P2-016 NEUROPATHOLOGICAL, NEUROPSYCHOLOGICAL, AND IMAGENOLOGICAL COMPARISON BETWEEN SPORADIC ALZHEIMER'S DISEASE AND PS1-E280A

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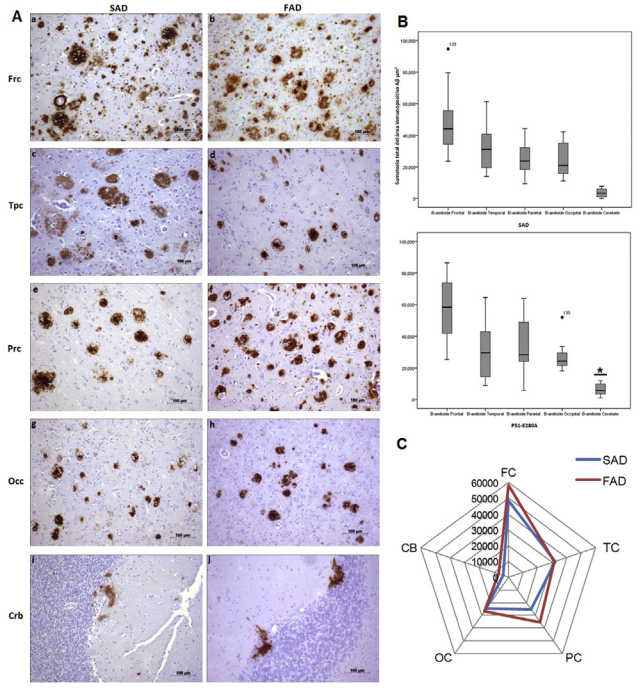
Background: In this descriptive study of a case series a comparison was made of neuropathological, neuropsychological and imaging characteristics from two groups of patients with Alzheimer's Disease (AD). One group with sporadic AD (SAD) and another group with familial AD (FAD). **Methods:** For that, 460 deparaffinized slides from brain tissue were obtained, together with clinical and imaging registries from the Brain Bank of the Neurosciences group of the University of Antioquia. **Results:** In the neuropathological comparison it was observed that SAD presents more but smaller A β deposits in temporal and occipital cortex compared with FAD. Also, SAD shows correlation between disease duration and neurofibril pathology in temporal cortex. FAD cases present larger deposits in frontal, temporal, parietal and cerebellar areas; together with higher A β immunosignal in the cerebellum. Regarding hyperphosphorylated Tau (pTau) quantification, FAD presents with higher levels in frontal, parietal, occipital and cerebellar areas. Also, It was observed correlations between brain weight, disease duration and pTau in temporal cortex in FAD. In the neuropsychological comparison there were significant differences in total scores for memory, particularly in immediate and long term recovery in a word list task. Imaging comparison showed no differences in cortical atrophy, hippocampal atrophy, fimbriosubicular distance and interuncal distance. **Conclusions:** In conclusion, there are neuropathological differences between FAD and SAD that could be related with the physiopathology of this hereditary variant of AD.



P2-017 THE EFFECT OF KAMI-UNTAN-TOU ON BETA-AMYLOID PROTEIN-INDUCED APOPTOSIS IN PC12 CELLS

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Background: Kami-Untan-Tou (KUT, Kracie: and Co., Tokyo, Japan) has been reported to improvement of the cognitive deficit of Alzheimer's disease (AD) in AD patients. The association between neuron death in AD and apoptosis has attracted attention, and studies in cultured cells have suggested that β-Amyloid protein induces cell death by apoptosis. A pathway for the induction of apoptosis is caspase cascade activation. In addition, caspase-3 activation due to β40-induced injury in PC12 cells has been reported. The protective effect of a Japanese herbal medicine, Kami-Untan-Tou (KUT) were investigated on beta-Amyloid protein (β40) -induced apoptosis in PC12 cells. **Methods:** Two biochemical methods, determination of the lactate dehydrogenase (LDH) release and activity of 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT) reduction of cells, were used. We evaluated the ROS activity using a H 2 DCFDA (Molecular Probes), and we also evaluated the caspase-3 activity of KUT on β40-induced apoptosis in PC12 cells using a fluorophotometer (Type 850, HITACHI). **Results:** KUT significantly inhibited the increase in LDH release following β40-induced cell injury and significantly increased MTT reduction, significantly increasing the cell survival rate. These results suggested the protective effects of KUT. In addition, the inhibition of β40-induced cell injury and the significant increase in the cell survival rate by KUT were continuous, suggesting continuous protective effects. KUT significantly inhibited ROS and caspase-3 activation due to β40-induced cell injury. **Conclusions:** In summary, the administration of KUT increased the cell survival rate, which suggested its protective effects on neurons. KUT significantly inhibited β40-induced cell injury, which suggested its protective effects against neuron injury. These results suggest a pathway via ROS and a pathway via caspase-3 activations are one of the mechanisms of the protective effect of KUT.

P2-018 SULFORAPHANE PROMOTES THE DEGRADATION OF PHOSPHORYLATED TAU VIA THE INDUCTION OF AUTOPHAGY

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Background: Tau becomes aberrantly phosphorylated and oligomerized which likely contributes to neuronal dysfunction and death in neurodegenerative diseases such as Alzheimer's disease (AD). The mechanism involved in clearing pathological, phosphorylated tau has not yet been fully defined. T here is a growing interest in understanding the processes by which pathological, phosphorylated tau is removed from neurons. **Methods:** CN1.4 mouse cortical neurons stably expressing inducible tau in the presence of doxycycline were treated with sulforaphane (SFN), anisothiocyanate obtained from a variety of cruciferous vegetables for indicated times. The levels of phosphorylated tau, tau or LC3-II, a key autophagy marker, were analyzed by immunoblotting. The autophagic vesicles were analyzed using transmission electron microscope (TEM). **Results:** SFN decreased the levels of phosphrylated tau via the induction of autophagy in neuronal cells. Treatment of neuronal cells with SFN not only increased the levels of LC3-II, but also decreased the levels of phsophorylated tau which occurred concurrently with ERK activation. Interestingly, blockade of ERK activation by pretreatment with the specific MEK inhibitors, PD184352 or U0126, inhibited the SFN-induced increase in LC3-II levels. Pretreatment with NAC (N-acetyl-L-cysteine), a well-known antioxidant, completely blocked the SFN-induced increase in LC3-II levels, the activation of