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Pyrroloquinoline quinone nutritional status revert synaptosomal mitochondrial dysfunction and behavioral alterations in a transgenic rat model of Alzheimer's disease

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Alzheimer's disease (AD) is associated to depressed brain energy supply and impaired cortical and hippocampal synaptic function. Previous reports showed no consistent time-course bioenergetic deficiencies of cortical and hippocampal presynaptic nerve terminals (synaptosomes) from commonly used mouse models with AD-like phenotypes (J20, Tg2576 and APP/PS) as compared to age-matched controls supporting the conclusion that the intrinsic bioenergetic capacities of presynaptic nerve terminals are maintained in these symptomatic AD mouse models. Intraneuronal accumulation of amyloid β (Aβ) has been linked to mild cognitive impairment that precedes AD onset. This neuropathological trait was recently mimicked in a novel animal model of AD, the hemizygous (+/-) transgenic (Tg) rats (McGill-R-Thy1-APP). We performed in Tg(+/-) and control (WT) animals a time-course analysis of the bioenergetic profile of isolated hippocampal mitochondria and synaptosomes to evaluate mitochondrial functionality and its impact on behavior. A clear bioenergetic dysfunction was observed in isolated hippocampal mitochondrias or synaptosomes in 6 months-old Tg(+/-) rats which correlates to higher levels of anxiety and impaired working and spatial reference memories as compared to their WT littermates. This phenotype was partially reverted when Tg(+/-) rats were fed a nutritionally complete diet with Pyrroloquinoline quinone (PQQ) added at 2 mg/Kg from weaning to 6 months-old. Collectively, these data demonstrate that the intrinsic bioenergetic capacities of presynaptic nerve terminals are not conserved in these AD rats lacking extracellular Aβ accumulation and that PQQ impacts a number of parameters related to normal mitochondrial function and cognition.

Biography

Laura Morelli has pursued his MS from School of Pharmacy and Biochemistry-UBA (Argentina), PhD from UBA (Argentina) in 1992 and Post-Doctoral Fellow at NYU School of Medicine (USA) In 1993. In 2001, he worked as Fulbright Senior Fellow at NYU School of Medicine (USA). From 2014 to present, he is working as a Principal Investigator and Group Leader at Laboratory of Amyloidosis and Neurodegeneration, Leloir Institute (Argentina).

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