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and with UFA content similar to detected in the parental strain (9.8% and 11%, respectively), although distributed mainly in PG. When the mutant strain was exposed to TTAB, the *P* value was 0.14, the UFA content increased (16.5%) and the amount of viable cells decreased from 10^{12} ufc ml⁻¹ to 10^{6} ufc ml⁻¹, demonstrating that the fluidizing effect of surfactant cannot be counteracted. The set of results indicate that an adequate level of CL is indispensable in the cell's response to TTAB

LI-P13

LIPID PROFILE IN BRAIN MITOCHONDRIA DURING DEVELOPMENT: SEXUAL DIFFERENCES

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During perinatal development, testosterone (Te) released by male testes has organizational functions responsible for sexual differences in mce adult brain. As neuronal remodeling during this period depends on mitochondrial metabolism, we studied mitochondrial lipid composition during postnatal development and it relationship with Te. We used C57BL/6 pregnant dams to separate pups by sex at postnatal day 0, 2, 4, 6, 8 and 10. At each time point we obtained blood samples and mitochondrial fraction (MF) from cerebral cortex. Plasma Te levels were measured by RIA, total lipids from MF were extracted, phospholipids (PL) separated by 2D-TLC and fatty acid composition quantified by c-GLC. We found a sex-independent variation of PC, PE and CL content during the analyzed period. In particular, CL, showed a differential fatty acid (FA) profile within sex, it unsaturation index (UI) is higher in males than in females at PND 0 and 2, due to the higher content of 20:4 and 22:6. The UI correlated well with Te levels androgenizing females with Te propionate (100 μ g of 2 mg/mL in corn oil) at PND 0. The sexual dimorphism we have found would be relevant for understanding the long-lasting deleterious effects in brain of the exposure to endocrine disruptors during development.

LI-P14 EXPRESSION OF FATTY ACID ELONGASES IN CELLS OF THE SEMINIFEROUS EPITHELIUM

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Fatty acid elongases play a crucial role in the biosynthesis of long-chain polyunsaturated fatty acids (PUFA) and in their further elongation to very long chain (VLC) PUFA. Male rat germ cell membranes contain glycerophospholipids with C18-C24 PUFA and sphingolipids with C26 to C32 PUFA. In the present study, the expression of seven members of the *Elovl* (elongation of very long chain fatty acids) gene family that encode elongases was surveyed in pachytene spermatocytes, round spermatids and Sertoli cells. The mRNAs of *Elovl1*, *Elovl2*, *Elovl4*, *Elovl5*, and *Elovl6* were detected in all of these cells , all of them lacked *Elovl3* expression, and *Elovl7* was expressed only in the latter. As the ELOVL4 protein was previously shown to be responsible for the elongation of >C24 PUFA in the retina, the expression of this protein was also evaluated. During postnatal development, ELOVL4 was not detectable in testis up to P21, i.e., its time of appearance concurred with that of the first spermatocytes. Thereafter, the protein was evidently present in spermatocytes and spermatids, and was also faintly detected in Sertoli cells. As *Elovl2* and *Elovl5* are essential to form PUFA, and *Elovl4* is required to elongate them, the joint expression of these elongases in spermatocytes and spermatids implies that they are functionally related, probably acting in sequence to produce the VLCPUFA of sphingolipids. This work was partially supported by Fondecyt 1140758(JRG).

LI-P15

A53T α-SYN REGULATES TRIACYLGLYCEROL CONTENT IN DOPAMINERGIC NEURONS EXPOSED TO OXIDATIVE STRESS

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Iron-induced oxidative stress and pathological α -synuclein (α -syn) aggregation contribute to the loss of dopaminergic neurons in Parkinson Disease (PD). In this work, we characterized the status of lipid metabolism in N27 dopaminergic neurons and in neurons stably expressing A53T α -syn (a dominant mutation found in familial early onset PD) exposed to iron-induced injury. N27 dopaminergic neurons incubated with iron for 24 hs (Fe, 1mM) displayed increased levels of reactive oxygen species (ROS), lipid peroxidation and elevated plasma membrane