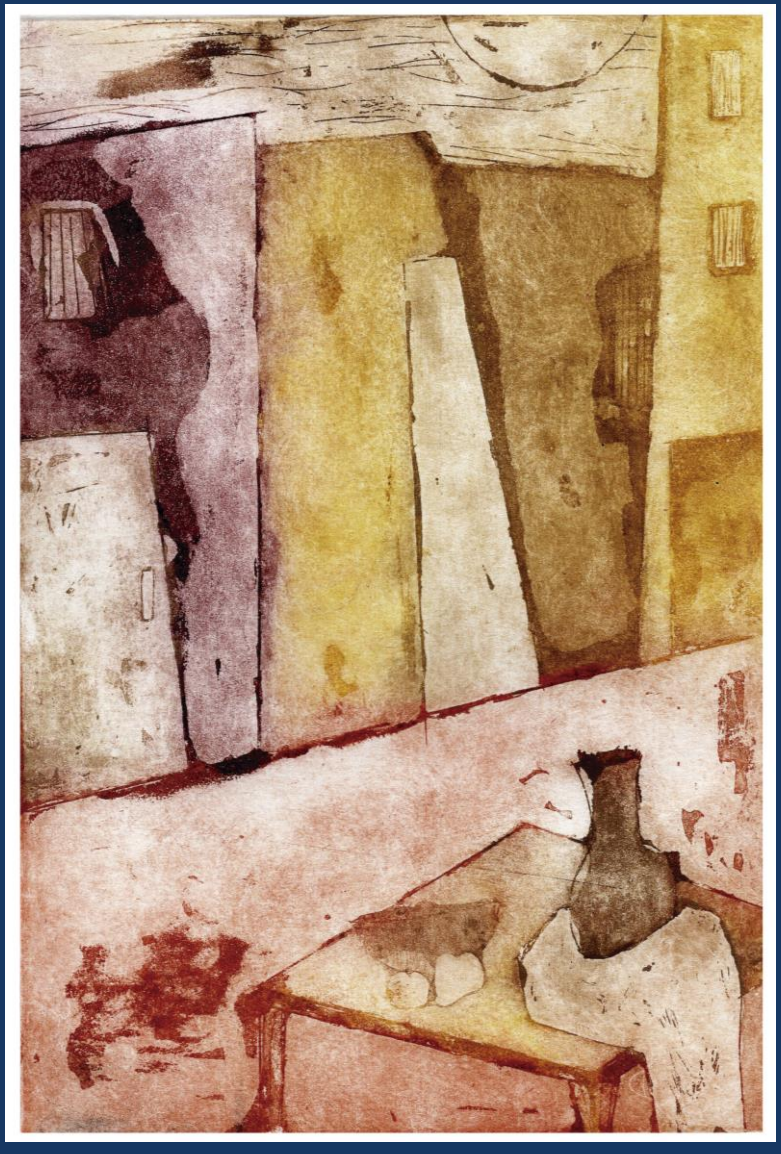
2019

medicina

BUENOS AIRES VOL. 79 Supl. IV - 2019

80° Aniversario



MEDICINA

Volumen 79, Supl. IV, págs. 1-338

medicina

BUENOS AIRES, VOL. 79 Supl. IV - 2019

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

MEDICINA (Buenos Aires) – Revista bimestral – ISSN 0025-7680 (Impresa) – ISSN 1669-9106 (En línea)

REVISTA BIMESTRAL
Registro de la Propiedad Intelectual N° 02683675
Personería Jurídica N° C-7497
Publicación de la Fundación Revista Medicina (Buenos Aires)
Propietario de la publicación: Fundación Revista Medicina
Queda hecho el depósito que establece la Ley 11723

Publicada con el apoyo del Ministerio de Ciencia, Tecnología e Innovación Productiva.
MEDICINA no tiene propósitos comerciales. El objeto de su creación ha sido propender al adelanto de la medicina argentina. Los beneficios que pudieran obtenerse serán aplicados exclusivamente a este fin.
Aparece en *MEDLINE (PubMed), ISI-THOMSON REUTERS (Journal Citation Report, Current Contents, Biological Abstracts, Biosis, Life Sciences), CABI (Global Health), ELSEVIER (Scopus, Embase, Excerpta Medica), SciELO, LATINDEX, BVS* (Biblioteca Virtual en Salud), *DOAJ, Google Scholar y Google Books.*
Incluida en el Núcleo Básico de Revistas Científicas Argentinas del CONICET.

Directores Responsables:
Basilio A. Kotsias, Eduardo L. De Vito, Isabel Narvaiz Kantor, Guillermo B. Semeniuk

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

Vol. 79, Supl. IV, Noviembre 2019

medicina
BUENOS AIRES, VOL 79 Supl. IV - 2019

REUNIÓN ANUAL DE SOCIEDADES DE BIOCIENCIA 2019

LXIV Reunión Anual de la
Sociedad Argentina de Investigación Clínica (SAIC)

LI Reunión Anual de la
Asociación Argentina de Farmacología Experimental (SAFE)

XXI Reunión Anual de la
Sociedad Argentina de Biología (SAB)

XXXI Reunión Anual de la
Sociedad Argentina de Protozoología (SAP)

IX Reunión Anual de la
Asociación Argentina de Nanomedicinas
(NANOMED-ar)

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

con la participación de
The Histochemical Society

13 - 16 de noviembre de 2019
Hotel 13 de Julio - Mar del Plata

EDITORES RESPONSABLES

Dra. Mónica Costas
Dra. Gabriela Marino
Dr. Pablo Azurmendi

transcript. Morphological analyses and live imaging axonal transport indicate that perturbations in the tau 3R:4R ratio in human neurons impaired axonal transport dynamics without altering neuronal morphology. In a mouse model of tauopathy (htau mice) local modulation of E10 inclusion in the prefrontal cortex improved cognitive deficit, restored neuronal firing patterns and reduced insoluble and hyperphosphorylated tau contents. Moreover, local shifting of 3R to 4R tau in the striatum improved motor coordination déficits in htau mice.
Together, our results evidence some of the (dys)functional consequences of tau 3R:4R imbalance and rise the potential use of RNA reprogramming to correct tau *mis*-splicing in human tauopathies.

NANOCLUSTER ORGANIZATION AND DYNAMICS OF SYNAPTIC PROTEINS IN SYNAPTOPATHIES

FRANCISCO BARRANTES

Laboratory of Molecular Neurobiology, Institute of Biomedical Research, UCA-CONICET. Buenos Aires, Argentina.

Synaptic transmission relies on an adequate balance of receptor synthesis, delivery to and removal from the cell membrane and anchorage by scaffolding and cytoskeletal components. Alteration of this homeostatic balance is at the root of various neurodegenerative diseases affecting the peripheral and central nervous synapses. In order to understand the interplay between the intervening molecules, it is necessary to define their supramolecular organization, dynamics and trafficking. Two independent superresolution microscopy techniques -STED and STORM- provide complementary information on the static supramolecular organization of neurotransmitter receptors and scaffolding proteins -often occurring in nanometer-sized aggregates ("nanoclusters") in central and peripheral synapses. These can be imaged with nanometer precision and the density, number of molecules per cluster and other structural parameters defined. Furthermore, the mobility of the synaptic proteins can be followed in living cells using single-particle tracking and nanoscopy. The alterations occurring in neurodegenerative or autoimmune synaptopathies will be exemplified.

SAIC SYMPOSIUM III

NUTRITION, METABOLISM, GENETIC, SOCIAL AND CULTURAL HABITS AS DETERMINANTS FOR ILLNESS VULNERABILITY

Chairs: Mariana Tellechea / Adriana Fraga

LIPIDS AT THE CROSSROAD OF α-SYNUCLEIN FUNCTION AND DYSFUNCTION: NEW INSIGHTS INTO NEURODEGENERATION

GABRIELA SALVADOR

Biochemical Research Institute of Bahía Blanca, INIBIBB-UNS-CONICET. Department of Biology, Biochemistry and Pharmacy. National University of the South. Bahía Blanca, Buenos Aires, Argentina.

Since its discovery, the study of the biological role of α-synuclein has been the subject of increasing interest. Its aggregation and accumulation in intracellular structures called Lewy bodies are a hallmark of a class of neurodegenerative disorders grouped as synucleinopathies, of which Parkinson´s disease is the most prevalent. The different ways in which α-synuclein interacts with lipids are not only an intriguing characteristic but also an open question related with its biological function and pathogenesis. In our lab, we are mainly focused on the study of lipid signaling and metabolism in different models of neuronal injury.
Phosphatidic acid, a bioactive lipid produced by the activation of Phospholipase D (PLD), governs multiple signaling pathways. We have previously demonstrated that PLD pathways are involved in neuronal degeneration and are, in particular, associated with synaptic injury induced by oxidative stress and inflammatory response. Based on these findings and taking into account the intersections between α-synuclein and lipid biology, we have recently investigated the role of PLD signaling in a synucleinopathy cellular model. The overexpression of wild type (WT) α-synuclein was found to trigger an inhibition of phosphatidic acid production through PLD1 downregulation as well as a decrease in ERK1/2 phosphorylation. Moreover, ERK1/2 subcellular localization and nuclear sequestration were shown to be modulated by the overexpression of α-synuclein in a PLD1-dependent manner. In addition to the changes observed in PLD signaling, neuroblastoma cells expressing WT α-synuclein were found to exhibit a degenerative-like phenotype characterized by a marked reduction in the neurofilament light subunit (NFL). This NFL loss has also been reported in studies performed in post-mortem brains from Lewy body dementia. The gain of function of PLD1 through the overexpression of its active form had the effect of restoring NFL expression in WT α-synuclein neurons.
Lipid metabolism was also altered in neurons overexpressing several forms of α-synuclein (WT or the mutant A53T). The most conspicuous evidence supporting a metabolic switch induced by the different forms of α-synuclein was the presence of lipid droplets. The accumulation of lipid droplets is a rare and unusual entity for the neuronal phenotype. In this respect, WT α-synuclein overexpression was observed to

trigger the nuclear localization of the lipogenic transcription factor SREBP-2 and enhancers of protein aggregation (manganese and bortezomib) were found to increase lipid droplets content. WT α-synuclein overexpression also induced Acyl-CoA synthetase activation, which explained, at least in part, the increase in triacylglycerol (usually stored in lipid droplets). The pharmacological inhibition of triacylglycerol synthesis turned neurons more vulnerable to the presence of WT α-synuclein.

Taken together, our findings reveal unforeseen roles for α-synuclein in lipid biology, namely i) PLD1 downregulation associated with NF loss and ii) a metabolic switch with increased triacylglycerol content. Both the decrease in phosphatidic acid levels by PLD1 inhibition and the increase in lipid droplets could be considered as early markers of the neurodegenerative process triggered in synucleopathies.

GLOBAL AND LOCAL NUTRITIONAL SITUATION: EFFECTS ON THE BURDEN OF CHRONIC DISEASES

ANABEL PALLARO

Professor of "Nutrition" Faculty of Pharmacy and Biochemistry. UBA. Buenos Aires, Argentina.

Noncommunicable diseases (NCDs) are the result of genetic, physiological, environmental and behaviours factors. NCDs are the biggest cause of death worldwide; 36 million die annually (63 % of global deaths), including 14 million before 70 y (WHO). Ninety % occur in low and middle-income countries and could have been prevented as they are linked to common causes as unhealthy diet, physical inactivity, tobacco and alcohol use. Thirty nine and 13% people over 18 years were overweight and obese in 2016, respectively; meanwhile, the prevalence of overweight and obesity among children/adolescents aged 5-19 y has risen dramatically from 4 % in 1975 to over 18 %. UN Agenda for Sustainable Development recognizes NCDs as a major challenge for sustainable development and committed to reduce one-third premature mortality from NCDs. In Argentina, ENNYS1 Survey (MOH,2004-5) demostrated that 40 % of children 6-72 months and 44 % of women 19-49 y showed excess weight, 34.1 % (6-23 mo), 9% (24-72 mo) and 18 % (19-49 y) anaemia and 14.3 % (6-23 mo) vitamin A deficiency. ENNYS2 Survey (2018-9) recently showed 41.1 % excess weight in boys and girls 5-17 y and 67.9 % in adults over 18 y and pointed out that it was 21 % higher in the lower income quintile. Exclusive breastfeeding, which assures food security, was low (43.7 %, ENNYS2).

As OMS defined obesity as excessive fat accumulation that may impair health, we found an increase in fat mass associated with childhood obesity by deuterium dilution technique in community studies; furthermore, it was also found in normal-weight children. Considering that excessive fatness may increase dyslipidemia and insulin resistance, its evaluation is useful to identify children at metabolic risk. Besides, the measurement of breast milk intake suggests that non-exclusive breastfed children (4 mo) may receive higher energy and protein intake and contribute to obesity. Additionally, the urinary sodium addressed for the excessive sodium intake in adults. The assessment of these less frequently evaluated risk factors would contribute as a tool for better diagnosis of NCDs.

DIABETES: ITS DEVELOPMENT AND SOCIOECONOMIC IMPACT AND HOW CAN BE EFFECTIVELY TACKLE THE PROBLEM

JUAN JOSÉ GAGLIARDINO

CENEXA. Centro de Endocrinología Experimental y Aplicada (UNLP-CONICET-CEAS CICPBA). Facultad de Ciencias Médicas UNLP. La Plata, Buenos Aires, Argentina

In the period 2005-2018 diabetes prevalence in Argentina increased 51 % attaining a value of 12.7 % in adult population. While traditionally its development was geared by a strong genetic component, the role of epigenetic factors has recently showed its important role. Among the latter, central obesity, different metabolic dysfunctions, sedentarism, poverty and low education level are just some of the factors that triggers the epigenetic components.

Late diagnosis of the disease, inappropriate treatment prescriptions and low patient´ adherence to their treatment allows the development of preventable diabetes chronic micro and macroangiopathic complications that increase the cost of treatment and decrease quality of life of people with diabetes. Otherwise, while microangiopatic complications which affect the retina, kidney and nerves are responsible of serious disabilities, the macrongiopatic ones (CVA, acute myocardial infarcts and lower limb amputations) are the main responsible of the patients' death. In this regard, despite new drugs and devices contributed to facilitate the control of diabetes and its associated cardiovascular risk factors (CVRF), less than half of the people with the disease attain treatment goals capable to prevent the development and progression of such complications, thus a large percentage of the diabetes population develop the above mentioned complications.

The picture described demonstrates that probably the health care team strategies currently implemented are not appropriate to tackle efficiently the diabetes problem. Thus, perhaps is the time to change the current diabetes