



Second Joint Meeting of the Argentine Society for Neuroscience (SAN: XXV Reunión Anual de la Sociedad Argentina de Investigación en Neurociencias) and the Argentine Workshop in Neuroscience (TAN: XII Taller Argentino de Neurociencias)

*Huerta Grande, 6 al 10 de octubre, 2010*







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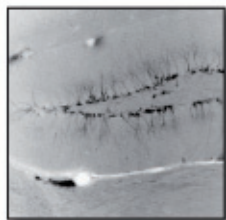




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## IIRCN ← Organizing Committee

*Dr. Maria Paula Faillace.* Investigador Adjunto (Consejo Nacional de Investigaciones Científicas y Técnicas), Laboratorio de Retina de Vertebrados, Departamento de Fisiología, Facultad de Medicina, Universidad de Buenos Aires e Instituto de Química y Fisicoquímica Biológicas. (IQUIFIB, CONICET).

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## Course ← Organizing Committee

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*Lic. Juan Kamienkowski.* Laboratorio de Neurociencia Integrativa. Departamento de Física, Facultad de Ciencias Exactas y Naturales, Universidad de Buenos Aires.

**Logistic Organization: Silvina Andrea Ceriani**



## → WELCOME!

*We are really happy of getting together at the 2nd Joint Meeting of Neuroscience in Argentina!*

After the success of the first Joint Meeting, this year's organizers gratefully acknowledge your participation in this second edition.

We are proud of being part of this endeavor with the main purpose of consolidating Argentinean neuroscientist's reunion, at least once a year, for sharing common interests, positive criticisms, ideas and projects. Moreover, we hope this meeting results a suitable place for encouraging scientific discussions and future collaborations.

We also thank researchers from other countries that came to kindly share their expertise with us.

We really expect you have a great time and that this experience serves to all of us for enriching our lives as scientists and (Why not?) for making new friends.

## → BIENVENIDOS!

*¡Estamos muy felices de encontrarnos en la Segunda Reunión Conjunta de Neurociencias en Argentina!*

Luego del éxito de la Primer Reunión Conjunta, los organizadores de este año agradecen su participación en esta segunda edición.

Estamos orgullosos de ser parte de este emprendimiento cuyo principal propósito es consolidar la reunión de los neurocientíficos argentinos, al menos una vez al año, para compartir intereses comunes, críticas constructivas, ideas y proyectos. Asimismo, esperamos que esta reunión sea un lugar propicio para alentar discusiones científicas y futuras colaboraciones.

Queremos también agradecer a los investigadores de otros países que amablemente aceptaron venir y compartir sus conocimientos con nosotros.

Realmente esperamos que la pasen muy bien y que esta experiencia nos sirva a todos para enriquecer nuestras vidas como científicos y (¿Por qué no?) para hacer nuevos amigos.



# COURSE PROGRAM

Physics and Biology:  
heading towards quantitative  
neuroscience

**WEDNESDAY, OCTOBER 6<sup>TH</sup>** ←

<b>8.00</b>	<b>Registration</b>
<b>9.30</b>	<b>Coffee break</b>
<b>10.00</b>	Introductory lecture: Physics and Biology: heading towards quantitative neuroscience (Manuel Eguia).
<b>10.30</b>	Pascal Martin, Institut Curie recherche, Paris, France. I: <i>"The psychophysical properties of hearing. Mechanical vibrations in the ear."</i>
<b>12.00</b>	Pascal Martin II: <i>"Mechano-electrical transduction by the inner ear's receptor cells, the hair cells"</i>
<b>13.30</b>	<b>Lunch.</b>
<b>14.30</b>	Pascal Martin III: <i>"Somatic electromotility by mammalian outer hair cells The "critical" cochlea: active oscillators as amplifying elements in hearing".</i>
<b>16:00</b>	Laurent Bourdieu, Institut de Biologie de l'Ecole Normale Supérieure, Paris, France. I: <i>"Sensory coding in the whisker to barrel pathway".</i>
<b>17:30</b>	<b>Coffee break.</b>
<b>18:00</b>	Laurent Bourdieu II: <i>"Recent optical developments in neuron network imaging".</i>
<b>19:30</b>	Laurent Bourdieu III: <i>"Late maturation of the direction selectivity map in the rat barrel cortex".</i>

<b>21.00</b>	<b>Dinner.</b>
<b>22:00</b>	Round table: new perspectives in neuroscience.

## THURSDAY, OCTOBER 7<sup>TH</sup> ←

<b>8.30</b>	<b>Breakfast with speakers.</b>
<b>10:00</b>	Dario Ringach, Jules Stein Eye Institute, David Geffen School of Medicine, University of California, Los Angeles, USA. I: <i>"Receptive fields and maps in early visual cortex: basic concepts"</i>
<b>11:30</b>	Dario Ringach II: <i>"Receptive fields and maps in early visual cortex: open questions"</i>
<b>13.00</b>	<b>Lunch.</b>
<b>14:00</b>	Dario Ringach III: <i>"Receptive fields and maps in early visual cortex: a statistical wiring model of primary visual cortex"</i>
<b>15:30</b>	Rodrigo Quian Quiroga, Department of Engineering, University of Leicester, United Kingdom. I: <i>"Processing of extracellular recordings: spike sorting"</i>
<b>17:00</b>	<b>Coffee break.</b>
<b>17:30</b>	Rodrigo Quian Quiroga II: <i>"Extracting information from neural populations: Basic principles and clinical applications"</i>
<b>19:00</b>	Matias Ison, Department of Engineering, University of Leicester, United Kingdom. <i>"A possible mechanism for generating sparse responses in the human medial temporal lobe"</i>
<b>21.00</b>	<b>Dinner.</b>
<b>22:00</b>	Closing round table: Bridging scales in neuroscience.



# II RCN MEETING PROGRAM

FRIDAY, OCTOBER 8<sup>TH</sup> ←

7.30	Registration
19.00   12.30	<p>Symposium I: <i>"Sensory Systems"</i></p> <p>Chairs: Belen Elgoyhen (Univerdidad de Buenos Aires) and Paul Fuchs (Johns Hopkins University)</p> <p><b>9.15: Paul Fuchs</b>, Department of Otolaryngology, Head and Neck Surgery, Johns Hopkins University School of Medicine, USA</p> <p><i>"Afferent and efferent synaptic physiology in cochlear hair cells"</i></p> <p><b>10.00: Randall Reed</b>, Molecular Biology &amp; Genetics, Johns Hopkins University, USA</p> <p><i>"Pathways responsible for converting smells into signals perceived by the brain and the role of these genes in wiring this extraordinary sensory system"</i></p>
10.45	<p><b>Coffee break</b></p> <p><b>11.00: Xinzhong Dong</b>, The Solomon H. Snyder Department of Neuroscience, Johns Hopkins University School of Medicine, USA <i>"Peripheral Pain sensing neurons: Identification of many genes specifically expressed in pain-sensing neurons in dorsal root ganglia"</i></p> <p><b>11.45: Darío Protti</b>, Bosch Institute, Department of Physiology,</p>

University of Sidney, Australia

*"Synaptic inputs generating receptive field properties in  
mammalian retinal ganglion cells"*

<b>12.45</b>	Lunch
<b>14.45 I 16.15</b>	<p>Young Investigators Colloquium I</p> <p><b>Jimena Ballestero</b>; INGEBI- CONICET, Buenos Aires, Argentina</p> <p><i>"Properties of the olivocochlear-outer hair cell synapse in the mouse cochlea"</i></p> <p><b>María Laura Ceci</b>, Laboratory of Telencephalic Development, Instituto Cajal (CSIC), Madrid. Spain</p> <p><i>"Analysis of early migratory pathways in whole embryos"</i></p> <p><b>María José De Rosa</b>, Instituto de Investigaciones Bioquímicas Bahía Blanca, Buenos Aires, Argentina</p> <p><i>"Neurotransmitter systems in lymphocytes"</i></p>
<b>16.30 I 19.00</b>	Poster Session I
<b>19.00 I 21.10</b>	<p>Symposium II: <i>"Stress system: Old diseases, new interactions"</i></p> <p>Chair: Marta Suárez, Facultad de Ciencias Exactas. Físicas y Naturales, Universidad Nacional de Córdoba.</p> <p><b>19.10 Ron de Kloet</b>, University of Leiden, Holland</p> <p><i>"Brain corticosteroid balance and adaptation: a hypothesis revisited"</i></p> <p><b>19.50 Alejandro De Nicola</b>, Facultad de Medicina, Universidad de Buenos Aires, Argentina</p> <p><i>"Stress, hypertension and the mineralocorticoid system"</i></p> <p><b>20.30 Katia Gysling Caselli</b>, Facultad de Ciencias Biológicas, Pontificia Universidad Católica de Chile, Chile</p> <p><i>"Drug addiction, corticotrophin releasing hormone and the ventral tegmental area"</i></p>
<b>21.30</b>	Dinner
<b>23.00</b>	SAN business meeting - Argentina Chapter SFN Meeting

<b>9.00   12.00</b>	<b>Poster Session II</b>
<b>12.00</b>	Lunch
<b>14.00   15.00</b>	<p>Ranwell Caputto Plenary talk</p> <p>Víctor Molina, IFEC-CONICET, Dpto de Farmacología, Facultad de Ciencias Químicas, Universidad Nacional de Córdoba, Argentina</p> <p><i>"From fear to a traumatic memory: modulating factors involved"</i></p>
<b>15.00   16.30</b>	<p>Young Investigators Colloquium II</p> <p><b>Joaquín Piriz</b>; Instituto de Fisiología Biología Molecular y Neurociencias (IFIBYNE), UBA, Buenos Aires, Argentina</p> <p><i>"Synaptic potentiation onto lateral habenula neurons in the learned helplessness model of depression"</i></p> <p><b>Lionel Müller Igaz</b>; Departamento de Fisiología, Facultad de Medicina, UBA, Buenos Aires, Argentina</p> <p><i>"Role of TDP-43 in neurodegenerative disease"</i></p> <p><b>María Noel Federman</b>; Laboratorio de Neurobiología de la Memoria, IFIBYNE-CONICET, FCEN-UBA, Argentina</p> <p><i>"Epigenetic mechanisms in long term memory"</i></p>
<b>16.30   20.00</b>	<p>Symposium III: <i>"Structural biology of neurodegenerative processes: alpha-synuclein and Parkinson disease"</i>.</p> <p>Chairs: Claudio Fernández (Instituto de Biología Molecular y Celular de Rosario) and Santiago Quiroga (Universidad Nacional de Córdoba)</p> <p><b>16.45 Soledad Celej</b>, Facultad de Ciencias Químicas, Universidad Nacional de Córdoba, Argentina</p> <p><i>"Native alpha-synuclein and its mutants involved in the family variant of Parkinson disease"</i></p> <p><b>17.30 José M. Souza</b>, Facultad de Medicina. Universidad de la República, Montevideo, Uruguay</p> <p><i>"Role of alpha-synuclein phosphorylation and nitration in its amyloidogenic capability"</i></p>

<b>18.15</b>	<b>Coffee break</b>
	<b>18.30 Elizabeth Jares-Erijman</b> , Facultad de Ciencias Exactas y Naturales, Universidad de Buenos Aires, Argentina <i>"Nobelfluorescent probes for protonic transfer and atomic force microscopy reveal alpha-synuclein aggregation early intermediaries"</i>
	<b>19.15 Claudio O. Fernández</b> , Instituto de Biología Molecular y Celular de Rosario, Argentina <i>"Alpha-synuclein structure, aggregation, and toxicity: rational designing of neurodegenerative process inhibitors"</i>
<b>20.15   21.15</b>	<b>SAN Plenary talk</b> <b>Rodrigo Quiñan Quiroga</b> , Department of Engineering, University of Leicester, Leicester, UK. <i>"The Jennifer Aniston neuron"</i>
<b>21.30</b>	Dinner
<b>23.00</b>	TAN business meeting
<b>24.00</b>	Neuroparty

## SUNDAY, OCTOBER 10<sup>TH</sup> ←

**9.00 | 12.30**

Symposium IV: *"Schizophrenia: From Cortical Development to Imaging of the Emotional and Social Brain"*.

Chair: Salvador Guinjoan (FLENI y Universidad de Buenos Aires)

**9.15 Kuei-Yang Tseng**, Department of Cellular and Molecular Pharmacology, Rosalind Franklin University of Medicine and Science, USA

*"Emerging properties of prefrontal cortex during normal and pathological adolescence"*

**10.00 Karl-Juergen Bär**, Department of Psychiatry,

University Hospital, Jena, Germany

*"The influence of respiration on heart rate variability in schizophrenia"*

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**10.45**

**Coffee break**

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**11.00 Mirta F. Villarreal**, Departamento de Neurología, FLENI y Facultad de Ciencias Exactas y Naturales, Universidad de Buenos Aires, Argentina

*"Functional MRI in schizophrenia: Imaging the brain or imagining the activity?"*

**11.45 Salvador M. Guinjoan**, Departamentos de Neurología y Psiquiatría, FLENI y Universidad de Buenos Aires, Argentina

*"Searching for Schizophrenia Endophenotypes: Autonomic Activity and Functional Brain Imaging during Tasks of Social Cognition in Patients and their Relatives"*

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**12.30 I 15.00**

Lunch and Poster Session III

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**15.15 I 16.15**

De Robertis Plenary talk

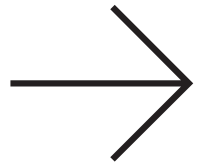
**Omar Macadar**. Departamento de Neurofisiología, Instituto de Investigaciones Biológicas "Clemente Estable". Montevideo, Uruguay.

*"Navigare Necesse (and Communication as well) in the Electric Fish dark habitat"*

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# COURSE ABSTRACTS







Wednesday,  
October 6<sup>th</sup>, 10:30.  
CLASS I, II, III

**Pascal Martin**

Laboratoire PCC (UMR168), Institut Curie recherche, Paris, France.

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[www.curie.fr/recherche/themes/detail\\_equipe.cfm/lang/\\_gb/id\\_equipe/323.htm](http://www.curie.fr/recherche/themes/detail_equipe.cfm/lang/_gb/id_equipe/323.htm)

»» Class I, II, III:

Active mechano-sensation by hair cells at the periphery of the auditory system

The ear functions as a sort of microphone that responds to sound-evoked mechanical vibrations by producing electrical signals that propagate along nervous pathways to the brain. The technical specifications of our hearing are remarkable. We can hear sounds that evoke mechanical vibrations with magnitudes comparable to those produced by thermal noise. Hearing is so sharply tuned to specific frequencies that a trained musician can distinguish tones differing in frequency by only 0.3%. Finally, from the faintest sounds that we can detect to sounds so intense that they hurt, the sound-pressure level increases by one millionfold, which corresponds to a trillionfold range in stimulus power. These striking characteristics of our hearing emerge because the ear is not a passive sensory receptor, but mobilizes internal resources of energy to augment audition in three ways. First, amplification renders hearing several hundred times as sensitive as would be expected for a passive system. The active process next exhibits tuning that sharpens our frequency discrimination. Finally, a compressive nonlinearity ensures that inputs spanning an enormous range of sound-pressure levels are systematically encoded by a modest range of mechanical vibrations and in turn of receptor potentials and nerve-fiber firing rates. The active process additionally exhibits the striking epiphenomenon of spontaneous otoacoustic emission, the production of sound by an ear in the absence of external stimulation. In these two lectures, I will describe experimental approaches at different scales: from psychophysics, to various types of neurophysiological recordings at the cellular level, down to the description of key molecular constituents. From these observations, I will unveil general physical principles that shed light on the active mechano-transduction process that shapes our sensation of sound at the ear's periphery.

»» General outline of lectures:

- 1- The psychophysical properties of hearing: threshold, frequency selectivity, dynamical range, distortion products.
- 2- Mechanical vibrations in the ear: middle ear mechanics, the cochlea as an

“acoustic prism” (frequency analysis and travelling waves), the cochlear amplifier.

**3-** Mechano-electrical transduction by the inner ear’s receptor cells, the hair cells (Part 1): the hair bundle as a mechanosensory antenna, the “gating-spring model” of mechano-electrical transduction, adaptation to saturating stimuli.

**4-** Mechano-electrical transduction by the inner ear’s receptor cells, the hair cells (Part 2): nonlinear hair-bundle mechanics (negative stiffness), active hair-bundle motility (spontaneous oscillations), the hair-bundle amplifier.

**5-** Somatic electromotility by mammalian outer hair cells

**6-** The “critical” cochlea: active oscillators as amplifying elements in hearing.

Wednesday,  
October 6<sup>th</sup>, 16:00.  
CLASS IV, V & VI

**Laurent Bourdieu**

Institut de Biologie de l'Ecole Normale Supérieure, IBENS, UMR ENS-CNRS-INSERM 8197, Ecole Normale Supérieure, Paris, France.

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»» CLASS IV:

Sensory coding in the whisker to barrel pathway

The rodent whisker system is a fascinating model for studying the neurobiology of tactile sensation at scales ranging from molecules to neuron networks and behaviour. In the last years, several studies have revealed how tactile features (“where” and “what”) are detected by rodents and encoded in their primary somatosensory cortex S1. I will more specifically describe experiments which address the detection of surface textures. Rats explore actively surfaces with the extremity of their whiskers, which exhibit a complex pattern of micro-displacements. The mechano-sensory signal at the whisker follicle results then from a further mechanical pre-filtering by the whisker rod, and is finally encoded in S1. The extraction of texture features by the whisker to barrel pathway combines therefore a physical problem of friction at the whisker to surface contact, a mechanical pre-filtering by the whisker and a neuronal encoding. I will present observations and alternative models describing this sensory integration and try to make an analogy with the sense of touch with fingers by primates. Finally, I will show that experiments on

anaesthetized animals using recently designed multi-whisker stimulators might provide complementary information to those obtained on awake behaving rats.

## »» CLASS V:

Optical developments in neuron network imaging

Two-photon fluorescence microscopy (TPFM) is a powerful tool for imaging deep inside living tissues with sub-cellular resolution. In this course, I will review the principle of TPFM and some of its major applications in neuroscience. I will then discuss some optical challenges in TPFM specifically raised by neuroscience. The first is its temporal resolution which is currently strongly limited to several tens of ms by the galvanometric mirrors which scan the laser beam. I will show that acousto-optic deflectors, which are non-mechanical devices, can bridge the gap between electrophysiology and optics. The second issue is the depth of imaging which is limited to a few hundreds of  $\mu\text{m}$  in the current setups. Attempts to increase this penetration depth have mainly try to combine TPFM and adaptive optics, a technique issued from astronomy. I will describe the different approaches undertaken and some preliminary results. I will finally show that, in our group, a side product of this research was the discovery of new method to image without any labelling myelinated axons in vivo. Finally, I will briefly show that TPFM is now used in several groups for recordings in awake behaving rodents.

## »» CLASS VI:

Late maturation of the direction selectivity map in the rat barrel cortex

In the neocortex, neuronal selectivities for multiple sensorimotor modalities are often distributed in topical maps thought to emerge during early postnatal development. Rodent somato-sensory cortex contains a somatotopic map for whisker identity, the well-known “barrel” map, but the existence of an intra-barrel map for whisker movement direction has proved controversial. We addressed this issue using in vivo two photon imaging of anaesthetized rats during single multi-directional stimulations. We confirmed the absence of direction map in juveniles, but discovered that one emerges in the adult long after all known critical periods in the somatosensory system. This map is remarkably specific, taking a pinwheel form centered near the barrel center and aligned to the barrel cortex somatotopy. We demonstrate by numerical simulation that the combination of spike-timing-dependent plasticity at synapses between layer 4 and layer 2/3 and realistic pad stimulation is sufficient to produce such a map. Its late emergence suggests that experience-dependent map formation and refinement continue in adulthood through intra-cortical mechanisms.

Thursday,  
October 7<sup>th</sup>, 10:00.  
CLASS VII, VIII, IX

**Dario Ringach**

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»» CLASS VII:

Receptive fields and maps in early visual cortex: Basic concepts

I will review some of the basic concepts of receptive fields in early visual cortex, including spatio temporal linearity, thresholding and gain control. We will discuss the notion of spatio-temporal inseparability and its role in direction selectivity. I will review the basic cell classes (simple and complex cells) and various reverse correlation techniques that have been used to “map” their properties along with associated models. We will conclude with a brief summary of the basic organization of neuronal properties in cortical maps, such as orientation and ocular dominance and methods for their measurement.

»» CLASS VIII:

Receptive fields and maps in early visual cortex: Open questions

I will describe some open questions in visual cortex. What is the role of cortical maps in visual processing? How specific are neuronal connections in the cortex? What is the relationship between neuronal structure (morphology) and function (visual properties)? How is the visual cortex wired during development? To what extent can visual cortex learn and/or reorganize in the adult? How do V1 population code visual information? What is the role of ongoing/spontaneous activity in cortical processing? How do eye movements affect the early representation of visual information? Why are there so many different visual areas? How do they all fit together?

»» CLASS IX:

Receptive fields and maps in early visual cortex: A statistical wiring model of primary visual cortex

Many cortical areas are organized into maps. These maps are so prevalent (and pretty!) that they have been assumed to form the basis for many computations in the brain. However, we do not really understand how these maps originate, how

they develop early in life and what function (if any) they play in cortical computation. I will discuss an ongoing debate of some of these issues with regards to functional maps in early visual cortex and offer a statistical view of cortical wiring that appears to account for a wide range of experimental data.

Thursday,  
October 7<sup>th</sup>, 15:30.  
CLASS X & XI

**Rodrigo Quian Quiroga**

Department of Engineering, University of Leicester, United Kingdom

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»» CLASS X:

Processing of extracellular recordings: spike sorting

Many questions in neuroscience depend on the analysis of neuronal spiking activity recorded under various behavioral conditions. For this reason the data acquired simultaneously from multiple neurons provide invaluable information to elucidate principles of neural information processing. The first step for processing these data is “spike sorting”: the grouping of spikes into clusters based on their shapes (given that each neuron tends to fire spikes of a particular shape, the resulting clusters correspond to the activity of different putative neurons).

In this lecture I will present “Wave\_clus”, a spike sorting method we have been developing in my lab. The method has 3 main steps: i) spikes are detected using an automatic amplitude threshold, ii) relevant features of the spike shapes are extracted using the wavelet transform, iii) the features are clustered using superparamagnetic clustering, a powerful method from statistical mechanics. The method is unsupervised and fast and I will show advantages in each of these steps compared to standard approaches. Finally, I will show its importance for the analysis of real single cell recordings from the human medial temporal lobe.

*References:*

*Spike Sorting.* Quian Quiroga R. *Scholarpedia* 2 (12): 3583. 2007.

*Unsupervised spike sorting with wavelets and superparamagnetic clustering.* Quian Quiroga R, Nadasdy Z and Ben-Shaul Y. *Neural Computation*, 16: 1661-1687; 2004.

For codes, tutorials and sample data see: <http://www2.le.ac.uk/departments/engineering/research/bioengineering/neuroengineering-lab/spike-sorting>

## »» CLASS XI:

Extracting information from neural populations: Basic principles and clinical applications

To a large extent, progress in neuroscience has been driven by the study of single cell responses averaged over several repetitions of stimuli or behaviors. However, the brain typically makes decisions based on single events by evaluating the activity of large neural populations. Therefore, to further understand how the brain processes information, it is important to shift from a single-neuron, multiple trial framework to multiple neuron, single-trial methodologies. Two related approaches – decoding and information theory – can be used to extract single-trial information from the activity of neural populations.

In this talk I will describe the principles of decoding algorithms and will show, with examples from single cell recordings in monkeys and humans, how the population analysis allowed by them give more information than traditional single-cell studies. I will also describe potential clinical applications, such as Brain-machine-interfaces, Neuroprosthetics and the possibility of new communication tools for 'locked-in' patients.

### *References:*

*Extracting information from neural populations: Information theory and decoding approaches. Quian Quiroga R and Panzeri S. Nature Reviews Neuroscience. 10: 173-185; 2009*

*Movement intention is better predicted than attention in the posterior parietal cortex. Quian Quiroga R, Snyder L, Batista A, Cui H and Andersen, R. Journal of Neuroscience 26: 3615-3620; 2006*

*Decoding visual inputs from multiple neurons in the human temporal lobe. Quian Quiroga R, Reddy L, Koch C and Fried I. Journal of Neurophysiology 98: 1997-2007; 2007.*

Thursday,  
October 7<sup>th</sup>, 19:00.  
CLASS XII

**Matias J. Ison**

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»» CLASS XII:

A possible mechanism for generating sparse responses in the human medial temporal lobe

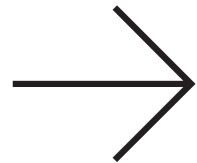
Neurons in the medial temporal lobe (MTL) have recently been shown to respond selectively to pictures of given individuals, objects and places. What are the underlying neural mechanisms leading to such degree of stimulus selectivity? In the first part of the lecture I will discuss a general machine learning approach to classify putative pyramidal cells and interneurons from a large dataset of extra-cellular recordings. Then, I will show that pyramidal cells recorded in vivo from the human medial temporal lobe are more selective than interneurons. I will also present evidence showing that hippocampal pyramidal cells exhibit the highest degree of selectivity within the MTL, reflecting the hierarchical processing of visual information.

Finally, I will discuss how we can interpret these findings as a plausible mechanism for generating sparse responses.





# IIRC MEETING ABSTRACTS



Friday,  
October 8<sup>th</sup>,  
9:00 | 12:30

## Symposium I: “**Sensory Systems**”

### → **HOW THE EAR TALKS TO THE BRAIN**

#### **Paul Albert Fuchs**

Otolaryngology-Head and Neck Surgery, and the Center for Sensory Biology,  
Johns Hopkins University School of Medicine, Baltimore Maryland USA

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Advances in molecular, cellular and genetic methods have enhanced our knowledge of cochlear physiology in the past two decades. After a general introduction to cochlear function, this paper will summarize recent findings on hair cell synaptic signaling obtained by intracellular ('patch-clamp') recording from afferent dendrites in the micro-dissected rodent organ of Corti. This includes recordings from **type I afferents** that transmit all acoustic information as we know it. Each type I neuron forms a single contact postsynaptic to a single 'ribbon' (active zone) on a single inner hair cell, and so all information on timing, intensity and frequency composition of sound must be transmitted through that solitary, limited connection. We now have confirmed that this is a rapid glutamatergic synapse employing AMPA-type receptors. An unexpected observation was that individual ribbons simultaneously release multiple vesicles to produce large, suprathreshold synaptic events in the afferent fiber. Intra-terminal recordings from **type II afferents** beneath outer hair cells show that these too are activated by glutamate binding to AMPA-type receptors. Additionally, type II afferents are highly sensitive to ATP, at least in the neonatal cochlea. Despite the fact that individual type II afferents contact dozens of outer hair cells, total synaptic transfer is an order of magnitude less than that from the single ribbon to a type I afferent, for equivalent patterns of excitation. These observations support the hypothesis that type II afferents are designed to respond only to very loud, perhaps traumatic levels of sound.

Glowatzki E and PA Fuchs (2002) Transmitter release at the hair cell ribbon synapse. *Nature Neuroscience* 5(2):147-154.

Weisz C, E Glowatzki and PA Fuchs (2009). The postsynaptic function of type II cochlear afferents. *Nature* 461:1027-1162.

→ ***Stem Cells And Smell Cells:  
Developmental Pathways And Cellular  
Specializations Underlying Olfaction***

**Randall Reed**

Center for Sensory Biology, Department of Molecular Biology and Genetics, Johns Hopkins University School of Medicine, Baltimore MD  
*E-mail: rreed@jhmi.edu*

Mammalian sensory systems are faced with two major challenges – they must detect information about the outside world with exquisite sensitivity and specificity while at the same time protecting themselves from the hostile environment that provides the sensory stimulation. The olfactory system achieves high sensitivity in part by developing highly specialized cilia and mechanisms for localizing transduction components to these hair like structures. We have identified genetic diseases associated with defects in these structures and model systems to study the underlying mechanisms. Additionally, olfactory neurons are directly exposed to the toxins, pathogens and environmental hazards that we breathe every day. The olfactory system has the capacity to regenerate new neurons throughout adult life from at least two distinct populations of progenitors. This regeneration is highly regulated during normal life and in disease states including acute trauma and inflammation. We are currently elucidating the origins and regulation of tissue-specific stem cells responsible for initial establishment and continually regeneration of olfactory neurons throughout adult life and repopulation of the tissue after environmental assault.

→ ***Cellular and Molecular Mechanisms of Itch Sensation***

Qin Liu<sup>1</sup>, Zongxiang Tang<sup>1</sup>, Lenka Surdenikova<sup>2,4</sup>, Seungil Kim<sup>5</sup>, Kush N. Patel<sup>1</sup>, Andrew Kim<sup>1</sup>, Fei Ru<sup>2</sup>, Yun Guan<sup>3</sup>, Hao-Jui Weng<sup>1</sup>, Yixun Geng<sup>1</sup>, Bradley J. Undem<sup>2</sup>, Marian Kollarik<sup>2</sup>, Zhou-Feng Chen<sup>5</sup>, David J. Anderson<sup>6,7</sup>, and Xinzhong Dong<sup>1,7 \*</sup>

<sup>1</sup>The Solomon H. Snyder Department of Neuroscience; <sup>2</sup>Department of Medicine; <sup>3</sup>Department of Anesthesiology & Critical Care Medicine; Johns Hopkins University, School of Medicine, Baltimore, MD; <sup>4</sup>Department of Pathophysiology, Jessenius Medical School, Martin, Slovakia; <sup>5</sup>Departments of Anesthesiology, Psychiatry, and Developmental Biology, Washington University School of Medicine Pain Center, St. Louis, MO; <sup>6</sup>Division of Biology, California Institute of Technology, Pasadena, CA; <sup>7</sup>Howard Hughes Medical Institute

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The cellular and molecular mechanisms mediating histamine-independent itch are largely unknown, despite its clinical importance. Itch induced by chloroquine

(CQ) is a common side effect of this widely used anti-malarial drug, limiting compliance and efficacy. Recently we identified the receptors for CQ-induced itch. These receptors, which include MrgprA3 in mouse and MrgprX1 in human, are members of an orphan G protein-coupled receptor family expressed exclusively in peripheral sensory neurons. Loss- and gain-of-function studies demonstrate that MrgprA3 is required for CQ responsiveness in mice. Furthermore, the expression of MrgprA3 defines a highly restricted subset of primary sensory neurons that co-express Gastrin-Releasing Peptide, a peptide whose receptor, GRPR, marks a subset of itch-specific second-order neurons in the dorsal spinal cord. Mrgprs may not only constitute novel targets for itch therapeutics, but may also provide molecular access to itch-selective primary sensory neurons.

→ ***Synaptic Inputs Generating Receptive Field Properties In Mammalian Retinal Ganglion Cells***

**Dario Protti**

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Retinal ganglion cells (RGCs) face the complex task of encoding the broad range of light intensities and contrast levels present in the visual world by using a limited spike rate. The output of RGCs depends on the balance of excitatory and inhibitory signals they receive from presynaptic circuits. Although inhibitory synaptic mechanisms are thought to contribute to adaptation, the relative contribution of synaptic interactions in the outer and inner plexiform layers is poorly understood. In addition, inhibition in the inner retina is often thought to shape the temporal properties of RGCs but not their spatial tuning. In this project we investigated the relative impact of excitatory and inhibitory synaptic inputs onto RGCs in relation to their spatial organisation in the retina of a New World primate (marmoset). Light evoked currents were measured in response to light stimulation by doing patch clamp recordings. Light increments elicited increases in excitation and inhibition in all ON-RGCs; excitation was size tuned in all RGCs whilst inhibition was spatially tuned in only a few cells. Light decrements caused size tuned excitation in most OFF-RGCs but rarely increase in direct inhibition. In a small proportion of OFF cells light decrements reduced a large tonic inhibitory current. Most ON and OFF cells displayed strong inhibition for anti-preferred contrast steps (decrements and increments respectively). In all RGCs tested, inhibitory inputs were reduced by a GABAA receptor antagonist. TTX, which blocks the activity of spiking amacrine cells, modified the spatial tuning of excitation and inhibition. We conclude that while the excitatory inputs to ON and OFF RGCs are similarly tuned, inhibitory inputs are asymmetric and that spatial tuning of both ON and OFF RGCs is at least partly shaped by inner retinal inhibition.

Friday,  
October 8<sup>th</sup>,  
19:00 | 21:30

## Symposium II: ***“Stress system: Old diseases, new interactions”***

### → ***Brain corticosteroid balance and adaptation: a hypothesis revisited***

#### **Ron de Kloet**

Department of Medical Pharmacology, University of Leiden, The Netherlands

E-mail: [kloet\\_e@chem.leidenuniv.nl](mailto:kloet_e@chem.leidenuniv.nl), [e.kloet@lacdr.leidenuniv.nl](mailto:e.kloet@lacdr.leidenuniv.nl)

In response to stressors, the brain activates neuropeptides eventually leading to the secretion of adrenal glucocorticoid hormones, which feed back to coordinate information processing aimed to promote adaptation and recovery. To exert this action the hormone binds to two types of receptors i.e. mineralocorticoid and glucocorticoid receptors (MR & GR) that operate in fast membrane and slower transcriptional events. The property of this complementary receptor system explains how one single glucocorticoid hormone can initially promote stress reactions, which are then slowly suppressed to prevent them from overshooting. By targeting multiple signal cascades and genes, the two receptor types function in a binary fashion, serving as a master switch in the control of neuronal network responses that underlie emotion, cognition and behavioural adaptation. Imbalance in this binary control mechanism can introduce a bias towards psychic dysfunction. This MR:GR balance hypothesis is examined here in the light of the ultradian rhythm of glucocorticoid secretion, the recently discovered MR and GR gene variants, glucocorticoid responsive pathways in brain and the efficacy of drugs targeting the stress system itself. de Kloet ER, Joëls M, Holsboer F (2005) *Nature Rev Neurosci.* 6: 463-475.

### → ***Impact of adrenocorticosteroids on hippocampal neuropathology and endocrine function in models of aging, hypertension and neurodegeneration***

**Alejandro F. De Nicola** <sup>1,2</sup>, Luciana Pietranera <sup>1,2</sup>, Flavia Saravia <sup>1,2</sup>, Maria Meyer <sup>1</sup>, Gisela Gargiullo <sup>1</sup> & Maria Claudia Gonzalez Deniselle <sup>1,2</sup>

<sup>1</sup> Instituto de Biología y Medicina Experimental-CONICET and <sup>2</sup> Department of Human Biochemistry, Faculty of Medicine, University of Buenos Aires.

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Adrenocorticosteroids exert pleiotropic effects on the brain, ranging from protective to neurotoxic. In animal models of aging, essential hypertension and motoneuron degeneration, changes of brain glucocorticoid (GR) and/or mineralocorticoid receptors (MR), coincide with hippocampal neuropathology and dysfunction of the hypothalamic-pituitary-adrenal axis (HPA). For instance, aging rats show a hypersensitive HPA axis and reduced glucocorticoid negative feedback, coexisting with down-regulation of hippocampal GR. Spontaneously hypertensive rats show a pronounced encephalopathy, which includes deficit of hippocampal neurogenesis, increased astrogliosis and reduced neuronal density in the hilus of the dentate gyrus. These changes are accompanied by hyperexpression of hippocampal and hypothalamic MR, and hyperresponse of the hypothalamic vasopressinergic system to mineralocorticoid administration. The motoneuron degeneration Wobbler mouse is a genetic mutant and model of amyotrophic lateral sclerosis, which presents a deficient hippocampal neurogenesis and increased astrogliosis; accompanied by increased levels of corticosterone and hyperresponse to stress. Scatchard analysis of hippocampal GR in Wobblers demonstrated decreased B<sub>max</sub> with normal K<sub>d</sub>, indicating GR down regulation. Literature reports have shown in humans suffering from amyotrophic lateral sclerosis a dysregulation of adrenal function, coupled to degeneration of hippocampal pyramidal cells and TDP43 inclusions in the dentate gyrus. Besides, it is already known that Alzheimer's patients showing decreased hippocampal volume present changes of cortisol secretion/negative feedback. Therefore, demonstrations in models of aging, neurodegeneration and hypertensive encephalopathy and humans with degenerative diseases, support that a pathological environment may increase the susceptibility of the hippocampus to adrenocorticosteroid damage, affecting among other parameters, neuroendocrine functions involving this brain structure.

→ ***Drug addiction, corticotrophin releasing hormone and the ventral tegmental area***

**Katia Gysling**

Millennium Science Nucleus in Stress and Addiction; Department of Cell and Molecular Biology, Faculty of Biological Sciences, Pontificia Universidad Católica de Chile.

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Epidemiological studies in human as well as studies in animal models of drug abuse have shown strong interaction between chronic or persistent stress and drug abuse. In addition, acute stressful stimuli induce relapse to drug seeking in abstinent individuals. Available evidence suggests that the ventral tegmental area (VTA) is a key nucleus for this interaction and that corticotrophin releasing hormone (CRH) is involved in the plastic changes induced by drug of abuse in

VTA glutamatergic synapses, at both pre and postsynaptic sites. CRH effects are mediated by the activation of CRH-R1 and CRH-R2 receptors at the postsynaptic site. Instead, it has been suggested that CRH acts mainly through CRH-R2 receptors at the presynaptic site. Acute exposure to most drug of abuse induces a CRH/CRH-R1-dependent strengthening of VTA glutamatergic synapses. The group of Roy Wise (Wang et al, 2005, 2007) has shown that exposure to an stressful stimulus increases CRH extracellular levels in the VTA of both control and cocaine-experienced rats. However, CRH increases glutamate and dopamine (DA) extracellular levels only in the VTA of cocaine-experienced rats by activating CRH-R2 receptors. Furthermore, they have shown that this increase sensitivity of VTA glutamate to CRH is responsible of stress-induced relapse to cocaine seeking. New anatomical and functional evidence showing that D1-DA and CRH-R2 receptors co-existing in VTA glutamatergic nerve terminals of subcortical origin may underlie the increased sensitivity to CRH in the VTA of cocaine-treated rats will be discussed.

Supported by FONDECYT 1070340 and MSI P06/008-F.

Saturday,  
October 9<sup>th</sup>, 15:00  
Ranwell Caputto  
Plenary talk

→ ***From fear to a traumatic memory: modulating factors involved***

**Víctor A. Molina**

Instituto de Farmacología Experimental (IFEC)-CONICET/ Universidad Nacional de Córdoba (UNC)

Fear memory induced by a threatening stimulus requires the emergence of a consolidation process, dependent on new protein synthesis necessary to stabilize transient synaptic changes following acquisition. It is well known that the basolateral complex of the amygdala (BLA) participates in fear memory formation. A traumatic experience prior to learning facilitates memory formation and the generation of LTP in BLA. The present findings support the notion that the reduced GABAergic neurotransmission in BLA induced by stress is a critical neurobiological event in such facilitating effect on fear memory. Similarly, we have shown that a similar stressful treatment prior to acquisition attenuated the vulnerability to midazolam's disruptive effect on fear memory reconsolidation. Moreover, D-cycloserine previous to memory reactivation restored the suscepti-

bility to this benzodiazepine ligand. In addition, a similar stressful situation prior to memory reactivation promoted fear memory. Modulating factors implicated in such interaction are discussed.

Saturday,  
October 9<sup>th</sup>,  
16:30 | 20:00

**Symposium III: “*Structural biology of neurodegenerative processes: alpha-synuclein and Parkinson disease*”**

→ ***Native alpha-synuclein and its mutants involved in the familial variants of Parkinson's disease***

**Soledad Celej**

Departamento de Química Biológica-CIQUIBIC, Facultad de Ciencias Químicas, Universidad Nacional de Córdoba. e-mail: [mcelej@mail.fcq.unc.edu.ar](mailto:mcelej@mail.fcq.unc.edu.ar)  
E-mail: [mcelej@fcq.unc.edu.ar](mailto:mcelej@fcq.unc.edu.ar)

Parkinson's disease (PD) is a movement disorder characterized by the presence in the mid-brain of intracellular proteinaceous inclusions, mainly formed by fibrillar alpha-synuclein (AS). AS is located presynaptically and functions in vesicle trafficking and release. Three missense point mutations (A53T, A30P and E46K) in the AS gene lead to early onset PD.

The amyloid fibrils have an unbranched rod-like morphology and are formed by interwound protofilaments. The core structure is a stack of  $\beta$ -sheets in which the strands are perpendicular to the main axis of the fibril. Of particular interest is the ability of the  $\beta$ -sheet network within this canonical cross- $\beta$  structure to adapt to point mutations. Fibrils formed by AS familial mutants exhibit subtle differences in ultrastructural features as compared to the WT protein. It will be shown that AS variants differ in their supramolecular fibrillar organization as sensed by a novel dual-emission fluorescence probe. These differences will be discussed in terms of distinctive polarity and hydration of the binding sites [Celej MS, Caarls W, Demchenko A, Jovin TM, Biochemistry (2009) 48, 7465–7472].

The sensitivity of this probe to structural alterations induced by point mutations is unprecedented and may contribute to understand various phenomena associated with amyloidosis: plasticity, polymorphism, and structure-toxicity relationship of prefibrillar intermediates.



→ ***Role of alpha-synuclein phosphorylation and nitration in its amyloidogenic capability***

**José M. Souza**

Departamento de Bioquímica, Facultad de Medicina, Universidad de la República, Montevideo, Uruguay

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Intracellular proteinaceous accumulations of alpha-synuclein (AS) characterize a number of neurodegenerative diseases called synucleinopathies. Lewy bodies of Parkinson disease represents an illustrate example of AS aggregates. AS is a presynaptic soluble protein having a random coil conformation. It forms antiparallel beta-sheet containing fibrils, presenting a similar morphology to other amyloid proteins. Posttranslational modifications of AS include oxidative-mediated modifications i.e. 3-nitrotyrosine formation as well as serine phosphorylation. 3-Nitrotyrosine modified-AS is due to the simultaneous formation of nitric oxide and superoxide, secondarily to inflammation or toxic related-drugs (i.e. rotenone). Casein kinases and G protein-coupled receptor kinases are capable of phosphorylate AS at serine-129. All these protein modifications have been found in AS intracellular accumulations observed in synucleinopathies. Tyrosine mononitrated-AS promotes fibril formation of unmodified AS; however, phosphorylation at serine-129 inhibits fibrillation. What is the role of these AS posttranslational modifications in synucleinopathies? Hypothetically, small amounts of AS may be nitrated to form 3-nitrotyrosine-modified AS which seed the fibrillation of unmodified AS. Lately, kinases colocalize within these fibrils and phosphorylate serine-129 which remains exposed in the fibrils.

Supported by FCE-ANII 491.

→ ***Nobel fluorescent probes for protonic transfer and atomic force microscopy reveal alpha-synuclein aggregation early intermediaries***

Jonathan Fauerbach<sup>1</sup>, Dmytro Yuschenko<sup>2</sup>, Reinhard Klement<sup>2</sup>, Thomas Jovin<sup>2</sup> and **Elizabeth Jares-Erijman<sup>1</sup>**

<sup>1</sup> Facultad de Ciencias Exactas y Naturales, Universidad de Buenos Aires Buenos Aires, Argentina; <sup>2</sup> Max Planck Institute for Biophysical Chemistry, Goettingen, Germany

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Parkinson's Disease (PD), the second most frequent neurodegenerative syndrome affecting >1% of the population above 60 years in age, is characterized by debilitating, progressive and irreversible neuromotor degeneration and impaired cognitive functions, secondary to the loss of dopaminergic neurons of the substantia nigra and other nuclei of the midbrain. PD remains incurable primarily because its

etiology is obscure, although intermediates (“oligomers”) on the aggregation pathway of the 140 aa presynaptic protein  $\alpha$ -synuclein (AS) are currently considered the most likely causes of cellular toxicity, dysfunction and death. Organic compounds such as thioflavin T are generally used to demonstrate terminal amyloid fibrils in cuvette and microplate assays or in cytochemical staining, but they are incapable of generating signals with prefibrillar structures. We have been interested in developing new technologies for the study of the elusive initial stages of AS aggregation. We have demonstrated that pyrene [1] and aminonaphthalenes [2] reveal changes in rotational dynamics (size) and microenvironment from the onset of aggregation, and that a biarsenical-tetracysteine expression probe [3,4] fused to AS (AS-TC; [5-7]) together with functionalized quantum dots serve as ultrasensitive sensors and initiators of AS aggregation in vitro and in cells [6]. In addition, we have visualized dynamic aggregated states of AS by STED and PALM superresolution microscopy [8].

Our more recent studies of AS aggregation have been based on an ESIPT probe family [9]. The first experiments of AS aggregation with such probes utilized an extrinsic dye (FE), which was able to distinguish between wild type and familial mutant fibrillar forms of AS [10]. We then switched to covalent adducts of two ESIPT probes (FE, FC), inserted at defined sequence positions (18, 90, 140) of functionally neutral A C substitutions in AS. Aggregation reactions were carried out with mixtures of unlabeled protein (wt) and a low reporter concentration of labeled protein. The latter demonstrated a variety of pronounced changes, during the early stages of AS aggregation, i.e before the appearance of an appreciable ThioT response [11]. In order to establish the structural identity of the early species, we sampled the reactions at various times by AFM, guided by the temporal course of the ESIPT T\*/N\* ratio. This evaluation led to the perception of a family of supramolecular structures arising and disappearing in a defined well-orchestrated sequence [12]. We propose that these intermediates and their “offspring” may promote interactions with other proteins and structures and thus constitute the toxic species leading to neuronal dysfunction and loss.

#### References

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→ ***Alpha-synuclein structure, aggregation, and toxicity: rational designing of neurodegenerative process inhibitors***

**Claudio O. Fernández**

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The misfolding of proteins into a toxic conformation is proposed to be at the molecular foundation of a number of neurodegenerative disorders including Alzheimer and Parkinson's disease. One common and defining feature of protein misfolding diseases is the formation and deposition of amyloid-like fibrils. The aggregation of the protein alpha-synuclein (AS) is a critical step in the etiology of Parkinson's disease (PD) and other neurodegenerative synucleinopathies. The study of the structural and toxic mechanisms related to AS amyloid formation is critical to advance in the design of a therapeutic strategy. The identification of aggregation inhibitors and the investigation of their mechanism of action are fundamental in the quest to mitigate the pathological consequences of amyloid formation. By the combined application of a battery of biophysical techniques we have addressed structural and molecular unresolved details related to the mechanistic rules that direct the anti-amyloid effect of small molecules on AS amyloid fibril formation.

Lamberto et al. (2009) *Proc Natl Acad Sci U S A* 106: 21057-21062

Acknowledgments: ANPCyT, Fundación Antorchas, CONICET, Max Planck Society, Alexander von Humboldt Foundation.

Saturday,  
October 9<sup>th</sup>, 20:15  
SAN Plenary talk

→ ***The Jennifer Aniston neuron***

**Rodrigo Quián Quiroga**

Department of Engineering, University of Leicester, United Kingdom

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We can easily recognize a person or an object in a fraction of a second even when seen under strikingly different conditions. How neurons are capable of creating such an invariant representation has been a hot topic of debate in Neuroscience. In epileptic patients candidates to surgery we analyzed the responses of neurons

in the human medial temporal lobe to picture presentations. Several technical improvements led to the finding of 'abstract' neurons that fired selectively to different pictures of familiar individuals (e.g. Jennifer Aniston) and even to their written names. If time permits, I will also show that these responses follow conscious perception and that from the firing of these neurons it was possible to predict what the subjects were seeing far above chance. Finally, I will discuss the possible function of these neurons.

Sunday,  
October 10<sup>th</sup>,  
9.00 | 12.30

**Symposium IV: *"Schizophrenia: From Cortical Development to Imaging of the Emotional and Social Brain"***

→ ***Age matters when dopamine and endocannabinoid receptors meet in the prefrontal cortex***

**Kuei Y. Tseng**

Department of Cellular & Molecular Pharmacology, RFUMS / The Chicago Medical School, North Chicago, USA

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Dopamine (DA) modulation of prefrontal cortex (PFC) function is refined during the periadolescent transition to adulthood. Disruption of this DA modulation is thought to underlie the onset of PFC deficits in schizophrenia. Here we conducted whole-cell recordings in brain slices obtained from pre- (PD<40) and post- (PD>45) pubertal rats to study how synaptic plasticity in the PFC changes during the periadolescence transition. We found that depending on the stages of cortical maturation, different forms of LTP and LTD can be induced in the PFC by taking into account the temporal order of correlated pre and postsynaptic events, known as spike-timing dependent plasticity (STDP). We found that glutamatergic synapses onto deep-layer pyramidal neurons in the PFC exhibit a form of NMDA/Ca<sup>2+</sup>/PKA-mediated LTD that is more pronounced in the adult, compared to the adolescent, PFC. A shift of LTD to LTP was observed only in the adult PFC when CB1 receptors are blocked. Importantly, DA modulation of STDP-LTP and LTD takes place only in the adult PFC by virtue of an upregulation of D1 and D2-dependent signaling during adolescence. We conclude that STDP at PFC excitatory synapses is developmentally regulated and that DA-dependent regulation of PFC synaptic plasticity acquires its distinctive mature form during the periadolescent transition period as a result of changes in the relative dominance of pre (CB1) and posts-

ynaptic signaling mechanisms. Overall, these results provide important insights into the synaptic mechanisms thought to be involved in the pathophysiology of cortical deficits in schizophrenia, a psychiatric condition with onset of symptoms often occurring during adolescence.

### → *The influence of respiration on heart rate variability in schizophrenia*

#### **Karl-Juergen Bär**

Department of Psychiatry and Psychotherapy, University Hospital, Jena, Germany  
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Recent studies revealed cardiac autonomic dysfunction in patients with acute schizophrenia, which appears to be mainly related to reduced vagal and increased sympathetic modulation. To understand the significance of cardiac autonomic function in patients with schizophrenia, evidence will be presented that first-degree relatives show an attenuated, yet identical pattern in autonomic dysfunction as patients, with decreased vagal modulation of heart rate and decreased baroreflex sensitivity. Furthermore, altered amygdala activity in patients with schizophrenia can influence respiratory patterns and consequently cardiovascular parameters. Thus, the examination of respiration and heart rate time series complexity as well as coupling of both signals will be shown for patients and their relatives.

In conclusion, the pattern of autonomic dysfunction seen in patients and relatives might indicate underlying disease-inherent genetic vulnerability, especially since autonomic parameters are heritable. In addition, these findings may be of value to identify the high-risk group of patients' relatives in regards to serious cardiovascular events so that early preventive measures can be taken. More importantly, a strong line of evidence suggests that the altered breathing pattern determines autonomic dysfunction seen in patients and relatives. Thus, the presentation will cover possible interventional strategies to improve autonomic dysfunction by means of biofeedback in these patients.

### → *Functional MRI in schizophrenia: Imaging the brain or imagining the activity?*

#### **Mirta Villarreal**

Departamento de Neurología, FLENI y Facultad de Ciencias Exactas y Naturales, Universidad de Buenos Aires  
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In recent years there has been explosive growth in the number of neuroimaging studies performed using functional Magnetic Resonance Imaging (fMRI). The field that has grown around the acquisition and analysis of fMRI data is intrinsic-

lly interdisciplinary and involves contributions from researchers in neuroscience, psychology, physics and physicist, among others. Functional MRI is a noninvasive technique for studying brain activity. In opposition of neurons electrical activity recording, it not concerns with the behavior of single neurons and the direct measurement of electrical signals, fMRI provides information of blood flow changes that accompany neuronal activity of a large group of neurons. Its advantage is the relatively high spatial resolution, its disadvantage is a temporal resolution limited by the slower rate of brain hemodynamics.

I will introduce the general aspects of fMRI: how are the acquisition procedures, the design of the paradigms and the statistical considerations needed for obtaining and interpreting the results. I will also talk about the spatial and temporal resolution as well as its critical sensibility to the head movement during the acquisition. I expect with this presentation to give you the basic tools needed to understand futures fMRI studies.

→ ***Searching for Schizophrenia Endophenotypes: Autonomic Activity and Functional Brain Imaging during Tasks of Social Cognition in Patients and their Relatives***

**Salvador Guinjoan**

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Schizophrenia is a brain disorder with high heritability. Recent efforts to detect genes associated to the development of this disease have been less successful than previously expected, probably because of neurobiological heterogeneity and the presence of phenocopies. The definition of intermediate phenotypes can help guide efforts to characterize genetic abnormalities associated to the disorder, by defining phenotypes of neuropsychological, biomolecular, anatomical, and behavioral nature. This presentation describes our efforts to define traits associated with the disease that are present in close relatives more frequently than in comparable persons from the general population. We have studied the presence of abnormalities in the performance of a variety of social cognitive tasks, along with peripheral autonomic activity and brain activation (as measured by functional magnetic resonance imaging) evoked by such tasks. We observed that patients display protracted autonomic responses to both generic stress and social cognitive tasks which, in the case of their relatives, seem restricted to the parasympathetic division of the autonomic nervous system. Brain activation during emotional processing and theory of mind tasks in healthy persons is largely symmetrical, involving structures associated with language (left hemisphere) and its emotional qualities (e.g., prosody; right hemisphere). Patients with schizophrenia appear to display fairly selective activation of left structures, and their unaffected siblings share characteristics of both groups. This pattern is in accordance with previous

evidence linking schizophrenia to abnormal brain lateralization, specifically involving the right hemisphere and its functions.

Sunday,  
October 10<sup>th</sup>, 15:5  
De Robertis Plenary talk

→ ***Navigare Necesse (and Communication as well) in the Electric Fish dark habitat***

**Omar Macadar**

Departamento de Neurofisiología, Instituto de Investigaciones Biológicas "Clemente Estable". Montevideo, Uruguay  
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South American electric fish offer a pioneer model system in Neuroethology, with remarkable advantages to study relevant questions in Neuroscience such as neural bases of behavior, sensory-motor integration, circuit organization of motor control systems and neural plasticity. Over the last 30 years, we have conducted our research aiming to understand the role of neural circuits in the control of behavior using the two most abundant species detected in Uruguay: *Gymnotus omarorum* and *Brachyhypopomus gauderio*.

Gymnotiformes are nocturnal fish that inhabit shallow muddy waters. Their main sensory system is active electroreception that includes a sensory component and an electrogenic one. The latter generates species-specific weak (<1 V), regular (15-1000 Hz), brief (2-10 mS) electric pulses that provide the carrier energy for electrosensation and also act as behavioral displays for intraspecific communication. Electric fish are champions in the control of timing in both, motor and sensory processing. The electric discharge depends on the firing of more than 1000 cells located all along the fish body and yet it is extremely brief and robust in waveform, indicating the existence of powerful synchronization mechanisms acting at peripheral and spinal levels.

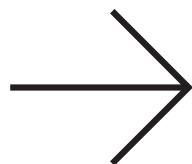
Discharge waveform and rate are under the influence of environmental and hormonal influences. Rapid increase of water temperature increase discharge rate and modifies waveform jeopardizing its communicative value. At the southern boundary of their distribution, Gymnotiforms show strong seasonality. Maintained high temperature induce breeding conditions both in gonads and the electric discharge waveform. Gonadal hormones, in turn, prevent the temperature-induced rapid changes in waveform, preserving the communicative value of the electric signal, essential for successful reproduction.

Acknowledgements need to be presented *in vivo*.





# YOUNG INVESTIGATORS ABSTRACTS





Friday, October 8<sup>th</sup>,  
14:45 | 16:15  
Young Investigators  
Colloquium I

### **Properties of the olivocochlear-outer hair cell synapse in the mouse cochlea**

**Ballester, Jimena A.**<sup>1</sup>; Zorrilla de San Martín, Javier<sup>1</sup>; Goutman, Juan<sup>1</sup>; Fuchs, Paul<sup>2</sup> A.; Elgoyhen, Ana Belén<sup>1</sup>; Katz, Eleonora<sup>1</sup>

<sup>1</sup> INGEBI- CONICET, Buenos Aires , Argentina

<sup>2</sup> Otolaryngology-Head and Neck Surgery, and the Center for Sensory Biology, Johns Hopkins University School of Medicine, Baltimore, Maryland, USA

In the Organ of Corti, the sensory epithelia of the mammalian auditory system, inner hair cells transduce sound stimuli while outer hair cells (OHCs) participate in the amplification of sound. OHC function is inhibited by efferent cholinergic olivocochlear (OC) fibers. OC fibers firing rate varies according to the type and intensity of sound. In turn, the degree of inhibition exerted by OC fibers on the auditory function changes with firing rates. In this work we study the properties of the OC synapse onto OHCs. Synaptic activity was recorded in voltage-clamped OHCs from the apical turn of the mouse cochlea (P10-12) during electrical stimulation of OC fibers. Activation of fibers by single shocks evoked inhibitory postsynaptic currents with a low rate of success. Paired-pulse protocols showed that this synapse facilitates with maximum efficacy at 10 ms pulse intervals. Trains of stimulation at different frequencies produced increasing levels of transmitter release proportional to the stimulus frequency due both to summation and facilitation of synaptic responses. These results show that this synapse facilitates at physiological OC fibers firing frequencies. This property could be relevant for encoding different degrees of OC inhibition in response to variable sound stimulation.

### **Analysis of early migratory pathways in whole embryos**

**Ceci, María Laura**, López-Mascaraque, Laura and de Carlos, Juan A.

Laboratory of Telencephalic Development, Instituto Cajal (CSIC), Madrid. Spain.

During early development (E10-E12), diverse cell populations migrate tangentially through the cortical preplate, from their site of origin toward their final destinations. To study their migratory pathways we injected fluorescent tracers in whole mice embryos and cultured them exo-utero during 24 hours in roller bottles. One of the most important populations for cortical development is the

Cajal-Retzius (C-R) cells. It has been proposed that this population could be originated in three different telencephalic areas (cortical hem –CH-, ventral pallium –VP- and septum –Sp-). Here, we show that C-R cells are mainly generated in CH and migrate along parallel routes to cover the complete neocortical mantle. If we implant pieces of CH in different brain locations of host embryos, the ectopically generated cells attain new migratory routes, indicating the important influence of the environment. Further, studies in Sey mutant mice are particularly informative since the lack of Pax6 seriously affects the migratory environment. As a result, in these animals the C-R cell population is disorganized as well as other cells that share their migratory pathway.

Spanish Ministerio de Educación y Ciencia (BFU2007-60351/BFI); OLFACTOSENSE Consortium (P-SEM-0255-2006); Fundación Castilla La Mancha (FISCAM-PI 2007/661).

### **Neurotransmitter systems in lymphocytes**

**De Rosa, María José**, Dionisio, L., Bouzat, C. and Esandi, M. C.

Instituto de Investigaciones Bioquímicas Bahía Blanca, Buenos Aires, Argentina

Neurotransmitters (NT) have been classically considered as neuron-secreted molecules. Yet, non-neuronal cells produce NT and express NT receptors. The physiological role of these extraneuronal systems is not clear.

We explored the presence of neuronal components of nicotinic cholinergic and GABAergic systems in lymphocytes and their functional significance.

We showed that lymphocytes express neuronal nicotinic  $\alpha 7$  and GABA subunits. Moreover, electrophysiological and fluorescence microscopy studies revealed that these subunits assemble into functional receptors. We also detected mRNAs of enzymes responsible of acetylcholine and GABA synthesis and proteins involved in GABA metabolism, storage and transport.

By studying the effect of nicotinic and GABAergic drugs in vital T-cell functions we demonstrated that nicotinic drugs decrease the number of cortisol-induced apoptotic cells. In addition,  $\alpha 7$  and GABA receptors negatively modulate T-cell proliferation.

Our results show that lymphocytes present similar NTs systems to those expressed in neurons. NTs regulate crucial T-cell functions acting in an autocrine/paracrine mode and may serve for communicating between brain and immune system. Pharmacological modulation of these systems may provide new approaches as new targets for inflammation control.

Saturday, October 9<sup>th</sup>,  
15:00 | 16:30  
Young Investigators  
Colloquium II

**Synaptic potentiation onto lateral habenula neurons in the learned helplessness model of depression**

**Piriz, Joaquin**

Instituto de Fisiología Biología Molecular y Neurociencias (IFIBYNE), UBA,  
CONICET

The cellular basis of depressive disorders is poorly understood<sup>1</sup>. Recent studies in monkeys indicate that neurons in the lateral habenula (LHb), a nucleus that mediates communication between forebrain and midbrain structures, can increase their activity when an animal fails to receive an expected positive reward or receives a stimulus that predicts aversive conditions (i.e. disappointment or anticipation of a negative outcome).

LHb neurons project to and modulate dopamine-rich regions such as the ventral-tegmental area (VTA)<sup>2</sup> that control reward-seeking behavior<sup>3</sup> and participate in depressive disorders<sup>4</sup>. In this study we show in the learned helplessness, a well established model of depression whereby animals show reduced escape from escapable foot shock<sup>5</sup>, that excitatory synapses onto LHb neurons projecting to the VTA are potentiated. Synaptic potentiation is due to an enhanced presynaptic release probability.

Depleting transmitter release by repeated electrical stimulation of LHb afferents, using a protocol that can be effective on depressed patients<sup>6</sup>, dramatically suppresses synaptic drive onto VTA-projecting LHb neurons in brain slices and significantly reduces learned helplessness behavior in rats. Our findings suggest an aberrant cellular process previously unexamined in the context of mood disorders that may be critical in the etiology of depression. Future studies aimed at determining the molecular signaling changes underlying the synaptic hyperactivity in the VTA-projecting LHb neurons may lead to novel and effective treatments potentially able to reverse some forms of depressive disorders.

<sup>1</sup> Krishnan, V. and Nestler, E. J., The molecular neurobiology of depression. *Nature* 455 (7215), 894 (2008).

<sup>2</sup> Christoph, G. R., Leonzio, R. J., and Wilcox, K. S., Stimulation of the lateral habenula inhibits dopamine-containing neurons in the substantia nigra and ventral tegmental area of the rat. *J Neurosci* 6 (3), 613 (1986); Ji, H. And Shepard, P. D., Lateral habenula stimulation

inhibits rat midbrain dopamine neurons through a GABA(A) receptor-mediated mechanism. *J Neurosci* 27 (26), 6923 (2007); Matsumoto, M. and Hikosaka, O., Lateral habenula as a source of negative reward signals in dopamine neurons. *Nature* 447 (7148), 1111 (2007).

<sup>3</sup> Martin-Soelch, C., Is depression associated with dysfunction of the central reward system? *Biochem Soc Trans* 37 (Pt 1), 313 (2009); Nestler, E. J. And Carlezon, W. A., Jr., The mesolimbic dopamine reward circuit in depression. *Biol Psychiatry* 59 (12), 1151 (2006); Schultz, W., Multiple dopamine functions at different time courses. *Annu Rev Neurosci* 30, 259 (2007).

<sup>4</sup> Krishnan, V. et al., Molecular adaptations underlying susceptibility and resistance to social defeat in brain reward regions. *Cell* 131 (2), 391 (2007).

<sup>5</sup> Seligman, M. E., Learned helplessness. *Annu Rev Med* 23, 407 (1972).

<sup>6</sup> Mayberg, H. S., Targeted electrode-based modulation of neural circuits for depression. *J Clin Invest* 119 (4), 717 (2009); Sartorius, A. et al., Remission of major depression under deep brain stimulation of the lateral habenula in a therapy-refractory patient. *Biol Psychiatry* 67 (2), e9.

\* This work was done during the speaker's postdoctoral training at the laboratory of Dr. Roberto Malinow at the University of California San Diego.

## **Role of TDP-43 in neurodegenerative disease**

### **Müller Igaz, Lionel**

Departament of Physiology, School of Medicine, University of Buenos Aires. Buenos Aires, Argentina.

TAR DNA-binding protein 43 (TDP-43) has been recently identified as the major disease protein in frontotemporal dementia (FTD) and amyotrophic lateral sclerosis (ALS). In these and other disorders (now termed "TDP-43 proteinopathies"), TDP-43 is redistributed from its normal nuclear localization to form cytoplasmic insoluble aggregates. Moreover, pathological TDP-43 is abnormally ubiquitinated, hyperphosphorylated and cleaved to generate C-terminal fragments (CTFs). TDP-43 aggregates are present as cytoplasmic, neuritic or nuclear inclusions and affected neurons show a dramatic depletion of normal nuclear TDP-43. To mimic this nuclear clearance, we overexpressed TDP-43 with a mutated nuclear localization signal in cultured cells that showed a reduction in endogenous nuclear TDP-43 and accumulations of insoluble cytoplasmic aggregates. We also show that insoluble TDP-43 CTFs are selectively enriched in affected cortical regions compared with spinal cord of both FTLD-U and ALS cases. Expression of TDP-43 CTFs is sufficient to generate cytoplasmic aggregates in cultured cells. Moreover, these insoluble CTFs are ubiquitinated and abnormally phosphorylated, and cells expressing TDP-43 CTFs display abnormal splicing, one of the few known physiological functions of TDP-43. In summary, these models will allow us to study the pathogenic mechanisms underlying TDP-43 proteinopathies, which in turn will be vital to develop new therapies for these disorders.

## **Epigenetic mechanisms in long term memory**

### **Federman, María Noel**

Laboratorio de Neurobiología de la Memoria, IFIBYNE-CONICET, FCEN-UBA

Long-term memory (LTM) consolidation requires mRNA and de novo protein synthesis. Transcriptional activation is controlled by transcription factors, their cofactors and repressors. Cofactors and repressors can regulate gene expression by interacting with basal transcription machinery, remodeling chromatin structure and chemically modifying histones. These last two processes are considered as epigenetic mechanisms.

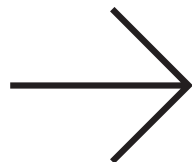
The acetylation is the most studied histone modification related to gene expression regulation. This process is regulated by lysine acetylases (KATs) and deacetylases (KDACs). We use histone acetylation as a model mechanism in order to study the role of epigenetic during LTM formation.

We present evidences supporting histone acetylation function during consolidation of context-signal memory in the crab *Chasmagnathus*, as well as during consolidation of novel object recognition memory in mice. Based on our results, we hypothesize that histone acetylation is a key mechanism in LTM consolidation, functioning as a molecular feature of stronger memories.





# POSTERS ABSTRACTS





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**P-7** - Oberholzer María Victoria - Muscarinic M1 modulation on basal neurotransmission in CA1 region of rat hippocampus

**P-10** - Zorrilla De San Martín Javier - Short term plasticity (STP) changes during development of the medial olivocochlear-inner hair cell synapse in the mouse cochlea.

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**P-5** - González Nicolás - M1 muscarinic receptor involvement in LTP induction in CA1 synapsis of rat hippocampus

**P-8** - Vattino Lucas - P/Q-type calcium channel  $\alpha 1$  subunit is necessary for ALS-IgGs spontaneous synaptic activity modulation on mouse neuromuscular junction

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**P-3** - Davies Sala M. Georgina - Electrical activity regulates neuronal maturation in the adult dentate gyrus

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**P-32** - Hernández Mariana V. - Characterization of ena/VASP domains with dominant negative function

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**P-38** - Luchelli Luciana - NPSAX: novel mRNA silencing foci that regulate local translation at the synapse

**P-41** - Philippe Valeria - Frizzled-7, a potential Wnt7b receptor, regulates dendrite morphogenesis.

**P-44** - Rolando Xavier Avilés-reyes - Evidences of ischemic tolerance induced by a model of sleep apnea by intermittent hypoxia

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- P-122** - Bacigalupo Masdeu Paola - Head shakes and hyperlocomotion induced by DOI, a serotonergic 5-HT<sub>2A</sub> agonist, as behavioral tools to study potential atypical antipsychotic actions.
- P-125** - Blake Mariano - Memory consolidation and reconsolidation of an inhibitory avoidance task in mice are impaired by acquisition / consolidation of a novel task
- P-128** - Calero Cecilia I - Histidine 141 is critical for GABA<sub>A</sub> receptor sensitivity to ascorbic acid.
- P-131** - Cid Mariana Paula - SYNAPSIN PHOSPHORYLATION AND GLUTAMATE RELEASE REGULATION BY THE GABA<sub>A</sub>ERGIC SYSTEM IN FRONTAL CORTEX SYNAPTOSOMES FROM RATS WITH EXPERIMENTAL AUTOIMMUNE ENCEPHALOMYELITIS.
- P-134** - Di Guilmi Mariano N. - Pregabalin modulation of calcium channels and neurotransmitter release.
- P-137** - Frick Luciana - Effects of systemic administration of fluoxetine and tianeptine on an operant conditioning task in rats
- P-140** - Hernando Guillermina - Activation and Desensitization of C. elegans Muscle Levamisole-Sensitive Nicotinic Receptors
- P-143** - Maur Damián Gustavo - Prenatal stress increases plasma corticosterone and hippocampal Nitric Oxide Synthase expression in rat's offspring.
- P-146** - Pascual Mariano M. - ChIP-RAPD as a possible new methodology to identify genes regulated by chromatin related proteins.
- P-149** - Reiff Rachel - The development of an agonist for the 9 10 nicotinic cholinergic receptor
- P-152** - Riberi Maria Ines - Increased GABA<sub>A</sub> receptors 1-subunit expression in chick fore-brain. Modulation by noradrenaline and acute stress.

### »» SESSION II

- P-120** - Araujo Nilza - DISSOCIATION OF ENVIRONMENTAL CONDITIONING TO HYPERACTIVITY AND DRUG EXPECTANCY IN RESPONSE TO STIMULI FORMERLY PAIRED WITH COCAINE IN MICE
- P-123** - Beltrán González Andrea - Modulation of GABAC receptors by reactive oxygen species
- P-126** - Boffi Juan Carlos - Ascorbic acid is a positive modulator of 9 10 nicotinic cholinergic receptors.
- P-129** - Campi Julieta - IN VIVO PHOTORELEASE OF GABA IN THE MOUSE CORTEX
- P-132** - Corradi Jeremías - A Novel Mechanism of Modulation of 5-HT<sub>3A</sub> Receptors by Hydrocortisone
- P-135** - Duarte Dalmolin Gerusa - The armed spider toxin Tx3-3 restores the analgesic effect of morphine in opioid- and neuropathic hyperalgesic state

- P-138** - Gori M Belén - NMDA RECEPTOR NR2A SUBUNIT EXPRESSION IN AN EXPERIMENTAL EPILEPSY MODEL. CYCLOPENTYLADENOSINE EFFECT
- P-141** - Krapacher Favio - Mice lacking p35 exhibit hyperactivity and paradoxical response to psychostimulants.
- P-144** - Mlewski Ec - A behavioral and biochemical comparison of 2 different sensitization protocols: chronic Vs two injection protocols (Tips).
- P-147** - Paz María Constanza - The amphetamine-induced neuroplastic changes involve the brain renin-angiotensin system (RAS)
- P-150** - Reiner Gabriela - ALLOSTERIC POSITIVE EFFECTS OF PHENOLIC COMPOUNDS ON GABAA RECEPTOR IN PRIMARY NEURONAL CULTURES.
- P-153** - Tolosa Ma. Fernanda - Variable magnetic fields modify function of Cys-loop receptors

### »» SESSION III

- P-121** - Bachor Tomás - Endothelins and cell proliferation in the subventricular zone
- P-124** - Bergé Ignacio - The Anthelmintic Agent Oxantel is a Partial Agonist of C. elegans Muscle Levamisole-Sensitive Nicotinic Receptor
- P-127** - Cabrera Ricardo - Caged Serotonin for Visible-Light Photodelivery
- P-130** - Cazulo Carolina - Neuroprotection by nicotinic agonism in a Parkinson's disease model: changes in the iron metabolism
- P-133** - Czerniczyniec Analía - Chronic exposure to paraquat induces cortical and striatal mitochondrial dysfunction.
- P-136** - Filevich Oscar - New Kids on the Block: Caged Glycine and Caged GABA.
- P-139** - Gutiérrez María Laura - Activity-dependent regulation of GABAA receptor function mediated by a phosphorylation mechanism
- P-142** - López-hill Ximena - Hyperlocomotion induced by MK-801, a N-methyl-D-aspartate (NMDA) receptor antagonist, imply a GABAergic disinhibition of the anterior thalamic nucleus.
- P-145** - Morris Winston - Glutamate release is involved in C. perfringens epsilon toxin neuropathology.
- P-148** - Raineri Mariana - Modafinil attenuates methamphetamine acute toxic effects in mice.
- P-151** - Revillo Damián - Ontogenetic differences in sensitivity to LiCl- and amphetamine-mediated taste avoidance in preweanling rats.

## → Sensory Systems

### »» SESSION I

- P-154** - Acosta Luis E - Modulation of gliogenesis by glia-glia and neuron-glia interactions in the olfactory pathway
- P-157** - Cattaneo Verónica - Analysis of the dynamic of postmitotic neuronal migration during corticogenesis in the chick optic tectum
- P-160** - López María Eugenia - Building the colour space of tetrachromatic species

»» SESSION II

**P-155** - Battista Ariadna - Purinergic signaling is involved in the regeneration of the zebra-fish retina.

**P-158** - Issolio Luis - Rod-Cone Interaction in the mesopic

**P-161** - Marachlian Emiliano - Olfactory processing in the antennal lobe of the honey bee: neural representation of mixtures and pure odors

»» SESSION III

**P-156** - Bellotti Mariela - The electrical response of Human Cells using the ECIS technique

**P-159** - Lipovsek Marcela - Adaptive evolution of  $\alpha 9\alpha 10$  nicotinic acetylcholine receptors.

**P-162** - Ricatti Jimena - Daily proliferative activity in neurogenic niches of zebrafish retina is regulated by purinergic signals

→ *Cognition,  
Behavior, and Memory*

»» SESSION I

**P-163** - García Sebastian - PREGNENOLONE INFUSED IN LATERAL SEPTUM IMPAIRS MEMORY ACQUISITION OF MALE RATS IN A PASSIVE AVOIDANCE TEST

**P-166** - Alzugaray Sabina - Impact of cognitive training on neuronal and glial markers in the hippocampus of non human primates (*Macaca fascicularis*)

**P-169** - Barraco Mariano - fNIRS and pupilometry as a measurement of mental effort

**P-172** - Bavassi Luz - Event-related potentials during a finger tapping task

**P-175** - Caceres Lucila Guadalupe - Neonatal X irradiation: Increases in PKC levels underlie behavioural changes.

**P-178** - Carbó-tano Martín - GABA like receptors distribution in the central nervous system of *Chasmagnathus*

**P-181** - Coletti Natalia - Overcoming the amnesia caused by blockade of hippocampal muscarinic receptors by previous exposures to an open field.

**P-184** - Cuenya Lucas - FRUSTRATION RESPONSES IN PSYCHOGENETICALLY SELECTED RATS: EFFECT OF PARTIAL REINFORCEMENT ON INSTRUMENTAL AND CONSUMMATORY SUCCESSIVE NEGATIVE CONTRAST IN MALE ROMAN HIGH AND LOW AVOIDANCE RATS

**P-187** - Décima Agustín - Attending a direction increases the performance in the opposite direction

**P-190** - Fiorentini Leticia - Qualitative and quantitative study of cognitive functioning in Multiple Sclerosis

**P-193** - Gabach Laura A. - SILDENAFIL PROMOTES COCAINE SENSITIZATION AND ENHANCES HIPPOCAMPAL LTP

**P-196** - Gonzalez Carolina - Formation and consolidation of an aversive memory: role of the Medial Prefrontal Cortex

- P-199** - Hernández José Ignacio - IS THERE A ROLE FOR OPIATES IN MEDIATING ACETALDEHYDE'S (ACD) UNCONDITIONED PROPERTIES DURING EARLY ONTOGENY?
- P-202** - Karadayian Analía - Chronic exposure to paraquat induces changes in body weight gain, anxiety like behavior and non social olfactory discrimination.
- P-205** - Kramar Cecilia - A new player: the role of retrosplenial cortex in memory processing.
- P-208** - Lagos Maria - Novel localization of NF-kappa B in mouse forebrain synaptic terminals. Activation dynamics during memory consolidation.
- P-211** - Locatelli Fernando - Nonassociative plasticity alters competitive interactions among mixture components in early olfactory processing
- P-214** - Marisa Ghersi - Ghrelin inhibited serotonin release from hippocampal slices.
- P-217** - Mayol -trancoso Rocío - Visual behavior during free viewing of images from different categories
- P-220** - Monteiro Gomes Guilherme - The novel peptidic toxin Tx3-1, extracted from the venom of the spider Phoneutria Nigrieventer, rescue memory deficits of mice submitted to a model of Alzheimer's Disease
- P-223** - Navajas Joaquín - Dynamics of conscious access during eye movements
- P-226** - Pautassi Ricardo - EFFECTS OF SEQUESTERING ACETALDEHYDE ON ALCOHOL-MEDIATED CONDITIONED PLACE PREFERENCE IN ADOLESCENT RATS
- P-229** - Poretti María Belén - Fluoxetine revert memory impairment in a depressive animal model.
- P-232** - Rodriguez Maria Laura Cecilia - Contribution of the Gabaergic system to the labilization-reconsolidation process in a neutral verbal memory paradigm
- P-235** - Schneider Elisa - Eye movements reflect syntactic organization of arithmetic thinking
- P-238** - Uran Soledad Lucia - Loud noise exposure induces associative memory and anxiety levels impairments.
- P-241** - Vigliecca Nora Silvana - APHASIA: WHEN JUST ONE NEUROPSYCHOLOGICAL CONDITION GENERATES MORE SYNDROMES THAN SYMPTOMS

## »» SESSION II

- P-164** - Acevedo Ma. Belén - AGE-RELATED DIFFERENCES AND ROLE OF NOVELTY IN THE EXPRESSION OF ETHANOL-INDUCED LOCOMOTOR ACTIVATION
- P-167** - Anllo Hernan - Shines and shadows of perception: The threshold for access to consciousness fluctuates with phases of Bipolar Disorder
- P-170** - Barraza José F. - Modal and amodal contour completion are driven by different mechanisms
- P-173** - Boccia Mariano M - Sildenafil enhances memory reconsolidation of an inhibitory avoidance task in mice
- P-176** - Caffaro Pedro - ROLE OF MUSCARINIC MECHANISMS IN LONG-TERM MEMORY STORAGE VS LONG-TERM MEMORY EXPRESSION
- P-179** - Carlini Valeria Paola - Neuronostatin administration impairs memory and induces anxiolytic effects in rats.
- P-182** - Corti Bielsa Gonzalo D. - Modeling information transfer in eusocial bees
- P-185** - De Giovanni Laura Noemí - Involvement of the Glutamatergic Neurotransmission in the Stress and Cocaine-Induced Reinstatement of Extinguished Cocaine-Induced Condi-

tioned Place Preference in rats

**P-188** - Delfina De Achával - SOCIAL FUNCTIONING AND COGNITION IN PATIENTS WITH SCHIZOPHRENIA, THEIR UNAFFECTED SIBLINGS AND HEALTHY CONTROLS: IMPACT ON QUALITY OF LIFE.

**P-191** - Fiorenza Natália - Molecular mechanisms in hippocampus and basolateral amygdala but not in parietal or cingulate cortex are involved in extinction of one-trial avoidance learning

**P-194** - Giachero Marcelo - INTERACTION BETWEEN A CONSOLIDATED FEAR MEMORY AND A STRESSFUL SITUATION DURING REACTIVATION. PHARMACOLOGICAL MANIPULATION IN THE BASOLATERAL AMYGDALA.

**P-197** - Graziano Martin - Neurophysiology of subjective confidence in a partial report paradigm

**P-200** - Justel Nadia - Anxiolytic effect of testosterone in an animal model of frustration

**P-203** - Kedikian Ximena - ZEBRAFISH AS A MODEL FOR ADDICTIVE BEHAVIORS

**P-206** - Krawczyk María - Hippocampal alpha7 nicotinic receptors modulate memory reconsolidation of an inhibitory avoidance task in mice

**P-209** - Landi Sofía - Neuroplasticity: changes in grey and white matter structure induced by long-term motor learning

**P-212** - Lucchina Luciana - Alterations in the inflammatory response in a mouse model of autism

**P-215** - Martijena Irene - EFFECT OF MK-801 ON FEAR CONDITIONING AND SUBSEQUENT ETHANOL-INDUCED CONDITIONED PLACE PREFERENCE IN ETHANOL WITHDRAWN RATS.

**P-218** - Miranda Morales Roberto Sebastián - Opioid system blockade inhibits operant behaviors in an infantile model of ethanol self-administration task.

**P-221** - Monti Carolina - Hippocampus synaptic plasticity related to contextual cues involved in chronic diazepam administration and withdrawal

**P-224** - Pastor Verónica - NICOTINE-INDUCED CONDITIONING PLACE PREFERENCE IN LOW AND HIGH NICOTINE-RESPONDER RATS

**P-227** - Peszano Valeria Natacha - SUCCESSIVE NEGATIVE CONTRAST IN RATS. EFFECTS OF FRONTAL CORTEX LESIONS

**P-230** - Radiske Andressa - Modulation of extinction memory persistence after its expression

**P-233** - San Martín Alvaro - Understanding learning Disability by enhanced Ras/MAPK signaling

**P-236** - Siele M. Eugenia - NF-kappa B like DNA binding activity during long-term memory consolidation in the honey bee

**P-239** - Vigliecca Nora Silvana - AUTOMATED AND ABBREVIATED NEUROPSYCHOLOGICAL TESTS OF FREE DISTRIBUTION: ATTENTION, MEMORY, GNOSIA, PRAXIA, APHASIA, AND EXECUTIVE FUNCTION

**P-242** - Villalta Jorge - Role of the posterior parietal cortex in online movement corrections

»» SESSION III

**P-165** - Alen Nadia - Acute stress induces the formation of long-term memories

**P-168** - Ballarini Fabricio - Behavioral tagging during an inhibitory avoidance task: identi-

cation of involved transmitter systems, learning tag-molecules in hippocampus-dependent long-term memory formation

**P-171** - Barttfeld Pablo - A big-world network in ASD: Dynamical connectivity analysis reflects a deficit in long-range connections and an excess of short-range connections

**P-174** - Bustos Silvia Gabriela - RESISTANT FEAR MEMORIES TO THE DISRUPTIVE EFFECT OF MIDAZOLAM ON MEMORY RECONSOLIDATION: INFLUENCE OF D-CYCLO-SERINE

**P-177** - Campetella Florencia - Electrophysiological markers in word association experiments: revisited

**P-180** - Cattaneo Veronica - Noonan syndrome animal model suggests mechanistic bases of gene dosage imbalance-dependent learning disability

**P-183** - Costanzo Elsa Y. - Asymmetrical Contribution of Brain Structures to Modulation of Emotion As Indicated by Differential Effects of Right Subgenual Cingulum Stimulation

**P-186** - De La Fuente Verónica - Calcineurin and Nuclear Factor of Activated T-cells (NFAT) involvement in fear memory consolidation and extinction

**P-189** - Fabio Maria Carolina - Emotional reactivity in adolescent animals with or without prenatal ethanol exposure

**P-192** - Fustiñana María Sol - Labilize or not labilize? That is the question

**P-195** - Goldin Andrea P. - Socrates' teaching brain: The Meno experiment

**P-198** - Hepp Yanil. - NMDA like receptors in Chasmagnathus granulatus

**P-201** - Kaczer Laura - Memory updating during reconsolidation in the crab Chasmagnathus

**P-204** - Klappenbach Martín - A dopamine antagonist impairs an aversive memory with a narrow window of effect in the crab Chasmagnathus

**P-207** - Lafon Belén - The Role of Awareness in Trace Conditioning Learning and Expression

**P-210** - Langer Federico - Mental imagery and emotion. A neurophilosophical approach to literary aesthetics.

**P-213** - Maldonado Noelia M. - Prior stress exposure facilitates the behavioural and molecular changes induced by a weak fear conditioning procedure.

**P-216** - Martin Elias Costa - A new paradigm to study speech recognition: semantic bistability.

**P-219** - Molinas Julieta - Visual context affects decision-making of the escape direction in the crab Chasmagnathus

**P-222** - Mora Martina - DECISION MAKING, COGNITIVE FUNCTIONING AND CARDIOVASCULAR RISK FACTORS

**P-225** - Patterson Sean I. - Mapping connective plasticity in 3 dimensions in the rat spinal cord after sciatic nerve axotomy

**P-228** - Petroni Agustín - Early ERPs (N170) Measures of Valence, Interference and Stimulus Type Discrimination: Association to Executive Function and Social Cognition

**P-231** - Renner Maria - New paradigm to measure retrieval induced forgetting in rats.

**P-234** - Sanchez Federico - Association between abstract category learning and executive function

**P-237** - Snitcofsky Marina - Rats either in their sleep- or awake-phase are rescued from amnesia of inhibitory avoidance caused by scopolamine, by previous exposures to the open field.

**P-240** - Vigliecca Nora Silvana - APHASIA: WHAT ARE WE MEASURING?

**P-243** - Villarreal Mirta - Brain Activity during Social Cognition tasks in Individuals with Schizophrenia, their Unaffected Siblings, and Healthy Controls.

## **\* Synaptic Transmission and Excitability**

Synaptic Transmission and Excitability

**Poster Number (1) Session 1**

Rapid endocytosis in mouse chromaffin cells is controlled by intracellular and extracellular calcium sources

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Endocytosis is critical for maintaining membrane homeostasis and secretion reliability in neuroendocrine cells. Chromaffin cell rapid endocytosis (RE) is a Ca<sup>2+</sup> dependent process that overcomes previous exocytosis at a Ca<sup>2+</sup> entry → 75pC, i.e. excess retrieval (EX). In order to study the Ca<sup>2+</sup> sources that regulate RE we used patch clamp capacitance measurements, and applied specific pharmacological agents. We found that the L-Ca<sup>2+</sup> channel blocker nitrendipine (10μM) completely inhibited RE. As in many cells Ca<sup>2+</sup> entry through L-Ca<sup>2+</sup> channels is coupled to Ca<sup>2+</sup> release from endoplasmic reticulum, we tested the effect of Ryanodine (Ry) on RE. Ry (100μM) partially inhibited RE (by 62%) and completely blocked excess retrieval. To identify if RE is activated by a global or local Ca<sup>2+</sup> signal, we replaced EGTA in the internal solution by the fast Ca<sup>2+</sup> buffer BAPTA. A partial inhibition of RE (by 25%) and a reduction of EX (by 66%) was observed. We conclude that RE is triggered by a localized Ca<sup>2+</sup> increase provoked by a combined action of Ca<sup>2+</sup> entry through L-Ca<sup>2+</sup> channels and Ca<sup>2+</sup> release from endoplasmic reticulum.

Synaptic Transmission and Excitability

**Poster Number (2) Session 2**

Mice lacking dopamine D2 autoreceptors are hyperactive and display increased motivation for natural rewards and sensitivity for cocaine

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We generated mice lacking presynaptic D2 autoreceptors (autoDrd2<sup>-/-</sup>). DA release in the dorsal striatum of autoDrd2<sup>-/-</sup> mice showed a significant increase ( $3.13 \pm 0.23 \mu\text{M}$ ) in comparison to Drd2flox/flox control mice ( $1.97 \pm 0.15 \mu\text{M}$ ), as measured by fast cyclic voltammetry on brain slices. The D2R agonist quinpirole inhibited electrically stimulated DA release in Drd2flox/flox mice with an IC<sub>50</sub> of  $19 \pm 0.3 \text{ nM}$  whereas this drug had no effect in autoDrd2<sup>-/-</sup> mice. Basal DA clearance was identical in brain slices of autoDrd2<sup>-/-</sup> and Drd2flox/flox mice. In addition, the significant changes in DA uptake inhibition elicited by the DA uptake blockers methylphenidate or cocaine did not differ between genotypes. At the behavioral level, autoDrd2<sup>-/-</sup> mice displayed increased locomotor activity and were insensitive to low doses of the D2R agonist quinpirole. Curiously, autoDrd2<sup>-/-</sup> mice also showed behavioral supersensitivity for cocaine and haloperidol compared to wild-type mice. Conditioned place preference for cocaine was also higher in autoDrd2<sup>-/-</sup> mice. Our results demonstrate the importance that D2 autoreceptors play in the regulation of DA transmission to set the level of spontaneous locomotor activity, motivation for natural rewards and sensitivity to drugs of abuse.

Synaptic Transmission and Excitability

**Poster Number (3) Session 3**

Electrical activity regulates neuronal maturation in the adult dentate gyrus

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Newborn granule cells (GCs) of the adult hippocampus develop over several weeks. Little is known about the role of intrinsic activity in GC development. To answer this question we designed a strategy to decrease intrinsic excitability of adult-born GCs and analyze their morphological and functional properties. We built a retrovirus encoding an inward rectifying potassium channel (Kir2.1) and green fluorescent protein (GFP), and delivered it into the dentate gyrus of young adult mice. Labeled cells were analyzed in brain slices by electrophysiology and confocal imaging after 2, 3 or 5 weeks. Electrophysiological recordings confirmed that Kir-expressing neurons displayed larger inward rectifier currents, decreased input resistance, hyperpolarized resting potential, and diminished levels of sub-threshold depolarization. In regard to neuronal phenotype, Kir neurons expressed higher levels doublecortin and reduced levels of calbindin at all time points, which was also associated to a reduced dendritic length. Kir neurons also exhibited a reduced frequency of both excitatory and inhibitory miniature events, and a striking reduction in survival. Our results demonstrate that intrinsic electrical activity is

essential for the correct maturation, integration and survival of adult-born GCs.

Synaptic Transmission and Excitability

**Poster Number (4) Session 1**

Calcium current alterations at the calyx of held in S218L KI mice mutant

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Familial hemiplegic migraine type-1 (FHM1) is caused by missense mutations in the CaV2.1 Ca<sup>2+</sup> channel. We used knock-in (KI) transgenic mice with the pathogenic FHM-1 mutation S218L to study Ca<sup>2+</sup> current alterations at the calyx of Held. Due to the fact that the human disease is expressed in a heterozygote (Het) mode, we decided to focalize our studies in this genotype. Using whole-cell patch-clamp recordings, a shift was found in the I-V curve at more negative potentials (WT: -10 mV; Het: -15 mV; KI: -20 mV). The steady-state activation curves were significantly different between WT and Het mice. Presynaptic calcium currents evoked at the calyx of Held of WT or Het mice by their own action potentials (APs) were similar in amplitude. However Het mice showed an increase in IpCa amplitudes when evoked by the longer AP waveforms characteristic of the pyramidal cells. Additionally, Ca<sup>2+</sup> current facilitation after 100 Hz train of APs is reduced in Het compared to WT mice. Our results suggest that: 1) longer time courses of pyramidal cell APs are a key factor for the expression of a synaptic gain of function in the mutated mice as it was demonstrated in other transgenic mice model (R192Q); 2) activation/inactivation properties of CaV2.1 Ca<sup>2+</sup> channels are modified by the mutation.

M1 muscarinic receptor involvement in LTP induction in CA1 synapsis of rat hippocampus

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To investigate the participation of M1 cholinergic muscarinic receptor in synaptic plasticity in the rat hippocampus, we recorded field excitatory postsynaptic potentials (fEPSPs) in CA1 area of hippocampal slices. Muscarinic toxin MT7 from Green mamba venom is a highly selective M1 antagonist. Long term potentiation (LTP) induced by a theta burst stimulation protocol (TBS) was blocked by both scopolamine (specific muscarinic antagonist) and pirenzepine (selective M1 and M4 antagonist). We used MT7 to elucidate M1 participation in this phenomena, to find out whether it was responsible for the observed blockade of LTP by classical muscarinic antagonists. Radioligand binding assays in rat hippocampal synaptosomal membranes were performed in order to corroborate MT7 specificity on muscarinic receptors. The toxin inhibited 3H-N-methylscopolamine (muscarinic ligand) binding with  $K_i = 0,70 \pm 0,08$  nM, with a maximal inhibition of  $38 \pm 5$  %, and did not significantly inhibit 3H-prazosin ( $\alpha$ -adrenergic ligand) binding. In electrophysiological assays, infusion of 10 nM MT7 on rat hippocampal slices, blocked LTP induction without significant effect on basal transmission or paired pulse facilitation. We propose that M1 receptors are necessary for the induction of LTP by TBS at CA1 synapses.

P/Q-type CA2+-channels are required for acid-sensing ion channels 1A (ASIC1A)-mediated inhibition of neuromuscular transmission in mice

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CNS changes in pH<sub>O</sub> have considerable influence on the physiology of neurons,

and in pathological conditions affect the outcome of neuronal injury. Acid-sensing ion channels (ASICs) have been proposed to regulate synaptic transmission in response to acidosis. Recently, we have described the presence of ASIC1a at the presynaptic terminals of neuromuscular junctions (NMJs), where they may exert an inhibition on neuromuscular transmission. Considering our evidence and the fact that several neurological diseases are characterized by an abnormally sustained synaptic transmission, the aim of this work was to investigate the modulatory role of ASIC1a in such pathological conditions. We initially studied miniature endplate potentials frequencies from mice lacking P/Q-type  $\text{Ca}^{2+}$ -channels (1A-/-) in the absence or presence of psalmotoxin. No significant difference was observed after the application of psalmotoxin (Student's t-test,  $p=0,278$ ). Neither evoked endplate potentials during paired-pulse nerve-stimulation at 20 and 50 Hz change significantly after the application of psalmotoxin (Student's t-test,  $p=0,314$  and  $p=0,834$ , respectively). In conclusion, the lack of expression of the P/Q-type  $\text{Ca}^{2+}$ -channels might preclude ASIC1a channels from inhibiting neuromuscular synaptic transmission in mice.

Synaptic Transmission and Excitability

### **Poster Number (7) Session 1**

#### Muscarinic M1 modulation on basal neurotransmission in CA1 region of rat hippocampus

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To investigate the putative M1 muscarinic receptor modulation in CA1 synapses of rat hippocampus, we used muscarinic toxins (MTs) from Green Mamba venom as selective M1 receptor ligands. MT1 and MT2 are M1 agonists and M4 antagonists. MT7 is a highly selective M1 antagonist. We have shown that MT2 enhanced field potentials (fEPSP) in CA1 region of rat hippocampus and this was blocked by both pirenzepine and atropine. We performed 3H-N-methylscopolamine (3H-NMS) and 3H-prazosin (3H-PRZ) binding assays in hippocampal synaptosomal membranes to evaluate MTs specificities. All toxins inhibited 3H-NMS binding with  $K_i=89\pm10$  nM for MT1;  $3,2\pm0,9$   $\mu\text{M}$  for MT2 and  $0,70\pm0,08$  nM for MT7. MT1 and MT2 inhibited 3H-PRZ binding with  $K_i=83\pm3$  nM and  $292\pm11$  nM. MT7 did not inhibit 3H-PRZ binding. Perfusion of  $1\mu\text{M}$  MT1 enhanced fEPSP by  $45\pm4$  %; this facilitation was blocked by 10nM MT7. Perfusion of  $10\mu\text{M}$  PRZ enhanced basal

fEPSP in  $16 \pm 3$  %. Blockade of MT2's effect by both pirenzepine and atropine and MT1's blockade by MT7 evidence M1 involvement in these effects. Since PRZ inhibited 3H-NMS binding ( $K_i = 5,1 \pm 1,2 \mu\text{M}$ ), we speculate that PRZ effect on fEPSP could be mediated by muscarinic receptors. These results show that M1 receptors positively modulate basal transmission in CA1 synapses.

Synaptic Transmission and Excitability

**Poster Number (8) Session 2**

P/Q-type calcium channel  $\alpha 1$  subunit is necessary for ALS-IgGs spontaneous synaptic activity modulation on mouse neuromuscular junction

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Amyotrophic Lateral Sclerosis (ALS) is a neurodegenerative disease that promotes motoneurons' dysfunction and death. Most patients present a sporadic form of unknown etiology. In spite of the existence of many hypotheses trying to explain this variant, there is ample evidence supporting immune-mediated mechanisms. The aim of our work was to evaluate whether the absence of the  $\alpha 1A$  subunit could affect the neuromuscular junction (NMJ) spontaneous synaptic activity modulation already observed with IgGs from our patients (ALS-IgGs). Electrophysiological experiments on mouse diaphragm were performed by intracellular recordings while immunofluorescence assays were carried out using ALS-IgGs as primary antibodies. We found that 63% of our ALS-IgGs (5/8) were able to significantly increase miniature end-plate potentials (MEPPs) frequencies and presented a strong immunoreactivity against NMJ. In addition, using  $\alpha 1A^{+/+}$  and  $\alpha 1A^{-/-}$  mice, we discovered a positive correlation between ALS-IgGs effects and the presence of P/Q-type voltage-dependent calcium channels (VDCCs). We found a significant decrease in NMJ immunoreactivity in  $\alpha 1A$ -deficient animals as well as a lack of ALS-IgGs effects over MEPPs frequencies. We concluded that the absence of the P/Q-type VDCC, or at least its  $\alpha 1$  subunit, was positively correlated with synaptic modulation activity in ALS IgGs samples.

Gaba fails to regulate the release of ACh at efferent-cochlear hair cell synapses in GABAB-knockout mice

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During neonatal development inner hair cells (IHCs) of the mammalian cochlea are transiently innervated by medial olivocochlear (MOC) efferent fibers. Acetylcholine (ACh) is the main neurotransmitter released at this synapse but  $\gamma$ -aminobutyric acid (GABA) is also present at MOC synaptic terminals. We have previously shown by electrophysiological and pharmacological methods that GABA modulates the cholinergic input at MOC-IHC synapses by acting on presynaptic GABAB receptors. To further evaluate the role of GABAB receptors in the modulation of ACh release, we recorded postsynaptic currents evoked by electrically stimulating the MOC efferent fibers contacting IHCs, in acutely isolated mouse organs of Corti from GABAB-knockout mice. Application of the specific GABAB agonist, baclofen (1 $\mu$ M), caused a significant reduction in the quantal content of evoked release in wild-type mice ( $37.3 \pm 6.1\%$  of control,  $n=4$ ,  $p < 0.01$ ) but did not affect this parameter in GABAB-knockout mice ( $103.1 \pm 1.6\%$  of control,  $n=2$ ,  $p > 0.05$ ). These preliminary results show that in the absence of GABAB receptors, GABA fails to regulate the release of ACh at MOC terminals. We are now evaluating whether P/Q or N-type  $\text{Ca}^{2+}$  channels, that support release at this synapse, are the targets of GABAB receptor activation.

Short term plasticity (STP) changes during development of the medial olivocochlear-inner hair cell synapse in the mouse cochlea

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In the organ of Corti, the sensory cells of the mammalian auditory system, inner hair cells (IHCs) transduce sound stimuli into electrical signals that are conveyed to the central nervous system. Before the onset of hearing, IHCs are transiently innervated by cholinergic medial olivo-cochlear (MOC) fibers. Synaptic activity was recorded in voltage-clamped IHCs from excised apical turns of the mouse cochlea at two developmental stages (postnatal days (P) 5-7 and 9-11), during electrical stimulation of the MOC fibers. Ten-pulse trains at 10, 20, 40 and 100 Hz applied to P5-7 MOC-IHC synapses led to  $2.9\pm0.7$ ;  $2.1\pm0.4$ ;  $2.4\pm0.5$  and  $2.0\pm0.4$  fold increase in synaptic efficacy, respectively, estimated as the ratio between the amplitude of the fifth and the first evoked synaptic current ( $S5/S1$ );  $n=7-8$ . Facilitation induced by the 40 and 100 Hz trains was followed by a slight depression. The same protocols applied to P9-11 synapses led to a progressive decrease of the  $S5/S1$  value ( $0.9\pm0.1$ ;  $0.8\pm0.1$ ;  $0.8\pm0.1$ ;  $0.5\pm0.1$  for the 10, 20, 40 and 100 Hz trains, respectively;  $n=12-19$ ). Depression upon high frequency stimulation at P9-11 was reversed to facilitation when reducing  $[Ca^{2+}]_o$ . Our results suggest differences in the coupling between  $Ca^{2+}$  influx and transmitter release at the two stages.

## **\* Cellular and Molecular Neurobiology**

Cellular and Molecular Neurobiology

**Poster Number (11) Session 1**

Activation of antioxidant defense mechanisms by docosahexaenoic acid and eicosaexaenoic acid prevents apoptosis of retina photoreceptors

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Oxidative stress participates in activating the apoptosis of photoreceptors (PRs) in retinal neurodegenerative diseases. We have shown that docosahexaenoic acid (DHA), the major retina polyunsaturated fatty acid, protects PRs from apoptosis induced by the oxidant paraquat (PQ) by activating the ERK pathway. Here we investigated if other fatty acids had a similar protective effect and if this protection involved the activation of antioxidant defense mechanisms in PRs. Rat retina neuronal cultures were supplemented with EPA (eicosapentaenoic acid), DHA, Palmitic, Oleic and Arachidonic acids and treated at day 3 with PQ. Only EPA and DHA prevented PR apoptosis. As EPA is a DHA precursor, we investigated whether

it protected PRs by itself or through its conversion to DHA. When we evaluated the fatty acid composition of EPA-supplemented neurons, EPA levels remained constant but DHA content significantly increased. Addition of hydrogen peroxide (H<sub>2</sub>O<sub>2</sub>) induced PR apoptosis and increased the production of reactive oxygen species (ROS), measured by DCFDA. DHA and EPA prevented PR apoptosis and decreased ROS formation after H<sub>2</sub>O<sub>2</sub> addition. This work suggests EPA protects PRs through its conversion to DHA and implies that DHA and EPA activate antioxidant defense mechanisms to rescue PRs.

Cellular and Molecular Neurobiology

**Poster Number (12) Session 2**

Calcineurin regulates PERK autophosphorylation in astrocytes under stress

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The accumulation of unfolded proteins into the Endoplasmic Reticulum (ER) activates a signal transduction cascade called Unfolding Protein Response (UPR), which attempts to restore homeostasis in the organelle. (PKR)-like-ER kinase (PERK) is an early stress response transmembrane protein that is generally inactive due to its association with the chaperone BIP. During ER stress, BIP is tritrated by the unfolded protein, leading PERK activation and phosphorylation of eukaryotic initiation factor-2 alpha (eIF2alpha), which attenuates protein synthesis. We demonstrated that calcineurin-A/B (CN-A/B), an heterotrimeric Ca<sup>2+</sup> phosphatase, associates with PERK, increasing its auto-phosphorylation and significantly enhancing inhibition of protein translation and cell viability in *Xenopus* oocytes. Moreover, we report that CN-A/B and PERK interaction is significantly increased after Oxygen and Glucose Deprivation (OGD) treatment in astrocytes. These cells deficient in CN-Abeta isoform have constitutive active UPR. OGD treatment did not further increase cell death or eIF2alpha phosphorylation in CN-Abeta<sup>-/-</sup> cells, but did so in both CN-Aalpha<sup>-/-</sup> and wild-type controls. Our finding indicates that the protective role of CN observed in *Xenopus* oocytes is extended to astrocytes stressed by OGD.



OPA1 cleavage mediates impaired mitochondrial dynamics in an experimental model of parkinsonism

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Neurotoxicity due to excessive manganese (Mn) accumulation in the brain leads to Manganism, a neurodegenerative pathology, whose symptoms resemble those of Idiopathic Parkinson's Disease. Mn is mainly accumulated in astrocyte's mitochondria which continuously undergo fusion/fission events to form a dynamic tubular network (DTN). OPA1 is a fusion GTPase involved in cristae remodeling and maintenance; its cleavage and complexes disassembly lead to DTN disintegration and cell death. Previously, we have reported mitochondrial morphology alterations and OPA1 processing in astrocytoma C6 cells Mn- induced apoptosis, an experimental model of Parkinsonism. The present work demonstrates that mitochondrial membrane potential collapse, and consequent opening of mPTP by Mn, has drastic consequences on DTN integrity. Cyclosporin A (mPTP inhibitor) nearly avoided mitochondria fragmentation and OPA1 cleavage. Moreover, C6 cells transiently transfected either WT or Q297V (disassembly-resistant mutant) OPA1 cDNA3 resulted in cell viability protection, reduced number of apoptotic nuclei and maintenance of DTN integrity. Our results suggest that OPA1 has a bi-functional role, promoting mitochondrial fusion, and regulating apoptosis by playing as an anti-apoptotic protein.

Two sides of the same coin: lysosomal and autophagic pathways in manganese-induced glial cell death

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Manganese (Mn) overexposure produces neurodegenerative damage associated with a high risk of developing Idiopathic Parkinson's Disease. There is increasing evidence that autophagy impairment and the subsequent accumulation of aggregated proteins are involved in the pathogenesis of neurodegenerative diseases. In

this work we studied the role of the lysosomal/autophagic pathways in Mn-induced Parkinsonism in glioma C6 cells. Cell viability measured by neutral red (a vital dye that accumulates in lysosomes) retention, resulted in a  $45\pm4\%$  ( $p<0.05$ ) indicating the possible involvement of different autophagic pathways. Furthermore, we demonstrated the participation of the lysosomal pathway measuring both an increase in lysosomes size (stained with LysoTracker Red), and in cell viability by inhibition of v-ATPase and Cathepsin D activities with Bafilomycin A1 and Pepstatin A, respectively. Our results suggest the involvement of the lysosomal/autophagic compartment in Mn cytotoxicity and shows a new link between Parkinson's disease and Manganism.

Cellular and Molecular Neurobiology

**Poster Number (15) Session 2**

UPA:uPAR complex co-localize with  $\alpha 5\beta 1$  integrin and is related with FAK activation

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We demonstrated that uPA (urokinase-type plasminogen activator) promotes neuronal migration and neuritogenesis. This response is unrelated with its proteolytic activity. The membrane receptor of uPA (uPAR) is a GPI-anchored protein and it was shown that it mediates the phosphorylation of focal adhesion kinase (FAK) in fibroblast cultures. This work explored whether a  $\alpha 5\beta 1$  integrin heterodimer is associated with uPA:uPAR and whether this complex participates in the FAK phosphorylation during neuronal migration and neuritogenesis. Explants of optic tecta from chicken embryos of 7 days were incubated for 16 hs and then were exposed to a 10 nM uPA pulse for 2-5 minutes. They were fixed and immunolabeled with uPAR,  $\alpha 5$ ,  $\alpha 6$ ,  $\beta 1$  integrin subunits and pFAK antibodies. Besides optic tecta were cut into small pieces, exposed to a 10 nM uPA pulse for 2-5 minutes and lysated in buffer with protease and phosphatase inhibitors. Western blots of whole cell lysates or immunoprecipitations (IP) were analysed. Confocal analysis showed a co-localization of uPAR with  $\alpha 5$  and  $\beta 1$ . The co-immunoreactivity was higher in the uPA stimulated neurons. In control explants pFAK was restricted to somas while in uPA stimulated neurons the pFAK expression was expanded to neuritis. The IP with anti-uPAR and the subsequent western blot for integrins confirm the morphological results. pFAK analysis by western confirm the increase of

FAK phosphorylation in the experimental conditions. These results show the interaction of  $\alpha 5\beta 1$  integrin with uPAR and suggest that this interaction promote the kinases activation. This work was supported by grant from UBACyT

Cellular and Molecular Neurobiology

**Poster Number (16) Session 3**

The subventricular region in streptozotocin-induced hyperglycemia

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Alterations of adult neurogenesis have been shown in rodents after long-term diabetes. We have now evaluated neurogenesis in the subventricular zone (SVZ) after short-term hyperglycemia. Male C57Bl/6J mice received streptozotocin (SZT), a pancreatic islet toxic, or vehicle injections only. STZ-mice reached hyperglycemia on day 1. Mice received 5-bromo-2'-deoxyuridine (BrdU) on day 3, and were perfused at day 4. Brains were processed for immunohistochemistry, using antibodies against BrdU, mini-chromosome maintenance protein 2 (Mcm2), glial fibrillary acidic protein (GFAP), nestin and cleaved caspase-3 (CC-3). Control and experimental mice showed the same distribution of GFAP+ and nestin+ cells. BrdU+ and Mcm2+ cells appeared along the lateral and dorsal walls of the lateral ventricle. STZ brains contained less BrdU+ nuclei than control brains. By contrast, the SVZ of STZ mice displayed a larger number of Mcm2+ cells than control brains. No labeling for CC-3 was detected, neither in control or STZ brains. Our findings showed that insulin depletion was immediately followed by alterations of the cell cycle in neural precursor cells of the SVZ. Since no evidence of cell death was detected, decrease in BrdU incorporation most likely reflected slower progression to the S phase. The increased Mcm2+ cells also point to a lengthened G1 phase. These early alterations of neurogenesis are probably related with development of psychiatric and neurological disease in diabetics.

Long term exposure to an enriched environment induces hippocampal changes and reduces soluble abeta levels in a transgenic mouse model of alzheimer

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Our aim was to explore the effects of environmental enrichment (EE) on the neurodegenerative process in an animal model of AD. Female transgenic mice (Tg, PDAPP-J20) carrying the Swe and Ind APP mutations and their non-transgenic siblings (NTg) were housed in EE or in standard conditions (SC) for 3 months (5 to 8 months of life). Soluble AB 1-40 and 1-42 brain levels were reduced in Tg in EE compared with Tg in SC. No differences were found in the number of AB plaques in the hippocampus. In situ hybridization for BDNF mRNA showed an increase in the dentate gyrus (DG) of Tg in EE compared with Tg in SC. Cell proliferation rate (Ki67 labeling) in the DG was significantly lower in Tg, with no effect of EE. Tg mice showed a decrease in the number of doublecortin+ cells in the DG. Survival of newborn BrdU+ cells (BrdU injected 21 days before euthanasia) showed an increase in both Tg and NTg with EE. Ratio of BrdU+NeuN+/BrdU+ cells was calculated, finding a decrease in Tg mice in SC and an increase with EE. Our results indicate that survival of hippocampal neuronal progenitors is promoted by EE in mice that model AD, correlating with increased levels of BDNF and lower levels of soluble AB. This might suggest an important role for social and sensorial stimuli in the pathogenesis of AD.

Axogenic effect of WNT3A factor in hippocampal neurons is mediated by the activation of IGF-1 receptor and PI3K

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The selection of the future axon in cultured hippocampal neuron requires the activation of IGF-1 receptor (IGF-1R), PI3k and the accumulation of PIP3 at the growth cone. Neuronal polarization is regulated by a combination of intrinsic programs of gene expression and by extrinsic factors as Wnts and IGF-1. Wnts, through Frizzled receptors, activate Dishevelled (Dvl), a first downstream effector that can signal through different pathways. In the nervous system, Wnt function as a regulator of neuronal development. We studied Wnt3a on neuronal differentiation, particularly on the regulation of axonal outgrowth. We observed that Wnt3a is necessary for axon formation through the activation of PI3k. Neurons cultured in the presence of Wnt3a or overexpressing Dvl show multiple axons. Importantly, Wnt3a activates PI3k in neurons and in purified growth cones suggesting that Wnt3a may signals through the same pathway as IGF-1/IGF-1R. In addition, we found that Wnt3a cross-activates IGF-1R in neurons and in growth cones and this effect is blocked by an IGF-1R blocking antibody. These findings suggest that Wnt proteins are important for the establishment of neuronal polarity and suggest a possible parallelism between the two signalling systems: Wnt-Fz-DVL and IGF-1-IGFR-PI3k on axon formation.

Cellular and Molecular Neurobiology

**Poster Number (19) Session 3**

Bacterial lipopolysaccharide-induced protection against light-induced retinal damage in rats

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We analyzed the effect of lipopolysaccharide (LPS) on light-induced retinal damage. Male Wistar rats were intravitreally injected with LPS in one eye and vehicle in the fellow eye, one day before an intense light exposure for 24 h. Electroretinograms (ERGs) were registered at several time points after light exposure, and retinal histology was examined 14 days after light. Apoptosis was examined through the assessment of mitochondrial bax/cytosolic bax ratio, and lipid peroxidation was evaluated through the TBARS method. Animals were injected with an iNOS inhibitor (aminoguanidine, AG) or a mitochondrial K<sup>+</sup> ATP channel blocker (5-hydroxidecanoic acid, 5HD), or a phosphatidylinositol 3-kinase (PI3K) inhibitor (wortmannin, WT) and electroretinographic and histological analysis were performed 7 days after light exposure. Light significantly decreased the ERG a- and b-wave amplitude, and the total retina, photoreceptor segments, outer nuclear

layer thickness, and the number of cells in ganglion cell layer, whereas LPS significantly prevented these alterations. Light exposure increased the mitochondrial bax/cytosolic bax ratio and TBARS levels, whereas LPS prevented these changes. The injection of WT (but not of AG or 5HD) completely blocked the functional and histological protection.

Cellular and Molecular Neurobiology

**Poster Number (20) Session 1**

Photoreceptor cell death in low light retinal exposure

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Retinal degenerations, including Retinitis Pigmentosa (RP) and vitamin A deficiencies, exhibit progressive death of rod and cone photoreceptors with subsequent loss of vision. Typical symptoms are night blindness, decreases in visual field and eventually blindness. In some types of RP as well as vitamin A deficiencies, photoreceptor death is due to constitutive activation of the phototransduction pathway, but the biochemical events underlying apoptosis are presently unknown. To answer this question, we are studying the molecular mechanisms involved in retinal degeneration in Wistar rats kept in moderate light (200 lux) to activate phototransduction for different periods of time. Our results showed a significant reduction in outer nuclear layer (ONL) thickness in rat retinas after ten days of continuous light exposure. Western blots detected the active form of caspase 3, and in preliminary results we observed degradation of rhodopsin and melanopsin, during the first five days of exposure. These results indicate that continuous activation of the phototransduction cascade initiates photoreceptor apoptosis within five days of light exposure, and involves a caspase 3-dependent mechanism which leads to measurable cell loss after ten days.

Cellular and Molecular Neurobiology

**Poster Number (21) Session 2**

Glyphosate affects the development of pyramidal hippocampal Neurons

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Glyphosate (active ingredient of Round-Up) is used as a non-selective organophosphorus herbicide for controlling the growth of weeds in forest and agricultural areas particularly in association with genetically modified plants. Mammals and humans are exposed to glyphosate containing herbicides by agriculture or when they enter the food chain. Up to date, this herbicide is considered innocuous and very few data about its toxicity is available. In this work, we study the potential effect of glyphosate on developmental neurons. Particularly, we examine the effect of glyphosate on neuronal differentiation and polarity which are critical processes for establishing the proper neural connections and the acquisition of an adequate neuronal plasticity. Cultured hippocampal neurons were exposed to the herbicide (as acid or salt) at different concentrations during 1 and 2 DIV and their morphology was analyzed by confocal microscopy. After 1 DIV, control neurons develop one clear axon and several minor processes. In contrast, most of glyphosate treated neurons do not polarized after 24h in culture. However, neurons exposed to the herbicide for 2 days show one axon and several dendrites but they exhibit a significant decrease in length and complexity compared to controls. In addition, neurons exposed to the herbicide showed changes in the growth cone morphology. These observations suggest that the exposure to sub-lethal doses of glyphosate causes neurotoxic effects on developmental neurons that could affect neuronal connectivity and function.

Cellular and Molecular Neurobiology

**Poster Number (22) Session 3**

Stress kinase JNK pathway in the regulation of axonal transport

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An efficient kinesin anterograde axonal transport system is essential for neuronal function and metabolism. Microtubule associated proteins such as tau, stabilizes the axonal microtubule network. Impairments on the transport machinery have been suggested in the progression of neurodegenerative diseases including Alzheimer Disease. To understand how JNK activation is involved in the initial steps that lead to transport defects we tested for changes on transport properties and tau modifications under JNK pathway activation. Live imaging experiments on hippocampal cultures were used to describe transport dynamics of fluorescent cargos when JNK was constitutively induced. Axonal transport movies were generated after co-transfection experiments in mature neurons with APP-YFP and psr -JNK (constitutively active) or psr -JNK1-KM (inhibited activity) and transport properties such as directionality, speed, processivity and particle proportion quantified. Co-transfection of p-JNK or p-JNK-KM with a fluorescent protein was used

for immunofluorescent analysis of tau accumulation and hyper-phosphorylation. Western blots from N2a neuroblastoma cells transfected with p-JNK or p-JNK-KM were used to analyze and quantify tau accumulation and hyper-phosphorylation, and for kinesin stability.

Cellular and Molecular Neurobiology

**Poster Number (23) Session 1**

Retinoic Acid stimulates the differentiation of retina photoreceptors in vitro

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Retinoic Acid (RA), a vitamin A prime metabolite, is crucial for correct eye development and photoreceptor (PR) differentiation. We here investigated its effects on early PR development and the intracellular pathways involved. Pure neuronal cultures obtained from 2 day old rat retinae were supplemented with or without 10 nM RA a few hours after plating. RA almost doubled the amount of cells expressing opsin and stimulated the expression of peripherin, a disc structural component, and the formation of apical processes in PRs at every time studied. We then assessed the activation of p38-MAPK, which is involved in RA receptor phosphorylation; RA readily increased phosphorylated p38 levels whereas total p38 remained unchanged. Addition of SB203580, a p38 inhibitor, led to a partial reversion of RA-stimulated opsin expression. We next studied the participation of PI3K, which is thought to have opposite effects to those of p38. Opsin expression was higher in cultures treated with RA plus Wortmannin or LY294002, PI3K inhibitors, than in those only with RA, suggesting RA might negatively regulate PI3K. In summary this work shows that RA promotes early development in vitro of PRs and this stimulation may occur, at least in part, through p38-MAPK activation and negative regulation of PI3K.

Cellular and Molecular Neurobiology

**Poster Number (24) Session 2**

The photoisomerase retinal g protein coupled receptor (RGR) is localized in the ganglion cell layer of the chicken retina

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Absorption of a photon by visual pigments induces isomerization of 11-cis-retinaldehyde (RAL) chromophore to all-trans-RAL. Sustained vision requires continuous regeneration of 11cis-RAL via a process called visual cycle. Subpopulation of retinal ganglion cells (RGCs) expressing the photopigment melanopsin have been shown to be intrinsically photoresponsive (ipRGCs) and responsible for the photic regulation of diverse non-visual functions. To date very little is known about how these ipRGCs do regenerate the chromophore. RGR has been localized to the RPE and Müller cells, and has been shown to interact with proteins involved in the visual cycle, and it is currently believed that RGR behaves as a cofactor modulating isomerohydrolase activity. In the present work we investigated the presence and expression of RGR in RGCs and inner retina of wild type (WT) and GUCY1\* birds, an animal model of blindness lacking functional photoreceptor cells. We found detectable levels of RGR mRNA by RT-PCR and of the RGR protein by Western Blot in both the WT and GUCY1\* chicken retinas as well as in RGC preparations. By immunofluorescence, we observed positive staining for RGR localized in the RGC layer. These results suggest that RGR may play a role in the regulation of the chromophore regeneration in the chicken RGCs.

Cellular and Molecular Neurobiology

**Poster Number (25) Session 3**

Derivation of dopaminergic neurons from human embryonic stem cells, yield efficiency obtained by two protocols

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Human embryonic stem cells (hESCs) are derived from the inner cell mass of the blastocyst of human pre-implantation embryos and can proliferate without apparent limit, while retaining the ability to differentiate into multiple cell types. Efficient derivation of hESCs into neural cells is critical for future cell-based regenerative therapies to treat neurodegenerative disorders such as Parkinson's Disease. The aim of this study was the optimization of a novel protocol to generate an efficient yield of dopaminergic neurons from hESC, and the characterization of the emerging phenotypes throughout the differentiation process when compared with the cell cultures obtained by the implementation of a classical derivation protocol. The nature of the cells and its changes in phenotype were assessed by immunocytochemistry and qPCR focusing on the temporal appearance of neuronal markers such as pax-6, nestin, Tuj1, tyrosine hydroxylase concomitant with the downregulation of stemness markers such as Oct-4 and Nanog. The obtained results showed that both protocols were capable of generating dopaminergic neurons at end point, yet the dynamics and final cellular yield of the protocols

differed. These changes can be ascribed to differences in the time and intensity of exposure to morphogen

Cellular and Molecular Neurobiology

**Poster Number (26) Session 1**

Activation of IGF-1 receptors and the PI3K signalling pathway are involved in adult retinal ganglion cells axonal regeneration

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Axonal damage is a key component of disorders such as stroke and brain, retina and spinal cord injuries, leading to the dysfunction of neuronal networks. In the CNS, injured axons are unable to regenerate and have a limited capacity to sprout and re-establish lost connections. It is accepted that the mechanisms involved on neuritic regeneration of the CNS neurons are similar to those important on the regulation of axonal outgrowth in development. We have previously shown that activation of the IGF-1 receptor and the PI3k-Akt signalling pathway are essential for axonal designation and outgrowth in hippocampal cells. The experiments of the present project have been designed to study the role of the IGF-1 receptor and the PI3k-Akt signalling pathway on the regeneration of adult RGCs (an excellent experimental model of the adult CNS) in culture. Our results indicated that: i) IGF-1R are enriched at the growth cone and distal third axon of regenerating adult RGCs; ii) Treatment of the RGCs with IGF-1 results in activation of the IGF-1R and PI3K at the axonal growth cone and the third distal axon and stimulate axonal outgrowth; and iii) Treatment with an antibody that blocks activation of the IGF-1R or with inhibitors of PI3K significantly inhibit axonal regeneration in RGCs.

Cellular and Molecular Neurobiology

**Poster Number (27) Session 2**

Reduced camp levels and Akt activation are implicated in the anti-apoptotic actions of mGluR3 agonists in astrocytes

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Metabotropic glutamate receptors (mGluR) are implicated in neuroplasticity and neuroprotection. We have previously shown that mGluR3 activation prevents NO-induced astrocyte death, by modulating levels of apoptotic mitochondrial pathway mediators. Since mGluR3 activation inhibits adenylate cyclase and induces Akt activation, we studied these pathways. A non-hydrolyzable analog of cAMP, dibutyryl-cAMP and an Akt1/2 inhibitor both abolished the anti-apoptotic effect of the mGluR3 agonists LY379268 or LY404039 on cultured astrocytes exposed to NO (p

Cellular and Molecular Neurobiology

**Poster Number (28) Session 3**

The regulation of MBP by its interaction with CA<sup>2+</sup>-calmodulin is specific for each isoform and cam binding-site deiminations impair MBP-CaM interaction

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MBP is essential for formation of myelin compact membranes in central nervous system. The interactions of MBP with different cell components are regulated by CaM and by various post-translational modifications. In this regard, we studied the MBP-CaM interaction by immobilized peptide array. There are six variants of MBP encoded by a single gene and generated by alternative splicing. We performed the peptide array with the amino acid sequence of the longest variant that contains all exons, MBP isoform 1, and we find that CaM binds to three distinct domains that lay in the exons involved in alternative splicing. These CaM-binding sites differ in the strength of interaction; the strongest is located between exons 6 and 7 of MBP isoforms 1, 2, 3 and 5. In isoforms 4 and 6 the junction of exon 5 with exon 7 generates a weak CaM-binding site. In addition, each CaM-binding site has three serines and two arginines that can undergo the most common post-translational modifications of MBP such as phosphorylation or deimination respectively. Substitution of arginine by citrulline in the CaM-binding sites impairs MBP-CaM interaction. Deimination of MBP has been correlated with early development, demyelination process and disease severity in MS. Further studies of the regulation of MBP by CaM and post-translational modifications will help to understand its role in signaling for ligodendrocyte differentiation and myelin formation, and its involvement in the pathogenesis of MS. Supported by CONICET, FONCyT and SECyT

Effects of prenatal ethanol exposure on radial glia cells and postmitotic neuroblasts migration in the fetal cerebral cortex

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Prenatal ethanol exposure (PEE) induces cortical dysplasias. Orderly migration of neuroblasts is crucial in cerebral cortex (CC) development. The transcription factor Pax6 is highly expressed in neuroepithelial and radial glia cells (RGCs). Female Wistar rats were fed a liquid diet with 5.9% (w/w) ethanol (EtOH), rendering moderate blood EtOH concentrations. Maternal gestational weight progression and fetal CC thickness were measured. Brain sections of fetal CC obtained at G12, G14, G16, G18 and PND3 were morphologically studied by means of digital image analysis after immunocytochemical experiments against vimentin, nestin, S-100b, Pax6 and doublecortin. RGCs expressing vimentin, nestin, S-100b and Pax6 were morphologically altered. The migration distance through the cortex and the number of doublecortin-ir neuroblasts were decreased. There was significant morphological defects on RGCs, a marked migratory process delay, a decreased number of postmitotic neuroblasts, and a decreased of Pax6-ir cells in the developing cortex of PEE fetuses. These alterations could be involved in the establishment of cortical dysplasias in the offspring of alcoholic mothers, such as those seen in the human fetal alcohol syndrome.

Effect of TIAM1 phosphorylation on dendritic morphology

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The antagonist relationship between Rho and Rac is essential for the establishment of cell polarity. RhoGTPases activity is controlled by GEFs. Tiam1/2 are Rac GEFs that interact with Par3. The overexpression of Tiam1/2 induces multiple axon formation while its suppression prevents axon outgrowth. In non-neural cells, RhoK (a downstream effector of Rho) phosphorylates Tiam1 / 2, preventing its associa-

tion with Par3 and the formation of the polarity complex Par3-Par6-aPKC. In this study we evaluated the effect of Tiam1 mutants mimicking RhoK phosphorylation on dendritic development. To this end, site-directed mutagenesis was performed to generate Tiam1/2 mutants on consensus sequence for Rho-kinase. Thus, by replacing the target residue by alanine (Tiam1-AA), a mutant was generated that can not be phosphorylated by RhoK, while substitution by glutamic (Tiam1-EE) result in a mutant that emulates RhoK induced phosphorylation. Primary cultures of hippocampal neurons were transfected with each construct to evaluate the effect of these mutants on neuronal morphology. Confocal microscopy revealed that the phosphomimic mutant (Tiam1-EE) inhibits dendritic growth, while both Tiam1 wild type and Tiam1-AA stimulate dendritic growth and branching, as well as the subcellular distribution of the axonal marker Tau. Pull-down assay were employed to assess the ability of each mutant to interact with Par3. The results obtained show that the phosphomimic mutant has a decreased ability to interact with Par3.

Cellular and Molecular Neurobiology

**Poster Number (31) Session 3**

Regulation of EphA4 phosphorylation is involved in the growth of retinal ganglion cells axons stimulated by EphA3

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Eph receptor tyrosine kinases and ephrins guide retinal ganglion cells (RGCs) axons in the tectum. Ephrin-As located in the caudal tectum repel temporal (T) axons by activating EphAs. We demonstrated that tectal EphA3 stimulates nasal (N) RGCs axon growth to caudal tectum. It is not known which molecule/s mediates the effect of EphA3. Our objectives were to investigate if 1) the RGCs that express higher levels of ephrin-As grow shorter axons but increase their growth when binding to EphA3; 2) inactivation of axonal EphA4 mimics EphA3 effect on axon growth. We used retinal explants from 7 days-old chicken embryos exposed to Kyl (EphA4 inhibitor) or EphA3-Fc, performed immunocytochemistry against Eph/ephrins-As and Western blot against phosphorylated EphA4. EphA4 is expressed in a shallow decreasing naso to temporal gradient. T RGCs express lower levels of ephrin-As and grow longer axons. N RGCs express higher levels of ephrin-As and grow longer axons with EphA4 inhibition or with EphA3-Fc. EphA3-Fc modifies the profile of EphA4 phosphorylation. This suggests that EphA4 activation by co-

expressed ephrin-As decreases axon growth and that EphA4 inhibition stimulates axon growth produced by EphA3. It is postulated that tectal EphA3 might stimulate N RGCs axon growth through binding with axonal ephrin-As and indirectly inhibiting EphA4 activity. Grants by CONICET and UBACyT.

Cellular and Molecular Neurobiology

**Poster Number (32) Session 1**

Characterization of Ena/VASP domains with dominant negative function

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Neurodegeneration is a process known to occur in metazoans. In previous work we found that the gene enabled (ena), encoding a protein involved in cytoskeleton remodeling, is implicated in progressive neurodegeneration in *Drosophila*. Silencing ena ortholog genes (the ena/VASP family) in mouse hippocampal neurons triggered neurite retraction and concomitant neuronal cell death through an apoptotic pathway. Since our ultimate goal is to confirm these results in a mouse model of late onset progressive neurodegeneration through deregulation of ena/VASP family members, we characterized potential dominant negative (DN) versions to identify the most effective one. Ena/VASP proteins share three well-defined domains, the EVH1 domain, a central proline-rich region, and EVH2. Thus we analyzed the expression of EVH1-GFP, EVH2-GFP and GFP (control) in transfected NIH 3T3 cells. Both constructs delocalized VASP with variable efficiency, however none of them affected Mena localization. Preliminary results after DN overexpression in mouse hippocampal neurons led to neurite retraction, recreating what was observed with RNAi pools. Interestingly, EVH1 displayed the most potent inhibition compared to EVH2, suggesting the EVH1 domain is a good candidate to generate an inducible transgenic model.

Cellular and Molecular Neurobiology

**Poster Number (33) Session 2**

The role of IQGAP1 in spine morphogenesis

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IQGAP1 is a scaffold protein that interacts with many proteins involved in cytoskeletal dynamics. In our laboratory we explored how IQGAP1 regulates cytoskeletal dynamics and its function in neurons. In this study we examined the subcellular distribution of IQGAP1 and its functions in relation to the cell cytoskeleton regulation, especially focused on the actin cytoskeleton. Due to the great influence of IQGAP1 on actin cytoskeleton organization, we analyzed the involvement of this protein on the establishment of neuronal polarity, morphogenesis and spine morphogenesis. Our observations showed the localization of IQGAP1 in dendritic spines, as well as an active participation in spine morphogenesis. We demonstrated that the interaction domain with microfilaments is essential for the formation of the spine head. The interaction of IQGAP1 with small GTPases is necessary for the generation of the spine neck. Finally, we have shown that the carboxy-terminal domain of IQGAP1 has effect on the dendritic spines length. Moreover, we identified the Arp2/3 complex as a downstream effector of IQGAP1 in spine morphogenesis events and the suppression of IQGAP1 decreases the dendritic spines density in culture. Taken together, our results demonstrate the involvement of the different domains of IQGAP1 in physiological processes, and dendritic spines morphogenesis.

Cellular and Molecular Neurobiology

**Poster Number (34) Session 3**

Genetic basis of human brain evolution: functional analysis of NPAS3 human accelerated regions

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It has been hypothesized that the unparalleled explosion in human cognitive capacities and behavioral repertoire is due the acquisition of new temporal and spatial expression patterns of preexisting genes rather than changes in the protein-coding sequences. In order to study differences in gene regulation which may have contributed to the evolution of human brain we are functionally characterizing some of the most rapidly evolving human elements yet identified (termed human accelerated regions [HARs]). We focused on the largest cluster of HARs located within 648 kb of the neuronal PAS domain protein 3 (NPAS3) gene. Using a transposon-based transgenic assay in zebrafish we tested the ability of NPAS3-HARs to function as developmental enhancers. Additionally, we performed a comparative expression analysis in transgenic mice. Two human and mouse NPAS3-HARs ortholog sequences were able to drive reproducible patterns of reporter expression across three mouse developmental stages partially recapitulating

the endogenous Npas3 expression pattern. These findings suggest the participation of tested sequences in NPAS3 developmental expression. This approach is allowing us to analyze the role of human NPAS3-HARs in gene regulation and their potential role in human brain evolution.

Cellular and Molecular Neurobiology

**Poster Number (35) Session 1**

Angiotensin II AT-2 receptor mRNA is increased by hypoxic-preconditioning (HP) in the brain and cerebellum of the neonatal rat

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Angiotensin II, an octapeptide compromised with mechanisms of organ injury and repair acts through the membrane receptors AT1-R and AT2-R. In previous experiments we observed a transient increase of AT2-R protein in the brain of HP animals. We examined by RT-PCR the temporal expression of the AT2-R mRNA in the brain and cerebellum of rat pups after being submitted to Hypoxia-Ischemia (HI) on day8 after birth and treated with HP 24h before injury . On Day 1 after HI the HP group showed a significant decrease of the transcript ( $p<0.05$ ) in both brain hemispheres. On Day 2 after HI it was observed a remarkable increase of AT2-R mRNA in the lesioned side of the brains of HI and HP animals ( $P<0.01$ ), that returned to normal after a week. In the cerebellum significant increases on Day 2 ( $P<0.01$ ) persisted until day7 in the HP group ( $p<0.05$ , Anova-1- Bonferro-ni ). The inflammatory response to the injury was assessed by IHC and confocal images of GFAP+ and Vim+ astrocytes in the Corpus Callosum were obtained and quantified. HP animals showed less inflammation and no co-localization of AT2-R label with astrocyte markers. In conclusion: AT2-R is up-regulated in the brain in response to injury and HP stimulates its cerebellar genomic expression. ANPCYT. PICT2006-451.

Cellular and Molecular Neurobiology

**Poster Number (36) Session 2**

Passive transfer of IGG anti-GM1 antibodies impairs peripheral nerve repair

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Anti-GM1 antibodies are associated with an acute immune neuropathy called Guillain-Barré syndrome (GBS). Some clinical studies associate the presence of these antibodies with poor recovery in GBS. The patients with incomplete recovery have failure of nerve repair, particularly axon regeneration. Our previous work indicates that monoclonal antibodies can inhibit axon regeneration by engaging cell surface gangliosides (Lehmann & Lopez et al., J Neurosci 2007). We asked whether passive transfer of human anti-GM1 antibodies from patients with GBS modulate axon regeneration in an animal model. Human anti-GM1 antibodies were compared with other GM1 ligands, cholera toxin B subunit and a monoclonal anti-GM1 antibody. Our results show that patient derived anti-GM1 antibodies and cholera toxin beta subunit impair axon regeneration/repair after PNS injury in mice. Comparative studies indicated that the antibody/ligand-mediated inhibition of axon regeneration is dependent on antibody/ligand characteristics such as affinity-avidity and fine specificity. These data indicate that circulating immune effectors such as human autoantibodies, which are exogenous to the nervous system, can modulate axon regeneration/nerve repair in autoimmune neurological disorders such as GBS.

Cellular and Molecular Neurobiology

**Poster Number (37) Session 3**

Myelin associated glycoprotein protects neurons from excitotoxicity

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Myelin-associated glycoprotein (MAG) is a molecule present in the periaxonal membrane of myelin that contributes to long-term axon-myelin stability, enhances axon cytoskeleton structure, and contributes to the integrity of nodes of Ranvier. Recently, MAG was found to protect axons from acute toxic axonopathic insults. We report here that MAG protection extends beyond the axon to neurons from which they emanate. Primary hippocampal neurons plated on a surface adsorbed with myelin proteins from rats and wild type mice protected hippocampal neurons from KA-induced toxicity, whereas myelin from Mag-null mice did not. Myelin protection was also mimicked by molecularly expressed soluble MAG. MAG-mediated protection from excitotoxicity was dependent on Nogo receptors and 1 integrin, but not on sialoglycans. Two in vivo models confirmed a role for

MAG in protection against excitotoxicity. Mag-null mice were hypersensitive to KA-induced seizures, and had increased lesion volumes upon intrastratial injection of N-methyl-D-aspartate (NMDA). NMDA-induced lesion volumes were reduced by intrastratial pre-injection of soluble MAG. The in vivo findings confirm that MAG protects neurons from excitotoxicity and demonstrate the potential of MAG to mitigate excitotoxic damage.

Cellular and Molecular Neurobiology

**Poster Number (38) Session 1**

NPSAX: novel mRNA silencing foci that regulate local translation at the synapse

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XRN1 is a RNA binding protein with 5' 3' exoribonuclease activity present in all cell types. In almost all cases, XRN1 concentrates in discrete foci, termed Processing Bodies (PBs). We found that in neurons, XRN1 doesn't colocalize with PBs, but form discrete structures associated to the postsynapse, that we call NPSAXs (Non PB Synapse-associated XRN1). We found that 40 % of synapses contain NPSAXs. The NPSAXs are dynamic; they increase in number when mRNAs are released from polysomes and disassemble after prolonged treatment with a drug that interrupts translation. Moreover, the NPSAXs respond to distinct synaptic stimuli. NMDAR activation provokes an enhancement in NPSAX, whereas mGluR stimulation provokes their dissolution. In all cases, we found that the stability of the NPSAXs upon synaptic stimulation correlates inversely with polysome stability. This suggests that the NPSAXs function as mRNA silencing foci, releasing transcripts to allow their translation, or harboring transcripts that are not being translated. A recently described strategy termed FUNCAT, which allow monitoring protein synthesis in situ, we found that local translation at the synapse is stimulated by mGluR activity, whereas NMDAR stimulation decreases local translation.

Cellular and Molecular Neurobiology

**Poster Number (39) Session 2**

Protein kinase D1 (PKD1) role in neuronal development in vivo

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Axons and dendrites differ their cytoskeleton and membrane components as well as in their function (Craig & Banker, 1994). Previous studies of our laboratory (Bisbal et al., 2008) indicate that PKD1 regulates Golgi-derived vesicle sorting and directs dendritic membrane proteins transport. This work addresses new evidence on the role of PKD1 during in situ neuronal development. Using in utero electroporation (Saito, 2006) we transfected neurons in E14 mice intact brains. As soon as 24hs post-transfection control cells (GFP) recapitulate normal migration with a leading and trailing processes properly established. On the other hand, neurons expressing a kinase deficient and inactive form of PKD1 (PKD1-kd) remain at a multipolar stage with longer processes. 48hs after electroporation these effects are more evident; showing some cases of morphologically aberrant PKD1-kd expressing neurons. After 4 days of expressing GFP neurons reached their final position. In the PKD1-kd case neurons remained in intermediate positions, some exhibiting branched leading processes. In summary, PKD1 has an essential role in proper neuronal migration and morphology establishment. Ongoing work intends to clarify dendritic membrane proteins missorted responsible for our observations.

Cellular and Molecular Neurobiology

**Poster Number (40) Session 3**

Differences in GABAA receptor subunit composition and sensitivity to ethanol in male and female hypothalamic neurons

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Pharmacological and electrophysiological properties of GABAA receptors are dependent on subunit conformation. Previous results from our lab revealed that the response to muscimol is greater in male hypothalamic neurons than female ones. To search for a sexually dimorphic subunit constitution of GABAA receptors, we conducted whole-cell patch clamp recordings in 2 or 9 DIV neurons. Responses to different GABA concentrations as well as the percentage of potentiation or inhibition of 10  $\mu$ M GABA-elicited currents in the presence of allosteric modulators such as diazepam, alfaxalone, propofol, Zn, furosemide and ethanol were recorded. There were no statistical sex differences in those parameters. However, ethanol potentiated the GABAergic current only in female neurons. In addition, mRNA from 2 DIV sexually discriminated cultures was extracted and RT-PCR was performed in order to amplify GABAA receptor subunits. The analysis showed that most subunits were present in both sexes, but the  $\alpha$ 4 subunit was exclusive

for females. Our data confirm that GABAA receptor subunits in hypothalamic neurons from E16 embryos are different in males and females even before the hormonal sexual differentiation. Supported by ANPCyT PICT26331 and 0456, CONICET PIP2010, MCyTCBA PID2008 and NIH RO1 15015

Cellular and Molecular Neurobiology

**Poster Number (41) Session 1**

Frizzled-7, a potential WNT7B receptor, regulates dendrite Morphogenesis

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Wnt factors play a crucial role in the embryonic development of all animal species. In the nervous system, WNTs regulates neuronal connectivity by controlling axon pathfinding, dendrite morphogenesis and synapses. The morphogenesis of dendrites is regulated by genetic factors, neuronal activity, and external molecular cues. WNTs are secreted glycoproteins that signal through their Frizzled receptors (FZ) and Dishevelled (DVL). They can signal through three different pathways: WNT/ $\beta$ -catenin pathway, the planar cell polarity and the calcium pathway. We demonstrated that Wnt7b regulates dendritic morphogenesis. Thus, Wnt-DVL signals through Rac and JNK to regulate dendrite development. In this study, we go further and we try to identify the Wnt7b receptor involved in the dendritic effect. Binding assays on cell surfaces show that Wnt7b interacts with extracellular domain of Fz7 (CRD-Fz7). In addition, Fz7 expressing neurons develop complex dendritic arbours compared to controls. Neurons show a significant increase in dendritic length and complexity. Importantly, this effect is blocked when neurons express the CRD domain of Fz7, which function as a dominant negative. These evidences suggest that Fz7 may act as a potential transmembrana receptor of Wnt7b to regulate dendrite morphogenesis.

Cellular and Molecular Neurobiology

**Poster Number (42) Session 2**

Effect of culture conditions on the phenotype of adult neural stem cells from the SVZ

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Adult neural stem cells (aNSC) from the subventricular zone (SVZ) generate new neurons throughout life. They can be cultured as floating neurospheres, and can generate secondary spheres upon mechanical dissociation. Culture conditions affect aNSC differentiation rate. For example, TGF- $\beta$ 1 is pro-neurogenic on hippocampal aNSCs. In order to consider these cells for potential regenerative therapies is essential to develop efficient and reproducible culture protocols. We aimed to characterize the effect of different culture conditions on cell phenotypes of aNSC cultures from SVZ and to study whether TGF- $\beta$ 1 has a pro-neurogenic role. We generated neurospheres from adult rat SVZ, and performed immunocytochemistry with markers for aNSCs (Nestin), astrocytes (GFAP), and neurons (Tuj). TGF- $\beta$ 1 was added to these cultures for 5 days. Cultures maintained without mechanical dissociation showed a higher Nestin- and a lower Tuj-positive fraction when grown with EGF, while the GFAP-positive fraction was not altered. When dissociated, cultures showed an initial higher fraction of Tuj-positive cells. Upon EGF treatment, the Nestin-positive cell fraction was also increased, while Tuj- and GFAP-fractions did not vary. TGF- $\beta$ 1 treatment of dissociated cultures increased the Tuj-positive cell fraction. Thus, EGF increases aNSCs fraction in these cultures, while mechanical dissociation and TGF- $\beta$ 1 increase neuronal phenotypes. These factors may be powerful tools to increase a desired cell phenotype within aNSC populations.

Cellular and Molecular Neurobiology

**Poster Number (43) Session 3**

Participation of PKD1, LIMK1 and BARS in the formation of Golgi outpost in hippocampals neurons

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Participation of PKD1, LIMK1 and BARS in the formation of Golgi outpost in hippocampals neurons Quassollo G, ,Gastaldi L, Wojnaki J, Remedi M, Cáceres A. INIMEC – CONICET, Argentina. Neurons are cells highly polarized that present structural and functional asymmetries generated and supported by the cytoskeleton and by the selective, active and constant transport of materials from the Golgi complex to the final destinations, which are fundamental for the maintenance of cellular morphology and his correct functioning. In the majority of mammal cells, the Golgi complex consists of a stacked array of cisternae and connecting tubules/vesicles of peri nuclear location related to the microtúbulos organizing center. In neurons, this arrangement of membranes is partially different since not only

consists of a set of peri nuclear cisterns, but also of discreet structures distributed along dendrites, named Golgi outposts. These structures, demonstrates the existence of a new platform in the membrane traffic and segregation within dendrites. Given the importance of these new structures, we proposed to determine the involvement of PKD1, LIMK1 and BARS proteins in the generation and distribution of Golgi outposts in hippocampal neuron dendrites.

Cellular and Molecular Neurobiology

**Poster Number (44) Session 1**

Evidences of ischemic tolerance induced by a model of sleep apnea by intermittent hypoxia

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Sleep apnea (SA) patients show reduced mortality after an ischemic stroke. This clinical observation probably evidences an ischemic tolerance phenomenon. To test this hypothesis, we exposed adult rats or mice (wild type, XIAP<sup>-/-</sup> or NF-κB reporter) to an experimental model of SA by intermittent hypoxia (IH) by cycling oxygen level (6 min 21% + 6 min 10%; 3 days; 8 h/day), during the sleep phase (Hx group). Control animals were exposed to room air (Nx). A subgroup of animals was subjected to a focal brain ischemia and sacrificed 3 or 7 days post lesion (dpl) (Nx+I or Hx+I). Our results showed that Heat Shock Proteins (HSP) mRNAs were significantly increased in hippocampus of Hx animals. Apoptosis Inhibitor Proteins (c-IAP, XIAP) showed an increase in Hx animals. The activity of NF-κB was increased in Hx animals as shown by the NF-κB reporter transgenic mice and increased levels of IκB. Reduced number of altered neurons with atypical NeuN staining in the ischemic penumbra were observed in Hx+I compared with Nx+I group. Fluoro Jade B staining also showed less number of degenerating neurons in the Hx+I wt animals. XIAP<sup>-/-</sup> mice lost the protection to a subsequent ischemia induced by Hx exposure. We conclude that IH induces partial protection to a following ischemia, probably involving HSPs and NF-κB target genes specifically XIAP. Grants: CONICET PIP 1728, IBRO RHF; PICT 2008-1590

Effect of prenatal exposure to the cannabinoid receptor agonist  
WIN55,212-2 on postmitotic neuroblast during periods of cortical migration

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The endocannabinoid system plays a modulatory role in specific processes of brain development such as radial and tangential migration and cell proliferation. Neurons of the cerebral cortex derive from two sources: projection neurons, which migrate radially from the neuroepithelium of the dorsal pallium and interneurons, which migrate tangentially from the ganglionic eminence. Cajal-Retzius (CR) cells are the earliest generated cortical neurons and migrate from the borders of the developing pallium. Doublecortin is a microtubule-associated protein expressed in postmitotic neurons during periods of migration. The aim of this study was to assess the effects of prenatal exposure to cannabinoid CB1/CB2 receptor agonist WIN55,212-2 (WIN) on postmitotic neuroblast in the fetal cortex. Pregnant Wistar rats were treated, from gestational day 5 to 16, at a daily WIN dose (0.75mg/kg). WIN-exposed rats showed altered disposition and orientation of tangential and radial migrating postmitotic neuroblast. No changes were observed in number of proliferating cells in the ventricular zone. Prenatal exposure to WIN increase the number of CR cells in the marginal zone. The effects of cannabinoids on cortical development may be due to its effects on the generation and/or migration of neuroblasts.

Modulation of astroglial networks in the cerebral cortex of the rat

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Astrocytes play a key role in the maintenance of neural function and tissue homeostasis. They have the ability to sense and modulate synaptic activity, remove substances from extracellular clefts, supply metabolic fuel to neurons, and regulate ion-water balance. In contrast to mature neurons, a distinguishable feature of mature astrocytes is that their cytoplasm is intercommunicated through gap-

junction channels that allow the passage of physiologically relevant molecules. These astroglial networks constitute a putative scaffold for long range signaling pathways and distribution of substances within the neuropil. We report a preliminary description of astroglial networks in the cerebral cortex of the rat, as part of a study aimed at the characterization of factors that may be involved in their modulation. We estimated the magnitude of astroglial networks evaluating the spread of a small fluorescent dye (Lucifer Yellow), after its administration into single astrocytes during patch-clamp (whole-cell) recordings in acute brain slices. We measured the number of coupled cells and their spatial extent working on image stacks (Z-axis). Administrative and technical support : Lic. B. Stuto (CONICET), Mrs. C. Juárez (CONICET). Support: Fundación Conectar, FONCYT (PICT 0694), CONICET.

Cellular and Molecular Neurobiology

**Poster Number (47) Session 1**

Melatonin induces the mitochondrial apoptotic pathway in C6 glioma cells

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Melatonin (MLT) is an indoleamine synthesized by the pineal gland. In vitro and in vivo experiments have shown that MLT influences mitochondrial homeostasis. Tumor cells present higher levels of ROS and oxidative damage markers than normal cells. In the present work we demonstrated that MLT (2mM, 48hs) induced cell death both in the presence and in the absence of serum, measured by MTT assay (40±4% and 63±5% respectively). Nuclear morphology analysis employing Hoechst 33258 dye showed condensation and DNA fragmentation at 24 and 48hs. Furthermore, we studied the effect of MLT on Bcl-2 family proteins expression. MLT increased Bax expression and decreased Bid proform, in a dose-dependent manner. Bcl-2 and Bcl-xL levels were also investigated. Analysis of mitochondrial integrity employing MitoTracker Red dye showed the occurrence of different morphologies: normal (tubular) mitochondria in controls, and intermediate (partially fragmented) and fragmented mitochondria in MLT-treated cells, in both, the presence or absence of serum. ROS enhancement seems not to be the only event responsible of MLT-induced apoptosis. Our results suggest that MLT is capable of inducing cell death in glioma cells by activating the mitochondrial apoptotic pathway.



The role of bone marrow mononuclear and mesenchymal stem cells in the demyelination-remyelination process

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We have previously described, in a model of crushed sciatic nerve, the migration of CD34+ bone marrow mononuclear cells (BMMC) exclusively to the ipsilateral nerve. The aim of this work was to characterize BMMC and mesenchymal stem cells (MSC) to evaluate changes in their phenotype after transplantation. We studied the colocalization of BMMC with different markers and analyzed MSC capacity to acquire a SC-phenotype (SC-MS) in vitro. Dyed BMMC were injected intravenously after crushing the sciatic nerve, 5 days afterwards, we evaluated their phenotype by immunohistochemistry. Western Blot, immunocytochemistry and flow cytometry analysis were performed to characterize the cells. Results show that CD34+ cells represent approximately 5% of the BMMCs and 35% of the MSC. MSC also express CD90, p75NTR, ED1 and nestin. When MSC were incubated in a prodifferentiating medium, some cells expressed SC' markers, but the differentiation was not complete. Transplanted BMMCs, co-localized with S100 $\beta$ , ED1 and CD34 in the injured nerve. The results may suggest that BMMC might help to remove debris during demyelination period or stimulate remyelination by transdifferentiation to SC. Further experiments are necessary to elucidate mechanisms involved and to decide the best cell population for future therapies.

DMT1 & schwann cells differentiation. Evidence for a Tf-independent iron uptake

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Schwann cells (SCs) are responsible for the myelination of the PNS. Down regulation of myelin genes occurs after axonal contact loss. We have described the de-differentiation phenotype SCs acquired when cultured in a serum free medium; similar to that of SCs precursors and immature SCs. Holotransferrin (hTf) prevented this de-differentiation, while apotransferrin (aTf) did not. This prodifferentiating effect suggests that Fe/hTf are involved in the axonal signal that occurs during

the maturation of SCs in vivo. Whereas Tf-mediated Fe uptake is considered to be the primary route of Fe uptake in cells, there is evidence for Tf independent mechanisms. Here we verify the existence of a divalent metal transporter (DMT1) described as a key player in Fe metabolism literature, but never before in SCs. The presence of the protein was demonstrated in sciatic homogenate, adult-rat myelin and cultured SCs by WB analysis and confirmed through colocalization by immunocytochemistry. We verified the existence of its mRNA by RT-PCR in SCs precursors, immature and mature SCs and in a sciatic nerve submitted to crush. These data confirms the existence of a Tf independent Fe uptake mechanism in SCs, which lead us to speculate the key role of Fe in the axonal signal that allows SCs differentiation and survival.

Cellular and Molecular Neurobiology

**Poster Number (50) Session 1**

Müller glial cells preserve stem cell features in retinal progenitors and induce their differentiation as mature photoreceptors

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Müller glial cells (MGC) are stem cells in the vertebrate retina and might be used to replace neurons lost in neurodegenerative diseases. We have shown that rat retina progenitor round cells (PRC) in co-culture with MGC preserve their proliferative state, can be re-seeded and can then adopt a photoreceptor (PHR) fate in secondary co-cultures. Here we analyzed whether MGC preserved proliferative PRC in successive passages, and then induced these PRC to differentiation as mature PHRs. Flow cytometry analyses established that about 5% of total round cells (RC) in secondary co-cultures were PRC, which showed stem cell markers, such as 5-bromo-2-deoxyuridine uptake and Pax6 expression, even after four passages. MGC induced PRC in secondary co-cultures to differentiate as PHRs: RC expressed Tuj1 (an early neuronal marker) and Western blot analysis revealed acetylated tubulin (an axon marker) levels augmented with time in vitro. PHR-like cells expressed Crx (an early PHR marker), opsin and peripherin (a disc structural protein) and responded to light by diminishing their intracellular cGMP levels, evidencing they had an active phototransduction pathway. Our results suggest that MGC maintain and/or generate proliferative PRC and later instruct them to differentiate into functional PHRs.

Cellular and Molecular Neurobiology

**Poster Number (51) Session 2**

Characterization of a sub-lethal systemic dose of Shiga toxin 2 in the nervous system

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Neurological damage caused by intoxication with Shiga toxin (Stx) from enterohemorrhagic *Escherichia coli* is one of the most fatal outcomes of Hemolytic Uremic Syndrome in children. We have previously shown that intracerebroventricular (icv) administration of Stx2 damages rat brain neurons and astrocytes. The aim of this work was to determine the action of intraperitoneal (ip) Stx2 in the Nervous System. Mice brains and colons were processed to perform transmission electron microscopy (TEM) and fluorescence studies after 8 days of treatment. Apoptotic neurons and damaged astrocytes were observed in Stx2 (0,002 LD50) treated groups by TEM in the corpus striatum, cerebral cortex and hippocampus. This was not observed in controls. In addition apoptotic and degenerated oligodendrocytes, damaged endothelial cells with perivascular edema, synaptic inhibition and mitochondrial disorganization were observed. The preceded pathologic changes were not observed in the icv treated group. Significant neurodegeneration was confirmed by the FluoroJade-B method in the same brain areas and in the myenteric plexus. This study shows differences in brain damage between the ip and icv administrations of Stx2. Systemic elements activated by Stx2 may increase the damage found in the Nervous System.

Cellular and Molecular Neurobiology

**Poster Number (52) Session 3**

Involvement of sonic hedgehog protein on mesencephalic neural crest cells migration

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Neural crest cells (NCC) exhibit high ability to migrate through the vertebrate embryo's body, where they colonize and differentiate into many derivatives, such

as craniofacial tissues, pigment cells, and neurons and glia of peripheral nervous system. The known factors regulating the dispersion of NCC are not sufficient to fully explain the oriented migration of this cell population. We propose that the precise NCC distribution is modulated by chemotactic mechanisms driven by concentration gradients of soluble molecules secreted by target sites. Sonic Hedgehog (Shh) protein -playing essential roles in organogenesis and differentiation during the embryo development- could be involved in these mechanisms. In this work, we have investigated morphological, dynamic and chemotactic parameters of mesencephalic NCC exposed to gradients of Shh using a computerized video-microscopy approach, as well as the expression of Shh-receptors (Ptch, Smo) on in vitro NCC. Cell directionality or proportion of NCC oriented toward or against de Shh gradients depending on the initial concentration of Shh. Oriented migration toward Shh was observed when NCC were exposed to 5 µg/ml, 10 µg/ml and no-tochord conditioned medium (Shh source). The absolute speed of cell migration was significantly reduced in all exposures to Shh. In addition, mesencephalic NCC express receptors corresponding to Shh signaling pathway. [Previous results corroborated the expression of Shh in the optic vesicle field, a cephalic target region of NCC]. Our in vitro results suggest that Shh may be involved as an in vivo signal cue modulating the oriented migration of NCC toward a cephalic target site (optic vesicle), providing evidence of a new biological role for the Shh morphogen during early embryogenesis.

## \* Chronobiology

Chronobiology

**Poster Number (53) Session 1**

Output signaling in the circadian clock, evolutionary and functional conservation between flies and crabs

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Species that live in the intertidal zone are exposed to the 24-h light-dark cycle and to cyclic ebb and flow of tidal waters. Accordingly, both circadian clocks and biological clocks that oscillate in synchrony with tide, namely circatidal clocks, have been described in several intertidal organisms. Whether these two biological timing systems share common mechanisms remains unknown. We have previously identified two members of the pigment-dispersing hormone family in *C. productus*,  $\beta$ -PDH-I and  $\beta$ -PDH II. The distribution of  $\beta$ -pdh I-expressing cells in

the crab's brain is similar to that of PDF in *Drosophila*. Furthermore,  $\beta$ -PDH I shares closer sequence homology with PDF than PDH II does, and it is expressed in neurons that also show CYCLE-like staining in the crab's brain. These results suggest that  $\beta$ -PDH I may represent a functional homolog of PDF, which is a critical signal to drive circadian outputs and to couple the circadian network. To further investigate this possibility we transformed *Drosophila pdf01* flies by overexpressing either  $\beta$ -PDH I or  $\beta$ -PDH II. Whereas the circadian phenotype of  $\beta$ -pdh I-expressing flies was undistinguishable from wild type flies,  $\beta$ -pdh II could only accomplish a modest rescue of the phenotypes associated with the loss of PDF function.

Chronobiology

**Poster Number (54) Session 2**

A time for everything: from circadian to interval timing (ecclesiastes 3: 1)

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Temporal perception is fundamental to environmental adaptation. To deal with timing, organisms have developed multiple systems that are active over a wide range of magnitude, the most important being circadian, interval and millisecond timing. We tested the hypothesis that interval timing in mice is sensitive to circadian modulations. Animals were trained following the peak-interval (PI) procedure. Results show significant differences in the estimation of 24-second intervals at different times of day -with a higher accuracy during the night- which were maintained under constant dark (DD) conditions. In addition, under constant light (LL) conditions, which abolish circadian activity, mice were unable to acquire temporal control in the PI procedure. Moreover, short time estimation in animals subjected to circadian desynchronizations (jetlag-like situations) was also affected. Taken together, our results indicate that short-time estimation is modulated by the circadian clock. We are currently studying the circadian regulation of dopamine levels in the striatum, as well as the expression of clock genes -such as *Per2*- in pre-frontal cortex and basal ganglia. Preliminary results indicate daily variations of dopamine transporter (DAT) levels, which are eliminated under LL conditions.

Internal desynchronization in a model of chronic jetlag in mice

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The effects of an abrupt phase shift of the light/dark cycle on the internal synchrony of the circadian system are well known in the chronobiological literature. Frequent phase shifts probably have a much more disruptive effect on the circadian system; this “chronic jet-lag” (CJL) state has been shown to affect survival and morbidity. Here we present activity data of mice under one particular experimental CJL protocol which sheds light and provides a new direction for analyzing this complex state under a concrete mathematical basis. Locomotor activity of C57 mice was recorded while they were subjected to a CJL schedule consisting in 6h advances of lights onset every 2 days for 30-60 days. Under the chronic jet-lag condition, 10/15 of animals showed two distinct activity components: one with a short period of 21.0h and another with a long period of 24.9h. After being released into DD, animals displayed an intermediate activity period of 24.1h. Computer simulations were performed with coupled Pavlidis oscillators forced by 24h pulses that were shifted variously with respect to size and frequency. Frequent phase shift schedules can be globally interpreted as a new Zeitgeber which elicits relative coordination or even internal dissociation between circadian components.

NeuroD1 protein dynamics in the adult rat pineal gland

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NeuroD1 is a transcription factor involved in the differentiation and functional maintenance of specific endocrine and neuronal cells, including rodent pinealocytes [1]. Several affected transcripts were identified in neonatal pineal gland from NeuroD1 KO mice using microarray analysis [1]. In contrast to mRNA levels [1],

the NeuroD1 protein could exhibit circadian variations in the adult rat pineal. Via immunohistochemistry, we studied the expression of pineal NeuroD1 protein over a 12:12 L:D cycle. We observed a NeuroD1 signal in pinealocytes and vimentin-positive astrocytes. While NeuroD1 was cytoplasmic in all positive cells and nuclear in a few vimentin-positive cells during the light phase, at ZT14 NeuroD1 was found in both subcellular compartments in pinealocytes and astrocytes. During early night, the disruption of the circadian system via superior cervical ganglionectomy sequestered NeuroD1 into the pinealocyte cytoplasm. Furthermore, using phalloidin and an anti-actin antibody we observed abundant actin filaments at night vs. globular actin during day time. These results suggest that NeuroD1 might modulate adult pinealocyte functions in a circadian fashion; the actin cytoskeleton might be involved. Reference [1] Muñoz et al. 2007. J Neurochem 102(3):887-899.

Chronobiology

**Poster Number (57) Session 2**

Time awareness in drosophila: the use of memory for understanding how time turns into a cue

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Increasing evidence from different model systems suggests that the circadian clock is a pervasive biological phenomenon. It would be exciting to unveil how deep it goes into brain processes and which functions are affected. Thus, if certain brain structure is sensing time it is reasonable to assume it is controlled and/or affected by the circadian clock. The main goal of the project is to understand how animals are aware of time; and the basic idea is making use of memory to uncover this ability. Since several mechanisms are conserved through evolution, many features can be studied in an organism as the fly *Drosophila melanogaster* which turns to be ideal for exploring a potential connection between the circuitries that underlie circadian rhythms and memory since both were deeply investigated in the last 30 years. An initial approach will evaluate a) whether memory processes are modulated by the circadian clock and b) possible mechanisms for this effect. Besides, opening the exploration of the mechanisms that might underlie time awareness, we will study whether circadian clock mutants are sensing time differently than a wild type strain. Ideally, this project will lead to the understanding of how time becomes a cue, which brain structure is sensing it and which are the outcomes.

PDF shapes the architecture of a key circadian pacemaker circuit

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The neuropeptide pigment-dispersing factor (PDF) is key to synchronize and support molecular oscillations within the circadian pacemaker groups in *Drosophila*. It is expressed in the lateral ventral neurons (LNvs), including the small-LNvs (sLNvs) and the large-LNvs (lLNvs). The sLNvs project to the dorsal protocerebrum, where most of the other circadian clusters are located, and are indispensable for maintaining behavioral rhythmicity under constant conditions. PDF immunoreactivity cycles in these dorsal terminals, and this rhythmic release sets the phase of most clock neurons. We have previously shown that the complexity of the dorsal arborizations from the sLNvs change throughout the day in a circadian fashion, and proposed it could represent a mechanism to encode and transmit time of day information. To understand this phenomenon and its relationship with PDF function we evaluated the degree of structural plasticity in a PDF null. Surprisingly, the axonal projections of 1 or 2 sLNvs showed a miss-routing phenotype, which is also present in a PDF-receptor mutant. When we reduced PDF levels acutely at specific stages we found not only that PDF is necessary in a precise moment for the correct development of the sLNv projections but also for the correct morphology of the lLNvs.

Circadian regulation of cytoplasmic mRNA-granules

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Gene expression is modulated by clocks, 5-20% of the mRNAs expressed in a given tissue show daily oscillations. Presumably, this is largely generated by changes in the transcription of those genes; however, the relative importance of mRNA post-transcriptional processing has not been established. Stress granules (SG) and P-bodies are cytoplasmic subdomains involved in the regulation of translation, mRNA decay and storage. They are formed by RNA and a number of factors



involved in mRNA cytoplasmic processing. Considering that some of the SG and P-body components are expressed under circadian basis, we have analyzed whether these foci show rhythmic changes. NIH3T3 cells were synchronized by serum shock and fixed after 7, 14, 21, and 28 h. SG and P-bodies were detected by ICC. P-body per cell (number) showed fluctuations peaking 28 h after synchronization, and reaching highest areas 14 h after the shock. We applied arsenite (oxidative stress) for inducing SG. The number of SG/cell did not change through time; however the area and perimeter of them showed daily variations with peak 14 h after serum shock. Further studies will be carried out with other markers to confirm these results; however they suggest that these foci, or a subpopulation of them, are regulated by a circadian clock.

Chronobiology

**Poster Number (60) Session 2**

#### RGC-5 cells as non-visual photoreceptors and circadian oscillators

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In mammals, non-visual responses to light are still observed in the absence of functional rods and cones. These responses have been shown to be mediated by a subset of retinal ganglion cells (RGCs) which are intrinsically photoresponsive and express the novel photopigment melanopsin (Opn4). In addition, diverse visual and non-visual opsins have been recently detected in the mammalian inner retina. The aim of this work was to study the retinal ganglion cell line (RGC-5) as autonomous circadian oscillators and photosensitive cells. We measured in RGC-5 cultures: light responses on the induction of c-Fos expression and on Ca<sup>2+</sup> mobilization as well as expression of different visual/non-visual opsins. Our observations revealed that a brief white light pulse significantly induced c-Fos expression in the cultures as compared to dark controls whereas a subset of RGC-5 cells exhibits a light-increase in intracellular Ca<sup>2+</sup> levels. In addition, we found in RGC-5 cells detectable mRNA levels for several opsins (S/ML Opn1, Opn3, Opn5, RGR,) but not for Opn4 or rhodopsin. Also, we observed that RGC-5 cells express several clock proteins as well as the mRNA for the key enzyme in melatonin synthesis AA-NAT. The results strongly suggest that oscillators and photoreceptors converge in RGC-5 cells.

CGMP modulation by PDE inhibition enhance photic response of the circadian system

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The circadian clock in mammals is located in the hypothalamic suprachiasmatic nuclei (SCN), which are synchronized mainly by the light-dark (LD) cycle. Light pulses (LP) in the late subjective night induce phase advances in circadian rhythms and the expression of clock genes (such as *per*) by the activation of the NO-GC-cGMP-PKG pathway. Pharmacological manipulation of cGMP by phosphodiesterase (PDE) inhibition increases light-induced (L-I) circadian responses. Vardenafil, Tadalafil or Sildenafil significantly increased L-I phase advances as well as accelerated circadian reentrainment after jet-lag. Sildenafil also increased the phase-shifting effect of saturating LP in male or female hamsters as well as in mice, and was also effective after oral administration. This inhibitor also accelerated reentrainment of temperature circadian rhythms. We also show that the cGMP-dependent pathway converges in L-I clock gene expression in the SCN, since inhibition of PKG significantly decreased L-I *Per1* expression in the subjective night. In summary, our results demonstrate the involvement of cGMP and its related signaling pathway on light-induced circadian entrainment, and provide promising tools to design treatments aimed at human circadian disruption.

Keeping up with the worm's rhythm: a story of homology and circadian variability

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Circadian rhythms are driven by endogenous biological clocks and are synchronized to environmental cues. Chronobiological study of *Caenorhabditis elegans* provides fundamental information about the basis of circadian rhythmicity in eukaryotes, due to its ease of use and manipulations, as well as of availability

of genetic data and mutant strains. We have designed an automated system to track individual nematodes and found that, under constant conditions, the circadian period was  $23.9 \pm 0.40$  h, is temperature-compensated and can be entrained by light. We studied mutants of clock gene homologues and input-related genes and found that mutations in the period gene homologue induced altered circadian periods ( $25.26 \pm 0.4$  h for the *lin-42(mg152)* mutant). A genetic rescue of this mutation, the transgenic strain VELS26 (*lin-42::gfp*) recovers a normal circadian period. Other mutant strains (2G8, CS67, MQ130, KG1180, RB765, PR691) provide additional evidence for the molecular basis of circadian rhythms in *C. elegans*. Our results represent a complete description of the locomotor activity rhythm in *C. elegans*, with a methodology that allowed us to uncover three of the key features of circadian systems: entrainment, free-running and temperature compensation.

## \* Computational Neuroscience

Computational Neuroscience

**Poster Number (63) Session 1**

### Onomatopoeia between sound and word

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As a first step in the study of the nature of the relationship between words and natural sounds, in this work we identified the acoustic similarities between noises and their onomatopoeias. We built a database including noises and onomatopoeias for different languages and speakers. From this database, we 1. explored mimetic keys in onomatopoeias, both in the temporal and spectral domains, and 2. developed an algorithm that allows finding the anatomy of the vocal tract that best reproduce a given spectrum. Our results suggest that a bulk of mimetic information can be found in specific speech elements within the onomatopoeias. Specifically, the occlusive consonant [k] can be regarded as the basic mimetic element used in the onomatopoeias associated to knocks and clicks.

The role of intrinsic conductances on the neural code

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Neurons transduce spatio-temporal input signals into output spike trains. Different neurons are selective to different input structures, thus giving rise to different neural codes. Here we explore the role of intrinsic membrane currents in shaping the selectivity to specific input features. We simulate a conductance-based neuron model (Hodgkin-Huxley or Morris-Lecar) and systematically vary the maximal permeability to different ionic species. The goal is to determine the effect of such variations on the stimulus feature that is most effective in inducing firing. The preferred stimulus of each model is estimated with statistical methods. We drive the cells with a white-noise input current, and apply covariance analysis techniques to obtain the preferred stimulus. We find that increasing the conductance of fast, voltage-dependent depolarizing currents sharpens the selectivity to rapid, shallow stimuli. In contrast, increasing the conductance of voltage-dependent potassium channels, amplifies the neural response to high-amplitude, slowly modulated stimuli. In conclusion, the balance between the permeability to depolarizing and hyperpolarizing membrane currents forges the amplitude and frequency preference of a neuron.

Synergy and redundancy in the neural code

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Neurons and neural pathways transmit information. The neural code that instantiates information transmission, however, is still a matter of debate: Do different neurons cooperate to convey information or do they transmit redundant messages? Previous studies have determined that these two alternatives are not mutually exclusive, i.e. synergy and redundancy are not opposite concepts, and can therefore coexist. However, so far no attempt has been made to quantify them individually. Here, we present novel definitions of synergy and redundancy, and

compare them with previous approaches, that only quantified the net balance between the two. We show that they are complementary -- rather than opposite -- properties, and apply them to address the role of correlations in the neural code. We also derive upper and lower bounds for the amount of synergy and redundancy. We finally extend these concepts to the analysis of generalised neural codes, irrespective of whether they refer to different neurons, different pathways in the nervous system or different response aspects of a single neuron.

Computational Neuroscience  
**Poster Number (66) Session 1**

Evaluation of neural encoding schemes in multifiber activity using information theory: an application to the vibrissal system

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Currently, the vibrissal texture encoding is studied through the neural activity of one neuron (or a few neurons) of trigeminal ganglion and/or cortex. Here we analyzed various neural encoding schemes which could be related to texture sensing in anesthetized rats during active touch. The multifiber discharge from a deep vibrissal nerve during the sweeps different materials with similar degrees of roughness and sandpapers of different grain size were analyzed. Three neural encoding schemes were proposed: RMS scheme, cumulative event count (CEC) and inter-event time (IET). These events were detected by using an event detection algorithm based on multiscale decomposition of the signal. All encoding schemes were evaluated quantitatively through the Shannon's mutual information formula. The CEC scheme provides more information than RMS scheme. By using the IET scheme, the information values were higher than those obtained with RMS and CEC schemes. In conclusion, information of sandpaper roughness was found to be better encoded by IET scheme, while for surfaces of different materials, CEC scheme was optimal. Finally, in this study we have demonstrated (and quantified) the existence of two biologically plausible neural coding schemes based on temporal patterns of events.

Temporal and spectral patterns related to texture discrimination

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This work explores the existence of new temporal and spectral patterns related to texture neural code. For this, we analyzed the multifiber afferent discharge of one vibrissal nerve during active whisking in anesthetized rats. We used sandpapers of different grain size as roughness discrimination surfaces. We have proposed two new methods for afferent activity analysis: a Time-Frequency (TF) analysis and Inter-event Time (IET) analysis. The last analysis allowed us to relate the electrophysiological activity with the physical characteristics of friction surfaces. For this, we also obtained three parameters that characterized the roughness surfaces: arithmetical deviation of assessed profile (Ra), average maximum height of profile (Rz) and distance between local peaks (DBLP). The TF analysis shows that smooth textures evoke lower frequency in the multifiber activity than rougher texture when differences are deemed significant. The IET analysis allows us extracting temporal patterns or 'events' related to texture discrimination coding. Finally, we concluded that temporal patterns found in the multifiber activity of a single vibrissa innervation would be related with the texture coding and that the DBLP would be the better parameter described by these patterns.

Brain and peripheral correlates of decision making in rapid chess

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Rapid chess is an ideal framework to study human decision making in a controlled environment. The proliferation of chess servers on the Internet has turned active chess, blitz and lightning, into a vast cognitive phenomenon involving engaged participants. In a previous work, we generated a database of response times and

estimated position values (score) downloading millions of games from Free Internet Chess Server (FICS, [www.freechess.org](http://www.freechess.org)). Scores were estimated using Stockfish Engine ([www.stockfishchess.com](http://www.stockfishchess.com)). We found that RTs were not stationary and can not be generated by a state function which had theoretical implications since they deny two basic assumptions of sequential decision making algorithms. Here we present a novel experimental set-up to study brain correlates of chess decision making in chess players. Robust behavioral markers obtained from FICS database are used as markers for brain and peripheral physiological responses during rapid chess games. Using verbal reports of introspection after the game (e.g. blunder occurrence, creativity, difficulty), we will explore the association between these conscious reports and event related brain potentials, heart rate, pupil size and blood flow.

Computational Neuroscience

**Poster Number (69) Session 1**

[A neurocomputational model for estimating contours from motion](#)

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Multiple physiological and psychophysical studies show that stimuli outside the receptive field of a neuron in the Primary visual cortex can modulate its response when presented concurrently with stimuli inside the receptive field. We present a neurocomputational model that extracts the contours of a moving figure that are only defined by the accretion and deletion of its texture elements. The model has three components: 1) the signal sensing through the receptive field of simple cells; 2) the lateral inhibitory and facilitatory interconnections, spatially constrained by the laws of the association fields (Field, 1993); and 3) the temporal component of these lateral connections. This is instantiated through signals with a fast inhibitory followed by a slow facilitatory phase, which are spatially constrained by the orientation of the cell. We performed model simulations by using sequences of artificial images. Results of these simulations show that the spatial interactions are not sufficient to extract efficiently the contours from these moving images. The temporal stage seems critical for the success of the model in the reconstruction of the complete contour. Because there is no experimental evidence about this third stage, we performed a psychophysical experiment where we show that this temporal facilitation is plausible.

The neural code of sensory neurons in leech

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In leeches, N-cells are mechanosensory receptors that transduce noxious stimuli into action potentials. Previous studies have characterized the electrophysiological properties of these neurons, in response to constant stimuli. Here, we broaden those analyses by considering time-dependent stimuli, more similar to those encountered in natural conditions. The goal was to determine the temporal evolution of the stimulus that is most effective in inducing firing. In addition, we evaluated the precision of the responses, by measuring the trial-to-trial variability in the firing times. To that end, we stimulated the neurons injecting Gaussian currents into the soma, while recording the membrane potential. With these data, we calculated the shape of the typical stimulus preceding spiking, and the amount of jitter in the generated responses. The typical stimulus is a depolarizing current, with a marked upward deflection immediately before spike generation. The deflection consists of an ascending ramp, with a duration of 30 ms, preceded by a hyperpolarizing phase lasting for 20 ms. The typical amount of jitter was half the spike width. We conclude that the most effective stimulus has a well defined temporal evolution. It is surprisingly stable in this species, and triggers extremely accurate responses.

Role of prefrontal gabaergic interneurons in the control of dopamine-dependent working memory

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Dopamine (DA) transmission in the prefrontal cortical (PFC) is critical for sustaining cognitive task-related processes including working memory and decision making. However, the cellular mechanism that contributes to these functions re-



mains elusive. Here, we developed a realistic computational model of the PFC to determine how a dysregulation of the prefrontal GABAergic interneuron system impacts the overall PFC network stability and DA-dependent working memory. Our model is composed by a network of 2,000 neurons with an inhibitory/excitatory ratio of 0.2 and includes physiological features of DA action such as the D1-NMDA synergism on both pyramidal neurons and interneurons as well as the D2-dependent facilitation of inhibitory tone via activation of local fast-spiking interneurons. Our model predicts that rewarded behavioral outcomes are associated with an initial elevation of PFC DA, which is required to sustain interneuronal firing increase. In fact, synchronous interneuron activation by DA improves PFC network signal detection ratio by virtue of attenuating recurrent persistent activity on pyramidal neurons. In contrast, reward omission and incorrect behavioral responses decrease DA levels below baseline and interneuron excitability. Notably, degradation of both, the DA system and the GABAergic function elicit hyperexcitability of pyramidal neurons and functional deficits that perturbed the formation and retention of working memory. This deficit is accounted by the presence of distractor stimuli perceived. Together, these results indicate that the signal detection ratio of the PFC network is dependent on DA level and sustained by a critical GABAergic tone.

Computational Neuroscience

**Poster Number (72) Session 1**

On spike correlations and information encoding

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We applied an information theoretic approach to gain insights of the role of spike correlations in the neuronal code. We used a modified version of the information components breakdown to quantify the contribution of individual members of the population, the interaction between them, and the overall information encoded by the ensemble of neurons, making especial emphasis on the separation between contributions due to the noise and signal spike correlations. We present examples of applications of this formalism to simultaneous recordings of multiple spike trains and provide an estimate of the impact of higher order correlations.

A simple auditory model for pitch extraction

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We present a simple model of the auditory periphery for pitch extraction. This consists of a biophysical model of the basilar membrane as a first stage, followed by a spike generator with a noisy threshold. The spiking mechanism behaves in a similar fashion to more common Poisson models used in auditory models. Nevertheless, it endows two parameters: threshold and noise intensity, which resemble the ones used for auditory fiber classification, i.e. threshold level and spontaneous rate. The pitch values are extracted from first order inter-spike intervals histograms. We show how the model segregates the stimulus components in both time and frequency domain. In addition, the fundamental frequency, if missing, emerges in some fibers of the population.

**\* Motor Systems**

Motor Systems

**Poster Number (74) Session 1**

Physiologically driven electronic avian vocal organ

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Songbirds are the best-suited animal model to study general mechanisms underlying learned motor behavior responsible for complex output, as it is birdsong. Between the central motor pattern generator and the complex output stands a highly nonlinear biomechanical periphery. The avian vocal organ of the Zebra Finch (*Taeniopygia guttata*) is capable of generating a large variety of acoustic signals, which range from simple whistles to highly complex sounds. A mathematical model of the zebra finch syrinx is proposed, in which the onset of labial oscillations and the acoustic features of the sound are tuned by parameters accounting for the bird's motor patterns. We build an electronic birdsong synthesizer by implementing the model in a Digital Signal Processor (DSP). This device is capable of reading physiological recordings, integrating the mathematical equations

representing the avian vocal organ's behavior, and producing synthetic birdsong in real time.

Motor Systems

**Poster Number (75) Session 2**

Mechanisms influencing long-term memory during visuomotor adaptation

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It has been postulated that initially during adaptation to a visual perturbation, learning is based on sensory prediction error, whereas once error approaches the level of baseline, use-dependent learning may be necessary to consolidate the new sensorimotor map based on repetition. Both types of learning have been hypothesized to be necessary for long-term memory, measured as faster learning rate on re-exposure to the perturbation. The aim of this study is to identify the time course of motor memory consolidation up to five hours of adapting to a visual perturbation, using resting-state magnetic resonance imaging. Normal subjects moved a cursor from a start point to a target presented on a computer screen, using a joystick. A 30 degree optical rotation was applied to perturb the eye-hand coordination. Preliminary psychophysics data show a positive correlation between the rate of adaptation on the first exposure to the perturbation and the retention, and a negative correlation with the rate of adaptation on the second day, suggesting that fast learners show less long-term memory than slow learners. These results suggest that the amount spent in repeating the newly learned visuomotor map once adaptation is achieved, may determine whether there will be a long-term memory for the perturbation.

Motor Systems

**Poster Number (76) Session 3**

Enhanced intrinsic excitability of striatal cholinergic interneurons in a rat model of parkinson's disease

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An enhanced cholinergic tone has been regarded as a core feature of Parkinson's

disease pathophysiology. However, the involved mechanisms remain to be clarified. Whole-cell patch clamp recordings of striatal cholinergic interneurons have been performed in slices of control rats and rats having a nigrostriatal lesion induced with 6-hydroxydopamine (6-OHDA), under current and voltage-clamp conditions, at 32-34°C. Computer simulations were run in Neuron. Cholinergic interneurons fire two times more spikes during current depolarizing pulses in rats with nigrostriatal lesion than in age matched adult controls ( $p < 0.01$ ). This is mainly because cholinergic interneurons of control rats show a marked adaptation during the current pulse that is almost absent in 6-OHDA rats. Thus, the interspike intervals are two times longer at the end than at the beginning of the current pulse in controls, but do not change along the pulse in the parkinsonian condition ( $p < 0.01$ ). Voltage clamp experiments and computer simulations show that a barium sensitive delayed outward current could be responsible for firing adaptation and the differences between control and lesion rats. Thus, a reduction of an outward current mediating firing adaptation may underlie the hypercholinergic state in the parkinsonian condition.

Motor Systems

**Poster Number (77) Session 1**

Synthetic birdsong generation with physiological signal recorded on muted zebra finch

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Songbirds are a widely studied animal model for understanding the mechanisms that are involved in learning a complex motor behavior. The generation of bird-song is relevant because of the ability of birds to produce complex output by controlling a highly nonlinear biomechanical periphery (the syrinx) with simple motor instructions. In this regard, the study of the syrinx is an important task in the path to unveiling the mechanisms underlying the generation of motor patterns in the central nervous system. To study the adaptability of the motor pattern generator system, experiments with altered auditory feedback are performed. In our case a muted bird feeds with physiological signals recorded in real time an electronic device that emulates the syrinx. Physiological signals measured under these conditions differ quantitatively from those measured in the intact bird. This entails the difficulty of identifying the motor gestures with which the bird is trying to produce the song. In this work we aim to generate synthetic Zebra Finch song by identi-

ifying the motor gestures of a muted bird with those of the intact animal, in order to drive an electronic syrxinx.

## **\* Neural Circuit Physiology**

Neural Circuit Physiology

**Poster Number (78) Session 1**

Fictive crawling motor-pattern regulation by a nonspiking premotor neuron

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Leeches display a limited behavioral repertoire. Crawling is a behavior that results from successive phases of contraction and elongation of the animal's body. A fictive version of this behavior is induced by dopamine. The underlying neuronal circuit is unknown. NS neurons are a pair of nonspiking neurons electrically connected to virtually all motoneurons that act as electric modulators of the motor output. The aim of this work is to investigate the role of NS neurons in dopamine-induced crawling. To achieve this, we performed intra and extracellular recordings of neurons active during the contraction and elongation phases while manipulating NS membrane potential. NS hyperpolarization produced an increase in inter burst period, as well as a decrease in average instant frequency. When depolarizing current was injected, the average instant frequency increased, while the average inter burst period was unaffected. These results suggest that NS neurons are connected to the central pattern generator of the crawling motor pattern. This information will help in analyzing the circuit organization of crawling.

Neural Circuit Physiology

**Poster Number (79) Session 2**

Vascular inflammation and oxidative stress in experimental migraine

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Several studies support the role of endothelial dysfunction and oxidative stress

in migraine, however not known clearly how they relate with the headache. With objective to determine the participation of oxidative stress trigeminal level we compare, in rats, plasma level of fibrinogen (F), nitric oxide (NO) and L-Citrulline with possible histological changes in trigeminal ganglions (TGs). Vascular inflammation was induced by subcutaneous injection of adrenaline for 30 days. F (mg/dL), NO ( $\mu\text{M}$ ) and L-Citrulline ( $\mu\text{M}$ ) were determined by spectrophotometry. TGs were analyzed by optical and electron microscopy. F ( $451.4 \pm 20$ ) significantly increased compared with control ( $239.1 \pm 38.2$ ) ( $p < 0.0001$ ). NO levels ( $11.8 \pm 2.4$ ) and L-citrulline ( $1.35 \pm 0.07$ ) decreased significantly ( $22.7 \pm 2$ ) ( $p < 0.01$ ) and ( $3.10 \pm 0.18$ ) ( $p < 0.0001$ ). Distortion of the morphology was observed in TGs with loss of pseudo-unipolar in nerve cells and disruption of cristae in mitochondria with cloudy swelling in glial cells. The decreased levels of NO and L-Citrulline (markers of oxidative stress) with changes in neurons and glial GTs in a model of vascular inflammation, indicating that systemic vascular disease would be able to generate modifications trigeminal and allow the study of these experimental migraine mechanisms.

Neural Circuit Physiology  
**Poster Number (80) Session 3**

### FPGA implementation of a dynamic-clamp system

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The dynamic-clamp electrophysiological technique allows the mimicking of the electrical effects of ion channels, controlled by the experimentalist, activating and inactivating into the membrane of an intracellularly recorded cell. Dynamic clamp relies on the establishing of a loop between the injected current and the recorded membrane potential. In this work, a very-fast dynamic-clamp system was implemented on a Field Programmable Gate Array (FPGA). The FPGA chip is an integrated circuit that contains configurable logic blocks and programmable interconnect between those blocks. The architecture of the developed instrument is based on two main modules: a state machine and a signal processing module. The state machine controls the ADC and DAC converters, along with the serial data transfer interface. The signal processing module implements all the algorithms needed to mimic several HH-type ion channel conductances. Extensive simulations of the device operation were performed under realistic measurement conditions. These results have shown an excellent system performance. There are numerous applications of this instrument, including the manipulation of ion channels and of synaptic inputs to a cell, as well as the construction of a network in which a biological cell interacts with an electronic neuron.

Neural Circuit Physiology  
**Poster Number (81) Session 1**

Morphological characterization of neural circuits involved in visual processing in crabs

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In nature, animals need to sense information of their environment in order to produce an adaptative behaviour. Crabs are highly visual and reactive animals that offer important advantages for studying the physiology of animal behaviour. In spite of this, the anatomy of their nervous system has barely been studied. Crabs have well-developed compound eyes located on movable stalks. Inside the eyes, below the retina, there is the optic lobe that is composed of three serially arranged retinotopic optic ganglia and a fourth neural structure - called lateral protocerebrum - which comprises a conglomerate of delineated neuropiles regions that serve not only vision but also several other modalities. By applying crystals of fluorescent dextrans directly onto the second retinotopic neuropile we stained the neural projections from this ganglion to the third retinotopic neuropile and to the distinct neural nuclei of the lateral protocerebrum. Based on these stainings, we present here a preliminar map of the connectivity of second optic neuropile with downstream ganglia. This morphological study is the anatomical counterpart of ongoing and future studies on the physiology of vision with in vivo calcium imaging in the crab nervous system.

Neural Circuit Physiology  
**Poster Number (82) Session 2**

In vivo CA++ imaging responses to visual stimuli in columnar elements of the optic neuropiles of an arthropod

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Crabs are highly visual and reactive animals that offer important advantages for studying the physiology of animal behaviour. Here, we developed a preparation that allows assessing the response of neural visual elements to visual stimulation using Ca++ imaging in the intact animal. First, as the retinal maps of crab optic neuropils were unknown, based on detailed tracing from retinal photoreceptors

and neuronal projections along the optic neuropiles we disclosed the crab retinal maps. Second, to perform  $\text{Ca}^{++}$  imaging in the intact animal the crab was restrained and a small window was opened on the cuticle of the eyestalk. Through this window, columnar neurons were stained with dextran Calcium Green, a  $\text{Ca}^{++}$  sensitive dye. We found consistent and clear responses from narrow field columnar elements from the lobula to different visual stimuli. The analysis of correspondence between the areas of columnar activation and the positions of stimulus presentation confirm the retinal maps derived from the anatomical studies. Ongoing studies conclusively show that these anatomically peripheral elements are able to undergo plastic changes when confronted with repetitive presentation of a moving visual danger stimulus.

Neural Circuit Physiology  
**Poster Number (83) Session 3**

Role of long term depression in the postnatal maturation of corticostriatal circuits in vivo

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Goal directed behavior and habit formation depend on corticostriatal circuit operation. Neuropsychiatric disorders as obsessive-compulsive and attention deficit hyperactivity disorder (ADHD) might stem from corticostriatal circuit malfunction due to altered postnatal maturation processes. Our aim is to establish the contribution of long term depression (LTD) to the maturation of the core properties of adult striatal neurons such as low spontaneous firing rate and elevated signal to noise ratio. Our hypothesis is that LTD plays a dominant role during infancy and adolescence lessening the efficacy of corticostriatal synapses that are not recruited during learning. We induced LTD in vivo by a saturation protocol in young and adult mice with or without neonatal dopamine (DA) depleting lesion -proposed as an ADHD model. Preliminary results show that it is possible to induce LTD in young and adult lesioned mice but not in control adults indicating a maturative role for physiological LTD which would depress corticostriatal synapses. Such LTD would be DA-dependent because adult lesioned mice still show experimentally induced LTD. These results are consistent with previous findings of elevated striatal activity in adult mice with neonatal DA depletion suggesting a role for LTD in signal to noise regulation.



Serotonergic modulation in sensory-motor networks of the leech

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Serotonin (5-HT) is a highly conserved neurotransmitter that modulates complex behaviors in different species. The distribution of release sites relative to the targets could establish a hierarchy of actions that determines the final modulatory effect. In the leech *Hirudo* sp., we showed that the release of 5-HT is not uniform within the ganglion but occurs at least in a discrete site close to the limit between the lateral and medial anterior packets (Calviño & Szczupak 2008). In order to analyze which is the source of serotonin involved in the fluoxetine effect, we performed an anatomical analysis of the serotonergic neurons relative to this site. We iontophoretically loaded the neurons with tetramethylrhodamine dextran and we found that both Rz and interneurons 21 and 61 are possible candidates since they all project neuropilar processes to this site. Since the reproductive ganglia (G5 and G6) have morphological variations in some serotonergic neurons, we took advantage of this situation in order to study the role of these neurons. Based on experiments performed with fluoxetine, we know that 5-HT release occurs at sexual ganglia, but it could be due to an external source. Therefore, taking into account the trace of serotonergic projections coming from adjacent ganglia, at least in G6, neurons 21 and/or 61 are the 5-HT source.

Research project: linking glutamatergic and dopaminergic theories of schizophrenia

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For years, the dopaminergic hypothesis has dominated theories of schizophrenia. Recently, an alternative hypothesis proposes a cortical interneuron disturbance, due to NMDA receptor hypofunction, as a primary event in the physiopathology of the disorder. Supporting this theory, we have recently shown that ablation of NMDA receptors exclusively in corticolimbic interneurons of transgenic mice results in histological, behavioral and electrophysiological phenotypes compatible

with a schizophrenia-like syndrome. Several phenotypes observed in the mutants (like novelty-induced hyperactivity, and working memory deficits) may result from a deregulation of the dopaminergic system. This project is aimed to determine the functional impact of early postnatal interneuron manipulation on corticolimbic control of the dopaminergic system. We speculate that the increased cortical activity observed in adult mutants results in abnormal control of the ventral tegmental area (VTA). To test this we will quantify behavioral-induced changes in immediate early gene expression in the VTA, perform electrophysiological recordings to explore cortical-VTA connectivity, and conduct pharmacological manipulations. We expect to provide a link between glutamatergic and dopaminergic theories of schizophrenia.

Neural Circuit Physiology

**Poster Number (86) Session 3**

Mechanisms underlying pathological oscillations in the rat  
6-hydroxydopamine model of Parkinson's disease

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Patients with Parkinson's disease and rats with nigrostriatal lesion induced by 6-hydroxydopamine, a model of Parkinson's disease, show enhanced basal ganglia oscillations in synchrony with cortical oscillations. The mechanisms underlying pathological oscillatory synchronization remain obscure. Recordings were performed in control rats and rats with nigrostriatal lesion under urethane anesthesia. In one set of experiments we explored the effect of blocking striatal NMDA receptors with the competitive antagonist AP-5 (100-200uM) on oscillatory activity in the external globus pallidus (GPe). In another set we studied the short latency response of subthalamic (STN) neurons to cortical stimulation. GPe activity was not modified by intrastriatal AP-5 administration in control rats. The abnormal synchronization between GPe and cortical oscillations as estimated by coherence analysis, was reduced by 80% during intrastriatal AP-5 administration in rats with nigrostriatal lesion (p

Neural Circuit Physiology  
**Poster Number (87) Session 1**

In vivo study of converging corticostriatal circuitry and its modulation by nigrostriatal projections

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Our behavior depends on complex computations performed by parallel corticostriatal circuits. Classical interpretation of basal ganglia function has proposed that each parallel circuit is involved in different aspects of behavior: dorsolateral circuits in motor functions, dorsomedial circuits in cognitive functions and ventral circuits in limbic functions. However, functional and behavioral data suggest that each corticostriatal circuit takes over behavioral control during different phases of motor-skill learning and instrumental-behavior training, indicating a more complex interaction among these circuits. In addition to the parallel connectivity it has also been proposed the existence of “spiriling” striato-nigro-striatal connections that would link ventromedial with dorsolateral regions of the striatum through dopaminergic inputs. These connections would be necessary for the normal progression of initially goal-directed driven behavior to habitual instrumental responses after extended training. However, physiological evidence of such connections is lacking. By means of in vivo simultaneous electrophysiological multisite striatal recordings and multisite cortical stimulation, we provide a new framework for studying complex aspects of basal ganglia circuitry and its dopaminergic influence.

Neural Circuit Physiology  
**Poster Number (88) Session 2**

Phase precession on pyramidal cells depends on position and theta phase

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Theta (8Hz) and gamma (30-90 Hz) oscillations coordinate circuit dynamics in the hippocampus and are prominent during navigation. Pyramidal cell activity is coupled with theta rhythm, but every place cell displays a systematic phase shift in relationship with theta when the animal goes through its place field (phase precession -PP). In addition, different sets of cells are related to distinct gamma pha-

ses during exploration. Nonetheless, we know very little of how theta and gamma oscillations coordinate neuronal activity. Our analysis of pyramidal cell activity inside their place fields shows that the PP slope depends on theta phase. Action potentials (AP) at different theta phases display variable magnitudes of PP, with maximum PP for AP near the trough of theta, where the highest firing rate is also found. In addition, the relationship between PP's slope and theta phase depends on the portion of the place field where the cell is firing. AP in the ascending portion of theta have a higher PP in the first half of the place field compared with the second half. Finally, AP associated with different phases of theta showed different gamma phase preference. This observations extend our knowledge of the hippocampal CA1 local circuitry.

Neural Circuit Physiology

**Poster Number (89) Session 3**

A postsynaptic mechanism for dopamine modulation of prepulse inhibition in the goldfish startle circuit

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Startle is a protective behavior against predation, part of the flight/fight response and therefore crucial for survival. Although reflexive, startle is a plastic behavior. One modification of the auditory startle-reflex is prepulse inhibition (PPI), experimentally produced when a startling stimulus (pulse) is preceded within 20-500 ms by a subthreshold stimulus (prepulse) of the same or another modality. Several neurotransmitters modulate PPI in mammals. Notably, dopaminergic (DA) agonists as apomorphine reduce PPI in rats, while antipsychotic drugs as haloperidol reverse this effect. Despite an extensive work on different neurotransmitters effects on behavioral PPI, the neural substrates and cellular mechanisms of its modulation are still unclear. As homologous brainstem circuits mediate mammalian and teleost startles, we decided to investigate the DA influence on PPI in the goldfish primary startle circuit, where an identified pair of reticulospinal neurons (the M-cells) decide the likelihood, timing, and direction of startle. We show that apomorphine decreases M-cell PPI a 25%, while haloperidol restores it to control levels; the PPI reduction is restricted to a time window around 50ms and is mediated by enhancement of an excitatory nonlinear behavior of the M-cell membrane.

Neural Circuit Physiology  
**Poster Number (90) Session 1**

Neural networks involved in learning: interaction between prefrontal cortex and the noradrenergic locus coeruleus

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The locus coeruleus (LC) is a brainstem neuromodulatory nucleus and the sole source of noradrenaline to the cerebral, cerebellar, and hippocampal cortices. Initially it was associated with arousal and now is also known to play an important role in behavioural control processes such as: learning and memory, unexpected uncertainty, behavioural flexibility and neuronal plasticity. In previous work, we developed a computational theory to simulate learning of several tasks where noradrenergic neurons of the LC were included modulating neuronal excitability on visual and somatosensory cortical neurons. However, the dynamics of interaction between LC and cortical neuron populations is still unknown. Here we studied the interaction between these structures, analysing the temporal correlation of neurons firing. Ten extracellular electrophysiological recordings were made simultaneously in the Prefrontal Cortex (PFC) and the LC of anesthetized rats. Over fifty independent records were obtained, and after spike sorting analysis 358 neurons were identified: 138 from PFC and 220 from LC. Correlation analysis was performed in all possible pairs of PFC/LC neurons finding short term, long term and both kinds of interactions in some cases, in 1ms and 300ms windows.

Neural Circuit Physiology  
**Poster Number (91) Session 2**

Positive and negative interactions in the prefrontal cortex-ventral tegmental area circuit occur at different temporal scales

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Prefrontal Cortex (PFC) and Ventral Tegmental Area (VTA) are key brain regions in the reward neural circuit, being essential in behavioural flexibility, working me-

mory and planning. Despite its critical role, the PFC –VTA interaction from a circuital perspective is poorly studied. Extracellular recordings of neural populations were performed in the PFC and the VTA simultaneously, in order to characterize their dynamics and interaction. Correlation coefficient (CC) between PFC-VTA neuron pairs were computed for all temporal scales from 1 msec to 3 sec. We found significant correlations at short (hundreds of msec) and long (2-3 sec) time scales. 72% of all pairs showed positive CCs while 33% exhibited negative CCs. Positive and negative interactions differ in the distribution of scales at which they most frequently occur. 91 pairs (12%) were positively correlated in one scale and negatively in another. Surprisingly, when positive and negative CCs appear in the same pair, positive CCs occur in a faster time scale than negative CCs. Coexisting fast and slow dynamics may reflect underlying short and long term processes. Our results describe the PFC-VTA interaction as a multiscale phenomenon, allowing a better understanding of the role this circuit has in the neural circuit of reward.

Neural Circuit Physiology

**Poster Number (92) Session 3**

Cortical connectivity in epilepsy revealed by using network analysis

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Localization of cortical areas involved in the epileptic seizures' generation is perhaps the most important issue still to be resolved in the treatment of this neurological disease, which affects approximately to 1-2% of the world population. By using graph theory and complex network analysis over electrocorticographic data we were able to identify several areas of remarkable functional connectivity in the temporal lobe of epileptic patients. Highly synchronized and connected areas were quantified by means of classical tools as the clustering coefficient and betweenness centrality. The use of these measures allow us characterize and classify different types of functional connectivity in the temporal cortex. Moreover, these results were compared against presurgical studies, as SPECT and MRI and also against post-surgical outcome. In this way the significance of these critical areas were assessed. Grounded in the results presented here we propose and discuss a new possible mechanism of the seizure onset and evolution.

Neural Circuit Physiology  
**Poster Number (93) Session 1**

Newly generated neurons convey higher levels of associativity to information processing in the adult dentate gyrus

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Adult neurogenesis provides a constant pool of new granule cells (GCs) to the dentate gyrus (DG) of the hippocampus. During maturation, these GCs present higher excitability than mature neurons and require weaker inputs to be recruited in response to activation of afferent inputs. These properties suggest that while mature GCs are presumably more selective in their responses, immature GCs could be recruited by several inputs, allowing associations to occur. To study differences in associativity between mature and immature GCs, we combined imaging and electrophysiological recordings from hippocampal slices obtained from adult mice after retroviral expression of RFP to identify immature GCs. GCs activity was monitored by loading the cells with a calcium dye. Two electrodes were placed to stimulate independent medial perforant path inputs at different intensities. The results showed that immature GCs presented higher levels of associativity than mature GCs, and that these levels were independent of inhibition in immature neurons, yet incremented by blockade of inhibition with picrotoxin in mature neurons. The increased associativity observed in immature GCs suggests that this population of neurons could serve the DG to produce temporal associations.

Neural Circuit Physiology  
**Poster Number (94) Session 2**

Firing frequency adaptation in leech motoneurons

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Animal locomotion arises from muscle contraction and relaxation cycles generated by central networks. Motoneurons are essential elements in understanding locomotion control. The leech has four types of muscles implicated in locomotion: longitudinal, circular, dorsoventral and oblique muscles (lm, cm, dvm & om). The contraction of each of these muscles produces characteristic movements. All these muscles are innervated by a set of excitatory and inhibitory motoneurons.

Among the excitatory ones that innervates the lm are the dorsal excitor 3 (MNDE-3) and the L motoneuron (MNL), innervating the dorsal and the dorsal and ventral lms, respectively. We have investigated the firing properties of MNL and MNDE-3 with current pulses applied in the soma of those motoneurons with different time duration (1, 5 or 30 seconds). Both motoneurons showed a differential firing frequency adaptation (FFA). MNDE-3 was able to fire at higher frequencies than MNL and showed a strong FFA, while MNL reaches lower firing frequencies but can sustain its activity for prolonged periods. Upon prolonged pulses, MNDE-3 switched from a high to a low firing regime. We are analyzing the activity of these motoneurons in the course of fictive locomotive behaviors.

Neural Circuit Physiology

**Poster Number (95) Session 3**

Cellular interactions during maintenance and regeneration of the olfactory pathway

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We propose to study neuron-glia interactions during sensory neuron integration to the mouse olfactory circuit. Olfactory sensory neurons are produced in adult life, extend axons along the olfactory nerve and synapse in the central nervous system (CNS). The olfactory nerve permits axon growth, which is attributed to the presence specialized glia -the olfactory ensheathing cells or OECs- through unknown mechanisms. OECs are gap junction-coupled, which alters their biophysical properties, suggesting that OEC function depends on connectivity. In addition, sensory neuron activity elicits Ca<sup>2+</sup> transients in OECs and Ca<sup>2+</sup> modulates both gap junction permeability and the effects of OECs on axon growth. We hypothesize that sensory neurons modulate OEC gap junction coupling and this in turn regulates sensory neuron integration. Our goals are to: 1. Study the effect of sensory neuron activity on OEC connectivity, with electrophysiology in tissue slices. 2. Assess the need of OEC gap junctions for integration of new sensory neurons, with electrophysiology and immunohistochemistry, in transgenic mice with reduced levels of gap junction proteins (connexins). This study will help both to understand olfactory circuit formation, and to design therapies using OEC transplants for CNS repair.



Neural Circuit Physiology  
**Poster Number (96) Session 1**

Acute sodium depletion during adolescence: cross-sensitization with stimulating properties of amphetamine, but not with ethanol activating effects

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Alterations in body sodium balance early in life promote changes in subsequent sodium appetite. Moreover, repeated sodium depletions in adulthood sensitize both salt appetite and stimulating properties of amphetamine (AMPH). These behavioral changes are accompanied by morphological alterations in dopaminergic neurons of nucleus accumbens. Thus, it is possible to postulate that sodium appetite and drugs of abuse share neurochemical substrates and, due to this, cross-sensitization takes place. The present experiment aimed to study sensitization to AMPH and ethanol (EtOH) induced by one episode of sodium depletion in adolescence. Male rats (34 days-old) were administered with diuretic furosemide (10mg/kg). After a week of recovery, rats were injected either with AMPH (0.5 mg/kg), EtOH (2,5 g/kg) or vehicle and tested in open-field activity. AMPH stimulating activity was significantly augmented in rats previously sodium-depleted. Motor activity induced by EtOH was not affected by prior diuretic treatment. These data suggest that cross-sensitization between sodium loss and drugs of abuse depend on the neural mechanism involved in drug's effect. Interestingly, acute sodium depletion in adolescence can alter reactivity to a drug, suggesting neuroplasticity of the mesolimbic system.

Neural Circuit Physiology  
**Poster Number (97) Session 2**

Modifications of dopamine system components in operant conditioning learning in rats

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The ventral tegmental area (VTA) is considered the master controller of reward-dependent tasks. The VTA projects dopaminergic axons to the medial Prefrontal

Cortex (mPFC) and the hippocampus (Hipp). Here we studied modifications in the VTA-mPFC-Hipp circuit during learning of an operant conditioning task, in which animals must press a lever to obtain food. Experimental groups were: animals learning the task (IT, 50-65% of responses) and animals that completely learned the task (Tr, 100% of responses). Tyrosine hydroxylase (TH) levels were assayed in the VTA. TH expression was increased only in IT animals ( $p < 0.001$ ). Dopamine (DA) was measured in the mPFC and Hipp by HPLC. DA was augmented a 49% in the mPFC of IT animals ( $p < 0.001$ ), and a 25% in Tr group ( $p < 0.05$ ). No changes were observed in the Hipp. Finally, systemic and mPFC-specific administration of entacapone, a catechol-O-methyl transferase inhibitor (COMT) improved animal performance in the operant conditioning task, in the first session ( $p < 0.05$ ), as well as in the second and third sessions ( $p < 0.001$ ). All groups reached 100% of responses. Therefore, during learning an operant conditioning there are several modifications of the dopamine system components in the VTA-mPFC-Hipp circuit associated to different stages of the task.

Neural Circuit Physiology  
**Poster Number (98) Session 3**

Exploring the relationship between vagal tone and event-related potentials in response to an affective picture task

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The present study is the first to investigate the relationship between vagal tone level and event-related potentials ERPs. Numerous studies have shown a relationship between vagal tone and the individual differences between a variety of psychophysiological, affective, and social outcomes. This suggests that vagal tone can be related to how people process relevant affective social information at the brain level. This study aimed to assess whether the ERP response varies between high and low vagal tone groups, in the face of salient affective information. In the experimental cohort, two groups were separated according to their vagal tone level. ERPs were recorded while individuals performed an affective picture task that included positive, neutral, and negative emotional stimuli. Differences between the high and low vagal tone groups were observed at the early posterior negativity

for both positive and negative valences, and at the late positive potential for all the categories. It can be concluded that differences between high and low vagal tone levels are related to differences in the ERPs at early, middle, and late latencies. The results are discussed with respect to the effect of differences between the vagal tone conditions on various stages of information-processing.

Neural Circuit Physiology

**Poster Number (99) Session 1**

N400 deficits from semantic matching of pictures in probands and first-degree relatives from multiplex schizophrenia families

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Endophenotypes is one emerging strategy in schizophrenia research that is being used to identify the functional importance of genetically transmitted, brain-based deficits present in this disease. Several ERPs, including N400, present deficits in relation to schizophrenia. In order to assess the genetic liability of N400 as a possible endophenotype, a picture semantic matching task was performed by 21 unaffected first-degree relatives of patients with schizophrenia, 21 DSM-IV diagnosed schizophrenia probands, and 21 control subjects, matched by age, gender and educational level. Significantly reduced N400 amplitude for congruent categories in N400 was found in probands and relatives in relation to controls. The latency onset and the maximum peak latency of N400 were delayed in both, relatives and probands groups compared to control. The voltage maps of incongruous-minus-congruous difference indicate a more reduced right restricted negativity in probands and relatives, when compared to a widely extended bilateral negativity in controls. These results demonstrate an electrophysiological deficit in semantic match processing in clinically unaffected first-degree relatives of patients with schizophrenia, suggesting a possible use of this marker as endophenotype.

Characterization of low-threshold voltage-dependent calcium conductances of nonspiking neurons in leech

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The nonspiking neurons (NS) of the leech midbody ganglion function as premotor units, modifying the membrane potential of all the motoneurons, having a massive effect on the motor output. These neurons present an extensive neuritic arbor, they do not fire conventional action potentials, but they express low-threshold voltage-dependent  $\text{Ca}^{2+}$  conductances (VDCC) and TEA-sensitive  $\text{K}^{+}$  conductances. NS neurons respond to electrical stimulation with a spike-like event, which is a graded phenomenon that depends on the intensity and duration of the electrical stimulus. The spike-like response has a threshold of -55 mV, and its amplitude is a logarithmic function of the extracellular  $\text{Ca}^{2+}$  concentration. Our hypothesis proposes that NS VDCCs amplify and channel the active propagation of synaptic inputs. With the aim of generating tools to analyze how these conductances affect the synaptic integration in these neurons, we analyzed a) the pattern of inactivation of the VDCCs and b) the pharmacological profile. NS VDCCs are severely inactivated at potentials above -40 mV; but prolonged depolarizing pulses proved that this inactivation is very slow (beyond 10 s). NS VDCCs were not affected by Amiloride (300  $\mu\text{M}$ ), and  $\text{Mg}^{2+}$  (ratios over 4:1),  $\text{Ni}^{2+}$  (250  $\mu\text{M}$ ) and NNC 55-0396 (100  $\mu\text{M}$ ) blocked it.

Localization of the cardiac ganglion in chasmagnathus. Gabaergic regulation

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Decapod heart contains a cardiac ganglion that consists of a few neurons supplied by inhibitory and excitatory inputs from the central nervous system. Heartbeat is inhibited by the application of GABA and cardiac inhibition induced by inhibitory neural processes activation is blocked by picrotoxin. Cardio inhibitory processes show GABA immunoreactivity. Bradycardia or reversible heart arrests have been reported in decapods to a variety of stimuli. Our objective is to study the regu-

latory neural substrate of this inhibitory cardiac response in the crab *Chasmagnathus*. The aim is to localize the cardiac ganglion, identify probable regulatory neurotransmitters and record heart rate during training in the presence of GABA antagonists. We investigated the cardiac system in the crab by means of a latex vascular filling technique that allowed the delineation of the arterial system. Next, we localized the crab cardiac ganglion by histology, finding a discrete zone where a few neurons can be easily visualized. Further on we obtained GABA immunoreactivity in cardiac tissue. Finally the administration of picrotoxin weakened the heart arrest upon the presentation of visual stimuli. These results point to GABA as an extrinsic inhibitory neurotransmitter involved in this response.

Neural Circuit Physiology

**Poster Number (102) Session 1**

Sensory processing by a nonspiking neuron in the leech

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A pair of nonspiking neurons in the leech nervous system, the NS cells, is present in each midbody ganglion and displays a very extensive arborization. These neurons are electrically coupled to virtually every excitatory motoneuron and they are part of the interneuronal layer that mediates between mechanosensory and motor neurons. Given the wide influence of these neurons on effector neurons, it has been of interest to analyze how sensory input is integrated by the NS neurons. In spite of the fact that NS neurons do not display Na-dependent spikes, under certain circumstances they fire low threshold spikes (LTS) that reveal that these neurons exhibit voltage-dependent-Ca-conductances (VCC). Using fluorescent calcium probes we observed that these LTS caused a widespread increase in  $Ca^{++}$ . To learn whether VCCs in NS cells were also activated by synaptic input, mechanosensory P cells were electrically stimulated. We observed that depolarizing synaptic responses evoked  $Ca^{++}$  transient, whose magnitude correlated with the synaptic potentials. Upon strong depolarizing responses the  $Ca^{++}$  signals were observed in the four primary branches and the principal trunk of the NS neuron. The results suggest that VCC play a role in the propagation of synaptic responses throughout the branches of the NS neuron.

## **\* Neuroendocrinology and Neuroimmunology**

Neuroendocrinology and Neuroimmunology

**Poster Number (103) Session 1**

### Modulation of BDNF expression by MC4 receptor activation in rat astrocytes

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Melanocortins (ACTH,  $\alpha$ -,  $\beta$ - and  $\gamma$ -MSH) are potent anti-inflammatory neuropeptides. Melanocortin 4 receptor (MC4R) is the only MCR expressed in astrocytes and mediates anti-inflammatory action of melanocortins. Our previous results showed that  $\alpha$ -MSH, through MC4R, increases CREB activation in astrocytes. Since anti-inflammatory effects could be therapeutical, we studied the mechanisms involved in MC4R activation in cultured rat astrocytes. Since MCRs are G protein-coupled receptors we determined the intracellular levels of cAMP in astrocytes after 20 min of stimulation. In a dose response curve we found that  $\alpha$ -MSH 1 $\mu$ M elicited the highest increase in intracellular cAMP levels. This effect was blocked by HS024, a MC4R selective antagonist which per se decreased cAMP production. On the other hand, the inflammatory stimulus lipopolysaccharide and interferon- $\gamma$  (LPS+IFN- $\gamma$ ) decreased cAMP levels but co-incubation with  $\alpha$ -MSH reverted this effect. In addition, treatment with 0.1, 1, and 10  $\mu$ M NDP-MSH (an  $\alpha$ -MSH analogue) increased brain-derived neurotrophic factor (BDNF) mRNA levels at 1 or 3 h ( $p < 0.05$ ). At 24 h mRNA levels returned to basal values. However, NDP-MSH significantly increased protein levels of BDNF at 24 h ( $p < 0.05$ ). In summary, MC4Rs are functional receptors that increase BDNF expression in astrocytes, suggesting that BDNF could be a mediator of MC4R-mediated effects.

Neuroendocrinology and Neuroimmunology

**Poster Number (104) Session 2**

### Participation of sex chromosome complement in the dimorphic expression of brain aromatase

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The sexual differentiation of mammalian central nervous system is the result of gonadal steroid action during the critical period (E18-P10) of brain development. Conversion of androgens to estrogens by neural aromatase (ARO) is a prerequisite for brain sexual differentiation. Recent evidences have shown effects of sex chromosome complement (SCC) on sexually dimorphic neural phenotypes. The aim of this study is to evaluate the effect of sex chromosomes on brain ARO expression at E16. To this end, we used the four core genotype mouse model with a deletion of the Sry gene on the Y chromosome (Y-) and a Sry transgene inserted on an autosome. The resulting genotypes are XX and XY- females (ovary-bearing) along with XXSry and XY-Sry males (testes-bearing). Using specific anti-ARO antiserum, the regional distribution was immunohistochemically evaluated in brain sections of E16 mice. ARO-immunoreactivity (AROir) were found in the stria terminalis, anterior amygdaloid area, medial and central nuclei of the amygdala. Levels of AROir in the stria terminalis were higher in mice that possessed the SCC XY, regardless of their gonadal sex. These results suggest that the dimorphic AROir expression in this region is likely controlled mainly by SCC. CONICET y ANPCyT

Neuroendocrinology and Neuroimmunology

**Poster Number (105) Session 3**

Early maternal separation influence on glucocorticoid receptors in adult rats under amitriptyline treatment

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Early environment has many long-term effects on animals. It may affect the regulation the HPA axis during adulthood leading to a differential glucocorticoid secretion. Two types of nuclear receptors for corticosteroids were identified: mineralocorticoid receptors (MR) and glucocorticoid receptor (GR). They mediate in a coordinate manner the steroid control of HPA activity and behaviour. The signalling through GR and MR has an important role in the interaction between limbic system and HPA axis. It's been proposed that alteration in the balance between the expression and functioning of these receptors would be an important factor in the pathogenesis of stress related disorders. The aim of this work was to measure the expression of GR and MR in Hippocampus of adult Wistar Rats treated with antidepressant Amitriptyline (10 mg/KG), which were previously exposed to 4.5 h of maternal separation during the first 3 weeks of life. Maternal separation showed a tendency to reduce the expression of GR on CA1 and CA3 regions of hippocampus because of the aversive environment during development, and this appeared to be reverted by the treatment with amitriptyline during adulthood. This may res-

tore the regulation of HPA axis, which is consistent with previous results of corticosterone secretion in the same conditions. MR expression and GR:MR ratio were not altered by maternal separation.

Neuroendocrinology and Neuroimmunology

**Poster Number (106) Session 1**

GABA receptor plasticity in lymphocytes

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The plasticity of GABA receptors has been studied in the nervous system. Neurons can modify the number and composition of their receptors in response to different stimuli, such as exposure to GABA and nicotine. We have reported that human lymphocytes express a functional neuronal-like GABAergic system which inhibits lymphocyte proliferation. In this work we aimed to determine if this non-neuronal system also shows plasticity. To this end, we studied changes in the expression level of subunits of ionotropic GABA receptors from Jurkat cells, a human T-leukemic cell line. By RT-PCR, we determined that these cells express  $\alpha 1$ ,  $\beta 3$ ,  $\delta$ ,  $\gamma 2$  and  $\rho 2$  subunits. To study plasticity, we incubated the cells with 100  $\mu$ M GABA or 10  $\mu$ M nicotine for different time periods. While the expression level of  $\alpha 1$  subunit increases after 15 hs, it decreases after 40 hs of incubation with these drugs. The high level of expression at 15 h-incubation is also evidenced by the detection of GABA-elicited currents in 35% of the tested cells. In contrast, currents cannot be detected from cells incubated during 40 hs with GABA. We conclude that lymphocyte GABA receptors show plasticity as neuronal receptors; and this plasticity may play a role in the modulation of immune response.

Neuroendocrinology and Neuroimmunology

**Poster Number (107) Session 2**

The expression of estrogen receptor  $\alpha$  isoform on the adenohypophyseal cell surface is regulated by 17 $\beta$ -estradiol

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17 $\beta$ -estradiol (E2) activates membrane estrogen receptors (ER), but its identity remains controversial. We sought to identify ER in plasma membrane of pituitary cells and to establish if E2 modifies this subcellular localization. Pituitary cell cultures from female rats were treated with 10nM of E2 and ER $\alpha$  and  $\beta$  agonists (PPT, DPN) for 0, 5, 15 and 30min and with the inhibitors ICI182780, PD98059 and Gö6976. ER $\alpha$  and  $\beta$  were detected by confocal microscopy (CM, using Concanavalin A as a membrane marker), flow cytometry (FC) and electron microscopy (EM). Also, PKC $\alpha$  and ERK1/2 were evaluated by western blot (WB). Statistical analysis: ANOVA-Fisher. By CM, ER $\alpha$  and  $\beta$  were detected in the nucleus and cytosol of control cells, E2 or PPT for 5min increased ER $\alpha$  in plasma membrane, co-localizing with Concanavalin A. Only ER $\alpha$  was detected on cell surface with EM. By CF in all experimental conditions, 8% of lactotrophs expressed ER $\alpha$  on the surface, but after 5min of stimulation a 20% of these positive-ER $\alpha$  cells showed a higher intensity of immunostaining. By MC and WB, PKC and ERK1/2 activation was observed. These effects were reversed by the inhibitors. These results evidence the presence of functional ER $\alpha$  in the plasma membrane of pituitary cells and suggest that E2 increased its expression.

Neuroendocrinology and Neuroimmunology

**Poster Number (108) Session 3**

Immunohistochemical localization of estrogen receptor alpha in the cerebral cortex of the caiman latirostris (crocodylia: alligatoridae)

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In mammals estrogens has been described as modulators of neuronal differentiation and synapse formation. Caiman latirostris is a South American alligator that present temperature (TSD) and hormone dependent sex determination (HSD). In the present study we have studied the distribution of estrogen receptor alpha (ER $\alpha$ ) in the cortex of neonatal caimans. Three groups of caimans were studied: females and males obtained by temperature sex determination (females and males TSD) and caimans feminized by in ovo administration of estradiol (E2) (females HSD). ER $\alpha$  was detected in all cortical areas studied, however only the medial cortex (MC) showed sex differences. The intensity of ER $\alpha$  staining was significantly higher in TSD females compared to males ( $p < 0.05$ ). Interestingly, HSD females showed the maximum amount of ER $\alpha$  immunostaining in the MC when compared with both TSD groups ( $p < 0.05$ ). In addition, the level of E2 was significantly higher in TSD females than TSD males

( $p < 0.05$ ). Double immunostaining showed that ER $\alpha$  is expressed in neural precursor cells and mature neurons. These observations demonstrate that the expression of ER $\alpha$  in the caiman cortex is sexually dimorphic and is present in early stages of neuronal differentiation.

Neuroendocrinology and Neuroimmunology

**Poster Number (109) Session 1**

Age-related changes in hippocampal estrogen receptor alpha in female mice

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A good amount of evidence indicates that estrogens modulate hippocampal activity, an area of brain related to cognition and memory, however little is known about the molecular mechanisms that regulate the expression of its mediators, the estrogen receptors (ERs). In the present study, to reveal the effects of normal aging on ER $\alpha$  expression in the hippocampus, we examined ER $\alpha$  expression using young (4 month, control group) and adult (11 months) intact CF-1 female mice. By microsurgery complete hippocampus were extracted, which were individually processed. The expression of ER $\alpha$  mRNA and relative transcriptional activity of promoters A, C and F were evaluated by real time RT-PCR. Adult mice showed a significant decrease in the expression of ER $\alpha$  total mRNA when compared with young controls ( $p < 0.05$ ). In considering the relative activity of each of the ER $\alpha$  gene promoters, a significant decrease was observed in the activity of promoters A and C ( $p < 0.001$ ) in adult animals, while F promoter relative activity remained unchanged ( $p = 0.99$ ). These results demonstrate that aging is associated with a decrease in the expression of ER $\alpha$  in the hippocampus. This down-regulation would be mediated, at least in part, by a decreased activity of promoters A and C.

Peripheral but not central chronic inflammatory stimulus, exacerbates central demyelination in an innate inflammatory model

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Multiple sclerosis (MS) is a neurodegenerative disease characterized by repeated demyelinating events. Remyelination occurs in the early stages, but it fails as the disease progresses. Interleukin-1B (IL-1B) has a dual role in MS: it is present in active MS lesions, but it also has potential as a pro-remyelinating and neuroprotective factor. We have developed an inflammatory rat model of reversible demyelination, by administering adenoviral vectors expressing IL-1B (AdIL-1B). Our hypothesis is that repeated central or systemic pro-inflammatory stimuli exacerbate previous demyelination due to a central demyelinating stimulus. To test our hypothesis, we re-administered AdIL-1B either in the striatum or i.v. Animals which received a systemic injection of Ad-IL-1B 30 days after the first injection (remyelination phase) showed exacerbated inflammation and demyelination, along with microglial and astroglial activation. However, if the second stimulus was administered centrally we observed lower inflammation and demyelination compared to the animals receiving a unique central stimulus. In conclusion, only peripheral IL-1B exacerbates the effects of the central lesion. Peripheral inflammation has a highly significant influence on central on-going inflammatory and demyelinating lesions.

Quantitative peptidomic for the study of neuroendocrinological activation after feeding in the insect rhodnius prolixus, vector of chagas disease

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In triatomine insects, vectors of Chagas Disease, blood feeding stimulates events associated with grow and development, as much as a rapid diuresis during which the transmission of the parasite *Tripanosoma cruzi* occurs. All these events are regulated by neuropeptides. The insect neuropeptidome is a promising target for a

novel generation of insecticides that offer improved selectivity and environmental compatibility. The understanding of quantitative modification of the neuropeptide set in response to feeding provides information for the development of molecules capable of disrupting the physiological processes regulated by feeding, in order to replace conventional insecticides. We performed quantitative peptidomic experiments, using nano-liquid chromatography and mass spectrometry, after dimethyl labeling of samples, which allows triplex labeling. We compared concentration of specific neuropeptides in *R. prolixus* central nervous system at three different physiological conditions (starved, 4 hs and 24 hs after feeding). We observed significant changes in the levels of Leucokinins, Neuropeptide-like precursor 1, Prohormone 2, Prohormone 3 and Short Neuropeptide F, suggesting the involvement of these molecules in the regulation of processes triggered by feeding.

Neuroendocrinology and Neuroimmunology

**Poster Number (112) Session 1**

Candida albicans infection in central nervous system (CNS):  
histopathological finding and glial reactivity

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Candida infection in CNS is a serious form of bloodstream infection with 50% of mortality. With the aim to explore the ethiopathogenic mechanisms in this mycosis, we developed an In Vivo model in C57Bl/6 mouse. The animals were injected iv with 5.105, 1.106 or 2,5.106 viable *C.albicans* and at 12, 24, 48 and 72h the brain was removed. CNS yeast infection was confirmed by CFU and fungal morphotypes were visualized in the lesions(PAS). The local inflammatory reaction was characterized by meningitis and microabscesses distributed in the brain parenchyma(HE). The immunostaining with anti-GFAP(astrocytes As marker), revealed the presence of reactive astrogliosis(IF). Interestingly we detected neuronal cell death associated to the infection (FJB/Ag-stain). We also developed an In Vitro model to explore the immune reactivity of glial cells against *C.albicans*. FACS analysis of primary cultures revealed the follow composition: As enriched culture >96% GFAP+ cells vs 80% vs M

Influences of natural variations in maternal care upon plasma oxytocin and hippocampal monoamine activity in neonatal rats

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In the first 2 weeks the rats are susceptible to environmental events, such as the quality of maternal care (MC). It was investigated plasma oxytocin (pOT), central monoaminergic (MA) activity in neonatal rats and the possible influences of MC received. At the end of 1st postpartum day (P0), 60 Wistar dams had their litters culled to 8 pups. From P1 to P10, dams had their MC analysed, in which licking behaviour was taken as a measurement of the MC. Dams showing high and low frequencies of licking were selected as High Licking (HL) and Low Licking (LL), respectively. At P13, a pair of pups was removed from the litter and immediately sacrificed. Blood trunk was taken for pOT RIA. The hippocampi (HP) were dissected for MAs and their metabolites HPLC analysis. The three-way ANOVA with Duncan Post Hoc indicated that HL male and female pups showed higher levels of pOT (p

Oxytocinergic system involvement during states of body hypertonicity

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Our previous results indicate that both isotonic and hypertonic (HI) volume expansion (VE) increase the plasma oxytocin (OT) concentration and the activity of hypothalamic oxytocinergic neurons in order to increase renal excretion and re-establish extracellular volume. According to this evidence, in a more recent work we have shown activation of the oxytocinergic system after hypertonic NaCl intake (but not isotonic); however this consumption also produced a small change in the extracellular VE. Taken together, these results provide further support to the idea that the presence of both stimuli simultaneously, VE and HI, are able to increase

the OT system activity. The aim of the present work was to study the OT system involvement during a hypertonic sodium overload (HSO) that doesn't induce any extracellular volume expansion. For that purpose, we analyzed the number of brain Fos-OT-immunoreactive neurons, and the renal, endocrine and behavioural responses during a s.c. HSO, without changes in the extracellular volume (measured by plasma protein concentration). Our results indicate that HSO significantly increased the OT plasma concentration and the activity of the OT supraoptic neurons. Confirming previous result the HSO increased significantly the water intake and the renal excretion. We conclude that a body hypertonic state itself without involving VE can increase the activity of OT neurons and the plasma OT release, possibly to modulate sodium/water intake/excretion and avoid an extracellular VE.

Neuroendocrinology and Neuroimmunology

**Poster Number (115) Session 1**

Long term continuous environmental enrichment decreases estrogen receptor alpha expression in the female mouse hippocampus

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Estrogens are neurotrophic factors that modulate synaptic plasticity and neurogenesis. In the hippocampus these actions are mediated by the estrogen receptor alpha (ERα). In the present study, we examined the effects of living in a long term continuous enriched environment on the expression of ERα in the female mouse hippocampus. Enrichment consisted of social interaction, stimulation of exploratory behaviour and physical activity. One group of female mice (n=12) was exposed to a continuous enriched environment during 9 months, while controls (n=12) were kept in a standard environment. All animals were sacrificed at the same age (11 months-old). By microsurgery complete hippocampus were extracted and individually processed. The expression of ERα mRNA and the relative transcriptional activity of promoters A, C and F were evaluated by real time RT-PCR. Enriched mice showed a significant decrease in the expression of ERα mRNA (p 0.05) on the relative activity of each of the ERα gene promoters. These results show that a long term continuous enriched environment produced a down-regulation of the expression of ERα in the hippocampus of female mice, that was not mediated by a differential promoter usage.

TLR-4 signaling pathway mediates LPS-induced proliferation of hyperplastic lactotrophs cells

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Intrapituitary cytokines production stimulated by lipopolysaccharide (LPS) activates HPA axis during pathological events. LPS is recognized by the TLR-4, which is involved in cancer progression. Previously we showed that LPS-induced lactotroph proliferation in estrogen-stimulated hyperplastic glands in vitro. In this work we studied TLR-4 presence on lactotrophs and the intracellular mechanism involved in response to LPS. Male rats were treated with or without estradiol benzoate for 60 days. Then, anterior pituitary gland were cultured and stimulated for 30min with LPS (100ng/ml). Statistic analysis: ANOVA-Fisher Test. TLR-4 localization in hyperplastic lactotrophs was confirmed for the first time by flow cytometry and confocal microscopy. Also, we observed a significant increase in TLR-4/CD14 expression, Akt and Erk1/2 activation and NF- $\kappa$ B nuclear translocation in response to LPS by western blot. Although, TLR-4 was found in normal lactotrophs, BrdU incorporation revealed no effects of LPS on these cells proliferation. Concluding, hyperplastic lactotrophs respond to LPS by activating TLR-4/CD14, Akt, Erk1/2 and NF $\kappa$ B to elicit cell proliferation. Estrogenic priming might influence lactotroph sensitivity to LPS, promoting proliferative injury.

Aggressive behavior and reproductive physiology in females of the social cichlid fish cichlasoma dimerus

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Circulating androgens are potent mediators of male and female aggressive behavior. Aggression and androgen levels have been strongly associated with do-

minance over conspecifics. The GnRH-3 neuronal population is located in the olfactory bulb of several vertebrate species and it is related with reproductive behavior. We studied the body coloration pattern and described and quantified the aggressive and reproductive behavior of *C. dimerus* adult females of during four stages of the reproductive phase: pre-spawning, spawning, larvae-hatching stage and free-swimming larvae stage. To study physiological parameters of the brain-pituitary-gonadal axis in these four stages, adult couples (N=20) were kept under constant temperature and a 14:10 hs photoperiod. Females were weighted and measured; plasmatic androgens, 17- estradiol and cortisol levels were measured by RIA. Liver, spleen and gonadosomatic index were also calculated. We study the histomorphology of the ovaries. The GnRH-3 neural population was studied by IHC and morphometrical analysis was done. Results showed that pre-spawning is the most aggressive stage in terms of the quantified interactions; androgen and estradiol levels are higher also at this stage. Cortisol levels are higher at the spawning stage. Taken together these results suggest that GnRH-3 may regulate the reproductive physiology and behavior of *C. dimerus*, and that androgens are related with the aggressive behavior of females.

Neuroendocrinology and Neuroimmunology

**Poster Number (118) Session 1**

The pineal complex structure of the social cichlid fish *cichlasoma dimerus*: photoperiodic modulation of reproductive physiology

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In teleosts, the pineal organ is a functional, non-image forming photoreceptive structure that transduces photoperiodic information into neural and neurohormonal messages. The neurohormonal signal is melatonin, a time-keeping molecule released during the night time. The pineal organ is an elongated structure with a stalk and an expanded end-vesicle, with an internal lumen and a parenchyma conformed by an epithelium containing pinealocytes. In this study, we described the structure of the pineal complex and identify photoreceptor and melatonin-secreting cells in this species. Photoreceptor cells were identified using cones and rods antisera and melatonin producing cells with serotonin and HIOMT antisera. To study the effect of photoperiod on the brain-pituitary-gonadal axis, adult animals were placed in individual tanks either in long (14L:10D) or short (8L:16D) photoperiods for 4 weeks (n=6 per sex and photoperiod). Animals were weighted, measured and sacrificed. Liver, spleen and gonadosomatic indexes were calculated.



ted. PRL, GH, SL,  $\beta$ -FSH and  $\beta$ -LH pituitary content was semiquantified by Western blot. The morphology of GnRH-3 neural populations was studied by IHC techniques. At the brain level we demonstrated that GnRH3 neuronal optical density of staining was reduced. At the pituitary level we showed that SP males showed lower levels of  $\beta$ -LH, PRL and GH in the pituitary. Taken all together these results suggest that in *C. dimerus* the photoperiod is a relevant environmental cue related to reproductive physiology.

## **★ Neurochemistry and Neuropharmacology**

Neurochemistry and Neuropharmacology

**Poster Number (119) Session 1**

### New insights into the mechanism of activation of Cys-loop receptors

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Cys-loop receptors are pentameric neurotransmitter-gated ion channels that contain an extracellular domain, which carries the binding sites, and a trans-membrane domain, which forms the ion channel. The transition zone between these domains, called coupling region, is essential for coupling agonist binding to the opening of the channel. To understand this process, we used the chimeric 7-5HT3A receptor as a model of homomeric Cys-loop receptors. We constructed a subunit with a mutation that disables the coupling region and a reporter mutation that alters unitary conductance, and co-expressed mutant and non-mutant subunits. By measuring single-channel currents we determined that the amplitude of each opening event reports the number of subunits with intact coupling regions in the pentameric receptor. We found that: i) the coupling region of each subunit contributes additively to channel lifetime of the receptor; ii) channel opening is profoundly impaired in receptors containing only one active interface; and iii) for channel gating each functional binding site requires direct connection to an active interface, indicating that changes at a binding site are confined to the same subunit. Our results provide a framework for defining mechanisms of activation for these receptors.

Dissociation of environmental conditioning to hyperactivity and drug expectancy in response to stimuli formerly paired with cocaine in mice

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We tested the hypothesis that conditioned activity phenomenon (CA) is independently influenced not only by a Pavlovian conditioning between the observation environment and the locomotor activating effect of the drug but also by a locomotor stimulant effect induced by reward expectancy. An ineffective dose (10 mg/kg, i.p.) of cocaine in inducing open-field hyperactivity in female mice was used. Mice repeatedly treated with cocaine before (C-EXP-S) or after (S-EXP-C) open-field exposure presented a gradual enhancement of open-field locomotion frequency (LO), as well as increased LO responses when challenged with saline or cocaine, as compared to the control group (S-EXP-S). CA and behavioral sensitization (BS) observed in the S-EXP-C group were not due to context independent sensitization since non-exposed animals with the same drug history did not develop these phenomena. In addition, the S-EXP-C association markedly potentiated the duration of BS. In conclusion, an environmental conditioning to the locomotor stimulant effect of cocaine is not necessary for the development of CA. Thus, an expectancy learning is probably developed due to an association between a previous or simultaneous exposure to an environment and cocaine pharmacological effects.

Endothelins and cell proliferation in the subventricular zone

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We have proposed that endothelins (ETs) are involved in regulation of prolifera-

tion and differentiation of adult neural precursor cells (NPCs). Thus, we evaluated changes on proliferation and NPC markers after blockade of the cell cycle or inhibition of endothelinergic receptors. Male C57Bl/6 mice were implanted with osmotic minipumps delivering cytosine arabinoside (AraC, 2% in 0.9% saline, flow rate 0.5  $\mu$ l/hr), tezosentan (TEZ, 0.025% in 0.9% saline, flow rate 0.5  $\mu$ l/hr), or their vehicles for 7 days. Mice received 5-bromo-2'-deoxyuridine (BrdU) on day 6. Brains were processed for immunohistochemistry with antibodies against BrdU, ETs and glial fibrillary acidic protein (GFAP). AraC eliminates rapidly dividing cells, leaving the slowly cycling true stem cells. After AraC, BrdU+ nuclei were not detected in the subventricular zone (SVZ). ET immunoreactivity was decreased, but appeared in cells showing GFAP and the interependymal processes characteristic of neural stem cells. TEZ blocking of endothelinergic receptors reduced BrdU+ nuclei in the SVZ, but increased BrdU+ nuclei along the migratory stream. We can conclude that ET+/GFAP+ cells remaining along the ventricular walls would represent true stem cells. ET would also be present in amplification cells, disappearing after AraC. TEZ-induced changes suggested that endothelinergic receptor activation might be necessary for division of true neural stems, whereas this activation would elicit the opposite response on neural amplification cells.

Neurochemistry and Neuropharmacology

**Poster Number (122) Session 1**

Head shakes and hyperlocomotion induced by DOI, a serotonergic 5-HT<sub>2A</sub> agonist, as behavioral tools to study potential atypical antipsychotic actions

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There is considerable interest in the development of new antipsychotics drugs, particularly with 5-HT<sub>2A</sub>/D<sub>2</sub> receptors mechanism involved in the atypical antipsychotics (AADs). We proposed that the behavioral effects induced by DOI could be used as an in vivo procedure for detecting potential AADs. We study the ability of Clozapine (Clz), which presents higher affinity to 5-HT<sub>2A</sub> than to D<sub>2</sub> receptors, to antagonize DOI-induced behavioral responses. DOI was injected in mice; Clz was administered before DOI. Amphetamine (AMPH) was included in the study. Locomotor activity was registered automatically (15 min) in an Open Field paradigm associated to the software Motor Behavioral Monitor. Head shakes (HS) were measured by direct observation. DOI increased the number of HS and also induced hyperlocomotion (HL). Clz blockaded HS without changing the HL. A locomotion increase was obtained after AMPH injection and Clz antagonized it. Our results indicate that DOI produced two behavioral responses at the same time that

were selectively blockaded by Clz according to its affinity for 5-HT<sub>2A</sub>/D<sub>2</sub> receptors. AMPH-HL confirmed that Clz is also capable to blockade a DA response. DOI behavioral effects could be used as a tool to determine AADs actions depending of 5-HT<sub>2A</sub>/D<sub>2</sub> receptor affinity.

Neurochemistry and Neuropharmacology  
**Poster Number (123) Session 2**

Modulation of GABAC receptors by reactive oxygen species

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Reactive oxygen species (ROS), such as hydrogen peroxide (H<sub>2</sub>O<sub>2</sub>), hydroxyl radical (OH $\cdot$ ) and superoxide anion (O<sub>2</sub> $\cdot$ -) are generated as by products of the cellular oxidative metabolism and secondary to the activation of NMDA and AMPA receptors. ROS have been implicated in normal aging and in neurodegenerative disorders including Parkinson and Alzheimer's disease and ischemia-reperfusion injury. Numerous neurotransmitter systems are modulated by ROS, including dopaminergic, serotonergic, adrenergic and GABAergic receptors. Even though ROS effects on GABA<sub>A</sub> receptor-mediated synaptic transmission has been reported, the modulation of GABAC receptors by these species has not been determined. The aim of the present study was to analyze if GABAC receptors can be regulated by ROS. Homomeric GABA<sub>A</sub> 1 receptors were expressed in *Xenopus laevis* oocytes and GABA-evoked (0.31  $\mu$ M) chloride currents were recorded by two-electrode voltage-clamp in the presence or absence of H<sub>2</sub>O<sub>2</sub> (1 mM). Preliminary results showed that the application of H<sub>2</sub>O<sub>2</sub> significantly potentiated GABA-evoked currents. H<sub>2</sub>O<sub>2</sub> effects were reversibly, voltage independent and strongly depended on GABA concentration. These results suggest that GABAC receptors can be modulated by ROS. The mechanisms of action are currently under study.

Neurochemistry and Neuropharmacology  
**Poster Number (124) Session 3**

The anthelmintic agent oxantel is a partial agonist of C. Elegans muscle levamisole-sensitive nicotinic receptor

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The availability of a sequenced genome with 60% of the genes having vertebrate counterparts has led to the adoption of *C. elegans* as a model of neuronal diseases and is used in drug screenings. *C. elegans* expresses two types of muscle nicotinic receptors (AChRs): levamisole-sensitive (L-AChR) and nicotine-sensitive (N-AChR). Using a primary culture method that allows in vitro differentiation of embryonic cells into muscle cells, we explore activation of *C. elegans* AChRs by the nematocide drug oxantel. Patch-clamp recordings showed that oxantel elicits single-channel openings of ~3.7 pA at -100 mV, similar to those of levamisole-activated nAChRs, indicating that activity corresponds to L-AChRs. Oxantel-activated channels are 2- and 3.7-fold briefer compared to ACh and levamisole respectively, and the opening frequency is 20-fold lower than that of ACh-activated channels. Macroscopic responses elicited by oxantel are

Neurochemistry and Neuropharmacology

**Poster Number (125) Session 1**

Memory consolidation and reconsolidation of an inhibitory avoidance task in mice are impaired by acquisition / consolidation of a novel task

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When subjects are exposed to new experiences, the information acquired could be stored and eventually produce behavioral modifications. Once acquired, the information needs to be progressively strengthened over time through a consolidation process during which memory is stabilized. If the information processing were perturbed while this consolidation process is taking place, the storage could be affected and, as a consequence, the formation of the memory trace could be either enhanced or impaired. The exposure of mice to a novel environment (a hole board, for example) after being trained on an inhibitory avoidance task, impairs not only consolidation, but also reconsolidation of the IA memory only if the novel context is perceived as new, but not if it is recognized as previously explored. The nature of the interference remains unknown. The impairing effect of the novel task was related to the duration of the exposition to it, and blockade of its acquisition by using a pre-training administration of scopolamine, allowed consolidation and reconsolidation of the first task. In this work, we present evidence that acquisition/consolidation of the novel task is necessary to cause the interference.

Ascorbic acid is a positive modulator of  $\alpha 9\alpha 10$  nicotinic cholinergic receptors

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Activity of inhibitory efferent cholinergic neurons projecting from the brainstem and synapsing cochlear hair cells containing  $\alpha 9\alpha 10$  nicotinic receptors, can ameliorate acoustic trauma. Consequently, compounds that increase  $\alpha 9\alpha 10$ -mediated responses have a potential therapeutic use in noise-induced hearing loss. We have analyzed the effect of ascorbic acid (ASC) in  $\alpha 9\alpha 10$  injected *X. laevis* oocytes by two-electrode voltage-clamp recordings. Responses to 10  $\mu$ M acetylcholine (ACh) were potentiated by ASC in a concentration-dependent manner (0.1-30mM ASC). At 3mM ASC, an  $81 \pm 6\%$  ( $n=7$ ) potentiation was observed. Potentiation was more pronounced at lower ( $305 \pm 40\%$ , 3  $\mu$ M ACh,  $n=8$ ) than at higher ( $138 \pm 35\%$ , 1mM ACh,  $n=8$ ) ACh concentrations. No significant changes in the half maximal concentration of ACh and Hill coefficients were observed at 3mM ASC ( $EC_{50} = 18 \pm 1$ ,  $nHill = 0.8 \pm 0.1$ ,  $n=5-9$ ). The ASC oxidized form, dihydroascorbate, had no effect on ACh-evoked responses at a 3mM concentration, suggesting that the reduced form of ASC is the active compound. Altogether, our results show that ASC potentiates  $\alpha 9\alpha 10$ -mediated responses and thus has a potential therapeutic use in noise-induced hearing loss.

Caged serotonin for visible-light photodelivery

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Caged compounds of a molecule to be photoreleased are important tools for neurophysiology and other areas of biological research. The possibility to deliver precise amounts of biomolecules, in this case, neurotransmitters, by means of a focused light spot in a small area of living tissue allows the researcher to substitute the invasive current injection or picosyringe-based techniques. Since late 2008, we

selected the biomolecule Serotonin (5HT) for the synthesis of a caged compound. The complex synthesized is Ru(bpy)<sub>2</sub>PMe<sub>3</sub>Serotonin, a ruthenium atom coordinating two bipyridines (bpy), a tuning ligand (PMe<sub>3</sub>), and Serotonin. The product obtained was characterized by RMN and UV-Vis spectrometry. Electrophysiological experiments were conducted in leech ganglia to confirm at the biological level the performance of the photoreleased serotonin. The records obtained are consistent with those of direct stimulation with free serotonin in solution by more traditional methods.

Neurochemistry and Neuropharmacology

**Poster Number (128) Session 1**

Histidine 141 is critical for GABA<sub>A</sub> receptor sensitivity to ascorbic acid

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Neurotransmitter receptors, including ionotropic GABA receptors, can be regulated by redox mechanisms. We demonstrated that homomeric  $\rho 1$  GABA<sub>A</sub> receptors (GABA<sub>A</sub>1R) are modulated by ascorbic acid (Asc) and other redox agents (Calero and Calvo, 2008). Asc modulation was partially explained by a chemical modification of the SH-groups at two extracellular cysteines forming the cys-loop (C177 and C191) of GABA<sub>A</sub>1R. However, we also showed an additional allosteric mechanism involved. Histidine 141 (H141) is essential for allosteric effects induced by Zn<sup>2+</sup> on GABA<sub>A</sub>1R. In order to determine if this aminoacidic residue also participates in the mechanism of Asc modulation, we used site-directed mutagenesis. Wild type and mutant receptors were expressed in *X. laevis* oocytes and GABA-evoked Cl<sup>-</sup> currents electrophysiologically recorded. Maximal responses mediated by H141D GABA<sub>A</sub>1R were significantly potentiated by 3 mM Asc, as observed for wt GABA<sub>A</sub>1R. However, Asc was unable to produce shifts in the EC<sub>50</sub> of D-R curves of receptors carrying the mutation. Our results suggest that H141 is involved in the allosteric effects evoked by Asc on GABA<sub>A</sub>1R.

In vivo photorelease of GABA in the mouse cortex

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Caged compounds are a useful tool for spatially and temporally controlled release of a molecule with biological activity. They consist roughly of two parts: a protecting group and a bioactive ligand. A union that can be broken with light joins both parts of the complex. By breaking this bond with light of the appropriate wavelength, the ligand recovers its activity. These compounds have been tested in several biological models and, based on the results, several modifications were made in order to increase their biocompatibility. The next step was to find a way to use these compounds in vivo, which presents the challenge of finding a way to photorelease once the complex is inside the animal's body. To do this, we have designed experiments with mice that include a conventional surgery to irradiate the cerebral cortex in order to photorelease GABA. After surgery, the mice were placed in a dark box and had the local field potentials (LFP) recorded. Once the complex was in contact with the cerebral cortex, laser pulses were delivered by a 4mW 473nm-wavelength DPDSS laser in order to release the caged compounds in mouse's brain. Through these experiments we have successfully inactivated cortical neurons using a blue light pulse.

Neuroprotection by nicotinic agonism in a Parkinson's disease model: changes in the iron metabolism

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Parkinson's disease (PD) is characterized by motor impairment associated to a progressive degeneration of dopaminergic neurons in the substantia nigra. Oxidative stress, in particular an imbalance in iron (Fe) homeostasis, plays an impor-



tant role in the pathophysiology of PD. Epidemiological studies show that tobacco smokers have about 50% lower incidence of PD. Experimental evidence suggests that this beneficial effect is mediated by nicotine, however the underlying mechanism is still unknown. To determine if the protection induced by nicotine is mediated by changes in the metabolism of Fe, we exposed rat mesencephalic primary cultures to rotenone as a PD model. In this paradigm, rotenone reduces tyrosine hydroxylase-positive neurons survival, while a pretreatment with nicotine significantly protects the cells. A non-specific antagonist of the nicotinic receptor reversed this beneficial effect. Exposure of cells to nicotine at a neuroprotective dose causes a decrease in the labile iron pool, suggesting that the mechanism underlying the neuroprotection by nicotine could be to reduce the availability of iron to participate in oxidative reactions. In fact, a treatment with an iron chelator prevented cell death after rotenone damage.

Neurochemistry and Neuropharmacology

**Poster Number (131) Session 1**

Synapsin phosphorylation and glutamate release regulation by the GABAergic system in frontal cortex synaptosomes from rats with experimental autoimmune encephalomyelitis

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Recently we found that the glutamate release and synapsin I phosphorylation were decreased in synaptosomes from rat cerebral cortex during the development of experimental autoimmune encephalomyelitis (EAE), the animal model of multiple sclerosis. In order to evaluate the events that may affect neuronal function in EAE synaptosomes, we analyzed the modulation of the GABAergic system on the glutamate release and synapsin phosphorylation, and the flunitrazepam sensitive-GABAA receptor density. We describe alterations of the GABAergic system of frontal cortex synaptosomes from EAE animals. In contrast to control animals where GABA induced a decrease in the glutamate release, which was abolished by picrotoxin, synaptosomes from EAE rats showed a loss in the inhibition of the glutamate release mediated by GABA. Furthermore, the flunitrazepam sensitive-GABAA receptor density was decreased during the acute stage of the disease in synaptosomes from EAE rats. On the other hand, it was studied if in EAE cortical synap-

tosomes GABA inhibited synapsin I phosphorylation via  $\text{Ca}^{2+}$ /calmodulin, which in turn decreased the glutamate release. The changes observed in the GABAergic inhibition were reverted in cortical synaptosomes from recovered EAE animals. These results suggest that the diminution of the flunitrazepam sensitive-GABA<sub>A</sub> receptor density could explain the observed failure of the GABAergic regulation on the glutamate release of synaptosomes from EAE rats, and could contribute to clinical symptoms and disease progression.

Neurochemistry and Neuropharmacology

**Poster Number (132) Session 2**

A novel mechanism of modulation of 5-HT<sub>3A</sub> receptors by Hydrocortisone

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Modulation of Cys-loop receptors by steroids is of physiological and therapeutic relevance. Nonetheless, its molecular mechanism has not been elucidated for 5-HT<sub>3</sub> receptors. We deciphered the mechanism of action of hydrocortisone (HC) at 5-HT<sub>3A</sub>Rs. Single-channel currents from the high conductance form (74.7 pA, -70 mV) appear as a series of long opening events forming bursts, which group into long clusters. Though very infrequently, subconductance events (2.4 pA) are detected within clusters. HC produces a concentration-dependent reduction in open and burst durations, evidencing open-channel block. In addition, it increases in a concentration- and slightly voltage-dependent manner the appearance of subconductance events. Dual effects are distinguished from macroscopic currents: HC reduces amplitude by acting from either open or closed states and increases decay rates from the open state. Thus, HC acts as a negative modulator of 5-HT<sub>3A</sub>R by different mechanisms: It acts as an open-channel blocker and it favors opening to a pre-existing subconductance level. The latter constitutes a novel mechanism of channel modulation.

Neurochemistry and Neuropharmacology

**Poster Number (133) Session 3**

Chronic exposure to paraquat induces cortical and striatal mitochondrial dysfunction

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Paraquat (PQ) is a redox cycling agent capable of generating reactive oxygen species and oxidative stress. It has been described as a potential environmental neurotoxin associated with neurodegenerative diseases. As mitochondrial dysfunction has been implicated in neurodegenerative diseases, the aim of this work was to evaluate the effect of chronic administration of PQ on cortical and striatal mitochondrial function. SD female rats received paraquat (10 mg/Kg i.p.) or saline once a week during a month. Results: State 4 oxygen consumption was increased by 55 and 74 % and state 3 oxygen consumption was decreased by 12% and 33% in cortical and striatal mitochondria from treated animals respectively. In addition, respiratory control of mitochondria from both brain areas decreased after PQ treatment. Evaluation of hydrogen peroxide production by cortical and striatal mitochondria from treated animals showed a significant increase of 13 and 48 % respectively. Also, PQ treatment induced mitochondrial depolarization in both brain areas. This work shows that chronic exposure to PQ induces cortical and striatal mitochondrial dysfunction that could be associated with first steps of neurodegenerative diseases.

Neurochemistry and Neuropharmacology

**Poster Number (134) Session 1**

Pregabalin modulation of calcium channels and neurotransmitter release

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In this work we studied the effects of the anticonvulsant and analgesic drug pregabalin (PGB) on excitatory postsynaptic currents (EPSCs) at principal neurons of the mouse Medial Nucleus of the Trapezoid Body (MNTB), as well as on presynaptic calcium currents (IpCa) at the calyx of Held. We found that the acute application of PGB reduced the amplitude of EPSCs in a dose-dependent manner with a maximal blocking effect of about 30%. A clinical high-concentration dose of PGB (e.g., 500  $\mu$ M) blocked CaV2.1 channel-mediated currents and decreased their facilitation during 100 Hz train, without changing their voltage-dependent activation. Furthermore, PGB also favored the recovery of CaV2.1 channels after inactivation at a clinically relevant low concentration of 100  $\mu$ M. These results suggest novel modulatory mechanisms mediated by the acute administration of PGB on fast excitatory synaptic transmission and might contribute to better understanding PGB anticonvulsant/analgesic clinical effects.

The armed spider toxin TX3-3 restores the analgesic effect of morphine in opioid- and neuropathic hyperalgesic state

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Neuropathic pain is somewhat resistant to alleviation by morphine. Besides, neuropathic hyperalgesia and development of opioid tolerance share similar mechanisms within the spinal cord. Here, we tested the effect of the voltage dependent calcium channels (VDCC) blocker, Tx3-3 in morphine analgesia in opioid-tolerant and neuropathic hyperalgesic states. Neuropathic pain was achieved as described by Malmberg et al. (Pain 76:215;1998). Opioid tolerance was induced according to Marshall et al. (Nature 234:223;1971). After neuropathy induction (7 days) or repeated morphine administration, male and female adult Swiss mice received Tx3-3 (30 pmol/site) or the calcium/calmodulin-dependent protein kinase II (CaMKII) inhibitor KN62 (10 nmol/site) by intrathecal (it) route 15 min before intraperitoneal (ip) morphine injection (10 mg/kg). Antinociception was assessed by tail-flick test. Experimental model of opioid tolerance and neuropathy caused hyperalgesia resistant to morphine. Spinal injection of Tx3-3 or KN62 reestablished the morphine effect in tolerant- and allowed morphine analgesia in neuropathic mice. These data show that the blockade of VDCC and, ultimately, the lack of CaMKII activation enable morphine analgesia in hyperalgesic states usually unresponsive to opioids.

New kids on the block: caged glycine and caged GABA

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Our lab has created a family of caged compounds based on the coordination chemistry of ruthenium bipyridyl complexes. We have caged 4-AP, glutamate, nicotine, serotonin, and other biologically important small molecules with effects over

ionotropic or metabotropic receptors. The photochemical properties of these complexes make them particularly useful for a variety of applications: their very fast nanosecond-range kinetics allows probing ligand-triggered ion channel conformational changes. Their coordination bonds are labile under long wavelength, visible light. Their high quantum efficiency  $\{\phi\}$  and high absorptivity coefficients  $\{\epsilon\}$  makes it possible to use very inexpensive, low power light sources, like violet (405 nm), blue (473 nm) or green (532 nm) laser pointers which in turn makes expensive quartz optics unnecessary. Finally, their high solubility and absence of nonspecific interactions allow for in-vivo experiments. Here we present two new members of this family: caged GABA (Ru(bpy)2PMe3GABA) and caged Glycine (Ru(bpy)2PMe3Glycine) their synthesis, chemical properties and application examples.

Neurochemistry and Neuropharmacology

**Poster Number (137) Session 1**

Effects of systemic administration of fluoxetine and tianeptine on an operant conditioning task in rats

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The medial Prefrontal Cortex (mPFC) is one of the brain structures that controls reward-directed behaviors. The mPFC is innervated by serotonergic axons and express serotonin (5-HT) receptors. Therefore, the serotonergic circuit comprising the mPFC could be involved in the learning of reward-dependent tasks. We studied the effects of systemic administration of Fluoxetine, a selective 5-HT reuptake inhibitor, and Tianeptine, a selective 5-HT reuptake enhancer, on the performance in a operant conditioning task. Adult Long Evans rats were trained in five sessions (25 trials each). Fluoxetine reduced the number of correct responses in the second and third session ( $p < 0.01$ ), compared to control subjects. At the fourth session, animals from both groups reached the 100% of responses. However, the latency times to respond were higher from the second ( $p < 0.001$ ) to the fourth ( $p < 0.01$ ) session. Surprisingly, Tianeptine also impaired the performance on the task. At the first and second sessions, the number of correct responses was lower ( $p < 0.01$  and  $p < 0.001$ ) but not for the third. Moreover, the latency times were higher in treated animals in both, the second ( $p < 0.001$ ) and third ( $p < 0.01$ ) session. Our results suggest a fine tuning of the serotonergic circuit in the operant conditioning learning.

NMDA receptor NR2A subunit expression in an experimental epilepsy model. Cyclopentyladenosine effect

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NMDA receptor (NMDAR) is involved in synaptic plasticity, memory and neurological diseases like epilepsy and it is the major mediator of excitotoxicity. NMDARs are heteromeric complexes formed by the subunit NR1 (essential) and one or more NR2 subunit (A–D). NR2A-containing NMDAR may play a crucial role in epileptic disorders. In this study we focused on NR2A subunit expression in hippocampus after repetitive seizures induced by the convulsant drug 3-mercaptopropionic acid (MP) and the effect of adenosine analogue, cyclopentyladenosine alone (CPA) or previous to MP (CPA+MP). Lots of Wistar rats (200–250 g) were administered during 4 days: a) MP 45 mg/kg; b) CPA 2 mg/kg; c) CPA+MP; d) Control (C). Hippocampus was homogenized at 5th day. Western blot assays were performed on the whole hippocampal tissue showing a significant increase in NR2A [removed]17.5%, P

Activity-dependent regulation of GABAA receptor function mediated by a phosphorylation mechanism

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Continuous activation of GABAA receptors under pathological and physiological conditions produces changes in receptor structure and/or function. Chronic exposure of neocortical cultures to GABA (48 h) induces down regulation of GABAA receptor number and uncoupling of GABA and benzodiazepine binding sites. A brief exposure to GABA (t<sub>1/2</sub> 3 min), however, induces uncoupling hours later without alterations in receptor number. This paradigm allowed us to study the uncoupling mechanism independently from the down regulation mechanism. In a previous study we found a decrease on mRNA and protein levels of certain GABAA receptor subunits and a reduction in the levels of  $\alpha 3$ -containing receptors. On the other

hand, we demonstrated that PKA and PKC inhibitors block uncoupling induced by a brief GABA exposure. Since GABAA receptor function and intracellular trafficking are regulated by phosphorylation, the aim of this work was to study possible changes on the receptor phosphorylation state. We observed an increase in the phosphorylation degree of  $\gamma 2$  without changes in the phosphorylation state of  $\beta 2$  subunits. These results suggest that activation of PKA and PKC pathways are part of the uncoupling mechanism induced by GABAA receptor stimulation, probably by phosphorylation of some receptor subunits.

Neurochemistry and Neuropharmacology

**Poster Number (140) Session 1**

Activation and desensitization of *c. Elegans* muscle levamisole-sensitive nicotinic receptors

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*Caenorhabditis elegans* is a model organism for the study of the nervous system and neuromuscular diseases. The levamisole-sensitive nicotinic acetylcholine receptor (L-AChR) is involved in *C. elegans* neuromuscular transmission. This receptor may be composed of three essential (UNC-63, UNC-38, and UNC-29) and two accessory subunits (LEV-1 and LEV-8). We here explored the contribution of the  $\alpha$ -type LEV-8 subunit to activation and desensitization of muscle L-AChRs from *C. elegans*. Single-channel activity of L-AChRs can be detected from muscle cultured cells of LEV-8 null mutant strain. Channel conductance is similar to that of wild-type L-AChRs (~36 pS) but channel lifetime is about 3-fold more prolonged. A more dramatic difference between channel activity from LEV-8 null mutant and wild-type muscle cells is the time-dependent reduction in the frequency of opening events, which suggests enhanced desensitization of L-AChRs lacking LEV-8. ACh-elicited macroscopic currents from the mutant strain decay ~4-fold faster than those from wild-type cells, again indicating enhanced desensitization. Thus, although LEV-8 is an accessory subunit, L-AChRs-mediated responses can be substantially modified whether or not it is incorporated into the pentameric receptor.

Mice lacking p35 exhibit hyperactivity and paradoxical response to psychostimulants

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Cyclin-dependent kinase 5(cdk5)/p35 kinase complex plays a critical role in dopaminergic neurotransmission. Dysregulation of dopamine (DA) signaling is associated with neurological and psychiatric disorders. Cdk5 requires association with p35 for its proper activation, we hypothesized that dysregulation of Cdk5 activity might have an effect on striatal-mediated behavior. We used a mutant mouse, deficient in p35 (p35 KO). We demonstrated that only juvenile p35 KO mice displayed spontaneous hyperactivity, responded with a paradoxical hypolocomotor effect to psychostimulant drugs and exhibited deficit on behavioral inhibition. Elevated levels of tyrosine-hydroxylase and high striatal DA synthesis and contents with a low DA turnover, which were reverted by psychostimulants, were also found in p35KO mice. Our results demonstrate that p35 deficiency is critically involved in the expression of a hyperactive behavioral phenotype with hyper-functioning of the dopaminergic system. Thus, p35 KO mice may represent a useful animal model for study mechanism underlying attention deficit hyperactivity disorder.

Hyperlocomotion induced by MK-801, a n-methyl-d-aspartate (NMDA) receptor antagonist, imply a gabaergic disinhibition of the anterior thalamic nucleus

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The behavioural changes observed after the systemic administration of NMDA receptor antagonists are used as a preclinical model to study the neurobiological basis of schizophrenia and the mechanism of action of antipsychotics. Despite its widespread use, the neural circuits and mechanisms are yet under study. We evaluated the behavioural effects induced by MK-801 systemic administration in rats with a concomitant microinjection of Muscimol (GABAA receptor agonist) into



the Anterior Thalamus Nucleus (ATN), a projection nucleus to cortical areas. ATN neurons are modulated by GABAergic projection from the Reticular Thalamus Nucleus. Locomotor pattern and activity were registered (15 min) using the Open Field paradigm associated to the Ethovision tracking software. Stereotypies and rearings were registered by direct observation. Results showed that Muscimol intra ATN decreased significantly the hyperlocomotion induced by MK-801 administration. Stereotypies and motor disorganization were reinstated. Rearings did not change. These data indicate that a desinhibition of ATN neurons mediates almost all the behavioral effects induced by the NMDA antagonism. The participation of the thalamic-cortical circuit in the mechanism of action of NMDA antagonists was also demonstrated.

Neurochemistry and Neuropharmacology

**Poster Number (143) Session 1**

Prenatal stress increases plasma corticosterone and hippocampal nitric oxide synthase expression in rat's offspring

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Stress during early stages of development induces long lasting neurochemical and neuroendocrine alterations that in turn develop into behavioural deficits. We have already established that prenatal stress (PS) induces a deficit in spatial and associative memory and alterations in anxiety-like behaviour, together with a reduction of nitric oxide synthase (NOS) activity in adulthood. The aim of the present study was to determine if PS has an effect on plasma corticosterone levels and in hippocampal NOS expression. Methods: Pregnant Wistar rats were restrained three times a day, 45 minutes each, during the last week of pregnancy. Offspring was sacrificed at postnatal day 7 (PN 7) and analyzed for hippocampal NOS mRNA by RT-PCR and for plasma corticosterone levels by ELISA. Results: nNOS mRNA levels were increased by PS. We also found an increased level of plasma corticosterone. Discussion: It has already been shown that different models of PS induce alterations in the HPA axis that in turn develop into behavioural alterations. We have found behavioural abnormalities in PS rats, together with alterations in nitric oxide synthase expression and activity, both in cerebellum and hippocampus. In this study we show that this modifications appear as early as PN 7. Indeed, plasma corticosterone and hippocampal nNOS mRNA levels were increased in PS rats. We are currently evaluating a possible correlation between these parameters, in order to establish its relationship with the behavioural abnormalities observed.

A behavioral and biochemical comparison of 2 different sensitization protocols: chronic vs two injection protocols (TIPS)

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Locomotor sensitization to psychostimulants depends on the temporal pattern of drug exposure. Studies show that a single exposure to psychostimulants induces behavioral sensitization. The goal of this study was to compare the behavioral response of 2 different sensitization protocols, chronic and two injection protocol (TIP). Adult mice were divided in 2 groups (chronic or TIP). Subjects from the chronic condition received 5 injection of cocaine (coc, 20mg/kg) or vehicle and those from TIP receive only one injection. Fourteen days after the first injection the subjects from each condition received a challenge of coc or vehicle. Both protocols produced locomotor sensitization. The temporal pattern induced by the chronic was significantly different than the one induced by the TIP. The maximum response in the chronic group was reached earlier. Twenty min after injection locomotor activity in the chronic group was significantly lower than in the TIP, and by the end of the test, the chronic group showed again significantly higher activity scores. The differences observed in the temporal pattern of the slope of the 2 protocols seems to be associated with biochemical changes induced by the differential exposure to the drug rather than differences in exposure to the testing environment.

Glutamate release is involved in C. Perfringens epsilon toxin neuropathology

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*Clostridium perfringens* type D epsilon toxin (ETX) affects different animal species and is considered a category B toxin by the Centre of Disease Control, for its potential role in bioterrorism. It affects the vasculature of the brain causing oedema and death. Some reports indicated that excessive neurotransmitter release

is also implicated in its pathology. Mice were inoculated intraperitoneally with several different neuroprotective drugs including Riluzole, an inhibitor of glutamate release, and then challenged with ETX (2 LD50). In addition, SD rats were injected via the right lateral ventricle, with either ETX (0.05 LD50) or vehicle. The brains of the mice were removed and processed by histology, stained with H&E and Fluor Jade-B. Rat brains were perfusion-fixed either with 2% glutaraldehyde and analyzed by transmission electron microscopy (TEM) or formalin 10% for LM. Toxin-inoculated mice –but no control mice- exhibited convulsive episodes and death, 5 to 30 minutes after ETX inoculation. When treated with Riluzole, mice survived 2 or 3 times longer, and some lived until the termination of the experiment. Both H&E and Fluor Jade-B stains revealed neurodegenerative changes in ETX but not in the controls. In the rats, increased amount of neurofilaments in the neuropile and glial processes between pre- and post-synapses were seen with TEM at the cortex, thalamus and striatum. These results suggest that ETX induces significant neurodegenerative changes through excessive glutamate release that may lead to death independently of the vascular damage.

Neurochemistry and Neuropharmacology

**Poster Number (146) Session 1**

ChIP-RAPD as a possible new methodology to identify genes regulated by chromatin related proteins

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Cells can respond to nicotine by activating specific pathways and translating some of them into changes in gene expression. Technical and experimental advances in DNA amplification, quantitative PCR, cDNA microarray and chromatin immunoprecipitation (ChIP) analysis have led to an increase in the number of studies identifying specific genes regulated by chromatin related proteins. Our aim was to characterize activated/repressed genes in response to nicotine when chromatin remodeling is involved. We decided to setup a genome screening method which sequentially combines two key techniques: 1) the ChIP assay, used to study protein-DNA interactions and mapping of histone modifications, 2) Random amplified polymorphic DNA (RAPD), which has been used to detect polymorphism in genetic mapping. To approach this strategy, we chose a simple nicotine responder model, PC12 cells. NGF, the strongest PC12 stimulus, was used to setup the technical conditions. Different antibodies against histone modifications were tested for ChIP. For RAPD, around 10 randomized primers were used to find different unknown amplicons. Preliminary results indicate that specific genes were detected.

ted with different antibodies. Sequentiation of the relevant bands generated with RAPD will allow us to identify these genes.

Neurochemistry and Neuropharmacology

**Poster Number (147) Session 2**

The amphetamine-induced neuroplastic changes involve the brain renin-angiotensin system (RAS)

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The enhanced response to psychostimulants relies on time-dependent neuroplastic changes involving long-lasting hyper-reactivity of dopaminergic pathways. A single exposure to amphetamine (AMPH) is sufficient to induce long-term changes in rats. Our study tested the hypothesis that Angiotensin II AT1 receptors are involved in the neuroadaptative changes induced by a single exposure to AMPH and that such changes are related to the development of neuroplasticity. Wistar male rats (250-300 g) were pretreated with an AT1 blocker, candesartan (3 mg/kg po) for five days and after that injected once with AMPH (5mg/kg ip). The Fos immunoreactive neurons (Fos-ir) in response to AMPH (0.5 mg/kg) were determined 3 weeks later. Other animals treated with AMPH (5mg/kg ip) were used 7 or 21 days later to determine the AT1 receptors expression by immunohistochemistry after AMPH (0.5 mg/kg) injection. Our results showed an increase in Fos-ir neurons in AMPH pretreated rats in NAcc and CPu and this response was prevented by the AT1 receptor blockade. AMPH produced an increase of AT1 receptors immunoreactivity in CPu and NAcc observed 7 and 21 days after treatment. The brain RAS should be considered for a better understanding of the mechanisms involved in psychostimulants-induced neuroplasticity.

Neurochemistry and Neuropharmacology

**Poster Number (148) Session 3**

Modafinil attenuates methamphetamine acute toxic effects in mice

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Long term Methamphetamine (METH) exposure has been shown to produce deleterious effects on brain function. Recent clinical data have supported the use off-label of Modafinil (MOD) for the treatment of psychostimulant addiction. Here we evaluate the potential neuroprotective role of MOD on METH neurotoxic effects in mice treated either with a single injection (2 or 7 mg/kg) or with a binge dosing protocol of METH (3x 7mg/Kg, ip. 3 h apart) co-administrated with MOD (2x 180 or 90 mg/Kg, 1 h before first and third METH injection); control groups received vehicles. As expected, acute METH administration induced hyperlocomotion and increased continuous sniffing compared to MOD and control group; MOD induced a dose dependent increase in locomotor activity without altering stereotyped behavior; MOD+METH group exhibited locomotor activity values similar to the saline group but showed enhanced stereotyped behavior. Six days after METH binge, reduced distance traveled was observed in METH treated animals, except for the group pretreated with MOD 90 mg/kg. Striatal dopamine content (measured by HPLC) and TH immunoreactivity decreased in METH group while MOD+METH showed similar values compared to the saline group. Our results suggest a possible protective role of MOD against METH acute toxicity. Supported by: PICT 2007-1009 (F.U.), PICT 31953, UBACyT M013 (S.W.)

Neurochemistry and Neuropharmacology  
**Poster Number (149) Session 1**

The development of an agonist for the  $\alpha 9\alpha 10$  nicotinic cholinergic receptor

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The  $\alpha 9\alpha 10$  nicotinic acetylcholine (ACh) receptor is expressed in outer hair cells of the organ of Corti, where it is involved in efferent modulation of sound amplification. With the exception of ACh, no effective agonists for  $\alpha 9\alpha 10$  are known. In this study several ACh analogs were designed based on previous investigations of molecular dynamics and tested as potential agonists for  $\alpha 9\alpha 10$  using electrophysiology recordings in *Xenopus* oocytes. Four quaternary ammonium compounds elicited agonist-induced responses. One particularly effective compound, 2-((3-methoxy-3-oxopropanoyl)oxy)-N,N,N-trimethylethanaminium iodide, produced nearly 150% of the maximal response to ACh (n=5). Compared with an EC50

of  $15.35 \pm 0.29 \mu\text{M}$  for ACh ( $n=7$ ), this compound had an EC<sub>50</sub> greater than 508.2  $\mu\text{M}$ , and required a concentration of 129  $\mu\text{M}$  to mimic the EC<sub>50</sub> response of ACh. This compound was chosen as a lead compound that will be modified to improve selectivity and potency of the agonist. The iodides of 2-((4-methoxy-4-oxobutanoyl)oxy)-N,N,N-trimethylethanaminium ( $n=3$ ), 2-((5-methoxy-5-oxopentanoyl)oxy)-N,N,N-trimethylethanaminium ( $n=4$ ), and 2-(2-methoxy-2-oxoacetoxy)-N,N,N-trimethylethanaminium ( $n=3$ ) acted as weaker agonists. These results indicate a step toward the development of a drug to target  $\alpha 9\alpha 10$ .

Neurochemistry and Neuropharmacology

**Poster Number (150) Session 2**

Allosteric positive effects of phenolic compounds on GABAA receptor in primary neuronal cultures

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We have previously demonstrated that thymol, a phenolic compound, possesses an important positive activity as modulator of GABAA receptor and GABA agonist. According to its similar structural properties with propofol, we have developed a pharmacophore model of activity on GABAA receptor for both compounds and derivatives (García et al., Neuropharmacol. 2006, 50:25). In the present work, we analyzed other different phenolic derivatives -carvacrol, eugenol and chlorothymol- which would confirm and/or improve this model. The effects on the GABAA receptor were evaluated by their ability to modulate the 3H-flunitrazepam binding on primary cultures of cortical neurons. The results showed an increment in the ligand binding induced by carvacrol and eugenol (EC<sub>50</sub>: 248Carv and 846Eug  $\mu\text{M}$ ), and an enhancement exerted by chlorothymol but only until approximately 100  $\mu\text{M}$ . Higher concentrations of chlorothymol reduced the binding possibly by the prevalence of an eventual cytotoxic effect. Bicuculline, a GABA antagonist, inhibited the stimulation produced by all compounds. Thus, the obtained results would indicate a positive modulation of GABAA receptor performed by all assayed compounds. Finally, the following studies are being directed to evaluate their effects on the neuronal viability. Supported by FONCyT, SECyT-UNC, IBRO, FIS 061212.

Ontogenetic differences in sensitivity to LiCl- and amphetamine-mediated taste avoidance in preweanling rats

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When amphetamine is associated with a tastant conditioned stimulus, rats learn to avoid the taste even with doses that promote conditioned place preference. This paradoxical effect has been explained in terms of fear conditioning when learning involves a gustatory stimulus. In the infant rat it has been defined a sensitive period (until postnatal day 10) in which infants learn appetitive effects to stimuli to which aversions are conditioned after this period. Exogenous administration of corticosterone reverses this effect. We investigated conditioning to amphetamine or LiCl, within and after the sensitive period and the role of corticosterone modulating this learning during this ontogenetic period. After the sensitive period infant rats acquired aversions to the taste paired with amphetamine or LiCl after two conditioning trials, but within the sensitive period aversions were conditioned only by LiCl, and after four conditioning trials. Amphetamine-mediated taste avoidance was not observed even when corticosterone was administered before conditioning. Additionally, during the sensitive period LiCl promoted conditioned taste preference. The present experimental framework may represent a useful tool for studying mechanisms underlying taste avoidance and aversion effects.

Increased GABAA receptors  $\alpha$ 1-subunit expression in chick forebrain. Modulation by noradrenaline and acute stress

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Gamma-aminobutyric acid (GABA), the major inhibitory neurotransmitter in the brain, mediates inhibition via GABAA receptors (GABAAR). The assembly of GABAAR as heteropentamers produces complex heterogeneity in their structure;

this is the major determinant of their pharmacology profile. Many studies investigating the abundance of GABAAR using subunit specific antibodies confirmed that receptors composed by  $\alpha 1$ ,  $\beta 2$ ,  $\gamma 2$  subunits are the most abundant in the brain. Our previous findings, in 4-6 day old chicks, show differences on behavioral and pharmacological responses and on GABAAR recruitment after Open Field and central administration of noradrenaline. In the present study, different doses of noradrenaline (0.0025, 0.0100, 0.0500, 1.000  $\mu\text{g}/\mu\text{l}$ ) and the Open Field induced an increased GABAAR  $\alpha 1$ -subunit expression on cell-surface detected by immunoperoxidase in brain tissue. In addition, we also observed a higher  $\alpha 1$ -subunit expression after to acute stress. These results suggest that the increased  $\alpha 1$ -subunit expression induced by noradrenaline is additive to response to an acute stress.

Neurochemistry and Neuropharmacology

**Poster Number (153) Session 2**

Variable magnetic fields modify function of Cys-loop receptors

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In today's world, technological developments bring social and economical benefits to the society; however, the health consequences of these developments can be difficult to predict and manage. With rapid advances in communication technologies, population is increasingly exposed to electromagnetic fields (EMFs), which have raised concern about potential health effects from exposure to them. We here studied the influence of low intensity EMFs on members of the Cys-loop receptor family, nicotinic (AChR) and 5HT<sub>3A</sub> receptor. We recorded macroscopic currents from cells expressing these receptors exposed to EMFs before or during agonist application. Peak currents are reduced by the exposure to EMFs at frequencies varying from 15 Hz to 120 kHz whereas the decay time constants are not affected. The decrease in the peak current is dependent on both the frequency of the field applied and the membrane potential. Interestingly, the effect of EMFs on 5HT<sub>3A</sub> receptor currents is 3-times more profound than on AChR currents, thus indicating different sensitivity of the receptors to EMFs. In conclusion, our results reveal that EMFs affect function of ligand-gated ion channels and open doors to understand the mechanistic of channel modulation.



## **\* Sensory Systems**

Sensory Systems

**Poster Number (154) Session 1**

Modulation of gliogenesis by glia-glia and neuron-glia interactions in the olfactory pathway

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The modulation of neurogenesis by sensory input and the degree of regeneration after damage reveal plasticity in the mammalian afferent olfactory pathway. It has been proposed that the presence of specialized glia, olfactory ensheathing cells (OECs), explains this plasticity. The question whether plastic changes in gliogenesis correlate with changes in neurogenesis of olfactory sensory neurons has not been addressed. We hypothesize that glia-glia and neuron-glia interactions are involved in the homeostasis of OEC population size. We expect to observe alterations in OEC number when glial gap junction connectivity is altered or when the rate of neurogenesis changes. We will: 1. Study correlations between gliogenesis and neurogenesis during development and in adult life in conditions that alter neurogenesis. 2. Perform lineage analyses to identify cell types in the progeny of proliferating OECs, in transgenic mice that allow us to label isolated cells. 3. Study the role of glial connectivity on gliogenesis, using transgenic mice with reduced levels of connexin 43, a protein that forms OEC gap junctions. These experiments will help to elucidate mechanisms of plasticity of the olfactory pathway and will help the design of therapies using OEC transplants for CNS repair.

Sensory Systems

**Poster Number (155) Session 2**

Purinergic signaling is involved in the regeneration of the zebrafish retina

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In the adult fish, damage of the mature retina activates tissue regeneration from different pools of intrinsic precursor cells. Extracellular purinergic signaling is cru-

cial for inducing cell proliferation and regeneration. We have previously demonstrated that cell proliferation is regulated by extracellular ADP via P2Y1 membrane receptors, because a P2Y1 receptor antagonist completely blocked lesion-induced increase in cell division. Injury-induced cell proliferation was not modified by antagonists for other ADP-, adenosine- or ATP-activated receptors. Here, we report the presence of mRNA (RT-PCR) and protein (immunofluorescence) for P2Y1 receptor in the zebrafish retina. We also described that zebrafish retina expresses mRNA for P2Y2, P2X2, P2X1, P2X7 and P2Y12 receptors. In addition, we evaluated the localization of P2Y1 receptor in non-lesioned and lesioned retinas by using specific markers of retinal cells. We found that this receptor is localized in the inner layers mainly at synaptic level. Colabeling with SV2 suggests that P2Y1 receptor is principally on postsynapsis. These results add supporting evidence for an important role of extracellular ADP which by acting via P2Y1 receptors regulates cell proliferative activity necessary for retinal regeneration.

Sensory Systems

**Poster Number (156) Session 3**

The electrical response of human cells using the ECIS technique

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The Electrical Cell-substrate Impedance Sensing (ECIS) technique allows us to obtain relevant physical information from the measurement of the electrical impedance developed by cells cultured in vitro on a microelectrode system. Using this method we can red in vitro on a microelectrode system. Using this method we can study, in a non-invasive way, the dynamics of some biological processes associated with cell growth. This technique is used to characterize various parameters of a monolayer cells and their reaction to different concentrations in the osmolarity of the medium. In the present work we studied the electrical response of human conjunctival epithel cell line (IOBA-NHC). We experimentally determined that cell monolayer contacts were modified determined that cell monolayer contacts were modified in in conditions of hyperosmolarity.

Sensory Systems

**Poster Number (157) Session 1**

Analysis of the dynamic of posmitotic neuronal migration during corticogenesis in the chick optic tectum

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The structural organization of cortical areas results from the dynamic of cellular behavior involved in development. The cellular behavior takes place in an integrated spatial and temporal organization. Such organization depends on gradients or waves tightly controlled signaling processes throughout the region. Recently we have shown in the chick optic tectum that specific features characterizing successive stages of development occur as a function of the time and space, spreading from cephalic to caudal. Given that radial migration of postmitotic neurons is a key process in the corticogenesis we analyzed fluctuations in the interphase zone between the generation and migration of neurons aimed at verify whether such phenomena shows a spatial organization in the mentioned ax. Standard mathematic methods allow us to demonstrate the occurrence of zones of high and low amplitude of fluctuations associated with high and low intensity of neuronal migration respectively. Moreover, our studies showed that the zone of high migratory activity strated on the cephalic ending and spread towards to the caudal one at non-constant rate and shaping a wave during the migratory phase of the neuroepithelium.

Sensory Systems

**Poster Number (158) Session 2**

Rod-cone interaction in the mesopic

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Rod-cone interactions are a fundamental operation in visual processing under mesopic light levels. They can be observed when the stimulation of cones surrounding a test reduces the sensitivity of the rods sensing that test, or from the temporal point of view, acting to decrease the temporal bandwidth of the visual

system. We present results obtained in two different kinds of psychophysical experiments made in the mesopic range evidencing these interactions. In one of them, the effect of a glare source was measured (10 degree to the visual axis) upon the brightness of a foveal test. The results showed a brightness reduction of the test due to the glare, but also they evidence that brightness reduction is stronger when the background luminance decreases. In the second experiment the sensitivity of the retina was studied when there are a sudden changes in adaptation. In this case the results showed a loss of sensitivity when moving from a steady dark adaptation condition to a transient mesopic condition. It also became clear that the speed of adaptation is greatest in an area of the retina between 6 and 9 degrees of eccentricity. Both sets of results show the phenomenon of rod-cone interaction and can be interpreted in terms of the cellular pathways that were proposed for this operation such as gap junctions and All cells.

Sensory Systems

**Poster Number (159) Session 3**

Adaptive evolution of  $\alpha 9\alpha 10$  nicotinic acetylcholine receptors

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The  $\alpha 9\alpha 10$  nicotinic acetylcholine receptor ( $\alpha 9\alpha 10R$ ) mediates efferent inhibition of cochlear hair cells in mammals and birds. This inhibition results from a  $Ca^{2+}$ -dependent  $K^{+}$  current activated by  $Ca^{2+}$  entry through  $\alpha 9\alpha 10Rs$ . Using codon-based likelihood models we found evidence of positive selection in the CHRNA10 (Franchini and Elgoyhen, 2006) and CHRNA9 genes within the mammalian lineage. We propose that, as a result of non-synonymous substitutions, mammalian and non-mammalian  $\alpha 9\alpha 10Rs$  should present differential functional properties. To test this hypothesis, we studied mammalian (rat) and avian (chicken)  $\alpha 9\alpha 10Rs$  expressed in *Xenopus* oocytes. We found that these evolution-driven modifications dramatically changed the receptor's properties when comparing rat to chicken. Unlike rat, chicken  $\alpha 10$  subunits formed a functional receptor. Strikingly, the  $Ca^{2+}$  permeability of the avian  $\alpha 9\alpha 10Rs$  was substantially lower than that of its mammalian counterpart. Moreover, aminoacid substitutions in the  $\alpha 9$  subunit (and not  $\alpha 10$ ) were responsible for the encountered differences in  $Ca^{2+}$  permeability. These results indicate a different evolutionary history of mammalian versus non-mammalian  $\alpha 9\alpha 10Rs$ , with important functional implications for the operation of the efferent system in each species

Building the colour space of tetrachromatic species

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Many animals (humans included) have the ability to distinguish objects based on the spectral distribution received by the eye. Having established the existence of colour vision, research focused on the question of how many types of photoreceptors are involved. Colour perception is a result of the comparison of differential excitation between cones with different spectral sensibilities at roughly the same retinal location. In the animal kingdom colour vision is classified as dichromatic, trichromatic, tetrachromatic (etc.) according to the number of lights required to match any spectral light. While most mammals have only two types of cones and we (old-world monkeys) have three, birds have five types of cone photoreceptors: four single cones and a double cone. They are said to have tetrachromatic vision. It is hard to know how the colour space is built in this kind of species and how it is used advantageously by the animal. Is the neural wiring genetically coded or experienced dependent? To address these questions we propose a set of behavioral experiments of colour discrimination by domestic chicks (*Gallus gallus*) reared in colour deprivation conditions. A model will be used to fit the behavioral data, so we can then look for physiological correlate of the postulated neural mechanisms.

Olfactory processing in the antennal lobe of the honey bee: neural representation of mixtures and pure odors

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The antennal lobe (AL) is the first processing centre for olfactory information in the insect brain. It receives information from sensory neurons and from it depart projection neurons that convey odor information to other brain areas. Some evidences suggest that experience affects the AL network in a way that the animal gains sensibility for relevant stimuli. We hypothesize that such ability should be

important in the context of odors mixtures, in which the presence of a relevant and meaningful odorant can be overshadowed by another confounding and irrelevant odor. We did calcium imaging in projection neurons of the antennal lobe and determined the activation patterns for single odors that are floral components, 1-hexanol, 2-octanone, acetophenone, geraniol, and the respective binary mixtures. We report our first results with representation of pure odors and mixtures in naive animals. All tested odors elicited differential and unique spatiotemporal activity patterns with some degree of overlap. Representation of the binary mixtures can be mainly predicted based on the patterns of the pure odors with only minor cases of synergistic and antagonistic interaction. Next experiments will study if appetitive learning with one component affects mixture representation.

Sensory Systems

**Poster Number (162) Session 3**

Daily proliferative activity in neurogenic niches of zebrafish retina is regulated by purinergic signals

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Growth and regeneration that occur in the adult teleost retina by neurogenesis have been helpful in identifying molecular and cellular mechanisms underlying cell proliferation and differentiation. We have found that cell proliferative activity in a neurogenic area known as the ciliary marginal zone (CMZ) of the adult zebrafish retina follows diurnal variations with highest values observed at daylight. ADP $\beta$ S-treatment significantly enhanced cell division at night to normally observed daylight levels. Diurnal increase of proliferation was completely abolished when extracellular nucleotide levels or their extracellular hydrolysis (by ectonucleotidases called NTPDases) were significantly disrupted. Likewise, proliferative activity was inhibited by using an antagonist of purinergic P2Y1 receptors. Finally, we showed that mRNA levels of NTPDases 1, 2 and 3 as well as of P2Y1 receptors are present in the zebrafish retina. NTPDases mRNA expression exhibited a two-fold increment in light versus dark conditions whereas P2Y1 receptor mRNA levels did not show significant daily variations. Our results demonstrate a key role for nucleotides as well as for NTPDases for the regulation of proliferative activity in the CMZ, associated with retinal growth, throughout the light:dark cycle.

## **\* Cognition, Behavior, and Memory**

Cognition, Behavior, and Memory

**Poster Number (163) Session 1**

Pregnenolone infused in lateral septum impairs memory acquisition of male rats in a passive avoidance test

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Pregnenolone (Preg) and Preg-Sulfate (Preg-S) are neuroactive steroids (NS) that have a role as memory modulators, among other functions. Our objective was to study the effects of these NS in an aversive memory paradigm (step down passive avoidance test) when injected in the lateral septum nucleus (LSN) of male rats. Three groups were used: 1) Control; 2) Preg 12  $\mu$ M; y 3) Preg-S 12  $\mu$ M. After 24 hs. they were tested for retention. The administration of reagents (1 $\mu$ L) was done via a cannula implanted in the right LSN 30 minutes before training. Statistical analysis was performed using an ANOVA-1 test, adopting a value of p

Cognition, Behavior, and Memory

**Poster Number (164) Session 2**

Age-related differences and role of novelty in the expression of ethanol-induced locomotor activation

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Adolescent rats exhibit ethanol-induced locomotor activity. Spontaneous and ethanol-induced activation in an inescapable environment have been hypothesized to predict ethanol reinforcement and self-administration. This work assessed age-related differences in ethanol's motor stimulating effects and analyzed the role of novelty in the expression of these effects. Adolescent (postnatal days, PD28) and adult rats (PD70) were given ethanol (0.0 or 2.5 g/kg; i.g.) and screened for motor activation in an open field, during post-intubation minutes 5 to 11. A subsequent experiment assessed ethanol-induced activity in adolescent rats that

had been previously habituated to the open field. Ethanol induced motor activating effects and this result was similar for adolescents and adults (Experiment 1). Repeated exposure to the test arena caused a significant decrease in locomotion in vehicle-treated adolescents, a result suggestive of habituation to the context. The adolescents given ethanol, however, exhibited robust behavioral stimulation even when the test arena was no longer novel (Experiment 2). These results indicate that adolescent and adult animals appear to perceive the pharmacological effects of high-dose ethanol as similarly activating. Ethanol-induced locomotor activity in adolescents does not depend on the novelty of the testing environment.

Cognition, Behavior, and Memory

**Poster Number (165) Session 3**

Acute stress induces the formation of long-term memories

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The formation of long-term memories (LTM) requires protein synthesis. We have demonstrated that learning of a task which only induces short-term memory (STM) can be stabilized into LTM if another event brings in the necessary proteins. As it is widely known that stress can enhance or disrupt synaptic plasticity and memory consolidation, we decided to investigate whether acute stress could promote the formation of LTM from a weak training. We also studied the dependence of the phenomenon on protein synthesis. For this purpose, rats were subjected to a weak training (inhibitory avoidance or spatial object recognition) that induces only STM. At different times close to training, subjects were exposed to a stressful situation (an elevated platform –EP– for 30 min), which elicits the release of corticosterone (assessed by RIA). In another set of experiments we infused a protein synthesis inhibitor in the hippocampus, before the exposure to the EP and trained rats with a weak protocol. We found that inhibition of protein synthesis prevented the promoting effect of the stressful event. In sum here we show that STM, derived from a weak training, can be stabilized into a LTM due to the influence of an adjacent stressful situation and this process requires the synthesis on new proteins.



Cognition, Behavior, and Memory

**Poster Number (166) Session 1**

Impact of cognitive training on neuronal and glial markers in the hippocampus of non human primates (macaca fascicularis)

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We present initial results regarding the impact of intensive cognitive training on specific brain areas in a non human primate (*Macaca fascicularis*). Such areas (within prefrontal and parietal cortical regions, as well as the hippocampus) have been involved in the processing of complex brain functions. Lipina and Colombo (Brain Res. 1134:180-186, 2007), showed that cognitive training selectively prevented cognitive impairment following neurotoxin MPTP administration. Since plasticity is an intrinsic property of the nervous system, and under the assumption that cognitive training produces functional changes at the participating neural circuits, we are in process of evaluating the underlying cellular and molecular changes. Immunohistochemical analysis is being performed following intensive cognitive training (Spatial Delayed Response and Detour tests administered twice daily, during three months). We began analyzing the hippocampal dentate gyrus using cell proliferation- and new neurons markers (BrdU, doublecortin), as well as non neuronal markers (aquaporin 4, Ezrin), aiming at the possible association of neuronal-glial changes. Administrative and technical support : Lic.B. Stuto (CONICET), Mrs.C. Juárez (CONICET). Support: Fund. Quirno, Fund. Conectar, FONCYT, CONICET.

Cognition, Behavior, and Memory

**Poster Number (167) Session 2**

Shines and shadows of perception: the threshold for access to consciousness fluctuates with phases of bipolar disorder

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Bipolar disorder is a psychiatric diagnosis describing mood disorders which alter-

nates episodes of mania (abnormally elevated levels of energy, cognitive abilities and mood) with depression. Although bipolar patients have cognitive impairments even in euthymia, patients report an increased perceptual sensitivity during mania (linked in some subgroup of patients to creativity) and shadowing of perception during depression. We hypothesized that the threshold of conscious access (i.e. the strength of a stimulus to access consciousness) may vary with the different phases of bipolar disorder. We measured conscious threshold using an iterative masking procedure. As the temporal interval between the stimulus and the mask decreases, stimulus visibility decreases following a sigmoidal function from which a threshold can be derived. To separate conscious and executive function processing, we measured the threshold in four different attentional control mechanisms. We observed a main effect of phase on the threshold when comparing across groups: 1) thresholds were higher for patients in the depressive phase than for controls, 2) thresholds were lower for patients in the mania phase than for controls and 3) thresholds were slightly higher for patients in the euthymic phase than for controls.

Cognition, Behavior, and Memory

**Poster Number (168) Session 3**

Behavioral tagging during an inhibitory avoidance task: identification of involved transmitter systems, learning tag-molecules in hippocampus-dependent long-term memory formation

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Long-term memory consolidation requires the synthesis of plasticity-related proteins (PRPs). We have demonstrated that learning of a task which only induces short-term memory (STM) can be stabilized into LTM if another event brings in the necessary proteins. A weak training which only would result in short-term memory (STM) can set a "tag" but does not induce protein synthesis. The promotion to a long-term memory (LTM) is possible if the tag could capture PRPs derived from a temporal-spatial closely-related event. Here, we studied the involvement of glutamatergic, dopaminergic and noradrenergic inputs on the setting of the "learning-tag" and PRP-synthesis using an inhibitory avoidance (IA) training and the exploration of a novel open field (OF). Throughout pharmacological interventions around OF and/or IA sessions, we found that hippocampal dopamine D1/

D5- and  $\beta$ -adrenergic receptors are specifically required to induce PRP-synthesis, while the glutamatergic N-methyl-D-aspartate receptors were required for setting the "learning-tags"; which machinery further required  $\alpha$ CAMKII and PKA but not ERK 1/2 activity; emphasizing the essential role of the interaction between PRPs and "learning-tags" for long-term memory formation.

Cognition, Behavior, and Memory

**Poster Number (169) Session 1**

FNIRS and pupilometry as a measurement of mental effort

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Functional near-infrared spectroscopy (fNIRS) is a non-invasive technique based on neurovascular coupling, which uses the effect of metabolic activity due to neural processing on the oxygenation of cerebral tissue. fNIRS measures hemodynamic changes associated with cortical activation. Despite NIR technologies have lower spatial resolution than other neuroimaging methods, it offers the advantage of portable systems. In this study a novel miniaturized wireless fNIRS instrument was used, with no head or body fixation requirement. Hemodynamic and pupil activity was recorded simultaneously during performance of an arithmetic and a memory task, with different levels of complexity. Results for both tasks show an increased pupil response with task complexity. However pupilometry is known to also correlate with other measurements of cognitive load. fNIRS results show significant main effects between oxy-hemoglobin [ $\text{O}_2\text{Hb}$ ] changes and the task complexity indicating that NIRS is capable of discriminating different complexity levels. We also detected high correlations between pupilometry and fNIRS response, which partially explains inter-subject and inter-trial variability. We conclude that fNIRS can be used to quantify mental effort in absence of explicit report.

Cognition, Behavior, and Memory

**Poster Number (170) Session 2**

Modal and amodal contour completion are driven by different mechanisms

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Are modal and amodal contour completion driven by the same mechanism? We present the results of two psychophysical experiments favoring the hypothesis that modal and amodal completion are driven by different mechanisms. We used random dot patterns producing Spatio-Temporal Boundary Formation and introduced disparity to produce modal and amodal percepts. In the first experiment we tested Vernier acuity for two motion-defined bars as a function of stimulus duration, for disparities of +161 and -168 arc sec. The stimuli moved at 4 deg/s and contained 8 dots/deg<sup>2</sup>. Eight observers participated in the experiment. Results show no differences between thresholds obtained with both disparities for the three values of stimulus duration. Importantly, thresholds decrease with increasing time. In the second experiment, the bars were displayed half of the time with positive and negative disparities and thus, transitioning from occluded to occluder (or vice versa). We present the stimulus with different temporal windows and found no temporal summation of modal and amodal information. Moreover, results show that thresholds obtained in this condition for a presentation time  $T$  equal the thresholds obtained with a single type of stimulus, modal or amodal, presented during  $T/2$ . This suggests that the transition resets the contour computation.

Cognition, Behavior, and Memory

### **Poster Number (171) Session 3**

A big-world network in ASD: dynamical connectivity analysis reflects a deficit in long-range connections and an excess of short-range connections

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Increasing evidence has fuelled the hypothesis that Autism Spectrum Disorder (ASD) is a condition of altered brain functional connectivity. Here we used electroencephalography (EEG) to assess dynamic brain connectivity in ASD focusing in the low-frequency (delta) range. We found that connectivity patterns were distinct in ASD and control populations and reflected a double dissociation: ASD subjects lacked long-range connections, with a most prominent deficit in fronto-occipital connections. Conversely, individuals with ASD showed increased short-range connections in lateral-frontal electrodes. This effect between categories showed a consistent parametric dependency: as ASD severity increased, short-range coherence was more pronounced and long-range coherence decreased. We show that

the networks in ASD subjects have less Clustering coefficient, greater Characteristic Path Length than controls -indicating that the topology of the network departs from small-world behaviour- and greater modularity. Together these results show that delta-band coherence reveal qualitative and quantitative aspects associated with ASD pathology.

Cognition, Behavior, and Memory

**Poster Number (172) Session 1**

Event-related potentials during a finger tapping task

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Finger tapping to a metronome is a paradigmatic task in sensorimotor synchronization, the specifically human ability to entrain movement to an external periodic stimulus. In this task the subject is instructed to tap in synchrony with a periodic sequence of brief tones, and the time difference (asynchrony) between each stimulus and its corresponding response is recorded. Despite being a very simple, spontaneous, and robust behavior in humans, finger tapping involves the superposition of several distinct systems like time perception, comparison, error correction, time production, and motor execution. With the aim of disentangling these overlapped contributions, we recorded high-density EEG event-related potentials and the concurrent behavioral asynchronies during a finger tapping task. We have found a) high bilateral activation in fronto-central sources around 200 ms before tapping; b) lower levels of activity in motor cortex but correlated with the observed asynchronies; and c) a fronto-central mismatch negativity after large asynchronies.

Cognition, Behavior, and Memory

**Poster Number (173) Session 2**

Sildenafil enhances memory reconsolidation of an inhibitory avoidance task in mice

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CF-1 male mice were trained in an inhibitory avoidance task. The immediate injection of sildenafil, a cyclic guanosine monophosphate (cGMP)-specific phosphodiesterase type 5 (PDE5) inhibitor (SIL 1 – 10 mg/kg, ip), immediately after the first retention test given 48 h after training, enhanced retention performance. Retention performance was unchanged in SIL-treated mice not undergoing memory reactivation session. SIL effects were dose- and time- dependent and also were long lasting. The enhancement effect of SIL was dependent on the age of the original memory. That is, there was an inverse correlation between the susceptibility of the memory trace and the time elapsed between training and the first retrieval session. Administration of SIL 30 min prior to the 2nd retention test to mice that received vehicle or SIL immediately after the 1st retention test did not affect retention performance arguing against the interpretation that the post-retrieval enhancing effects of SIL on subsequent retention test might result from state dependency. The results suggest that sildenafil influences retention by modulating time-dependent mechanisms involved in memory processes occurring after retrieval. A possible participation of the NO/Guanylyl cyclase/cGMP system also is suggested.

Cognition, Behavior, and Memory

### **Poster Number (174) Session 3**

#### Resistant fear memories to the disruptive effect of midazolam on memory reconsolidation: influence of D-cycloserine

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The retrieval of a consolidated memory results into a labile phase, which is vulnerable to the interference by benzodiazepines. The aim of the present study was to assess MDZ vulnerability after retrieval of a strong contextual fear memories or after retrieval of a d contextual fear memory in animals that had experienced a stressful situation prior to acquisition. Male Wistar rats were subjected on day 1 to a stressful session and on day 2 submitted to a contextual fear conditioning paradigm (3 shocks, 0.25 mA) and 7 days after training re-exposed to the training context (A) for 3 or 5 min. A separate group of rats were conditioned with a strong training protocol (5 shocks, 0.25 mA) and 7 days after re-exposed to the training context (A) for 3 or 5 min. Immediately after retrieval all rats were administered (i.p.) either with SAL or MDZ 1.5 mg/kg,. One day later, rats were tested in A. The results showed that MDZ does not affect reconsolidation of a 7-day fear memory in stressed animals and in stronger memories regardless of the duration of the re-exposure period and the MDZ doses used. In addition, we tested the influence

of pre-reactivation D-cycloserine (DCS) on MDZ's effect on fear memory reconsolidation. Our evidence showed that: a) Previous stress or a strong memory prevents MDZ's disruptive effect on fear memory reconsolidation and b) DCS prior to reactivation promotes retrieval-induced lability in such resistant memory traces.

Cognition, Behavior, and Memory

**Poster Number (175) Session 1**

Neonatal X irradiation: increases in PKC levels underlie behavioural changes

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Developing Central Nervous System is vulnerable to radiation-induced reactive oxygen species. The consequent oxidative stress is capable to produce changes at different levels in the hippocampus (Hip). We have found that irradiated rats (Rx) have better associative memory in the inhibitory avoidance test (IA). Different molecular targets could be involved in this behavioural change including protein kinase C (PKC), a well known kinase related with memory and learning processes as well as anxiety mechanisms. The aim of the present work was to evaluate the role of PKC in the behavioural changes induced by X-radiation. Neonatal male Wistar rats were X-irradiated (5 Gy) in their cephalic ends up to 48h of postnatal life. Elevated Plus Maze (EPM) as well as PKC total activity and isoforms levels were assessed in Hip of 30-days-old rats. We found a decrease in anxiety, as observed in the EPM, as well as an increase in total PKC activity and PKC  $\beta 1$  isoform levels in the Rx group. These results suggest that PKC could underlie behavioural changes induced by X-rays on the Hip. The better performance in the IA and the decrease in anxiety levels in the Rx group can be attributed to the increase in PKC activity, being the PKC  $\beta 1$  one of the isoforms that would account for this increase.

Cognition, Behavior, and Memory

**Poster Number (176) Session 2**

Role of muscarinic mechanisms in long-term memory storage vs long-term memory expression

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In the crab *Chasmagnathus*, long-term memory (LTM) expression -the process of taking control over behavior of a reactivated trace- may be considered a distinct attribute of LTM storage. Positive modulation during reconsolidation is proposed as an experimental approach to distinguish between memories that are not behaviorally expressed and those memories that were not stored at long term or altered in other retrieval mechanisms. In *Chasmagnathus* was described that the muscarinic antagonist scopolamine (SCP-0,1mg/kg), has amnesic effect when is administered either pre or post training: traditional interpretation is that SCP disrupt LTM storage. Here, we tested whether SCP amnesic effects can be explained as LTM storage deficit or LTM expression deficit. Results showed that the SCP amnesic effects are the consequence of a memory trace that is not behaviorally expressed but is potentially reactivated and labilized. However, a higher SCP dose (5mg/kg) induces amnesia that is result of a LTM storage deficit or an alteration in other retrieval mechanisms. The experimental approach allows us to dissect two different natures of amnesias induced by SCP in a dose-dependent way. This study would be relevant to interpret controversial results about the role of muscarinic mechanism in memory.

Cognition, Behavior, and Memory  
**Poster Number (177) Session 3**

### Electrophysiological markers in word association experiments: revisited

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In 1919 Carl Jung proposed the word association technique as a method to explore patient's brain states. Inspired by this classic idea in psychology, we used free word association to investigate the physiology of transitions between mental states. We recorded high density EEG while participants made free associations to a controlled list of frequent nouns in Spanish. Our main objective was to investigate which markers in the EEG are predictive of the semantic distance of the association. Results show that 1) there is a sequence of components which unfolds over 400 ms tightly locked to stimulus presentation, 2) the amplitude of a relatively early (300 ms) and phasic component correlates negatively with semantic distance 3) the amplitude of a late (500-600 ms) sustained component correlates positively with semantic distance and 4) Rhythmic activity in the alpha band prior to word presentation covaries with semantic distance. These results suggest a model by which the distance of the transition between states is determined by prior internal parametric states (simile to a temperature), decreases with the amplitude of a



component which indicates semantic content (like the depth of a potential well). The late marker component be a physiological marker of the transition, i.e. the excursion in semantic space.

Cognition, Behavior, and Memory

**Poster Number (178) Session 1**

GABA like receptors distribution in the central nervous system of chasmagnathus

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Gamma-amino butyric acid (GABA) is the most abundant inhibitory neurotransmitter in the central nervous system (CNS). A considerable amount of evidence showed the role of the GABAA receptor in diverse behavioral paradigms. We have already demonstrated that compounds that enhance the action of GABA impair the consolidation and reconsolidation processes, and compounds that reduce the action of GABA have the opposite action on the context learning in the crab Chasmagnathus. Our objective is to study the differential role of the GABAergic system in acquisition, consolidation, reconsolidation and extinction memory in this paradigm. In particular, we are interested in the plastic modification of the GABAergic system as consequence of the different memory processes. To achieve this objective it was necessary to characterize the GABAergic CNS in the crab. Here we show the first approaches in the study of the GABAergic system using commercial antibodies raised against GABA and the mammalian beta 2/3 subunit of the GABAA receptor in immunohistochemistry experiments and western blot assays. Taken together, these results show that it would be possible to analyze the plastic modifications suffered at receptor level as a consequence of the different phases or memory processes.

Neuronostatin administration impairs memory and induces anxiolytic effects in rats

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Neuronostatin (NST) is a 13 amino acid peptide encoded in the somatostatin (SOM) pro-hormone. It was recently discovered and it is produced throughout the body, in particular in brain areas known to be important in metabolic and autonomic regulation. It has been shown that NST have similar effects to SOM in feeding behavior effects; however, NST effects on memory, hippocampal excitability and anxiety have not been described. In the present work we investigated the effect of intra-hippocampal NST administration upon memory retention, hippocampal excitability, anxiety-like and feeding behavior in Wistar male rats. In our study, we have demonstrated acute intra-hippocampal NST administration induces anxiolytic effects and impairs memory retention, but it did not induce changes in food intake and drinking. We have also shown that NST 0.3 nmol/ $\mu$ l administration into the CA1 hippocampus decreased the threshold values to generate LTP, while that NST 3.0 nmol/ $\mu$ l administration blocked the LTP generation. These results provide behavioral evidence that NST may modulate higher brain functions, suggesting that the peptide influences anxiolytic and memory processes that involve the hippocampus and that the hippocampus don't participates in the NST effects on hunger at the doses studied.

Noonan syndrome animal model suggests mechanistic bases of gene dosage imbalance-dependent learning disability

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Human genetic disorders caused by gene dosage alterations usually produce lear-

ning disability. In the cases of trisomy, many studies have been seeking for the dosage-sensitive genes, such that the phenotype they confer is altered by gene-copy number. Studying the molecular pathogenesis of learning disability of Noonan Syndrome (NS) in a *Drosophila* model, we identify a potentially novel mechanism causing gene dosage dependent learning disability. The over expression of different clinical relevant Shp2 alleles (mutations associated with NS), as well as a wild type allele, produced learning disability. Such effect was caused independently of developmental alterations. Interestingly, the gene product of Shp2 was not required for normal learning ability, since the disruption of the endogenous Shp2 gene through RNA-interference (RNAi) did not affect learning ability. The capability of the RNAi to affect its target gene was confirmed by its effect on long-term memory. This evidence supports the idea that even when a gene is not necessary for normal learning, its over expression or an increase in protein function, can lead to learning disability presumably through unspecific interaction with the normal mechanisms. Support by: ANPCyT; FUNDALMA.

Cognition, Behavior, and Memory  
**Poster Number (181) Session 1**

Overcoming the amnesia caused by blockade of hippocampal muscarinic receptors by previous exposures to an open field

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Cholinergic muscarinic transmission in rodent hippocampus participates in learning and memory. We found that two, but not one, exposures of rats to an open field (OF), facilitates performance in a step-down inhibitory avoidance of an over-threshold footshock (IA). The muscarinic antagonist scopolamine injected intrahippocampus, caused amnesia. We evaluated the influence of OF exposures over IA in adult male and female Wistar rats maintained in an inverted light cycle. Rats explored the OF by 3 min; 24 h later they experienced a 2nd OF session; 1h later they were trained in IA (with threshold footshock). Immediately after training we injected scopolamine or saline bilaterally intrahippocampus; test was performed 24 h later. Scopolamine caused amnesia, but male and female rats previously exposed twice to the OF were not amnesic. The OF exposures did not improved IA performance under these conditions, at variance with rats trained with a stronger footshock. Although blockade of hippocampal muscarinic receptors caused amnesia, a previous experience allowed to overcome the influence of that blockade. This could be due to different previous synapse recruitment, task depending on same

brain structure (Ballarini et al., 2009), state of the system (day/night cycle), among other explanations.

Cognition, Behavior, and Memory

**Poster Number (182) Session 2**

Modeling information transfer in eusocial bees

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The understanding of collective decision making in honeybee (*Apis mellifera*) societies requires knowing the information that individuals acquire and store on the external environment. The social foraging of nectar involves a subtle coordination through recruitment displays (involving different sensory modalities) and the resource distribution within these social networks. In these decentralized information systems, individual experiences affect interactions between hive members. Although theoretical models address the relevance of these social interactions in the context of collective decision making, frequently they ignore the proper role that cognitive processes, such as learning and memory, could play in such collective phenomena. Therefore, the purpose of our project concerns the integration of these two aspects. With artificial neural networks as our conceptual framework, our goal is to model certain forms of individual experience and generate a plausible structure of the social network resulting from the resource distribution (empirical data suggest a hierarchical structure coupled with age-related subcastes, and hence, dynamic). We will show our preliminary results, for which we use a 1st generation Rescorla-Wagner model in a demographically structured population.

Cognition, Behavior, and Memory

**Poster Number (183) Session 3**

Asymmetrical contribution of brain structures to modulation of emotion as indicated by differential effects of right subgenual cingulum stimulation

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This report describes the recovery of a previously treatment-resistant depressive patient following right unilateral deep brain stimulation of Broadmann's area 25, along with partial recovery during bilateral stimulation and clinical worsening with left unilateral stimulation. The role of the right and left hemispheres in emotion regulation of depressive disorder pathogenesis and clinical recovery are discussed.

Cognition, Behavior, and Memory

***Poster Number (184) Session 1***

Frustration responses in psychogenetically selected rats: effect of partial reinforcement on instrumental and consummatory successive negative contrast in male roman high and low avoidance rats

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Frustration is induced by the sudden devaluation of a reinforcer in presence of greater reinforcement expectancies (Amsel, 1992). This emotional response seems to be similar to anxiety (Gray and McNaughton, 2000), and can be attenuated by loss similar previous experiences (Pellegrini et al., 2004). In this study we used instrumental (iSNC) and consummatory (cSNC) successive negative contrast and partial reinforcement (PR) procedures in order to compare the performance of two strains of rats psychogenetically selected on the basis of their emotional reactivity: the Roman High (RHA-I, low anxiety and high novelty seeking) and Low (RLA-I, high anxiety and low novelty seeking) Avoidance rats. In experiment 1 animals were exposed to a straight alley where they were changed from 12 pellets to 2 pellets. The iSNC only appeared in RLA-I rats, an effect that was abolished by previous PR experience. In experiment 2, CSNc was induced by reducing a sucrose concentration from 22% to 4%. The contrast effect was obtained in both RHA-I and RLA-I rats, and it was attenuated by previous exposure to PR. These data seem to support the implication of emotional responses in both SNC and PR situations, and suggest that instrumental procedures are more sensitive to differences in emotional responses between Roman strains.

Involvement of the glutamatergic neurotransmission in the stress and cocaine-induced reinstatement of extinguished cocaine-induced conditioned place preference in rats

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We have previously showed that MK 801 abrogated both, the development and the expression of stress-induced reinstatement in extinguished cocaine-induced conditioned place preference (CPP), in rats. This effect was context-dependent and persistently observed on subsequent cocaine-induced reinstatements (CR). Our goal was to determine if the memory reconsolidation process could be involved in the long lasting blockade of MK-801 on CR. Male Wistar rats (220-300g) were conditioned with cocaine (10-mg/kg ip) during four alternated drug/vehicle sessions and later extinguished with successive context/vehicle associations. In the reinstatement day, a group of animals was immobilized for 30-min while the control group was left undisturbed. Subsequently, all groups were tested in the CPP and, either immediately or 3 h after, injected with MK 801 (0.1 mg/kg ip) or vehicle, to be evaluated for CR following 3 days. MK 801 administered immediately after stress-induced reinstatement blocked the CR, whereas it was not observed when the animals were injected 3 h after the test. The long-term blockade of CR by MK 801 administered immediately after the first reinstatement could be attributed to a disruption of the glutamate-dependent memory reconsolidation process of the cocaine-induced CPP. Supported by: FONCYT, CONICET and SeCyT.

Calcineurin and nuclear factor of activated t-cells (NFAT) involvement in fear memory consolidation and extinction

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In fear conditioning, aversive stimuli are readily associated with contextual features. A brief re-exposure to the training context causes fear memory reconsolidation, whereas a prolonged re-exposure induces memory extinction. The regulation

of hippocampal gene expression plays a key role in contextual memory consolidation and reconsolidation. However, the mechanisms that determine whether memory will reconsolidate or extinguish are not known. We had already seen that NF- $\kappa$ B is required for fear memory reconsolidation. In the present work, we show that Calcineurin phosphatase (CaN) inhibits NF- $\kappa$ B, translocates to the nucleus and induces NFAT nuclear translocation, in the transition between reconsolidation and extinction. Accordingly, the hippocampal inhibition of both CaN and NFAT independently impairs memory extinction but not reconsolidation. We propose that CaN and NFAT are involved in the weakening of the original memory in extinction. We were also interested in study if inhibition of either of these two proteins has a facilitating role in consolidation of memory. Preliminary results show that inhibition of CaN immediately post training facilitates the consolidation of fear memory, whereas inhibition of NFAT shows no effect.

Cognition, Behavior, and Memory

**Poster Number (187) Session 1**

Attending a direction increases the performance in the opposite direction

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Previous studies showed that feature-based attention increases the selectivity of population responses in primate visual cortex. They found that a direction selective cell response increases when the attention to a particular direction of motion coincides with their preferred direction regardless of the stimulus direction presented on its receptive field. We wonder whether this effect has some kind of perceptual correlate. We performed an experiment in which three patches were displayed. Subjects were asked to respond, while attending to a patch containing a random dot pattern which could be moving coherently in some direction  $\theta$  (Condition A), or randomly (Condition B), about two other patches, one of them having some dots moving coherently in the opposite direction of  $\theta$ , and the other with no coherent movement at all. She/he had to decide which patch had coherent movement. We used a masking technique to desensitize the neurons that have the opposite direction of  $\theta$  as their preferred direction in order to relatively increase the response of neurons that have  $\theta$  as their preferred direction. Results show that, instead of what the feature-based hypothesis would predict, performance in Condition A is higher than in Condition B.

Cognition, Behavior, and Memory

**Poster Number (188) Session 2**

Social functioning and cognition in patients with schizophrenia, their unaffected siblings and healthy controls: impact on quality of life

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Patients with schizophrenia show deficits in many cognitive domains and social functioning, social skills, and self management skills in their daily life activities. The present study evaluated the relationship between general and social cognition, mental state and social functioning, and impact on quality of life. 22 patients with chronic disorder of schizophrenia were evaluated and compared with 19 first-degree relatives and 19 healthy controls. The assessments used were: Word Accentuation Test, MCCB (Matrices Consensus Cognitive Battery), UPSA (University of California Performance Skills Assessment), TABS (Test of Adaptive Behavior in Schizophrenia), SSPA (Social Skills Performance Assessment), PANSS, SF36. Patients showed significant differences from controls and siblings in MCCB total score (p

Cognition, Behavior, and Memory

**Poster Number (189) Session 3**

Emotional reactivity in adolescent animals with or without prenatal ethanol exposure

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Problematic use of alcohol, which mainly starts at early adolescence, is associated with a heightened probability of Alcohol use Disorders later in life. We recently underscored the facilitative effects of gestational alcohol on adolescent alcohol affinity (Fabio et al., 2010). This study aimed at analyzing differences in emotional reactivity underlying this facilitative effect. Pregnant Wistar rats were given ethanol (2 g/kg, ig) or vehicle on gestational days 17-20. Their adolescent offspring was challenged with ethanol (2.5 or 0.0 g/kg) or left untreated and screened for motor



activation in an open-field arena. Afterwards, they were assessed for appetitive (50-60khz) and aversive (20-30khz) emission of ultrasonic vocalizations (USVs). We observed ethanol-induced activation and heightened USVs (both appetitive and aversive) in animals challenged with ethanol, and these effects did not differ across prenatal treatments. A subsequent experiment revealed similar blood alcohol levels at adolescence, after ethanol or vehicle prenatal exposure. Under these experimental conditions, the facilitative effects of gestational alcohol exposure on adolescent alcohol affinity cannot be explained in terms of differential emotional reactivity caused by prenatal exposure.

Cognition, Behavior, and Memory

**Poster Number (190) Session 1**

Qualitative and quantitative study of cognitive functioning in multiple sclerosis

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Multiple Sclerosis (MS) patients show a pattern of neuropsychological dysfunction, with deficits in long term memory, working memory and other executive functions, among other symptoms. These abilities are necessary for the acquisition of "equivalence relations" (ER), which is a paradigm that has been widely used to study the formation of categories. The aim of the present study is to evaluate MS patients with a single case methodology, in order to provide converging evidence to previous studies that reported an association between performance on categorization tasks and executive skills in both healthy subjects and neurological patients (O'Hora et al.). Five patients with clinically definite MS (2 males and 3 females) with ages between 29 and 54, and an educational range between 11 and 17 years, were evaluated with an ER task and the Brief Repeatable Battery in Multiple Sclerosis (BRB-MS). The results showed a strong relationship between the performance on ER task and working memory skills, independently of global cognitive impairment. References: O'Hora, D.; Pelaez, M.; Barnes-Holmes, D. (2005) Derived Relational Responding and Performance on Verbal Subtests of the WAIS-III. *The Psychological Record*. 22:155-175

Molecular mechanisms in hippocampus and basolateral amygdala but not in parietal or cingulate cortex are involved in extinction of one-trial avoidance learning

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The extinction of one-trial avoidance involves the dorsal hippocampus (DH) and basolateral amygdala (BLA), two areas that participate in its original consolidation. The posterior parietal (PARIE) cortices also participate in consolidation of this task but their role in extinction has not been explored. Here we study the effect on the extinction of one-trial avoidance in rats of different drugs infused into DH, BLA or PARIE 5min before the first of four daily unreinforced test sessions: the NMDA receptor antagonist, AP5 (5.0microg/side), and the inhibitors of calcium-calmodulin dependent kinase II (CaMKII), KN-93 (0.3microg/side), or of the cAMP-dependent protein kinase (PKA), Rp-cAMPs (0.5microg/side) hindered extinction when given into DH or BLA. Levels of pPKA and pCaMKII increased in DH after the first extinction trial; in BLA only CaMKII increase was seen. Thus, this pathway appears to participate in extinction in BLA at the "basal" levels, and at enhanced levels in DH. None of the treatments affected extinction when given into PARIE. The present findings indicate that DH and BLA are important for the extinction at the time of the first unreinforced retrieval session and that CaMKII and PKA signaling pathway are necessary for the development of extinction.

Labilize or not labilize? That is the question

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Once a memory is consolidated, the presentation of a reminder can induce reconsolidation, a process that has inherent stages: reactivation, labilización and resabilization of the memory trace. We are studying the labilization process in two contextual associative models in phylogenetically distant species: context-signal memory in the crab *Chasmagnathus* and fear conditioning in mouse. Here, we de-

veloped firstly a pavlovian-conditioning protocol in crabs designed to increase the contingency between the context and the stimulus. We found that this new paradigm is context specific, and implies protein synthesis and GABA receptors. We also found that a brief reexposure is sufficient to induce the reconsolidation, which is dependent on NF- $\kappa$ B activation. These results demonstrate that the protocol is suitable for the study of memory labilization. In the second part of this study, we analysed the role of the proteasome system in memory labilization. A previous study indicated that the labilization process involved protein degradation by the proteasome. Here we found that the inhibition of the proteasome system block labilization, impairing drugs amnesic effect during memory reconsolidation.

Cognition, Behavior, and Memory

**Poster Number (193) Session 1**

Sildenafil promotes cocaine sensitization and enhances hippocampal LTP

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Phosphodiesterase 5 inhibitors (PDE5-i) are blockers of the cGMP-specific enzyme that hydrolyzes cGMP. The recreational use of PDE5-i has been described and linked to other illicit drugs use. Nitric oxide (NO) activates the GC/cGMP signaling pathway. Cocaine (COC) sensitization is associated to nitric oxide synthase activity and enhancement of hippocampal synaptic plasticity (HSP). Then, we can hypothesize that the NO/cGMP signaling pathway in the brain can initiate, contribute or exacerbate addictive behaviors, being PDE5-i potential candidates for these actions for them selfs or when they are used concomitantly with other drugs. In the present work, we administrated rats with the PDE5-i, sildenafil, before COC along 5 days, in order to examine the impact of the inhibition of cGMP degradation in the development of COC sensitization and the associated HSP. Our results indicate that sildenafil enhances HSP and when it is co-administered with COC increases the percentage of sensitized rats, being this increase correlated to an enhanced HSP. These findings suggest that NO/cGMP signalling pathway has an important role in the development of COC sensitization and in the changes induced by COC on HSP that may contribute to the behavioural effects after repeated administration.

Cognition, Behavior, and Memory  
**Poster Number (194) Session 2**

Interaction between a consolidated fear memory and a stressful situation during reactivation. Pharmacological manipulation in the basolateral amygdala

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Consolidated memories may result into a labile one after retrieval. It was proposed that this memory plasticity can allow incorporate new environmental information to the original trace. One of the aims of the present research was to assess whether a stressful stimulus prior to reactivation would influence such trace. Adult male Wistar rats were subjected to a contextual fear conditioning paradigm using a single footshock (weak training session). One day after training, rats were subjected to a stressful situation (restraint for 30 min.). Half of the rats were re-exposed to the original context of conditioning (test 1) for 3 minutes one day after the stress. There was an increase of freezing only in those animals re-exposed to the associated context, which persisted in test 2 performed ten days after stress exposure. Midazolam intra-basolateral amygdala (BLA) previous to stress prevented such increase. Similarly, NMDA antagonist intra-BLA prior to reactivation attenuated stress-induced increase of freezing. The intra-BLA infusion of an inhibitor of protein degradation (B-lac) did not prevent the enhancement of fear memory in stressed animals. Although there is a clear interaction between stress and fear memory that leads to an improved fear memory, such increase seems not to be dependent on protein degradation in BLA.

Cognition, Behavior, and Memory  
**Poster Number (195) Session 3**

Socrates's teaching brain: the meno experiment

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All animals learn but, with only very minor exceptions, only humans teach. Paradoxically, while neuroscience and psychology have extensively investigated the workings of the learning brain, very little is known about the teaching brain. Learning and teaching are entangled operations. One of the first descriptions of this process is the famous dialo-

gue between Socrates and a young slave of Meno, reported by Plato 2400 years ago to prove that “nobody learns from a teacher” but only remembers (anamnesis). In fact, this dialog has been considered –without ever being submitted to explicit proof, as one of the greatest icons and landmarks in the history of education. To empirically determine the plausibility and universality of the dialog, we presented an almost literal version of it to educated adults and adolescents. Our results show a remarkable agreement between Socratic and empiric dialogs. To examine the effectiveness of the Socratic dialog to generalization, after concluding the dialog we posed participants the same question which drives the dialog (how to double the area of a square), but changed its scale. Almost half of the participants failed to respond correctly, indicating that strict following of the dialog does not lead to abstract learning.

Cognition, Behavior, and Memory  
**Poster Number (196) Session 1**

Formation and consolidation of an aversive memory: role of the medial prefrontal cortex

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Systems consolidation establishes that memories are initially stored in the hippocampus and later transferred to the cortex for persistent storage. Nevertheless, an alternative view proposes that the neocortex may have a crucial role from initial steps of memory formation. Using c-Fos immunocytochemistry, we studied the activation of cortical areas produced by an inhibitory avoidance (IA) training in rats. We found that after a strong training, which generates a persistent memory, certain cortical regions are selectively activated, including the medial prefrontal cortex (mPFC). Since this activation might indicate sites involved in memory processing, we decided to study the role of mPFC in the formation and storage of aversive memories. We infused anisomycin or emetine (protein synthesis inhibitors) in mPFC near the time of training and 12h later. These treatments produced amnesia for IA learning measured 2, 7 or 14 days after acquisition. These results indicate that protein synthesis during both time periods are necessary for memory formation of this task and suggest a potential role for mPFC in memory processing at both early and late stages.

Neurophysiology of subjective confidence in a partial report paradigm

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A vast ensemble of stimuli are continuously being processed in parallel by the sensory system, most of which elicit only a brief transient sensory response which fades after few hundreds of milliseconds without reaching working memory or consciousness. This sensory representation is referred as iconic memory. Previously we found a strong dissociation between the objective performance and the subjective confidence in the response in a partial report paradigm,, allowing the understanding of the brain dynamics in the construction of subjective confidence. Here we report a high-density EEG experiment in which we infer elements of the EEG response which are indicative of subjective confidence. We found that an early response during encoding partially correlates with perceived confidence. However, the bulk of the weight of subjective confidence is determined during a late, N400-like waveform, during the retrieval stage. A reconstruction of the cortical source locates the N400 waveform at the occipito-temporal cortex. This experiment shows that we can find markers of access to internal, subjective states, that are uncoupled from objective response and stimulus properties of the task, and we propose that this can be used with decoding methods of EEG to infer subjective mental states.

NMDA like receptors in chasmagnathus granulatus

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The NMDA glutamate receptors (NMDARs) mediate a majority of excitatory neurotransmission in the nervous system. Molecular and electrophysiological properties suggest that they may be the Hebbian´ coincidence detectors, and has

been implicated in synaptic plasticity, memory acquisition and learning, in both vertebrates and invertebrates. In the crab *Chasmagnathus granulatus*, the systemic administration of NMDAR antagonists (MK-801, APV) affects the consolidation and reconsolidation of the Context-Signal Memory, indicating that NMDA like receptors have been involved in long-term memory in this invertebrate model. Therefore, the first step was the characterization of the NMDA like receptors in the crabs. Immunohistochemistry techniques were applied to identify the distribution of the NR1 and NR2 like receptor subunits through the nervous system. With Western Blot techniques we evaluated the antibody specificity, as well as the expression pattern and molecular weight of these receptor subunits. The variation of the receptors number or their membrane insertion, as consequence of learning y/o drug treatments was studied.

Cognition, Behavior, and Memory

**Poster Number (199) Session 1**

Is there a role for opiates in mediating acetaldehyde's (ACD) unconditioned properties during early ontogeny?

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ACD, the first metabolite of ethanol, produces ethanol-like effects such as hypothermia, motor stimulation, anxiolysis, conditioned-place preferences and conditioned taste aversions. Animals self-administrate large amounts of ACD directly into the brain. However, little is known about ACD effects during early ontogeny. The catalase system (wich metabolize EtOH in the brain) has a progressive fall across ontogeny. Intracysternal administration of EtOH supports appetitive conditioning in newborn rats. This phenomenon is blocked when the catalase system is inhibited. Recently, we have found that IC administration of ACD also supports appetitive conditioning. Here, we analyzed the role of opiates in the reinforcing strength of ACD. Newborn administered with naloxone (0 or 5 mg/kg, s.c.) were then exposed to lemon odor in contingency with ACD (0 or 0.35  $\mu$ mol, i.c). One hour later, pups were tested with an artificial nipple scented with lemon odor. Mean grasp duration (MGD) was considered as dependent variable. Data indicated that MGD was increased when lemon was paired with ACD's effects, verifying appetitive responsiveness mediated by this metabolite. Nevertheless NLX failed to modify attachment parameters. Further studies will help to unravel the mechanisms in ACD effects.

Anxiolytic effect of testosterone in an animal model of frustration

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Sexual response has anxiolytic effects on behavior. The evidence shows that one of the possible mechanisms involved in this phenomenon is the increase in the release of testosterone (T) produced by the sexual situation, because the finding shows that systemic and intracerebral administration of this hormone causes anxiolytic effects. Frustration situations that involve reductions or omissions of appetitive reinforcers cause anxiety reactions and are sensible to the administration of anxiolytics and whether the animals were able to ejaculate or be with a receptive female before the downshift of a reward in a consummatory successive negative contrast procedure (cSNC). The aim of the present study was evaluated the T's effect in the reduction or omission of an expected appetitive reinforcer. With this objective we realized a chronic administration of 25mg/kg T in a cSNC and consummatory extinction (cE) procedures. The findings suggest that the subjects with T's administration show a minor frustration effect and a faster recovery than control animals in the cSNC and a faster extinction in the cE. The results are discussed in relationship with the emotional testosterone's effects and the functional equivalence between aversive stimulus and omission of an appetitive stimulus.

Memory updating during reconsolidation in the crab chasmagnathus

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The reconsolidation phenomenon describes that a stable and consolidated memory recalled by a reminder enters a vulnerability phase (labilization), followed by a process of re-stabilization (reconsolidation). Although much has been learned regarding the mechanisms of reconsolidation, the search for an endogenous function of this process remains a fundamental question. It has been suggested that reconsolidation might enable memories to be modified or updated. Here, we



address this issue in the framework of reconsolidation of an aversive contextual learning in the crab *Chasmagnathus*. Our preliminary results show that after being reactivated by a reminder, crabs can incorporate new information to an active memory trace. Interestingly, this information consists of an appetitive stimulus, which also elicits a contextual association. Thus, we hypothesize that by means of the labilization-reconsolidation mechanism, a same context could be able to support two opposite meanings in a same memory trace.

Cognition, Behavior, and Memory

**Poster Number (202) Session 1**

Chronic exposure to paraquat induces changes in body weight gain, anxiety like behavior and non social olfactory discrimination

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Recently, paraquat (PQ) has been described as a potential environmental neurotoxin associated with risk for neurodegenerative diseases often developing after chronic exposure. It is known that chronic PQ exposure induces changes in the neurobehavior compromising the motor and sensitive functions. The aim of this study was to evaluate the possible cognitive and sensitive changes weekly in an experimental model of PQ toxicity during a month. Materials and Methods: SD female rats (n=12) were divided in two groups which received either PQ (10 mg/Kg i.p.) or saline once a week during four weeks. Body weight gain, anxiety like behavior and non social olfactory discrimination were monitored throughout the treatment. Results: PQ treated and control mice gained body weight on average. However, the body weight gain of the PQ-treated group was lower than control groups in the last week (p

Zebrafish as a model for addictive behaviors

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Rats and mice were extensively used to study the addictive properties of drugs of abuse. In recent years, zebrafish (*Danio rerio*) was increasingly utilized in neurobehavioral research, because it has proved to be a reliable vertebrate model system and is an excellent model for use in forward genetics. Extensive research has shown that there are genetic, neural, and endocrine homologies between zebrafish and mammals. Nicotine is the principal psychoactive component present in tobacco and is responsible for the reinforcing properties that lead to dependence, addiction and high incidence of relapse. Addiction studies in rodents are done using two experimental models: the conditioned place preference (CPP), and drug self-administration. Using a CPP paradigm, and knowing that zebrafish has a natural preference for dark environments, we setup a biased procedure to evaluate nicotine-induced behaviors. After conditioning, treated fishes showed a significant increase in the time spent in the drug-paired side. This results show that adult zebrafish is a good model to study the rewarding properties of nicotine and support the possible use of zebrafish as a model to understand the genetic and molecular mechanisms underlying addictive behaviors.

A dopamine antagonist impairs an aversive memory with a narrow window of effect in the crab *Chasmagnathus*

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Several studies in insects converge on the idea that dopamine (DA) mediates the negative reinforcement in aversive conditioning. Here we studied the role of DA in a novel aversive learning paradigm in the crab *Chasmagnathus granulatus*. It is shown that the administration of chlorpromazine (D2-like DA antagonist) 30 min before training impairs memory retention at test session, performed the next day. However, this amnesic effect is not found when the drug is applied 15 or 45

min before training or 1h after it, revealing a very narrow time window of effect. These results are in agreement with the idea that DA could act as the internal reinforcement of the aversive stimulus in associative learning. Our next step will be to analyze the action of DA in an appetitive paradigm, with the hypothesis that it would interfere with the consolidation of an appetitive memory. As it was found for octopamine (Kaczer & Maldonado, 2009), another biogenic amine, we propose that DA would have a contrasting role in memory processes of opposite sign.

Cognition, Behavior, and Memory  
**Poster Number (205) Session 1**

A new player: the role of retrosplenial cortex in memory processing

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A widely accepted view concerning memory processing (termed “system consolidation”) suggests the existence of a dialogue between the hippocampus (Hp) and other regions. Although the Hp is crucial in the formation of new declarative memories, other brain regions probably mediate permanent storage of remote memories. We identified changes in the expression of the immediate early gene (IEG) c-Fos at 1, 12 and 24 hours after inhibitory avoidance (IA) training in the retrosplenial cortex (RSCx) by immunohistochemistry. Taking into account these results, and because this region is connected with the Hp, we studied the expression of plasticity-related proteins. Using western blot analysis, we performed a temporal expression profile of IEGs in RSCx of naive, shocked or IA-trained animals. There is an increase in IEG protein levels at specific times after learning, but not after a non-associative shock. We are currently performing pharmacological experiments to clarify the role played by RSCx in these mechanisms, and to define functional RSCx-Hp interactions. These results suggest a role for this cortex in memory formation or storage.

Hippocampal  $\alpha 7$  nicotinic receptors modulate memory reconsolidation of an inhibitory avoidance task in mice

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CF-1 male mice were trained in an inhibitory avoidance task using either a mild or a high footshock. A retention test was given 48 hours later. Immediately after it, mice were given intra-dorsal hippocampus infusions of either choline (Ch, an  $\alpha 7$  nicotinic acetylcholine receptor agonist, 0.08–1.30  $\mu\text{g}/\text{hippocampus}$ ), or methyllycaconitine (MLA, an  $\alpha 7$  nicotinic acetylcholine receptor antagonist, 1.0–30.0  $\mu\text{g}/\text{hippocampus}$ ). Memory retention was tested again 24 h later. MLA impaired retention performance regardless of footshock intensity and its effects were long lasting. Ch impaired retention performance only in those mice trained with a high footshock. On the contrary, Ch enhanced retention performance when mice were trained with a mild footshock. These effects were long lasting and dose- and time-dependent. Retention performance was not affected in drug-treated mice that were not subjected to memory reactivation, suggesting that the performance effects could not be attributable to non-specific effects of the drugs. Methyllycaconitine effects were dose-dependently reversed by choline. Our results suggest that hippocampal  $\alpha 7$ nAChRs play a critical role in reconsolidation of an inhibitory avoidance response in mice, and may also have important implications for dynamic memory processes.

The role of awareness in trace conditioning learning and expression

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Pavlovian trace conditioning depends on the temporal gap between the conditioned and unconditioned stimuli. While generally trace conditioning requires explicit knowledge of the temporal contingency, certain exceptions have been reported using emotionally salient subliminal stimuli as conditioned stimulus. Fear trace conditioning can be learned even when the conditioned stimulus is masked only when it has a strong emotional content. The objective of the current experiment is to delimit the role of consciousness in trace learning. Specifically we investigate whether 1) A subliminal (hence unconscious) stimulus may elicit a trace response when the explicit rule is conscious and 2) whether extensive practice may bypass the necessity of consciousness. We have designed a modified version of the eye-blink trace conditioning, using words as CS+ and CS- stimuli. Compression of these words in a stream of distracters can be used parametrically to control the conscious access of these words. The trace task is performed with a concurrent task (tone discrimination) to factor attentional contributions. Preliminary results show that after conscious conditioning and explicit acquisition of the contingency, a subliminal abstract stimulus can trigger a trace response.

Cognition, Behavior, and Memory

**Poster Number (208) Session 1**

Novel localization of NF-kappa B in mouse forebrain synaptic terminals.  
Activation dynamics during memory consolidation

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NF-kappa B activation has recently been shown to be necessary for long-term memory consolidation and reconsolidation, in the inhibitory avoidance learning in the mouse hippocampus. NF-kappa B is activated in the cell nucleus in specific temporal windows in both consolidation and reconsolidation. Previous results showed that the transcription factor is also present in synaptic terminals. This work focuses in the dynamics of its synaptic activation during consolidation, in two different synaptic localizations: soluble in the synaptic content and, in a pool described for the first time in this work, bound to the cell membrane. The function of this novel localization is discussed.

Cognition, Behavior, and Memory

**Poster Number (209) Session 2**

Neuroplasticity: changes in grey and white matter structure induced by long-term motor learning

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Learning a motor skill is reflected in structural changes, such as increases of dendritic arborization and the generation of new anatomical projections. At a macroanatomical level, structural changes can be detectable through the use of non-invasive magnetic resonance imaging techniques. This project was aimed at detecting changes in brain structure induced by motor skill training. For this purpose, we obtained T1 and diffusion weighted images from a group of 12 participants before and after one week of training on a visuomotor adaptation task with their right hand. On average, adaptation was achieved within 4 days and was accompanied by an increase in grey matter density in a region of the left primary motor cortex, consistent with the trained hand representation. In addition, we found an increase in the fractional anisotropy (FA) of the corticospinal tract and in the superior longitudinal fasciculus. FA is thought to reflect greater myelination or the outgrowth of axons collaterals and dendrites. These results indicate that specific alterations in grey and white matter can occur rapidly with only one week of training and they constitute the first experimental evidence that the formation of a new visuomotor map is associated with the structural modification of fronto-parietal cortices.

Cognition, Behavior, and Memory

**Poster Number (210) Session 3**

Mental imagery and emotion. A neurophilosophical approach to literary aesthetics

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In the Republic Plato inaugurated the philosophical study of artistic aesthetics. One of his enduring contributions was to show that aesthetics is dependent upon an explicit theory of mind. In the last decade neuroaesthetics set forth to uncover the neuronal substrate and mechanisms of artistic beauty and artistic effects (Chatterjee: 2010, Skov & Martin: 2009), with a focus on pictorial (Cela-Conde,

et al: 2004, Nadal, et al: 2009, Zeki: 1999, Ramachandran: 2004) and musical (Blood & Zatorre: 2001, Levitin: 2006, Zatorre & Halpern: 2005) artworks. Here we offer a philosophical ground for neuroaesthetics and expands its realm by presenting a model for literary neuroaesthetics based on the relationship between mental imagery and emotions.

Cognition, Behavior, and Memory

**Poster Number (211) Session 1**

Nonassociative plasticity alters competitive interactions among mixture components in early olfactory processing

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Nonassociative plasticity modifies olfactory processing both in the mammalian olfactory bulb and in the insect antennal lobe (AL). However, mechanisms and effects of the changes are not well understood. Using calcium imaging in the honey bee AL, we show that stimulation with a binary odor mixture (A+X) sets up a spatiotemporal activity pattern that is intermediate to the patterns for the two pure components. Unreinforced exposure to one component (A) shifts the pattern for the mixture such that it becomes less similar to the pattern for A and more like the pattern for X. The results suggest that nonassociative plasticity modifies the neural network in the AL in a way that affects local, competitive interactions among mixture components. We also tested in behavioral analyses that unreinforced exposure of A causes the mixture A+X to become perceptually more similar to X. We then use a computational model to evaluate the most likely targets for modification of the network. We show that hebbian modification of synapses from inhibitory local interneurons to projection neurons most reliably produces the observed shift in the pattern for the mixture. These results are consistent with a model in which plasticity in the AL acts to filter out irrelevant cues in complex olfactory stimuli.

Alterations in the inflammatory response in a mouse model of autism

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Autism spectrum disorder (ASD) is a severe neurodevelopmental disorder characterized by significant impairments in social, communicative, cognitive and behavioral functioning. Based on recent work, we hypothesized that glial activation is a common factor underlying different proposed etiologies for ASD. To investigate the inflammatory and glial alterations in a mouse model of ASD, we injected pregnant mice with 600, 400mg/kg valproic acid (VPA) or saline at E13. VPA-treated mice showed a delay in the development of the righting reflex, and a decrease in social interaction and an increase in anxiety-related behaviors in adulthood. These results validate our mouse model of ASD. Interestingly, animals prenatally exposed to VPA showed an enhanced response to an inflammatory challenge in adulthood, in comparison with control animals. A LPS dose that failed to elicit an inflammatory response in control mice, induced in turn a significant increment in plasma corticosterone and in the spleen levels of the pro-inflammatory cytokines IL-1b and IL-6 in the spleen. However, IL-6 levels in the hippocampus were augmented 2hs after LPS injection in all experimental groups. These results show that animals treated with VPA at E13 show altered behavior related to ASD and an exacerbated inflammation.

Prior stress exposure facilitates the behavioural and molecular changes induced by a weak fear conditioning procedure

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Exposure to threatening stimuli results in excessive fear and anxiety responses, a process defined as Emotional Sensitization. The Basolateral Complex of the amygdala (BLA) plays a key role in the processing of aversive information. It is known that the amygdalar MAPK signaling pathway is crucially involved in the



formation of fear memories. The purpose of this study was to assess the stress influence on the behavioural and neurochemical changes associated with weak contextual fear training (CFC). Male Wistar rats (280-300g) were used to perform the experiments. Animals were injected either systemically or intra-BLA with midazolam (MDZ) prior to restraint, and one day later subjected to CFC (0,3mAx1 footshock). The behavioural test was conducted 24 h after CFC. We observed high freezing levels only in VEH stressed animals, and MDZ prevented the enhanced fear expression induced by stress. Western blot showed a significant pERK2 increase in BLA 30 min after CFC just in stressed animals. This effect was attenuated in MDZ pretreated rats. We suggest that ERK2 activation in BLA would be necessary to install the negative emotional state induced by stress whereas stimulating BLA GABAergic neurotransmission impaired the promoting influence of the aversive stimulus.

Cognition, Behavior, and Memory

**Poster Number (214) Session 1**

Ghrelin inhibited serotonin release from hippocampal slices

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Ghrelin (Ghr) is a peptide produced peripherally and centrally. It participates in the modulation of different biological processes. In our laboratory we have shown that a) Ghr administration, either intracerebroventricular or directly into the hippocampus(Hi) enhanced memory consolidation in a step down test (SDT) in rats b) the effect of Ghr upon memory decreases in animals pretreated with a 5-HT reuptake inhibitor fluoxetine suggesting that Ghr effects in the Hi could be related to the availability of 5-HT. It has been demonstrated that Ghr inhibits 5-HT release (R) from rat hypothalamic synaptosomes. Taking in mind these evidences, we studied the R of radioactive 5-HT to the superfusion media from hippocampal slices treated with two doses of Ghr (0.3 and 3nm/ul). Ghr inhibited significantly the 5HT R in relation to those superfused with artificial cerebrospinal fluid (ACSF) ( $33.2 \pm 4.3$  vs  $1.8 \pm 1.8$  Ghr 3). In a other set of experiments, Ghr was infused in the CA1 area of Hi of the rats immediately after training in the SDT and the 5-HT R from slices was studied 24 hs after Ghr injection showing that in this condition also the 5HT R was inhibited ( $107.0 \pm 11.4$  vs  $2.5 \pm 2.5$  Ghr 3). In conclusion, results provide additional evidence about the neurobiological bases of Ghr action.

Effect of MK-801 on fear conditioning and subsequent ethanol-induced conditioned place preference in ethanol withdrawn rats

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Withdrawal from chronic ethanol (ETOH) administration facilitated the formation of contextual fear memory. The retrieval of such memory induced a significant increase in ETOH consumption and the expression of conditioned place preference (CPP) induced by ETOH solely in withdrawn rats. Here we examined whether the NMDA receptor antagonist MK-801 given prior fear conditioning would attenuate both the fear response and the subsequent expression of CPP induced by ETOH in withdrawn rats. Male rats made dependent via an ETOH containing liquid diet for 14 days and the basal test for CPP was performed 3 days after withdrawal. The next day, rats were injected with MK-801 (0.1 mg/kg ip) prior to fear conditioning and the CPP conditioning began 24 h after training. One day after the last conditioning CPP session, the fear response was evaluated and the CPP test was performed 2 h later. ETOH produced neither CPP nor place aversion in both control and ETOH withdrawn non-fear trained rats. MK-801, at a dose that did not affect the acquisition of contextual fear conditioning in control rats, prevented the increase of fear response and blocked the subsequent CPP induced by ETOH in withdrawn rats. These effects were not observed in withdrawn rats that did not receive fear memory recall.

A new paradigm to study speech recognition: semantic bistability

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In this work we explore a new form of perceptual bistability in speech recognition. We present the subjects with a continuous stream of alternating pairs of syllables: TACOTACOTACOTACO. This string can either be parsed as a sequence of TACOs or as COTAs which results in an ambiguity that produces alternations between the

two interpretations. Changing the time interval between syllables we can bias the subject's perception towards either TACO or COTA. As a first result we show that the phenomenon exhibits all the characteristic traits of perceptual bistability: 1) The mean duration time of each percept follows Levelt's propositions (a set of rules derived for binocular rivalry which have been shown to hold for all known bistable stimuli), 2) The distribution of perception times for one interpretation is a log-normal distribution, 3) The duration of successive perception times for the same interpretation are not correlated. Additionally we found that the dynamics of the oscillations is affected by lexical characteristics of the rivaling words. This makes this type of stimuli particularly interesting because they can be used to probe the internal organization of the lexicon as well as constrain possible mechanisms for spoken word recognition.

Cognition, Behavior, and Memory

**Poster Number (217) Session 1**

Visual behavior during free viewing of images from different categories

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A number of studies have examined visual exploration to establish a relationship between eye movements and stimulus properties, but recent evidence suggests that cultural and cognitive factors affect visual behavior. The aim of this work is to establish whether there are differences in visual behavior in images from different categories (natural or artificial). A total of 8 subjects freely viewed 7 different image categories construction, landscape, fractal, pink and white noise, gray or blank images; their eye movements were recorded with an eye-tracking system. A Kruskal-Wallis test between fixation duration distributions of the pair landscape-fractal images ( $p \sim 0.9$ ) produced a quantitatively similar distribution, while construction-landscape have a much weaker relationship. The mean number of fixations for each type of image was significantly different (c.i. 95%). Even though pink noise images share most frequency components as natural images due to its low frequency components, the mean number of fixation in landscape was significantly different from pink noise (t-test p

Opioid system blockade inhibits operant behaviors in an infantile model of ethanol self-administration task

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This study analyzed ethanol operant self-administration, in infants. Involvement of opioid system in the acquisition or expression of this experience was inquired. Infant rats (PDs14-17) have to display a target behavior (nose-poke) to gain access to 5% sucrose, 3.75% ethanol or water. On PD18 an extinction session was included. Sucrose and ethanol promoted higher levels of responses than water. During extinction, seeking behavior was only observed in pups reinforced with sucrose or ethanol. In a second study, only ethanol was used as reinforcer. At PDs16-17, 6-hr before training, pups were re-exposed to ethanol under opioid antagonism effects (naloxone, 1 mg/kg). During training, pups pre-exposed to ethanol under naloxone effects, failed to increase nose-poking responses to ethanol. Re-exposure conditions had no effects at extinction. In a third experiment a re-exposure trial was included at PD18. Prior extinction, pups were injected with naloxone and re-exposed to ethanol. The main result was a reduction of seeking behavior during extinction, in pups re-exposed to the drug -under naloxone effects- at PDs16, 17 and 18. These results indicate that opioid system seems to be modulating ethanol reinforcing effects, in terms of both appetitive and consummatory behaviors.

Cognition, Behavior, and Memory

**Poster Number (219) Session 3**

Visual context affects decision-making of the escape direction in the crab chasmagnathus

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Crabs are elusive prey with a short-latency (~100 ms) escape behavior, a critical feature to avoid predation. To a sudden approaching object, all crabs react by running in the opposite direction. Thus, the direction of escape is highly determined by the direction of the object approach. However, when an object is coming straight from above, the proper side to run becomes less certain. Even so, the ani-

mal must decide which side to run (crabs run sideways). We began to investigate this decision making processes, which to a large extent is thought to take place in a group of identified giant neurons of the crab's brain. We used a running simulator device located inside an arrangement of 5 computer screens surrounding the animal. This allows us to precisely record the locomotor responses elicited by the image of an approaching object (an expanding black square) generated in the upper screen. We found that when the luminance of the screens surrounding the crab at the horizontal level were equal, 50 % of the animals ran to the left and 50 % to the right. Upon repeated stimulus presentations, each individual tended to maintain the same escape direction, indicating the existence of individual directional preferences. On the other hand, upon different situations of visual contextual asymmetries, we found in several cases differences in escape side chosen by the animals. These behavioral results establish solid bases for begin investigating the neuronal mechanisms involved in such decision making processes.

Cognition, Behavior, and Memory

**Poster Number (220) Session 1**

The novel peptidic toxin TX3-1, extracted from the venom of the spider *Phoneutria nigriventris*, rescue memory deficits of mice submitted to a model of Alzheimer's disease

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A-type K currents (IA) have main role in mnemonic process, regulating neuronal excitability and synaptic plasticity. In an animal model of Alzheimer's Disease (AD) IA are increased, thus compromising the synaptic transmission. The present study aimed to evaluate the effect of Tx3-1 in memory of naïve and in mice submitted to a model of AD. Male Swiss mice were submitted the experimental model of AD (A $\beta$  peptide 25-35, 3 nmol), and the behavioral test was carried on 6 days after injection of A $\beta$  peptides. Cognitive behavior was evaluated through object recognition paradigm. Immediately after training session, Tx3-1 (10 - 300 pmol/site), 4-AP, a non-selective blocker of voltage activated K channels (30 - 300 pmol/site) or PBS were administered by icv route. Administration of Tx3-1 (300 pmol/site) improved and rescued memory in naïve and in mice submitted to the AD model. 4-AP (30-300 pmol/site) did not improved memory retention and, at least in doses tested, caused toxic side effects. These results shown that Tx3-1 improved and rescue memory deficit in animal model of AD, suggesting the involvement of IA currents in the cognitive deficit of Alzheimer disease.

Hippocampus synaptic plasticity related to contextual cues involved in chronic diazepam administration and withdrawal

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Contextual cues linked to drug experience have been frequently associated to craving and relapse. Hippocampal synaptic plasticity has been related to learning and adaptive processes developed during chronic administration of drug abuse. Leading authors suggest a common neurobiological mechanism mediating drug addiction and memory. We demonstrated that the environmental context associated with withdrawal experience was able to evoke the similar behavioral alteration observed after chronic benzodiazepines administration. Also an increased hippocampal synaptic plasticity and expression of Arc protein during withdrawal and during the re-exposure to the context associated with anxiety expression was demonstrated. The goal of this work is to find out if memory induced by drugs of abuse and evoked by the contextual cues linked to the withdrawal experience, could be affected by changes in the contextual cues. In order to associate this memory with the hippocampus temporary participation and continue identifying the cellular mechanisms supporting the plastic change, responsible for the maintenance of this memory trace. Preliminary results indicate that changing the contextual cues during re-exposure prevented the retrieval of the memory and the plastic phenomenon observed previously.

Decision making, cognitive functioning and cardiovascular risk factors

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The aim of this study was to determine the relationship between decision-making (DM), general cognitive functioning, emotional and cardiovascular risk factors. Three groups of participants (cardiovascular disease, N=14, depression, N=17, and

healthy, N=7) were evaluated with the IOWA gambling and Game of Dice tasks, Montreal Cognitive Assessment (MOCA), Beck Depression Inventory, SF-36, Weight, height, IMC, Waist perimeter, arterial blood pressure, total cholesterol, LDL cholesterol, HDL cholesterol, triglycerides and glucose. Positive correlations were observed between HDL level and quality of life measures, while the latter correlated with fasting glycemia. We observed an inverse correlation ( $r = -0.717$   $p < 0.013$ ) between MOCA and glycemia and, surprisingly, a positive correlation with LDL levels ( $r = 0.740$   $p < 0.036$ ). The global performance between the two DM tests had a high correlation ( $r = 0.734$   $p < 0.003$ ) Among depression patients MOCA correlated with GD ( $R = 0.702$ ;  $p < 0.001$ ) whereas IOWA was inversely related to fasting glycemia ( $r = -0.758$   $p < 0.011$ ). In the DM tests, cardiac patients have a better performance than depression patients. General cognitive performance and DM seem to have a complex relationship with some CRFs parameters, and both impact on perception of quality of life.

Cognition, Behavior, and Memory

**Poster Number (223) Session 1**

### Dynamics of conscious access during eye movements

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When looking at a scene we produce spontaneously a discrete sequence of brief fixations. Bits of information from all fixations are integrated to produce a coherent percept of the scene. This experiment was aimed to understand the temporal kernel of conscious access to information in a single fixation. Participants had to make a rapid sequence of three fixations: 1) to a point at the left side of the screen, 2) to the center and 3) to the right side of the screen. A stream of letters presented on the center commenced before participant's gaze was directed to it and continued until their eyes reached the right side. They were asked to report the content of what they had seen. We could infer the temporal kernel of conscious access relative to the onset and offset of the fixation and whether sequential information could be buffered in a single fixation. This temporal window begins around 80ms from the fixation onset, revealing the existence of a refractory period locked to the beginning of the fixation. The kernel increased smoothly and reached a cutoff during the saccade. These results indicate that it is possible to acquire information from a visual scene not only during fixations but also during saccades, contrary to what was expected from the literature (i.e. saccadic suppression).

Cognition, Behavior, and Memory

**Poster Number (224) Session 2**

Nicotine-induced conditioning place preference in low and high nicotine-responder rats

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Horizontal locomotor activity in rats is a behavioral index of nicotine effect on the brain. Previous studies showed that rats with high locomotor responses to an acute injection of nicotine are more prone to self-administer the drug than low responders, demonstrating an increased motivational effect of the drug by a pre-exposure. We designed the present study to determine if individual differences in initial responsiveness to nicotine are related to the rewarding properties of the drug. We evaluated locomotor response to a single injection of nicotine in adolescent Sprague-Dawley rats. Animals were classified in high (HR) or low (LR) nicotine responders based on their locomotor responses to the drug. Then, we trained the animals in a Conditioned-place preference (CPP) paradigm, in order to evaluate nicotine rewarding properties in these two behavioral groups. As expected, our results indicate that the animals differed in their locomotor response to a single injection of nicotine, with some exhibiting a greater response than others. However, the animals classified as HR and LR developed CPP with the same strength and efficiency, suggesting that a nicotine preexposure have no effect on the posterior rewarding properties of the drug.

Cognition, Behavior, and Memory

**Poster Number (225) Session 3**

Mapping connective plasticity in 3 dimensions in the rat spinal cord after sciatic nerve axotomy

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Certain forms of peripheral nerve damage are known to result in a neuropathy characterized by chronic pain, despite functional recovery of the connections to somatic sensory and motor targets. It has been hypothesized that this phenome-



non is due in large part to rearrangements of the associated central projections at the level of the spinal cord, and at higher levels. We have used markers for pre-synaptic sensory axons and post-synaptic dendrites together with confocal microscopy in an attempt to analyze on a global scale the localization and nature of these rearrangements after a widely used form of peripheral lesion, the sciatic nerve axotomy. Our results indicate that even a limited lesion is capable of producing widespread rearrangement of the central connections that appears to affect simultaneously both the incoming and intrinsic neurites. Use of confocal microscopy permits the re-construction in three dimensions of the changes due to axotomy for a widespread evaluation of how the central nervous system may reire itself after injury, applicable to other experimental models of nervous sytem damage.

Cognition, Behavior, and Memory

**Poster Number (226) Session 1**

Effects of sequestering acetaldehyde on alcohol-mediated conditioned place preference in adolescent rats

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The transition from controlled to problematic alcohol drinking is significantly affected by the age in which alcohol consumption begins and by the perception of alcohol's motivational reinforcing effects. It is thus important to assess the neurobiology of alcohol motivational effects during adolescence. Recent studies revealed alcohol-induced conditioned place preference (CPP) in adolescent rats. Acetaldehyde, the first metabolic product of ethanol oxidation, seems to be involved in alcohol's appetitive effects. Administration of d-penicillamine (DP), a drug that sequesters acetaldehyde, prevented the acquisition of CPP by alcohol in adult and infant rats. The present study assessed the role of acetaldehyde in the motivational effects of alcohol in adolescent rats. Rats (postnatal days 28-30, PDs 28-30) were given DP (0.0 or 75 mg/kg) 20 min before pairings of 1.0 g/kg alcohol and a sandpaper surface (conditioned stimulus, CS). The rats were exposed to the CS during alcohol post-administration time 5-20 min. At test, the rats given sandpaper-ethanol pairings exhibited greater preference for the CS than unpaired controls, and this effect -- indicative of appetitive reinforcement by ethanol -- was unaffected by pre-training administration of DP. These results confirm previous data suggesting that adolescent rats are sensitive to the reinforcing effect of ethanol. In adolescents and under the present conditions, this motivational effect was not blocked by an acetaldehyde-chelating agent.

Successive negative contrast in rats. Effects of frontal cortex lesions

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When animals trained to receive a reward have a surprising decrease in the quality or magnitude of this reinforcement, instead of adjusting its performance to that of animals always receive a small reward, show a much greater decreasing in performance, phenomenon called Successive Negative Contrast (SNC). SNC has been found both in consummatory (cSNC) as in instrumental (iSNC) procedures, and the existence of different neural substrates has been postulated. It has been shown that lesions in the limbic system have different effects on the cSNC and iSNC, but the evidence is controversial. The frontal cortex (FC) is a phylogenetically novel area in vertebrates, and potentially also might be involved in the SNC (since this phenomenon is observed only in mammals). By performing different chemical lesions in FC with quinolinic acid we evaluate the role of this area in the SNC. In this work we present the results of ongoing experiments, including evidence of cSNC and iSNC, and the preliminary results of the effect of lesions of the FC on the iSNC.

Early ERPs (N170) measures of valence, interference and stimulus type discrimination: association to executive function and social cognition

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A vast literature has described how early cortical activity is modulated by changes in stimulus properties, including its emotional content. These studies mostly relied on fixed stimulus attributes (e.g. intensity of an emotion) without explicitly exploring the impact that these stimuli may have on each individual. The aim of this study was to fill this gap, investigating the interaction between stimulus type

and neuropsychological evaluation. Scalp ERPs were recorded in healthy controls while they classified faces and/or words according to its emotional valence (positive or negative). Modulation of N170 by three factors (stimulus type, valence and interference) were quantified to calculate indexes of stimulus, valence and interference discrimination. Subjects also completed three neuropsychological batteries: general neuropsychology, executive functions, and social cognition. Our results showed a strong association between complex cognitive processes like theory of mind tasks and early brain cortical measures of emotional valence discrimination. They also showed an association between N170 stimulus type discrimination and cognitive task segregation abilities. Interestingly, emotional decision making may also be related to ERP emotional discrimination.

Cognition, Behavior, and Memory

**Poster Number (229) Session 1**

Fluoxetine revert memory impairment in a depressive animal model

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Psychiatric disorders, including depression, are complex and heterogeneous clinical entities. The depressive state is characterized by a core conjunct of psychopathological phenomena, among which, the short and long-term memory impairment is included (Chestnut et al, 2008). The launch of fluoxetine (F) to the market, a selective serotonin reuptake inhibitor, was the beginning of a new era of safe and effective treatment for depression disease; nevertheless its impact upon memory processes has not been explained. In the present study we investigated its effects upon memory in a depression animal model (olfactory bulbectomy). Albino's Swiss male mice were divided in two groups: sham (C) and bulbectomized (BO). In order to determine a depressive behavior, after 15 days of surgery they were evaluated in tail suspension test and then, orally treated with saline (S) or F (10 mg/Kg) during 28 days. The last day of treatment, memory retention was evaluated using the objects recognition test. C-F and BO-S animals showed memory impairment in relation to C-S animals ( $F_{68,2}= 39.5$ ,  $p < 0.05$ ), and BO-F animals showed memory enhancement in relation to BO-S ( $F_{134,4}= 3.2$ ,  $p < 0.05$ ). In conclusion, F revert memory impairment in BO animals and, its effects varies according to the experimental model.

Modulation of extinction memory persistence after its expression

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Memory expression may lead to two opposing protein-synthesis-dependent processes; extinction and reconsolidation. The knowledge about these two processes has accumulated in recent years, but their possible interaction was not evaluated yet. To analyze whether inhibition of protein synthesis after reactivation affects the persistence of extinction memory rats were trained in inhibitory avoidance task (IA) and, beginning 24 h posttraining, submitted to one daily extinction session for 5 days. 24 h after the last extinction session, memory was reactivated and either immediately or 6 h later the animals received intra-CA1 infusions of the protein synthesis inhibitor anisomycin (ANI; 160 ug/side). Here we show that immediately after reactivation, ANI impaired further expression of extinction allowing reappearance of the extinguished IA response. This effect was observed 24 h and 168 h but not 3 h after reactivation. Our results are in agreement with models proposing that extinction results from a new learning that suppresses expression of the original memory but does not erase it and support the hypothesis that, at least for the case of fear extinction memory, inhibition of reconsolidation does not affect storage but hinders retrieval persistently.

New paradigm to measure retrieval induced forgetting in rats

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Retrieval-induced forgetting (RIF) refers to the observation that repeated retrieval of a given item causes loss of retrieval access to other related items. When retrieving specific events, the cues that guide retrieval are related to other traces that may interfere in the process of retrieving the desired item. RIF has been attributed

to inhibitory control mechanisms that are recruited to overcome interference caused by competing memory traces. In humans it has been shown, using declarative memory tasks, that retrieval of specific items will induce forgetting of category related but not unrelated items, suggesting that RIF is caused by inhibitory processes specific to the situation of recall. RIF has been studied exclusively in humans limiting the ability to understand the mechanisms involved. Then, our aim is to develop an animal protocol that parallel the human task used to study RIF. Novel object recognition tests exploit an animal's natural tendency to explore novel or relatively less familiar stimulus. It is considered an equivalent of declarative memory in humans. Based on this task, we developed a paradigm that allowed us to measure RIF in rats. This new test will allow us to study the neural circuitry and mechanisms involved in this particular type of memory modulation.

Cognition, Behavior, and Memory

**Poster Number (232) Session 1**

Contribution of the Gabaergic system to the labilization-reconsolidation process in a neutral verbal memory paradigm

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The reconsolidation hypothesis states that a consolidated memory could become susceptible to modulation for a discrete period of time, after a reminder presentation, which begins with a learned cue and triggers the labilization-reconsolidation process. This process was previously described in a declarative memory and specific reminder parametrical conditions were determined. We explored the Gabaergic system role in this process, by administering benzodiazepines, in a neutral verbal memory paradigm. Experimental design consists in a 3-day experiment: Day 1: learning of five cue and response syllables. Day 2: reminder presentation and clonazepam or placebo administration. Day 3: verbal memory evaluation. We determined a dose-response curve by administering different clonazepam doses and found an effective dose of 0.25mg. Results indicate that when this dose was administered, in reconsolidation phase, declarative memory was facilitated. When the reminder conditions were changed, placebo was used or memory was tested 4 hs after the reminder, the facilitating effect was not observed. This unexpected effect could be explained by the paradigm and/or the low dose used. Future experiments will include an aversive paradigm looking for the amnesic effect typically described for this drug.

Understanding learning disability by enhanced Ras/MAPK signaling

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Learning disability is a common cognitive alteration in a group of disorders in which a higher activity of MAPK is a hallmark. This group of genetic disorders, including Noonan syndrome (NS), are caused by mutations in genes encoding for components of the Ras/MAPK signaling pathway. Recently, we identified that different *csn* gain-of-function alleles, including the *csnN308D* which models the commonest NS mutation, impair a fundamental property of learning called the spacing effect. The spacing effect makes reference to a longer-lasting memory induced by spaced experiences over time (spaced training) compared with experiences in close temporal proximity (massed training). The defect in spacing effect precludes the induction of long-term memory as a result of a deregulation of a CSW-dependent MAPK signaling during training. Here we present some preliminary studies on the effect of mutations on components of the Ras/MAPK signaling pathway. We identify loss-of-function alleles of a few genes that can reduce long-term memory, but did not affect learning after a single training session. Moreover, such genes could be involved in the spacing effect because their memory phenotype it was sensitive to variations of the inter-trial intervals, whereas the control genotype did not.

Asociation between abstract category learning and executive function

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Stimulus equivalence paradigm has been widely applied to the study of abstract category learning. An equivalence class is formed when, having learned baseline relations (AB, BC), the subject is able to respond to derived relations: reflexivity (A-A), symmetry (B-A) and transitivity (A-C). The main goal of the current work was to analyze if executive functions are implicated in equivalence class learning. Subjects were trained to learn baseline relations, and tested for derived relations of

symmetry, transitivity and symmetry-transitivity combined. Additionally, subject performance in three components of executive functions was assessed: working memory (N-back task), inhibitory control (Stroop task) and cognitive flexibility (Dot mixed task). Subjects who completed training successfully obtained better scores in all executive function tasks, while subjects who passed all test of derived relations obtained higher working memory scores. Additionally, significant correlations were observed between performance and response times in derived relation tests and working memory scores. Results suggest that equivalence class learning is associated with working memory because it requires evoking and manipulating previously learned information.

Cognition, Behavior, and Memory  
**Poster Number (235) Session 1**

### Eye movements reflect syntactic organization of arithmetic thinking

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Mathematics shares with language an essential reliance on the human capacity for recursion, permitting the generation of an infinite range of embedded expressions from a finite set of elements. Here, we study the role of syntax in arithmetic thinking, a much neglected component of numerical cognition, by examining whether adult humans can, at a glance extract the syntactic organization of nested arithmetic formulas. We measured eye movement sequences during the calculation of left-branching and right-branching arithmetic problems such as  $\{(3-1)+2\}+4$  versus  $4+\{2+(3-1)\}$ . Fixation sequences were organized in clusters corresponding to syntactic levels. These sequences were stereotyped across all subjects and reflected a linear exploration of the hierarchical tree. In the second part of the study we investigate, by a factorial response time analysis, whether an arithmetic problem can be parsed in a sequence of unitary operations determined by the syntactic tree. For instance  $\{(3-1)+2\}+4$  in 3-1, 2+2 and 4+4). Our findings provide evidence for a syntactic organization of arithmetic thinking which is reminiscent of the organization observed in language and paves the way for further comparative analysis of the role of recursion in language and mathematics.

NF-kappa B like DNA binding activity during long-term memory consolidation in the honey bee

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Long-term memory consolidation requires de novo protein synthesis. Activation of transcription factors from the Rel/NF-kB family is crucial for memory consolidation in rodents and crabs. The recent sequencing of the honey bee *Apis mellifera* genome allowed identification of plausible orthologues for almost all NF-kB family members. As in mammals, activation of NF-kB plays also role during immune response in the honey bee. In the present study we investigate the role of this factor during consolidation of olfactory memory in honey bees. First, we calibrated conditions to measure activity (DNA binding) of NF-kB from brain extracts using and electro mobility shift assay (EMSA). Second, we observed an increase in DNA binding after a training protocol that induces long-term memory formation. Third, we are performing pharmacological experiments using an inhibitor of NF-kB activation (sulfasalazine) to test the functional role of NF-kB during memory formation. The access to the honey bee genome and the nucleotide sequence admit future experiments using RNA interference to specifically down regulate and test the contribution of different NF-KB family members. These results would represent the first report about participation of NF-kB in memory formation in insects.

Rats either in their sleep- or awake-phase are rescued from amnesia of inhibitory avoidance caused by scopolamine, by previous exposures to the open field

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We reported that two open field (OF) sessions previous to training in inhibitory



avoidance (IA), facilitated learning of this task in the rat. When inverted light/dark cycle-rats (IC) were bilaterally injected with scopolamine intrahippocampus immediately after IA training in their awake phase, they were amnesic as expected; however, rats previously exposed twice to the OF were not. It was reported a promoting effect of previous exposure to a single OF session, when rats in their sleep phase (straight cycle, SC) were trained with an underthreshold shock (Moncada & Viola, 2007). To investigate a putative influence of light cycle, we also evaluated SC-animals during their sleep phase. Two OF exposures had a similar effect on IA performance compared to IC-animals trained in their awake phase. IA training latencies for SC-animals were higher than for IC-rats. SC-rats appeared to be rescued from the amnesic effect of scopolamine on IA performance by previous exposure to 1 OF 24 h before, or were clearly rescued by two OF sessions. Although the influence of novelty and/or manipulation of the rats could be involved in these facilitatory effects, it appears that attentional and awareness states could be also relevant; further investigations are necessary to clarify this issue.

Cognition, Behavior, and Memory  
**Poster Number (238) Session 1**

Loud noise exposure induces associative memory and anxiety levels impairments

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Living organisms are exposed to noise levels that might produce extra-auditory effects, including behavioral alterations. The aim of the present work was to test if loud noise exposure can affect associative memory and anxiety. Male Wistar rats of 15 days were exposed to white noise (95-97dB, 2h/day) and separated into 2 groups, acute (AE, 2h/day) and chronic exposure (CE, 2h/day for 15 d). Passive avoidance test (PA) was used to evaluate the associative memory whereas open field (OF) and elevated plus maze (EPM) was used as anxiety-related tests in 30-days-old rats. PA and OF were performed at short (1 h, ST) and long-term (24 h, LT). Results showed an impairment in PA test in exposed CE animals, with a decreased latency to enter the dark compartment, both at ST and LT. On the other hand, OF and EPM results show a decrease in anxiety level in CE exposed rats. Latency to enter the centre when the animals were first place in the OF was decreased in CE, both at ST and LT. The number of entries (E) as well as the total time (%T) spent on open arms in EPM were increased. These results suggest that CE to loud noise can induce associative memory impairments and anxiety-related changes, supporting the high vulnerability of developing rodents to stressors such as noise exposure.

Cognition, Behavior, and Memory

**Poster Number (239) Session 2**

Automated and abbreviated neuropsychological tests of free distribution: attention, memory, gnosis, praxis, aphasia, and executive function

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This site attempts to be a common workspace for professionals interested in the diagnosis, rehabilitation and neuropsychological research. The selected tests have satisfactory psychometric properties and have been validated by both the brain injury site and the ability to discriminate healthy subjects from patients as well as different groups of patients with mild cognitive impairment or dementia. The tests are freely distributed for research and clinical application. Bearing in mind the population of aphasic patients, pre-school children and illiterates, the tests were designed with minor verbal components. Adapted visual tests of memory (figures and faces) and cancellation are on line assessed. All the tests designed in our lab are on line and off line assessed. Among the latter ones there are two dichotic listening tests (fused words and digits), a test of aphasia or minimum verbal performance (which includes most of the items of the mini mental test), and several card sorting tests designed to assess: praxis in the use of the mouse (either acquisition or execution), visual discrimination (correspondence), and executive function (the nine-card sorting test which assesses spontaneous and induced performance to get single or combined arrays of three visual sorting principles).

Cognition, Behavior, and Memory

**Poster Number (240) Session 3**

Aphasia: what are we measuring?

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In the psychological scope, the diagnosis of patients based on the conceptual definition of the disorder (not the empirical one) is common. Consequently, real cases are forced to fit into certain ideal categories. In the case of aphasia it is assumed that these typologies (syndromes) are constructed from the gathering of basic components or symptoms (usually six). At the same time, there are "syndromes"

which consist of only one symptom such as anomic aphasia which mainly presents with failures in naming. By assuming that the aphasic symptoms were six and that they could be grouped in all possible ways, ranging from zero to six symptoms, real people should be allowed to fall into 64 possible syndromes or combinations of symptoms. Nevertheless, only eight "typical" syndromes are generally recognized. Do the other combinations not deserve to be computed? Additionally, the syndromes are suspected to be based on the injury or dysfunction of certain brain areas also considered as "typical". These areas are not simultaneously described (empirically correlated to) the syndrome because the syndrome is assumed to be certain a priori. Both typical syndromes and typical lesions have been originated in the study of illustrative "cases" that historically generated the "rule".

Cognition, Behavior, and Memory

**Poster Number (241) Session 1**

Aphasia: when just one neuropsychological condition generates more syndromes than symptoms

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In the classical (clinical-neuroanatomical) approach to aphasia various language functions are simultaneously evaluated in order to detect typical aphasic syndromes or pure language impairments. Four basic language functions (repetition, comprehension, expression, and naming) and two complementary ones (reading and writing) are usually studied as the elementary aphasic components (symptoms) which will make up the syndrome. However, at least eight aphasic syndromes (Broca, Wernicke, conduction, anomic, transcortical sensorial, transcortical motor, transcortical mixed, subcortical, etc.) and at least three pure language impairments (alexia, agraphia, verbal deafness, etc.) are usually deduced from the elementary components. While the studied functions are at most six, the diagnoses are at least eleven. Additionally, the diagnoses are not exhaustive and they keep increasing. Would not it be more useful to describe the presence and magnitude of symptoms than try to find a perfect archetype for each combination of symptoms? The practice of easily assigning a proper name to some interesting combinations of clinical features can be seen as an abuse of the authority criterion if just incipient or case studies are shown as validation. Worldwide accepted" is not synonymous of "valid."

Role of the posterior parietal cortex in online movement corrections

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Unpredictable modifications in the environment, like the sudden displacement of an object to be grasped, require adjusting well practiced movements by updating the initial motor command online, as the movement progresses. Single pulses of transcranial magnetic stimulation (TMS) applied over the posterior parietal cortex (PPC) disrupts the adjustment of reaching movements aimed at targets displaced during the movement but not to stationary targets (Desmurget et al., 1999). Here, we examined the possibility that PPC may also be involved in online corrections to proprioceptive perturbations applied during a reaching movement. For this purpose subjects made reaching movements to a target located 12cm away of the starting point. Once every 5 trials, a lateral perturbation force was applied against the arm. TMS was applied at 0, 100 and 200 ms relative to the perturbation onset and its effect on different kinematics measurement was computed. Preliminary results indicate that TMS applied at 0 ms had a marginally significant effect on the time point of maximal tangential velocity during the second half of the movement ( $p=0.088$ , ANOVA RM), which is consistent with a modulation of predictive online corrections to the proprioceptive perturbation. Desmurget et al., 1999. *Nat Neurosci* 2:563-567

Cognition, Behavior, and Memory  
**Poster Number (243) Session 3**

Brain activity during social cognition tasks in individuals with schizophrenia, their unaffected siblings, and healthy controls

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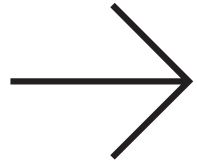
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Several studies have shown that patients with schizophrenia have impaired performance in various aspects of social cognition, such as emotion processing (EP) and theory of mind (TOM). The present study examined brain activation patterns during social cognition tasks in patients with schizophrenia and their nonpsychotic siblings, trying to determine whether these alterations reflect a heritable trait.

Twelve patients with schizophrenia (age  $31.3 \pm 6.5$ ), twelve non-psychotic relatives (age  $31.8 \pm 3.5$ ), and twelve matched comparison subjects (age  $30.1 \pm 9.2$ ) underwent BOLD functional magnetic resonance imaging during visual presentation of different social cognition paradigms. Random effects analysis was done for each task within groups, and later a group analysis was done. Beyond the activation of some independent brain areas generally associated with emotional response, social cognition performances brought about activations in language areas. The intensity of the activations was minimum in the emotional processing task, and maximum in the detection of complex mental states in eyes. Patients' activations were predominantly left and unilateral. Healthy controls also activated symmetric brain structures on the right side. Unaffected siblings showed bilateral activation in the same brain structures but asymmetrically distributed. These results support the idea that schizophrenia is an illness characterized by abnormalities in the process of brain lateralization.



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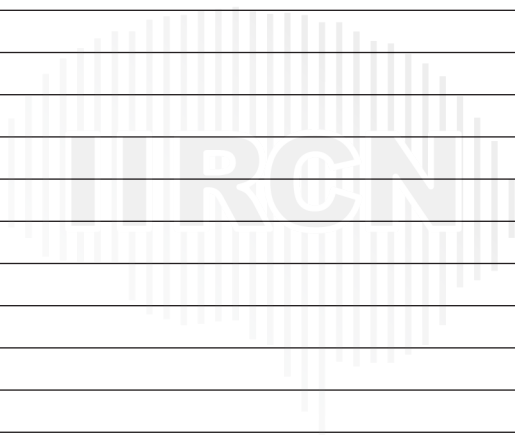
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The logo for the International Institute for Rural Reconstruction (IIRC) is centered on the page. It features the acronym "IIRC" in a bold, sans-serif font. The letters are white and are set against a dark, circular background that has a textured, slightly grainy appearance. The overall design is simple and professional.

///////////////// NOTES





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