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Daniela Kantor

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REUNIÓN CONJUNTA SAIC SAI SAFIS 2018

LXIII REUNIÓN ANUAL DE LA

SOCIEDAD ARGENTINA DE INVESTIGACIÓN CLÍNICA (SAIC)

LXVI REUNIÓN ANUAL DE LA

SOCIEDAD ARGENTINA DE INMUNOLOGÍA (SAI)

REUNIÓN ANUAL DE LA

SOCIEDAD ARGENTINA DE FISIOLOGÍA (SAFIS)

CON LA PARTICIPACIÓN DE

SOCIEDAD ARGENTINA DE VIROLOGÍA (SAV)

ASOCIACIÓN ARGENTINA DE NANOMEDICINAS (NANOMED-ar)

14-17 de noviembre de 2018
Hotel 13 de Julio – Mar del Plata

EDITORES RESPONSABLES

Claudia Pérez Leirós
Pablo Baldi
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JOINT MEETING SAIC SAI SAFIS 2018

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November 14-17, 2018
Hotel 13 de Julio – Mar del Plata

RESPONSIBLE EDITORS

Claudia Pérez Leirós
Pablo Baldi
Alberto Crottogini

LA TAPA

Los palos rosas, 2015

Daniela Kantor

Técnica: Acrílico sobre bastidor. Medidas: 35 x 70 cm

Daniela Kantor es diseñadora gráfica (FADU-UBA), historietista, ilustradora y pintora. Desde 2014 es docente en la materia Ilustración, cátedra Roldán, FADU, y da talleres para niños (Filbita 2017, taller de comics librerías Matilda-Tigre, taller de historietas CCK, etc.) Estudió con el maestro Alberto Breccia dibujo de historieta y con Carlos Gorriarena realizó el Curso de color. Asistió al Taller de acuarela y pastel de Carlos Nine y realizó clínicas de pintura con Mariano Sapia y Tulio de Sagastizábal. Además de ilustrar muchos libros para niños y adolescentes (Editoriales Troquel, Abran Cancha, Puerto de Palos, Santillana, etc.), es parte de la revista de historietas El tripero, publica en revistas (Barcelona, Zona de obras, Crisis, suplemento Ñ, entre otras). Publicó su primera novela gráfica: Mujer primeriza (2014). Su proyecto de segundo libro de historietas Naturalella obtuvo la primera mención del Premio Nueva Historieta Argentina (2016) y fue publicado en parte en Dis-tinta, el compilado de Liniers y Martín Pérez (Ed. Sudamericana, 2016). Expone sus pinturas desde 2003; recientemente exhibió en Cic.edu.ar

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PALABRAS DE BIENVENIDA

Estimados colegas y amigos,

Nos complace darles la bienvenida a la Reunión Conjunta SAIC SAI SAFIS 2018 de la **Sociedad Argentina de Investigación Clínica (SAIC),** la **Sociedad Argentina de Inmunología (SAI)** y la **Sociedad Argentina de Fisiología (SAFIS),** que este año también cuenta con la participación de la **Sociedad Argentina de Virología (SAV)** y la **Asociación Argentina de Nanomedicinas (NANOMED-ar).**

El Programa Científico es abarcador y cubre los aspectos más sobresalientes e innovadores de las diferentes disciplinas. Contamos con la presencia de investigadores argentinos y extranjeros de la mayor jerarquía internacional que expondrán los avances de su trabajo en conferencias y simposios. Además, se han inscripto más de 760 trabajos de estudiantes de doctorado, becarios, investigadores, médicos residentes y otros profesionales del ámbito de la Salud con los últimos resultados de sus investigaciones, los que serán expuestos en forma de comunicaciones orales y pósters. Se han seleccionado algunos de estos trabajos para su presentación en simposios para favorecer el intercambio con los pares extranjeros. Asimismo, Jurados de expertos han pre-seleccionado trabajos para competir por distintos premios: se otorgarán los Premios León Cherny al mejor trabajo multidisciplinario, Honorio Bigand al mejor proyecto presentado por investigadores jóvenes, Eduardo Soto al mejor trabajo en Neurociencias, Irene Faryna de Raveglia en Oncología, Leonardo Satz en Inmunología, SAFIS Jóvenes Investigadores en Fisiología, Camilión de Hurtado en Fisiopatología Cardiovascular y César Milstein en Enfermedad de Chagas. Se otorgará un premio de la American Society for Microbiologists en el área de Infectología y Menciones a los mejores pósters por áreas de la SAIC. Estos premios constituyen un estímulo para los grupos de investigación argentinos que mejoran la calidad de sus trabajos año tras año y se otorgan merced al generoso aporte de las fundaciones Cherny, Bigand, de la Dra Pasquini, de la Familia Camilión de Hurtado y de las empresas ETC Internacional y Novartis Argentina SA. Habrá también minicursos, encuentros con expertos y exposición comercial.

El principal objetivo de esta Reunión Conjunta es ofrecer a los asistentes el marco académico propicio para alentar la interacción entre científicos argentinos y con pares extranjeros que investigan las bases moleculares y bioquímicas de las enfermedades humanas. Nuestras sociedades reúnen a investigadores y académicos de las distintas ramas de la Biomedicina, con un importante enfoque en la medicina traslacional. Desde la organización alentamos la discusión y formación científica en un clima de intercambio cordial y multidisciplinario.

Aprovechamos la oportunidad para agradecer a las comisiones directivas de las sociedades participantes quienes, en un año de crecientes complicaciones económicas y de funcionamiento, han trabajado con enorme dedicación y responsabilidad para el éxito de esta Reunión. Nuestro agradecimiento a las instituciones oficiales y no gubernamentales que apoyaron la organización de este evento a través de subsidios u otros aportes; a las empresas y entidades que auspiciaron y acompañan con su presencia este Congreso; a las empresas organizadoras y a la gerencia del Hotel 13 de Julio por su amabilidad y profesionalismo.

Esperamos que disfruten de este encuentro en sus aspectos científicos y académicos como también en salidas sociales aprovechando las instalaciones turísticas de esta espléndida ciudad de Mar del Plata.

Dra. Claudia Pérez Leirós	**Dr. Pablo Baldi**	**Dr. Alberto Crottogini**
Presidente SAIC	Presidente SAI	Presidente SAFIS

WELCOME WORDS

We are pleased to welcome you to the SAIC SAI SAFIS 2018 Joint Meeting, organized by Sociedad Argentina de Investigación Clínica (SAIC), Sociedad Argentina de Inmunología (SAI) and Sociedad Argentina de Fisiología (SAFIS), with the participation of Sociedad Argentina de Virología (SAV) and Asociación Argentina de Nanomedicinas (NANOMED-ar).

The scientific program is comprehensive, spanning the most glowing and innovative aspects of the diverse fields. Outstanding international experts from Argentina and from abroad will discuss their recent advances in the setting of conferences and symposia. In addition, PhD and postdoctoral fellows, young investigators, resident physicians and other health professionals will address the recent results of their research in over 760 communications during poster and oral sessions. A number of these works have been selected for presentation in symposia, in order to foster interactions of their authors with foreign colleagues. Likewise, expert juries have pre-selected communications to compete for the following awards: The León Cherny Award to the best multidisciplinary research, The Honorio Bigand Award to the best project presented by young investigators, The Eduardo Soto Award to the best research in Neuroscience, The Irene Faryna de Raveglia Award in Oncology, The Leonardo Satz Award in Immunology, The SAFIS Young Investigators in Physiology Award, The Camilión de Hurtado Award in Cardiovascular Pathophysiology and The César Milstein Award in Chagas Disease. A Prize in the field of Infectology from The American Society for Microbioloy, as well as Mentions from SAIC to the best posters, will also be awarded. These awards convey a motivation to the Argentine research groups that progressively improve the quality of their investigations, and are granted thanks to the generosity of the Cherny and Bigand Foundations, Dr. Pasqualini, the Camilión de Hurtado Family and the companies ETC Internacional y Novartis Argentina SA. Minicourses, Meeting with the Expert Sessions and a commercial exhibit will also take place during the Joint Meeting.

The main goal of this Joint Meeting is providing the attendees with an appropriate academic framework to encourage interactions between Argentine scientists and colleagues from abroad who investigate the molecular and biochemical bases of human ailments. The members from our societies are investigators and academics from diverse biomedical areas with a strong focus in translational medicine. From the Organizing Committee, we firmly encourage scientific discussion and training in an atmosphere of warm, multidisciplinary interaction.

We take advantage of this opportunity to thank the Boards of the participating Societies which, in a year of increasing economic and managing complications have worked with enormous commitment and responsibility for the success of this Meeting. Our gratitude, as well, to the official and private institutions that supported the organization of this event with grants or other financial contributions; to the sponsoring and organizing companies and entities; and to the staff of 13 de Julio Hotel for their kindness and professionalism.

We wholeheartedly hope that you enjoy this Meeting in its scientific, academic and social aspects, while profiting the attractions of this beautiful, splendid Mar del Plata.

Dr. Claudia Pérez Leirós
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REGENERATING CNS MYELIN - FROM MECHANISMS TO EXPERIMENTAL MEDICINE

Robin J.M. Franklin

Wellcome Trust-MRC Cambridge Stem Cell Institute, University of Cambridge, United Kingdom

Remyelination, the process by which new myelin sheaths are restored to demyelinated axons, represents one of the most compelling examples of adult multipotent stem cells contributing to regeneration of the injured CNS. This process can occur with remarkable efficiency in multiple sclerosis (MS), and in experimental models, revealing an impressive ability of the adult CNS to repair itself. However, the inconsistency of remyelination in MS, and the loss of axonal integrity that results from its failure, makes enhancement of remyelination an important therapeutic objective. There is now compelling evidence that ageing is the major contributor to the declining efficiency of remyelination and that this is largely due to a failure of stem cell differentiation. This talk will cover some of our recent studies on how ageing effects many aspects of CNS remyelination, including the divergent properties of CNS progenitors of different developmental origin and how changes in the mechanical properties of the ageing brain change the properties of CNS progenitors.

EMBO Keynote Lecture

MAINTENANCE AND REACTIVATION OF IMMUNOLOGICAL MEMORY

Andreas Radbruch

Deutsches Rheumaforschungszentrum Berlin, a Leibniz institute, and Charité University Medicine Berlin; radbruch@drfz.de

Recent observations have fundamentally challenged the classical view that immunological memory is maintained by coherent populations of circulating and proliferating immune memory cells. Distinct populations of memory T lymphocytes and memory plasma cells residing in epithelial tissues and in the bone marrow have been described. They provide first-line protection and long term memory to prevailing antigenic challenges of the environment. We have now also identified memory B lymphocytes of the bone marrow as a population distinct from their splenic counterparts in terms of repertoire and phenotype. Immune memory cells of the bone marrow are individually docking onto stromal cells, implying that stromal cells determine the capacity of immunological memory. There they rest in terms of mobility and activity. These resident memory lymphocytes apparently are not maintained by (homeostatic) proliferation. As we could show for memory plasma cells, their survival is dependent on cell contact to the stromal cell, inducing PI3K signaling, and on the cytokines April or BAFF from their environment, inducing NFkB signaling. In synergy, both signaling pathways in memory plasma cells upregulate expression of the vital transcription factor IRF4 and prevent caspase-induced apoptosis. Memory T and B lymphocytes are maintained by PI3K signalling as well in the bone, suggesting that stromal cells play a pivotal role for the persistence of immunological memory, by preventing apoptosis of the memory cells through contact-dependent PI3K signaling. In secondary immune reactions, resident quiescent T and B lymphocytes obviously have to be mobilized from their memory niches. We could show for resident CD4+ memory T lymphocytes that this mobilization leads (a) to the formation of “Immune clusters” in the bone marrow, resulting in amplification of the specific memory lymphocytes, and (b) to the emigration of specific resident memory T lymphocytes into the blood, and their participation in the secondary immune reaction.

SAIC CONFERENCE ‘ALBERTO TAQUINI’

SIGNALING NEW THERAPEUTIC APPROACHES IN HEPATOCELLULAR CHOLESTASIS

Marcelo G. Roma

IFISE-CONICET, Universidad Nacional de Rosario

Hepatocellular cholestasis is associated with a functional failure in the capability of hepatocytes to produce bile. It is often due to a functional impairment in the main transporters involved in the canalicular efflux of solutes acting as driving force for bile flow generation (*e.g.*, bile salts and gluthation, transported via Bsep and Mrp2, respec-

ELIMINATION AND CONTROL OF MEASLES IN ARGENTINA IN A GLOBAL CONTEXT

Elsa Baumeister

Servicio Virosis Respiratorias, Laboratorio Nacional de Referencia de Sarampión y Rubéola de OMS. Centro Nacional de Influenza de OMS. Departamento Virología. INEI-ANLIS "Dr. Carlos G. Malbrán", Buenos Aires. Virología Clínica, Facultad de Ciencias Exactas, UNLP

Measles (M) is a highly contagious and serious disease caused by a paramyxovirus. Before 1980, an estimated 2.6 million deaths occurred each year. M is still one of the leading causes of death among young children worldwide, despite the availability of a safe and effective vaccine. Approximately 134,200 deaths due to M occurred in 2015. The WHO Directing Council established in 1994 the elimination goal for 2000 and in 2001, the Initiative to ensure that no child dies because of M. Among the goals proposed for 2020 are reducing Measles deaths by 95% and achieving the elimination of M in at least 5 WHO regions. The WHO estimates that systematic vaccination against M prevented 17.1 million deaths worldwide between 2000 and 2014, with a decrease of 79% in deaths. In 2016, PAHO / WHO determined that America had eliminated the endemic transmission of M but in 2016 Europe reported 3553 cases with vaccination coverage less than 95%. The last endemic case of M in Argentina was reported in 2000 and endemic transmission was stopped in 2002, without deaths since 1998. Since 2002, there have been 26 documented imported cases of M or associated with importation, 22 between 2010 and 2018. In the current situation of low endemicity, new challenges are presented: cases are sporadic; it is difficult to detect them, obtain the appropriate samples and epidemiological information. The performance of the serological assays is getting lower forcing genomic detection assays into the diagnosis. Sequencing is essential to establish and confirm transmission chains, explain import phenomena or post-vacunal cases. In populations highly vaccinated with low endemicity, the absence of natural "booster" by exposure to the virus will lead antibodies to decline over time and vaccinated people may be reinfected. Improving clinical notification, laboratory confirmation and gathering epidemiological information is essential.

SAIC SYMPOSIUM: METABOLISM AND ITS IMPACT ON DISEASE OUTCOME

MOLECULAR MECHANISMS INVOLVED IN PROSTATE TUMOR GROWTH AND PROGRESSION ASSOCIATED TO METABOLIC SYNDROME. GENES AND MIRNAS RELATED TO CTBP1 PATHWAY.

Adriana De Siervi

Laboratorio de Oncología Molecular y Nuevos Blancos Terapéuticos (IBYME-CONICET)

Prostate cancer (PCa) is the second commonest diagnosed malignancy and the fifth leading cause of cancer mortality in men. Metabolic Syndrome (MeS) is linked to increased PCa risk and aggressiveness by unknown mechanisms. C-terminal binding protein 1 (CTBP1) is a transcriptional co-repressor of tumor suppressor genes that is activated by low NAD+/NADH ratio. Our group established a MeS-like/PCa mice model that identified CTBP1 as a novel link associating both diseases. We found that CTBP1 is overexpressed in high grade human PCa, and its depletion decreased PCa growth in MeS mice. To understand the molecular mechanism underlying the link between MeS and PCa mediated by CTBP1, we investigated PCa development and progression in MeS mice models. We identified an mRNAs and miRNAs profile regulated by CTBP1 that is associated to PCa development and progression in xenografts generated in MeS mice using expression microarrays and bioinformatic analysis. CTBP1 diminished the prostate cellular adhesion and altered the cellular morphology, which induced mesenchymal phenotype and filopodia number in a mechanism mediated by the hsa-miR-196b-5p. Moreover, CTBP1 depletion in primary tumors significantly decreased circulating tumor cells and spontaneous metastasis with an increment of hsa-miR-30b-5p plasma circulating miRNA in MeS mice. Finally, MeS increased hypertrophy, hyperplasia, inflammation and mRNA/miRNA expression of adipose tissue which induced CTBP1 expression and PCa cell proliferation. Our studies uncover the role of CTBP1/MeS in PCa development and progression. Targeting of CTBP1 expression might be considered for PCa management and therapy in the subset of patients with MeS.

DIET AND THE BRAIN. A VIEW FROM BASIC BIOMEDICINE.

Juan Beauquis

Laboratorio de Neurobiología del Envejecimiento IBYME-CONICET y Dpto. de Química Biológica, FCEN, UBA.

In the last few years, increased attention has been put in understanding the modulatory role of diet on the central nervous system. On the one hand, epidemiological evidence suggests that consumption of "western diets"

(high intake of fat, carbohydrates and industrially processed food) constitutes a risk factor for neurodegenerative and neurovascular disorders. On the other hand, evidence shows that dietary restriction (DR) ameliorates the impact of age-associated diseases, such as Alzheimer's (AD). However, underlying mechanisms are not clear yet and could involve the modulation of multiple biological pathways. Our objective is to describe and understand histological, biochemical and molecular consequences in the brain and associated behavioral changes in response to A) the consumption of a high-fat diet (HFD) and B) dietary restriction in animal models. Our results show that C57BL/6 mice that were fed a moderately HFD (45% of kCal from fat vs. 12% in control diet) since weaning displayed peripheral and central inflammation along with impaired insulin signaling without overweight. Cognitive deficits were found in HFD mice, concomitantly with decreased hippocampal adult neurogenesis. Using an in vitro model of fatty acid exposure on microglia we found that secreted exosomes, as a mean for intercellular communication, induced dendritic remodeling on primary hippocampal neurons. Finally, using a transgenic model of AD we studied the neuroprotective capability of periodic DR. We found that DR for 6 weeks was associated with decreased activation of hippocampal microglia, increased neurogenesis and reversal of cognitive impairment in AD mice. We studied the communication between astrocytes and microglia in vitro and found that astrocytes under nutrient restriction are able to prevent amyloid-induced microglial activation. Our results suggest that diet has a significant role on brain function and structure, with degenerative or protective effects, and that glial cells are possible effector cells and potential therapeutic targets.

FAT DIET SWEET DIET: FROM LIPIDS TO HEART, AN ENDLESS TRASLATIONAL JOURNEY

Gabriela Berg

Laboratorio de Lípidos y Aterosclerosis. Instituto de Fisiopatología y Bioquímica Clínica, Facultad de Farmacia y Bioquímica-Universidad de Buenos Aires, Argentina

Alterations in plasma lipoproteins levels are hallmarks of cardiovascular disease (CVD). Several co-morbidities including diabetes and obesity have been shown to increase CVD risk, in part due to changes in lipoprotein profile, characteristic of insulin resistance (IR) states. The interplay among synthesis, catabolism and structural modifications, conditions the levels and atherogenicity of lipoproteins. The increase of VLDL remnants in plasma is the first step of lipoprotein metabolism after fat-rich meal intake, as in the case of glycaemia after carbohydrate-rich meal intake. In two different models of IR in rats, we demonstrated an increased secretion of larger and TG over-enriched VLDL particles from the liver, as well as a reduction in lipoprotein lipase (LPL) activity from adipose tissue and heart. Consequently, the delayed catabolism of VLDL and its remnants increases the residence time of these lipoproteins in circulation. In parallel, we reported for the first time an increase in endothelial lipase (EL) activity in the same model, counterbalanced with decreased LPL. The enzyme contribution to the lipoprotein profile differs according to each tissue; heart enzymes are the major responsible of plasma TG behaviour, meanwhile adipose tissue EL mainly conditioned HDL levels. However, when evaluating human epicardial adipose tissue LPL activity, we observed that although it decreased with IR in coronary patients, remained increased compared to controls, supporting that not only transcriptional but also post-translational modifications are involved in the enzyme regulation. These results support our previous findings, which showed that plasma LPL activity is reduced in patients with IR and obesity, meanwhile plasma EL present increased activity in the highest obesity and IR degrees, accounting for the hypertriglyceridemia and the decreased HDL-cholesterol levels. Our studies provide new insights into the role of lipolytic enzymes and its regulation in determining lipoproteins levels and characteristics, beyond fatty acids supply to different tissues.

SELECTED ABSTRACT FOR SYMPOSIUM

CARDIAC HYPERTROPHY IN OBESITY: LEPTIN-TRH INTERACTION.

Aisicovich, Maia; Peres Diaz Ludmila; Schuman Mariano; Landa Maria; Garcia Silvia.

Instituto de Investigaciones Médicas A Lanari UBA-CONICET.

Cardiac TRH induce left ventricular hypertrophy (LVH) and fibrosis, its inhibition prevent hypertrophy.The adiponectin leptin induces TRH in CNS. We hypothesized that in obesity, the increase of TRH induced by hyperleptinemia is responsible of the LVH, until now mostly attributed to pressure load. We studied obese Agouti mice suffering hypertension with hyperleptinemia and found LVH with increased TRH gene expression. Consequently we found higher ($p<0.05$) fibrotic and hypertrophic markers vs lean (BL/6J). As pressure could explain results we treated obese mice with diuretic (hydroclorothiazide 20 mg/kg/day) from weaning (n=9), the diuretic group was normotensive in contrast to control obese mice. Nevertheless both groups developed ($p<0.05$): LVH, higher TRH gene and elevated fibrotic and hypertrophic markers suggesting that LVH is not induced by hypertension.