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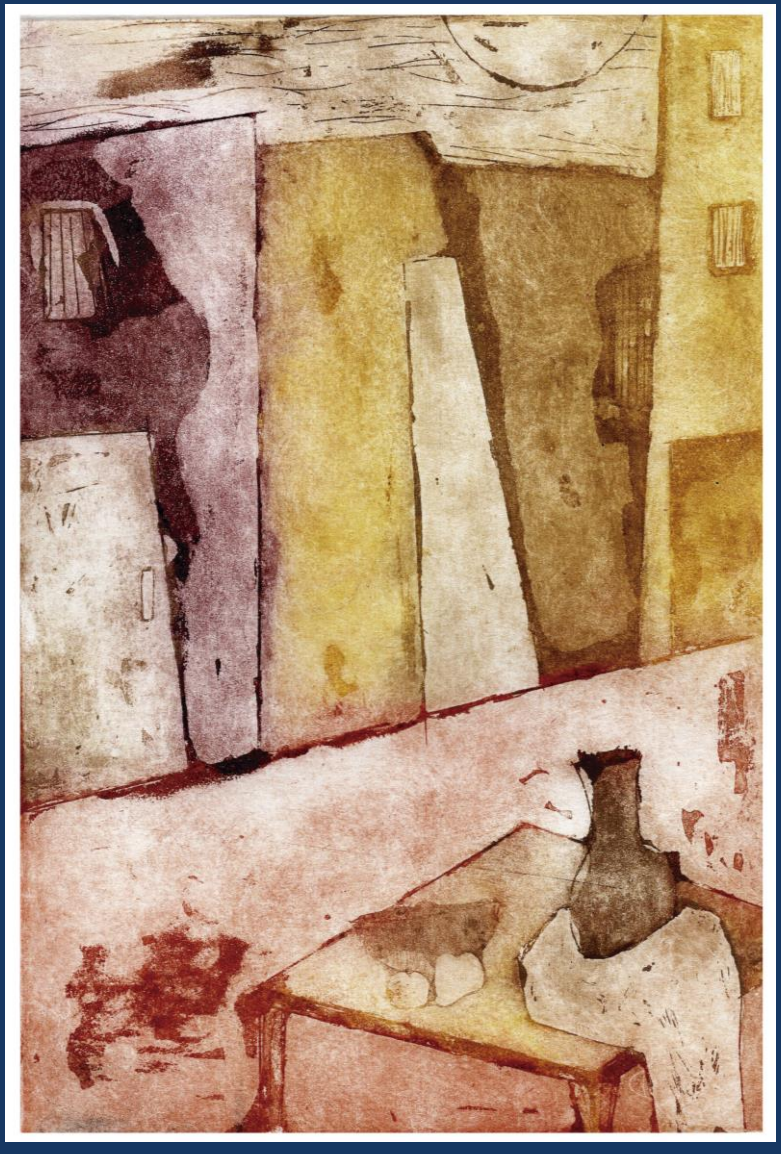

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medicina

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La Tapa (Ver pág. 4)
Atardecer en la tarde
Antonella Ricagni

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features of NMDARs is the high specificity of their antagonists, making them important targets for drug design. The aim of this work is to synthesize and evaluate in vitro the neuroprotective effect of two additional peptides (EAR-17 and EAR-19). The methodological design has three general aspects as follows: In-silico design and molecular coupling of the two peptides with the GluN2B subunit of the NMDAR. Synthesize the two peptides by solid phase and subsequently characterize, purify and determine their secondary. Electrophysiological evaluation in tsA 201 (HEK293T) and hippocampal neurons. The neuroprotection of peptides EAR-17 and EAR-19 in the oxygen-glucose deprivation model (OGD), for which primary cultures of hippocampal neurons were used to perform a molecular approach through the activation of caspase-3 and calcium imaging techniques. As a result of the docking of the peptides EAR-17 and EAR-19 with the GluN2B subunit of the NMDAR, it was found that the affinity of the peptides varies according to the receptor conditions. In addition to the interactions established between the peptides and the GluN2B subunit, there are dipole to dipole electrostatic interactions, among which the hydrogen bond predominates. In general, the process of synthesis and purification has been optimal, allowing to have pure peptide species, with a reaction efficiency greater than 50 %. This great efficiency confirms a synthesis process without the presence of adducts of the peptides. The electrophysiological evaluation confirmed the antagonistic effect of the EAR 17 and EAR 19 peptides on the current evoked by the NMDAR. For each peptide, the IC 50 was established, finding that the EAR 19 has more affinity for the NMDAR. In the EPSC assay, EAR-17 and EAR-19 decreased in the postsynaptic current on the hippocampal neurons, without recovering the initial current of the EPSCs although they are repeated continuously in the same period of time. The vehicle has no action on hippocampal neurons. In neuroprotection, the OGD-model in hippocampal neurons activated the path of cell death by caspases, which is a consequence of the irregular entry of calcium mediated by the NMDAR. At the same time, we observed significant changes when the OGD was performed with or without the presence of the peptides, with significant differences observed if the peptides were added at the beginning of the OGD or in the OGD recovery process. In this study, we identified two peptidic molecules called EAR-17 and EAR-19 that have the possibility of being antagonists of the GluN2B subunit of the NMDAR and therefore the possibility of modulating the permeability to the calcium ion inducing a neuroprotection effect. We suggest continuing the evaluation of EAR-17 and EAR-19 with other in-vitro and in-vivo approaches.

0793 - NEW FINDINGS FOR OLD PLAYERS: IMIDAZOLIUM SALTS AS PROTECTIVE DRUGS IN C. ELEGANS MODELS OF STRESS AND NEURODEGENERATION

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Abstract/Resumen: Imidazolium salts are attractive pharmacological agents that have been linked to a wide range of biological effects, including antitumoral, antimicrobial, anthelmintic and anti-inflammatory. In this study, we aim to evaluate the role of these compounds as antioxidant and anti-aging agents. We synthesized imidazolium salts and analyzed their ability to improve oxidative stress (OS) resistance. We used an established model in biomedical research, the free-living nematode C. elegans, and exposed them to the oxidizing agent $FeSO_4$. We identified a derivate, 1-Mesithyl-3-(3-sulfonatopropyl) imidazolium (MSI), that enhances animal resistance to OS. To delineate MSI roles, we split this work into two goals: i) to describe MSI action mechanisms and, ii) to evaluate MSI role in neurodegenerative models. To gain insight into its mechanism of action, we evaluated MSI ability to activate DAF-16 (FOXO in vertebrates), a transcription factor relevant for cytoprotective defense mechanisms. Unexpectedly, our experiments revealed that MSI stress protection was not dependent on DAF-16. These results support the idea that other transcription factors (such as SKN-1 (Nrf-2 in vertebrates), HSF-1), could be involved in MSI protection. We are currently performing experiments to identify the role of these molecular players, in MSI-induced stress resistance. The second goal is held by the theory that links OS to aging and neurodegeneration. We are currently evaluating if MSI increases lifespan, healthspan, and improves biological markers of neurodegeneration in a C. elegans model of Alzheimer disease. This strain expresses Aß1-42 in muscle and shows age-dependent protein aggregation and paralysis. Our preliminary results show that MSI delays paralysis in this strain. Additional research is needed to underpin the protective role of MSI and to determine if these effects can be extrapolated in other neurodegenerative scenarios.

0968 - EVALUATION OF SERUM CYTOKINES LEVELS AS POTENTIAL PERIPHERAL MARKER OF MILD COGNITIVE IMPAIRMENT IN WOMEN.

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Abstract/Resumen: Mild cognitive impairment (MCI) is a transitional stage between cognitive changes of normal aging and early-stage dementia. MCI is recognized as a pathological condition that typically precedes Alzheiner´s disease. A fraction of 20-40 % of MCI individuals will progress to dementia within 3 years following the initial diagnosis. Previously, we demonstrated that the cognitive deficit observed in Balb/c mice exposure to chronic mild stress was correlated with a decrease of IFN-g and an increase of IL-4 in hippocampus and peripheral lymph nodes. The aim of this work was evaluated the levels of cytokines IFN-g, IL-1b, IL-4 and IL-6 in serum of subjects with MCI and control as a possible peripheral marker of cognitive deficit. In this pilot study participated six female subjects (aged 60-70 years) from Junin´s city. The participants were randomly selected taking into account the inclusion and exclusion and criteria according to the protocol approved by the COENOBA and the Central Ethics Committee of the Province of Buenos Aires (exp. 2,919-1,593/17). The subjects were evaluated by neuropsychological tests following the diagnostic criteria of Petersen. Criteria: the control group showed scores in all cognitive functions evaluated in normal range for patient age and schooling (Z score between -1 and 1). The MCI group (MCI of the amnesic type) showed values equal or less than Z -1.5 in the memory tests. The cytokines levels (pg/ml) were measure in serum by ELISA. We found an increase in IL-4 level (C: 0.7 ± 0.4; MCI: 7.0 ± 1.7; $p<0.05$) in MCI respect to control subjects. We did not find a significant difference in IFN-g (C: 51.4 ± 11.5; MCI: 38.3 ± 2.1), IL-1b (C: 13.9 ± 1.7; MCI: 19.0 ± 2.3) and IL-6 (C: 5.7 ± 1.6; MCI: 7.3 ± 2.7) between the MCI group compared to control group. We conclude that changes in these cytokines serum levels could be related to the cognitive deficit observed in MCI subjects.

Medicina Regenerativa y Terapia celular/ Regenerative Medicine and Cell Therapy I

Chairs: Andrea Fellet | Daniel Grasso

0125 - REJUVENATION BY PARTIAL CELL REPROGRAMMING: TRANSFER OF THE YAMANAKA GENES TO FIBROBLASTS FROM YOUNG AND OLD RATS