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Lipids

LI-C01
NUCLEAR LIPID METABOLISM IS DIFFERENTLY REGULATED BY POLYUNSATURATED FATTY ACIDS DURING AGING

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Former studies from our lab demonstrated an active nuclear lipid metabolism in central nervous system that is modified by aging. We detected several nuclear enzymatic activities related to glycerolipid metabolism, such as lipid phosphate phosphatase (LPP), diacylglycerol lipase (DAGL), monoacylglycerol lipase (MAGL), phospholipase A (PLA) and lysophosphate phosphatase (LPAPase). Interestingly, we also observed that they could be regulated by retinoic acid and polyunsaturated fatty acids (PUFA) through an unknown non-genomic mechanism in adult nuclei. Therefore, the aim of this work was to study the modulation of these enzymatic activities by arachidonic acid (AA) and docosahexaenoic acid (DHA) in nuclei from cerebellum of aged rats. To this end, rat cerebellums (28 mo) were homogenized and highly purified nuclei were isolated by sucrose-density ultracentrifugation. Using the respective radiolabelled substrates co-incubated with these PUFA, we observed that AA and DHA promote a major DAG availability by increasing and decreasing LPPs and DAGL activity, respectively. A minor MAG availability was also observed due to a diminution on PLA and LPAPase activities. These results demonstrate a different PUFA-regulated lipid metabolism in aged nuclei with respect to adults which could be involved in signaling events related to the epigenetic changes during aging.

LI-C02
SK1 AS KEY G1-G0 TRANSITION MODULATOR IN RENAL EPITHELIAL CELL

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Sphingosine Kinase (SK) is a key enzyme involved in the synthesis of sphingosine-1-Phosphate (S1P), a lipid mediator that regulates several cellular processes. S1P has been characterized as a dual signaling molecule with the ability to activate different effectors. We demonstrated that S1P biosynthesis present a gradual decrease during kidney maturation and cell proliferation. In this report we evaluate the SK activity in renal epithelial cell cycle modulation and in the transit to cell differentiation For this, MDCK cells were cultured at low density to allow cell cycle progression and were treated with D,L-threo-dihydrosphingosine (tDHS), a SK1 inhibitor. SK inhibition induced a decrease in cell number after 24 h of incubation with no alteration in cell viability. Besides, treatment for 24 h with tDHS caused cell cycle arrest in G0/G1 phase with cyclin D1 accumulation. Cell cycle arrest was accompanied with hypophosphorilation of Rb protein. These results suggest that intracellular S1P was involved in cell cycle arrest. Moreover, SK inhibition induced an increase in the percentage of cell in G0 phase after tDHS treatment accompanied by cellular morphological changes. These suggest that S1P is not only involved in cell cycle arrest (with induction of cell quiescence), but also participates in cell differentiation.

LI-C03
CIRCADIAN REGULATION OF CLOCK GENE EXPRESSION AND PHOSPHOLIPID BIOSYNTHESIS IN GLIOBLASTOMA CELLS

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Circadian clocks present even in immortalized cell lines, temporarily regulated diverse physiological processes including cell proliferation and apoptosis while disruption of circadian rhythms can alter cell cycle to potentiate tumorigenesis. Here we analyzed whether the immortalized human glioblastoma T98G cells subject to proliferation (P) in the presence of serum, or maintained quiescent (Q) keep a functional clock, after synchronization, temporally regulating gene expression and phospholipid (PL) metabolism. We examined the expression of clock genes (Bmal1, Per1, Rev-Erbα) and PL synthesizing enzyme genes (choline kinase α: Chokα and CTP:phosphoethanolamine cytidylyltransferase 2:Pcyt-2), and the metabolic labeling of PLs. Cells grown in 10% FBS-DMEM for 3 days were synchronized with a 20 min shock of dexamethasone (100 nM) (time 0), maintained with (P) or without FBS-DMEM