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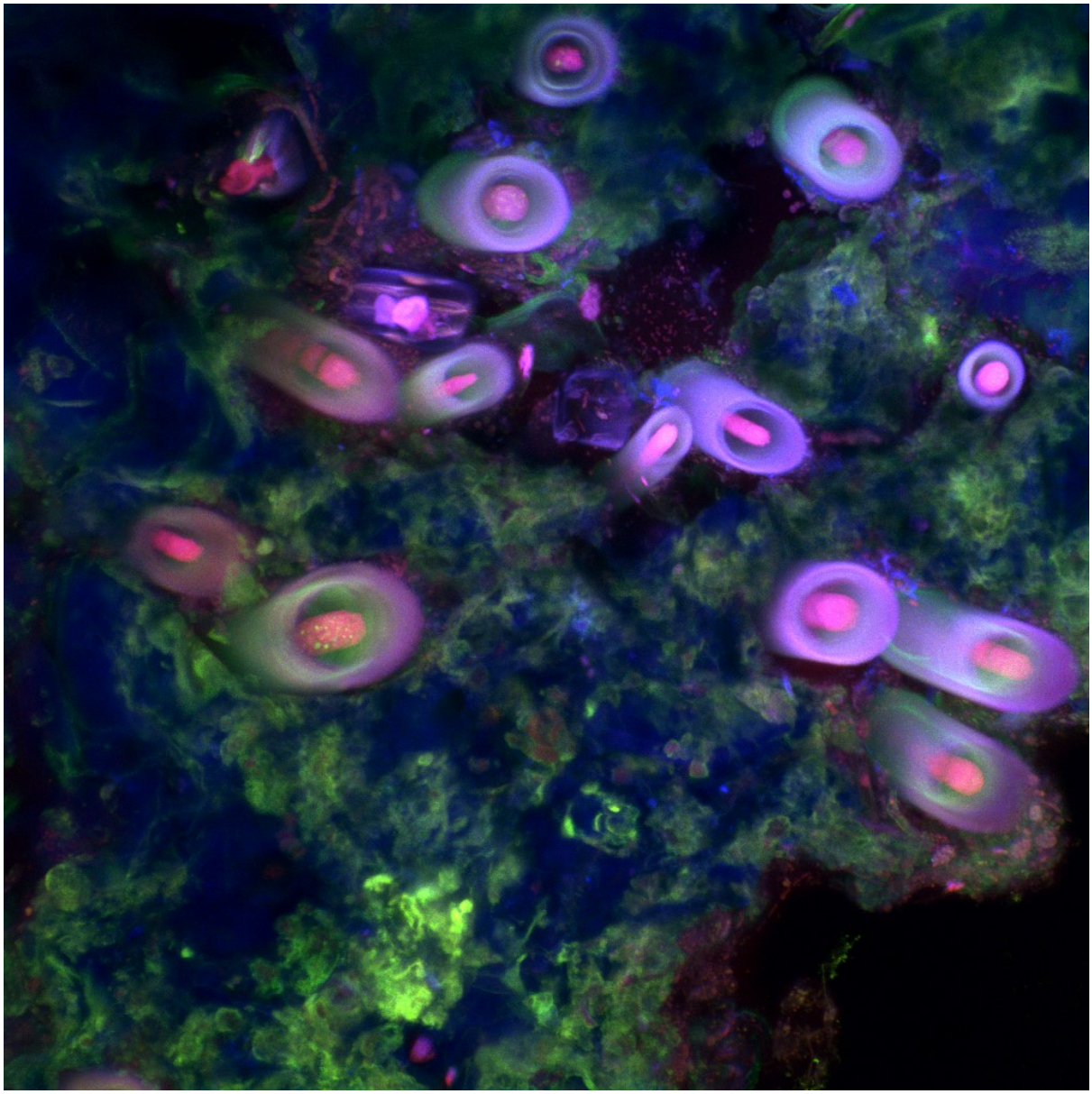
LVI SAIB Meeting – XV SAMIGE Meeting



SAIB-SAMIGE Joint Meeting 2020 – *Online*

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LVI Annual Meeting
Argentine Society for Biochemistry and Molecular Biology
(SAIB)

XV Annual Meeting
Argentinean Society for General Microbiology
(SAMIGE)

SAIB-SAMIGE – Online
Joint Meeting 2020

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2 and produces the contraction of an actomyosin ring in the neighboring cell. The contraction squeezes the cell out apically while drawing together neighboring cells and preventing any gaps in the epithelial barrier. Previously, we demonstrated that sphingosine kinase 2 (SphK2) is involved in MDCK differentiated cells. However, the origin of the S1P is controversial. The goal of this work was to study the source of the S1P synthesis production that triggers cell extrusion. To this end, we developed a microscopy fluorescence-based assay for monitoring S1P endogenous production, based on a shift in the NBD-Sph spectral emission after SphK activity to form NBD-S1P. MDCK differentiated cells showed a very low NBD-S1P signal level; whereas, extruding cells in an upper plane of the monolayer showed a significant increase of NBD-S1P signal. On the other hand, we found a change in the SphK2 subcellular localization that could be linked to the S1P synthesis. The results show that cell extrusion is triggered by the single-cell synthesis of S1P, synthesized by SphK2 of the extruding cell itself.

LI-C05-81
MENADIONE-INDUCED OXIDATIVE STRESS ALTERS LIPID METABOLISM OF THE MATURE ADIPOCYTE

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Obesity is closely related to metabolic disturbances, with the latter majorly caused by adipose tissue dysfunction. Oxidative stress (OS), a major characteristic of dysfunctional adipose tissue, is considered a primary contributing factor to the etiopathogenesis of obesity and associated comorbidities. However, the biochemical mechanisms by which OS alters adipocyte biology still require to be fully uncovered. We have previously demonstrated that menadione, a synthetic vitamer of vitamin K known to generate intracellular oxygen species, impairs adipogenesis by inhibiting the PI3K/Akt pathway. Our goal in this work was to study the effect of menadione-induced OS on mature adipocytes. For this purpose, differentiated 3T3-L1 adipocytes were exposed to menadione (20 and 50 μM) for 5 h, and different biochemical parameters were assessed. The exposure to menadione resulted in increased cell oxidants (65% and 122% of control, for 20 and 50 μM, respectively). However, none of the concentrations of menadione tested had any significant effect on either cell viability or morphology. The expression of adipogenic markers was evaluated by Western blot. Menadione-induced OS caused a significant decrease in the expression of PPARγ (95% and 99%, for 20 and 50 μM menadione, respectively), FAS (70% and 88%, for 20 and 50 μM menadione, respectively), C/EBPα (75% and 93%, for 20 and 50 μM menadione, respectively), and FABP4 (30% for 50 μM menadione). No changes were detected in intracellular triglyceride levels after the incubation in the presence of menadione. However, when the exposure to menadione-dependent OS was extended to 24 h, the intracellular triglyceride content was augmented by 53% and 68% upon the exposure to 20 and 50 μM menadione, respectively. At the same time, ACC (the rate-limiting enzyme in fatty acid synthesis) was activated (32% and 38% decreased phosphorylation, for 20 and 50 μM menadione, respectively). On the other hand, menadione-triggered OS also activated lipolysis (40% for 50 μM menadione). Together, our results show that OS acutely modulates both the expression and activity of different lipo/adipogenic proteins, activating fatty acids' metabolic turnover, with enhanced lipolysis, which is overcome by fatty acid synthesis, resulting in an increased triglyceride content. Our next goal is the unraveling of the cellular signaling responsible for these metabolic changes observed.

LI-C06-244
URSOLIC ACID INTERFERES LIPID DROPLET METABOLISM AND INHIBITS ROTAVIRUS INFECTION

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Rotavirus (RV) is one of the main causes of acute gastroenteritis and hospitalizations in young children, mainly affecting developing countries. Since there is no specific treatment, the development of an efficient method for RV elimination is still a priority. We demonstrated that ursolic acid (UA), a natural triterpenoid, exerts anti-RV activity, negatively affecting the early stages of the viral cycle. Moreover, UA comprises a broad anti-RV since the yields of the simian SA11, the porcine RRV, and the bovine NCDV RV strains were diminished in the presence of the compound *in vitro*. Once the virus reaches the cytosol, viral protein translation and genome replication begin. Immediately after the viroplasms (VPs) formation occurs. The VPs are electrodense structures that constitute the platform for the assembly of new viral particles. One of the main components of the VPs is the lipid droplets (LDs), dynamic organelles mainly associated with lipid storage within the cells. We and others observed that during RV infection, there is an accumulation of LDs in the cells. We analysed if the anti-RV effect of UA was due to its ability to modulate the LD metabolism and/or the thermodynamic aspects related to LDs generation and growth. To evaluate the influence of UA on LDs formation, we used Langmuir monolayers as a model. Monolayers of a phosphatidylcholine and triglyceride (PC-TG) mixture –two of the main LDs components– were prepared, and the generation of TG-aggregates (lenses) was monitored using Brewster Angle Microscopy. Our results showed that UA exerts an effect on PC-TG mixtures, yielding membranes thermodynamically more prone to form lenses. Therefore, the number of lenses increased with UA content. However, the lenses became thinner in those conditions. These observations, translated to the cellular environment, suggest that although UA would induce the formation of "blisters" inside the ER-membrane (the initial stage of LD biogenesis), it would interfere with their budding-off. Accordingly, we observed that short treatments with UA significantly decreased the number and size of LDs within the cells. Also, we analysed the lipolytic rate of LDs in the presence