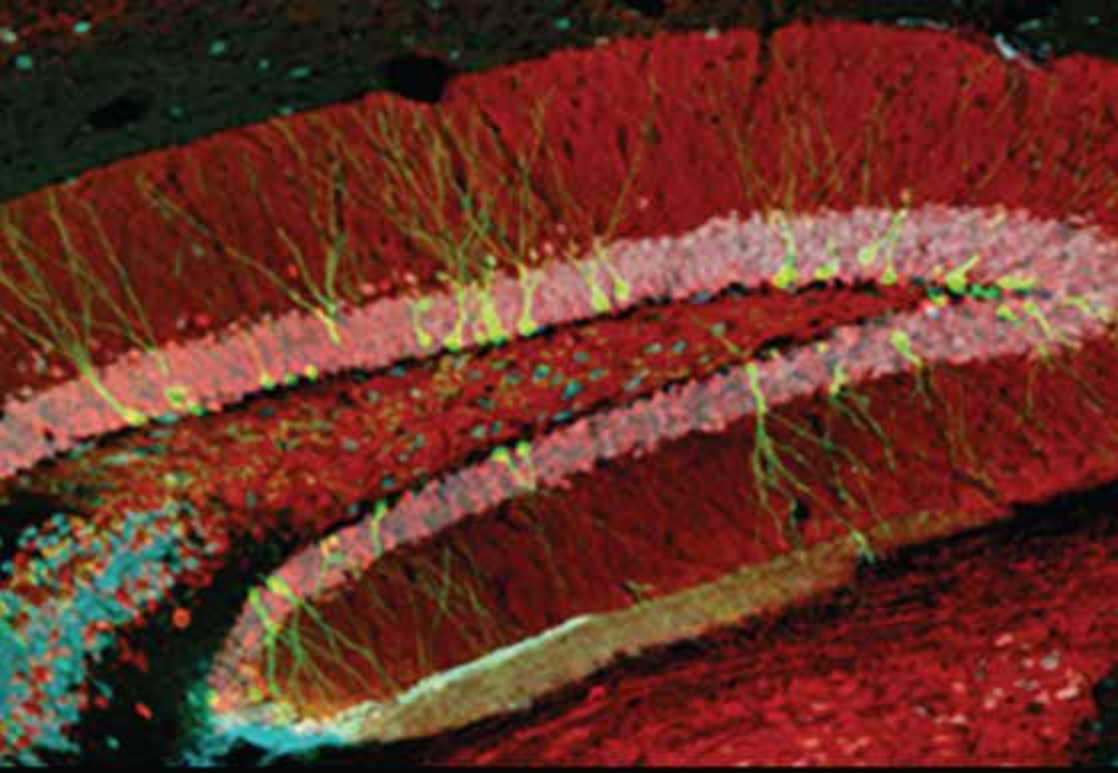

SAN
SOCIEDAD ARGENTINA DE INVESTIGACIÓN EN NEUROCIENCIAS

XXIX ANNUAL MEETING
AND SAN-ISN SMALL CONFERENCE AND COURSE

"New mechanisms of neuro-glial interaction: Their contribution to nervous system development and repair"

September 29 | October 3, 2014
Huerta Grande, Córdoba, Argentina

COMMITTEES

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A. Javier Ramos

Tomás Falzone

Fernanda Ledda

Gustavo Paratcha

Analia Reinés

Meeting Organizing Committee:

Tomás Falzone

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Foto de tapa: Imagen obtenida por microscopía confocal, correspondiente a un corte de hipocampo obtenido de ratones adultos. En verde fluorescente pueden observarse neuronas granulares generadas en el cerebro adulto, marcadas por transduccion retroviral.

Créditos: Georgina Davies-Sala, Lab Schinder.

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Dear SAN members, friends and colleagues,

On behalf of the 2014 organizing committee and the SAN Board of Directors, it is my great pleasure to welcome you all to our XXIX Annual Meeting and to the SAN-ISN special small conference. As in previous years, Huerta Grande, Córdoba, is the place where we will gather. This retreat in the middle of the hills gives the unique opportunity to host us all in one place and provides the right atmosphere for scientific discussions.

With an increasing ageing population, neuroscience is a strategic area of research in the 21st century. Although far from the large-scale investments such as the USA BRAIN Initiative or the European Human Brain projects, the Argentinean neuroscience community continues to grow and SAN has now near 500 members. This mainly derives from the personal effort of each one of our members who, through a strong passion for their work, set free will to their imagination and explore new horizons, even with limited resources. The excellence of Argentinean neuroscience is reflected by the increasing number of publications in high-profile journals and the prestigious awards received by SAN members in 2014, such as the L´ORÉAL Unesco For Women in Science for Latin America and recognitions from the Humboldt Foundation.

We at SAN are committed to aid in deciphering the complexity of the brain and the nervous system, promoting high quality science and encouraging translational research that will ultimately benefit society through a better understanding of neurological diseases. This can only be achieved by bringing together scientists with diverse scientific background and expertise, different nationalities and at different stages of their career. We hope that the XXIX Annual Meeting will fulfill this objective. Thank you all invited speakers for coming from far away to share your newest discoveries. Thank you all neuroscience students for your energy and refreshing and renovating ideas. Thank you SAN members for your continuing support to the society. And last, but not least, thank all sponsors that make our Annual Meetings a reality.

Enjoy the meeting, establish new collaborations, come up with innovative ideas, meet old friends and make new ones.

Ana Belén Elgoyhen
SAN President

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PROGRAM

ISN International Society for Neurochemistry

SAN-ISN Small Conference and course

"New mechanisms of neuro-glial interaction: Their contribution to nervous system development and repair"

DAY 1: Monday September 29th

8:00-9:30	Registration
9:30-10:15	Welcome words by the SAN-ISN small conference and course organizers Description of the general organization of the course and initial orientation for the Project-writing workshop for students
10:15-10:45	Coffee break and post your poster
10:45-12:15	Lecture I: ***"Tripartite synapse: new glial roles"*** **Vladimir Parpura,** University of Alabama at Birmingham, USA
12:30-14:00	Lunch
14:30-16:00	Lecture II: "***Astrocytes: key function in the balance between excitatory and inhibitory synaptic inputs"*** **Flavia Gomes,** Universidade Federal do Rio de Janeiro, Brasil
16:00-17:30	Lecture III: ***"Characterization of astrocytes phenotypes modulating disease progression in inherited ALS"*** **Luis Barbeito,** Institut Pasteur, Montevideo, Uruguay
17:30-18:30	Poster viewing I & refreshments
18:30-21:00	Group activity meeting I: Project writing workshop for students
21:30	Dinner

DAY 2: Tuesday September 30th

7:30-8:30	Breakfast
8:30-10:00	Lecture IV: ***"Glia and the formation/plasticity of neural circuits"*** **Gabriel Corfas,** Director, Kresge Hearing Research Institute, University of Michigan, USA
10:30-11:00	Coffee Break and poster viewing II
11.00-12:30	Lecture V: ***"Functional role of microglial cells in Parkinson´s Disease"*** **Fernando Pitossi,** Instituto Leloir, Buenos Aires, Argentina
13:00-14:30	Lunch
14:30-16:30	Lecture VI: ***"The role of Schwann cells in degenerative and regenerative axonal programs"*** **Felipe Court,** Pontificia Universidad Católica de Chile, Santiago, Chile
16:30-17:00	Coffee Break and poster viewing III
17:00-18:30	Group activity meeting II: Project writing workshop for students
18:30-20:30	Group activity meeting II: Project writing workshop, student´s presentations
20:30-21:30	Round table and final conclusions of the SAN-ISN small conference and course
21:30	Dinner

XXIX CONGRESO ANUAL DE LA SOCIEDAD ARGENTINA DE INVESTIGACION EN NEUROCIENCIAS

DAY 1: Wednesday October 1st

09:00 Registration

10:00 Welcome by Organizers

10:10-12:30 **Symposium I: "The Good and the Bad of Neurotrophins"**
Chairs: **Laura Montroull y Andrea Cragnolini,** Instituto de Investigaciones Biológicas y Tecnológicas, Universidad Nacional de Córdoba, CONICET, Argentina

10:20-10:50 *"XIAP Regulates Sub-Lethal Caspase Activity in Axons and Synapses"*
Philip Barker, McGill University, Montreal, Canada

10:50-11:20 *"The multifaceted role of the p75 neurotrophin receptor in the brain"*
Wilma Friedman, Rutgers University, USA

11:20-11:50 ***"Role of the endocytic system in BDNF-mediated dendritic branching"***
Francisca Bronfman, Pontificia Universidad Católica de Chile, Santiago, Chile

11:50-12:20 ***"Endogenous BDNF/proBDNF level modification in neuronal death and survival"***
Daniel Mascó, Universidad Nacional de Córdoba-CONICET, Córdoba, Argentina

12:30-14:00 Lunch

14:30-16:00 **Short talks selected from poster abstracts** (parallel sessions):
Room A – Chair: **A. Javier** Ramos. Instituto de Biología Celular y Neurociencia (IBCN-CONICET), Universidad de Buenos Aires
Room B – Chair: **Fernanda Ledda.** Instituto de Biología Celular y Neurociencia (IBCN-CONICET), Universidad de Buenos Aires

16:15-19:15 **Poster Session I, Networking & Coffee break**
16:15-17:45 ***EVEN NUMBER poster presentation***
17:45-19:15 ***ODD NUMBER poster presentation***

19:15-20:15 **Eduardo de Robertis Plenary Lecture**
Chair: **Ana Belén Elgoyhen,** Instituto de Investigaciones en Ingeniería genética y Biología Molecular, Dr Héctor N. Torres, Argentina
"Unexpected interactions in the basal ganglia"
Bernardo Sabatini, Neurobiology Department, Harvard Medical School, Howard

Hughes Medical Institute, USA

20:15-20:45 **SAN Award to the Best Doctoral Thesis in Neuroscience 2014**
Chair: **Juan Goutman,** Instituto de Investigaciones en Ingeniería genética y Biología Molecular, Dr Héctor N. Torres, Argentina
"Stress-Induced Cocaine Sensitization: A Study of Glutamate Homeostasis and its Interaction with the Dopaminergic System in Nucleus Accumbens"
Constanza García Keller, Dpto de Farmacología, Fac. Ciencias Químicas, Universidad Nacional de Córdoba, Argentina - Department of Neurosciences, Medical University of South Carolina, USA

21:00-22:45 Dinner
Dinner activity: eat with the big shots I!
Students and postdocs can sign up to share the table with lecturers and symposia speakers

23:00 Asamblea Ordinaria SAN/ SFN Chapter

DAY 2: Thursday October 2nd

7:30-8:15 Breakfast

8:15-12:50 **SAN–ISN Symposium: "Deconstructing Adult Neurogenesis: From Neural Stem Cells to Neuronal Networks in Health and Disease"**
Chair: **Alejandro Schinder,** Fundación Instituto Leloir, Buenos Aires

8:15-9:00 ***"A novel view of neurogenesis and memory encoding in the dentate gyrus"***
Alejandro Schinder, Laboratory of Neuronal Plasticity, Fundación Instituto Leloir, Buenos Aires

9:00-9:50 ***"Analysis of neural stem cells in the adult mammalian brain, one cell at a time"***
Hongjun Song, Institute for Cell Engineering and Department of Neuroscience, Johns Hopkins University School of Medicine, Baltimore

9:50-10:40 ***"Brains in metamorphosis: physiological and forced neurogenesis in the adult brain"***
Benedikt Berninger, Adult Neurogenesis & Cellular Reprogramming, Institute of Physiological Chemistry University Medical Center, Johannes Gutenberg, University Mainz

10:40-11:10 Coffee Break

11:10-12:00 *"Adult Neurogenesis and Psychiatric Neurodevelopmental Disorders"*
Guoli Ming, Institute for Cell Engineering and Department of Neuroscience, Johns Hopkins University School of Medicine, Baltimore

12:00-12:50 ***"From pluripotent stem cells to cortical circuits"***
Pierre Vanderhaeghen, Institute for Interdisciplinary Research and Institute of Neuroscience, Free University of Brussels

13:00-14:45 Lunch

15:00-17:30 **Poster Session & Networking II & Coffee break**

15:00-16:15 ***ODD NUMBER poster presentation***

16:15-17:30 ***EVEN NUMBER poster presentation***

17:30-19:00 **IBRO Special Lectures**
Chair: **Marta Hallak**, Centro de Investigaciones en Química Biológica de Córdoba, CONICET

17:30-18:00 ***"The Global Mission of IBRO, and IBRO 2015 in Rio de Janeiro"***
Sten Grillner, IBRO Secretary-General, Karolinska Institute, Stockholm, Sweden

18:00-19:00 *"**The Blueprint of the Vertebrate Motor System – from Microcircuits to Selection of Behaviour"***
Sten Grillner, IBRO Secretary-General, Karolinska Institute, Stockholm, Sweden

19:00-19:30 Coffee Break

19:30-21:00 **Young Investigator Symposium**
Chair: **Tomás Falzone**, CONICET, Universidad de Buenos Aires

19:30-19:50 ***"Impact of axonal, autoreceptor mediated, synaptic events on cerebellar interneuron's activity"***
Javier Zorrilla de San Martin, Université Paris Descartes, France

19:50-20:10 *"Caspase-3 and Calpains become active during (and play a role in) injury-induced axonal degeneration but are not inhibited during NAD+-mediated protection"*
Nicolás Unsaín, McGill University, Canada/Instituto de Investigacion Médica Mercedes y Martín Ferreyra, CONICET, Argentina

20:10-20:30 *"Amyloid Precursor Protein Is an Autonomous Growth Cone Adhesion Molecule Engaged in Contact Guidance"*
Lucas Sosa, University of Colorado School of Medicine, USA/Centro de Investigaciones en Química Biológica de Córdoba, CONICET , Argentina

20:30-20:50 *"Neurogenin3 is a key regulator in serotonergic vs. glutamatergic neuronal cell fate"*

Abel Carcagno, Fundación Instituto Leloir, Argentina

21:00 Dinner

Dinner activity: eat with the big shots II!

23:00 PARTY!!!!

DAY 3: Friday October 3rd

8:00-9:00 Breakfast

9:00-11:00 **Symposium II: "Ion channels from development to behavior"**

Chair: **Nara Muraro**, Fundación Instituto Leloir, Argentina

9:00-9:30 ***"Inhibitory aminergic signaling in C.elegans: characterization and in vivo manipulation"*** **Diego Rayes**, Instituto de Investigaciones Bioquímicas de Bahía Blanca, Bahía Blanca, Argentina

9:30-10:00 ***"How do Drosophila clock neurons fire up?"***

Nara I. Muraro, Fundación Instituto Leloir, Argentina

10:00-10:30 *"Pumilio-2 regulates translation of Nav1.6 to mediate homeostasis of membrane excitability"*

Richard Baines, Faculty of Life Sciences, University of Manchester, UK

10:30-11:00 *"Activity-dependent regulation of coordinated ion channel expression: from mRNA to network output"*

David Schulz, Department of Biological Sciences, University of Missouri, USA

11:00-11:30 Coffee break

11:30-12:30 **Ranwel Caputto Plenary Lecture**

Chair: **Arturo Romano**, Instituto de Fisiología Biología Molecular y Neurociencias, Universidad de Buenos Aires

"Motor coordinates to study birdsong"

Gabriel Mindlin, Departamento de Física, Facultad de Ciencias Exactas y Naturales, UBA, Argentina

12:30 Closing remarks by organizers

13:00 Farewell Barbecue

16:00 Meeting adjourns

Recipients of Young ISN Neurochemistry Awards

Anabela Palandri, Córdoba, Argentina
María Paula Avalos, Córdoba, Argentina
Andrea Susana Guzmán, Córdoba, Argentina
María Vanessa Cadena, Buenos Aires, Argentina
Nonthue Alejandra Uccelli, Berazategui, Argentina
Jerónimo Lukin, Buenos Aires, Argentina
Laura Montroull, Córdoba, Argentina
Nicolás Unsain, Córdoba, Argentina
Luis Ernesto Acosta, Buenos Aires, Argentina
María Florencia Almeira Gubiani, Buenos Aires, Argentina
Diego Damián Alvarez, Buenos Aires, Argentina
Marcos Andrés Campolongo, Buenos Aires, Argentina
Micaela Daiana García, San Martín, Argentina
María Paula Ibáñez Rodríguez, Mendoza, Argentina
Nadia Kazlauskas, Buenos Aires, Argentina
Noelia Giselle Lino, Buenos Aires, Argentina
Magdalena Miranda, Buenos Aires, Argentina
Javier Andrés Muñiz, Buenos Aires, Argentina
Patricio Roberto Pavía, Buenos Aires, Argentina
Gonzalo Miguel Piñero, Buenos Aires, Argentina
María Celeste Solange Rivero Echeto, Buenos Aires, Argentina
Gerardo Ariel Rosciszewski, Buenos Aires, Argentina
María Micaela Sartoretti, Buenos Aires, Argentina
Silvio Temprana, Buenos Aires, Argentina
Lucila Brocardo, Bernal, Argentina
Octavio Gianatiempo, Buenos Aires, Argentina
Florencia Martina Soler García, San Luis, Argentina
Betina González, Buenos Aires, Argentina
Veronica Murta, Buenos Aires, Argentina
Constanza García Keller, Córdoba, Argentina
Alejandra Ivetth **Pérez** Alvarez, Caracas, Venezuela
Sonia Carolina Guerrero Prieto, São Bernardo do Campo, Brasil
Wagno Alcantara de Santana, Salvador, Brasil
Angelica Jhoan Alarcon Garcia, Bogotá, Colombia
Claudia Angelica Bonilla Escobar, Santo Andre, Brasil
Irene Riveros Barrera, Bogotá, Colombia
Cristian Ivan Giraldo León, Bogotá, Colombia
Nicolás Iván Bertone Cueto, Buenos Aires, Argentina

ABSTRACTS

Tripartite synapse: new glial roles

Vladimir Parpura

Part 1: Mechanisms of glutamate release from astrocytes

Astrocytes can release the excitatory transmitter glutamate which is capable of modulating activity in nearby neurons. This astrocytic glutamate release can occur through six known mechanisms: **(i) reversal of uptake by glutamate (ii) anion channel opening, (iii) Ca^{2+}-dependent exocytosis, (iv) glutamate exchange via the cystine-glutamate antiporter, (v) release through ionotropic purinergic receptors and (vi) functional unpaired connexons, 'hemichannels', on the cell surface. Although these various pathways have been defined, it is not clear how often and to what extent astrocytes employ different mechanisms. It will be necessary to determine whether the same glutamate release mechanisms that operate under physiological conditions operate during pathological conditions or whether there are specific release mechanisms that operate under particular conditions.**

Part 2: Vesicular release of glutamate mediates bidirectional signaling between astrocytes and neurons

The major excitatory neurotransmitter in the CNS, glutamate, can be released exocytotically by neurons and astrocytes. Glutamate released from neurons can affect adjacent astrocytes by changing their intracellular Ca^{2+} dynamics and, vice versa, glutamate released from astrocytes can cause variety of responses in neurons such as: an elevation of $[Ca^{2+}]_i$, a slow inward current, an increase of excitability, modulation of synaptic transmission, synchronization of synaptic events, and long-term potentiation, or some combination of these. This astrocyte-neuron signaling pathway might be a wide-spread phenomenon throughout the brain with astrocytes possessing the means to be active participants in many functions of the CNS. Thus, it appears that the vesicular release of glutamate can serve as a common denominator for two of the major cellular components of the CNS, astrocytes and neurons, in brain function.

Astrocytes: key function in the balance between excitatory and inhibitory synaptic inputs

Flavia Gomes

Assembly of synapses requires proper coordination between pre- and post-synaptic elements. Identification of cellular and molecular events in synapse formation and maintenance is a key step to understand human perception, learning, memory, and cognition. Astrocytes play important role in the development and maintenance of neuronal circuitry. Here we will discuss how astrocytes regulate the balance between excitatory and inhibitory synaptic inputs, critical for control brain function. We previously demonstrated that astrocytes induce excitatory synapses through transforming growth factor beta 1 (TGF-β1) pathway and control of the levels of the aminoacid, D-serine. Recently, we showed that inhibitory synapses are also induced by astrocyte secreted TGF-β1. TGF-β1 -induction of inhibitory synapse is dependent of glutamatergic activity and activation of CaM kinase II, which thus induces localization and cluster formation of the synaptic adhesion protein, Neuroligin 2, in inhibitory postsynaptic terminals. We will discuss that the balance between excitatory and inhibitory inputs might be provided by astrocytes signals, at least partly achieved via TGF-β1 downstream pathways.

Characterization of astrocytes phenotypes modulating disease progression in inherited ALS

Luis Barbeito

Motor neuron loss and reactive astrocytosis are pathological hallmarks of Amyotrophic Lateral Sclerosis, a paralytic neurodegenerative disease that can be triggered by mutations in Cu,Zn-superoxide dismutase-1 (SOD1). Dysfunctional astrocytes contribute to ALS pathogenesis, inducing motoneuron damage and accelerating disease progression. **However, it is unknown whether ALS progression is associated to the appearance of a specific astrocytic phenotype with neurotoxic potential.** We have recently reported the isolation of astrocytes with aberrant phenotype (referred as AbAs) from primary spinal cord cultures of symptomatic rats expressing the $SOD1^{G93A}$ mutation. Isolation was based on AbA's marked proliferative capacity and lack of replicative senescence, which allowed oligoclonal cell expansion during over 1 year. AbAs displayed astrocytic markers including GFAP, S100β, glutamine synthase and connexin 43, but lacked the GLT1 glutamate transporter and the glial progenitor marker NG2 glycoprotein. Notably, AbAs secreted soluble factors that induced motoneuron death with a 10-fold higher potency than neonatal $SOD1^{G93A}$astrocytes. AbA-like aberrant astrocytes expressing S100b and connexin43 but lacking NG2 were identified nearby motoneurons, its number increasing sharply after disease onset. In conclusion, AbA cells appear as a yet-unknown astrocyte population arising during ALS progression, with unprecedented proliferative and neurotoxic capacity, being potential cellular targets for slowing ALS progression.

Glia and the formation/plasticity of neural circuits

Gabriel Corfas

Since their initial discovery in the 1800s until recently, glial cells were considered to be the "connective tissue" of the nervous system, their formation was believed to be regulated by predetermined biochemical and cellular processes, and they were viewed as static components. Research during the last few years is providing a very different picture, one in which glia are actively involved in the development and plasticity of the nervous system. In my lecture I will discuss our recent findings on how experience changes glia, how glia regulate the formation of synapses, and the consequences that altered glia have on physiology, behavior and cognitive function. We will also discuss how trophic factor signaling pathways regulate neuron-glia interactions and the implications of these interactions for normal brain function and disease.

Functional role of microglial cells in Parkinson´s Disease

Fernando Pitossi

Parkinson ´s Disease (PD) is the second most common neurodegenerative disease in the population. Unfortunately, the aetiology of over 80% of PD cases is unknown. Lacking evidence on the aetiology, the study of the pathophysiology of the disease becomes more relevant to identify novel therapeutic targets. One patho-physiological feature consistently found in animal models and PD patients is robust microglial activation. However, microglia activation could mediate neurodegenerative or neuroprotective effects depending on the array of molecules associated with this activation and the molecular and cellular context in which they act. As a consequence, microglial activation remains an unreliable therapeutic target in PD treatment. We believe that identifying parameters that could determine a univocal role of microglial activation on neuronal cell death in the substantia nigra (SN), the main region affected in PD, is crucial to define new therapeutic targets against PD and select PD patients to be enrolled in anti-PD trials based on immunomodulation. We have found that microglial activation in the degenerating SN is "primed". Microglial cells can be shifted to a pro-inflammatory state by, not only central, but also sub toxic levels of systemic inflammation. This shift can dramatically exacerbate on-going neurodegeneration in the SN leading to increased and earlier motor symptoms, via Interleukin-1beta (IL-1) overproduction. In addition, we have observed that sustained but not acute expression of IL-1 or Tumor necrosis factor-alpha (TNF) in the SN leads to dopaminergic neuronal demise, motor symptoms and microglial activation. TNF effects are dose-dependent since, using a combination of knock-in mice, adenoviral vectors and the CRE/lox system we could demonstrate that low levels of TNF can be neuroprotective for nigral neurons, while higher levels could be detrimental.In conclusion, we have identified parameters that determine a given effect of pro-inflammatory cytokines on neuronal viability, paving the way to test new hypothesis, study the effects of immunomodulatory treatments, and identify downstream effector molecules on these newly generated models of PD.

The role of Schwann cells in degenerative and regenerative axonal programs

Felipe Court

Nervous system function relies in the coordinated action of neurons and glial cells. In recent years, the importance of glial cells for several aspects of nervous system function has been underscored. Phenomena like synaptic activity, conduction of action potentials, neuronal growth and regeneration, to name a few, are fine tuned by glial cells. We have proposed a model in which the axon has certain autonomy from the neuronal cell body, and its associated glial cell is a major regulator of local axonal programs, including a regenerative program of axonal extension (Court and Alvarez, 2005), a destruction program activated by various stimuli (Barrientos et al., 2011; Villegas et al., 2014) and unpublished data) and local protein synthesis in the axon (Court et al., 2008). Several intercellular mechanisms have been shown to operate on a local basis in the neuron-glia unit, including contact-mediated signaling and extracellular free ligands. Recently, another regulatory mechanism has emerged in which a cell releases vesicles containing RNAs and proteins, that are taken up by the recipient cell and the cargo is incorporated into the target cell (Simons and Raposo, 2009). Vesicular-mediated transfer of molecular cargoes between glial cells and neurons has been described in the nervous system (Lopez-Verrilli and Court, 2012; Lopez-Verrilli and Court, 2013). We have demonstrated vesicular-mediated transfer of ribosomes from Schwann cells (SCs), the peripheral glial cell type, to axons in vivo after axonal damage as well as during axonal regeneration (Court et al., 2008; Court et al., 2011). Recently, we have found that exosomes secreted by SCs and selectively internalized by axons increase neurite growth substantially and greatly enhance axonal regeneration in vitro and in vivo (Lopez-Verrilli et al., 2013). We have now used a combination of next-generation sequencing, proteomics and bioinformatic analysis to identify RNAs and proteins present in SC-exosomes, and to search for candidates mediating the functional effect of SC-exosomes over axonal regeneration. This mode of interaction provides a new dimension to the understanding of the intercellular regulation at large, and we foresee that a number of phenomena of the nervous system still poorly understood will be studied under this new light.

Eduardo de Robertis Plenary Lecture

Wednesday 1st, 19:15-20:15
Chair: **Ana Belén Elgoyhen**, Instituto de Investigaciones en Ingeniería genética y Biología Molecular, Dr Héctor N. Torres, Argentina

"Unexpected interactions in the basal ganglia"
Bernardo Sabatini
Neurobiology Department, Harvard Medical School, Howard Hughes Medical Institute, USA

The basal ganglia (BG) are a phylogenetically conserved set of subcortical nuclei necessary for coordinated motor action and reward learning. Accepted models postulate that the BG modulate cerebral cortex indirectly via an inhibitory output to thalamus, bidirectionally controlled from within the BG by direct (dSPNs) and indirect (iSPNs) pathway striatal projection neurons2-4. The BG thalamic output sculpts cortical activity by interacting with signals from sensory and motor systems5. Here we describe a direct projection from the globus pallidus externus (GP), a central nucleus of the BG, to frontal regions of the cerebral cortex (FC). Two cell types make up the GP-FC projection, distinguished by their electrophysiological properties, cortical projection patterns and expression of choline acetyltransferase (ChAT), a genetic marker for neurons that release the neurotransmitter acetylcholine (ACh). Despite these differences, ChAT+ cells, which have historically been identified as an extension of the nucleus basalis (NB), as well as ChAT- cells, release the inhibitory neurotransmitter GABA (γ-aminobutyric acid) and are inhibited by iSPNs and dSPNs of dorsal striatum. Thus GP-FC cells comprise a direct GABAergic/cholinergic projection that places frontal cortex under the inhibitory control of the striatum. Furthermore, iSPN inhibition of GP-FC cells is sensitive to dopamine 2 receptor signaling, revealing a pathway by which drugs that target dopamine receptors for the treatment of neuropsychiatric disorders can act in the BG to modulate frontal cortices.

IBRO Special Lectures

Thursday 2nd, 18:00-19:00
Chair: **Marta Hallak,** Centro de Investigaciones en Química Biológica de Córdoba, CONICET

"The Blueprint of the Vertebrate Motor System – from Microcircuits to Selection of Behaviour"
Sten Grillner
IBRO Secretary-General, Karolinska Institute, Stockholm, Sweden

The lamprey diverged from the vertebrate line of evolution leading up to mammals 560 million years ago. What is common in the organization of the nervous system of lamprey and mammals must have been present already very early in vertebrate evolution. We have previously shown that the basic organization of the brainstem spinal cord is conserved and also the midbrain control of eye and orienting movements. Recently we have shown in a series of studies that the detailed organization of the basal ganglia and related habenula complex is conserved with regard to transmitters, neuropeptides, expression of ion channel subtypes, neuronal activity pattern and connectivity (e.g. Stephenson Jones et al 2013). We now show that also the organization of pallium (corresponding to cortex) is similar in that we have specific projection neurons to the tectum/superior colliculus, the midbrain tegmentum and to reticulospinal neurons in the hindbrain. Moreover, stimulation of the pallial region can elicit eye, orienting, locomotor and oral movements and these effects are elicited by monosynaptic effects on the different motor centres. In conclusion, the basic features of the vertebrate motor system existed already at the dawn of vertebrate evolution.
Stephenson-Jones M, AA Kardamakis, B Robertson and S Grillner (2013) PNAS 110; 3670-09.

Ranwel Caputto Plenary Lecture

Friday 3rd, 11:30-12:30
Chair: **Arturo Romano**, Instituto de Fisiología Biología Molecular y Neurociencias, Universidad de Buenos Aires

"Motor coordinates to study birdsong"
Gabriel Mindlin
Departamento de Física, Facultad de Ciencias Exactas y Naturales, UBA, Argentina

Fundamental unresolved problems of motor coding and sensorimotor integration include what information about behavior is represented at different levels of the motor pathway. Insight into this issue is essential for understanding complex learned behaviors such as speech or birdsong. A major challenge in motor coding has been to identify an appropriate framework for characterizing behavior.
In this work we discuss a novel approach linking biomechanics and neurophysiology to explore motor control of songbirds. We developed a model of song based on gestures that can be related to physiological parameters the birds can control. This physical model for the vocal structures allowed a reduction in the dimensionality of the singing behavior
This is a powerful approach for studying sensorimotor integration and represents a significant methodological advantage. Our results also show how dynamical systems models can provide insight into neurophysiological analysis of vocal motor control. In particular, our work challenges the actual understanding of how the motor pathway of the songbird systems works and proposes a novel perspective to study neural coding for song production. It also illustrates the turbulent relationship between physics and biology...

Symposium I: "The Good and the Bad of Neurotrophins"

Wednesday 1st, 10:10-12:30
Chairs: **Laura Montroull y Andrea Cragnolini**, Instituto de Investigaciones Biológicas y Tecnológicas, Universidad Nacional de Córdoba, CONICET, Argentina

Wednesday 1st, 10:20-10:50

"XIAP Regulates Sub-Lethal Caspase Activity in Axons and Synapses"

Philip Barker
Montreal Neurological Institute, McGill University, 3801 University St., Montreal, Canada, *H3A 2B4*

The ability of neurons to receive, process and transmit information relies on a highly complex and dynamic architecture. Most mammalian neurons initially produce an extensive set of processes that are subsequently pruned to retain only those that form part of the mature neuronal network. Significant, albeit less dramatic, re-sculpting also occurs throughout the neuron's life-span. The mechanisms that allow some neurites to be destroyed while others, derived from the same cell, are retained have not been extensively studied, perhaps because it was assumed that neurites withered away through a passive process. However, it is now certain that neurites are destroyed through phylogenetically conserved signaling mechanisms that induce local caspase activity. In addition, recent studies have indicated that activity-dependent changes in synaptic transmission also rely on sublethal caspase activity. The mechanisms that activate and regulate sublethal caspase activity in the nervous system remain poorly understood. Nerve growth factor (NGF) is a crucial survival signal for sensory and sympathetic neurons and we have examined the molecular signaling events that drive cell death and axonal degeneration after NGF withdrawal in peripheral neurons. Our studies show that X-linked inhibitor of apoptosis (XIAP) functions as a key regulator of caspase activity during developmental axonal degeneration. Based on these findings, we have explored the hypothesis that XIAP is required to regulate sublethal caspase activity in the central nervous system and our results demonstrate that XIAP is a potent regulator of activity-dependent changes in synapse function. Together, these studies show that XIAP plays a critical physiological role regulating caspase-dependent sub-lethal morphogenic events in the developing and adult nervous system.

Wednesday 1st, 10:50-11:20

"The multifaceted role of the p75 neurotrophin receptor in the brain"

Wilma Friedman

Department of Biological Sciences – RUTGERS University. Newark, NJ, USA

The p75NTR is induced in the brain following seizures and other types of brain injury and mediates neuronal death. The signaling mechanisms involve activation of the intrinsic caspase pathway, as well as suppression of Trk signaling by induction of the PTEN phosphatase which prevents Akt activation. However, p75NTR is also widely expressed in many brain regions during development, including the external granule layer of the cerebellum, where it does not appear to mediate neuronal death. Therefore, in addition to investigating the apoptotic role of p75NTR after brain injury, we have explored some of the non-apoptotic functions of this receptor during development.

Wednesday 1st, 11:20-11:50

"Role of the endocytic system in BDNF-mediated dendritic branching"

Francisca Bronfman

Departamento de Fisiología, Facultad de Ciencias Biológicas,
Pontificia Universidad Católica de Chile. Santiago, Chile.

Dendritic arborization of neurons is regulated by brain-derived neurotrophic factor (BDNF) together with its receptor TrkB. Endocytosis is required for dendritic branching and regulates TrkB signaling, but how post-endocytic trafficking determine the neuronal response to BDNF is not well understood. The monomeric GTPase Rab11 regulates the dynamics of recycling endosomes and local delivery of receptors to specific dendritic compartments. Our aim was to study whether Rab11-dependent trafficking of TrkB in dendrites regulates BDNF-induced dendritic branching in rat hippocampal neurons. We report that TrkB in dendrites is a cargo for Rab11 endosomes and both Rab11 and its effector MyoVb, an actin-based molecular motor, are required for BDNF/TrkB-induced dendritic branching. In turn, BDNF induces accumulation of Rab11-positive endosomes and GTP-bound Rab11 in dendrites. The expression of a constitutively active mutant of Rab11 is sufficient to increase dendritic branching by increasing TrkB localization in dendrites and enhancing sensitization to endogenous BDNF. On the other hand, increased Rab11-mediated TrkB signaling in dendrites is also required for BDNF-mediated nuclear activation of the transcription factor CREB. We propose a model where dendritic activation of Rab11 would increase recycling by promoting the interaction of Rab11 with Myosin Vb, increasing the amount of Rab11-positive endosomes in secondary dendrites, a process that would require actin filaments. Because Rab11 endosomes carry TrkB, sustained Rab11 activity results in increased endogenous TrkB receptor levels in dendrites, implying that this mechanism potentiates TrkB signaling in dendrites. Thus, this process provides a positive feedback mechanism to induce BDNF-dependent protrusion and the outgrowth of dendritic branches. On the other hand, TrkB signaling in Rab11 endosomes associated to cell bodies is required for nuclear signaling, process that allows the transcription of genes, including Rab11, required for increasing dendritic complexity.

Wednesday 1st, 11:50-12:20

"Endogenous BDNF/proBDNF level modification in neuronal death and survival"

Daniel Mascó

Universidad Nacional de Córdoba-CONICET, Córdoba, Argentina

Our research focusses on brain-derived neurotrophic factor (BDNF), a molecule known to be essential for a number of processes, including synaptic plasticity and positive events like neuronal survival. However, these functions has been challenged because the possibility that the same proteins may induce cell death. Animal models have revealed that its levels increase in pathological conditions such as Status Epilepticus. An important goal is then to explore whether the increase of BDNF levels or its interaction with neurotrophin receptors (TrkB and/or p75ntr) over prolonged periods of time are responsible for cell death.

SAN–ISN Symposium: "Deconstructing Adult Neurogenesis: From Neural Stem Cells to Neuronal Networks in Health and Disease"

Thursday 2nd, 8:15-12:50
Chair: **Alejandro Schinder**, Fundación Instituto Leloir, Buenos Aires, Argentina

Thursday 2nd, 8:15-9:00

"A novel view of neurogenesis and memory encoding in the dentate gyrus"

Alejandro Schinder
Laboratory of Neuronal Plasticity, Fundación Instituto Leloir, Buenos Aires, Argentina

The adult brain contains self-renewing neural stem cells that generate neurons through life. Extensive evidence has demonstrated that adult neurogenesis is highly regulated by brain function and that it is involved in information processing in specific circuits. For instance, ablation of adult hippocampal neurogenesis can impair spatial learning. We are interested in the specific modifications of hippocampal circuits produced by the incorporation of newly generated dentate granule cells (GCs). The impact of adult-born GCs on hippocampal function is greatly determined by their number, intrinsic properties, connectivity, and synaptic properties. In recent years, we have combined different approaches to investigate how adult-born GCs connect within the preexisting hippocampal network, building a spatio-temporal map of input/output connectivity. It takes almost two months to transit the road from neural stem cell to fully mature neuron. During this long transition, developing GCs go through different phases with distinctive functional properties. Work by our lab and several other groups is converging into the notion that adult neurogenesis may serve as a mechanism for the continuous generation of new cohorts of young and very plastic neurons that integrate in the network in a manner that is shaped by ongoing experience. In my talk I will focus our most recent experimental data on how local microcircuits change by adult neurogenesis, discuss its implications in hippocampal function, and propose a novel conceptual model on how newborn GCs may contribute to memory encoding.

Thursday 2nd, 9:00-9:50

"Analysis of neural stem cells in the adult mammalian brain, one cell at a time"

Hongjun Song

Institute for Cell Engineering and Department of Neuroscience,
Johns Hopkins University School of Medicine, Baltimore, USA

New neurons arise from neural stem cells throughout life in the dentate gyrus of the hippocampus. Traditionally, adult neural stem cells and neurogenesis have been investigated at the population level. Single-cell analyses provide high resolution information about heterogeneous stem cell properties and dynamic developmental process. I will present latest data from our ongoing studies to explore single-cell genetic lineage-tracing and single-cell RNA-seq of adult neural stem cell in the mouse hippocampus and their development.

Thursday 2nd, 9:50-10:40

"Brains in metamorphosis: physiological and forced neurogenesis in the adult brain"

Benedikt Berninger

Adult Neurogenesis & Cellular Reprogramming, Institute of Physiological Chemistry
University Medical Center, Johannes Gutenberg, University Mainz, Germany

In my talk I will discuss the topic of how new neurons integrate into the adult brain from two distinct angles. In the first part I will discuss the influence of experience on the incorporation of newly generated neurons in the adult dentate gyrus. I will describe how using a rabies virus-mediated synaptic tracing technique we found that the pattern of presynaptic connectivity impinging on newly generated neurons is strongly affected by exposure to an enriched environment during a critical period following its birth.
In the second part, I will discuss recent work showing that forced expression of the transcription factor Sox2 can lineage-convert reactive NG2 glia of the adult cerebral cortex into immature doublecortin-positive neurons in a model of traumatic cortical injury. I will discuss the implications of the NG2 glial origin of these de novo generated induced neurons for their connectivity.

Thursday 2nd, 11:10-12:00

"Adult Neurogenesis and Psychiatric Neurodevelopmental Disorders"

Guoli Ming

Institute for Cell Engineering and Department of Neuroscience, Johns Hopkins University School of Medicine, Baltimore, USA

Adult neurogenesis occurs in discrete brain regions and recapitulates the complete process of neuronal development in a mature central nervous system, from proliferation and fate specification of adult neural progenitors, morphogenesis, migration, axon/dendritic development, and finally synapse formation, culminating in the full integration of new neurons into the existing circuitry. Mounting evidence over the past two decades suggest that new neurons born in the adult brain exhibit unique characteristics and participate in specific brain functions. Furthermore, cumulating studies suggest that aberrant adult neurogenesis may contribute to neurological and mental disorders. Psychiatric mental disorders, including schizophrenia and autism, are neurodevelopmental disorders with prominent genetic predisposition. I will discuss our recent work focusing on understanding the function of DISC1, a risk gene for schizophrenia and other major mental disorders, in neuronal development and the underlying signaling mechanisms using adult hippocampal neurogenesis as a model system.

Thursday 2nd, 12:00-12:50

"From pluripotent stem cells to cortical circuits"

Pierre Vanderhaeghen
Institute for Interdisciplinary Research and Institute of Neuroscience,
Free University of Brussels, Belgium

The cerebral cortex consists of several hundreds of different types of neurons, organized into specific cortical layers and areas, that display specific profiles of gene expression, morphology, excitability and connectivity.
Embryonic stem (ES) and induced (IPS) pluripotent stem cells constitute a promising tool for the modelling and treatment of human neural diseases. We previously discovered an intrinsic pathway by which pluripotent stem cells, whether of mouse or human origin, recapitulate in vitro the major milestones of cortical development, leading to the sequential generation of a diverse repertoire of pyramidal neurons that display most salient features of genuine cortical neurons.
Here we will describe how corticogenesis from pluripotent stem cells can be used to model neurodevelopmental diseases that display human-specific features, and how the transplantation of ES/iPS cell-derived cortical neurons can lead to functional integration into developing and damaged cortical circuits.

Symposium II: "Ion channels from development to behavior"

Friday 3rd, 9:00-11:00

Chair: **Nara Muraro**, Fundación Instituto Leloir, Argentina

Friday 3rd, 9:00-9:30

"Synaptic engineering: An ionic switch to C.elegans behavior"

Diego Rayes

Instituto de Investigaciones Bioquímicas de Bahía Blanca, Bahía Blanca, Argentina

Mapping the neural connections of nervous systems is often considered to be a fundamental step in understanding behavior. However, a neural connectivity map carries no information about the activity of neurons and the nature of the connections that each neuron makes. Neurons are embedded in neural networks, which require a delicate balance between excitation and inhibition to maintain network stability. Homeostatic processes, conserved from invertebrates to humans, can adjust synaptic and neuronal excitability to keep neural circuits functioning within their stable dynamic range. In these circuits, ligand-gated ion channels (LGICs) are the principal signaling components that mediate fast inhibitory and excitatory neurotransmission. Is it possible to reverse the behavioral output of a neural circuit by changing the ion selectivity of LGICs and the sign of a synapse? Do intrinsic developmental constraints or homeostatic and behavioral feedback mechanisms prevent switches in the sign of a synapse within a network? We are interested in addressing these questions using the neuronal circuit that mediates the escape response of the nematode Caenorhabditis elegans, the only animal with a completely defined neural wiring diagram . In this circuit tyraminergic neurons coordinate the suppression of head movements with backward locomotion through the activation of a group of Cys-loop biogenic amine-gated chloride channels recently described, the LGCCs. We analyzed the molecular and behavioral consequences of changing the ion selectivity of one of these LGCCs, LGC-55, from anionic to cationic. Our data show that the C. elegans connectome is established independently of the nature of synaptic activity or behavioral output and suggest that switches in LGIC ion selectivity could provide an evolutionary mechanism to change behavior.

Friday 3rd, 9:30-10:00

"How do Drosophila clock neurons fire up?"

Nara I. Muraro

Fundación Instituto Leloir, Argentina

Circadian rhythms have been extensively studied in the fruit fly where many clock genes that interlock through negative feedback loops and generate daily oscillations have been described. Clock genes are expressed in approximately 150 clock neurons in the Drosophila melanogaster brain, of which a particular subset, the pigment dispersing factor-expressing lateral neurons (LNvs) have been found to play a central role.

Still, little is known on the electrical properties of Drosophila clock neurons. The large subtype of LNvs (lLNvs) show spontaneous action potential firing organized in bursts and firing activity that follows a circadian pattern. This daily cycling of neuronal activity could be crucial to confer time of day information to other neurons by altering the release of neurotransmitters or neuropeptides, however, the mechanisms that allow this change in firing activity are not known. We have performed a behavioral genetic screen through the down regulation of candidate voltage-gated ion channels using RNA interference specifically in LNvs. Among the positive hits of the screen, the hyperpolarization-activated cation current Ih and the T-type calcium channel DmαG are being studied further. The role of these currents in Drosophila neurons has not been explored much; however, they have been shown to be key in generating complex neuronal behaviors such as bursting in mammalian neurons. We are using whole-cell patch clamp electrophysiology in ex-vivo

Drosophila brains to study the role of these ion channels in the establishment of lLNvs physiology. Moreover, not only intrinsic, but also synaptic factors, such as Acetylcholine and GABA are contributing to the establishment of the lLNvs firing mode.

Friday 3rd, 10:00-10:30

"Pumilio-2 regulates translation of Nav1.6 to mediate homeostasis of membrane excitability"

Richard Baines

Faculty of Life Sciences, University of Manchester, UK

The ability to regulate intrinsic membrane excitability, in order to maintain consistency of action potential firing, is critical for stable neural circuit activity. Without such mechanisms, Hebbian-based synaptic plasticity could push circuits toward activity-saturation or, alternatively, quiescence. Although now well documented, the underlying molecular components of these homeostatic mechanisms remain poorly understood. Our previous work in the fruit fly, Drosophila melanogaster, identified Pumilio, a translational repressor, as an essential component of one such mechanism. In response to changing synaptic excitation, Pum regulates the translation of the Drosophila voltage-gated sodium channel leading to a concomitant adjustment in action potential firing.

We have recently shown that Pum2 is central to a highly similar homeostatic mechanism regulating membrane excitability in rat visual cortical pyramidal neurons. Using RNA interference, we observed that loss of Pum2 leads to increased sodium current (INa) and action potential firing, mimicking the response by these neurons to being deprived of synaptic depolarisation. By contrast, increased synaptic depolarisation results in increased Pum2 expression and subsequent reduction in INa and membrane excitability. We further show that Pum2 is able to directly bind the predominant voltage-gated sodium channel transcript (NaV1.6) expressed in these neurons and, through doing so, regulates translation of this key determinant of membrane excitability. Taken together, our results show that Pum forms part of a ubiquitous homeostatic mechanism that matches neuron membrane excitability to synaptic depolarization.

Friday 3rd, 10:30-11:00

"Activity-dependent regulation of coordinated ion channel expression: from mRNA to network output"

David Schulz

Department of Biological Sciences, University of Missouri, USA

The nervous system faces an extremely difficult task. It must be flexible, both during development and in adult life, so that it can respond to a variety of environmental demands and produce adaptive behavior. At the same time, it must be stable, so neural circuits that produce behavior function throughout the lifetime of the animal, and stable changes produced by learning endure. Given the challenges of both normal channel protein turnover and short-term plasticity, how is the balance of membrane conductances maintained over long-term timescales to ensure stable electrophysiological phenotype? One possible mechanism is to dynamically regulate production of channel protein via feedback that constrains relationships at the channel mRNA level. Our recent hypothesis is that mRNA relationships emerge as a result of activity-dependent homeostatic tuning rules to ensure an appropriate ratio of mRNA for key ion channels is maintained. We have quantified multiple ion channel mRNAs from single identified motor neurons of the crustacean stomatogastric ganglion and identified distinct, cell-specific correlations among mRNAs for different suites of voltage-dependent channels. We have also determined that these correlations among channel mRNAs are dynamically maintained by an activity-dependent process. These results suggest that cell-specific regulation of steady-state mRNA levels may be a mechanism underlying functional cellular identity. Furthermore, the feedback from cellular activity to coordinated transcriptome-level interactions represents a novel aspect of regulation of neuronal output with implications for long-term stability of neuron function.

Thursday 2nd, 19:30-21:00
Chair: **Tomás Falzone**, CONICET, Universidad de Buenos Aires

Thursday 2nd, 19:30-19:50

"Impact of axonal, autoreceptor mediated, synaptic events on cerebellar interneuron's activity"

Javier Zorrilla de San Martin
Laboratoire de Physiologie Cérébrale, Université Paris 5, 45 Rue des Saints Pères, 75006 Paris, France

The existence of axonal ionotropic receptors in different neuronal types have been known since the early 60's but their role was mainly associated to the modulation of axonal activity and were not considered as an input to be integrated with inputs originating in other neurons. Juvenile cerebellar Molecular Layer Interneurons (MLIs) express presynaptic GABAARs which are activated every time the synapse releases GABA, therefore acting as autoreceptors. In this study, we used local caged-Ca2+ and caged-GABA photolysis in MLI's presynaptic varicosities to induce GABA release and activate GABAA autoreceptor mediated responses. The use of a minimized laser spot (λ=405 nm) for the photolysis allowed us to explore the heterogeneities among synapses and estimate key parameters for the axonal physiology. We measured the axonal space constant (τ=56±9 µm) evoking GABA-release in synapses located at different positions while performing somatic patch clamp recordings. Moreover, fitting these data with a computational model we estimated a mean autoreceptor synaptic conductance of 3 nS. Autoreceptor activation had variable consequences when using the estimated physiological [Cl-]i in current clamp mode. Depending on membrane potential it ranged from small depolarizations (amp: 1 to 7 mV) and action potential triggering to spiking inhibition. Furthermore, subthreshold depolarizations showed a strong sensitivity to TTX, unveiling an amplification mechanism that affects their amplitude and kinetics. Finally, single MLI filling with green Alexa followed by immunolabeling of vesicular GABA transporter and 3D analysis of confocal images showed a concentration of presynaptic specializationsat <100 µm of the somatodendritic compartment, a distance compatible with the space constant of the axon. These results support the view by which the MLI axons can act as input compartments providing signals that can be integrated locally in the axon and at cellular level with inputs coming from other neurons.

Thursday 2nd, 19:50-20:10

"Caspase-3 and Calpains become active during (and play a role in) injury-induced axonal degeneration but are not inhibited during NAD+-mediated protection"

Nicolás Unsain[1,2], Aaron D Johnstone[2] and Phil A Barker[2]

[1]Current affiliation: Instituto Investigación Médica Mercedes y Martín Ferreyra (INIMEC-CONICET), Córdoba, Argentina.

[2]Montreal Neurological Institute, McGill University, Montreal, Canadá.

After nerve injury, the distal portion of severed axons undergoes a degenerative process known as Wallerian degeneration (WD) that leads to axonal fragmentation. Treatment of axons with nicotinamide adenine dinucleotide (NAD+) protects against WD, indicating that the axonal degeneration induced in a regulated process. Our understanding of the molecular mechanism underlying this protection is still rudimentary.

Caspases are aspartate-directed proteases that play critical roles in the regulation and execution of apoptotic cell death during development and tissue maintenance. Initial studies suggested that axonal degeneration proceeded independently of caspases, but recent genetic loss-of-function studies have shown that caspases play a crucial role in developmental axonal pruning (Unsain et al., 2013. Cell Reports). On the other hand, calpains are calcium dependent proteases that play important roles in the execution of necrotic cell death. The precise role of these proteases in injury-induced axonal degeneration is a matter of current debate.

In this study, we use pure samples of axons undergoing degeneration (Unsain et al., 2014, in press. JoVE) to unveil a novel interplay among caspase-3, calpains and the NAD+-sensitive system. We observed that caspase-3 becomes activated in axons early during WD, together with calpains. Calpains, in turn, cleave the N-terminus of caspase-3 and facilitate its activation. Caspase-3 or calpain inhibition greatly delays the detachment of axonal debris from the substrate. We further show that in NAD+-protected axons caspase-3 or calpain activities are normal, suggesting that NAD+-mediated protection does not rely on the inhibition of these proteases. These results show an intriguing relationship between caspases, calpains and the mechanism underlying NAD+-mediated protection.

Thursday 2nd, 20:10-20:30

"Amyloid Precursor Protein Is an Autonomous Growth Cone Adhesion Molecule Engaged in Contact Guidance"

Lucas J. Sosa [1,2], J. Bergman[1], A. Estrada-Bernal[1], T. J. Glorioso[1], J. M. Kittelson[1], and K. H. Pfenninger[1]

[1]Department of Pediatrics and Colorado Intellectual and Developmental Disabilities Research Center, University of Colorado School of Medicine, Aurora, Colorado, United States of America. [2]. CIQUIBIC-Dpto Química Biológica, Facultad de Ciencias Químicas, Universidad Nacional de Córdoba, Argentina

Amyloid precursor protein (APP) is a transmembrane glycoprotein, which is best known for its involvement in the pathogenesis of Alzheimer disease. Encoded on human chromosome 21 APP is overexpressed in Down syndrome (DS) brain and, thus, may contribute to DS-associated intellectual disability. However the physiological function of APP during brain development is poorly understood. APP is a prominent component of the adult as well as the developing brain. It is enriched in axonal growth cones (GCs) and has been implicated in cell adhesion and motility. We tested the hypothesis that APP is an extracellular matrix adhesion molecule. To this end we plated wild-type, APP-, or β1-integrin (Itgb1)- misexpressing mouse hippocampal neurons on matrices of either laminin, recombinant L1, or synthetic peptides binding specifically to Itgb1 or APP. We measured GC adhesion, initial axonal outgrowth, and substrate preference on alternating matrix stripes. Our results shows that substrates of APP-binding peptide alone sustain neurite outgrowth; APP dosage controls GC adhesion to laminin and APP-binding peptide as well as axonal outgrowth in Itgb1– independent manner; and APP directs GCs in contact guidance assays. It follows that APP is an independently operating cell adhesion molecule that affects the GC's phenotype on APP-binding matrices including laminin, and that it is likely to affect axon pathfinding in vivo.

Thursday 2nd, 20:30-20:50

"Neurogenin3 is a key regulator in serotonergic vs. glutamatergic neuronal cell fate"

Abel Carcagno, Daniela Di Bella and Guillermo Lanuza
Fundación Instituto Leloir, Buenos Aires, Argentina

The production of functionally diverse neuronal cell types at their correct locations requires the acquisition of specific progenitor identities in response to extrinsic positional cues. In the developing neural tube of amniotes, hindbrain serotonergic (5-HT) neurons and spinal V3 glutamatergic interneurons are produced from ventral progenitors, which possess a common transcriptional identity but are confined to distinct anterior-posterior territories. It is not completely understood how discrete progenitor pools expressing a seemingly identical molecular code give rise to divergent neuronal fates.

In this study, we identify that the expression of the transcription factor Neurogenin3 (Neurog3) in the spinal cord controls the correct specification of ventral neural tube cells. Gain-of-function experiments in the chick embryo show that Neurog3 represses the expression of the 5-HT determinant Ascl1 through a mechanism that is dependent on the activity of Hes proteins.

Conversely, the spinal cord of Neurog3 mutant mice displays abnormal elevated levels of Ascl1, which triggers the ectopic induction of the serotonergic differentiation program sequentially controlled by the transcription factors Gata2, Lmx1b and Pet1.

The ectopic spinal 5-HT neuron production in Neurog3 mutant mice resembles the serotonergic system of aquatic vertebrates, which interestingly lack Neurog3 expression.

In summary, our results show that Neurog3 serves as a mechanism for interpreting anterior-posterior signaling to impose the caudal border for the serotonergic rafe system in amniotes, and explain how equivalent progenitors within the hindbrain and the spinal cord can produce distinct functional neuron cell types.

MEETING |SAN AWARD TO THE BEST DOCTORAL THESIS IN NEUROSCIENCE 2014

Wednesday 1st, 20:15-20:45
Chair: **Juan Goutman**, Instituto de Investigaciones en Ingeniería genética y Biología Molecular, Dr Héctor N. Torres, Argentina

"Stress-Induced Cocaine Sensitization: A Study of Glutamate Homeostasis and its Interaction with the Dopaminergic System in Nucleus Accumbens"

Constanza García Keller
Dpto de Farmacología, Fac. Ciencias Químicas, Universidad Nacional de Córdoba, Argentina - Department of Neurosciences, Medical University of South Carolina, USA

In both, animals and humans, the repeated administration of psychostimulants increases the behavioral response to a new exposure. This phenomenon is called "sensitization" and extends to the stress-induced influence in stimulant behavioral effects of psychostimulants, called "cross-sensitization". Thus, drug addiction is a multifactorial disorder where individual previous experiences, i.e. influence of stress, interact and modulate the individual response to addictive drugs and increase the vulnerability to drug addiction (Chen and Anthony, 2004).

Acute or repeated psychostimulant treatment or acute or repeated stress induces long-term changes in behavioral and neurochemical expression of sensitization to the drug. Stress-induced mesocorticolimbic dopaminergic neuroadaptations that are thought to be initiated by the fact that both addictive drugs and stress increase the release of corticotrophin-releasing factor (CRF) into the ventral tegmental area (VTA), augment the response of dopamine neurons to glutamatergic inputs (Saal et al., 2003; Ungless et al., 2003). These adaptations in VTA glutamate transmission by stress are thought to mediate enduring changes in both dopamine and glutamate transmission in the nucleus accumbens (NAc, Pacchioni et al, 2007;Kalivas and Stewart, 1991).

The NAc is a heterogeneous structure that can be separated histologically into Core and Shell subdivisions (Pennartz et al., 1994). Dopamine release in Shell and Core is differentially sensitive to drugs of abuse (Di Chiara, 2002) and a role for glutamate in Core has been shown in the expression of cocaine-induced behavioral sensitization (Pierce et al., 1996). Here we endeavor to elucidate the plastic changes in NAc induced by a single restraint stress expressed tree weeks after the cocaine administration. Notably, certain stress-induced long-lasting neuroadaptations in the transmission or glutamatergic and dopaminergic pharmacology observed in the Core are similar to those observed after the cocaine withdrawal (Pierce et al, 1996). Specifically, we identified a role of AMPA receptor in the NAc), alteration in glutamate homeostasis, reflected in basal and synaptic glutamate released, decreased glutamate transporter (GLT-1) expression, and the differential impact in postsynaptic neurons of NAc Core and Shell (Conrad et al, 2008; Backer et al, 2003; Kalivas, 2009).Even more relevant is that pharmacological therapies proposed for the treatment of addiction to different drugs of abuse, including cocaine, in animal models (Knackstedt et al, 2010a;) and humans (LaRowe et al, 2007), could also be used in populations exposed to stressful events that are potentially vulnerable to developing comorbid substance abuse disorder. The similarity between acute stress-induced glutamatergic neuroadaptations in NAcore and those produced by the self-administration of addictive drugs, poses common points of pharmacological intervention that may be particularly useful in treating population suffering from stress disorders and substance use disorder.

LISTS

Room A

ST1.-Retrieval or reconsolidation of a fear memory can be independently affected by an appetitive experience

Roque Ignacio Ferrer Monti[1°], Joaquín Matías Alfei[1°], Adrián Marcelo Bueno[1°], Victor Alejandro Molina[2°]
[1°] Laboratorio de Psicología Experimental, Facultad de Psicología, Universidad Nacional de Córdoba, Argentina; [2°]Instituto de Farmacología Experimental Córdoba (IFEC-CONICET), Facultad de Ciencias Químicas, Universidad Nacional de Córdoba, Argentina
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ST2.-Evidence of postnatal astro and microgliosis in the valproic acid model of autism

Nadia Kazlauskas, Luciana Lucchina, Marcos Campolongo, Amaicha Depino
Institute for Physiology, Molecular Biology and Neurosciences, CONICET-UBA; Department of Physiology, Molecular and Cellular Biology, FCEyN, University of Buenos Aires
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ST3.-Early social stimulation and the stress response in an animal model of autism

Marcos Campolongo, Nadia Kazlauskas, Amaicha Depino
Departamento de Fisiología, Biología Molecular y Celular, FCEyN, Universidad de Buenos Aires; Instituto de Fisiología, Biología Molecular y Neurociencias, CONICET-UBA, Buenos Aires, Argentina
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ST4.Guess who's learning too!

Angeles Salles[1°], María del Carmen Krawczyk[2°], Mariano Boccia[2°], Arturo Romano[1°], Ramiro Freudenthal[1°]
[1°]IFIBYNE, CONICET- FCEN, UBA; [2°]FFyB, UBA
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ST5.-Dopamine signaling: the missing link between circadian and interval timing

Ivana Leda Bussi[1°], Gloria Levin[2°], Diego Golombek[1°], Patricia Agostino[1°]
[1°]Laboratorio de Cronobiología, Universidad Nacional de Quilmes/CONICET, Buenos Aires, Argentina; [2°]Hospital de Niños Ricardo Gutiérrez, CEDIE-CONICET , Buenos Aires, Argentina
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ST6.-Nanoparticles for targeted drug delivery to glial cells after brain ischemia

Veronica Murta[1°], Priscila Schilrreff P[2°], Mariana Seib[2°], María José Morrilla[2°], Alberto Javier Ramos[1°]
[1°]Instituto de Biología Celular y Neurociencias "Profesor Eduardo De Robertis", CONICET, Facultad de Medicina, Universidad de Buenos AiresA; [2°]Programa de Nanomedicinas, Universidad de Quilmes
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Room B

ST7.-Two signaling pathways mediate presynaptic voltage gated calcium channels inhibition by ghrelin receptor activation

Eduardo Javier López Soto, Francina Agosti, Valentina Martínez Damonte, Emilio Román Mustafa, Silvia Rodríguez, Jesica Raingo
Laboratorio de Electrofisiología, Instituto Multidisciplinario de Biología Celular (IMBICE- CICPBA/CONICET)
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ST8.-Cell reprogramming to model epilepsy

Mariana Laura Casalia[1°], Juan Cruz Casabona[1°], Veronica Cavaliere Candedo[1°], Isabel Farias[1°], Joaquin Gonzalez[1°], Ramiro Quinta[1°], Juana Pasquini2°, Marcelo Kauffman[2°], Fernando Pitossi[3°],
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ST9.-The Leucine-rich repeat transmembrane protein Lrig1 restricts hippocampal dendrite complexity modulating neurotrophin-induced TrkB signaling

Fernando Cruz Alsina, Francisco Javier Hita, Paula Fontanet, Dolores Irala, Fernanda Ledda, Gustavo Paratcha
Division of Molecular and Cellular Neuroscience, Institute of Cellular Biology and Neuroscience Prof. Dr. E. De Robertis (IBCN)-CONICET, School of Medicine. University of Buenos Aires (UBA), Buenos Aires, Argentina
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ST10.-First evidence of the proteasome fast axonal transport mediated by molecular motors and membrane interaction

María G. Otero[1°], Trinidad MM. Saez[2°], Matías Alloatti[1°], Lucas E. Cromberg[1°], Victorio M. Pozo Devoto[1°], Tomas L. Falzone[2°]
[1°]Instituto de Biología Celular y Neurociencia (IBCN-CONICET); [2°]Instituto de Biología y Medicina Experimental (IBYME-CONICET)
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ST11.-From axonal transport to physiology. Neuronal specific dependence for Kif5b, an ubiquitous molecular motor

Lucas Eneas Cromberg, Trinidad Maria de los Milagros Saez, Maria Gabriela Otero, Victorio Pozo Devoto, Matías Alloatti, Tomás Falzone
Instituto de Biología Celular y Neurociencias
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ST12.- SRm160, a splicing factor behind the clock

Esteban J. Beckwith, Agustina P. Bertolin, M. Fernanda Ceriani, Marcelo Yanovsky
Fundación Instituto Leloir, IIBBA-CONICET, Buenos Aires, Argentina
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Cellular and Molecular Neurobiology

P1.-GHSR1a constitutive activity decreases presynaptic voltage-gated calcium channels level in plasma membrane

Francina Agosti[1°], Eduardo Javier López Soto[1°], Valentina Martínez Damonte[1°], Alejandra Gandini[2°], Silvia Rodríguez[1°], Jacky Marie[3°], Michel Vignes[3°], Ricardo Félix[2°], Jesica Raingo[1°]
[1°]Laboratorio de Electrofisiología, Instituto Multidisciplinario de Biología Celular (IMBICE- CICPBA/CONICET); [2°]Departmento de Biología Celular, Centro de Investigación y de Estudios Avanzados del Instituto Politécnico Nacional.; [3°]Université Montpellier 2;
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P2.-Association study of 6 polymorphisms –SNP- related to the developmental coordination disorder (DCD) in colombian children and adolescents from 7 to 16 years: pilot study

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P3.-Demyelination-remyelination in the CNS: ligand-dependent participation of the Notch signaling pathway

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P4.-The Leucine-rich repeat transmembrane protein Lrig1 restricts hippocampal dendrite complexity modulating neurotrophin-induced TrkB signaling

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P5.-Coronin-1a is involved in neuronal filopodia formation induced by M6a

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P6.-Activity-dependent neuronal maturation in the adult hippocampus

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P7.-Morphological changes in subcortical white matter following cognitive training in *Macaca fascicularis*

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P8.-Neurodegeneration associated to copper and cholesterol administration in Wistar rats

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P9.-Effect of Constant Low Light Exposure on Rat Retina: A Model of Retinal Degeneration

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P10.-Measuring synaptic protein acetylation

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P11.-Network of photoreceptors: An analysis of a model of high complexity

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P12.-Cell reprogramming to model epilepsy

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P13.-Maternal protein malnutrition affects morphological and neurological development in mouse littermates

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P14.-Improved design of Angiotensin II AT2 riboprobes

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P15.-Role of Wnt5a in the neuronal development and its participation on the glyphosate induced neurotoxicity

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P16.-From axonal transport to physiology. Neuronal specific dependence for Kif5b, an ubiquitous molecular motor

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P17.-Behavioural phenotypes rescued in Tau Knock-out mice by human Tau re-expression

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P18.-GHSR1a constitutive and ligand evoked activity inhibits GABAergic transmission in primary neuronal cultures

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P19.-Different roles of the TrkB and p75NTR neurotrophin receptors in the Preconditioning effect in a coculture model of Status epilepticus in vitro.

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P20.-Gene regulatory network controlling late neurogenesis in the developing spinal cord

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P21.-Differential Regulation of Myelin Basic Protein Isoforms and Deiminated Isomers by Calmodulin

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P22.-Selective Oxidation of Alpha-Synuclein Promotes its Cytotoxicity

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P23.-Hypothalamic tanycytes mediate ghrelin uptake into brain tissue

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P24.-Inhibition of CDK5 alters Dopamine Transporter endocytosis in N2A neuroblastoma cells

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P25.-WNT7B and FRIZZLED7 are involved in the regulation of dendrite architecture by the non-canonical WNT pathways

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P26.-Novel modulators of the neurotrophic actions of NGF and its receptor TrkA

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P27.-Pea3 transcription factors are key mediators of hippocampal dendrite growth during development

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P28.-Filopodia formation driven by M6a depends upon M6a´s oligomerization

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P29.-Phosphorylation of M6a at Serine-267 induces filopodia formation in rat hippocampal neurons

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P30.-The pineal gland: thinking outside the box

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P31.-Progesterone effects on transcription factors that drive oligodendrogenesis after spinal cord injury

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P32.-Array Tomography as a Tool for Tracking the Distribution of ASIC1a at the Neuromuscular Junction

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P33-Two signaling pathways mediate presynaptic voltage gated calcium channels inhibition by ghrelin receptor activation

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P34.-TDP-43 transgenic mouse models display altered brain polysomal profiles

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P35.-Study of the adaptive evolution of the α9α10 cholinergic nicotinic receptor

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P36.-Arc is required for pattern separation across the spatial and the object domains in the dentate gyrus and perirhinal cortex

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P37.-Regional differences of astrocyte response to neurotrophins

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P38.-To die or to not die... The fight between TrkB and p75ntr signaling

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P39.-Sensing Light by Horizontal Cells in the Chicken Retina: A New Player in the Photoreceptive System

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P40.-Nanoparticles for targeted drug delivery to glial cells after brain ischemia

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P41.-Study of the impact of ghrelin receptor dimerization with other G-protein coupled receptors on calcium channel modulation

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P42.-First evidence of the proteasome fast axonal transport mediated by molecular motors and membrane interaction

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P43.-Myelin-Associated Glycoprotein modulates programmed cell death of motoneurons during early postnatal development via NgR/p75NTR receptor-mediated activation of RhoA signaling pathway

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P44.-Microglial Alterations Before And After Plaque Deposition in PDAPP-J20 MICE, Model of Alzheimer's Disease

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P45.-Remyelination by Bone Marrow Mononuclear Cells: efficiency in cell tracking vs efficiency in cell functionality.

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P46.-Effects of propranolol applied before and after the processing of novelty as a modulator of frustration

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P47.-Electroretinographic signals in a model of retinal degeneration in rats

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P48.-Genetic characterization of the promoter polymorphism in SLC6A4 gene (5HTTLPR) in a sample of Colombian population with Major Depression: Pilot Study

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P49.-Guillain Barre Syndrome-Associated Anti-Glycan Antibodies Alter Growth Cone Tubulin Cytoskeleton Via RhoA/ROCK Pathway from Growing DRGs Neurons

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P50.-New properties of motor protein dependent transport in the axonal guidance of the telencephalic axonal tracts

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P51.-Guess who's learning too!

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P52.-The small G-protein RAS modulates olfactory memory and synaptic plasticity of neuromuscular junction in Drosophila

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P53.-Astrocyte Heterogeneity Derived from a Single Spinal Cord Progenitor Domain

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P54.-PEDF expression in normal and illuminated rat retina

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P55.-Analysis of expression and function of voltage-activated potassium KCNQ channels on mouse eye

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P56.-Abolition of the Sex Difference in Ngn3 By Estradiol is Depending on Sex Chromosome Complement

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Chronobiology

P57.-Photic Sinchronization of Circadian Rhythms in Mammals: The Role of Second Messengers Downstream Protein-Kinase G

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P58.-SRm160, a splicing factor behind the clock

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P59.-Circadian Rhythmomas: Chronobiology of a Glioma Cell Line

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P60.-Dopamine signaling: the missing link between circadian and interval timing

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Cognition, Behavior, and Memory

P61.-Influence of maternal experience on behavioral response to the maternal separation stress in mother rats: Preliminary results

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P62.-The Metacognitive Abilities of Children and Adults

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P63.-The Occurrence of a Temporal Prediction Error During Reinforced Reactivation is Critical to Induce Memory Destabilization

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P64.-Conditional overexpression of the neurodegenerative disease-related protein TDP-43 leads to cognitive and social abnormalities in transgenic mice

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P65.-Is major depression a matter of size?

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P66.-The Effect of Palatable Solutions in the Memory Impairment Induced by Sleep Deprivation in Rats

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P67.-Functional corticostriatal disconnection and behavioral exploitation-exploration imbalance emerge as intermediate phenotypes for a neonatal dopamine dysfunction

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P68.-Early social stimulation and the stress response in an animal model of autism

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P69.-Two-trial spaced training in Drosophila reveals that repetition and spacing in learning improves memory by similar mechanisms

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P70.-Cocaine induced sensitization is associated with decrease activity of the Wnt/βcatenin pathway in Dorsal Striatum

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P71.-The influence of stress on the structural plasticity associated with fear extinction memory

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P72.-Effect of Intra-Core, but Not Intra-Shell, mGlu I Antagonist Administration in Restraint Stress-Induced Reinstatement on Extinguished Cocaine-Conditioned Animals

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P73.-Involvement of δCamkII protein in persistent forms of memory

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P74.-Sex differences in human reconsolidation of episodic memories

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P75.-Retrieval or reconsolidation of a fear memory can be independently affected by an appetitive experience

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P76.-Development of a retrieval-induced forgetting paradigm in rodents to model adaptive forgetting in the mammalian brain

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P77.-Neurosteroids and Gabaergic Activity on Lateral Septum

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P78.-Ghrelin increases memory consolidation through hippocampal mechanisms dependent of glutamate release and NR2B subunits of the NMDA receptor

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P79.-The promoting influence of stress on hippocampal structural plasticity and on fear memory is modulated by GABAergic signaling within the Basolateral Amygdala Complex

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P80.-Intergenerational effects of perinatal protein malnutrition on maternal and offspring behavior

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P81.-Stress induced by maternal manipulation during late gestation increases ethanol intake in offspring

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P82.-Hippocampal alpha7 nicotinic receptors modulate memory reconsolidation of an inhibitory avoidance task in mice: possible participation of the MAPK pathway

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P83.-Studing the function of the reconsolidation process: Analysis of the reactivation of the Contextual Pavlovian Conditioning memory triggered by the prediction error in the crab Neohelice granulata

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P84.-A novel spherical treadmill apparatus for the head-fixed behaving rat

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P85.-Rearing in an Enriched Environment Can Prevent Most Behavioral Alterations Induced by Acute Noise Exposure, Independently of the Exposure Age

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P86.-Role of Medial Prefrontal Cortex and Perirrhinal Cortex during Reconsolidation in Object Recognition Memory Task in Rats

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P87.-Assessment of memory extinction of an inhibitory avoidance task

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P88.-Disruption of fear memory reconsolidation by an appetitive stimulus in ethanol withdrawn rats pre-treated with D-cycloserine

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P89.-Positive emotional induction interferes with the reconsolidation of autobiographical memories

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P90.-Cortical interneuron impaired function and psychiatric diseases.

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Computational Neuroscience

P91.-Role of the hilar cells of the dentate gyrus in pattern separation and storage in the hippocampus: a computational model

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P92.-Pressure patterns in birdsong as the activity of the telencephalon is thermally manipulated

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P93.-Specificity quantification of texture discrimination processes in vibrissal system

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P94.-Permutation entropy applied to the characterization of the clinical evolution of epileptic patients under pharmacological treatment

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P95.-Discriminability measures and time–frequency features: An application to vibrissal tactile discrimination

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P96.- From bipolar to unipolar recordings

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Motor Systems

P97.-Selective neurons in the nucleus HVC of the domestic canary

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P98.-Experimental validation of a minimal model for birdsong production

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P99.-Similar extrapyramidal side effects with typical and atypical antipsychotics chronic treatment

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P100.-Cortical Responses to Speech Production

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P101.-Electrophysiological characterization of muscle activation in 6-OHDA rat model of Parkinson's disease

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Neural Circuit Physiology

P102.-Neural activity alterations and functional connectivity deficits in a developmental mouse model of schizophrenia

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P103.-Optical photorelease of dopamine in freely moving animals

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P104.-Circulating or cerebrospinal fluid ghrelin regulates different neuronal circuits within the dorsal vagal complex

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P105.-Ghrelin signaling is required for escalation in high-fat intake during repeated binge eating episodes

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P106.-Unveiling the CRF Neurons of the Amygdala: Neuroanatomical and Functional Characterization using a Novel Transgenic Mouse Model

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P107.-Unbiased prediction of the degree of midbrain dopaminergic neuron loss in parkinsonian mice using behavioral parameters

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P108.-Analysing limited electrophysiological data by artificial sample generation

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P109.-Dorsal raphe nucleus lesion modulates sodium appetite

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P110.-Development of a feedback circuit for input specification in adult-born dentate granule cells

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P111.-Theta-oscillations in visual cortex emerge with experience to convey expected reward time and reward rate

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Neurochemistry and Neuropharmacology

P112.- JM-20, a new hybrid benzodiazepine - dihydropyridine molecule prevents neuronal cell death in different in vitro model of Parkinson's diseases

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P113.-Stress and vulnerability to develop cocaine addiction: role of glutamatergic transmission

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P114.-Modulation of GABAA-rho1 receptors by L-cysteine

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P115.-Serotoninergic alterations in CB1R knockout mice

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P116.-Previous stress diminishes the interfering effect of midazolam on fear memory reconsolidation. Effect of intra basolateral amygdala D-cycloserine administration

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P117.-Methamphetamine and modafinil differentially alter mRNA expression of epigenetic regulators in the mouse prefrontal cortex

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P118.-Activation of Cannabinoid CB1 Receptor within Nucleus Accumbens Core Underlies Restraint Stress-Induced Reinstatement in Extinguished Cocaine–Conditioned Animal

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P119.-Genotoxicity and alteration of spontaneous and evoked activity of the nervous system induced by repeated exposure to low levels of chlorpyriphos

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P120.-Chronic intermittent intake of caffeine and cocaine in mice induced differential effects on locomotor sensitization and glutamatergic gene expression in mPFC

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P121.-Perinatal protein deprivation facilitates morphine cross-sensitization to cocaine in adult rats

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P122.-Repeated intermittent treatment with cocaine, caffeine or their combination alters intrinsic and synaptic properties of ventrobasal thalamic neurons

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P123.-Blockage of ANG II AT2 receptors modifies cerebelar foliation process

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Neuroendocrinology and Neuroimmunology

P124.-Expression of key steroidogenic enzymes in developing brain: hormonal compensation of sex chromosomes-induced sex differences

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P125.-Neuronal regulation of the stress response in *C. elegans*: Role of the neurotransmitter tyramine

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P126.-Evidence of postnatal astro and microgliosis in the valproic acid model of autism

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P127.-Does L-DOPA have neuroendocrine effects?

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P128.-Long-term effects of lifelong aerobic exercise on the stress response in middle-aged and old rats

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P129.-Toll-like receptors TLR2 and TLR4 are involved in reactive gliosis and microglial activation after ischemic brain injury

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Sensory Systems

P130.-Connexin 43 contributes to gap junction coupling in olfactory ensheathing glia

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P131.-Behavioural characterization of the response to polarization motion stimuli in an arthropod

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P132.-Differential calcium responses of visual columnar neurons to different parameters of visual motion stimuli

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P133.-Effects of loud noise on the efferent system of the inner ear

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P134.-A new preparation for extracellular electrophysiological recordings at the level of the optic nerve in the crab *Neohelice granulata*

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P135.-Wide-field stimulation system for measuring of visuomotor behaviors in arthropods

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Synaptic Transmission and Excitability

P136.-Transcranial ultrasound modulates ketamine-xylazine effects in mice

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P137.-ATP and adenosine modulate acetylcholine release through P2Y and P1 receptors at the efferent-inner hair cell synapse in the developing inner ear

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P138.-Carbonic anhydrase pH regulation modulates short term plasticity at the mouse neuromuscular junction

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P139.-Characterization of responses mediated by low threshold calcium conductances in a nonspiking neuron

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P140.-Carbonic anhydrase modulates short term plasticity at central synapses

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P141-Voltage-gated Ca2+ channels (VGCC) that support ACh release at the mouse efferent-inner hair cell synapse during early stages of development

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P142.-Localized calcium signals in inner hair cells of the developing inner ear following efferent fiber stimulation

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P143.-Can neurosecretion be independent of calcium entry? Some new answers for an old question

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P144.-Activation of presynaptic GABAB receptors enables sustained transmission at high rate of stimulation in cholinergic olivocochlear-hair cell synapses

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P145.-Properties of the olivocochlear efferent synapse relevant for the regulation of the auditory periphery

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BAW 2014

P146.-BAW 2014, La Plata: "Electric Brain"

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P147.-Brain Awarenes Week Córdoba - Getting to know our brain

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P148.-What do you have in your head? An approach to neuroscience in middle schools of Rio Ceballos and Villa Carlos Paz

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P 149.-The Brain Awareness Week in Bariloche

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