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SAIB - SAMIGE Joint meeting
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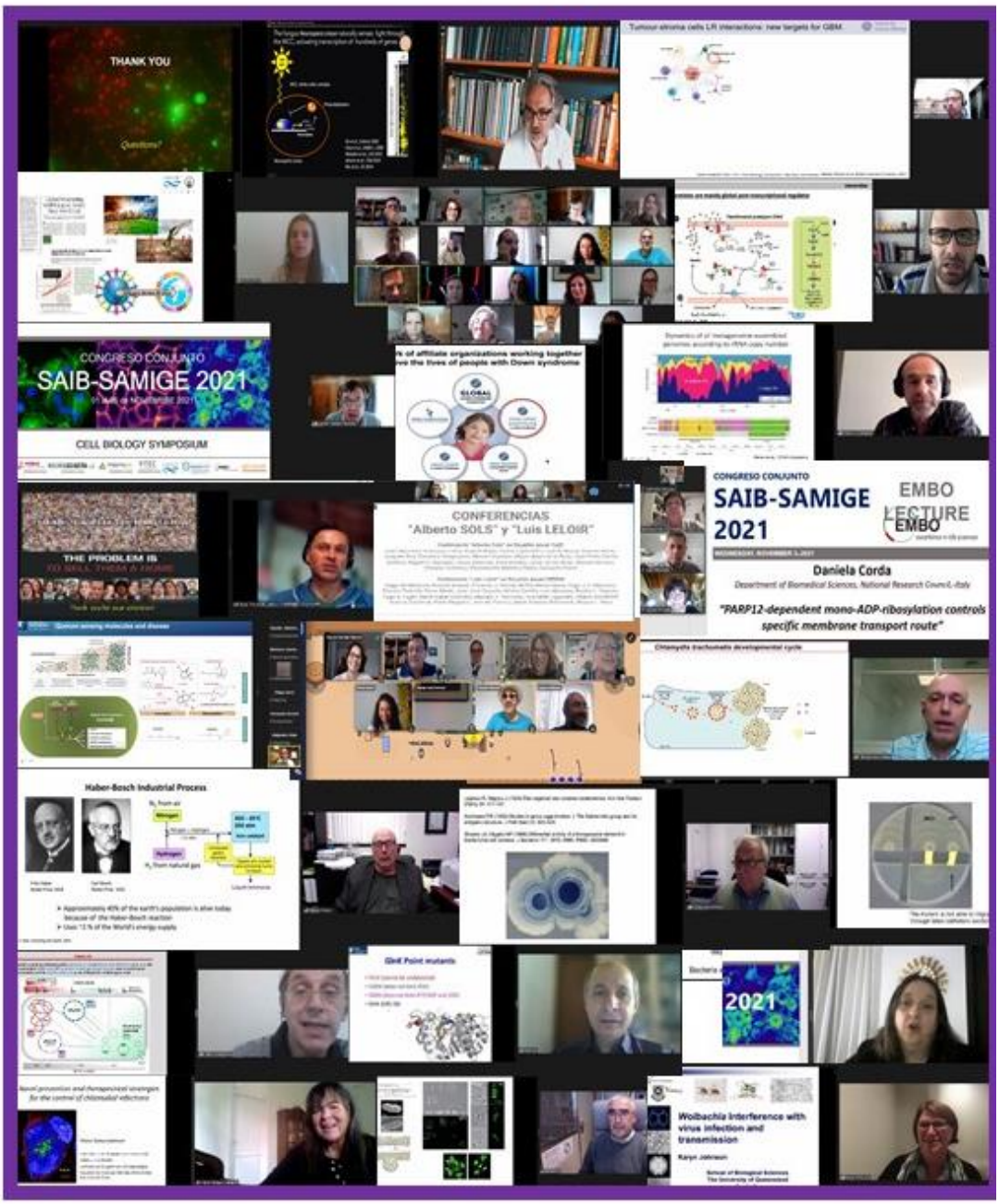

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LVII Annual Meeting of the Argentine Society for Biochemistry and Molecular Biology Research (SAIB)

XVI Annual Meeting of the Argentinean Society for General Microbiology (SAMIGE)

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directly impacts on copepod reproduction, supporting the importance of this study to improve the production of native aquatic species for aquaculture purposes.

LI-P02-55
BLOCKING VERY LOW-DENSITY LIPOPROTEIN (VLDL) SECRETION, BY MICROSOMAL TRIACYLGLYCEROL TRANSFER PROTEIN (MTP) INHIBITION, FAVORS TUMOR DEVELOPMENT

Comanzo CG [1], Vera MC [1], Lucci A[1,2], Heit Barbini FJ[3], Lorenzetti F[1], Ferretti AC[2], Ceballos MP[1], Alvarez ML[1,2,3], Carrillo MC[1,2], Quiroga AD [1,2,3]
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It has been shown that dysregulation in lipid metabolism is a general molecular phenomenon during the progression of hepatocarcinogenesis. The mechanisms by which lipid accumulation occurs during the cellular hepatocarcinoma development are not fully understood. Microsomal triacylglycerol transfer protein (MTP) locates in the lumen of the endoplasmic reticulum and participates in the secretion of lipids from the liver as VLDL. The MTP inhibitor lomitapide binds directly to MTP thereby inhibiting the synthesis of triglyceride-rich VLDL in the liver. The objective of this work was to study the effect of the inhibition of the VLDL secretion on liver tumor development. Adult male C57BL/6 mice were subjected to a model of chemical hepatocarcinogenesis. Animals were randomly divided into two groups. One group (Control) received vehicle (methylcellulose, gastric probe) and another group received 5 mg/kg bw/day lomitapide (gastric probe) for 3 weeks. At the end of the treatment, mice were sacrificed, livers were excised and weighed and tumors counted from the liver´s surface. After treatment, lomitapide-treated mice showed increased liver/body weights ratio (2-fold) and more tumors (2-fold) than control mice. As expected, plasma levels of triacylglycerol and ApoB-100 were decreased (-40% and -60%, respectively) in lomitapide-treated mice compared to control mice. Liver histology analysis showed no differences between groups on tissue and tumor architecture; however, lomitapide-treated mice presented less remaining normal liver parenchyma. Conclusion: these studies demonstrate that inhibition of lipid secretion from the liver could lead to increased tumor development, and MTP may be participating in tumor growth, and represent the first steps in the evaluation of the role of MTP in cancer development.

LI-P03-69
IN VIVO FERROPTOSIS INDUCES LIPID CACOSTASIS: IMPLICATIONS FOR NEURODEGENERATION ASSOCIATED WITH PARKINSON´S DISEASE

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Ferroptosis is a recently discovered type of cell death that results from iron (Fe)-dependent lipid peroxide accumulation and has been proposed as one of the main mechanisms responsible for neuronal death in Parkinson´s disease (PD). In this connection, Fe accumulation in several brain regions, and specifically in the *substantia nigra* has been reported in PD patients. We have previously demonstrated that dopaminergic neurons exposed to α-synuclein overexpression and Fe overload display lipid dyshomeostasis that results in triacylglycerol accumulation and exacerbated phospholipid hydrolysis. In this work, our goal was to characterize the brain lipid profile in an *in vivo* model of ferroptosis. For this purpose, C57BL/6 mice were subjected to Fe overload by performing a four-doses scheme of intraperitoneal administration (Fe-saccharate -800 or 1332 mg/kg- or vehicle). During treatment (16 days), animal welfare and locomotor activity were periodically evaluated. After sacrifice, biochemical parameters were determined in several organs (brain, liver and kidney). Motor skills were assessed by using open field and footprint tests. Mice exposed to Fe overload (1332 mg/kg) showed a 60% diminution of total distance traveled, associated with a greater thigmotaxis (20%; $p < 0.05$) and a slightly delayed right footprint. These alterations in motor skills were related to increased α-synuclein expression. A buildup of oxidative stress markers associated with ferroptosis, such as lipid peroxide levels and conjugated dienes and trienes products derived from fatty acid oxidation (200% and 500%, respectively), was detected in the brain of Fe-treated animals compared to controls ($p<0.001$). Liver and kidney presented a similar profile of oxidative stress markers. Brain lipid content was altered in Fe-treated mice. Whereas increased cholesterol ($p< 0.05$) and diacylglycerol ($p<0.001$) levels were detected, their acylated forms were decreased ($p<0.05$). Total brain phospholipid levels remained unaltered in the ferroptosis model. Changes in neutral lipid profile were paradoxically associated with diminished expression of lipases such as calcium-independent phospholipase A2 and adipose-triacylglycerol lipase. Our results demonstrate that lipid cacostasis is associated with brain Fe accumulation, ferroptosis and motor impairment. The imbalance in lipid acylation/deacylation processes and cholesterol accumulation reported here could be considered as biomarkers of Fe-induced neurodegeneration and ferroptosis.