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LI-01
PLD AND PI3K SIGNALING IN NEURONAL OXIDATIVE STRESS

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Lipid signaling cascades have important roles in the regulation of cellular fate. Our studies provide new insights into the regulation and physiological role of lipid messengers during neuronal oxidative stress (OS). Specifically, we have studied neuronal signal events derived from phosphatidylcholine (PC) and phosphatidylinositol (PI).
Synaptic OS triggers phospholipase D (PLD) activation and, consequently, a rise in phosphatidic acid and diacylglycerol (DAG) generation from PC. These lipid messengers activate downstream signaling cascades as ERK1/2 and conventional PKCs and regulate glutamate transport in the synaptic cleft of adult rat brains. Studies in aged brains reveal an increased synaptic susceptibility to OS and an impairment in the DAG-mediated signaling pathways. Tyrosine phosphorylation associated with PI phosphorylation and phosphoinositide 3 kinase (PI3K) activation are stimulated in OS-exposed hippocampal neurons and synapses. PI3K activation and its downstream effector kinase, Akt, trigger pleiotropic neuroprotective mechanisms against OS by suppressing FOXO3A transcriptional activity, inhibiting GSK3β and upregulating glutathione metabolism.
In summary, we have characterized signaling events elicited by PLD and PI3K activation, which produce lipid messengers that control smart strategies for preventing neuronal death triggered by OS.

LI-02
INSIGHTS INTO THE MECHANISM OF INTERACTION BETWEEN FABPS AND BIOLOGICAL MEMBRANES. A COMPUTATIONAL APPROACH

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The role of fatty acid binding proteins (FABPs) as intracellular fatty acid (FA) transporters may require their interaction with membranes; not only to deliver long-chain fatty acids to target surfaces, but also to remove them from the membranes. In vitro studies have shown that different FABPs, transfer FA to or from membranes by two different mechanisms. Liver FABPs do it by a supposed aqueous phase diffusion ("diffusional" mechanism); but, in marked contrast, a larger number of FABPs, including adipocyte, intestinal, brain, and heart/muscle types, transfer their FAs by directly interacting with a membrane ("collisional" mechanism). However, the process in which both kind of proteins adsorbs to the membrane remains to be elucidated. Based on computational analysis, we confirmed that recruitment to membranes is facilitated, in a first step, by electrostatic interactions; and this analysis can quantitatively differentiate among the mechanisms of membrane association proposed, and determinate the most energetically favorable configuration for the membrane-associated states of different FABPs. Moreover, we have identified the aminoacids putatively responsible for both, collisional and diffusional mechanisms; and, also, we pointed out how the structures could be punctually modified to adapt their function. Finnally, using molecular dynamics simulations we studied the interactions of apoFABPs with lipid membranes of anionic and zwitterionic phospholipids, even for FABPs of unknown behavior, using molecular modeling methods as predictive tools in biophysics.

LI-03
ROLE OF MONOUNSATURATED FATTY ACIDS IN LIVER-ADIPOSE TISSUE CROSS-TALK AND METABOLIC REGULATION

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The maintenance of metabolic health requires complex regulation of metabolic processes in several tissues. The coordination of this metabolic regulation involves extensive crosstalk among tissues. Signaling factors that are secreted into the circulation and impart systemic metabolic effects include molecules such as hepatokines, adipokines and lipokines. Many of these factors regulate lipid metabolism, including de novo lipogenesis. Stearoyl-CoA desaturases (SCDs) catalyze the delta 9-desaturation of the saturated fatty acids palmitate and stearate to the monounsaturated fatty acids (MUFAs) palmitoleate (16:1n7) and oleate (18:1n9), respectively. These MUFAs, mainly oleate, are predominant components of cellular and circulating free fatty acids, triglycerides, cholesterol esters and membrane phospholipids. In mice, genetic ablation of SCD-1 isoform reduces hepatic de novo lipogenesis (DNL) and protects against high carbohydrate diet-induced adiposity and liver steatosis. To understand the mechanism by which hepatic MUFA production influences adiposity, we created two liver-specific transgenic mouse models in the SCD1 global knockout that express either human SCD5 or mouse SCD3, that synthesize 18:1n9 and 16:1n7