissue explants. *Supported by FONCyT [PICT2017-2535] and PGI-UNS [24/B272] to GMO and by the MCIyU, Spain [BFU2017–87095-R] to JdM.*

LI-P05
IMPLICATION OF SPHINGOLIPIDS IN EPITHELIAL–MESENCHYMAL TRANSITION PROCESS IN RENAL COLLECTING DUCTS OF AGED RATS

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The epithelial–mesenchymal transition (EMT) is a process in which the cells lose their epithelial phenotype and acquire the characteristics of mesenchymal cells, which includes loss of cell–cell binding. Renal function declines progressively with age, and the EMT process has been suggested as a mechanism that drives renal fibrosis, with the consequent loss of tissue functions, which occurs mainly in old age. In previous works, we demonstrated that the inhibition of sphingomyelin synthase 1—the enzyme responsible for the synthesis of sphingomyelin (SM) at the Golgi Ap level—induces an EMT process in CD cells from the renal papilla of young, 70-day-old rats. We also demonstrated that the EMT occurs spontaneously in renal papillary CD cells of middle-aged rats (6–8 months), denoted by an impairment of cell–cell adhesion, a higher number of CD cells expressing the mesenchymal protein vimentin, and the *de novo* synthesis of α-smooth muscle actin (α-SMA), another mesenchymal biomarker. These results motivated us to study the possible implication of sphingolipids, and in particular SM, in the occurrence of EMT in renal papilla CD cells during aging. Taking into account that the cells in culture behave as in intact tissue, primary cultures of CD cells isolated from the renal papilla of young and middle-aged rats were performed. Since the occurrence of the EMT process was observed in 6-month-old rats, we performed a recovery experiment using the exogenous addition of 10 μM C12-SM to primary cultured CD cells from middle-aged rats. For this purpose, we simultaneously evaluated the intercellular adhesions by α- catenin immunostaining, and the expression of the mesenchymal biomarker α-SMA. After the addition of exogenous SM, the intercellular spaces between the CD cells disappeared, and α- catenin lined the lateral cell membranes, reflecting the presence of mature adherens junctions. Moreover, the percentage of CD cells that express α-SMA decreased (young vs middle-aged, $p = 0.0006$). We also analyzed the total SM content in CD cells isolated from young, middle-aged and aged-rats (15 months old) by thin-layer chromatography (TLC) and densitometry. Surprisingly, although we observed an EMT in CD cells from middle-aged rats, the quantitative results showed a decrease in SM content only in CD cells isolated from the renal papilla of aged rats (young vs aged-rats, $p = 0.0030$). Taking into account our previous and present results, we conclude that the epithelial-mesenchymal phenotypic conversion that spontaneously