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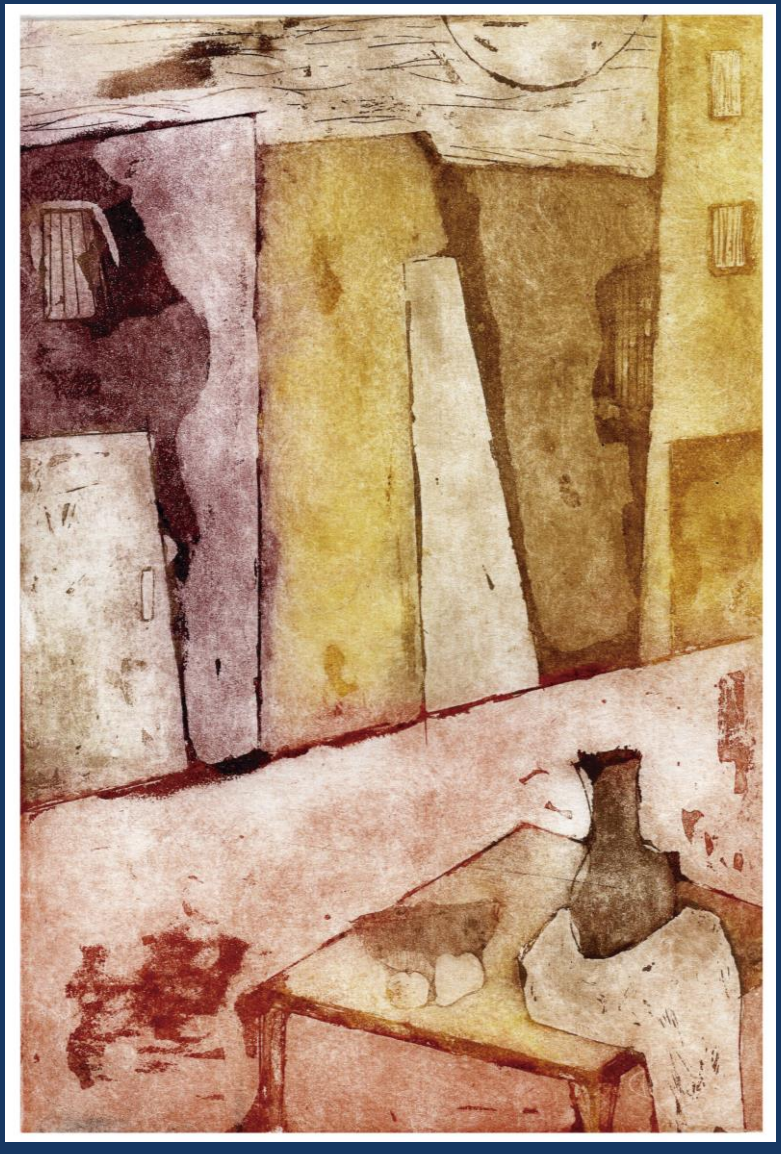

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La Tapa (Ver pág. 4)
Atardecer en la tarde
Antonella Ricagni

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Directores Responsables:
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Secretaría de Redacción: Ethel Di Vita, Instituto de Investigaciones Médicas Alfredo Lanari, Combatientes de Malvinas 3150, 1427 Buenos Aires, Argentina
Tel. 5287-3827 Int. 73919 y 4523-6619
e-mail: revmedbuenosaires@gmail.com – http//: www.medicinabuenosaires.com

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IX Reunión Anual de la
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(NANOMED-ar)

VI Reunión Científica Regional de la Asociación Argentina de Ciencia y Tecnología de Animales de Laboratorio (AACyTAL)

con la participación de
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13 - 16 de noviembre de 2019
Hotel 13 de Julio - Mar del Plata

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25 µM forskolin reduced dex-induced GR activity in a 30 % ($p<0.05$). This discrepancy indicates that H2r regulation of GR activity is not strictly mediated by cAMP pathway, suggesting the involvement of other signaling partners. It has been described that H2r activation with amthamine also triggers ERK1/2 phosphorylation. In fact, treatment with the MEK inhibitor UO126 prevented amthamine potentiation of GR activity, pointing to ERK as a relevant player in the potentiation effect. Moreover, pretreatment with 10 µM of the H2r inverse agonists, famotidine and ranitidine, both of which decrease cAMP levels and increase ERK phosphorylation, boosted dex-induced GR activity to almost the quadruple. Trying to elucidate the role of other signaling proteins, cells were transfected with Rap-GAP, an inactivator of the small G-protein Rap. In this system, amthamine also lost its potentiating effect. The whole of our results points to a dual parallel regulation of the GR transcriptional activity: an inhibitory effect mediated by cAMP and an enhancing effect mediated by Rap and ERK proteins. Considering the co-expression of H2r and GR in several physiological systems and the widespread use of their ligands, the interaction described herein could have an impact on glucocorticoid based therapy and grants further research.

0810 - TREATMENT OF IRRADIATED MICE WITH ORAL RADIOPROTECTOR ATTENUATES INSULT.

Veronica MARTINEZ MARIGNAC (1) | Leonel MONDRAGON(2) | Lucia CERVANTES(3) | Gloria OERTLIN(1) | Fernanda CANTERO(3) | Jose Luis FAVANT(1)

IBIOGEM, CICYTTP (CONICET, ENTRE RIOS PROVINCE AND UADER) (1); RIVER PLATE ADVENTIST UNIVERSITY- UAP (2); NATIONAL UNIVERSITY OF ENTRE RÍOS, UNER, ENGINEERING FACULTY. (3)

Abstract/Resumen: Ionizing radiation directly affects DNA structure by inducing primarily DNA double strand breaks (DSBs), and secondarily production of reactive oxygen species (ROS) that oxidize proteins, lipids, and also induce several different damages to DNA, like generation of abasic sites and single strand breaks (SSB). Consequently, all these changes induce cell death and mitotic failure. The important use of IR in X ray exams and in radiotherapy and its undesirable effects took us to validate a murine model in order to evaluate DNA damage of X Rays and characterize natural and food supplements compounds with radio mitigation properties. Essiac Genuine tea has been used widely in the homeopathy market as a popular anticancer and antioxidant tonic. Due to the reported ROS scavenging properties of Essiac formula, we evaluate DNA damaged mitigation in 50 male Balb/c mice under 25-100m Sv-Gy, which is an average effective dose received by most X Ray exams during a year of radioimaging services by its personnel. The tea formula resulted in a significant reduction of DNA damaged of mice under the formula evidenced by Comet Assay ($p<0.01$) and acridine orange assay for micronuclei and DNA fragmentation evaluation ($p<0.02$) as well as in a normalization of the complete blood count (CBC). The tea did not show any cytotoxicity at the used doses, glucose and animal weight was similar between treatments. We not only demonstrated that Essiac tea is not toxic and acts as a radioprotector of IR X rays at doses to which are exposed the X ray personnel though we also optimized a murine model for further analysis of other natural compounds and supplements (e.g. Ascorbic Acid).

0819 - ESSENTIAL OILS AS SOURCES OF POTENTIAL ANTHELMINTIC COMPOUNDS TESTED ON THE MODEL ORGANISM CAENORHABDITIS ELEGANS

Guillermina HERNANDO | Ornella TURANI | Cecilia BOUZAT

CCT-INIBIBB, DEPARTAMENTO DE BBYF, UNS-CONICET

Abstract/Resumen: Parasitic nematodes are of major significance as human pathogens and have important economic impact worldwide due to considerable losses in livestock and food crops. Drug treatment of nematode infections are the pillar of worm control in human and veterinary medicine. Due to the appearance of drug resistant nematodes, there is a need of developing novel drugs. As parasitic nematodes are not ideal laboratory animals, the non-parasitic nematode Caenorhabditis elegans, has emerged as a model organism for drug discovery. Essential oils (EOs) are natural products produced by aromatic plants. EOs are complex mixtures that usually contain two or three major phytochemicals, which can be terpenes and/or aromatic compounds. We used paralysis assays of wild-type and mutant C. elegans strain and electrophysiological recordings to identify EO with potential anthelmintic activities, reveal the active components, the target sites and mechanisms of action. We found that EOs belonging to six different orders produced rapid paralysis of C. elegans and we establish the half maximal effective concentration values between 0.02-1.2 percent of EOs. We also found that all EOs inhibit egg hatching. Thus, EOs can mediate both rapid and long-term anthelmintic effects. We determined that trans-cinnamaldehyde (TC), a major component of C. verum EO, produces both paralysis and egg-hatching inhibition. By testing mutant worms, we identified that muscle L-AChR and GABA receptors are EO and TC targets in vivo. Electrophysiological studies from C. elegans cultured muscle cells identified the mechanism underlying the antiparasitic effect. Thus, by modulating two receptors with key roles in worm motility, these EO emerge as novel sources of anthelmintic compounds.

0864 - UNRAVELING THE MOLECULAR MECHANISM OF DII, A NEW ANTHELMINTIC DRUG

Sebastián GIUNTI | Pamela AZCONA | Gabriela BLANCO | Gustavo SILBESTRI | Diego RAYES | María José DE ROSA

INIBIBB-CONICET, DEPTO. BIOLOGÍA, BIOQUÍMICA Y FARMACIA-UNS

Abstract/Resumen: Nematode parasites cause substantial morbidity to billions of people and considerable losses in livestock and food crops. The repertoire of effective anthelmintic compounds is very limited, as drug development has been delayed for decades. By using C. elegans as a model for parasitic nematodes, we previously identified a new imidazole derivative, diisopropylphenyl-imidazole (DII), as a promising candidate for anthelmintic agent. DII lethal effects rely on a previously unidentified muscle nicotinic receptor (AChR), different from the classical levamisole-sensitive AChR. This novel AChR is composed by UNC-29 (a non-alpha subunit incapable of forming homomeric receptors) and other unidentified subunits. To elucidate its stoichiometry, we performed an initial screening of strains containing null mutations in different AChR subunits. By exposing these animals to DII (600 µm), we found a null mutant in acr-23 (an alpha nicotinic subunit) that is even more resistant to DII than UNC-29 null mutants. Since the mutants used in the initial screening had not been outcrossed to the wild-type (wt), we performed this outcross four times, selecting (by genotyping) those animals that contain the deletion in acr-23. Surprisingly, these outcrossed animals are as sensitive to DII as the wt. Moreover, when we outcrossed the original mutant strain to the wt selecting by their resistance to DII, we obtained animals that contain wild-type acr-23 alleles. This strongly suggests that another mutation, different from acr-23 deletion, causes the DII resistance. The drug resistance of these mutants appears to be DII-specific, as it is as sensitive to the classic anthelmintic levamisole as the wt. We are now focused on determining the gene that underlies this DII resistant phenotype. Parasite resistance to traditional nematocidal drugs has become a global concern. Therefore, the identification of new anthelmintics with novel targets, as DII, is mandatory to circumvent this growing problem.

0955 - NEBIVOLOL AND N-ACETYLCYSTEINE IN A MODEL OF GENETIC HYPERTENSION.