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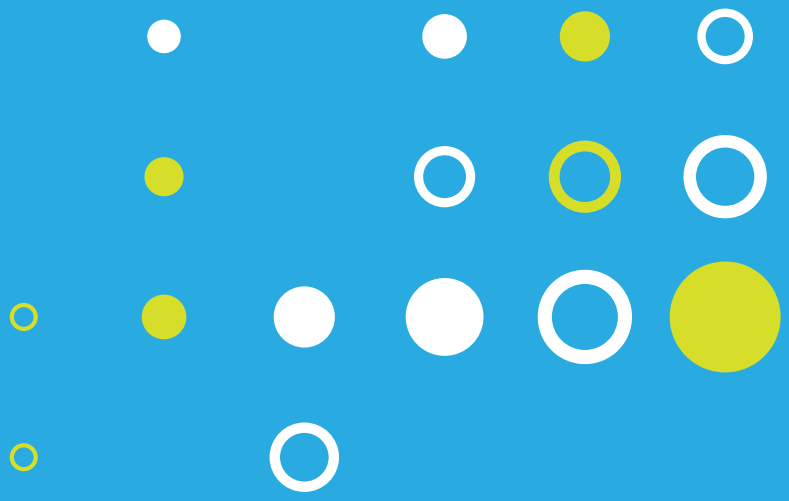

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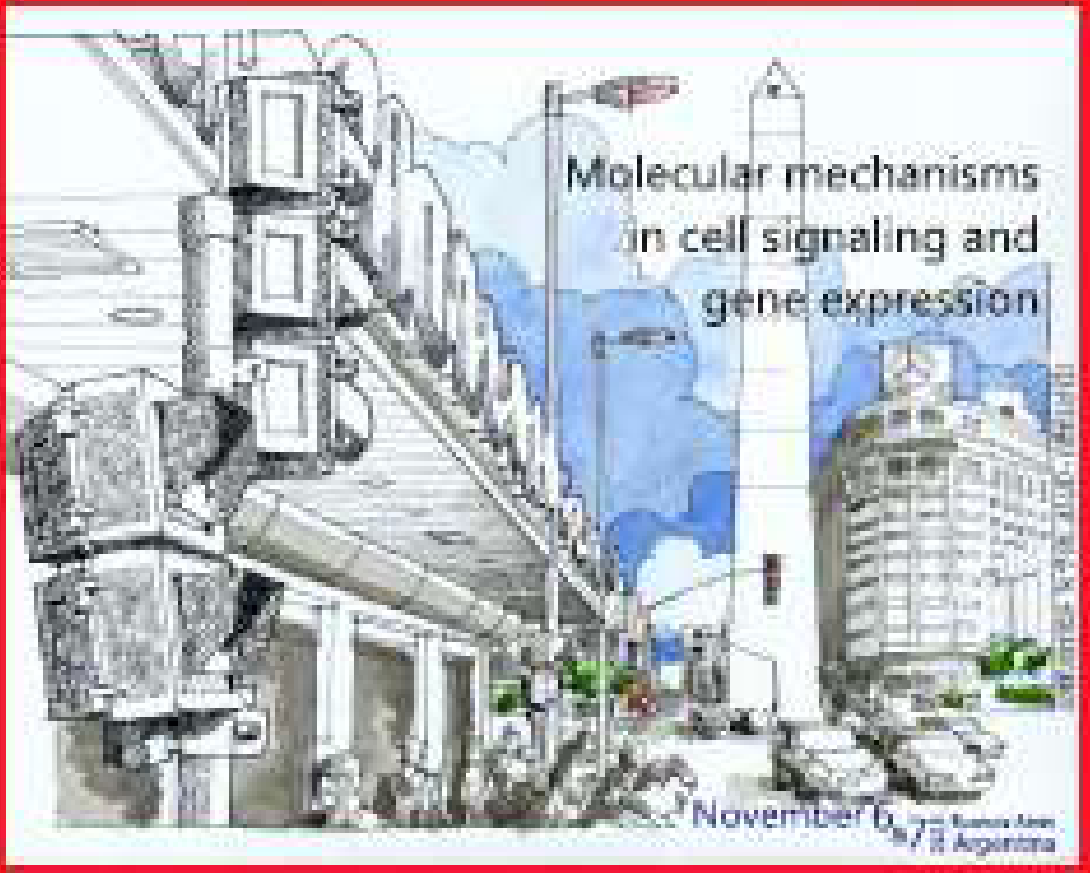

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«Molecular mechanisms in cell signaling and gene expression»

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LI-P13.
DGK-θ AT THE SYNAPSE: A ROLE FOR THE METABOLISM OF DAG IN SYNAPTIC VESICLE RECYCLING?
Goldschmidt H[1], Tu-Sekine B[1], Anggono V[2], Huganir R[2], Raben D[1]. Dept.of[1]Biological Chemistry and [2]Neuroscience, The Johns Hopkins School of Medicine,USA. E-mail: hanabobana21@gmail.com

Diacylglycerol kinase-θ (DGK-θ) is one of ten mammalian DGK isoforms that regulate the generation and metabolism of two important lipid second messengers: diacylgylcerol (DAG) and phosphatidic acid (PtdOH). Although DGK-θ is primarily expressed in the brain, no physiological role has been identified for this isoform in the central nervous system (CNS). Our data demonstrate this enzyme is involved in regulating glutamate release from cortical neurons. Both shRNA-knockdown of DGK-θ and neurons derived from DGK-θ knock-out mice exhibit a decreased rate of synaptic vesicle (SV) endocytosis compared to control neurons. Importantly, the rate of SV endocytosis is recovered by ectopic expression of DGK-θ in neurons depleted of the endogenous enzyme. In contrast to SV endocytosis, our most recent data show that the rate exocytosis is elevated in DGK-θ knock-out neurons. Our data establish a role for DGK-θ at the presynaptic nerve terminal in the regulation of SV recycling, and suggest that DGK- θsupports synaptic transmission during periods of elevated or sustained neuronal activity.

LI-P14.
PHOSPHOLIPID REMODELING IN DOPAMINERGIC NEURONS: ROLE OF α-SYNUCLEIN VARIANTS AND IRON OVERLOAD
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Increased levels of α-synuclein (α-syn) and iron-overload are pathognomonic signs of dopaminergic neurons in Parkinson's disease (PD) patients. Moreover, iron and fatty acid (FA) availability are predisposing factors for pathological α-syn aggregation. In this work, we characterized the phospholipid remodeling pathways that regulate FA availability in dopaminergic neurons overexpressing α-syn variants (WT and A53T) and exposed to iron-overload. Increased cellular oxidant and lipid peroxidation levels were observed in dopaminergic neurons exposed to iron-overload. The inhibition of calcium-independent phospholipase A^2 (deacylation pathway) provoked an increase in the extent of cellular damage induced by iron-overload. In this connection, phospholipid acylation was differentially affected by iron overload and the presence of α-syn variants. FA incorporation into phosphatidylcholine (PC) and phosphatidylethanolamine (PE) was increased in dopaminergic neurons harboring WT α-syn. This acylation profile was not altered by iron-overload. Neurons expressing A53T α-syn (a variant present in autosomic dominant PD and with high iron affinity) showed a diminished FA esterification in PC and PE. This effect was enhanced in iron overloaded neurons. Our results show that FA availability is differentially regulated by α-syn variants and iron overload in this *in vitro* model of PD.

LI-P15.
CONSEQUENCES OF THALLIUM EXPOSURE ON MDCK CELLS LIPIDS METABOLISM
Morel Gómez E[1], Casali C[1], Faggionato D[1], Verstraeten SV[2], Fernández Tome MC[1].
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Thallium (Tl) is a toxic heavy metal that among other symptoms causes renal damage. In the present study we investigated the effects of Tl(I) and Tl(III) on lipids metabolism in a renal epithelial (MDCK) cell line. Cells were exposed to 10 or 100 μM of Tl(I) or Tl(III) for either 24 or 48 h. The profile of phospholipids (PL), cholesterol (Cho) and triacylglycerides (TG) was evaluated. No changes were observed at 10 μM Tl(I), but increased total PL (24 h: 28%, 48 h: 40%) and Cho (24 h: 30%, 48 h: 52%) contents were found at 100 μM Tl(I). TG content was decreased by 30% and increased by 80% after 24 and 48 h of Tl(I) treatment, respectively. Tl(III) (100 μM) increased both PL and Cho content around 100% after 24 h and 300% after 48 h of incubation. TG content was increased by 60 and 600% after 24 and 48 h, respectively. Such an increase was also evidenced in lipid droplet size. PL profile showed decreased phosphatidylethanolamine and increased phosphatidylcholine contents. After 48 h, both cations decreased the fluidity of the outer monolayer of plasma membrane, increased that of the inner monolayer, and increased the annular fluidity. Tl(III) caused marked alterations in cells ultrastructure. The increase in cell membranes components (PL and Cho) and fatty acid storage (TG) as well as the changes in membrane properties may contribute to renal cell dysfunction in Tl-exposed people.

LI-P16.
HUMAN MILK: EVALUATION OF CREAMATOCRIT METHOD FOR DETERMING CALORIC CONTENT
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[1]INIBIOLP(CCT-LP,CONICET,UNLP); [2]Dto Cs Biol,Fac Cs Ex,UNLP; [3]Bc de Leche Mat. HIGASMLP. [4]CEREN-CIC/PBA. E-mail: martins.sisti@gmail.com

Human milk is a fluid capable of providing all the nutrients and defenses necessary for the newborns (NB). The neonatology service of the HIGA San Martin La Plata City has its own Mother's Milk Bank, whose milk is classified according to gestational age in which the birth took place (to find homogeneity with the recipient's mother milk); colostrum (up to 7 days old), transitional milk (7 to 14) and mature milk (over 14). Categorized milk is classified by its caloric content (CC), by the creamatocrit method (CM) which only considers milk lipids (L) whereas proteins (P) and carbohydrates (C) are not quantified (CM:L66.8+290).This allows selecting the milk which best adapts to the NB's needs. The aim of the present study was to determine if CM (at present applied in the Milk Bank) is appropriated to evaluate milk CC. For this purpose milk CC was determined by CM and analytical methods (P:Kjeldakl, L:Gerber, C:Antrona-Sulfuric) and Atwater coefficients. Mature milk had 0.40-0.65 kcal/ml (both methods). However, CM was equivalent to CC only when milk P and C were within normal ranges, if not CC was over/under estimated. In conclusion, we propose milk CC should consider all macrocomponents, in benefit of those neonates undergoing critical stage. IR Spectrometry would be the most convenient method since it is simple, quick, non-destructive and sensitive.