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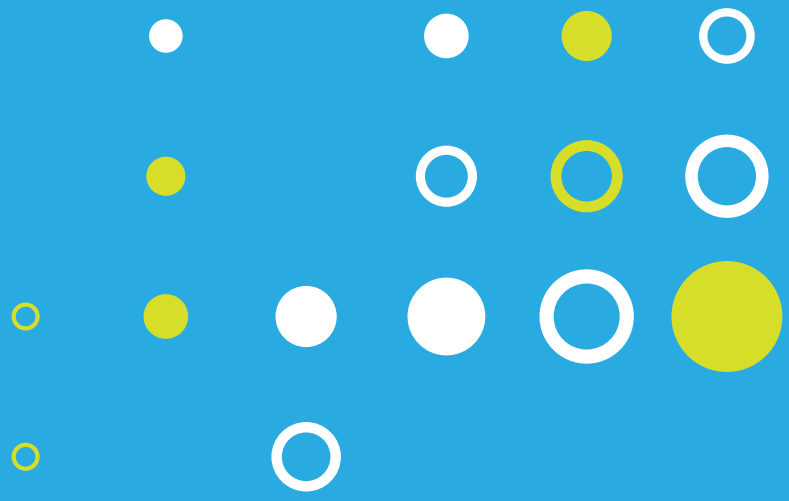

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República Argentina

NS-P05.

COOPER, CERULOPLASMINE, AND OXIDATIVE STRESS BIOMARKERS LEVELS IN HUMAN NEURODEGENERATIVE DISORDERS

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Cu imbalance and oxidative stress (OS) are documented in brain tissue of Alzheimer patients (AD). However, their utility as prognostic parameters in peripheral blood is still poorly understood. We studied the levels of TBARS, nitrate + nitrite [NOx], protein carbonyls (PCs), total glutathione (GSH), tocopherol (Toc), antioxidant enzymes, Cu, Se, Zn, and ceruloplasmine (CRP) in plasma and erythrocytes from AD, Parkinson (PD) and vascular dementia patients (VD). Results were compared with first order relatives (R) and a control population. All OS markers were altered in AD, PD and VD compared with the corresponding control group. Superoxide dismutase, GSH-reductase, and catalase activities in erythrocytes, as well as TBARS, PCs, and [NOx] in plasma were increased; while GSH and Toc in AD, PD and VD were decreased. Cu level was increased in severe AD and PD patients, and in all stages of VD. R of AD and VD also exhibited increased [Cu] not associated to substantial and clear alterations in OS biomarkers. CRP was increased only in VD samples. We conclude that peripheral OS biomarkers should be poor discriminating indexes for the screening of the type and severity of neurodegenerative disorders; however, the CRP/Cu ratio may be useful in estimating the progression from a sub-clinical condition to a symptomatological illness, especially in R of VD.

NS-P06.

GSK3â AND ERK ARE ACTIVATED DOWNSTREAM OF PI3K IN SYNAPTIC ENDINGS DURING OXIDATIVE INJURY

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Iron induced oxidative injury is comparable to that of ß-amyloid peptide on the brain of Alzheimer's disease patients. Our purpose was to evaluate the state of PI3K pathway and its downstream effectors Akt and GSK3ß in cerebral cortex synaptosomes exposed to Fe^{2+} (50 µM) for different periods of time (5, 30 and 60 min). The increase in Akt phosphorylation in serine 473 and threonine 308 was temporally coincident with PI3K activation (5 min). GSK3ß, the downstream effector of Akt, was also phosphorylated after 5 and 30 min of iron exposure and this phosphorylation was inhibited by LY294002. Additionally, Erk activation was also observed after 5 and 30 min of insult exposure, and this activation was PI3K-dependent. Immunoprecipitations carried out with anti-cSrc demonstrated a strong association between activated Akt and this tyrosine kinase induced by oxidative stress. Our results demonstrate that oxidative stress triggers the activation of different synaptic signaling pathways that operate downstream PI3K/Akt.