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54th Annual Meeting Agentine Society for Biochemistry and Molecular Biology

LIV Reunion Anual Sociedad Argentina de Investigación en Bioquímica y Biología Molecular

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BIOCELL 42 (suppl. 4), 2018
ISSN 1667-5746 (online version)

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BIOCELL 42 (suppl. 4), 2018
ISSN 1667-5746 (online version)

NEUROSCIENCE

NS-P01
DIFFERENTIAL COCHLEAR HAIR CELL DEGENERATION IN MICE WITH IMPAIRED POTASSIUM RECYCLING

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Potassium ion (K^+) is essential for sound transduction in mammalian inner ear. KCNQ4, a voltage-gated K^+ channel, is expressed in cochlear hair cells (HCs) and in the central auditory pathway. KCNQ4 mutations lead to DFNA2, a progressive sensorineural deafness, due to chronic depolarization of HCs. Our aim is to analyze the progression of HC loss over time generated by absence of KCNQ4 channel in a mouse model lacking its expression (*Kcnq4-/-*). Quantitative PCR on wild-type mouse revealed the strongest *Kcnq4* mRNA level in basal cochlear turn, while it decreases ~50% in middle-apical turns. By using immunofluorescence on cochlear whole-mounts, we estimated cell number average and plotted cytocochleograms. We observed the highest outer hair cell (OHC) degeneration in basal turn starting early (3 weeksold(W)), which progresses to middle and apical turns in older mice (10-58W). Moreover, cell death progression correlated with different OHC stereociliar disarrangement patterns. Degeneration differed according to OHC row: the middle one exhibited the maximum decrease at 10W in *Kcnq4-/-* mice. Furthermore, inner HCs reached total loss in basal initial segment at 40W and cell loss also progresses to middle turn with age. Our results indicate that both HCs degenerate but starting at different ages, contributing to elucidate the mechanisms leading to profound hearing loss in DFNA2 patients.

NS-P02
AKT/FOXO3A PATHWAY: SIGNALING TARGET FOR α-SYNUCLEIN OVEREXPRESSION AND MANEB MEDIATED NEUROTOXICITY

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α-synuclein (α-syn) overexpression and manganese-based pesticides such as Maneb (Mb) have been both implicated as etiological factors of Parkinson's disease. We have previously reported the neuroprotective role of Akt/FoxO3a in amyloid β- and Fe-induced injury. In this work, we studied the role of the above-mentioned pathway in the effect of Mb and/or α-syn overexpression on IMR-32 human neuroblastoma cells. For this purpose, we exposed these neurons for different times (24-72 h) to increasing Mb concentrations (6-24 μM) and evaluated the redox status, Akt/FoxO3a subcellular localization and phosphorylation levels, and cell viability. The same parameters were evaluated in neurons stably overexpressing the wild type form of α-syn and exposed to either Mb or its vehicle.
Mb exposure provoked a time- and concentration-dependent decrease in neuronal viability. This cytotoxic effect was mediated by the increase in reactive oxygen species (ROS), lipid peroxides and membrane cell permeability (LDH release). Intriguingly, Mb exposure in α-syn-overexpressing neurons showed decreased ROS content and LDH release, with no changes in lipid peroxides. Mb was also found to induce changes in α-syn aggregation and phosphorylation, as measured with the intracellular probe Thioflavin S and by immunocytochemistry.
On the other hand, Mb exposure and α-syn overexpression unconnectedly triggered the increase in Akt and FoxO3a nuclear localization. However, Mb exposure in α-syn overexpressing neurons enhanced FoxO3a nuclear localization without increasing cell death. We hypothesize that FoxO3a might be an α-syn target related with its unexpected protective role.

NS-P03
ANALYSIS OF THE BEHAVIOR OF NEURAL STEM CELLS UNDER OXIDATIVE STRESS INDUCED BY IRON AND COPPER

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Traditionally, it was thought that the mammalian nervous system lacked the ability to self-repair after injuries or neurodegeneration. We now know that the adult brain does indeed hold the capacity to regenerate, albeit to a limited extent. Endogenous neural stem cells (NSCs) could be a regenerative source for the damaged neural cells but because their number and regenerative ability are limited, they cannot fully repair the damaged tissue. Factors present in the injured microenvironment (such as inflammatory mediators and reactive oxygen species (ROS)) influence survival, self-renewal, migration and neuronal differentiation of both endogenous NSCs and transplanted exogenous stem cells.
In order to test this hypothesis, we used the transition metals iron and copper to induce oxidative stress in NSCs. To determine the appropriate concentration and exposure time, the viability of the cultures treated with metals was assessed using the MTT assay and by Trypan Blue staining. To evaluate the extent of metal-induced effects on NSCs, cell morphology and generation of ROS (measured by using the probe DCFH-DA) were analyzed. Moreover, the type of cell death after the exposure to iron and copper was evaluated by differential nuclear staining with fluorescent dyes acridine orange and ethidium bromide. We demonstrate that both metals can stimulate the production of ROS in NSCs cultures an induce apoptosis or necrosis of the stem cells.
Additionally, we investigated the effects of iron and copper on the ability of NSCs to proliferate and generate new neurospheres or to differentiate into neurons. We have observed that both metals affect NSCs survival under proliferation conditions. Furthermore, by