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2nd FALAN Congress

XII Reunión Anual de la Sociedad Chilena de Neurociencias
XV Jornadas de la Sociedad de Neurociencias del Uruguay
XXXI Congreso Anual de la Sociedad Argentina de Investigación en Neurociencias
XXXIX Reunião Anual da la Sociedade Brasileira de Neurociências e Comportamento

October 17-20, 2016
Buenos Aires
Argentina

Puente de la mujer, Puerto Madero (Arq. Santiago Calatrava)

SAN
SOCIEDAD ARGENTINA DE INVESTIGACIÓN EN NEUROCIENCIAS

FALAN
Federation of Latin American and Caribbean Neuroscience Societies

PROGRAM

#FALAN2016

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Welcome to FALAN 2016!

Dear colleagues:

Welcome to the 2nd Congress of the Federation of Latin-American and Caribbean Societies for Neuroscience (FALAN). Welcome to Buenos Aires.
We are very happy to have you at the 2nd FALAN Congress together with hundreds of neuroscientists from Latin America and from all over the world. You are now part of the history of neuroscience development in the region.

The first Latin-American meeting held in Buzios in 2008 was the initial step towards the creation of FALAN. Once established, the first formal FALAN meeting was held in Cancun-México in 2012. Today, under the auspices of FALAN Buenos Aires Meeting several events will be held: the¨XXXIX Reunião Anual da la Sociedade Brasileira de Neurociências e Comportamento¨, the ¨XII Reunión Anual de la Sociedad Chilena de Neurociencias¨, the ¨XV Jornadas de la Sociedad de Neurociencias del Uruguay¨ and the ¨XXXI Congreso Anual de la Sociedad Argentina de Investigación en Neurociencias. The rest of FALAN Members Societies are also supporting and promoting this Congress as one of the major activities of year 2016. IBRO and the Latin-American Regional Committee (LARC) are providing strong economic and logistic assistance. We are very grateful for this support.

The 2nd FALAN Congress reflects the vigorous and fast growing pace of neuroscience in the region, and the institutional strengthening of FALAN. Given the scientific and institutional significance of the 2nd FALAN Congress we warmly welcome you.
The aim of the Congress is to promote neuroscience in the region and, at the same time, to provide the Latin-American neuroscience community an environment in which the research and sharing of ideas and techniques will pave the way for a stronger interaction between Latin-American scientists.

The meeting has been organized by FALAN together with the Sociedad Argentina de Investigación en Neurociencias (SAN) and a scientific committee on which most FALAN societies were represented. These international committees have created a high quality program where all the important areas and relevant topics in neuroscience are present. Lectures and symposia of high academic standards on specific subjects were included. Furthermore, as a very important part of the meeting, more than 800 posters will be presented.

We would like to acknowledge the effort of the participants, speakers and members of the different committees for their time and effort and thank them once more for finding their own support for the registration fees and the travel expenses to attend this venue.
Welcome all and enjoy the meeting.

Osvaldo D. Uchitel
President of FALAN

Arturo Romano
Chair of the Organizing Committee

FALAN Executive Committee

(January 2016 to January 2018)

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Participating Societies

BRASIL
Sociedade Brasileira de Neurociências e Comportamento
XXXIX Reunião Anual da la Sociedade Brasileira de Neurociências e Comportamento

CHILE
Sociedad Chilena de Neurociencia
XII Reunión Anual de la Sociedad Chilena de Neurociencias

URUGUAY
Sociedad de Neurociencia del Uruguay
XV Jornadas de la Sociedad de Neurociencias del Uruguay

ARGENTINA
Sociedad Argentina de investigación en Neurociencias
XXXI Congreso Anual de la Sociedad Argentina de Investigación en Neurociencias

Invited Societies

COLOMBIA
Colegio Colombiano de Neurociencias (COLNE)

CUBA
Sociedad de Neurociencias de Cuba (SONECUB)

MEXICO
Sociedad Mexicana de Ciencias Fisiológicas (SMCF)

MEXICO
Sociedad Mexicana de Neurociencias y Neurobiología

COSTA RICA
Programa de Investigación en Neurociencias de Costa Rica

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SPON

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ISN
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BIO analitica

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PROGRAM AT A GLANCE

Day 1 - October 17, 2016

SCHEDULE	ROOM A	ROOM B	ROOM C
8:30 - 11:00	REGISTRATION		
11:00 - 13:00	**Symposium 1** "Novel insights into hypothalamic mechanisms controlling body homeostasis" - Chair: M. Perello	**Symposium 2** "Sleep to remember: sleep, memory and consciousness" Chairs: C. Forcato and F. Beijamini	**Symposium 3** "Macromolecular signalling complexes in neurons" Chairs: M. Shapiro and F. Barrantes
13:00 - 13:30	BREAK		
13:30 - 15:00	**Special Event 1** Meet the professor: "Neurosciences at lunch with a good friend: Prof J. Nichols" Chairs: E. Del-Bel and F. De Miguel		**Special Event 2** "Neuroscience and Education: Primer time to fill the bridge" Chair: M. Sigman
15:00 - 17:00	**Symposium 4** "Professor John G. Nicholls celebration symposium. Function, repair and training of the nervous system" Chairs: E. Del-Bel and F. De Miguel	**Symposium 5** "New insights into synaptic plasticity" Chair: A. Rodriguez-Moreno	**Symposium 6** "Patient derived induced pluripotent stem like cells as models for neudegenerative diseases" Chair: L.J. Falomir Lockhart
17:00 - 18:00	**OPENING CEREMONY** ROOM F		
18:00 - 19:00	**PLENARY LECTURE 1: Larry Swanson** (USA): "Architecture of the cortical association network supporting voluntary behavior and cognition" ROOM F		

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Day 2 - October 18, 2016

	ROOM A	ROOM B	ROOM C	ROOM D
8:30 - 9:30	**Special Lecture 1** Rodrigo Andrade "Using optogenetics to interrogate serotonergic synaptic transmission in the mammalian brain"	**Special Lecture 2** Jorge Bergado "Time and timing in neurophysiology. Lessons from synaptic tagging"	**Special Lecture 3** Juan Carlos Brenes "Effects of environmental enrichment on brain plasticity, cognition and social communication in rats"	
9:30 - 11:00	POSTER PRESENTATION 1 - ROOM F			
11:00 - 13:00	**Symposium 7** IBRO Alumni Symposium: "Basic and translation research in neurodegenerative disease: fom molecules to animal models" Chair: V. Della Maggiore	**Symposium 8** "The consequences of memory retrieval: reconsolidation, extinction or nothing at all" Chair: P. Bekinschtein	**Symposium 9** "Regulation and function of gap junctions and hemichannels in the nervous system" Chairs: J.C. Saez - A. Pereda	**Symposium 10** "Neuromathematics" Chair: A.C. Roque
13:00 - 13:30	B R E A K			
13:30 - 15:00	**Special Event 3** "How can neuroscience research impact the global burden of disease" Chair: P. Valdes-Sosa		**Special Event 4** Workshop: "How to get published" Chair: J. Lerma	**Leica Microsystems** "Avances tecnología confocal y súper resolución"
15:00 - 17:00	**Symposium 11** "Auditory processing from the cochlea to the cortex and back" Chairs: E. Katz and M.E. Gomez Casati	**Symposium 12** "New concepts in oligodendrocyte function in neurological diseases" Chairs: B. Fuss and C. Hedin-Pereira	**Symposium 13** "ISN Symposium on Neural Control of Appetite - From genes to circuits and behaviour" Chairs: I.E. de Araujo and V.F. Bumaschny	**Symposium 14** "Behavioral, neurochemical and molecular approaches to study fear anxiety and posttraumatic stress disorder" Chair: A.M. Gallegos
1700 - 18:30	POSTER PRESENTATION 1 - ROOM F			
18:30 - 19:30	**Special Lecture 4:** Newton Canteras "The many paths to fear"	**Special Lecture 5:** Zulma Dueñas Gómez "Exploring neuroendocrine mechanisms of sexual dimorphism in early stress response: a translational approach"	**Special Lecture 6:** Cecilia Hidalgo "Calcium signaling, cellular oxidative tone and synaptic plasticity"	
19:30	**SAN Society Meeting**	**SBNeC Society Meeting**	**SCN Society Meeting**	**SNU Society Meeting**

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Day 3 - October 19, 2016

SCHEDULE	ROOM A	ROOM B	ROOM C	ROOM D
8:30 - 9:30	**Special Lecture 7** Diogo O. Gomes de Souza "Neuroprotective effect of guanosine in experimental models of brain diseases"	**Special Lecture 8** Conference Distintion SCN: Nibaldo Inestrosa "Wnt signaling and Alzheimer's Disease"		
9:30 - 11:00	POSTER PRESENTATION 2 - ROOM F			
11:00 - 13:00	**Symposium 15** "Neurophysiology of temporal processing in the brain" Chairs: P. Agostino and H. Merchant	**Symposium 16** "Integrative sensory motor function: from motor commands to cognition" Chair: P. Maldonado	**Symposium 17** "Cellular and molecular mechanisms of neuronal plasticity" Chairs: F. Rossi - N. Vitureira	
13:00 - 14:00	BREAK			
14:00 - 15:00	**FENS PLENARY LECTURE 2:** Pierre Magistretti: "Neuron- glia metabolic coupling : roles in plasticity and neuroprotection" ROOM A+B+C			
15:00 - 16:30	POSTER PRESENTATION 2 - ROOM F			
16:30 - 18:30	**Symposium 18** Young Investigators Symposia I Chair: Jaime Fornaguera	**Symposium 19** "Neuroimmunoendocrinology of the circadian system" Chair: D. Golombek	**Symposium 20** "Dopamine neurons: connectivity, functional connectivity and susceptibility" Chair: J. P. Bolam	**Symposium 21** "Neuroesteroids, cardioesteroids and oxidative cell signalling as target in neuroinflammation and possible role in neurodegenerative disease" Chairs: F. Benetti
18:30 - 19:30	**Special Lecture 9** R. Caputto Conference: Jorge Medina "Modulation of the duration of aversive and appetitive memories"		**Special Lecture 10** Clemente Estable Conference: José Roberto Sotelo "Schwann cell to axon RNA transfer"	

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Day 4 - October 20, 2016

SCHEDULE	ROOM A	ROOM B	ROOM C
8:30 - 9:30	**Special Lecture 11** E. de Robertis Conference: Alejandro Schinder "Activity and Neurogenesis-mediated Circuit Remodeling in the Hippocampus"	**Special Lecture 12** Elio Garcia Austt Conference: Pablo Torterolo "Melanin concentrating hormone in mesopontine raphe nuclei: role in REM sleep and depression"	**Special Lecture 13** Mitchell Valdes-Sosa "Neural mechanisms for the configuration of selective attention"
9:30 - 11:00	POSTER PRESENTATION 3 - ROOM F		
11:00 - 13:00	**Symposium 22** "The interplay of neuronal activity, synaptogenesis and plasticity" Chair: D. Refojo		**Symposium 23** "The glial cell-neuron regulatory cross talk" Chair: R. von Bernhardi
13:00 - 13:30	BREAK		
13:30 - 15:00	**Special Event 5** "Latin American Brain Mapping Network" LABMAN - Chair: V. della Maggiore	**FALAN Meeting**	**Special Event 6** Workshop: "Submitting your work to an international journal: the peer review system and what we expect in a good paper" - Chair: P. Bolam
15:00 - 17:00	**Symposium 24** Young Investigators Symposia II Chair: Antonia Marin Burgin	**Symposium 25** "Parkinsons disease: from neuronal death to therapeutics" Chairs: J. Ferrario and G. Murer	**Symposium 26** "Neuroframes symposium - Freud revisited: computational psychiatry" Chair: J. Sitt
17:00 - 18:30	POSTER PRESENTATION 3 - ROOM F		
18:30 - 19:30	**PLENARY LECTURE 3:** **Carlos Belmonte** (Spain): "TRP channels, an early alert system for environmental challenges" ROOM A+B+C		
19:30 ›››	**CLOSING CEREMONY** - ROOM A+B+C		
	PARTY - ROOM F		

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SATELLITE EVENTS

OCTOBER 15TH

MOTORIZED STEREOTAXIC NEUROSURGERY FOR CHRONIC ELECTROPHYSIOLOGICAL RECORDINGS IN RODENTS

Location: La Cascada & Anexo, Fray Justo Santamería de Oro 2529, Palermo, Buenos Aires
Time: 8:00-13:00

Lecturers:
LILIANA FRANCIS TURNER, PHD, *FACULTAD DE CIENCIAS, UNIVERSIDAD DEL TOLIMA, COLOMBIA*
EDGARD MORYA, PHD, *EDMOND AND LILY SAFRA INTERNATIONAL NEUROSCIENCE INSTITUTE, SANTOS DUMONT INSTITUTE, BRAZIL*
Motorized stereotaxic neurosurgery and electrophysiological recordings combined with behavior research is fundamental to understand basic mechanisms and develop news approaches in neuroscience and neuroengineering. This workshop will explore those advanced scientific tools mainly for young students interested in how to use in future projects.
We encorauge young students facing dificulties in how to use use such tools to investigate the nervous system electrophysiology to book early, as places are limited due to the hands on. To apply you need a valid Falan Congress registration and the the participants will be selected accordingly with CV and letter of interest.

Realization:
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Edmon and Lily Safra - International Institute of Neuroscience
Scientific and Technical Support:
NeuroStar
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SBNEC SATELLITE SYMPOSIUM – BRAIN DISEASES: NEUROENERGETICS AND NEUROPROTECTION

Location: Facultad de Ciencias Exactas y Naturales de la UBA, Ciudad Universitaria, Pabellón 2 (Room 8)
Time: 8:00-13:00
CHAIR: DIOGO ONOFRE GOMES DE SOUZA, *UNIVERSIDADE FEDERAL DO RIO GRANDE DO SUL, DEPARTMENT: BIOCHEMISTRY, BRAZIL*
Recent advances in neuroenergetics have highlighted the importance of neuronastrocyte metabolic interactions. Astrocytes contribute to a variety of neuronal functions, including synapse formation and plasticity, energetic support and redox status. Disturbances of these neuron-astrocyte interactions are likely to play an important role in brain diseases. This Symposium will discuss a wide range of approaches in this theme.

THE ROLE OF ASTROCYTE ALTERATIONS IN EARLY CHANGES IN THE DYNAMICS OF CULTURED CEREBELLAR NETWORKS

Location: Facultad de Ciencias Exactas y Naturales de la UBA, Ciudad Universitaria, Pabellón 2 (Room 9)
Time: 9:00 12:30
CHAIR: ARI BARZILAI, *DEPARTMENT OF NEUROBIOLOGY, GEORGE S. WISE, FACULTY OF LIFE SCIENCES; SAGOL SCHOOL OF NEUROSCIENCE, TEL-AVIV UNIVERSITY, ISRAEL*
An aberrant response to DNA lesions is implicated in many human brain degenerative disorders. Varioustypes of DNA lesions activate a cellular process known as the DNA damage response (DDR). Mutations affecting the proteins involved in the DDR can lead to severe genomic instability syndromes that involve varying degrees of sensitivity to genotoxic stress, and also to tissue degeneration, cancer predisposition, and premature aging. Malfunctioning DDR was found in various brain degenerative disorders such as Alzheimer's, Parkinson's and Huntington. One of the key components of the DDR is the protein ATM, which is inactivated in the genomic instability disorder ataxia-telangiectasia (A-T). In order to study the effect of malfunctioning DDR on neuronal circuits, we used calcium imaging and immunocystochemical staining to compare the morphology and the dynamics of primary cerebellar cultures grown from postnatal Atm-deficient and wild-type (WT) mice. Cerebellar networks exhibited spontaneous network events after two weeks in-vitro. Compared to WT circuits, Atm-deficient circuits displayed a lower number of global synchronizations and a larger number of sparse synchronizations, i.e. synchronous events involving less than a dozen cells. In WT networks we observed significantly high global burst similarity compared to the Atm-/-network. In addition, nodes with a high functional connectivity degree could be observed in the WT
networks but not in the Atm-/- networks. To understand A-T on the cellular level we tested the hypothesis that A-T is at least partially a glial disease. Immunocystochemical staining of astrocytes revealed a significantly less complex cell arborization in Atm-deficient versus WT circuits, as measured by the number of branches originating from cell bodies as well as their length. To further study the interrelations between neurons and astrocytes, we generated chimeric networks in which the neurons and astrocytes were extracted from different animals. We found that functional and viable chimera cultures could be prepared only from P8 cerebellar neurons and astrocytes. Chimera cultures made from combinations of P8 cerebellar neurons and P2 cortical glia or from P8 cerebellar neurons and P2 cerebellar glia did not survive and the neurons died within 3 to 4 days of plating. Our results clearly show that Atm-/- astroglial cell replacement with WT astrocytes fully restores the dynamics of neural networks in chimera neuron-glia networks extracted from Atm-deficient mice. In contrast, Atm-/- astrocytes failed to support the survival and the functionality of the WT neurons. These results support the notion that neuronal network failures in genetic brain degenerative diseases are correlated with impairment of astroglial cell functionality.

OCTOBER 16TH

CURSO SAN: "THE DOORS OF MEMORY: THE ROLE OF SLEEP ON MEMORY FORMATION AND MODIFICATION"

Location: Facultad de Ciencias Exactas y Naturales de la UBA, Ciudad Universitaria, Pabellón 2, Aula 12.

ORGANIZES: DR. CECILIA FORCATO (ARGENTINA) - DR. FELIPE BEIJAMINI (BRAZIL)

Purpose and nature of the course

This is the first Latin American Meeting of Sleep and Memory dealing with one of the most frontier topics in Neuroscience: the role of sleep in memory formation and modification. It will be held in the National University of Quilmes (UNQ), Buenos Aires on 16th October 2016 as a Satellite Event of the FALAN 2016 (Federation of Latin America and Caribbean Neuroscience, http://falan-ibrolarc.org/drupal/es). It counts with the support of the International Brain Research Organization (IBRO), Brazilian Sleep Society, the Brazilian Society of Neuroscience and Behaviour, the National University of Quilmes (UNQ), and the Argentinian Society of Neuroscience.

WORKSHOP
BCI, MOTOR IMAGERY, GAMES, VIRTUAL REALITY, EYE TRAKING, VIDEOSYNC AND EEG ANALYSIS -BEHAVIOR NEUROSCIENCE-

Location: La Cascada & Anexo, Fray Justo Santamería de Oro 2529, Palermo, Buenos Aires

Time: 8:30 13:00

Lecturers:

DANIEL GOMES DA SILVA MACHADO[1], PAULO RODRIGO BAZÁN[1], MARIA ADELIA ALBANO DE ARATANHA[1]

[1]*BRAIN SUPPORT - BRAZIL*

Pascal Mangold from Mangold International - Germany

Pierluigi Castellone from Brain Products - Germany

The use of brain-computer interface (BCI) technology has been currently proven to provide new insights in studying important brain processes such as learning, brain plasticity and neurorehabilitation. The association with virtual reality, makes it possible to extrapolate lab environment providing new possibilities for neuroscientific research. This workshop will explore the use of open source softwares to acquire cortical activity with an EEG and use it to control an avatar in a virtual environment through a well known neurophysiological pattern called motor imagery. Furthermore, we will discuss how to merge and interpret data coming from different sources/devices, such as eye tracking, EEG and video cameras. In addition we will bring the state of art software (BrainVision Analyser 2.0) in ERP analysis and will demonstrate how to perform an optimal ERP study.

Realization:

Brain Support Corporation

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PROGRAM IN DETAIL

PLENARY LECTURES
SPECIAL LECTURES

Plenary Lectures

OCTOBER 17TH

PL1. ARCHITECTURE OF THE CORTICAL ASSOCIATION NETWORK SUPPORTING VOLUNTARY BEHAVIOR AND COGNITION

LARRY SWANSON, *UNIVERSITY OF SOUTHERN CALIFORNIA, USA*

October 17, ROOM F, 18:00-19:00
Chair: **ALEJANDRO SCHINDER**
FUNDACION INSTITUTO LELOIR, ARGENTINA

The nervous system controls and integrates two basic functions: behavioral interactions with the environment and coordination of internal bodily functions. The basic design features of this system—and thus its functional organization--remain unclear, in stark contrast to the other systems forming the animal body (cardiovascular, respiratory, digestive, and so on). Today's lecture will present a strategy for revealing organizing principles of the mammalian nervous system. It is based on systematic, data-driven network analysis tools that have now been applied to the rostral end of the rodent central nervous system, the cerebral hemispheres, which mediate cognition and the voluntary control of behavior. This initial analysis is based on weighted and directed axonal connections between all 73 parts of the cerebral cortex and all 45 parts of the cerebral nuclei (basal ganglia); in other words, it is based on complete cerebral cortical association and cerebral nuclei connectomes. Network analysis reveals that all cortical gray matter regions are arranged in four modules with small world connectivity, whereas in contrast all cerebral nuclei regions are also arranged in four modules, but with little indication of small world organization. The functional implications of these and other results will be discussed along with future research directions progressing down the neuraxis toward the spinal cord and peripheral nervous system.

OCTOBER 19TH

PL2. NEURON- GLIA METABOLIC COUPLING: ROLES IN PLASTICITY AND NEUROPROTECTION

PIERRE MAGISTRETTI, *EPFL, LAUSANNE, SWITZERLAND*

October 19, ROOM A+B+C, 14:00-15:00
Chair: **ARTURO ROMANO**
IFIBYNE-CONICET, FCEN, UBA, ARGENTINA

A tight metabolic coupling between astrocytes and neurons is a key feature of brain energy metabolism (Magistretti and Allaman, 2015). Over the years we have described two basic mechanisms of neurometabolic coupling. First the glycogenolytic effect of VIP - restricted to cortical columns - and of noradrenaline - spanning across functionally distinct cortical areas - indicating a regulation of brain homeostasis by neurotransmitters acting on astrocytes, as glycogen is exclusively localized in these cells. Second, the glutamate-stimulated aerobic glycolysis in astrocytes. This metabolic response is mediated by the sodium-coupled reuptake of glutamate by astrocytes and the ensuing activation of the Na-K-ATPase. glycogenolysis and aerobic glycolysis result in the release of lactate from astrocytes as an energy substrate for neurons (Magistretti and Allaman, 2015).
We have recently revealed a second function of lactate, as a signaling molecule for plasticity, long-term memory consolidation and for maintenance of LTP in the hippocampus (Suzuki et al, 2011). In the basolateral amygdala as well, lactate is necessary for the formation of an appetitive memory such as conditioned place preference for cocaine (Boury-Jamot etal, 2015).
At the molecular level lactate stimulates the expression of synaptic plasticity-related genes such as Arc, Zif268 and BDNF through a mechanism involving NMDA receptor activity and its downstream signaling cascade Erk1/2 (Yang et al, 2014).

OCTOBER 20TH

PL3. TRP CHANNELS, AN EARLY ALERT SYSTEM FOR ENVIRONMENTAL CHALLENGES

CARLOS BELMONTE, *INSTITUTO DE NEUROCIENCIAS, UNIVERSIDAD MIGUEL HERNANDEZ-CSIC, SAN JUAN DE ALICANTE, SPAIN*

October 20, Room A+B+C, 18:30-19:30
Chair: **BELÉN ELGOYHEN**, *INGEBI-CONICET, ARGENTINA*

The ability to sense potentially dangerous physical and chemical changes of the surrounding environment (temperature, mechanical pressures, low humidity, harmful chemicals) represents a fundamental attribute required by living organism, including humans, to ensure survival. Evolutionary pressures determined the development in animal species of specific sensory systems capable of transducing relevant physical and chemical properties of external stimuli into electrical signals which are processed to ultimately initiate or adjust specific behaviors. Sensory transduction is mediated by transducing proteins expressed by different functional types of sensory receptor cells. TRP channels constitute a large superfamily of cation channel forming proteins with a variety of functional properties and diverse cellular and physiological roles. The first TRP channel discovered in mammalian sensory neurons was Transient Receptor Potential Vanilloid 1 (TRPV1). Flourishing research over the past decades revealed that other members of the TRP ion channel family and in particular TRPM8 and TRPA1 act as detectors for heat, cold and humidity environmental

stimuli, mechanical force, chemicals including exogenous plant and environmental compounds, bacterial toxins as well as endogenous inflammatory molecules. Thus, these channels form a multimodal transducer system for early detection of environmental sensory stimuli, which may potentially represent a threat for survival.

Special Lectures

OCTOBER 18TH

SL01. USING OPTOGENETICS TO INTERROGATE SEROTONERGIC SYNAPTIC TRANSMISSION IN THE MAMMALIAN BRAIN

RODRIGO ANDRADE - *WAYNE STATE UNIVERSITY SCHOOL OF MEDICINE, USA*
CHAIR: KATIA GYSLING - *DEPTO. DEPARTMENT OF CELLULAR AND MOLECULAR BIOLOGY, PONTIFICIA UNIVERSIDAD CATOLICA DE CHILE*
ROOM A - 8:30-9:30

Brain serotonin is synthesized by a few thousand neurons located in the brainstem that in turn innervate the entire neural axis. This highly divergent serotonergic input is thought to regulate neuronal networks and mediate the behavioral effects of serotonin. Until recently efforts to understand serotonergic synaptic transmission have been hampered by limitations in our ability to use electrical stimulation on such a divergent axonal projection. The advent of optogenetics, which affords the ability to selective stimulate genetically defined neuronal populations, offers an avenue to bypass this limitation.

SL02. TIME AND TIMING IN NEUROPHYSIOLOGY. LESSONS FROM SYNAPTIC TAGGING

JORGE A. BERGADO - *CIREN (CENTRO INTERNACIONAL DE RESTAURACIÓN NEUROLÓGICA), LA HABANA, CUBA*
CHAIR: GUSTAVO MURER - *FAC. DE MEDICINA, UBA, ARGENTINA*
ROOM A - 8:30-9:30

Time windows are frequent in the neurosciences, and their contribution to allow or prevent experience-dependent plasticity is relevant. Time windows may last a few seconds (like in classical conditioning), hours (memory consolidation), or years (imprinting and other forms of experience expectant plasticity). Synaptic tagging is also an example of the existence of a time window during which a transient modification in synaptic plasticity can be made endurable if a temporally associated event reinforces it. We have recently obtained evidence confirming the relevance of timing for memory (or neural plasticity) improvements. In a first series of experiments we demonstrate that the exploration of a novel environment rescues spatial memory affected by a strong food shock, but only if the exploration is allowed 15 minutes after training, and not five hours later. In the same line, we demonstrate that memory improving effects of erythropoietin on fimbria-fornix lesioned animals (a lesion that causes a severe and permanent loss of spatial memory) are only expressed when the trophic factor is administered within minutes after the training sessions and not five hours after that or several days before. The importance of a correct temporal organization of interventions may be of a great relevance for developing successful strategies in a wide range of human activity, from the School system toRestorative Neurology.

SL03. EFFECTS OF ENVIRONMENTAL ENRICHMENT ON BRAIN PLASTICITY, COGNITION, AND SOCIAL COMMUNICATION IN RATS

JUAN CARLOS BRENES - *INSTIT. FOR PSYCHOLOGICAL RESEARCH, NEUROSCI. RESEARCH CENTER, UNIVERSITY OF COSTA RICA*
CHAIR: JAIME FORNAGUERA - *UNIVERSIDAD DE COSTA RICA*
ROOM C - 8:30-9:30

Environmental enrichment (EE) is one of the most used paradigms to model neurobehavioral consequences of environmental stimulation in rodents. EE exerts beneficial effects on brain plasticity, cognition, and stress-coping responses. EE leads to a brain that can better counteract deficits and insults induced to resemble several neurological and psychiatric disorders. Here, evidence will be presented and discussed about how EE –as a whole or some of its components–, differentially affects non-associative learning (e.g., open-field habituation), spatial, episodic, and working memory, anxiety, social communication (i.e., ultrasonic vocalizations, USV), and amphetamine-induced locomotor activity and appetitive USV in rats. At the neurobiological level, expression of several genes and microRNAs related with neural plasticity on different brain regions will be shown. Discussion will be oriented to the use of preclinical studies including EE as potential treatment in models for neuropsychiatric disorders. Knowing about how animals react to different environmental conditions would contribute to explain why environmental stimulation in humans (rehabilitation and behavioral therapies, exercise, and preventive or palliative treatments) use to benefit some subjects but not others, an important enigma about the complex relationship between experience and neurobehavioral plasticity.

SL04. THE PERIAQUEDUCTAL GRAY AND PRIMAL EMOTIONAL PROCESSING CRITICAL TO INFLUENCE COMPLEX DEFENSIVE RESPONSES, FEAR LEARNING AND REWARD SEEKING

NEWTON CANTERAS[1], **SIMONE C. MOTTA**[1], **ANTÔNIO CAROBREZ**[2]
[1]DEPARTAMENTO DE ANATOMIA, INSTITUTO DE CIÊNCIAS BIOMÉDICAS, UNIVERSIDADE DE SÃO PAULO, SÃO; [2]DEPARTAMENTO DE FARMACOLOGIA, CCB, UNIVERSIDADE FEDERAL DE SANTA CATARINA, FLORIANÓPOLIS, BRASIL
CHAIR: FRANCISCO SILVEIRA GUIMARÃES - *MEDICAL SCHOOL OF RIBEIRAO PRETO-USP, BRAZIL*
ROOM A - 18:30-19:30

The periaqueductal gray (PAG) has been commonly recognized as a downstream site in neural networks for the expression of a variety of behaviors and thought to provide stereotyped responses. However, a growing body of evidence suggests that the PAG may exert more complex modulation in a number of behavioral responses and work as a unique hub supplying primal emotional tone to influence prosencephalic sites mediating complex aversive and appetitive responses. Of particular relevance, we review how the PAG is involved in influencing feelings of fear and terror in humans and complex forms of defensive responses, such as circa-trike and risk assessment responses in animals. In addition, we discuss putative dorsal PAG ascending paths that are likely to convey information related to threatening events to cortico-hippocampal-amygdalar circuits involved in the processing of fear learning. Finally, we discuss the evidence supporting the role of PAG in reward seeking and note the lateral PAG as part of the circuitry related to goal-oriented responses mediating the motivation to hunt and perhaps drug seeking behavior.

SL05. EXPLORING NEUROENDOCRINE MECHANISMS OF SEXUAL DIMORPHISM IN EARLY STRESS RESPONSE: A TRANSLATIONAL APPROACH

ZULMA DUEÑAS[1], JUAN CARLOS CAICEDO-MERA[2]
[1]ASSOCIATE PROFESSOR; [2]UNIVERSIDAD EXTERNADO DE COLOMBIA
CHAIR: ANDREA MILENA GARCÍA - *COLOMBIA*
ROOM B - 18:30-19:30

Sexual dimorphism in early stress response is a relevant field whose molecular mechanisms remain unclear. Despite several research have demonstrated differential hormonal actions and neurological changes related to gender, there are few studies that explore behavioral and biochemical aspects through integrative approaches. In this study, hormonal interactions of ovaric steroids and glucocorticoids in two neurons lines and behavioral effects of early stress protocol in a rat model were analyzed, in order to explore possible neuroendocrine mechanisms that explain dimorphic expressions of stress response. The CAD and SH-SY5Y neurons cultures treated with different doses of dexamethasone, 17β-Estradiol and progesterone showed bimodal dose-dependent effects on cell viability, consist on protective effects in low doses range (1 to 100 μM) and proapoptotic effects in high doses ranges (500 to 1000μM) when they were used alone. Some costimulation treatements at high doses(estradiol + dexamethasone and estradiol + progesterone) showed increased damage in CAD cells, while protective effect induced by 50 μM of estradiol were able to antagonize dexamethasone induced damage in SH-SY5Y cells. In rats, that received a maternal separation protocol of three hours in the morning and three hours in the afternoon during lactation period, showed that separate females expressed anxiety and hypoactivity behaviors while separate males group shows the opposite.

SL06. CALCIUM SIGNALING, CELLULAR OXIDATIVE TONE AND SYNAPTIC PLASTICITY

CECILIA HIDALGO - *BIOMEDICAL NEUROSCIENCE INSTITUTE, CEMC & ICBM, F. MEDICINE, UNIVERSIDAD DE CHILE*
CHAIR: OSVALDO UCHITEL - *IFIBYNE-CONICET, FCEN, UBA, ARGENTINA*
ROOM C - 18:30-19:30

Calcium signals, including signals generated by the highly redox-sensitive ryanodine receptor (RyR) calcium release channels, are essential for hippocampal synaptic plasticity and memory tasks. RyR inhibition - or incubation with the Alzheimer's disease associated amyloid beta oligomers (AbOs) - prevents BDNF-induced dendritic spine remodeling in primary hippocampal neurons and the associated RyR protein increase. Primary hippocampal neurons transfected with RyR2 shRNA display significant inhibition of RyR-mediated calcium release and lack BDNF-induced spine remodeling, which requires reactive oxygen species production. Additionally, LTP induction by TBS and performance of hippocampal-dependent memory tasks upregulate RyR2, while RyR inhibition prevents LTP induction by TBS. Moreover, decreasing RyR2 protein content by injecting rats intra-hippocampus with RyR2 antisense oligonucleotides or with AbOs leads to impaired performance in learning and memory tasks. We suggest that calcium signals generated via calcium release mediated by redox-modified RyR2 channels are essential for synaptic plasticity and hippocampal-dependent spatial memory processes, and that deficient RyR2-mediated calcium signaling contributes to AbOs-induced memory deficits.
Supported by BNI-09-015F; FONDECYT 1140545.

OCTOBER 19TH

SL07. NEUROPROTECTIVE EFFECT OF GUANOSINE IN EXPERIMENTAL MODELS OF BRAIN DISEASES

DIOGO ONOFRE SOUZA - *DEPARTAMENTO DE BIOQUÍMICA, ICBS, UFRGS, BRAZIL*
CHAIR: JORGE ALBERTO QUILLFELDT - *UNIVERSIDADE FEDERAL DO RIO GRANDE DO SUL (UFRGS), BRAZIL*
ROOM A - 8:30-9:30

Glutamate is the main excitatory neurotransmitter in mammalian CNS. However, the neurotoxicity caused by pathological high levels of extracellular brain glutamate is involved in the pathogenesis of various acute and chronic brain injuries. The maintenance of extracellular glutamate levels below toxic concentrations, thus favoring the physiological glutamatergic tonus, is exerted by glutamate uptake through transporters located mainly in astrocytes cell membranes.
Our group has given strong evidence that the guanine-based purinergic system is effectively neuroprotective against glutamate toxicity, in acute and chronic animal models, both in vitro and in vivo studies. Our results indicate that the neuroprotective guanine-based purine is the nucleoside guanosine (Guo). In vivo studies, Guo (i.c.v., i.p. or orally administered) protect against seizures (induced by QA), brain ischemia and hepatic encephalopathy.
Searching for mechanisms implicated in this neuroprotection, we pointed that Guo stimulates the astrocytic glutamate uptake in astrocyte cultures (from

newborn, adult and old rats). Additionally, in models of brain injury that is accompanied by a decrease in brain glutamate uptake (measured in brain slices), Guo simultaneously exerts neuroprotective effects and avoids the decrease in glutamate uptake.

SL08. WNT SIGNALING AND ALZHEIMER´S DISEASE

NIBALDO INESTROSA - *CENTRO DE ENVEJECIMIENTO Y REGENERACIÓN (CARE UC), CHILE*
CHAIR: JOSÉ BACIGALUPO - *UNIVERSIDAD DE CHILE, FACULTAD DE CIENCIAS, CHILE*
ROOM B - 8:30-9:30

Wnt signaling pathway is implicate in neural development and function, including dendrite morphogenesis, axonal growth and fine-tuning of synapses, defining the synaptic plasticity of neuronal circuits. Activation of Wnt signaling regulates synaptic structure and function in hippocampal excitatory neurons, promoting the PSD-95 clustering, development of dendritic spine morphogenesis as well as the increases in glutamatergic neurotransmission. Early studies indicated that the activation of Wnt signaling prevents the neurotoxicity induced by amyloid- (A) peptide aggregates. Current evidence associates Wnt dysfunction to Alzheimer disease´s (AD), namely: β-catenin levels are reduce in AD patients carrying presenilin-1-inherited mutations, the secreted Wnt antagonist Dickkopf-1 is elevated in postmortem AD brains, and a variant of the LRP6 is associated with late-onset AD. In this seminar, I will present our recent work on the biology of the Wnt signaling in the nervous system, as well as, in vivo studies on the effect of Wnt signaling in AD animal models, including the double transgenic APPswe/PS-1 mouse, and the Chilean natural model Octodon degus. Results point to a neuroprotective potential of the Wnt cascades as a therapeutic approach to control AD.

SL09. DURATION MODULATION OF AVERSIVE MEMORIES AND APPETITIVE

JORGE MEDINA - *FACULTAD DE MEDICINA, UNIVERSIDAD DE BUENOS AIRES, ARGENTINA*
CHAIR: PEDRO BEKINSTEIN - *FAC. DE MEDICINA, UBA, ARGENTINA*
ROOM A - 18:30-19:30

Persistence is the most characteristic attribute of long-term memory (LTM). However, little is known about the mechanisms that make LTMs last longer than others. We found that a novel protein synthesis- and BDNF-dependent late phase in the hippocampus is critical for persistence, but not formation of fear LTM storage. Moreover, increasing BDNF levels in the hippocampus 12 hr after training is sufficient to induce memory persistence, transforming a non-lasting LTM trace into a persistent one. We also found that persistence of LTM depends on the activation of VTA/hippocampus connections controlling BDNF expression, and is modulated by noradrenergic and serotonergic influences. Persistence of a cocaine-associated memory is regulated in a manner opposite to that observed in fear memories. The role of this late consolidation phase in the hippocampus on systems consolidation processes will be discussed alongside with other research interests we have in the present days. For instance, which are the strategies to maintain or attenuate memories.

SL10. SCHWANN CELL TO AXON RNA TRANSFER

JOSÉ SOTELO - *INSTITUTO DE INVESTIGACIONES BIOLÓGICAS CLEMENTE ESTABLE, MONTEVIDEO, URUGUAY*
CHAIR: MARÍA CASTELLÓ - *IIBCE, URUGUAY*
ROOM C - 18:30-19:30

The existence of RNA in axons now has accumulated abundant experimental evidence. Much of the disputes turned now to the origin of these axonal RNAs. The neuronal soma as the source of most axonal RNAs is indisputable. However, the surrounding glial cells emerged as a supplemental source of axonal RNAs. Here, we focus on addressing the glial origin of axonal RNAs and ribosomes. We describe this process in both invertebrate axons and vertebrate axons. Court et al showed that Schwann cell to axon ribosomes transfer exists. Moreover, we showed Glia to axon RNA transfer in Peripheral axons (2013). Carsten (2013) also showed that Oligodendroglia transfer RNA to central axons. Recently, Ion Torrent massive sequencing of immunoprecipitated (Schwann cell synthesized) Bromo-uridine-mRNAs yielded hundreds of axonal mRNAs (i.e. neurofilaments, ankirin, actin, etc.). This implies important consequences respect the integration of glial and axonal function. This evolving field will certainly impact in the understanding of the cell biology and physiopathology of the axon. Moreover, if axonal protein synthesis can be controlled by the interacting glia, the possibilities for human clinical interventions in nerve injury and neurodegeneration are greatly increased.

OCTOBER 20TH

SL11. ACTIVITY AND NEUROGENESIS-MEDIATED CIRCUIT REMODELING IN THE HIPPOCAMPUS

ALEJANDRO SCHINDER - *INSTITUTO LELOIR, ARGENTINA*
CHAIR: DANIEL CALVO - *INGEBI-CONICET, ARGENTINA*
ROOM A - 8:30-9:30

The dentate gyrus is the first relay station in information flow from the entorhinal cortex towards the hippocampus, and it plays a crucial role in memory processing. A remarkable feature of the dentate circuitry is the unique degree of plasticity conveyed by its ability to generate and integrate new principal neurons (granule cells, GCs) through life. Adult-born GCs are important for specific forms of memory, such as those that demand fine discrimination of subtle differences, particularly during spatial tasks. My laboratory has focused on understanding the modifications of local dentate networks produced by the incorporation of newly generated GCs, their interaction with the microenvironment (niche), and their functional implications.
Adult-born GCs develop and connect over several weeks before they become mature. Our recent findings reveal

that new GCs may play distinct roles in memory encoding as they walk through the road of development. In addition, developing GCs undergo two critical periods of high sensitivity to electrical signals arising from their local microenvironment. At these times, their functional profile becomes tagged by behavior, resulting in long-lasting changes in connectivity and function. In my talk I will discuss recent approaches combining opto- and chemogenetics that we have used to understand the function of developing GCs and the mechanisms that transduce behavioral experiences into changes in the integration and plasticity of new GCs.

SL12. MELANIN CONCENTRATING HORMONE IN MESOPONTINE RAPHE NUCLEI: ROLE IN REM SLEEP AND DEPRESSION

PABLO TORTEROLO - *DEPARTMENT OF PHYSIOLOGY, SCHOOL OF MEDICINE, UNIVERSIDAD DE LA REPÚBLICA, URUGUAY*
CHAIR: PATRICIA LAGOS - *FACULTAD DE MEDICINA, UDELAR, URUGUAY*
ROOM B - 8:30-9:30

The melanin-concentrating hormone (MCH) is a neuromodulator synthesized by neurons of the postero-lateral hypothalamus. MCHergic neurons project to the serotonergic dorsal (DR) and median (MR) raphe nuclei. These nuclei have a major role both in the control of REM sleep and in the pathophysiology of Major Depression (MD). In this lecture I will summarize and evaluate our experimental data about the functional interactions between the MCHergic systems and the raphe nuclei, in the control of REM sleep and MD.
Our main findings are the following. MCHergic receptors are present in the serotonergic neurons of the DR and MR. Microinjections of MCH into the DR promote REM sleep in the rat, while immunoneutralization of this peptide within the DR, decreases the time spent in this state. Moreover, microinjections of MCH into the DR and MR promote a depressive-like behaviour. This effect is blocked by the intra-DR microinjection of a specific MCH receptor antagonist, and prevented by the systemic administration of antidepressant drugs (either fluoxetine or nortriptyline). Using electrophysiological and microdialysis techniques, we also demonstrated that MCH decreases the activity of serotonergic DR and MR neurons.
In conclusion, there is substantive experimental data suggesting that by modulating the neuronal activity of the DR and MR, the MCHergic system plays a role in the control of REM sleep and in the pathophysiology of MD.

SL13. NEURAL BASIS OF ATTENTION TO MULTI-PART, HIERARCHICALLY ORGANIZED, OBJECTS

MITCHELL VALDÉS-SOSA - *CUBAN CENTER FOR NEUROSCIENCE, CUBA*
CHAIR: MARÍA EUGENIA PEDREIRA - *IFIBYNE-CONICET, FCEN, UBA, ARGENTINA*
ROOM C - 8:30-9:30

Visual attention can be directed at an object as a whole (the global level) or to its parts (the local level). The cortical circuitry enabling these attentional configurations is not fully understood. This topic has been studied with hierarchical Navon figures, global letters made out of local letters. Using a novel paradigm we separated the presentation of these two levels in time. We found that seeing a shape at the global or local level momentarily blocks from awareness additional shapes from the other level, an interference not present for shapes from the same level. Using event related potentials we show that this attentional selection modulates early potentials with probable sources in visual extra-striate cortex. Moreover, by examining local activation patterns with functional MRI, we found a divergent specialization for the abstract information provided by the hierarchical figures. Information about shape (invariant to changes in level) was carried preferentially by lateral ventral-occipito-temporal cortex (VOT), overlapping object- and face-selective cortex. Conversely, information about level (invariant to changes in shape) was preferentially carried by medial VOT, and occipital areas partly covering house/scene-selective cortex This suggests a shared circuitry processing scene-layout and the internal structure of multipart objects, which is exploited by attention to control the access of shapes into awareness.

SYMP

#FALAN2016

PROGRAM IN DETAIL

SYMPOSIA

Symposia

OCTOBER 17TH

SY1. NOVEL INSIGHTS INTO HYPOTHALAMIC MECHANISMS CONTROLLING BODY HOMEOSTASIS

ROOM A - 11:00-13:00

CHAIR: MARIO PERELLO (ARGENTINA)

The hypothalamus is a functionally and structurally complex brain structure that constantly integrates a variety of peripheral signals and generates combined physiological responses essential for the body homeostasis regulation. In this symposium, we will present an update of some novel aspects of these very sophisticated and recently elucidated hypothalamic mechanisms.

SPEAKER: JAVIER STERN, *MEDICAL COLLEGE OF GEORGIA, AUGUSTA UNIVERSITY, GEORGIA, USA*

NON-CONVENTIONAL MODALITIES OF NEUROTRANSMISSION IN THE HYPOTHALAMUS: WHERE THE TORTOISE AND THE HARE MEET

It is classically considered that the proper functioning of the central nervous system is dependent upon communication between pairs of neurons, which is mediated by chemical neurotransmission at well-defined synaptic structures. However, research in the past decade has gradually expanded the repertoire of cell-cell signaling mechanisms, to include modalities that operate at very different spatio-temporal scales from classical temporally fast and spatially constrained synapses. An emerging model for the study of these distinct forms of neurotransmission is the hypothalamus, a brain region in which communication among functionally distinct neuronal types, ranging from cell-to-cell to interpopulation signaling, is critical for the generation of multimodal homeostatic responses. In my talk I will present recent data from our laboratory regarding how classical and non-conventional neurotransmission modalities work in concert in the regulation of hypothalamic neuronal activity, highlighting the key role that glial cells play in these interactions. I will discuss the functional relevance of these signaling modalities in the context of hypothalamic generation of cardiovascular and energy balance homeostatic responses.

SPEAKER: JOSE DONATO JR. - *UNIVERSITY OF SAO PAULO, BRASIL*

LEPTIN SIGNALING IN METABOLIC ADAPTATIONS OF PREGNANCY

During pregnancy, women normally increase their food intake and body fat mass, and exhibit insulin resistance. However, an increasing number of women are developing metabolic imbalances during pregnancy, including excessive gestational weight gain and gestational diabetes mellitus. Despite the negative health impacts of pregnancy-induced metabolic imbalances, their molecular causes remain unclear. In this talk, I will summarize our recent findings that identified the molecular mechanisms responsible for orchestrating the metabolic changes observed during pregnancy. In summary, we found that increased hypothalamic expression of SOCS3 is a key mechanism responsible for triggering pregnancy-induced leptin resistance and metabolic adaptations.

SPEAKER: MARIO PERELLO, *LABORATORIO DE NEUROFISIOLOGIA, INSTITUTO MULTIDISCIPLINARIO DE BIOLOGIA CELULAR, ARGENTINA*

NEURONAL CIRCUITS BY WHICH GHRELIN REGULATES STRESS AND EATING BEHAVIORS

The understanding of the neurobiological bases underlying food intake behaviors is essential to understand the normal physiology and also important for the further development of treatments for people suffering eating disorders. In order to get insights into the complex neural mechanisms regulating food intake, our laboratory has focused on the study of a stomach-derived hormone, named ghrelin, which is recognized as the only known orexigenic circulating peptide as well as a potent stress signal to the brain. Over the last years, our work has helped to define essential roles for ghrelin in mediating reward-based eating as well as in stress-related responses. Our data support the notion that the neuronal targets mediating ghrelin's role as an orexigenic vs. a stress signal are anatomically dissociated. Recently, we have also shown that neuronal targets mediating ghrelin's role on food reward or homeostatic eating are also dissociated. Thus, we propose that ghrelin impacts on first order neuronal targets of specific neuronal circuits that mediate each ghrelin´s role, and then these neuronal circuits are integrated in order to display coordinated responses.

SPEAKER: MATTHIAS TSCHÖP, *INST. FOR DIABETES AND OBESITY, BAVARIA, GERMANY*

THE METABOLIC SYNDROME: A BRAIN DISEASE?

All metabolic processes, from single cell substrate oxidation to complex behaviors, are under the control of specific CNS circuits, aiming to maintain homeostasis. Afferent signals include gut hormones, adipokines and nutrient components, while efferent information primarily originates from the hypothalamic nuclei and involves components of the autonomic nervous system as well as the classic endocrine axes. We recently observed that diet-induced metabolic diseases, such as obesity and type 2 diabetes, are associated with (and preceded by) pathological processes in these hypothalamic control centers. Such pathophysiology concerns the hypothalamic cell matrix beyond key neuronal populations and includes astrocytosis, microgliosis, hypervascularisation as well as increased presence of pro-inflammatory cytokines. Specific targeting of such "hypothalamic inflammation" using novel gut-peptide based delivery of glucocorticoids to key metabolic disease regions improved both local pathophysiology and systemic metabolic health. Such a novel unimolecular dual agonism and steroid delivery approach may not only offer superior therapeutic option for at least some patient subpopulations, but also suggests a pathogenetic relevance for this novel hypothalamic syndrome.

SY2. SLEEP, MEMORY AND CONSCIOUSNESS

ROOM B - 11:00-13:00

CHAIRS: CECILIA FORCATO (ARGENTINA) AND FELIPE BEIJAMINI (BRAZIL)

The study of the role of sleep in memory formation, its interaction with stress and the processing of information during sleep is a frontier topic in neuroscience.

In this Symposium we will discuss the active role of sleep in memory consolidation as well as the interaction between sleep and stress and the processing of information in this state of reduced consciousness.

SPEAKER: JESSICA PAYNE, *DEPARTMENT OF PSYCHOLOGY, UNIVERSITY OF NOTRE DAME, USA*

STRESS, SLEEP, AND MEMORY CONSOLIDATION: INDEPENDENT AND INTERACTIVE EFFECTS

Separate lines of research demonstrate that elevated cortisol can selectively benefit the consolidation of emotional memories, as can the occurrence of sleep soon after learning. The first part of my talk will examine the separate roles that stress and sleep play in the formation of emotional memories. In the second part, I will discuss new evidence, from behavioral, psychophysiological, and neuroimaging studies, suggesting that stress and arousal interact with sleep to benefit memory consolidation, particularly for negative arousing information. I will conclude by presenting a model suggesting that stress hormones may help 'tag' attended information as important to remember at the time of encoding, thus enabling subsequent, sleep-based processes to optimally consolidate information in a selective manner.

SPEAKER: TRISTAN BEKINSCHTEIN, *DEPARTMENT OF PSYCHOLOGY, UNIVERSITY OF CAMBRIDGE; UK*

FRAGMENTATION AND RESILIENCE OF COGNITIVE PROCESSES AS WE FALL ASLEEP

Little we know about the time when we fall into Morpheus' arms. The transition from wake to sleep is a thoroughly unexplored biological process that in humans is enormously variable and has not been characterized beyond some electrophysiological reports. In this program of research we have decided to build a framework to define the dynamics of the transition when people are falling asleep as they take different types of decisions. How does cognition fragment as we fall asleep? In a first series of experiments the findings show the different styles of transition, and how we stop responding but continue to take decisions, even deep into sleep stage 2. In a second wave of experiments we show how the system adapts to the change of resources when drowsy and shifts the cognitive processes between brain networks and cortical areas. Preliminary findings show that when drowsy we lose attention to the left side of the worlds (attentional hemineglect); we also continue to channel semantic decision to frontoparietal networks when asleep (markers of intention in stage 2); and we keep the threshold of perceptual detection but lose precision and shift the neural markers of that process from perceptual to decision making areas. These findings together represent the first attempt to understand the true plasticity of the brain when we change between fully conscious to less alert states, a transition that happens several times per day to every person in the world.

SPEAKER: JAN BORN, *DEPARTMENT OF MEDICAL PSYCHOLOGY AND BEHAVIORAL NEUROBIOLOGY, UNIVERSITY OF TÜBINGEN, GERMANY*

MECHANISMS OF SLEEP-DEPENDENT MEMORY FORMATION – DEVELOPMENTAL ASPECTS

Sleep favors the consolidation of memory. Recent studies have elucidated some of the neurophysiological mechanims underlying this consolidation process during sleep, especially in the hippocampus-dependent declarative memory system. This system is capable of rapidly forming an initial memory representation for an episode upon its one-time occurrence, and is thus at the basis of the formation of any long-term memory. Consolidation of hippocampus-dependent memories represents an active systems consolidation process that takes place mainly during slow wave sleep (SWS) rather than REM sleep. It critically relies on the neural reactivation of newly encoded memory representations which originates from hippocampal circuitry and is thought to promote the gradual redistribution of the representations towards extra-hippocampal, mainly neocortical networks serving as long-term store. This talk will cover developmental aspects of active systems consolidation during sleep. Compared with adults, children display longer and deeper SWS with increased <1 Hz slow oscillatory EEG activity and spindle activity. In parallel, memory consolidation during sleep in the hippocampus-dependent declarative memory system is enhanced in children, which goes along with a stronger transformation of the initial memory representations. In this way, sleep in children appears to particularly support the formation and storage of abstracted schema-like memories.

SPEAKER: SIDARTA RIBEIRO, *BRAIN INSTITUTE, BRAZIL*

Sleep-dependent plasticity and memory change: Strengthening, forgetting, and restructuring

SY3. MACROMOLECULAR SIGNALING COMPLEXES IN NEURONS

ROOM C - 11:00-13:00

CHAIR: MARK SHAPIRO (USA)

Signaling cascades that regulate neuronal activity use limited number of second messengers, therefore, in order for intracellular signaling to function with high fidelity, a precise spatiotemporal localization of intracellular signals must exist. Leading scientists from three continents will discuss current hot topics and technical advances in the field of neuronal localised intracellular signaling.

SPEAKER: NIKITA GAMPER, *FACULTY OF BIOLOGICAL SCIENCES, UNIVERSITY OF LEEDS, LEEDS, UK; DEPARTMENT OF PHARMACOLOGY, HEBEI MEDICAL UNIVERSITY, SHIJIAZHUANG, CHINA*

COUPLING OF CALCIUM-ACTIVATED CHLORIDE CHANNEL TMEM16A TO LOCALIZED CALCIUM SIGNALS IN SENSORY NEURONS

Ca2+-activated Cl- channels TMEM16A (ANO1) are expressed in nociceptive ('pain') sensory neurons where these are thought to play an excitatory role. Accordingly,

TMEM16A activation was shown to contribute to inflammatory and thermal pain. Since there are many types of intracellular Ca2+ signals, nociceptors must be able to differentiate between those originating from the tissue-damaging stimuli and 'other' Ca2+ signals. We found that TMEM16A in nociceptive dorsal root ganglion (DRG) neurons couple to two distinct localised Ca2+ sources: i) G protein coupled receptor (GPCR)-mediated release of Ca2+ form the endoplasmic reticulum (ER), and ii) Ca2+ influx via the TRPV1 channels. Intriguingly, Ca2+ influx through the voltage-gated Ca2+ channels was ineffective to activate TMEM16A. Coupling of TMEM16A to the ER Ca2+ release was mediated by signaling complexes assembled at the plasma membrane (PM)-ER junctions. The complex ensures close apposition and physical association of PM's TMEM16A channels and GPCR (i.e. bradykinin B2 and PAR2 receptors) with ER's IP3 receptors, which serve as Ca2+ sources for TMEM16A activation. Disrupting these complexes resulted in 'promiscuous' activation of TMEM16A by global cytosolic Ca2+ signals which, in turn, increased excitability of nociceptors. In sum, we postulate the existence of multiprotein signaling complexes, which bring together TMEM16A with their dedicated Ca2+ sources while protecting the channels form from 'irrelevant' Ca2+ signals.

SPEAKER: FRANCISCO J. BARRANTES

LABORATORY OF MOLECULAR NEUROBIOLOGY, INSTITUTE OF BIOMEDICAL RESEARCH, UCA-CONICET, ARGENTINA

NANOCLUSTER ORGANIZATION AND DYNAMICS OF SYNAPTIC PROTEINS

Synaptic transmission relies on an adequate balance of receptor synthesis, delivery to and removal from the cell membrane and anchorage by scaffolding and cytoskeletal components. In order to understand the interplay between these intervening molecules, it is necessary to define their supramolecular organization, dynamics and trafficking. Here we interrogate neuronal and muscle-type nicotinic acetylcholine receptors (nAChRs) and other synaptic components using a combination of ensemble averaging methods and single-molecule experimental techniques. Two independent superresolution microscopy techniques -STED and STORM/GSDIM- provide snapshot information on the "social" supramolecular organization of receptors in a clonal cell line heterologously expressing muscle-type nAChR and in hippocampal neuronal cells. In both cases nanometer-sized aggregates ("nanoclusters") can be imaged with nanometer precision and their density, number of molecules per cluster and other structural parameters defined. Cholesterol levels affect the surface architecture and dynamics of the nAChR nanodomains and individual macromolecules, the mobility of which can be followed in living cells using single-particle tracking techniques. The possible functional implications of these spatio-temporal properties of synaptic macromolecules will be discussed.

Supported by grants PICT 2011-0604 and 2015-2654 from Mincyt and PIP 11220150100858 from CONICET.

SPEAKER: MARK S. SHAPIRO[1], CHASE M. CARVER[1], FRANK CHOVEAU[1], JIE ZHANG[1]

[1]UNIVERSITY OF TEXAS HEALTH SCIENCE CENTER AT SAN ANTONIO, SAN ANTONIO, TX USA

CLUSTERING AND FUNCTIONAL COUPLING OF DIVERSE ION CHANNELS AND SIGNALING PROTEINS REVEALED BY SUPER-RESOLUTION STORM MICROSCOPY IN NEURONS

Neuronal ion channels are exquisitely regulated by intracellular signaling molecules which typically use scaffold proteins, such as A-kinase anchoring proteins (AKAPs) to orchestrate protein assemblies for spatiotemporal specificity. "M-type" K+ channels (KCNQ2-5) play key roles in regulating neuronal excitability. "L-type" Cav1 Ca2+ channels are critical for synaptic plasticity and excitation/transcription coupling. In sensory neurons, TRPV1 cation channels respond to heat, acidity or chemical ligands, initiating nociception. AKAP79/150 recruits PKA, PKC, calcineurin and receptors into signaling complexes centered on these three types of channels. However, optical observation of such individual complexes containing these proteins has not been achieved due to the intrinsic diffraction limit of light (~250 nm). I will show how we have addressed those questions using super-resolution STochastic Optical Reconstruction Microscopy (STORM) and electrophysiology. We also probed if AKAP79/150 directs "super-complexes" involving multiple channels. Indeed, we find AKAP150-mediated super-clusters in sensory neurons, showing AKAP79/150-mediated physical coupling of multiple and distinct ion channels. Moreover, we find functional coupling of these diverse channels, dependent on AKAP79/150. Our findings illustrate the novel role of AKAP79/150 as a coupler of different proteins to convey cross-talk between channel activities in controlling the physiological responses of neurons.

SPEAKER: RAMON LATORRE, *CENTRO INTERDISCIPLINARIO DE NEUROCIENCIA DE VALPARAÍSO, UNIVERSIDAD DE VALPARAÍSO, CHILE*

ALLOSTERISM AND STRUCTURE IN THERMALLY-ACTIVATED TRANSIENT RECEPTOR POTENTIAL CHANNELS

The molecular sensors for temperature changes in living organisms are a large family known as thermosensitive Transient Receptor Potential (TRP) ion channels. These membrane proteins are polymodal receptors in the sense that they can be activated by cold or hot temperatures, depending on the channel subtype, voltage, and ligands. The stimuli sensors are allosterically coupled to a pore domain, increasing the probability of finding the channel in its ion conductive conformation. We will discuss the allosteric coupling between the temperature and voltage sensor modules and the pore domain, to then discuss the thermodynamic foundations of thermo-TRP channel activation. A structural overview of the molecular determinants of temperature sensing is provided. We also posit an anisotropic thermal diffusion model that may explain the large temperature sensitivity of TRP channels. Additionally, we discuss the effect of several ligands on TRP channels function, and the evidence regarding their mechanisms of action.

SY4. PROFESSOR JOHN G NICHOLLS CELEBRATION SYMPOSIUM: FUNCTION, REPAIR AND TRAINING OF THE NERVOUS SYSTEM

ROOM A - 15:00-17:00

CHAIR: ELAINE DEL-BEL (BRAZIL) AND FRANCISCO DE MIGUEL (MEXICO)
In this symposium, lecturers will talk about their own scientific research, teaching experiences and how it was influenced by working with John Nicholls.

ORGANIZERS:

E. DEL-BEL (*USP-RIBEIRAO PRETO, BR*), **F. F. DE-MIGUEL** *(UNAM, MEXICO),* **OSVALDO UCHITEL** *(FALAN PRESIDENT),* **PIERRE MAGISTRETTI** *(IBRO PRESIDENT)*

Introduction: **FRANCISCO F. DE-MIGUEL**

SPEAKER: LIRIA MASUDA-NAKAGAWA, *UNIVERSITY OF CAMBRIDGE, DEPT OF GENETICS, UK*

FUNCTIONAL CIRCUITRY OF A SENSORY DISCRIMINATION AND LEARNING CENTER IN A SIMPLE BRAIN, LARVAL DROSOPHILA

Discrimination of sensory signals underlies memory formation and retrieval. In insects and mammals, sensory signals are represented in the higher brain, highly selectively. The aim of our work is to understand the circuit mechanisms that regulate selectivity and sparseness of sensory representations.

The mushroom bodies (MBs) of insect brains are higher order brain centers essential for associative olfactory learning. The relatively simple Drosophila larval MB calyx, the sensory input region, is organized in glomeruli, each receiving stereotypic input from a single projection or other input neuron. This allows a sensory map of all olfactory sensory neurons in the calyx. In contrast to stereotypic PN innervation, innervation of calyx glomeruli by MB neurons, Kenyon cells (KC) appears random. This pattern of connectivity is consistent with a model in which KC dendrites process olfactory input by a combinatorial mechanism that can discriminate a large number of odors.

However, the activity of the calyx must subject to regulation. We are now addressing how inhibition and other potential modulatory neurons regulate the activity in the calyx. By using the larval brain EM connectome, we are now dissecting the circuits that provide inputs and outputs to the calyx, and testing the roles of novel neurons by functional imaging and behavior. Our data will help reveal the logic of information processing that determines and regulates the selectivity of sensory representation in the MBs.

SPEAKER: JUÁN FERNANDEZ, *FACULTAD DE CIENCIAS. UNIVERSIDAD DE CHILE*

The early zebrafish embryo as a model for the study of cytoplasmic movements

SPEAKER: ROMMY VON BERNHARDI, *NEUROLOGY, SCHOOL OF MEDICINE, PONTIFICIA UNIVERSIDAD CATÓLICA DE CHILE, SANTIAGO, CHILE*

MICROGLIAL CELL DYSREGULATION IN BRAIN AGING AND NEURODEGENERATION

Aging is the main risk factor for Alzheimer's disease. We have developed the "glia-dysregulation" hypothesis that proposes that age-related impairment of microglia regulation is involved in AD pathogenesis. We have found that age-related changes on TGF-beta1 results in microglia dysregulation, neuroinflammation and increased neurotoxicity. Astrocytes regulation of microglia cytotoxicity and Aβ removal is mediated by TGF-beta1. However, TGF-beta1/Smad signaling is reduced in adult mice. Reduced activation of TGF-beta1/Smad is associated with changes on the activation of microglia: impaired expression of SR-A, which in turn associated with altered cytokine profiles in plasma and in the hippocampus as mice age. Modulation is at least partially dependent on the activation of TGF-beta1/Smad pathway and is impaired in chronic inflammation. Phagocytosis of Aβ is induced by inflammation and TTGF-beta1 only in microglia obtained from young mice, and is prevented by Smad inhibition. Our results show that the TGF-beta1/Smad pathway regulates the expression of scavenger receptors and the activation pattern of microglia, which is impaired in aging and chronic inflammatory preconditioning. The impairment reduces protective activation while facilitating microglia-mediated neurodegenerative changes and cognitive impairment.

Support: Grant FONDECYT 1131025.

SPEAKER: JAIME EUGENIN, *UNIVERSIDAD DE SANTIAGO DE CHILE*

THE ALTERATION OF NEONATAL RAPHE NEURONS BY PRENATAL-PERINATAL NICOTINE. MEANING FOR SUDDEN INFANT DEATH SYNDROME

Prenatal nicotine exposure is proposed as a probable link between smoking habit during pregnancy and Sudden Infant Death Syndrome (SIDS) in humans. We demonstrated that nicotine (60 mg Kg-1 day-1) administered subcutaneously with osmotic minipumps in CF1 mice from gestational days 5-7 to early postnatal life leads to blunted ventilatory responses to hypercarbia, and reduced central chemoreception in P0-P5 neonates. Because SIDS infants show several abnormalities in the serotonergic structures in the brainstem, we studied the effects of prenatal-perinatal nicotine on raphe neurons in mice. We found that in nicotine-exposed neonates, caudal raphe neurons are hypoactive and show a reduced innervation of the ventral respiratory column. In addition, the respiratory responses evoked by serotonin agonists and the expression of 5HT1A receptors are altered. Therefore, prenatal nicotine exposure modifies the respiratory rhythm and impairs the central chemoreception during the early postnatal life as a consequence of serotonergic system alteration. These results are relevant to understand possible pathogenic mechanisms of SIDS.

SPEAKER: ELAINE DEL-BEL, *USP RIBEIRAO PRETO. BRAZIL*

Identification of gene expression during CNS regeneration

From courses to studying regeneration and later with Walter Stuhmer

SPEAKER: FRANCISCO F. DE MIGUEL, *INSTITUTO DE FISIOLOGÍA CELULAR, UNAM. MEXICO*

Release of transmitters from synapses and cell bodies

Serotonin release from the neuronal cell body

Concluding remarks **RUBIA WEFFORT / CILENE LINO-DE-OLIVEIRA / ELAINE DEL-BEL**
Experience as students and partners.

SY5. NEW INSIGHTS INTO SYNAPTIC PLASTICITY

ROOM B - 15:00-17:00

CHAIR: ANTONIO RODRIGUEZ-MORENO (SPAIN)
Synaptic plasticity is one of the main properties of the brain. Understanding the mechanisms and functions of plasticity in development, learning and memory, as well as recovery after brain injury is an important topic with wide appeal, and this controversial topic will promote an interesting debate and contribute to clarity in a sometimes confusing field.

SPEAKER: ANTONIO RODRIGUEZ-MORENO, *UNIVERSIDAD PABLO DE OLAVIDE, SPAIN*
SPIKE TIMING-DEPENDENT PLASTICITY IN THE CORTEX AND THE HIPPOCAMPUS
Spike timing-dependent plasticity (STDP) is a Hebbian learning rule important for synaptic refinement during development and for learning and memory in the adult. We have investigated the requirements for induction of spike timing-dependent long-term potentiation (t-LTP) and spike timing-dependent long-term depression (t-LTD) in the hippocampus and the cortex and the mechanisms of these two forms of plasticity. We found that both t-LTP and t-LTD can be induced at L4-L2/3 cortical synapses as well as at hippocampal CA3-CA1 synapses by pairing presynaptic activity with single postsynaptic action potentials at low stimulation frequency (0.2 Hz). Both t-LTP and t-LTD require NMDA-type glutamate receptors for their induction, but the location and properties of these receptors are different: While t-LTP requires postsynaptic ionotropic NMDA receptor function, t-LTD does not. Both t-LTP and t-LTD require postsynaptic Ca2+ for their induction. Induction of t-LTD also requires metabotropic glutamate receptor activation, phospholipase C activation, postsynaptic IP3 receptor-mediated Ca2+ release from internal stores, postsynaptic endocannabinoid (eCB) synthesis, activation of CB1 receptors and astrocytic signalling, possibly via release of the gliotransmitters glutamate (in the cortex) and D-serine (in the hippocampus). We furthermore found that presynaptic calcineurin is required for t-LTD induction.

SPEAKER: MARCO FUENZALIDA, *INSTITUTO DE FISIOLOGÍA, FACULTAD DE CIENCIAS, UNIVERSIDAD DE VALPARAÍSO; CENTRO DE NEUROBIOLOGÍA Y PLASTICIDAD CEREBRAL, UNIVERSIDAD DE VALPARAÍSO, CHILE*
ACTIVITY-DEPENDENT SYNAPTIC PLASTICITY OF GABAERGIC SYNAPSES
Long-term changes in synaptic transmission are considered the cellular basis of learning and memory. Over the last decade, many studies have revealed that the precise order and timing between pre- and post-synaptic activity ("spike-timing-dependent plasticity; STDP") is crucial for the sign and magnitude of long-term potentiation (LTP) or long-term depression (LTD) at many synapses. Neuromodulatory systems including the dopaminergic, serotoninergic and cholinergic system can modulate the strengthening or weakening of synaptic transmission by regulating the magnitude of LTP/LTD. Here, we will summarized and discusses mechanistic aspects of neuromodulation of activity dependent synaptic plasticity in inhibitory synapses, with an emphasis on cholinergic and endocannabinoids system and its role in regulating STDP-iLTD in healthy brain and disease.

SPEAKER: FRANCISCO URBANO, *INSTITUTO DE FISIOLOGÍA, BIOLOGÍA MOLECULAR Y NEUROCIENCIAS (IFIBYNE-CONICET-UBA), ARGENTINA*
Psychostimulant-induced alterations on thalamic GABAergic plasticity
The effects of cocaine on thalamic GABAergic transmission resemble those described in several psychiatric and neurological pathologies included in the thalamocortical dysrhythmia syndrome, characterized by an anomalous coherence between high and low EEG frequencies. The presence of low frequencies in awake individuals is thought to cause aberrant processing of sensory inputs. Our group has compared cocaine and methylphenidate on synaptic transmission during repetitive stimulation. We found that cocaine administration, either acute or sub-chronic, led to an increase in paired pulse ratio values during electrical stimulation of GABAergic afferent to ventrobasal neurons at either 10 Hz or 40 Hz. Furthermore, only cocaine sub-chronic administration induced changes in 10 Hz/10 pulse trains of stimulation. Comparatively, the effects of methylphenidate are subtle, suggesting the existence of a cocaine-mediated serotonergic modulation of the inhibitory synapse between the thalamic reticular nucleus and the Ventrobasal nucleus.

SY6. PATIENT-DERIVED INDUCED PLURIPOTENT STEM-LIKE CELLS AS MODELS FOR NEURODEGENERATIVE DISESES

ROOM C - 15:00-17:00

CHAIR: LISANDRO J. FALOMIR LOCKHART (ARGENTINA)
Discovery of cellular induced pluripotency and reprogramming extended the horizons of medicine for the near future. Significant effort has been made to understand and control these processes, and now we can manipulate readily accessible cells from patients to resemble those from inaccessible tissues, such as the brain. Reproducing complex diseases in a dish allow us to study their molecular basis.

SPEAKER: GERSON CHADI
NEUROREGENERATION CENTER. DEPARTMENT OF NEUROLOGY. UNIVERSITY OF SAO PAULO SCHOOL OF MEDICINE. BRAZIL
MOLECULAR MODELING OF HUMAN INDUCED PLURIPOTENT STEM CELL-DERIVED MOTOR NEURONS FROM FIBROBLASTS OF MOTOR NERVES REVEALS PATHOPHYSIOLOGICAL MECHANISMS OF SPORADIC FORM OF AMYOTROPHIC LATERAL SCLEROSIS
The detailed mechanisms related to neurodegeneration in neurodegenerative disorders are still unknown. The methodology to study genetic, molecular and cellular events of human neurological diseases is current under development, increasing the expectation the discovery

of therapeutic targets that allow effective translation of proposed clinical trials. Amyotrophic Lateral Sclerosis (ALS) is a fatal neurodegenerative disease that leads to widespread motor neuron death, general palsy and respiratory failure. We have developed the methodology to allow gene expression modeling of sporadic ALS, the most prevalent form of disease, by employing human induced pluripotent stem cells-differentiated motor neurons (generated from fibroblasts of still functional motor nerves) linked to DAVID Functional Annotation Bioinformatics Microarray Analysis using a whole human genome platform. DAVID analyses of differentially expressed genes identified molecular function/biological process-related genes through Gene Ontology terms, summarized by REVIGO, and also genes related to KEGG signaling pathways. Specific software for Protein Interaction Network Analysis showed the degree of interaction of deregulated gene expression. The overall analysis showed a strong association between mitochondrial function and cellular processes possibly related to motor neuron degeneration. Supported by: FAPESP and CNPq, Brazil.

SPEAKER: LISANDRO J. FALOMIR LOCKHART, *INIBIOLP (UNLP, CCT-LA PLATA, CONICET), ARGENTIN*

METABOLIC AND DIFFERENTIATION IMPAIRMENT IN PARKINSON'S DISEASE PATIENT-DERIVED IPSCS WITH A TRIPLICATION EVENT INCLUDING THE SNCA LOCUS

Parkinson's disease (PD) is the 2nd most common neurodegenerative disorder. Its pathologic hallmark is the functional loss of dopaminergic neurons and the appearance of intracellular amyloid aggregates, constituted mainly by α-Synuclein (aSyn) protein. Although most PD cases are sporadic, mutations are known and usually correlated with early onset. We studied metabolic changes and neuronal differentiation of induced Pluripotent Stem-like cells (iPSCs) that were derived from patients with a triplication of the SNCA gene (SNCAx3) and age-matched healthy controls under normal and environmentally stressed conditions to model in vitro gene-environment interactions which may play a role in the initiation and progression of PD.

The iPSCs lines were initially committed to a neuronal linage, where SNCAx3 cells showed impaired viability, energetic metabolism and stress resistance to starvation and toxicants. A two-steps differentiation protocol was then employed to obtain neurons. SNCAx3 cells exhibited a delayed and decreased capacity to differentiate into neurons. Differentiated SNCAx3 cells showed decreased neurite outgrowth and lower electrophysiological activity. Knockdown by shRNAi against aSyn systematically and significantly ameliorated SNCAx3 defects.

Results suggest a two-fold aSyn overexpression is sufficient to set the stage for decreased developmental fitness, accelerated aging, impaired neuronal differentiation and increased neuronal cell loss.

SPEAKER: GUSTAVO TISCORNIA[1-2], DINO MATIAS[1], FABIO MONTEIRO[1]

[1]CENTER FOR BIOMEDICAL RESEARCH/DCBM, U. ALGARVE, PORTUGAL; [2]CLÍNICA EUGIN, BARCELONA, SPAIN

EXPLORING NEURONOPATHIC GAUCHER'S DISEASE THROUGH INDUCED PLURIPOTENT STEM CELL MODELING

Gaucher's Disease (GD) is a recessively inherited lysosomal storage disorder caused by mutations in the enzyme acid -glucocerebrosidase (GBA). Mutations cause miss-folding of the enzyme, leading to multiple cellular effects and ultimately decreased GBA activity in lysosomes. Disruption of the glucolipid pathway affected results in accumulation of the GBA substrate (glucocerebroside) in lysosomes, leading to altered lysosomal function and systemic effects, including a particular form of neuronopathic Gaucher Disease which presents an early onset neural degeneration leading to death during early childhood. We have derived and are characterizing induced pluripotent stem cell (iPSc) lines of several genotypes (L444P/G202R, L444P/L444P, L444P/P415R, G325R/C342G) in order to model of the neuropathic form of Gaucher's Disease. Our Gaucher iPSc are fully pluripotent, differentiate into the three germ layers, form teratomas, have a normal karyotype and show the same mutations and low GBA activity as the original fibroblasts they were derived from. We are using Gaucher iPSc derived neurons to gain insight into the mechanism of the disease, with particular interest in the recently established connection between Gaucher's disease and Parkinson's disease, and as a platform to test chemical compounds capable of increasing GBA activity. Overexpression of wt GBA in GD neurons does not decrease alpha-synuclein, supporting a gain of function mechanism of the GBA mutation.

OCTOBER 18TH

SY7. IBRO ALUMNI SYMPOSIUM: BASIC AND TRANSLATIONAL RESEARCH IN NEURODEGENERATIVE DISEASE: FROM MOLECULES TO ANIMAL MODELS

ROOM A - 11:00-13:00

CHAIR: VALERIA DELLA MAGGIORE (ARGENTINA)

This symposium reunites five speakers that specialize on the pathophysiology of neurodegenerative disorders including Alzheimer's, Parkinson's and Prion diseases. The talks will discuss state-of-the-art work based on structural and molecular biology, neurogenetics, cellular, and transgenic mouse models, aimed at elucidating the etiology of these disorders and devising potential therapeutic strategies.

SPEAKER: LIONEL MULLER IGAZ, *IFIBIO HOUSSAY (CONICET), UNIVERSITY OF BUENOS AIRES SCHOOL OF MEDICINE, ARGENTINA*

CONDITIONAL MOUSE MODELS OF TDP-43 PROTEINOPATHIES

TDP-43 mislocalization and aggregation are hallmark features of amyotrophic lateral sclerosis and frontotemporal dementia (FTD). We have previously shown in mice that inducible overexpression of a cytoplasmically-localized form of TDP-43 (TDP-43-dNLS) in forebrain neurons evokes neuropathological changes that recapitulate several features of TDP-43 proteinopathies. In the present study, we performed a battery of behavioral tests to evaluate motor, cognitive and social phenotypes in this model. We found that transgene (Tg) induction by doxycycline removal at weaning led to motor abnormalities including hyperlocomotion,

increased spasticity and impaired coordination and balance. Cognitive assessment demonstrated impaired recognition and spatial memory. Remarkably, TDP-43-dNLS mice displayed deficits in social behavior, mimicking a key aspect of FTD. In order to analyze if these symptoms were reversible, we suppressed Tg expression for 14 d in young mice, which showed an established behavioral phenotype but modest neurodegeneration, and found that motor and cognitive deficits were ameliorated; however, social performance remained altered. In older mice exhibiting overt neurodegeneration, the motoric phenotypes were not reversible. These results indicate that TDP-43-dNLS mice display several core behavioral features of FTD with motor neuron disease and might serve as a valuable tool to unveil the underlying mechanisms of this and other TDP-43 proteinopathies.

SPEAKER: HELENA CIMAROSTI[1], ANA CRISTINA GUERRA DE SOUZA[1-2], JEREMY HENLEY[2]

[1]DEPARTMENT OF PHARMACOLOGY, FEDERAL UNIVERSITY OF SANTA CATARINA, BRAZIL; [2]SCHOOL OF BIOCHEMISTRY, UNIVERSITY OF BRISTOL, UK

PROTEIN SUMOYLATION IN ALZHEIMER'S DISEASE

Alzheimer's disease (AD) is the most common cause of chronic dementia among the elderly, with an estimated ~40 million patients diagnosed worldwide, a number predicted to almost double every 20 years. Therefore, the mechanisms underlying neuronal death in AD are the focus of intense research.

SUMOylation acts as a biochemical switch in many pathways, regulating the function of several proteins, and is thus crucial in all eukaryotic cells. It has emerged recently that SUMOylation is involved in neuronal signalling cascades and is implicated in many neurodegenerative diseases, including AD.

We are currently investigating the effects of manipulating SUMOylation and deSUMOylation pathways in cultured neurons and animal models of AD. In particular, we are focusing on the role of potential SUMO targets relevant to mitochondrial dysfunction and neuronal death, e.g. dynamin-related protein 1 and voltage-gated calcium channels. This work will reveal if SUMOylation represents a potentially tractable target for therapeutic intervention and may also identify novel SUMO substrates for drug development.

Funding: IBRO Return Home, ISN-CAEN Return Home and Newton Advanced Fellowships

SPEAKER: GLAUCIA HAJJ[1], GIOVANNA BRITO[1], FERNANDA LUPINACCI[1], FLAVIO BERALDO[2], TIAGO SANTOS[1], MARTIN ROFFE[1], VILMA MARTINS[1]

[1]AC CAMARGO CANCER CENTER; [2]ROBARTS RESEARCH CENTER, BRASIL

CELLULAR PRION IS A RESISTANCE FACTOR FOR THE DEVELOPMENT OF TYPE 2 DIABETES

Prion protein (PrPC) was initially described for its involvement in Transmissible Spongiform Enchephalopathies. Later on, PrPC was demonstrated to be a cell surface molecule involved in many physiological processes, such as lipid raft organization and vesicle trafficking. Given this range of effects and its possible proximity of the insulin receptor in lipid rafts, we decided to analyze PrPc influence on insulin response. Herein we describe that PrPC KO animals present symptoms associated to the development of type 2 diabetes (T2D): hyperglycemia, hyperinsulinemia and obesity; upon a high fat diet. Conversely, animals that overexpress PrPC (TG20) have increased resistance to develop T2D. Primary cultured PrPC KO fibroblasts presented reduced glucose uptake in response to insulin and TG20 fibroblasts an increased glucose uptake. The modulation of glucose uptake is due to a difference in the translocation of the glucose transporter Glut4 to the membrane upon insulin stimulation. PrPc KO cells display reduced Glut4 translocation while TG20 cells presented increased translocation when compared to wild-type cells. Thus, our results indicate that PrPc could be a susceptibility factor for the development of T2D and metabolic syndrome. Strikingly, in cellular models of prion diseases, in which PrPc is converted to its factious form, there is also an impairment of glucose uptake, thus adding new evidence to the possible mechanisms of this disease.

SPEAKER: TOMAS FALZONE, *INSTITUTO DE BIOLOGIA CELULAR Y NEUROCIENCIAS (IBCN); INSTITUTO DE BIOLOGIA Y MEDICINA EXPERIMENTAL (IBYME); FACULTAD DE MEDICINA, UNIVERSIDAD DE BUENOS AIRES (UBA), ARGENTINA*

ASYNUCLEIN IN THE WAY OF MITOCHONDRIAL TRANSPORT AND MORPHOLOGY: DISRUPTED MITOCHONDRIAL HOMEOSTASIS IN HUMAN-DERIVED NEURONS WITH PARKINSON'S DISEASE MUTATIONS

Parkinson's Disease (PD) is characterized pathologically by a progressive loss of neurons and the accumulation of eosinophilic intracellular inclusions, termed Lewy bodies. αsynuclein (αSyn) was the first protein identified with dominant inheritance in familial PD (fPD). Later, many genes contributing to fPD have been identified from which Pink1, Parkin, DJ1 and VPS35 have a direct role in controlling mitochondria, suggesting a mayor role of this pathway in disease. Although genetic mutations account for a small proportion of PD cases; there are pathological, pharmacological and genetic evidence supporting a common sporadic form of disease (sPD) involving defects in neuronal mitochondrial homeostasis, although, the mechanism by which αSyn impairs mitochondrial function remains unknown. To test the αSyn role in the mitochondrial associated pathologies we generated human models with αSyn overexpression to study the axonal mitochondrial transport and morphology in human neurons derived from hESC or modified hiPSC. We provide novel evidence of a differential effect of αSyn mutations in a common pathological pathway involving the control of mitochondrial fragmentation in human neurons. Moreover, by genome edition we uncover a new physiological role for αSyn in the neuronal maintenance of mitochondrial size and distribution in axons. This knowledge provides an important contribution to the role that αSyn induce in early neuropathology and highlight a therapeutic strategy for PD.

SPEAKER: ELENA AVALE, *INGEBI-CONICET. BUENOS AIRES, ARGENTINA*

PHENOTYPIC RESCUE IN A MOUSE MODEL OF TAUOPATHY USING TRANS-SPLICING RNA REPROGRAMMING

Tauopathies are neurodegenerative diseases characterized by the presence of neuronal aggregates of the protein tau in insoluble neurofibrillary tangles. Tau is a microtubule-

associated protein, predominant in axons, which participates in microtubule dynamics and transport. Alternative splicing of exon 10 (E10) in the Tau transcript produces protein isoforms with three (3R) or four (4R) microtubule binding repeats, expressed in equal amounts in the normal adult human brain. Several tauopathies are associated with mutations affecting E10 alternative splicing, leading to an imbalance between 3R/4R isoforms concomitant with the neurodegenerative process. We developed an RNA reprogramming strategy to modulate Tau isoforms in vivo and tested it in a mouse model of tauopathy (hTau). Htau mice produce an excess of 3R Tau, displaying insoluble Tau accumulation in cortical areas and cognitive impairment from 9 months old. Tau 3R/4R balance was restored in the prefrontal cortex of adult hTau mice, inducing a trans-splicing reaction between the endogenous Tau transcript and exogenous RNA pre-trans-splicing molecule (PTM), locally delivered into the brain by lentiviral vectors. Rescued mice showed a reduction of insoluble Tau in the cortex, with a significant functional recovery, evidenced by biochemical, electrophysiological and behavioural analyses. Our results indicate that restoring Tau isoforms balance prevents tauopathy, rising new perspectives for future therapeutic interventions.

SY8. THE CONSEQUENCES OF MEMORY RETRIEVAL: RECONSOLIDATION, EXTINCTION OR NOTHING AT ALL

ROOM B - 11:00-13:00

CHAIR: PEDRO BEKINSCHTEIN (ARGENTINA)

During the past 15 years, the memory research field has increased interest in examining the consequences of retrieving a memory. The finding that inhibition of protein synthesis after retrieval was able to impair the original memory led to the construction of the destabilization-reconsolidation theory. There is accumulating evidence that, under certain conditions, retrieval can result in memory reconsolidation. However, for an associative memory, retrieval can also engage extinction of the original association. Recently, a handful of studies have started to identify the boundaries between reconsolidation and extinction with quite surprising results. In addition, there is also new evidence that indicates that memory expression might not be a required condition for reconsolidation to occur. In this symposium, we will discuss these subjects with pioneering scientists that have actively tried to identified the system, cellular and molecular establishment of the boundaries between reconsolidation and extinction and the relationship between retrieval, reconsolidation and extinction.

SPEAKER: EMILIANO MERLO, *DEPARTMENT OF PSYCHOLOGY, UNIVERSITY OF CAMBRIDGE, AND BCNI. UK*

RETRIEVAL-INDUCED PLASTICITY: RECONSOLIDATION, EXTINCTION AND NO MAN'S LAND

Fully consolidated memories can last for up to the entire animal's life, but they are not immutable. Memory persistence is critically influenced by retrieval episodes. In fear conditioned rats, a single presentation of the conditioned stimulus (CS) induces memory reconsolidation and fear memory persistence, while repeated CS presentations result in loss of fear through extinction. These two opposite behavioural outcomes are operationally linked by the number of cue presentations at memory retrieval, but the behavioural properties and mechanistic determinants of the transition are not known. In this talk I will present behavioural and molecular biological evidence supporting a three phase transition between reconsolidation and extinction critically controlled by the number of CS presentations. Reconsolidation and extinction are mutually exclusive processes, separated by an insensitive or 'limbo' state where neither of them is engaged.

SPEAKER: NOELIA WEISSTAUB[1], FACUNDO MORICI[1], FRANCISCO GALLO[1], MAGDALENA MIRANDA[1], BELEN ZANONI SAAD[1], PEDRO BEKINSCHTEIN[1]

[1]*IFIBIO UBA-CONICET, ARGENTINA*

CORTICAL SEROTONIN CONTROLS RETRIEVAL AND RECONSOLIDATION OF RECOGNITION MEMORY

Episodic memories contain information about our personal experiences. But memories would be useless if we could not retrieve them. Memory retrieval requires the correct selection of a particular trace to be expressed. However, many memories share cues, so how does the brain control interference between similar memories during retrieval? A system including the medial Prefrontal Cortex (mPFC) has been proposed to mediate response selection and control interference.

Serotonin is an important modulator of mPFC function, however it is not clear the role that this system in general and the serotonin 2a receptors (5-HT2aR) in particular play in memory interference processes. We employed the object-in-context (OIC) task, a recognition memory paradigm in rats to answer this question. We found that infusion of MDL 11,939, a 5-HT2aR specific antagonist, in the mPFC before retrieval affects its ability to control memory interference during the OIC task. Modulation of mPFC activity by 5-HT2aR also regulates the reconsolidation of the memory traces. Infusion of a protein synthesis inhibitor like emetine in the PRH after the retrieval blocked reconsolidation of only one of the object memories. However, infusion of 5-HT2aR antagonist in mPFC before the retrieval make both memory traces susceptible to emetine. These results suggest that 5-HT2a receptors in mPFC control memory reactivation allowing the expression and reconsolidation of the most relevant memory trace in the PRH.

SPEAKER: VERONICA DE LA FUENTE, *DFBMC-FCEN-UBA / IFIBYNE-UBA-CONICET, ARGENTINA*

WHAT CAN SMALL-ANIMAL POSITRON EMISSION TOMOGRAPHY TELL US ABOUT MEMORY EXPRESSION, LABILIZATION AND RECONSOLIDATION?

Common techniques for studying memory have traditionally involved drugs affecting global processes. In the past 15 years, drugs affecting more specific cellular mechanisms have been incorporated. The same rationale applies for brain areas affected by these drugs, while early studies involved systemic administrations, recent ones are directed to target specific areas or cell types. With the advent of imaging techniques that allowed whole brain studies, memory has

started to be considered in a "brain wide" manner, mostly in humans. However, there were no available techniques that allowed the study of whole brain activity in small animals until few years ago. In this talk I will focus on our approach using small-animal Positron Emission Tomography (PET) to study brain areas involved in labilization / reconsolidation of fear memory using a contextual fear conditioning paradigm in mice. We found differences in glucose consumption mainly in zones comprising the ectorhinal cortex, the temporal association cortex, hippocampus and amygdala in animals that labilized / reconsolidated vs animals that only evoked the memory or animals that did not evoked it at all. Our work opens new insights in the study of brain activity dynamics using a novel technique, which in combination with others like immunofluorescence, chemogenetics and electrophysiology will help to unravel the pending question about circuits involved in the processing of information.

SY9. REGULATION AND FUNCTION OF GAP JUNCTIONS AND HEMICHANNELS IN THE NERVOUS SYSTEM

ROOM C - 11:00-13:00

CHAIR: JUAN CARLOS SAEZ (CHILE) AND ALBERTO PEREDA (USA)

Gap junctions are clusters of intercellular channels widely expressed in the nervous system that are formed by the apposition of two hemichannels. Hemichannels can also function independently providing conduits for the release or uptake of molecules. We will discuss recent progress regarding cellular and molecular mechanisms underlying their function under normal and pathological conditions.

SPEAKER: FANNY MOMBOISSE[1-2], XIMENA BAEZ[1], AGUSTÍN MARTÍNEZ[1], ANA MARÍA CÁRDENAS[1]

[1]CENTRO INTERDISCIPLINARIO DE NEUROCIENCIAS DE VALPARAÍSO,UNIVERSIDAD DE VALPARAÍSO; [2]VIRAL PATHOGENESIS UNIT, DEPARTMENT OF VIROLOGY, INSTITUT PASTEUR, 75015 PARIS, FRANCE

PANNEXIN-1 REGULATES CATECHOLAMINE RELEASE FROM NEUROENDOCRINE CHROMAFFIN CELLS VIA A FUNCTIONAL COUPLING WITH THE ALFA7 NICOTINIC RECEPTOR

Pannexins are glycoproteins that form high conductance channels that amplify ATP release and/or Ca2+ signals. As hormone release from neuroendocrine chromaffin cells is highly regulated by extracellular ATP and intracellular Ca2+, we explored the role of pannexin channels in this process. We found that bovine chromaffin cells express Pannexin-1 (Panx1) at their plasma membrane, and that Panx1 channels participate in the Ca2+ signaling and catecholamine release induced by the activation of nicotinic receptors, but not in those induced by Ca2+ release from intracellular stores or by membrane depolarization with high K+, suggesting a functional coupling between Panx1 channels and nicotinic receptors. In this regard, we observed by dye uptake assay that choline, an agonist of alpha7 nicotinic receptors, promotes the opening of Panx1 channels, whereas alpha7 nicotinic receptor antagonists inhibit Panx1 channel opening. Also, the dye uptake induced by nicotinic agonists depends on the extracellular Ca2+ and is completed abolished by intracellular BAPTA, but not by EGTA. We propose a new partnership involving Panx1 and alpha 7 nicotinic receptors, in which the activation of alfa7 nicotinic receptors leads to Ca2+ microdomains formation that allow Panx1 channel opening and thus contributing to the catecholamine release.

Supported by grants P09-022-F from ICM-ECONOMIA, Chile.

SPEAKER: SEBASTIAN CURTI, *FACULTAD DE MEDICINA, UNIVERSIDAD DE LA REPÚBLICA, MONTEVIDEO, URUGUAY*

FUNCTIONAL INTERACTION BETWEEN VOLTAGE GATED CHANNELS AND GAP JUNCTIONS IN THE MAMMALIAN BRAIN

Gap junctions mediate electrical transmission between neurons which endows neural networks with a variety of relevant properties. In the mesencephalic trigeminal (MesV) nucleus of the rat we found that the passive properties of these cells in conjunction with the A-type K+ current and the persistent Na+ current support frequency selectivity of transmission instead of the classical lowpass filter properties. By tuning electrical synapses for the transmission of signals like subthreshold oscillations and spikes this property strongly promotes the synchronic activation of pairs of coupled neurons. Moreover, electrical synapses also support coincidence detection, which enables neurons to selectively respond to temporally correlated inputs as opposed to asynchronous depolarizations. Coincidence detection allows neural circuit to better represent temporal information and it also might operate as a noise reduction mechanism. Remarkably, electrophysiological experiments and computer simulations show that modulation of the H-current by cGMP produces an increase in MesV neurons excitability and a dramatic enhancement of coincidence detection. These results reinforces the notion that electrical transmission is strongly shaped by voltage gated conductances and modulation of these conductances might induce significant changes on the efficacy of this modality of synaptic transmission.

SPEAKER: AGUSTIN MARTINEZ[1-2], ISAAC GARCIA[1], AMAURY PUPO[1], BERNARDO PINTO[1], COSCAR JARA[1], JAIME MARIPILLAN[1], CARLOS GONZALEZ[1]

[1]CENTRO INTERDISCIPLINARIO DE NEUROCIENCIA DE VALPARAÍSO; [2]INSTITUTO DE NEUROCIENCIA, FACULTAD DE CIENCIAS, UNIVERSIDAD DE VALPARAÍSO, VALPARAÍSO, CHILE

CONNEXINOPATHIES: A FUNCTIONAL AND STRUCTURAL GLIMPSE

Connexinopathies are genetic diseases caused by mutations in connexin (Cx) genes, like nonsyndromic or syndromic deafness (Cx26, Cx30), Charcot Marie Tooth disease (Cx32), occulodentodigital dysplasia and cardiopathies (Cx43), and cataracts (Cx46, Cx50). Based in functional and structural studies made by us and other groups, we looked for similarities and differences between Cxs regarding the positions of mutations associated to the respective diseases and its functional consequences on gap junction channels (GJCs) and hemichannels (HCs). To know the location of mutations, we produced several molecular models for different Cxs by homology modeling, taking the crystal structure of Cx26 GJC as template. After this analysis we can conclude the following: 1.- Independent of the disease and Cx, all mutations generate partial or total loss of function of the GJCs, with not clear correlation between

the severity of disease and the level of GJCs loss of function. 2.- Mutations generating loss of GJCs function have no clear pattern of clustering at any structural domain, suggesting that GJC functionality is very sensitive to minor changes in Cxs protein. 3.- Syndromic deafness mutations of Cx26 produce gain of function HCs. All mutations eliciting gain of HCs function are clustered in the pore-associated domains, which are critical regions for gating and regulation.
Supported by FONDECYT 1130855 (A.D.M) and FONDECYT 3150634 (I.G.). CINV (P09-022-F).

SPEAKER: MARIA GARCIA-ROBLES[1], JUAN ORELLANA[2], ROBERTO ELIZONDO[1], JUAN CARLOS SAÉZ[2]
[1]UNIVERSITY OF CONCEPCION; [2]PONTIFICIA UNIVERSIDAD CATÓLICA DE CHILE

GLUCOSE INCREASES OPENING OF HEMICHANNELS THROUGH A GLYCOLYTIC-DEPENDENT MECHANISM

Tanycytes are specialized ependymal cells that interact with hypothalamic neurons of the arcuate nucleus; they express glucosensing proteins, including glucose transporter 2, glucokinase (GK) and ATP-sensitive K+ (KATP) channels, indicating their involvement in hypothalamic glucosensing. Additionally, both intracellular GK localization and its activity are modulated by GK regulatory protein in tanycytes (GKRP). Here, we examined whether extracellular glucose modulates the intracellular free Ca2+ concentration (Ca2+ signal) in cultured tanycytes. Fura-2AM time-lapse fluorescence images revealed that glucose increases the intracellular Ca2+ signal in a concentration-dependent manner. Glucose transportation, primarily via glucose transporters, and metabolism via anaerobic glycolysis increased connexin43 (Cx43) hemichannel activity, evaluated by ethidium uptake, through a KATP channel-dependent pathway. Glucose metabolism through GK limited this process since the adenovirus-mediated GKRP overexpression decreased hemichannel activity promoted by glucose. Accordingly, ATP export into the extracellular medium increased, resulting in the activation of purinergic P2Y1 receptors followed by inositol triphosphate receptor activation and Ca2+ release from intracellular stores. The present study establishes that in tanycytes Cx43 hemichannels can be rapidly activated under physiological conditions by the sequential activation and redistribution of proteins involving in glucosensing.

SY10. NEUROMATHEMATICS

ROOM D - 11:00-13:00

CHAIR: ANTONIO C. ROQUE (BRAZIL)

Recent advances in the Neurosciences make evident the need for the development of new mathematical objects and theories to accommodate the vast amount of data and build bridges across the different scales, from the cellular to the systems level. This symposium will put together top researchers working on present day frontiers in the interface between mathematics and neuroscience, and will provide an overview on recent developments in the field.

SPEAKER: ARIEL HAIMOVICI[1], MATTEO MARSILI[2]
[1]INTERNATIONAL CENTRE FOR THEORETICAL PHYSICS, TRIESTE, ITALY; [2]DTO. DE FÍSICA, FCEYN, UNIVERSIDAD DE BUENOS AIRES, ARGENTINA

OPTIMAL SAMPLING IN COMPLEX SYSTEMS

The study of complex systems, in particular the brain, involves the analysis and modeling of non trivial interactions between many degrees of freedom. Despite the advances in technology providing large data sets, the high dimensionality of the system implies that the phase space of states is usually strongly under sampled. Therefore the need to find reduced representations of the data via methods such as clustering or selection of variables. I will discuss a measure of information content in the data to be used as a guiding principle for dimensionality reduction schemes.

SPEAKER: DANIEL FRAIMAN, *UNIVERSIDAD DE SAN ANDRÉS; CONICET, ARGENTINA*

STATISTICS OF BRAIN NETWORKS

The study of random graphs and networks had an explosive development in the last couple of decades. Meanwhile, techniques for the statistical analysis of these networks were less developed. In this talk we will focus on brain networks and will study some statistical problems in a nonparametric framework.

We will address the following questions: Given one or more samples of brain networks, How to calculate a representative brain network? How to define a notion of variability for networks? How to identify a network outlier? How to test if the groups of networks have the same probability law? How to perform classification?

Answers to these questions provide an important step in the development of potential neuroimaging-based tools for diagnosis.

ANTONIO ROQUE[1], LUDMILA BROCHINI[1], NILTON KAMIJI[1], ARIADNE COSTA[2], RENAN SHIMOURA[1], VINÍCIUS CORDEIRO[1], MIGUEL ABADI[1], OSAME KINOUCHI[1], JORGE STOLFI [2]
[1]UNIVERSIDADE DE SAO PAULO; [2]UNIVERSIDADE ESTADUAL DE CAMPINAS, BRASIL

A STOCHASTIC CORTICAL NEURAL NETWORK MODEL

Experimental evidence suggest that neurons and neural circuits display stochastic variability. Recently, Galves and Löcherbach (1) introduced a leaky stochastic spiking neuron model in which the firing of a neuron is a random event with probability given by a monotonically increasing function of its membrane potential. This talk will present some recent analytical and simulation studies on the behavior of networks of Galves-Löcherbach neurons. Analytical results of a simple mean-field version of the network model show that it displays a variety of stationary regimes with continuous and discontinuous phase transitions depending on parameters of the firing function. Simulations of a layered model of the local cortical network with excitatory and inhibitory versions of the stochastic model display asynchronous and irregular activity with low firing rates comparable to deterministic models and experimental data. These results suggest that the Galves-Löcherbach model can be a useful model for studies of networks of spiking neurons because it enables exact analytical results and simple computer simulations.

(1) Galves, A., Löcherbach, E. (2013). Infinite systems of interacting chains with memory of variable length: a stochastic model for biological neural nets. J. Stat. Phys. 151, 896-921.

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SPEAKER: ALINE DUARTE[1], RICARDO FRAIMAN[2], ANTONIO GALVES[1], GUILHERME OST[1], CLAUDIA D. VARGAS[3]

[1]INSTITUTO DE MATEMÁTICA E ESTATISTICA DA UNIVERSIDADE DE SÃO PAULO, SAO PAULO, BRAZIL; [2]CENTRO DE MATEMATICA DA UNIVERSIDADE DE LA REPÚBICA, MONTEVIDEO, URUGUAY; [3]INSTITUTO DE BIOFÍSICA CARLOS CHAGAS FILHO, UNIVERSIDADE FEDERAL DO RIO DE JANEIRO, BRASIL

RETRIEVING CONTEXT TREES FROM EEG DATA

In the current presentation I will outline how we are using optogenetics to address long standing questions concerning the physiology of serotonergic synaptic transmission in the brain. I will present the results of optogenetic experiments that are allowing us to dissect the synaptic basis underlying serotonergic "autoinhibition" in the Dorsal Raphe nucleus, the ability of serotonin neurons to regulate their own excitability through serotonin release. One important reason why we want to study serotonergic synaptic transmission is because many antidepressants are thought to exert their therapeutic effects by regulating the synaptic effects of serotonin. Therefore in the second part of the talk I will then present results of experiments aimed at elucidating how serotonergic synaptic transmission is regulated by chronic administration of fluoxetine and other antidepressants.

SY11. AUDITORY PROCESSING: FROM THE COCHLEA TO THE CORTEX AND BACK

ROOM A - 15:00-17:00

CHAIR: ELEONORA KATZ + MARÍA EUGENIA GOMEZ CASATI (ARGENTINA)

This symposium will provide an updated view of some key points in auditory processing. Namely, sound encoding at the cochlear hair cell-afferent neuron synapse; the synaptic organization of midbrain nuclei where auditory signals are further processed before reaching the cortex and, back to the cochlea with cortico-olivocochlear fibers that modulate the gain of the system by an inhibitory synapse.

SPEAKER: PAUL FUCHS[1], STEPHEN ZACHARY[1]

[1]JOHNS HOPKINS UNIVERSITY SCHOOL OF MEDICINE, USA

EFFERENT INHIBITION OF THE COCHLEA

The mammalian cochlea is subject to feedback (centrifugal) control by central cholinergic neurons that are driven by sensory input. Smaller, unmyelinated lateral olivocochlear (LOC) axons project from the lateral brainstem olivary complex to form synapses with the peripheral dendrites of type I cochlear afferents beneath inner hair cells. Larger caliber, myelinated (MOC) axons from the medial olivary complex form large contacts on outer hair cells in the mature cochlea. Prior to the onset of hearing (~P12 in rodents), the MOCs make transient connections with inner hair cells.

LOCs onto type I afferent dendrites are predominantly cholinergic, but other neurotransmitters also may participate. Both the transient and mature MOC synapses release ACh that binds to a9/a10-containing receptors on hair cells. Calcium through the AChR activates calcium-dependent (SK) potassium channels. Thus hair cells are inhibited (hyperpolarized and shunted) by MOC activity to suppress transmitter release, and strongly reduce outer hair cell electromotility that supports cochlear amplification.

A near-membrane postsynaptic cistern aligns with efferent contacts on hair cells. This cistern may serve as calcium store, segregating synaptic calcium signals at adjoining efferent and afferent contacts. Synaptic calcium 'crosstalk' remains an issue of ongoing study, presumed to mediate developmental and age-related changes in the efferent and afferent innervation of cochlear hair cells.

SPEAKER: PAUL H. DELANO[1,2,3]

[1]FISIOLOGÍA Y BIOFÍSICA, ICBM, FACULTAD DE MEDICINA, UNIVERSIDAD DE CHILE; [2]DEPARTAMENTO DE OTORRINOLARINGOLOGÍA, HOSPITAL CLÍNICO DE LA UNIVERSIDAD DE CHILE; [3]AUDITORY AND COGNITION CENTER (AUCO)

MODULATION OF COCHLEAR SENSITIVITY DURING SELECTIVE ATTENTION: A POSSIBLE FUNCTION OF CORTICO-OLIVOCOCHLEAR PATHWAYS

The auditory efferent system comprises descending projections from the auditory cortex to the medial geniculate body, inferior colliculus, cochlear nucleus and superior olivary complex that form a neural network with multiple feedback loops. Top-down attentional filtering of peripheral auditory responses by higher structures of the brain has been proposed as one of the functions of the auditory efferent system. In this talk, electrophysiological evidence showing a reduction of cochlear sensitivity during selective visual attention in chinchillas and behavioral evidence of altered attention in alpha-9 nicotinic receptor knock-out (KO) mice will be presented. In addition, the corticofugal effects of auditory-cortex inactivation with lidocaine and cryoloops in animal models and the consequences of auditory-cortex electrical microstimulation on the strength of the olivocochlear reflex will be shown. Data will be framed in a network model including the different neural loops of the auditory efferent system from auditory cortex to the cochlear receptor. Finally, the next steps to unravel the role of the cortico-olivocochlear network in selective attention paradigms, including animal models and human experiments will be discussed.

Funded by CONICYT, PIA, Proyecto Anillo ACT1403, FONDECYT 1161155, REDES 150134, and Fundación Guillermo Puelma.

SPEAKER: JUAN GOUTMAN, *INGEBI, ARGENTINA*

SYNAPTIC MECHANISMS OF SOUND ENCODING IN THE MAMMALIAN INNER EA

Inner hair cell (IHC) are specialized sensory cells responsible for converting sounds into synaptic signals. Auditory nerve neurons receive input form this synapse, representing the output of the cochlea. As in other sensory modalities, persistent acoustic stimuli produce adaptation, observed as a reduction in spike rate at the auditory nerve. This phenomenon would contribute to setting the dynamic range for sensing varying sound intensities. We evaluated the hypothesis that the IHC-auditory nerve neurons synapse was responsible for adaptation, by performing simultaneous recordings from these two cell types.

Upon IHC depolarization, we observed an initial increase,

followed by a fast decay in synaptic responses, closely resembling adaptation in the auditory nerve. Interestingly, this phenomenon was invariant with presynaptic stimulation strength. Also, decay kinetics did not change even though the response amplitude varied ~5-fold. The application of a second depolarizing step shortly after adaptation occurred, showed an additional burst of activity, indicating that vesicles remained. The role of postsynaptic receptors desensitization was evaluated by applying similar protocols in the presence of cyclothiazide (CTZ). In CTZ, adaptation occurred with a ~5-fold slower decay time, but differences emerged when comparing IHC stimulation strengths. Both pre-(vesicle depletion) and post-synaptic (desensitization) mechanisms would contribute to synaptic adaptation at IHC synapses.

SPEAKER: KARL KANDLER, *UNIVERSITY OF PITTSBURGH SCHOOL OF MEDICINE, USA*

REORGANIZATION OF LOCAL SYNAPTIC CONNECTIONS IN THE AUDITORY MIDBRAIN DURING DEVELOPMENT AND DISEASE

The inferior colliculus (IC) in the mammalian midbrain is a major subcortical auditory integration center receiving inputs from almost all auditory nuclei. The IC also contains a dense network of local connections, which are thought to provide gain control and contribute to the selectivity for complex acoustic features. To better understand the development, functional organization, and plasticity of local IC connections we used laser-scanning photostimulation with caged glutamate to characterize the spatial distribution and strength of synaptic excitatory and inhibitory input maps to neurons in the central nucleus of the IC in mice.

We found that intrinsic networks are already present at birth. At this age, excitatory and inhibitory input maps largely overlapped with each other and were aligned along the isofrequency axis of the central nucleus of the IC. During development, the size of input maps increased during the first week that was followed by map shrinkage after hearing, which resulted in a predominance of inhibitory inputs maps. Exposure of 3 week-old mice to loud sound (45 min at 16 KHz, 116 dB) lead to a reorganization of local inputs to both excitatory and inhibitory IC neurons. The type of reorganization correlated with the presence or absence deficits in sound gap detection, which often is considered a behavioral sign of tinnitus. This suggest that reorganization of local excitatory and inhibitory IC connection may contribute to the generation of tinnitus.

SY12. NEW CONCEPTS IN OLIGODENDROCYTE FUNCTION IN NEUROLOGICAL DISEASES

ROOM B - 15:00-17:00

CHAIR: BABETTE FUSS (USA)

This symposium will present novel findings related to the role of oligodendrocytes, the myelinating cells of the CNS, as potential targets for the treatment of a variety of neurological diseases. These include the demyelinating disease Multiple Sclerosis and a number of behavioral and neuropsychiatric disorders more recently recognized to involve oligodendrocyte/myelin dysfunction and/or loss.

SPEAKER: CECILIA HEDIN-PEREIRA, *FUNDAÇÃO OSWALDO CRUZ-VPPLR-BRASIL*

MYELIN AND OLIGODENDROGENESIS IN A MOUSE DEPRESSION MODEL

Major depression is the most common neuropsychiatric disorder, but little is known about its pathophysiology. There are various etiologies that may explain the different causes for depression and the deregulation of the hypothalamic-pituitary-adrenal axis has been described as a major trigger for depression. Psychiatric disorders are also associated with changes in white matter, thus suggesting that the oligodendrocytes participate in aspects of these diseases. After demyelinating lesions, subventricular zone (SVZ) generates new oligodendrocyte progenitors which migrate to the injury site and differentiate into mature oligodendrocytes able to remyelinate. The paradigm of depression I will describe in this talk mimics chronic stress in mice by the administration of exogenous corticosterone associated with chronic social isolation. I will discuss our results showing that this protocol of chronic stress induction promotes behavioral and biochemical changes that characterize this as a model for depression. With regard to cellular and molecular changes we demonstrate in this model a dramatic disruption in corpus callosum myelin besides myelinated axon loss. Further, I will discuss the regenerative process for myelin recovery which is perturbed in this animal model suggesting the development of therapies directed to these fundamental components of neural circuits.

SPEAKER: BABETTE FUSS, *VIRGINIA COMMONWEALTH UNIVERSITY, USA*

THE ATX-LPA AXIS AS A REGULATOR OF CNS MYELINATION AND REMYELINATION

Multiple Sclerosis (MS) is the major human demyelinating disease affecting the central nervous system (CNS) and the most common non-traumatic debilitating neurologic disease in young adults. Current therapies for MS are effective in modifying the disease course and in managing symptoms. However, permanent neurologic disability still occurs, and is thought to be caused primarily by the degeneration of chronically demyelinated and hence more vulnerable axons. Thus, promoting remyelination represents one of the critical therapeutic objectives for restoring neurologic function in MS. A promising approach toward the design of a myelin restoring therapy lies in the characterization of molecular signaling axes that can promote the developmental differentiation of the myelinating cells of the CNS, oligodendrocytes, but are misregulated within the MS CNS. In this regard, our recent studies identified the glycoprotein autotaxin (ATX), also known as ENPP2, PD-Iα/ATX or lysoPLD, as an extracellularly located factor that can stimulate oligodendrocyte differentiation via its enzymatic activity generating the lipid signaling molecule lysophosphatidic acid (LPA). In MS, on the other hand, ATX mRNA and protein levels appear reduced within the CNS parenchyma. The ATX-LPA axis, therefore, represents a promising target for stimulating remyelination under pathological conditions.

SPEAKER: JUANA MARIA PASQUINI[1], VICTORIA ROSATO SIRI[1], BRENDA VALEIRAS[1], ANALIA REINES[2], DAVID COTTER[3]

[1]DEPARTMENT OF BIOLOGICAL CHEMISTRY AND IQUIFIB, SCHOOL OF PHARMACY AND BIOCHEMISTRY, UNIVERSIDAD OF BUENOS AIRES, ARGENTINA; [2]DE ROBERTIS INSTITUTE CONICET BUENOS AIRES, ARGENTINA; [3]DEPARTMENT OF PSYCHIATRY, ROYAL COLLEGE OF SURGEONS IN IRELAND, DUBLIN, IRELAND

MYELIN ALTERATIONS AND BEHAVIORAL DISORDERS

The aim of our studies was to determine whether early myelin alterations can impact adult behavior in rats through demyelination and hypomyelination models. On the one hand, rats of either sex were exposed to cuprizone (CPZ) before weaning or after weaning. After treatment, rats were returned to a normal diet until P90, when behavioral studies were performed. On the other hand, rats of either sex were fed an iron deficiency (ID) diet (4mg Fe/kg) from gestational day 5 and until P21 and then returned to a normal diet until P90.Interestingly, CPZ-AW correlated with significant behavioral and neurochemical changes in a gender-dependent manner, altering the number of social activities and the latency to the first social interaction, and highly compromising recognition-related activities in males. These results suggest that the timing of demyelination significantly influences the development of altered behavior, particularly in adult males. Studies on ID rats showed reduced expression of myelin-associated proteins and core metabolic pathways within the frontal cortex. These findings are consistent with changes observed in the schizophrenic brain and provide clues as to how ID may contribute to increased risk of schizophrenia. Behavioral tests were used to explore the relationship between a schizophrenia-like phenotype and the ID model, with results proving social impairment and poor performance during novelty-induced exploration.

SPEAKER: PATRICK LONG[1], MANABU MAKINODAN[2], XIANGYING MENG[3], PATRICK KANOLD[3], GABRIEL CORFAS[1]

[1]KRESGE HEARING RESEARCH INSTITUTE, UNIVERSITY OF MICHIGAN; [2]DEPARTMENT OF PSYCHIATRY, NARA MEDICAL UNIVERSITY SCHOOL OF MEDICINE; [3]DEPARTMENT OF BIOLOGY, UNIVERSITY OF MARYLAND, USA

THE EFFECTS OF EXPERIENCE ON BRAIN MYELINATION: MECHANISMS AND IMPLICATIONS

Since it's initial discovery in the 1800's until recently, myelin was considered a simple insulator for axons, and its formation was believed to be regulated by predetermined biochemical and cellular processes. Both oligodendrocytes and the myelin they generate were considered to be static components of the nervous system. However, recent studies have revealed that oligodendrocyte development and myelination are highly plastic processes that continue throughout adult life, contributing to experience- and activity-dependent plasticity, learning and memory.

I will present recent advances in the understanding of the mechanisms that regulate oligodendrocyte maturation and CNS myelination, and the impact these processes have on adult brain function. Specifically, we have found that the NRG1-ErbB receptor pathway plays a critical role in CNS myelination, promoting oligodendrocyte maturation and myelination. Loss of oligodendrocyte ErbB receptor function results in CNS hypomyelination, behavioral abnormalities and dysfunction in neurotransmitter systems. Furthermore, we also found that juvenile social isolation leads to defects in prefrontal cortex myelin maturation, that this is due to alterations in NRG1-ErbB signaling, and that this hypomyelination is responsible, at least in part, for behavioral abnormalities in adulthood. Together, these studies provide insights into the plasticity of myelin and oligodendrocytes and the importance of myelin plasticity on brain function.

SY13. ISN SYMPOSIUM: NEURAL CONTROL OF APPETITE - FROM GENES TO CIRCUITS AND BEHAVIOR

ROOM C - 15:00-17:00

CHAIR: IVAN E. DE ARAUJO (USA) AND VIVIANA F. BUMASCHNY (ARGENTINA)

The inability to maintain a stable body weight in the presence of abundant calories is a hallmark of mammalian species. This symposium will review novel insights into different neural circuits underlying appetite that are influenced by food reward and body energy stores. Emphasis will be placed on how cutting-edge neurobiological tools may assist in understanding the physiopathology of obesity.

SPEAKER: LUIS TELLEZ, *YALE UNIVERSITY SCHOOL OF MEDICINE; THE JOHN B. PIERCE LABORATORY , USA*

CIRCUIT LOGIC OF FOOD REWARD
[SYMPOSIUM: NEURAL CONTROL OF APPETITE - FROM GENES TO CIRCUITS AND BEHAVIOUR]

While we tend to attribute the rewarding effects of sugars to their sweetness, several lines of evidence indicate that these effects actually mainly arise from the energy sugars provide. Unlike artificial sweeteners, sugar exerts its potent reinforcing effects via both gustatory and postingestive pathways. However, the neural mechanisms mediating sugar's dual control over behaviour remain elusive. This talk will review emerging evidence suggesting that separate basal ganglia neuronal streams mediate the hedonic and nutritional actions of sugar. These findings imply that brain cells within the reward circuitry are primarily sensitive to the energy contents of foods, helping to prioritize energy seeking over hedonic value. These findings provide novel insights into the neural mechanisms underlying perseverant sugar consumption despite widespread availability of low-calorie sweeteners.

SPEAKER: LICIO VELLOSO, *UNIVERSITY OF CAMPINAS, BRASIL*

HYPOTHALAMIC DYSFUNCTION IN OBESITY

Energy homeostasis involves a complex network of hypothalamic and extra hypothalamic neurons that transduce hormonal, nutrient and neuronal signals into responses that ultimately match caloric intake to energy expenditure and thereby promote stability of body fat stores. Growing evidence suggests that rather than reflecting a failure to regulate caloric intake, common forms of obesity involve fundamental changes to this homeostatic system that favor the defense of an elevated level of body adiposity. This talk will review emerging evidence that during high-fat feeding, obesity pathogenesis involves fundamental alteration of hypothalamic systems that regulate food intake and energy expenditure. The changes in hypothalamic systems involve activation of inflammatory signaling, induction of endoplasmic reticulum stress, and

eventually induction of apoptosis of key neurons involved in the control of food intake and energy expenditure.

SPEAKER: DENIS BURDAKOV, *THE FRANCIS CRICK INSTITUTE, UK*
CONTROL OF EATING AND EXPLORATION BY HYPOTHALAMIC CIRCUITS
This lecture will present new data on how molecularly-defined hypothalamic neurons, such as orexin cells, control processes vital for life, such as eating and exploration. In vivo chemogenetic and behavioural evidence will be presented, in conjunction with in vitro optogenetic circuit mapping, to illustrate how wider hypothalamic circuits may coordinate diverse neural signals to ensure effective adaptive behaviour.

SPEAKER: VIVIANA F. BUMASCHNY, *INSTITUTO DE FISIOLOGÍA Y BIOFÍSICA BERNARDO HOUSSAY (IFIBIO, UBA-CONICET), ARGENTINA*
PROOPIOMELANOCORTIN CONTROL OF FOOD INTAKE
The global obesity epidemic has reached over 600 million people. Obesity predisposes to cardiovascular disease and type 2 diabetes mellitus by central and peripheral mechanisms, causing metabolic syndrome, which increases the risk of mortality. Despite great effort is made to develop new therapies, a major difficulty associated to obesity treatments, is that patients initially lose weight but they later experience a rebound. In this talk we will discuss emerging evidence, collected from genetically engineered mice, revealing that the plasticity of energy balance neural circuits is lost in overweighted animals, preventing them to achieve a normal body weight after treatment. We will focus on the subpopulation of hypothalamic GABAergic Proopiomelanocortin (POMC) neurons, which we found that play a critical role in the control of food intake and glucose homeostasis.

SY14. BEHAVIORAL, NEUROCHEMICAL AND MOLECULAR APPROACHES TO STUDY FEAR ANXIETY AND POSTTRAUMATIC STRESS DISORDER

ROOM D - 15:00-17:00
CHAIR: ANDREA MORA GALLEGOS (PUERTO RICO)

Anxiety, fear and post-traumatic stress are among the major disorders of modern humanity. There are different approaches to assess them.
Behavioral, neurochemical and molecular analysis bring information about the related neuronal mechanisms and the brain areas involved in. This Symposium will address different analysis levels to better under stand the above mentioned disorders and how they could influence processes like learning and behavior.

SPEAKER: JOSE RODRIGUEZ-ROMAGUERA[1], HIROSHI NOMURA[1], J. ELLIOTT ROBINSON[1], RANDALL UNG[1], SHANNA RESENDEZ[1], VIJAY MOHAN K NAMBOODIRI[1], JAMES OTIS[1], OKSANA KOSYK[1], GARRET D STUBER[1]
[1]DEPARTMENTS OF PSYCHIATRY, UNIVERSITY OF NORTH CAROLINA, USA
ENCODING AND REGULATION OF ANXIETY STATES BY PNOC EXPRESSING NEURONS WITHIN THE BNST
The choreography of complex emotional states, such as the anxiety of being in and the drive to avoid dangerous contexts, is critical for an organism's survival. Specifically, the bed nucleus of the stria terminalis (BNST) is critical for processing threat-related stimuli. Therefore understanding the precise neural circuits within the BNST that encode and regulate anxiety is crucial. Using a transgenic mouse line that co-expresses Cre in neurons with endogenous Pre-pronociceptin (PNOC) expression, we are able to target and studythis novel population of BNST neurons. We found this population to be a subset of BNST GABAergic neurons that projects both locally and distally to the medial amygdala and the medial preoptic area. Using in vivo calcium imaging in combination with the genetically-encoded calcium indicator GCaMP6s revealed that these neurons are preferentially activated by distinct anxiogenic stimuli (i.e. TMT exposure and open arm exposure in an elevated plus maze). Further experiments using in vivo optogenetics reinforced this notion, as photoactivation of BNST-PNOC neurons increased anxiety in the elevated plus maze (avoidance of the open arms), whereas photoinhibition decreased anxiety. However, photoactivation of BNST-PNOC neurons outside an anxiogenic context did not induce avoidance behavior, as shown in a real-time preference assay. Together, these experiments highlight the specificity of BNST-PNOC neurons in processing threat-related stimuli.

SPEAKER: JAMES PORTER[1], MARANGELIE CRIADO-MARRERO[1], BETHZALY VELAZQUEZ[1], ROBERTO J. MORALES SILVA[2], CÉSAR TORRES[2], RAMÓN MISLA[1], BENJAMÍN LÓPEZ[1]
[1]DEPT OF BASIC SCIENCES, PONCE RESEARCH INSTITUTE, PONCE HEALTH SCIENCES UNIVERSITY; [2]DEPT OF BIOLOGY, UNIVERSITY OF PUERTO RICO-PONCE
FKBP5 IN THE MEDIAL PREFRONTAL CORTEX MODULATES FEAR CONDITIONING AND EXTINCTION
Dysfunction of the hypothalamic-pituitary-adrenal axis and the ensuing impaired response to stress contributes to numerous mental health disorders including posttraumatic stress disorder (PTSD). The protein FKBP5 regulates the activation of the glucocorticoid receptor by decreasing its affinity for glucocorticoids. In soldiers, low FKBP5 mRNA expression in blood cells is associated with an increased risk of worse PTSD symptoms. Additionally, polymorphisms in the FKBP5 gene are associated with increased risk of developing PTSD in adults who were abused as children suggesting that dysfunctional expression of FKBP5 may contribute to PTSD. Although these data suggest that FKBP5 plays a role in PTSD, altered FKBP5 expression in various brain structures could contribute to PTSD by increasing aversive learning and/or impairing fear extinction. Given the prominent effects of stress on the medial prefrontal cortex (mPFC) and the importance of the mPFC in the modulation of fear, we tested whether signaling via FKBP5 in the mPFC modulates aversive learning. In this presentation, I will discuss our recent data showing that fear conditioning and extinction alter FKBP5 expression in the mPFC and that reducing FKBP5 expression in the mPFC modulates both fear conditioning and extinction. Our findings highlight the importance of FKBP5 expression in the mPFC in aversive learning and memory and suggest that dysfunctional expression of FKBP5 in the mPFC could contribute to PTSD.

SPEAKER: ANDREA MORA-GALLEGOS, *NEUROSCIENCE RESEARCH CENTER; UNIVERSITY OF COSTA RICA, COSTA RICA*

HOUSING EFFECTS AND THE REVERSION OF THOSE CONDITIONS ON FEAR CONDITIONING AND ANXIETY

Environmental enrichment (EE) and social isolation (SI), have lasting effects on brain and behavioral parameters related to emotional memory. We focused on differences between EE and SI rats and possible effects produced by the reversion of those conditions on fear, anxiety (Open Field Test-OFT and Plus Maze-PM) and dopamine (DA) on prefrontal cortex (PFC), and amygdala (AMY) with HPLC-EC analysis. In experiment 1, anxiety tests were carried out before, during and after two months of housing conditions. At two months FC procedure was conducted and rats were sacrificed for neurochemical analysis. In experiment 2, housing conditions were reverted (EE to SI and SI to EE), and maintained for two months, keeping their respective control groups. Anxiety tests were done as in experiment 1 and fear to the context (FCC) was conducted at the end of housing. In experiment 1 EE rats were less reactive on anxiety tests and FC. Anxiety effects are reflected in OFT locomotion and exploration and also in PM behaviors. In FC EE rats showed higher freezing as an adaptive defensive behavior. EE rats showed higher DA turnover in PFC and in AMY than SI rats. In experiment 2, consistent results with experiment 1 in OFT and PM were found. SI-EE behaviors were more alike to EE and that EE-SI behaviors were more alike to SI. Reverted conditions do not produced differences in FCC or DA. Our results confirm the positive effects of EE at behavioral and neurochemical levels maintained at 4 months of age.

OCTOBER 19TH

SY15. NEUROPHYSIOLOGY OF TEMPORAL PROCESSING IN THE BRAIN

ROOM A - 11:00-13:00

CHAIR: PATRICIA AGOSTINO (ARGENTINA) AND HUGO MERCHANT (MEXICO)

A fundamental component of cognition is the perception of the passage of time. In particular, temporal processing within the 10-2 to 102 seconds is crucial for many complex behaviors, such as speech comprehension, motor control, and decision-making. The goal for this symposium is to present studies of temporal processing in humans and animals using a diversity of experimental and analytical tools.

SPEAKER: HUGO MERCHANT[1], GERMAN MENDOZA [1], JUAN CARLOS MENDEZ [1]

[1]INSTITUTO DE NEUROBIOLOGÍA, UNAM CAMPUS JURIQUILLA, MEXICO

NEURAL UNDERPINNINGS OF TIME PERCEPTION IN THE PRIMATE: PREFRONTAL AND PREMOTOR SINGLE UNIT ACTIVITY DURING A CATEGORIZATION TASK OF TEMPORAL MAGNITUDES

Categorization in arguably the most common perceptual act and consists in the differential response to object or events that belong to separate classes. In the present study, we investigated the functional properties of neurons in the primate prefrontal and premotor cortex during the categorization of temporal intervals. The results show that both areas encode all the crucial parameters needed for appropriate categorization; namely, the accumulation of temporal information, the linear tuning to elapsed time, the categorical segregation of intervals, and the evaluation of the categorization outcome as a feedback signal to improve the perceptual performance in the psychometric task. These results suggest that the premotor-prefrontal loop plays a fundamental role, not only in the representation of the passage of time, but also in the assignment of categories based on the current rules of a task. Hence, this circuit has the ability to segregate the one-dimensional feature continuum that defines time, into short and long categories based on arbitrary boundaries or prototypes.

SPEAKER: SOFIA SOARES [1], BASSAM ATALLAH [1], THIAGO GOUVEA [1], TIAGO MONTEIRO [1], ASMA MOTIWALA [1], JOSEPH PATON [1]

[1] CHAMPALIMAUD RESEARCH , PORTUGAL

BASAL GANGLIA CONTRIBUTIONS TO A TIME-BASED DECISION

We trained rats and mice to judge whether the duration of time intervals were longer or shorter than 1.5 seconds while recording and manipulating activity of neurons the dorsal striatum and dopamine (DA) neurons in the substantia nigra pars reticulata (SNc). I will describe how elapsed time, the critical decision variable in this task, is encoded by population dynamics of striatal neurons. This representation predicts the duration judgements produced by the animal. Strikingly, cooling striatal tissue led to underestimation of interval duration, suggesting that striatal dynamics underlie animals' timing behavior. But which endogenous mechanisms might cause striatal dynamics to fluctuate? DA neurons in the SNc receive input from and project densely to the dorsal striatum and have been implicated in timing. Using fiber photometry, we found that higher/lower dopaminergic activity within a trial predicted under/overestimation of interval duration. These signals were consistent with the predicted impact of variability in timekeeping on reward prediction error coding by DA neurons. Surprisingly, optogenetic activation caused underestimation of interval duration, indicating that midbrain DA neurons not only reflect variability in timing, but exert control over it. These data suggest that reciprocal interactions between dopamine neurons and striatal networks can cause variability in time estimation, with broad implications for reinforcement based decision-making.

SPEAKER: VICTOR DE LAFUENTE, *UNIVERSIDAD NACIONAL AUTÓNOMA DE MÉXICO, MÉXICO*

TEMPORAL RHYTHM PERCEPTION IN MONKEYS AND HUMANS

Timing is a fundamental variable for behavior. The mechanisms allowing human and non-human primates to synchronize their actions with periodic external events are not yet completely understood. We characterized the ability of rhesus monkeys and humans to perceive and maintain rhythms of different paces in the absence of

sensory cues or motor actions. In our rhythm task subjects had to observe and then internally follow [imagine] a visual stimulus that periodically changed its location along a circular perimeter. Crucially, they had to maintain this visuospatial rhythm in the absence of movements. Our results show that the probability to remain in synchrony with the rhythm decreased and the variability in the timing estimates increased as a function of elapsed time. These trends were well captured by the generalized Weber´s law. Additionally, the pattern of errors show that subjects tended to lag behind fast rhythms and to get ahead of slow ones, suggesting that a mean frequency might be incorporated as prior information. Overall, our results demonstrate that rhythm perception and maintenance is a cognitive ability that we share with rhesus monkeys and that this ability does not depend on overt motor commands.

SPEAKER: RODRIGO LAJE, *UNIVERSITY OF QUILMES; CONICET; ARGENTINA*
TEMPORAL PROCESSING IN THE MILLISECONDS AND SECONDS RANGE
The mechanisms underlying time processing in the brain in the hundred milliseconds and few seconds range are still largely unknown, one possible reason being that there might be different overlapping mechanisms playing all at once. I'll show theoretical and experimental data supporting one of such mechanisms—a population clock where time is intrinsically encoded in the nonstationary spatiotemporal dynamics of a neural network. This framework reconciles divergent experimental observations like the apparently noisy spontaneous cortical activity and the robustness of a trained spatiotemporal pattern, both coexisting within the same neural subpopulation.

SY16. INTEGRATIVE SENSORY MOTOR FUNCTION: FROM MOTOR COMMANDS TO COGNITION

ROOM B - 11:00-13:00
CHAIR: PEDRO MALDONADO (CHILE)

Motor and sensory processes of the brain are not functional separated. Sensory processing is required for proper motor function, and motor commands are also an intrinsic component of sensory processes. In this symposium, we discuss several mechanism that shed light on how the motor and sensory systems interact in complex behaviors that range from an escape response to cognitive abilities.

SPEAKER: MICHEL BORDE [1], VIRGINIA COMAS [1]
[1] DEPTO. DE FISIOLOGÍA, FACULTAD DE MEDICINA, UDELAR, URUGUAY
INCREASE IN SENSORY SAMPLING TRIGGERED BY AN IDENTIFIED MOTOR COMMAND IN A LOWER VERTEBRATE
Despite recent advances that have elucidated the effects of collateral of motor commands on sensory processing structures, the neural mechanisms underlying the modulation of active sensory systems by internal motor-derived signals remains poorly understood. The modulation of the active electrosensory neural system triggered by a motor command described in Gymnotus omarorum, a pulse type weakly electric fish, emerged as a vertebrate model system to analyze such high-level motor-sensory interactions. In this species, discharge of a single action potential at Mauthner cells (MC), a pair of reticulospinal command neurons for escape in teleosts, evokes an abrupt and prolonged increase in the rate of the electric organ discharge (EOD), the output signal of the electrogenic component of the active electrosensory system. Temporal correlation of motor and sensory consequences of MC activation suggests that the neural network responsible for sensory modulation must comply with, at least, two functional requirements: short latency and long duration of the sensory modulation.
Neural strategies that may have evolved in this species to meet those functional requirements were investigated in vivo and in vitro using morphological and electrophysiological techniques.
We provide evidence indicating that the adequate timing and duration of the MC-initiated increase in sensory sampling during escape is achieved thanks to a combination of network, cellular and synaptic specific arrangements.

SPEAKER: AGUSTIN IBANEZ, *INSTITUTO DE NEUROCIENCIA COGNITIVA Y TRASLACIONAL (INCYT, INECO, FAVALORO, CONICET); CENTER FOR SOCIAL AND COGNITIVE NEUROSCIENCE, SCHOOL OF PSYCHOLOGY, UNIVERSIDAD ADOLFO IBANEZ, CHILE*
EARLY DETECTION OF INTENTIONAL HARM IN THE HUMAN AMYGDALA
A decisive element of moral cognition is the detection of harm and its assessment as intentional or unintentional. Moral evaluation engages brain networks supporting mentalizing, intentionality, empathic concern and evaluation. We will present relevant evidence from our laboratory, including behavioral studies of neurodegenerative conditions and psychiatric disorders, intracranial recordings, lesion studies, high-density electroencephalography, neuroimaging, and functional connectivity. These studies converge in 3 issues: 1) intentional harms are process at very early stages by coticolimbic networks in terms of stimulus salience; 2) intentional harms involucrate more broad and high-level regions; 3) impairments in the detection of intentional harms are partially dependent on a broad fronto-insular-temporal network (FITN) responsible for (a) on-the-fly context-based prediction making, (b) coordination of the internal (bodily) and external (task-related) milieus, and (c) consolidation of associations between context and target stimuli. Results support the 'many roads' view of the amygdala and its frontotemporal connections, highlighting its role in the rapid encoding of intention and salience –critical components of mentalizing and moral evaluation. Finally, we identify new challenges for this synergistic framework in order to be applied in psychiatric and neurological translational science.

SPEAKER: PEDRO MALDONADO, *BNI, UNIVERSIDAD DE CHILE, CHILE*
NEURONAL MECHANISMS OF ACTIVE SENSING IN VISION AND TOUCH
Motor and sensory processes of the brain are not functional separated. Sensory processing is required for proper motor function, and motor commands are also an intrinsic component of sensory processes. In this symposium, we discuss several mechanism that shed light on how the motor and sensory systems interact in complex behaviors that range from an escape response to cognitive abilities, and will discuss how these mechanisms are implemented

through long-range synchronization and use in active sensing.

SPEAKER: KATIA-SIMONE ROCHA[1], LUANA DANTAS[1], RAFAELA FAUSTINO[1], SERGIO NEUENSCHWANDER[1]

[1]VISLAB, BRAIN INSTITUTE - UFRN, 59056-450 NATAL, BRAZIL

HOW DO GRATING STIMULI BIAS OUR CONCEPTS ON CORTICAL GAMMA SYNCHRONIZATION? A STUDY IN CAPUCHIN MONKEY V1

Gamma have been implicated in perceptual binding and visual attention. So far, most of the evidence has been derived from analysis of responses to moving gratings. However, a key step for understanding whether gamma contributes to visual processing is to obtain data during free viewing of ecologically meaningful scenes. Recent studies using a more naturalistic approach in the visual cortex led to diverging conclusions. In humans, gamma was absent from ECoG responses to natural images and noise. Similarly, analysis of spiking activity in V1 of capuchin monkeys revealed strong beta but no gamma components in responses to pictures. An analysis of ECoG signals in the macaque showed, on the contrary, surprisingly strong gamma responses to static images. Here we record spiking and local field potential signals from V1 of capuchins in response to gratings and natural stimuli during both maintained fixation and free viewing. Our results show that large gratings capable of activating selectively the cortex induce strong and stable gamma oscillations (from 48 to 63 Hz, over 3 monkeys), confirming previous results in the macaque and humans. In contrast, gamma is absent from free viewing of natural images and movies presented on a monitor screen. Similar results were obtained with real world scenes, such as viewing of other monkeys, humans or real objects. Overall, our findings weaken the notion that gamma is necessary for visual processing and question its role in attention.

SY17. CELLULAR AND MOLECULAR MECHANISMS OF NEURONAL PLASTICITY

ROOM C - 11:00-13:00

CHAIR: FRANCESCO ROSSI AND NATHALIA VITUREIRA (URUGUAY)

The Symposium will focus on different aspect of neuronal plasticity: from learning and memory to pathology, from neuronal to astrocyte signaling, and from molecular mechanisms to behavior. This symposium brings together Latin-American and international researchers in this field to share their outstanding recent findings with the neuroscience community.

SPEAKER: MAURO COSTA-MATTIOLI, *BAYLOR COLLEGE OF MEDICINE, HOUSTON, TEXAS, USA*

MECHANISM IN SYNAPTIC PLASTICITY IN HEALTH AND DISEASE

SPEAKER: URSULA WYNEKEN[1], LUARTE ALEJANDRO[1], GOMEZ CRISTOBAL,[1] JUAN PABLO RAMIREZ[1]

[1]UNIVERSIDAD DE LOS ANDES, CHILE

ASTROCYTE-DERIVED EXOSOMES IN NEURAL PLASTICITY

Small extracellular vesicles (i.e. exosomes) are novel mediators of inter-cellular signaling that influence target cell function by means of their molecular content consisting of proteins, lipids and non-coding RNAs (such as microRNAs (miRNAs)). Their role in communication between cells of the CNS and with the body outside the brain is mostly unexplored. Thus, exosomes from primary astrocyte cultures were isolated by differential centrifugation and characterized by protein markers, size and equilibrium density. These exosomes contained the astrocyte-specific glycolitic enzyme Aldolase C and astrocytes expressing Aldolase C-GFP induced an increase in the content of miR-26a in them. When exosomes were added to neuronal cultures, they were taken up by hippocampal neurons and decreased dendritic length and complexity in a miR-26a-5p dependet manner. Moreover, miR-26a-5p recapitulated the morphological effect of exosomes and this was prevented by the corresponding antago-MiR. To address whether exosomes were released by astrocytes in vivo, Aldolase C-GFP was transferred to forebrain astrocytes by in utero electroporation. The endogenous and recombinant Aldolase C forms could be detected in exosomes obtained from cerebrospinal fluid and blood plasma. These findings confirm for the first time that a proportion of plasma exosomes originate in astrocytes under physiological conditions and open new avenues for the use of brain-derived exosomes as biomarkers in neurological diseases.

SPEAKER: ALVARO ARDILES, *CENTRO INTERDISCIPLINARIO DE NEUROCIENCIA DE VALPARAÍSO, UNIVERSIDAD DE VALPARAÍSO, CHILE; ESCUELA DE MEDICINA, FACULTAD DE MEDICINA, UNIVERSIDAD DE VALPARAÍSO, CHILE*

PANNEXIN 1, A NEW ACTOR IN SYNAPTIC PHYSIOLOGY

Pannexin 1 (Panx1) is a membrane protein that forms non-selective functional single channels, which are expressed in many areas of the brain, especially in pyramidal cells from hippocampus and cerebral cortex where it exhibits a preferential distribution in postsynaptic membranes accumulating in postsynaptic densities. Research based on their postsynaptic localization indicates that Panx1 channels stabilize synaptic plasticity and is needed for learning. In this regard, the absence of Panx1 modifies the threshold for the induction of excitatory synaptic plasticity, facilitating the induction of NMDAR-dependent long-term potentiation (LTP) and precluding the induction of NMDAR-dependent long-term depression (LTD). Accordingly, the loss of Panx1 leads to impaired object recognition memory and spatial memory reversion, consistent with an altered behavioral flexibility. Furthermore, Panx1 deletion induces changes in synaptic morphology and protein composition. Here we show evidences supporting a critical role of Panx1 channels in synaptic physiology.

SPEAKER: ROBERTO DE PASQUALE, *UNIVERSITY OF SÃO PAULO, BRASIL*

SYNAPTIC METAPLASTICITY IN THE VISUAL CORTEX: INTERACTION BETWEEN VISUAL EXPERIENCE AND REACTIVE OXYGEN SPECIES

The idea that the synapse's previous history of activity determines its current plasticity has been defined as

metaplasticity. In the visual cortex, sensory experience and reactive oxygen species (ROS) produce metaplastic changes related to the history of synaptic activity. ROS are highly reactive molecules. In neurons , they are produced by cellular metabolism and in part by sustained synaptic activity. ROS are important for intracellular signalling, given that knock out animals for the ROS producer enzyme NOX2 (gp91phox-/-) do not exhibit long-term potentiation (LTP) and long-term depression (LTD). Normally, deprivation of visual experience favors the induction of LTP and reduces the probability of obtaining LTD. However, in gp91phox-/- animals, dark rearing promotes LTD as the only possible form of plasticity inducible in these animals. These results suggest that the effects of dark exposure depend on the general levels of metabolic and synaptic activity, as dark rearing allows the occurrance of synaptic depression when such activity is downregulated. Moreover, my findings poited out that ROS and visual experience interact to determine metaplastic changes by altering the functionality of the NMDA receptor.

SY18. YOUNG INVESTIGATOR SYMPOSIA I

ROOM A - 16:30-18:30

CHAIR: JAIME FORNAGUERAS (COSTA RICA)

SPEAKER: MICAELA LÓPEZ-LEÓN[1], MARIA FLORENCIA ZAPPA VILLAR[1], MARIANA G. GARCÍA[2], GUSTAVO R. MOREL[1], GUILLERMO MAZZOLINI[2], RODOLFO G. GOYA[1], PAULA C. REGGIANI[1]

[1]INIBIOLP, SCHOOL OF MEDICINE, HISTOLOGY B, UNLP; [2]GENE THERAPY LABORATORY, SCHOOL OF BIOMEDICAL SCIENCES, AUSTRAL UNIVERSITY

RESTORATIVE EFFECTS OF HUMAN MESENCHYMAL STEM CELL THERAPY ON SPATIAL MEMORY IN SENILE RATS

Brain aging is associated with a progressive increase in the incidence of neurodegenerative diseases and deterioration of spatial learning and memory in aging rats and humans. Here, we investigated the therapeutic potential of human adult bone marrow-derived mesenchymal stem cells (BM-MSCs) to treat cognitive impairment in Senile rats (27 months). Female rats were divided into 3 groups (N=8 each): Young-intact (3 months), Senile-Intact and Senile-MSC (intracerebroventricular injected with Dil-labeled human BM-MSCs). Using the Barnes maze we assessed hippocampus-dependent learning and spatial memory before and after cell injection. Additionally, we performed time-course studies for MSCs integration and viability in the brain and assessed a set of hippocampal cell markers.

Human BM-MSC therapy significantly increased goal hole and goal sector exploration activity in senile rats as compared with intact counterparts. Immature neuron number in the hippocampal dentate gyrus (DG) fell sharply in the senile animals as compared with young counterparts and was comparable in the hippocampal DG of both old groups. Time-course studies (24 days) revealed that MSCs integrated into ependymal cell layer and occasionally in the brain parenchyma.

The results suggest that human BM-MSC therapy partially reverses the decline in cognitive performance that occurs in senile rats. We conclude that human BM-MSC are a promising biological tool for the treatment of age-related spatial memory deficits.

SPEAKER: JACQUE PAK KAN IP [1], IKUE NAGAKURA [1], JEREMY PETRAVICZ [1], ERIK A.C. WIEMER [2], MIRGANKA SUR[1]

[1] PICOWER INSTITUTE FOR LEARNING AND MEMORY, MASSACHUSETTS INSTITUTE OF TECHNOLOGY; [2] INSTITUTE OF HEMATOLOGY, ERASMUS UNIVERSITY ROTTERDAM

PROBING SYNAPTIC DEFECTS IN 16P11.2 DELETION SYNDROME IN VIVO

Microdeletion of a region in the chromosome 16p11.2 increases susceptibility to autism. One candidate gene in this microdeletion region is the major vault protein (MVP), which has been implicated in the regulation of several cellular processes including transport mechanisms and multidrug resistance. We found that MVP expression levels in MVP+/- mice closely phenocopy those of 16p11.2 mice, suggesting MVP+/- mice may serve as a model of MVP function in 16p11.2 microdeletion. However, the function of MVP in the central nervous system, in particular its role in brain function and plasticity, has not been investigated. To determine the role of MVP in experience-dependent synaptic and circuit plasticity, we first measured ocular dominance plasticity (ODP) in primary visual cortex (V1). We found that MVP +/- mice showed impairment in strengthening of open eye responses in V1 after 7 days monocular deprivation (MD), resulting in reduced overall plasticity. Furthermore, electrophysiology experiments suggested a decrease in the number of functional synapses. To further investigate the synaptic defects in MVP+/- mice, we employed time-lapse in vivo two-photon microscopy. Collectively, we find a highly specific role for MVP as a critical molecule in the homeostatic component of activity-dependent synaptic plasticity. Thus, this study helps reveal a new mechanism for an autism-related gene in brain function.

SPEAKER: CLAUDIO PEREZ-LEIGHTON[1,2], BEATRIZ ALVAREZ[1], GAC LILY[1], HERNANDEZ MARIA PAZ[3], MORSELLI EUGENIA[3], JENNIFER TESKE [2,4,5,6]

[1]CIMIS, FACULTAD DE MEDICINA, UNIVERSIDAD ANDRES BELLO, SANTIAGO, CHILE; [2]DEPARTMENT OF FOOD SCIENCE AND NUTRITION, UNIVERSITY OF MINNESOTA, MN, USA; [3]FACULTAD DE CIENCIAS BIOLÓGICAS, PONTIFICIA UNIVERSIDAD CATOLICA DE CHILE, SANTIAGO, CHILE; [4]DEPARTMENT OF NUTRITIONAL SCIENCES, UNIVERSITY OF ARIZONA, AZ, USA; [5]MINNEAPOLIS VA HEALTHCARE SYSTEM, MN, USA; [6]MINNESOTA OBESITY CENTER, UNIVERSITY OF MINNESOTA, MN, USA

Regulation of physical activity and food choice in the context of obesity: role of orexins and opioid/non-opioid dynophins peptides

The orexin/dynorphin (ox/dyn) neurons release orexin and dynorphin (DYN), neuropeptides that affect food intake and energy expenditure. Most research has focused on the orexin peptides and less is known about the role of DYN peptides released from these neurons. The hypothalamic paraventricular nucleus (PVN) modulates feeding behavior and physical activity, and our recent work has focused in the role of non-opioid peptide DYN-A2-17 in PVN in mice. Our preliminary data shows that injection of DYN-A2-17 in PVN increases physical activity, energy expenditure and wheel-running activity. Next, we tested whether DYN-A2-17

the opioid DYN peptide DYN-A1-13 and orexin-A modulate hedonic food intake. Mice were acclimated to short-term access (2 h) to four snacks of human consumption and standard rodent chow. After establishing baseline preferences for snacks, mice were injected with each peptide at their different concentrations. Data suggest DYN-A1-13 increases intake of both non-preferred and preferred snacks, DYN-A2-17 increases intake of only the preferred snack while orexin-A increases chow intake. Finally, preliminary data shows that DYN-A2-17 increases intracellular calcium in hypothalamic mice cell line, suggesting it is excitatory. Together, these experiments will improve our understanding of the mechanisms by which the orexin/dynorphin neurons control energy balance.

SPEAKER: JULIA CLARKE[1], NATALIA LYRA E SILVA[1], CLAUDIA FIGUEIREDO[1], WILLIAM L. KLEIN[2], DOUGLAS P. MUNOZ[3], LICIO A. VELLOSO[4], SERGIO T FERREIRA[1], FERNANDA G. DE FELICE[1]

[1]FEDERAL UNIVERSITY OF RIO DE JANEIRO; [2]NORTHWESTERN UNIVERSITY; [3]QUEENS UNIVERSITY ; [4]STATE UNIVERSITY OF CAMPINAS

ALZHEIMER-ASSOCIATED ABETA OLIGOMERS IMPACT THE CENTRAL NERVOUS SYSTEM TO INDUCE PERIPHERAL METABOLIC DEREGULATION

Alzheimer's disease (AD) is associated with peripheral metabolic disorders. Clinical/epidemiological data indicate increased risk of diabetes in AD patients. Here, we show that intracerebroventricular infusion of AD-associated Abeta oligomers (AbOs) in mice triggered peripheral glucose intolerance, a phenomenon further verified in two transgenic mouse models of AD. Systemically injected AbOs failed to induce glucose intolerance, suggesting AβOs target brain regions involved in peripheral metabolic control. Accordingly, we show that AbOs affected hypothalamic neurons in culture, inducing eukaryotic translation initiation factor 2alpha phosphorylation (eIF2a-P). AbOs further induced eIF2a-P and activated pro-inflammatory IKKb/NF-kB signaling in the hypothalamus of mice and macaques. AbOs failed to trigger peripheral glucose intolerance in tumor necrosis factor-TNF-alpha receptor 1 knockout mice. Pharmacological inhibition of brain inflammation and endoplasmic reticulum stress prevented glucose intolerance in mice, indicating that AbOs act via a central route to affect peripheral glucose homeostasis. While the hypothalamus has been largely ignored in the AD field, our findings indicate that AbOs affect this brain region and reveal novel shared molecular mechanisms between hypothalamic dysfunction in metabolic disorders and AD.

SPEAKER: MARTA M. MORAWSKA[1], FABIAN BUECHELE[1], CARLOS G. MOREIRA[1], LUKAS L. IMBACH[1], DANIELA NOAIN[1], CHRISTIAN R. BAUMANN[1]

[1]NEUROLOGY DEPARTMENT, UNIVERSITY HOSPITAL ZURICH, ZURICH, SWITZERLAND

SLEEP MODULATION ALLEVIATES AXONAL DAMAGE AND COGNITIVE DECLINE AFTER RODENT TRAUMATIC BRAIN INJURY

Traumatic brain injury (TBI) is a major cause of death and disability worldwide. It produces diffuse axonal injury (DAI), which contributes to cognitive impairment, but effective disease-modifying treatment strategies are missing. We have recently developed a rat model of closed skull TBI that reproduces human TBI consequences, including DAI and clinical sequelae such as memory impairment. Here, we investigated whether sleep modulation after trauma has an impact on DAI and memory outcome. We assessed cognition with the novel object recognition test and stained for amyloid precursor protein, a DAI marker. We found that both sleep induction and restriction acutely after TBI enhanced encephalographic slow-wave activity, markedly reduced diffuse axonal damage in the cortex and hippocampus, and improved memory impairment 2 weeks after trauma. These results suggest that enhancing slow-wave sleep acutely after trauma may have a beneficial disease-modifying effect in subjects with acute TBI.

SPEAKER: ANDREA GOLDIN, *LABORATORIO DE NEUROCIENCIA, UNIVERSIDAD TORCUATO DI TELLA - CONICET*

NEUROSCIENCE FOR (BETTER) EDUCATION

Executive functions (EF) imply processes critical for purposeful, goal-directed behaviour. In children, evidence derived from laboratory measures indicates that training can improve EF. For the first time, we explicitly examined this hypothesis based on real-world measures, especially of educational achievement. We developed a set of computerized brain training games ("Mate Marote") and we investigated whether they might yield transfer on typically developing children in interventions deployed at their own schools.

The games do elicit transfer of some EF, which cascade to real-world measures of school performance. More importantly, an intervention on 6-year-olds equalized academic outcomes across children who regularly attended school and those who did not because of social and familiar circumstances.

SY19. NEUROIMMUNOENDOCRINOLOGY OF THE CIRCADIAN SYSTEM

ROOM B - 16:30-18:30

CHAIR: DIEGO GOLOMBEK (ARGENTINA)

Circadian rhythms in immune, endocrine and metabolic parameters, are controlled by a central suprachiasmatic clock. Desynchronization schedules severely affect humoral and autonomic rhythms, resulting in diverse pathological dysfunctions. Immune and endocrine signals feedback and regulate the clock. This symposium will present neuroimmunoendocrine interactions with the circadian timing system.

SPEAKER: RUUD BUIJS[1], NATALI GUERRERO[1], EVA SOTO[1]

[1]INSTITUTO INVESTIGACIONES BIOMEDICAS UNAM, MEXICO

INTERACTION BETWEEN THE BRAIN AND THE IMMUNE SYSTEM: THE AUTONOMIC REFLEX

The brain is responsible for maintaining homeostasis of the organism, herein the hypothalamus has a special role in integrating information from body and brain, adjusting its output constantly via hormones and the autonomic nervous system to set optimal every compartment of the body. Also the immune system is under strong control of the

brain. Beyond the conventional systemic responses such as fever, HPA axis activation and sickness behavior evoked by the brain during inflammation, the autonomic nervous system is now recognized to exert powerful effects on the inflammatory response. Which branches of the autonomic nervous system are able to decrease inflammation remains controversial. Whether this anti-inflammatory reflex is parasympathetic or sympathetic, is subject of hot debate. Either way, the existence of a strong autonomic influence on the immune system is now an undeniable fact. Therefore, attention needs to be given to those areas of the brain that can modify the autonomic output to the immune system. Here we will analyze the participating elements whereby special attention is given to the hypothalamus as main structure driving the autonomic output of the brain. We will discuss evidence that our biological clock the suprachiasmatic nucleus has a special role in the setting of this reflex determining the intensity of the autonomic reflex. This influence is of such magnitude that SCN disturbances by shift work conditions result in an increased inflammation

SPEAKER: JUAN JOSÉ CHIESA[1], LEANDRO PABLO CASIRAGHI[1], MALENA LIS MUL FEDELE[1], FERNANDA RUTH ROMÁN[1], ANA ALZAMENDI[2], ANDRÉS GIOVAMBATTISTA[2], BELÉN CERLIANI[2], SILVINA RICHARD[2], DIEGO ANDRÉS GOLOMBEK[1], NATALIA PALADINO[1]

[1]DEPARTAMENTO DE CIENCIA Y TECNOLOGÍA, UNIVERSIDAD NACIONAL DE QUILMES; [2]INSTITUTO MULTIDISCIPLINARIO DE BIOLOGÍA CELULAR, CENTRO CIENTÍFICO TECNOLÓGICO, CONICET , ARGENTINA

CIRCADIAN DESYNCHRONIZATION IN A MURINE MODEL OF CHRONIC JET-LAG: EFFECTS ON METABOLISM AND IN EXPERIMENTAL TUMORIGENESIS

Circadian misalignment may lead to pathological states when chronically established. We characterized a C57bl/6 mice model of behavioral desynchronization generated by a 6-h advance every 2 days of the light:dark (LD) cycle, a chronic jet-lag protocol (CJL). We found abnormal body weight gain in these animals when compared to those housed under normal LD cycles. This phenotype was not observed when animals had volitional access to running wheel, as well as when they were restricted to feed during darkness. Also we found some alterations in lipid metabolism, such as increased circulating triglycerides and adipocyte size. This loss of circadian homeostasis of energy balance could emerge from desynchronization between behavior, food assimilation, and metabolism.

In addition we studied the effect of CJL in experimental tumorigenesis in C57bl/6 mice, and in its regulation by immune factors. We found enhanced tumor development in mice under CJL, with higher growth rate and lower latency when compared to controls under LD cycles. An LD variation (higher diurnal levels) of pro-inflammatory cytokines Interleukin 1b, 6, and Tumor Necrosis Factor (TNF)-a, was determined in tumor tissue, but not present in animals under CJL. In addition, expression of clock genes in the tumor tissue was altered in this group. Desynchronization of immune variables, as well as peripheral circadian gene deregulation generated by CJL at the tumor environment, may be implicated in enhanced tumor growth.

SPEAKER: HORACIO DE LA IGLESIA[1], ÂNGELA KATSUYAMA[1]

[1]UNIVERSITY OF WASHINGTON, USA

INTERNAL DESYNCHRONIZATION OF CIRCADIAN RHYTHMS AND THE IMMUNE SYSTEM

Internal desynchronization of circadian rhythms is a common outcome of challenges to the circadian system that are associated with travel though time zones, shifts work, unusual-dark (LD) cycles and other environmental disruptions. Our laboratory has developed an animal model of internal desynchronization in which the internal misalignment of circadian rhythms is induced by exposure of rats to a 22-h LD cycle. This artificially short LD cycle leads to forced desynchrony of internal rhythms in which days in which the rhythms are aligned and misaligned are predictable. Using this animal model we show these animals exhibit disrupted neuroendocrine rhythms, including a dysregulation of the hypothalamo-pituitary-adrenal axis, and an abnormal response to immune challenges. Our findings may shed light into the mechanisms underlying higher disease propensity in humans exposed to circadian challenges.

SPEAKER: REGINA P MARKUS, *LABORATORY OF CHRONOPHARMACOLOGY, INSTITUTE BIOSCIENCE, UNIVEERSITY OF SÃO PAULO, BRAZIL*

IMMUNE-PINEAL AXIS - THE ROLE OF MELATONIN SYNTHESIZED BY GLIA CELLS IN RODENT MODELS AND HUMANS

The immune-pineal axis hypothesis proposes that a danger or pathogen associated molecular pattern reduces or even blocks nocturnal pineal melatonin synthesis, and eventually leads to the synthesis of melatonin by monocytes, including macrophages, colostral and glia cells. Recently we showed that local synthesized melatonin impairs neuronal death in rat cerebellum challenged with LPS. In addition, it will be discussed data that show that survival to glliobastomes is significantly related to an index that takes into account the expression of enzymes of the biosynthetic and metabolic pathway of melatonin. In summary, the relevance of chronobiotic and immune-competent sources of melatonin are not independently modulated. Indeed, they are strictly regulated by pathophysiological mechanisms.

SY20. DOPAMINE NEURONS: CONNECTIVITY, FUNCTIONAL CONNECTIVITY AND SUSCEPTIBILITY

ROOM C - 16:30-18:30

CHAIR: J. PAUL BOLAM (UK)

We will address new findings about how inputs to dopamine neurons control their firing (Henny), how brainstem cholinergic inputs modulate their responsiveness (Mena-Segovia) and how their output may underlie their susceptibility in PD (Bolam). Finally, we will discuss how the symptoms of PD may not entirely be due to the loss of DA (Schiaveto de Souza).

SPEAKER: J. PAUL BOLAM, *MRC UNIT, DEPT PHARMACOLOGY, UNIVERSITY OF OXFORD, UK*

DOPAMINE NEURONS, SYNAPSES AND SUSCEPTIBILITY IN PARKINSON'S DISEASE

There are many hypothesised mechanisms to account for the selective vulnerability of dopamine neurons in Parkinson's disease (PD). One factor that may contribute to this is that the axon and synaptic output of SNc dopamine neurons are remarkably different to all other neurons in the brain in that individual dopamine neurons give rise to hundreds of thousands of synapses in the striatum. We propose that this massive axonal arbour will impose a high energetic demand for normal cell biological functions and the generation and propagation of action potentials (AP) and the subsequent recovery of the membrane potential. Any stressor, e.g. oxidative stress, genetic mutations, or mitochondrial poisons, will have a preferential effect on these neurons because they are energetically 'on-the-edge' and the perturbations leading to energy demand out-stripping supply and eventual cell death. To test this hypothesis we generated a biology-based computational model of the axons of dopamine neurons and examined the energetic impact imposed by their extensive, unmyelinated axonal arbour. The main finding is that the energy demand associated with AP conduction is related in a supra-linear manner to the axonal size and complexity. Thus those neurons that show a greater vulnerability have a disproportionately greater energy cost for action potential propagation. This higher energy demand, together with unique molecular and functional features, may underlie their selective vulnerability in PD.

SPEAKER: PABLO HENNY, *PONTIFICIA UNIVERSIDAD CATOLICA DE CHILE, CHILE*

THE HEART OF A DOPAMINERGIC NEURON

The axon initial segment (AIS) is a specialized subcellular region enriched in voltage gated channels and where action potentials initiate. No studies have addressed the structural, molecular or synaptic characteristics of the AIS in dopaminergic neurons (DAN) nor its role in firing. Using various neuroanatomical tools, we identify the neurochemical and structural features of the AIS in mouse substantia nigra compacta (SNc) DANs, along with the expression of voltage gated channels that may endow them with their typical firing. While the AIS of SNc DANs shares various structural characteristics described in other neurons, evidence is shown that DANs AIS can receive direct synaptic innervation, a characteristic only reported in specific populations of cortical neurons. In situ hybridization for voltage gated sodium channels showed that most SNc neurons expressed Nav1.2, a subunit thought to increase threshold for firing. Using in vivo juxtacellular recording, labeling and 3D reconstructions of the somatodendritic and AIS compartments of individual DANs, we show how the size and location of the AIS closely predicts firing frequency. Computational modelling of the firing behavior of DANs show that firing rate arises from the interaction between AIS and somatodendritic compartments and their respective oscillatory and firing properties. The model also shows that size and location respectively contribute to firing frequency and action potential back propagation success.

SPEAKER: JUAN MENA-SEGOVIA, *RUTGERS UNIVERSITY, USA*

CHOLINERGIC SIGNALING IN THE VTA: FUNCTIONAL IMPLICATIONS FOR DOPAMINE SUBCIRCUITS

Dopamine neurons in the ventral tegmental area (VTA) receive cholinergic innervation from brainstem structures associated with either movement or reward. While cholinergic neurons of the pedunculopontine nucleus (PPN) carry an associative/motor signal, those of the laterodorsal tegmental nucleus (LDT) convey limbic information. In this talk I will present our recent results using optogenetic methods combined with in vivo juxtacellular recording/labeling to dissect the influence of brainstem cholinergic innervation of distinct neuronal subpopulations in the VTA. We found that LDT cholinergic axons selectively enhance the bursting activity of mesolimbic dopamine neurons that are excited by aversive stimulation. In contrast, PPN cholinergic axons activate and change the discharge properties of VTA neurons that are integrated in distinct functional circuits and are inhibited by aversive stimulation. While both structures conveyed a reinforcing signal, they had opposite roles in locomotion. Our results demonstrate that two modes of cholinergic transmission operate in the VTA and segregate neurons involved in different reward circuits.

SPEAKER: ALBERT SCHIAVETO DE SOUZA, *FUNDAÇÃO UNIVERSIDADE FEDERAL DE MATO GROSSO DO SUL, BRAZIL*

Influence of non-dopaminergic transmission on symptoms in rodent models of PD

SY21. NEUROESTEROIDS, CARDIOESTEROIDS AND OXIDATIVE CELL SIGNALLING AS TARGET IN NEUROINFLAMMATION AND POSSIBLE ROLE IN NEURODEGENERATIVE DISEASE

ROOM D - 16:30-18:30

CHAIR: FERNANDO BENETTI (BRAZIL)

The increased longevity of the world's population has been accompanied by an exponential growth in the number of people with neurodegenerative diseases. Several studies have shown multiple actions of progesterone, estrogen, neuroactive steroids, cardiosteroids as well cell oxidation, controll signaling pathways involved in neuronal death, thus are potential candidates for prevent these brain disease.

SPEAKER: WEBER DA SILVA, *UNIVERSIDADE ESTADUAL DO CENTRO-OESTE, BRAZIL*

NEUROINFLAMATION AND MNEMONIC DEFICITS

The increased longevity of the world's population has been accompanied by an exponential growth in the number of people with neurodegenerative diseases. Several studies have shown multiple actions of progesterone, estrogen, neuroactive steroids, cardiosteroids as well cell oxidation, controll signaling pathways involved in neuronal death, thus are potential candidates for prevent these brain disease.

SPEAKER: ALEJANDRO DE NICOLA[1,2], LAURA GARAY[1,2], MARIA MEYER[1], AGUSTINA LARA[1], GISELLA GARGIULO-MONACHELLI[1,3], MARIA CLAUDIA GONZALEZ DESNISELLE[1,2]

[1]INSTITUTO DE BIOLOGIA Y MEDICINA EXPERIMENTAL-CONICET; [2]DEPT. DE FISIOLOGIA Y BIOFISICA, FACULTAD DE MEDICINA, UBA; [3]HOSPITAL DE

AGUDOS JUAN A. FERNANDEZ, ARGENTINA

PROGESTINS AS ANTI-INFLAMMATORY FACTORS IN NEUROLOGICAL DISORDERS

An increasing number of reports supports that progesterone provides neuroprotection against CNS diseases. In the experimental autoimmune encephalomyelitis (EAE) model of multiple sclerosis (MS), progesterone treatment decreased cell infiltration, changed microglia phenotype and reduced the proinflammatory mediators TNFalpha, TLR4 and iNOS in the spinal cord. Concomitantly, progesterone increased myelin proteins and oligodendrocyte progenitors. To elucidate possible mediators of these effects, we analyzed the mRNA of neurosteroidogenic enzymes, considering that locally synthesized steroids bring neuroprotection by autocrine/paracrine mechanisms. We found that in EAE mice progesterone treatment restored the mRNA for the steroidogenic acute regulatory protein (Star), voltage-dependent anion channel (VDAC), P450scc (cholesterol side-chain cleavage), 5alpha-reductase, 3alpha-hydroxysteroid dehydrogenase and aromatase. We also found that the 18 Kd translocator protein (TSPO), a marker of reactive microgliosis was decreased, consequent with the inhibition of microglia reactivity. EAE mice showed pathological mitochondrial morphology and reduced expression of fission and fusion proteins, parameters restored by progesterone. These data indicate that progesterone neuroprotection include the recovery of neurosteroidodogenesis. In this way, endogenously synthesized neurosteroids may reinforce the anti-inflammatory and promyelinating effects of exogenous progesterone found in MS mice.

SPEAKER: LUIS MIGUEL GARCIA-SEGURA, *INSTITUTO CAJAL, CSIC, SPAIN*

ROLE OF ESTROGEN RECEPTORS IN THE REGULATION OF NEUROINFLAMMATION

Neuroprotective actions of 17beta-estradiol (estradiol) are in part mediated by direct actions on neurons. Astrocytes and microglia, which play an essential role in the maintenance of the homeostasis of neural tissue, express estrogen receptors and are also involved in the neuroprotective actions of estradiol in the brain. Estradiol controls gliosis and regulates neuroinflammation, edema and glutamate transport acting on astrocytes and microglia. In addition, the hormone regulates the release of neurotrophic factors and other neuroprotective molecules by astrocytes. In addition, reactive astrocytes are a local source of neuroprotective estradiol for the injured brain. Since estradiol therapy is not free from peripheral risks, alternatives for the hormone have been explored. Some selective estrogen receptor modulators (SERMs), which are already in use in clinical practice for the treatment of breast cancer, osteoporosis or menopausal symptoms, exert similar actions to estradiol on astrocytes and microglia. Therefore, SERMs represent therapeutic alternatives to estradiol for the activation of astroglia and microglia-mediated neuroprotective mechanisms.

SPEAKER: CRISTOFORO SCAVONE[1], ELISA M KAWAMOTO[1]

[1]DEPARTMENT OF PHARMACOLOGY, INSTITUTE OF BIOMEDICAL SCIENCE, UNIVERSITY OF SÃO PAULO, AVEN, BRAZIL

NEUROINFLAMMATION AND BRAIN PLASTICITY INDUCED BY CARDIOSTEROIDS

Endogenous steroids has been shown to play important roles in the modulation of renal sodium transport, arterial pressure, cell growth, differentiation, apoptosis, and the control of various central nervous functions. Na,K-ATPase (NKA) is constituted of 3 subunits, with each subunit having a number of isoforms that provide functional versatility across different cell types. Cardiotonic steroids (CTS) are specific ligands of the alpha subunit. CTS dose-dependently inhibit NKA activity. Recent studies have now shed new light on the function of CTS as hormones, which activate a signaling function of NKA. Ouabain (OUA) has been described as a new hormone synthesized in the adrenal cortex and hypothalamus. Several studies identify OUA as a physiological inducer of calcium oscillation and Src-Ras-MAPK pathways, and indicate a novel and important role for the OUA/NAK complex as a regulator of TNF, NFKB activity and BDNF levels. The non-inhibitory concentrations of OUA have been shown to be protective against some types of injury, such as kainic acid and Shiga toxin. OUA has anti-inflammatory and anti-apoptotic effects in the hippocampus challenged with LPS induced inflammation. The ability of OUA to suppress inflammatory process and maintain hippocampal BDNF levels suggests that NKA signaling cascade could be a new strategy for pharmacological interventions aimed at promoting longevity and healthy aging, as well as for the treatment of neurodegenerative disoders. FAPESP & CNPq.

OCTOBER 20TH

SY22. THE INTERPLAY OF NEURONAL ACTIVITY, SYNAPTOGENESIS AND PLASTICITY

ROOM A - 11:00-13:00

CHAIR: DAMIAN REFOJO (ARGENTINA)

Environmental inputs shape the wiring of the brain influencing timing, dynamics and efficiency of the synaptic connectivity. In this context we proposed to deeply but broadly discuss how different aspects of neuronal activity controls synaptogenesis, cell fate, plasticity and intracellular signaling processes and inversely how those processes influence how neurons perceive and process activity.

SPEAKER: LAURA BORODINSKY, *DEPARTMENT OF PHYSIOLOGY & MEMBRANE BIOLOGY, UNIVERSITY OF CALIFORNIA DAVIS; SHRINERS HOSPITAL FOR CHILDREN NORTHERN CALIFORNIA ; USA*

ENVIRONMENTAL REGULATION OF SPINAL CORD DEVELOPMENT

During development, differentiation of neurons is necessary for generating the cell populations that will makeup the mature nervous system. The patterning of the embryonic nervous system is driven by a developmental program. Whether the environment intervenes in this program, resulting in plastic changes in neuronal differentiation, is an understudied aspect of neural development. It is well established that environmental temperature regulates the rate of development in ectotherms, yet the specific

impact temperature has on nervous system development is unknown. Previous studies demonstrated that embryonic Xenopus spinal neurons exhibit calcium spike activity that is important for neurotransmitter specification. We hypothesize that environmental cues modulate embryonic calcium activity in developing neurons thus regulate neuronal differentiation. We find that in the embryonic ventral spinal cord, calcium spike frequency increases 1.5-fold in response to acute exposure to cold temperature. This increase is blocked by inhibiting the cold-sensitive transient receptor potential cation channel M8. Data show that the temperature in which embryos are grown regulates the number of motor neurons, correlating with calcium activity-dependent changes in motor neuron specification. This study suggests that the environment participates in neuronal differentiation allowing for the establishment of the best-equipped neuronal circuit.

SPEAKER: JUAN BURRONE[1], WINNIE WEFELMEYER[1], ALEJANDRO PAN VAZQUEZ[1]

[1]KING'S COLLEGE LONDON, UK

PLASTICITY AND DEVELOPMENT OF THE AXON INITIAL SEGMENT AND ITS SYNAPSES

The axon initial segment (AIS) is a structure at the proximal end of the axon with a high density of sodium channels that defines the site of action potential generation. It is also the target of inhibitory synapses formed by a specific GABAergic interneuron, the Chandelier cell. Here, we describe how activity affects this local microcircuit of axo-axonic synapses onto the AIS and begin to characterise how it forms. We find that the structure of the AIS is plastic and can change its position along the axon in hippocampal CA1 pyramidal neurons, resulting in a modulation of the cell's excitability. Importantly, GABAergic synapses do not translocate with the AIS, resulting in a partial mismatch between axo-axonic synapses and the AIS. We are currently characterising how this intriguing circuit is formed and shaped by neuronal activity, by using a transgenic mouse line that labels chandelier cells in the cortex, together with a live label of postsynaptic GABAergic compartments in pyramidal neurons. We found a critical temporal window of synapse formation at the AIS (P14-P16), which follows the gross morphological maturation of the Chandelier axonal arbour. Surprisingly, innervation of the AIS continued after this early synaptogenesis period, beyond P22. Here, both presynaptic and postsynaptic compartments gradually increased in number along the AIS, up to P40. We are currently performing in vivo imaging to visualise these dynamic processes as they occur in the brain.

SPEAKER: DAMIAN REFOJO, *IBIOBA-MAX PLANCK, ARGENTINA*

NEDDYLATION, A NEW POSTTRANSLATIONAL MODIFICATION IN THE SYNAPSE

Neddylation is an ubiquitylation-like pathway that controls cell cycle and proliferation by covalently conjugating Nedd8 to specific targets. Even though Nedd8 (NEDD8 neural precursor cell expressed, developmentally down-regulated 8) was originally cloned from brain tissue its role in neurons, nonreplicating postmitotic cells, remains almost entirely unexplored.

We recently found that Nedd8 is ubiquitously expressed in the brain and that neddylation increases along postnatal brain development and with neuronal maturation. Nedd8 conjugation is active in mature synapses, where many proteins are neddylated both at pre- and post-synaptic compartments. Interestingly, Neddylation is essential for normal development of excitatory (but not inhibitory) synapses during neuronal maturation and as well as spine stability in mature neurons.

Using different biochemical tools, we found that neddylated PSD-95 was present in spines and that neddylation on Lys202 of PSD-95 is required for the proactive role of the scaffolding protein in spine maturation and AMPA synaptic transmission. Finally, we developed Nae1CamKIIα-CreERT2 mice, in which neddylation is conditionally ablated in adult excitatory forebrain neurons. These mice showed synaptic loss, impaired neurotransmission and severe cognitive deficits.

Further studies suggesting a substantive role of neddylation on synaptic transmission and plasticity will be discussed.

SPEAKER: GINA TURRIGIANO, *BRANDEIS UNVIERSITY, USA*

DISRUPTED SYNAPTIC SCALING IN RODENT MODELS OF AUTISM-SPECTRUM DISORDERS

Synaptic scaling is a form of homeostatic plasticity that stabilizes neuronal firing rates by globally adjusting excitatory synaptic strengths. We recently showed that synaptic scaling is impaired in a mouse model of Rett syndrome, which shares some features with autism spectrum disorders. To determine whether this defect might generalize to other ASD models we investigated the role of the scaffolding protein Shank3 in synaptic

scaling; human mutations in shank3 are strongly associated with ASDs and other neurological disorders. We find that cell-autonomous knockdown of shank3 to roughly 50% of wildtype levels completely blocks synaptic scaling. This block can be rescued with wildtype shank3, but not by reintroducing shank3 harboring some disease-associated human mutations. Further, we find that synaptic scaling can be pharmacologically rescued in shank3 knockdown neurons. Together our data suggest that loss of homeostatic plasticity may be a common feature of disorders such as ASDs that are characterized by imbalances in excitation and inhibition, and raise the possibility that some defects may be treatable through pharmacological rescue of synaptic scaling.

SY23. THE GLIAL CELL-NEURON REGULATORY CROSSTALK

ROOM C - 11:00-13:00

CHAIR: ROMMY VON BERNHARDI (CHILE)

Glia serve pivotal functions both in the healthy and the diseased CNS. Although their individual properties are known, much less is known about how glia regulate neuronal function. We will address this fundamental problem by discussing in vitro and in vivo evidence on key mechanisms including CNS injury, regulation of synaptic function, and glia-mediated neuro-protection and -degeneration.

SPEAKER: FRANCISCO GUIMARAES, *MEDICAL SCHOOL OF RIBEIRAO PRETO-USP, RIBEIRAO PRETO, SP BRAZIL*

MICROGLIA AND THE ANTIPSYCHOTIC EFFECT OF CANNABIDIOL

Cannabidiol (CBD) is a major cannabinoid present in Cannabis sativa that lacks the psychotomimetic effects and abuse potential of the main component of the plant, delta-9-tetrahydrocannabinol (THC), On the contrary, CBD attenuates the psychotomimetic and anxiogenic effects produced by high doses of THC. In the last decade several preclinical and clinical studies have confirmed that CBD has antipsychotic and anxiolytic properties. Using animal models of psychiatry disorders, we showed that its anxiolytic effects depend on different pharmacological mechanisms, which include facilitation of 5HT1A-mediated neurotransmission, blockade of anandamide metabolism/uptake, and facilitation of adult hippocampal neurogenesis. The molecular mechanisms associated with CBD antipsychotic effects, however, are still unclear. Chronic treatment with an NMDA non-competitive receptor antagonist (MK801) has been proposed as an animal model of schizophrenia. Using this model in mice, we showed that repeated CBD treatment prevents the behavioural and molecular changes induced by MK801. Moreover, it also prevented microglial activation in medial prefrontal cortex and hippocampus. These effects could depend on CBD activation of PPARs-gamma receptors, since an antagonist of these receptors blocked the LPS-induced activation of microglial cells in vitro. Together, these findings suggest that anti-inflammatory effects of CBD could be responsible for the antipsychotic properties of this drug.

SPEAKER: FRANK KIRCHHOFF, *UNIVERSITY OF SAARLAND, HOMBURG, GERMANY*

THE DIVERSITY OF GLIAL RESPONSES IN ACUTE CNS INJURIES – LESSONS FROM TRANSGENIC MICE

Acute brain injuries activate signaling cascades essential for scar formation. Here, we report that acute lesions associated with a disruption of the blood-brain barrier (BBB) trigger re-programming of the oligodendrocyte lineage. Differentiated oligodendrocytes and their precursor cells can generate another neuroglial cell type: astrocytes.

By in vivo 2P-LSM analysis we followed oligodendrocytes after injury in PLP-DsRed1/GFAP-EGFP transgenic mice. Adjacent to the lesion site, oligodendrocytes first turned into an intermediate cell stage with astro- and oligodendroglial gene expression properties (AO cells). Subsequently, portions of AO cells differentiated into astrocytes, while others stayed in the oligodendrocyte lineage. In split-Cre mice, AO cells showed a clear glia-restricted differentiation potential that also depended on local cues. At the lesion higher expression levels of glial differentiation factors were detected. And indeed, local injection of IL-6 promoted the formation of AO cells.

In summary, our findings highlight the plastic potential of oligodendrocytes in acute brain trauma.

SPEAKER: ALEXANDRE OLIVEIRA, *UNIVERSITY OF CAMPINAS, BRAZIL*

MESENCHYMAL STEM CELL THERAPY FOLLOWING INTRASPINAL AXOTOMY: EFFECTS ON GLIAL CELLS AND NEURONAL SURVIVAL

Degeneration of motoneurons may occur after spinal cord trauma in response to direct cell body lesion or proximal axotomy. Of interest, surviving neurons display the ability to regrow their axon, and some of them surpass the glial scar formed at the lesion site. Nevertheless, the exact mechanisms behind survival and regeneration are elusive, but possibly related to the early inflammatory response post trauma. In this scenario, the use of mesenchymal stem cell (MSC) treatment following injury is advantageous since such cells produce a variety of molecules including neurotrophic factors and interleukins. In turn, MSCs theoretically have the ability to drive immune response towards Th2 polarization, giving raise to anti-inflammatory conditions that possibly contribute to neuronal survival and regeneration. Our intent is to show newly obtained data regarding immunomodulatory effects of MSC therapy in response to ventral funiculus penetrating injury. We will address the basis of aphatomy experimental model, regarding motoneuron degeneration and glial scar formation. In addition, the effects of MSC engrafting to the lesion site, combined with the use of fibrin matrix scaffold, will be detailed regarding acute neuroprotection, synaptic circuits preservation and local mRNA levels of VEGF, BDNF, iNOS2, arginase-1, TNF-α, IL-1β, IL-6, IL-10, IL-4, IL-13 and TGF-β. Astroglial and microglial reaction will also be discussed and related to positive effects of cell therapy.

SPEAKER: ROMMY VON BERNHARDI, *NEUROLOGY, SCHOOL OF MEDICINE, PONTIFICIA UNIVERSIDAD CATÓLICA DE CHILE, SANTIAGO, CHILE*

REGULATION OF MICROGLIA-MEDIATED NEURODEGENERATION

Activation of glia is responsible for the neuroinflammation observed in Alzheimer's disease (AD). We have proposed that AD is caused by impaired activation of glia leading to neurodegeneration. Scavenger Receptor A (SR-A) has an important role in Beta-amyloid (Abeta) uptake, and we propose that they serve a key role in glial activation. We showed age-related changes of SR-A and analyzed SR-A dependent regulation of glial activation in AD using a SR-A-/- mouse model and a triple transgenic mice generated in our lab that accumulates Aβ and is KO for SR-A (APP/PS1/SR-A-/-). We evaluated the participation of SR-A on microglial activation in response to stimulation with LPS. We observed a modification on the expression pattern of activation markers. In functional terms, APP/PS1/SR-A-/- microglia showed a decreased LPS-induced production of NO, increased secretion of inflammatory cytokines and degreased levels of anti-inflammatory cytokines compared with APP/PS1. Hippocampal IL1beta and TNFalfa were also elevated in adult APP/PS1/SR-A-/- compared with APP/PS1 mice. Abeta phagocytosis was analyzed both in culture and by flow cytometry of freshly obtained cells from adult animals. APP/PS1/SR-A-/- neonatal microglia showed a reduction of Aβ uptake by neonatal and adult mice. Our results indicate that SR-A has a key role in the pathological neuroinflammatory processes in AD, potentiating microglia-mediated neurodegeneration.

Support: Grant FONDECYT 1131025

SY24. YOUNG INVESTIGATOR SYMPOSIA II

ROOM A - 15:00-17:00

CHAIR: ANTONIA MARIN-BURGIN

SPEAKER: THIAGO CUNHA, *UNIVERSITY OF SAO PAULO SCHOOL OF MEDICINE OF RIBEIRAO PRETO , BRAZIL*

NEURO-IMMUNE-GLIA INTERACTIONS IN THE SENSORY GANGLIA ACCOUNT FOR THE DEVELOPMENT OF ACUTE HERPETIC NEURALGIA

Herpetic neuralgia is the most important symptom of herpes zoster disease, which is caused by Varicella zoster. Nevertheless, the pathophysiological mechanisms involved in herpetic neuralgia are not totally elucidated. Here, we examined the neuro-immune interactions at the sensory ganglia that account for the genesis of herpetic neuralgia by using a murine model of Herpes simplex virus type-1 (HSV-1) infection. The cutaneous HSV-1 infection of mice results in the development of a zosteriform-like skin lesion followed by a time-dependent increase in pain-like responses (mechanical allodynia). Leukocytes, composed mainly of macrophages and neutrophils, infiltrate infected DRGs and account for the development of herpetic neuralgia. Infiltrating leukocytes are responsible for driving the production of TNF, which in turn mediates development of herpetic neuralgia through down-regulation of the inwardly rectifying K+ channel, Kir4.1, in satellite glial cells. These results revealed that neuro-immune interactions at the sensory ganglia play a critical role in the genesis of herpetic neuralgia. In conclusion, the present study elucidates novel mechanisms involved in the genesis of herpetic pain and open new avenues in its control.

SPEAKER: MARÍA FLORENCIA ZAPPA VILLAR[1], GUSTAVO RAMÓN MOREL[1], MARIANA GABRIELA GARCÍA[2], JOAQUÍN PARDO[1], MICAELA LÓPEZ-LEÓN[1], LUCÍA TRÍPODI[1], GUILLERMO MAZZOLINI[2], RODOLFO GUSTAVO GOYA[1], PAULA CECILIA REGGIANI[1]

[1]INIBIOLP-HISTOLOGY B, SCHOOL OF MEDICAL SCIENCES, NATIONAL UNIVERSITY OF LA PLATA; [2]GENE THERAPY LAB, SCHOOL OF BIOMEDICAL SCIENCES, AUSTRAL UNIVERSITY, BUENOS AIRES

THERAPEUTIC POTENTIAL OF HUMAN MESENCHYMAL STEM CELLS AND INSULIN-LIKE GROWTH FACTOR-I GENE THERAPY IN ANIMAL MODELS OF NEURODEGENERATION

Our objective is to develop therapeutic strategies for neurodegenerative disorders, as Alzheimer's Disease (AD), and brain aging. Gene therapy combined with the use potent neuroprotective molecules, like Insulin-like growth factor 1 (IGF-1), emerge as promising tool for this purpose. We constructed an adenoviral vector for rat IGF-1 and implemented intracerebroventricular (ICV) IGF-I restorative gene therapy in the brain of aging rats. The treatment improved the spatial memory accuracy and, in the hippocampus, increased the number of immature neurons and modified astrocytes branching and number.

More recently we implemented human mesenchymal stem cell (MSC) therapy in a rat sporadic AD-model (by ICV injection of streptozotocin) as well as in aging rats. First, we explored the therapeutic effect of MSC ICV injection. In our AD rat model, ICV cell therapy improved the rat spatial and recognition memories performances. Comparable results were observed when we used a similar experimental design in aging rats. Also, in this study we found that cell therapy improves cognitive deficits. Finally, we assessed the effect of repeated intravenous administrations of MSC on cognitive performance in the AD rat model. This treatment improved memory, depression-like and anxiety-like behaviors. In sum, our results are agree with the emerging evidence that supports the use of MSCs for regenerative applications in neurodegenerative disorders and brain aging.

SPEAKER: KARINE MATHILDE CAMPESTRINI DALLAGNOL[1], ALINE PERTILE REMOR[1], RODRIGO AUGUSTO DA SILVA[1], RUI PREDIGER[1], ALEXANDRA LATINI[1], ADERBAL AGUIAR[1]

[1]LABOX, UNIVERSIDADE FEDERAL DE SANTA CATARINA, FLORIANÓPOLIS-SC, BRAZIL

RUNNING FOR REST: EXERCISE ATTENUATES IMPAIRED COGNITION, SICKNESS BEHAVIOR AND NEUROINFLAMMATION IN THE HIPPOCAMPUS OF AGED ANIMALS

Exercise improves mental health and synaptic function in the aged brain. However, the molecular mechanisms involved in exercise-induced healthy brain aging are not well understood. Evidence supports the role of neurogenesis and neuroplasticity in exercise-induced neuroplasticity. The RE1-silencing transcription factor (REST) and an anti-inflammatory role of exercise are also candidate mechanisms. We evaluate the effect of 8 weeks of voluntary exercise on running wheels (RW) on sickness behavior, cognition, neurogenesis and hippocampal gene expression of brain-derived neurotrophic factor (BDNF), REST, and interleukins IL-1 beta and IL-10 of adult and aged mice and rats. The aged animals exhibited impaired cognition, depressive-like and sickness behavior: decreased mobility in the RW and open field and severe immobility in the tail suspension test. The gene expression of REST, IL-1 beta, and IL-10 was increased in the hippocampus of aged mice. Exercise was a cognitive enhancer, anxiolytic and antidepressant and improved motor behavior in aged animals. Exercise also boosted neurogenesis, BDNF (and signalling) and REST expression and decreased IL-1 beta and IL-10 expression in the hippocampus of aged animals. These results support the beneficial role of REST in the aged brain, which can be further enhanced by regular exercise.

SPEAKER: LEZIO S. BUENO-JUNIOR[1], RAFAEL N. RUGGIERO[1], JOSE E. PEIXOTO-SANTOS[1], DANILO B. MARQUES[1], MILTON A. V. AVILA[1], CLEITON LOPES-AGUIAR[1], JOAO P. LEITE[1]

[1] RIBEIRAO PRETO MEDICAL SCHOOL, UNIVERSITY OF SAO PAULO, BRAZIL

THALAMO-PREFRONTAL RESONANCE OF HIPPOCAMPAL INPUTS IS PLASTIC AND ATTENUATED BY THALAMIC SILENCING

The prefrontal cortex (PFC) receives overlapping terminals from CA1/subiculum (CA1/sub) and mediodorsal thalamus (MD). Because the PFC reciprocates its thalamic afferents, CA1/sub inputs could plastically reverberate in the PFC-MD loop, which we examined through unit activity and synaptic plasticity monitoring. Rats were implanted with electrodes in CA1/sub (electrical stimulation), MD and PFC (recording) for a chronic session with paired-pulse stimulation, and high-frequency stimulation (HFS). Both PFC and MD firing responded to CA1/sub pulses with phasic

increases, then a transient decrease (<400 ms). Specifically in the PFC, we observed a delayed-onset increase (400-800 ms) that was potentiated after HFS. CA1/sub pulses elicited distinct field responses in PFC and MD, which underwent long-term potentiation. Those responses were correlated with c-Fos and Zif-268 expression throughout the circuit. We further asked whether MD optogenetic inhibition modulates the CA1/sub-PFC recruitment. A rat expressing green light-driven archaerhodopsins in the MD was implanted as above, except for an optrode into MD. When randomly paired with CA1/sub electrical pulses, MD light pulses attenuated PFC delayed-onset responses. Thus, hippocampal inputs seem to plastically resonate within the thalamo-prefrontal loop. These findings contribute to the systems-level understanding of limbic-prefrontal functions (e.g., working memory), and dysfunctions (e.g., psychoses and seizure amplification).

SPEAKER: CYNTHIA KATCHE[1], JORGE H. MEDINA[1]

[1]IBCN - UBA - CONICET

REQUIREMENT OF AN EARLY ACTIVATION OF BDNF/C-FOS CASCADE IN THE RETROSPLENIAL CORTEX FOR THE PERSISTENCE OF A LONG-LASTING AVERSIVE MEMORY

During the past few years there has been growing interest in the role of the retrosplenial cortex (RSC) in memory processing. However, little is known about the molecular changes that take place in this brain region during memory formation. In the present work, we studied the early posttraining participation of RSC in the formation of a long-lasting memory in rats. We found an increase in c-Fos levels in the anterior part of the RSC (aRSC) after inhibitory avoidance (IA) training. Interestingly, this increase was associated with memory durability, since blocking c-Fos expression using specific antisense-oligonucleotides (ASO) impaired long-lasting retention 7 days after training without affecting memory expression 2 days after training. In addition, we showed that BDNF is one of the upstream signals for c-Fos expression required for memory persistence, since blocking BDNF synthesis prevents IA training induced-increase in c-Fos levels in aRSC and affects memory persistence. In addition, we found that injection of BDNF into aRSC around training was sufficient to establish a persistent memory and that this effect was prevented by c-fos ASO infusion into the same structure. These findings reveal an early posttraining involvement of aRSC in the processing of a long-lasting aversive memory.

SPEAKER: EMILIO KROPFF, *FUNDACIÓN INSTITUTO LELOIR - IIBBA - CONICET*

SPEED CELLS AND SPATIAL NAVIGATION IN THE ENTORHINAL CORTEX

Grid cells in the mammalian Entorhinal Cortex provide a metric for space, and it has been proposed that they are at the core of a mechanism for orientation based on self-motion cues. Such a mechanism would also need a robust speed signal. Here we present speed cells, a population of entorhinal neurons dedicated to code for running speed in a linear, context-invariant and prospective way.

SY25. PARKINSON'S DISEASE: FROM NEURONAL DEATH TO THERAPEUTICS

ROOM B - 15:00-17:00

CHAIR: JUAN FERRARIO AND GUSTAVO MURER (ARGENTINA)

The etiology of Parkinson's Disease (PD) is unknown and its treatment is still unresolved due L-DOPA side effects. We cover promising and challenging areas in basic research of PD: neuronal death, immunotherapy, genetic approaches (MV & CH) and pathophysiology of Dyskinesias (RM). Authors are top ranked and produce resonant contributions. We are willing to favor poster discussion and networking.

SPEAKER: CLAUDIO HETZ, *BIOMEDICAL NEUROSCIENCE INSTITUTE, FACULTY OF MEDICINE, CHILE; CENTER FOR GEROSCIENCE, BRAIN HEALTH AND METABOLISM, SANTIAGO, CHILE; DEPARTMENT OF IMMUNOLOGY AND INFECTIOUS DISEASES, HARVARD SCHOOL OF PUBLIC HEALTH, USA; THE BUCK INSTITUTE FOR RESEARCH ON AGING, USA*

ENDOPLASMIC RETICULUM PROTEOSTASIS ALTERATIONS IN BRAIN DISEASES

Most neurodegenerative diseases share a common neuropathology, primarily featuring the presence of abnormal protein inclusions containing specific misfolded proteins. Recent evidence indicates that alteration in organelle function is a common pathological feature of protein misfolding disorders. The endoplasmic reticulum (ER) is an essential compartment for protein folding, maturation, and secretion. Signs of ER stress have been extensively described in most experimental models of neurological disorders. To cope with ER stress, cells activate an integrated signaling response termed the Unfolded Protein Response (UPR), which aims to reestablish homeostasis through transcriptional upregulation of genes involved in protein folding, quality control and degradation pathways. Here we discuss our efforts to assess the role of the UPR in neurodegenerative diseases including ALS and Parkinson. An emerging concept will be discussed where the impact of the UPR to neurodegeneration depends on the disease context and the specific signaling branch analyzed. Finally, strategies to alleviate ER stress using gene therapy and pharmacological approaches will be discussed.

SPEAKER: LUZ SUAREZ[1], OSCAR SOLIS[1], ROSARIO MORATALLA[1]

[1]INSTITUTO CAJAL, CSIC, CIBERNED, MADRID, SPAIN

OPPOSITE STRUCTURAL AND SYNAPTIC PLASTICITY IN D1- AND D2-PROJECTION NEURONS IN L-DOPA-INDUCED DYSKINESIAS

The synaptic organization of striatal medium-spiny neurons (MSNs) confers to dopamine a central role modulating glutamatergic-signaling from cortex and thalamus differentially in both output-pathways, striatonigral (D1-MSN) and striatopallidal (D2-MSN). The loss of dopamine fibers in Parkinson´s disease as well as chronic L-DOPA that induced dyskinesia produce severe alterations in the functioning of corticostriatal synapses. However, the specific changes in both types of MSNs underlying these alterations is still unclear. Using BAC-transgenic mice to identify striatal projection neurons, we demonstrate that spine-pruning caused by DA-depletion in Parkinson´s

disease affects mature spines similarly in D1- and D2-MSNs, enhancing the excitability of both striatal-pathways but reducing synaptic-strength selectively in D2-MSN. L-DOPA treatment restores spine density, synaptic-transmission and excitability to normal values selectively in D2-MSNs. However, chronic L-DOPA-treatment also modifies DR-sensitization, enhancing D1R-signaling but reducing D2R-mediated responses. All these alterations could contribute to the loss of bidirectional synaptic-plasticity observed in dyskinesia.

SPEAKER: MIQUEL VILA, *VALL D'HEBRON RESEARCH INSTITUTE (BARCELONA, SPAIN); CATALAN INSTITUTION FOR RESEARCH AND ADVANCED STUDIES (ICREA, BARCELONA, SPAIN); CENTRO DE INVESTIGACIÓN BIOMÉDICA EN RED EN ENFERMEDADES NEURODEGENERATIVAS (CIBERNED); UNIVERSIDAD AUTÓNOMA DE BARCELONA, SPAIN*

DOES ALPHA-SYNUCLEIN PATHOLOGY SPREAD IN THE BRAIN?

Formation and accumulation of abnormal protein aggregates are a central hallmark of several neurodegenerative diseases. In Parkinson's disease (PD), the aggregation-prone protein alpha-synuclein accumulates in several areas of the central and peripheral nervous system. Mounting evidence suggests that neuropathological alpha-synuclein lesions in PD may self-propagate and spread progressively between interconnected brain regions by a cell-to-cell transmission mechanism, thereby potentially contributing to the progression and extension of the disease.

SY26. NEUROFRAMES SYMPOSIUM - FREUD REVISITED: COMPUTATIONAL PSYCHIATRY

ROOM C - 15:00-17:00

CHAIR: JACOBO SITT & DAN SHULZ (FRANCE)

Since the onset of Psychiatry, clinical interviews are the base of mental disease diagnosis. Computational Psychiatry aims at objectively quantifying and modeling patient's signs and symptoms.

The ultimate objective of this new discipline is to develop clinical evaluation tools complementary to the expert's opinion. Here, we will present this new discipline and successful implementations.

SPEAKER: SIDARTA RIBEIRO, *BRAIN INSTITUTE, FEDERAL UNIVERSITY OF RIO GRANDE DO NORTE, BRAZIL*

A QUANTITATIVE REASSESSMENT OF DREAMS AS THE ROYAL ROAD TO THE UNCONSCIOUS

Emil Kraepelin, Eugen Bleuler and Sigmund Freud disagreed on many things, but they agreed that dreaming and psychosis are related, and that dream interpretation is relevant for psychiatric diagnosis. These notions lost traction in the 20th century but underwent a recent revival due to quantitative investigations of the structure of psychotic speech. This presentation will review results showing that the graph-theoretical analysis of dream reports is particularly useful for the differential diagnosis of psychosis. Indeed, dream reports are more informative than reports on several other kinds of long or short-term memories. The presentation will conclude with a discussion of alternative explanations for this fact.

SPEAKER: DIEGO FERNANDEZ SLEZAK, *UNIVERSIDAD DE BUENOS AIRES; CONICET, ARGENTINA*

AUTOMATED CHARACTERIZATION OF MENTAL STATES: A NATURAL LANGUAGE PROCESSING APPROACH

Nowadays, psychiatric disorders are assessed by qualitative semi-structured interviews and diagnosed without any modern machine-learning computational support. We will show how using semantic and morpho-syntactic features of text produced by patients may capture markers of psychiatric and neurological conditions. These techniques open new challenges in the development of Computational Clinical Decision Support Tools to assist the psychiatric practice, by the integration of automatic text transcription and natural language processing for mental state inference using cloud-based services providing a world-wide scalable support.

SPEAKER: RAPHAEL LE BOUC[1,2,3], RIGOUX LIONEL[1,2], PESSIGLIONE MATHIAS[1,2]

[1]MOTIVATION, BRAIN AND BEHAVIOR TEAM, INSTITUT DU CERVEAU ET DE LA MOELLE EPINIERE; [2]NSERM UMR1127, CNRS UMR 7225, UNIVERSITÉ PIERRE ET MARIE CURIE-PARIS 6; [3]URGENCES CEREBRO-VASCULAIRES, HÔPITAL DE LA PITIÉ-SALPÊTRIÈRE, AP-HP, PARIS, FRANCE

COMPUTATIONAL DISSECTION OF DOPAMINE MOTOR AND MOTIVATIONAL FUNCTIONS IN HUMANS

Motor dysfunction (e.g. bradykinesia) and motivational deficit (i.e. apathy) are hallmarks of Parkinson's disease (PD). Yet, it remains unclear whether these symptoms arise from a same dopaminergic dysfunction. Here, we develop a computational model that articulates motor control to economic decision theory, to dissect the motor and motivational functions of dopamine. This model can capture different aspects of the behavior: choice (which action is selected) and vigor (action speed and intensity). It was used to characterize the behavior of 24 PD patients, tested both medicated and unmedicated, in two tasks: an incentive motivation task that involved producing a physical effort, knowing that it would be multiplied by reward level to calculate the payoff, and a choice task that involved choosing between high reward/high effort and low reward/low effort options. Model-free analyses in both tasks showed the same two effects: dopamine depletion 1) decreased the amount of effort that patients were willing to produce for a given reward and 2) slowed down the production of this effort, regardless of reward level. Our model captured these effects with two independent parameters: reward sensitivity and motor activation rate. These parameters were respectively predictive of medication effects on clinical measures of apathy and motor dysfunction. We suggest that such computational phenotyping might help characterizing deficits and refining treatments in neuropsychiatric disorders.

SPEAKER: JACOBO SITT, *INSERM, FRANCE; ICM INSTITUTE, PARIS, FRANCE*

NEUROFRAMES SYMPOSIUM - FREUD REVISITED: COMPUTATIONAL PSYCHIATRY

Since the onset of Psychiatry, clinical interviews are the base of mental disease diagnosis. Computational Psychiatry aims at objectively quantifying and modeling patient's signs and symptoms.

The ultimate objective of this new discipline is to develop clinical evaluation tools complementary to the expert's opinion. Here, we will present this new discipline and successful implementations.

SPEAKER: FABIEN VINCKIER[1,2,3], RAPHAEL GAILLARD[1,4,5]

[1]SERVICE DE PSYCHIATRIE, CENTRE HOSPITALIER SAINTE-ANNE, UNIVERSITÉ PARIS DESCARTES; [2]MOTIVATION, BRAIN, AND BEHAVIOR LAB, INSTITUT DU CERVEAU ET DE LA MOELLE ÉPINIÈRE; [3]INSERM U975, CNRS UMR 7225, UPMC-P6, UMR S 1127, PARIS CEDEX 13, FRANCE; [4]DEPARTMENT OF PSYCHIATRY AND BEHAVIOURAL AND CLINICAL NEUROSCIENCE INSTITUTE, CAMBRIDGE; [5]LABORATOIRE DE "PHYSIOPATHOLOGIE DES MALADIES PSYCHIATRIQUES, CPN; FRANCE

CONFIDENCE AND PSYCHOSIS: A NEURO-COMPUTATIONAL ACCOUNT OF CONTINGENCY LEARNING DISRUPTION BY NMDA BLOCKADE

A state of pathological uncertainty about environmental regularities might represent a key step in the pathway to psychotic illness. Early psychosis can be investigated in healthy volunteers under ketamine, an NMDA receptor antagonist. Here, we explored the effects of ketamine on contingency learning using a placebo-controlled, double-blind, crossover design. During functional magnetic resonance imaging, participants performed an instrumental learning task, in which cue-outcome contingencies were probabilistic and reversed between blocks. Bayesian model comparison indicated that in such an unstable environment, reinforcement learning parameters are downregulated depending on confidence level, an adaptive mechanism that was specifically disrupted by ketamine administration. Drug effects were underpinned by altered neural activity in a fronto-parietal network, which reflected the confidence-based shift to exploitation of learned contingencies. Our findings suggest that an early characteristic of psychosis lies in a persistent doubt that undermines the stabilization of behavioral policy resulting in a failure to exploit regularities in the environment.

EVEN

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PROGRAM IN DETAIL

SPECIAL EVENTS

Special Events

OCTOBER 17TH

SE1. MEET THE PROFESSOR "NEUROSCIENCE AT LUNCH WITH A GOOD FRIEND" PROFESSOR JOHN G. NICHOLLS

ROOM A - 13:30-15:00

CHAIRS:

E. DEL-BEL, *FORP-USP-BRAZIL*

F.F. DE MIGUEL, *UNAM-MEXICO*

Professor John Nicholls will give an informal lecture about his views on neurosciences and other issues, followed by an informal discussion. This is one of the activities for which John is most remembered by students everywhere in the world. This event will give an unique opportunity for new and former students to have a view of Neuroscience form one of its pillars.

The lecture will be held during lunch time; simple food and beverages will be available.

SE2. NEUROSCIENCE AND EDUCATION: PRIME TIME TO BUILD THE BRIDGE

ROOM C - 13:30-15:00

CHAIR: MARIANO SIGMAN *UNIVERSIDAD DI TELLA, ARGENTINA*

As neuroscience gains social traction and entices media attention, the notion that education has much to benefit from brain research becomes increasingly popular. However, it has been argued that the fundamental bridge toward education is cognitive psychology, not neuroscience. In this symposium we will present specific cases in which neuroscience synergizes with other disciplines to serve education, ranging from very general physiological aspects of human learning such as nutrition, exercise and sleep, to brain architectures that shape the way we acquire language and reading, and neuroscience tools that increasingly allow the early detection of cognitive deficits, especially in preverbal infants. Neuroscience methods, tools and theoretical frameworks have broadened our understanding of the mind in a way that is highly relevant to educational practice. Although the bridge's cement is still fresh, we argue why it is prime time to march over it.

- Presentation 1: Physiology in School Learning: Eat, Sleep, Exercise - SPEAKER: SIDARTA RIBEIRO *(BRAZIL)*

- Presentation 2: Mechanisms of sleep-dependent learning - SPEAKER: JAN BORN *(GERMANY)*

- Presentation 3: Time and Numbers: From Lab to School - SPEAKER: ALEJANDRO MAICHINE *(URUGUAY)*

- Presentation 4: The illusion of knowledge - SPEAKER: MARIANO SIGMAN *(ARGENTINA)*

OCTOBER 18TH

SE3. HOW CAN NEUROSCIENCE RESEARCH IMPACT THE GLOBAL BURDEN OF DISEASE

ROOM A - 13:30-15:00

CHAIR: PEDRO VALDES-SOSA *JOINT CHINA-CUBA LABORATORY FOR FRONTIER RESEARCH IN TRANSLATIONAL NEUROTECHNOLOGY, CUBA*

ACCELERATING THE IMPACT NEUROSCIENCE RESEARCH ON GLOBAL HEALTH

A frequently voiced opinion is that Neuroscience Research has little direct contact with public health. We also believe that basic research has its own internal logic and that often serendipitous findings have enormous practical implications. However, we cannot ignore that real world problems can drive very fundamental questions and that keeping these in mind can facilitate translational research. The interplay of basic research and population brain health is illustrated by experience of the Cuban Neuorscience Center. These issues are of particular relevance today due to the huge funding dedicated to the great brain projects on a global scale. Awareness of the possibilities will save time—and brains.

- Presentation 1: **Learning disabilities in children. State of the art and future challenges - SPEAKER: VIVIAN REIGOSO-CRESPO** *(CUBA)*

- Presentation 2: **Population-based neuroimaging and GWAS: OCTAGENE study and clinical implications - SPEAKER: EDSON AMARO JR.** *(BRAZIL)*

- Presentation 3: **A role for the International Brain Research Organization (IBRO) in helping shape global health policy decisions - SPEAKER: LARRY W. SWANSON** *(IBRO)*

SE4. WORKSHOP: HOW TO GET PUBLISHED

ROOM C - 13:30-15:00

CHAIR: JUAN LERMA, *INSTITUTO DE NEUROCIENCIAS, ALICANTE, SPAIN*

OCTOBER 20TH

SE5. LABMAN: LATIN AMERICAN BRAIN MAPPING NETWORK

ROOM A - 13:30-15:00

CHAIR: VALERIA DELLA MAGGIORE, *IFIBIO HOUSAY, DEPARTAMENTO DE FISIOLOGÍA, FACULTAD DE MEDICINA, UBA, ARGENTINA*

Neuroscience and neuroimaging research in Latin America is hindered by a lack of critical mass within any single country. LABMAN (www.labman.org) is an initiative intended to formalize disparate collaborative threads into a Latin American network via exchange of software, data, personnel, training and ideas through training and collaboration. This meeting is intended at recruiting more laboratories and members interested in human brain mapping research, discussing current and future collaborative projects in the region and sharing information regarding new developments. Please join us!

SE6. WORKSHOP
SUBMITTING YOUR WORK TO AN INTERNATIONAL JOURNAL: THE PEER-REVIEW SYSTEM AND WHAT WE EXPECT IN A GOOD PAPER

ROOM C - 13:30-15:00

CHAIR: J. PAUL BOLAM, *CO-EDITOR-IN-CHIEF EUROPEAN JOURNAL OF NEUROSCIENCE; MRC BRAIN NETWORK DYNAMICS UNIT; DEPARTMENT OF PHARMACOLOGY, OXFORD, UK*

POST

Posters Schedule

SESSION 1
Day 2 - October 18th
Posters 1-280

SESSION 2
Day 3 - October 19th
Posters 281-554

SESSION 3
Day 4 - October 20th
Posters 555-827

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PROGRAM IN DETAIL

POSTERS

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POSTER SESSION 1

October 18, 2016

ADVOCACY AND EDUCATION

S1P1. THE VISUAL BRAIN

MARIA MERCEDES BENEDETTO[1*], PAULA VIRGINIA SUBIRADA CALDARONE[1], MARIA CONSTANZA PAZ[1], MARIA LUZ QUINTEROS[1], MAGALI EVELIN RIDANO[1], PABLO FEDERICO BARCELONA[1], MARIA CECILIA SANCHEZ[1]

[1] DEPARTAMENTO DE QUIMICA BIOLOGICA-CIQUIBIC (CONICET), FCQ, UNC.

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S1P2. WHAT´S IN YOUR HEAD? BRAINS GO TO COLLEGE. 2ND BAW SATELLITE IN THE SOUTH OF THE METROPOLITAN AREA OF BUENOS AIRES

CARLOS SEBASTIAN CALDART VALLE[1*], MALENA LIS MUL FEDELE[1], IVANA LEDA BUSSI[1], LEANDRO PABLO CASIRAGHI[2]

[1] LABORATORIO DE CRONOBIOLOGÍA, DEPARTAMENTO DE CIENCIA Y TECNOLOGÍA, UNIVERSIDAD NACIONAL DE QUILMES. BERNAL; [2] LABORATORIO DE NEUROCIENCIA, UNIVERSIDAD TORCUATO DI TELLA

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S1P3. LA PLATA BAW 2016: MY BRAIN CONTROLS ME

SANTIAGO CORDISCO GONZALEZ[1*]

[1] ELECTROPHYSIOLOGY LABORATORY, IMBICE

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S1P4. CONHECER NEURO: TALKING ABOUT NEURAL DEVELOPMENT TO TEENAGERS

ALAN COSTA[1*], PENHA DALTRO-SANTOS[1], MARTA RODRIGUES[1], GUSTAVO TAVEIRA[1], CASSIANA BALDUCI[1], EVERTON COSTA[1], MICHAEL ROCHA[1], LUIZ FELGUEIRAS[1], CAMILA PINTO[1], ANA FERREIRA[1], JAMMILY BIGNON[1], FRANK COSTA[1]

[1] UERJ.

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BEHAVIOR, NEUROETHOLOGY, MEMORY AND COGNITION

S1P5. ULTRA-PROCESSED FOODS SHOULD NOT BE LABELLED AS HEALTHY: EVIDENCE FROM NUTRITIONAL TRAFFIC LIGHT LABEL

LAURA KRUTMAN[1], FILIPE BRAGA[2], JÉSSICA R. DE ANDRADE[3], RAFAEL DELGADO[4], FÁBIO DA S. GOMES[5], MIRTES PEREIRA[1], LETÍCIA DE OLIVEIRA[1], SONIA RODRÍGUEZ-RUIZ[4], M CARMEN FERNÁNDEZ-SANTAELLA[4], ELIANE VOLCHAN[2], ISABEL A. DAVID.[1*]

[1] LABORATÓRIO DE NEUROFISIOLOGIA DO COMPORTAMENTO, CMB, UFF, RJ, BRASIL; [2] LABORATÓRIO DE NEUROBIOLOGIA II, IBCCF, UFRJ, RJ, BRASIL; [3] LABORATÓRIO INTEGRADO DE PESQUISA EM ESTRESSE, IPUB, UFRJ, RJ, BRASIL; [4]LABORATÓRIO DE PSICOFISIOLOGIA HUMANA Y SALUD, CIMCYC, UGR, GRANADA, ESPANHA; [5]UNIDADE TÉCNICA DE ALIMENTAÇÃO, NUTRIÇÃO E CÂNCER, INCA, RJ, BRASIL.

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S1P6. ALTERED NEURAL ACTIVITY IN CLINICAL RISK INDIVIDUALS DURING CROSS MODAL TASK

BELEN ABURTO[1*], ROLANDO CASTILLO[1], ROCÍO MAYOL[1], SEBASTIAN CORRAL[1], ROCÍO LOYOLA[1], ANTÍGONA MARTÍNEZ[2], JOSÉ CORTÉS-BRIONES[3], HERNAN SILVA[4], PABLO GASPAR[4].

[1] UNIVERSIDAD DE CHILE; [2] COLUMBIA UNIVERSITY; [3]YALE UNIVERSITY; [4]CLÍNICA PSIQUIATRICA DEL HOSPITAL CLÍNICO, UNIVERSIDAD DE CHILE.

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S1P7. DEFICITS IN TEMPORAL PROCESSING IN A MOUSE MODEL OF AUTISM

JULIETA ACOSTA[1*], MARCOS CAMPOLONGO[2], CHRISTIAN HÖCHT3, AMAICHA DEPINO2, DIEGO A. GOLOMBEK1, PATRICIA V. AGOSTINO1,

[1] LABORATORIO DE CRONOBIOLOGÍA, UNIVERSIDAD NACIONAL DE QUILMES/CONICET; [2] INSTITUTO DE FISIOLOGÍA, BIOLOGÍA MOLECULAR Y NEUROCIENCIAS, CONICET-UBA; [3]CÁTEDRA DE FARMACOLOGÍA, FACULTAD DE FARMACIA Y BIOQUÍMICA, UBA.

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S1P8. THE ROLE OF THE VENTRAL ANTEROMEDIAL THALAMIC NUCLEUS IN THE NEURAL CIRCUIT OF FEAR USING AN OLFACTORY AVERSIVE CONDITIONING PARADIGM

LUCÍA RAILY ACUÑA[1*], ANTONIO P. CAROBREZ[1]

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S1P9. TEMPORAL PROCESSING AND UHDRS CORRELATION IN HUNTINGTON'S DISEASE

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S1P10. ANALYSIS OF THE YOHIMBINE EFFECTS ON THE EMOTIONAL BEHAVIOR OF MALE RATS IN THE ELEVATED PLUS MAZE

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S1P11. MULTIPLES REPRODUCTIVE EXPERIENCES IN RATS PROTECT THE MOTHER AGAINST THE CONSEQUENCES OF DISRUPTING THE NATURAL DAM-PUP INTERACTION

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S1P12. ELECTROPHYSIOLOGICAL TOOLS FOR THE STUDY OF INTEROCEPTION DURING PROSOCIAL BEHAVIORS

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S1P13. EMOTIONAL MODULATION ON THETA AND ALPHA BAND POWER: A TEMPORAL ANALYSIS

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S1P14. IS TIME-COURSE OF ENDOGENOUS COVERT ORIENTING OF ATTENTION EQUIVALENT TO TIME-COURSE OF VOLUNTARY COVERT ORIENTING OF ATTENTION?

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S1P15. ENRICHED ENVIRONMENT REVERTS BEHAVIORAL DEFICITS IN A MOUSE MODEL OF PERINATALK PROTEIN MALNUTRITION

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S1P16. HIPPOCAMPAL NF-KAPPA B ACTIVITY IS REQUIRED FOR NOVEL OBJECT RECOGNITION MEMORY RECONSOLIDATION

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S1P17. THE TIME COURSE OF ASSOCIATIVE MEMORY RETRIEVAL DURING A PAIR ASSOCIATION TASK

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S1P18. SPATIAL COGNITION IN HIGH ABILITY CHILDREN: SEARCHING FOR A PHYSIOLOGICAL MARKER USING ELETROENCEFALOGRAPHY

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S1P19. CONTEXT-DEPENDENT EFFECTS OF RIMONABANT ON ETHANOL-INDUCED CONDITIONED PLACE PREFERENCE IN FEMALE MICE

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S1P20. UNDERSTANDING DUAL TASK PERFORMANCE IN HUMANS: ELECTROPHYSIOLOGICAL CORRELATES OF INTERFERENCES AND COSTS BETWEEN MOTOR AND WORKING MEMORY TASKS

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S1P21. ANXIOGENIC-LIKE BEHAVIOR INDUCED BY ACUTE RESTRAINT STRESS IS ASSOCIATED WITH DECREASE OF GABA LEVELS AND NEURAL ACTIVATION IN ZEBRAFISH BRAIN

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S1P22. SEARCHING FOR THE OPTIC FLOW PROCESSING CENTER IN CRABS

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S1P23. SUCROSE BEVERAGE CONSUMPTION AND ITS REPERCUSSION ON THE INTAKE OF HIGH-CALORIC FOOD AND BODY WEIGHT

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S1P24. VIOLENT VIDEO GAMES INTERFERE IN ADVANTAGEOUS DEFENSIVE BEHAVIOR

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S1P25. STOP OR MOVE UNDER GUN THREAT: IMPLICIT DEFENSIVE STRATEGIES IN HUMANS

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S1P26. LOOKING FOR NEURAL CORRELATES OF A RECONSOLIDATED DECLARATIVE MEMORY. AN FMRI STUDY

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S1P27. EFFECTS OF TEMPORARY INACTIVATION OF THE INFRALIMBIC SUBREGION OF THE MEDIAL PREFRONTAL CORTEX ON CONTEXTUAL FEAR MEMORY CONSOLIDATION IN RATS

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S1P28. INVOLVEMENT OF MEMORY AND INFLAMMATION IN EFFECT OF MELATONIN IN A MODEL OF DEMENTIA ANIMAL INDUCED BY PEPTIDE ASS 1-42

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S1P29. SLEEP ENHANCES CONTEXT DEPENDANT EXTINCTION MEMORY

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S1P30. SLOW WAVE SLEEP IN THE POSTPARTUM RAT AND ITS LITTER WEIGHT GAIN ARE PROMOTED AFTER DOPAMINERGIC ANTAGONISTIC TREATMENT

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S1P31. HISTAMINE IN THE BASOLATERAL AMYGDALA PROMOTES INHIBITORY AVOIDANCE LEARNING INDEPENDENTLY OF HIPPOCAMPUS

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S1P32. EFFECTS OF PHARMACOLOGICAL INTERVENTIONS AROUND THE EXPRESSION OF GENERALIZED FEAR ON THE ORIGINAL AVERSIVE MEMORY

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S1P33. UNDERSTANDING PREDICTABILITY AND PROVERB READING USING LINEAR MIXED MODELS AND TIME-FREQUENCY ANALYSIS

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S1P34. A NEURONAL AMINE-GATED CHLORIDE CHANNEL GOVERNS SATIETY IN C.ELEGANS

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S1P35. THE ROLE OF POSITIVITY ON WORKING MEMORY'S LOAD EFFECT

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S1P36. CAFFEINE PROTECTS AGAINST THE IMPAIRMENT IN AVERSIVE MEMORY ACQUISITION INDUCED BY ACUTE EXPOSURE WITH METHYLMERCURY IN RATS

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S1P37. THE INFLUENCE OF KETAMINE ON THE EXPRESSION OF LEARNED FEAR RESPONSES IN FEMALE RATS TESTED IN DIFFERENT HORMONAL PERIODS

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S1P38. THE INFLUENCE OF 2APB IN THE SOMATIC AND SENSORIMOTOR DEVELOPMENT OF RATS AFTER NEONATAL ANOXIA

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S1P39. CONFLICT AT RESPONSE LEVEL: HOW THE STROOP AND AFFORDANCE EFFECTS INTERACT?

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S1P40. NEURAL SIGNATURES OF MODIFIED DECLARATIVE MEMORIES AS RESULT OF RECONSOLIDATION PROCESS

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S1P41. EXPRESSION LEVELS OF 5-HT1A RECEPTORS IN HIPPOCAMPUS AND DORSAL RAPHE NUCLEUS HAVE CORRELATION WITH RESILIENCE TO STRESS

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S1P42. ISOFLAVONES IMPROVES THE EPISODIC-LIKE MEMORY IN MIDDLE-AGE FEMALE RATS

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S1P43. SUB-ACUTE EXERCISE REDUCES INTRACELLULAR SIGNALING PATHWAYS LINKED TO INFLAMMATION AND CELL DEATH AND IMPROVES LEARNING AND MEMORY IN AGED RATS

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S1P44. DIFFERENT TRAINING INTENSITIES DURING CONTEXTUAL FEAR CONDITIONING GUIDING THE RATE OF SYNAPTIC CONSOLIDATION

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S1P45. KNOCKING OUT DOPAMINE D2 RECEPTORS IN THE AMYGDALA IMPAIRS RISK EVALUATION

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S1P46. "WHOSE CHAIR IS IT, ANYWAY?" PART II: THE ROLE OF FIRST POSSESSION AND SOCIAL RECOGNITION OF PROPERTY IN THE RESOLUTION OF OWNERSHIP CONFLICTS BY CHILDREN AND ADULTS

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S1P47. BEHAVIORAL MODULATION IN ZEBRAFISH FEMALES OF DIFFERENT REPRODUCTIVE STATES, THROUGH VISUAL AND CHEMICAL SIGNALS

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S1P48. CHANGES IN NMDAR SUBUNITS LEVELS AFTER AN OBJECT RECOGNITION TASK

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S1P49. EFFECT OF DOPAMINERGIC BLOCKADE IN THALAMIC RETICULAR NUCLEUS ON NON-SPACIAL AND SPACIAL MEMORY IN RAT

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S1P50. EVALUATION OF PCREB/CREB EXPRESSION AND CATECHOLAMINE CONTENT IN HIGH AND LOW CONTEXTUAL FEAR CONDITIONING RATS

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S1P51. TEMPORAL DYNAMICS OF THE MAGNOCELLULAR ALTERATIONS IN CLINICAL HIGH RISK OF PSYCHOSIS

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S1P52. RELATIONSHIP BETWEEN PLASMA CORTICOSTERONE LEVELS AND CONTEXTUAL FEAR MEMORY DISCRIMINATION IN RATS

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S1P53. EFFECTS OF ENVIRONMENTAL ENRICHMENT ON COGNITIVE AND EMOTIONAL RESPONSES AND HISTOLOGICAL BRAIN CHANGES IN WISTAR RATS SUBMITTED TO MATERNAL SEPARATION

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S1P54. HIGH BUTTER AND SUGAR DIET ALTERS RESPONSE TO ETHANOL INDEPENDENT OF OBESITY

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S1P55. THE ROLE OF THE HISTAMINE H1 RECEPTOR IN THE HIPPOCAMPAL THETA OSCILLATION DURING SPATIAL NAVIGATION

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S1P56. ANXIOGENIC-LIKE EFFECTS INDUCED BY NITRIC OXIDE WITHIN THE RIGHT MEDIAL PREFRONTAL CORTEX DEPEND ON NMDA (N-METHYL-D-ASPARTATE) RECEPTOR ACTIVATION IN MICE

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S1P57. ENVIRONMENTAL ENRICHMENT INCREASES BALB/C NATURAL PREFERENCE BY CLOSED ARMS IN ELEVATED PLUS MAZE

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S1P58. ENRICHED ENVIRONMENT INFLUENCES ON THE NUMBER OF TELENCEPHALIC CELLS AND ZOOTECHNICAL PERFORMANCE IN ANGELFISH (PTEROPHYLLUM SCALARE)

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S1P59. ANXIETY-LIKE BEHAVIOR AND INCREASED NEURONAL ACTIVITY INDUCED BY CHRONIC STRESS AND MASTICATORY MUSCLE DYSFUNCTION

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S1P60. HEDONIC IMPLICATIONS IN NEGATIVE DISCREPANCY BETWEEN EXPECTED AND OBTAINED REWARD: AN ANIMAL MODEL FOR STUDYING FRUSTRATION

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S1P61. SELECTIVE ATTENTION AND DISTRACTIBILITY AFTER 12 WEEK OF TREATMENT WITH ALPRAZOLAM IN GENERAL ANXIETY PATIENTS USING THE CONTINUOUS PERFORMANCE TASK

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S1P62. OMEGA-3 SUPPLEMENTS ALTERS BIOMARKERS EXPRESSION IN THE AUTISM SPECTRUM DISORDER

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S1P63. USE OF STEROIDS AND THEIR EFFECTS ON AGGRESSION AND NEURON BODY CELLS DENSITY

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S1P64. USE OF STEROIDS AND THEIR EFFECTS ON BEHAVIOR AND NEURONAL QUANTIFICATION OF SWISS MICE

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S1P65. CREATION AND STANDARDIZATION OF A METHOD TO STUDY LEARNED HELPLESSNESS IN FLIES: THE PREFERENCE TEST

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S1P66. VOLUNTARY EXERCISE REDUCES THE NUMBER OF SEIZURES AND ALTERS THE BRAIN-DERIVED NEUROTROPHIC FACTOR (BDNF) AND TYROSINE KINASE B RECEPTOR (TRKB) EXPRESSIONS IN HIPPOCAMPUS OF WISTAR RATS WITH EPILEPSY

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S1P67. LET'S TALK ABOUT THE EARTH: CHILDREN LEARN ABOUT CONCEPTUAL EARTH MODELS FROM THEIR PEERS

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S1P68. OXIDATIVE STRESS IN LACTATING RATS WITH VARIATIONS IN THE MATERNAL BEHAVIOR

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S1P69. ERIODICTYOL PROMOTES NEUROPROTECTION ON NEURONAL DAMAGE, MOTOR E MEMORY DEFICITS INDUCED BY PERMANENT FOCAL BRAIN ISCHEMIA IN MICE

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S1P70. THE RELATIONSHIP BETWEEN PERCEPTUAL CLOSURE AND EXECUTIVE FUNCTIONS

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S1P71. HYPERCHOLESTEROLEMIA INDUCES BBB DISRUPTION: A COMPARISON BETWEEN C57BL/6 WILD-TYPE AND LDLR-/- MICE

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S1P72. EARLY LIFE INTERVENTIONS AFFECT MEMORY RECONSOLIDATION IN MALE RATS

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S1P73. ANALYZING FUNCTIONAL IMPAIRMENT IN ALZHEIMER DISEASE

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S1P74. EFFECTS OF FOLIC ACID SUPPLEMENTATION DURING PREGNANCY IN PUP RATS SUBMITTED OR NOT TO NEONATAL HYPOXIA-ISCHEMIA

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S1P75. CLUSTERING COEFFICIENT PREDICTS MEAN SUBJECTS' REACTION TIME

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S1P76. EFFECT OF MUSIC IN ESPACIAL MEMORY IN RODENTS

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S1P77. EFECT OF MUSICAL IMPROVISATION IN VISUAL EMOTIONAL MEMORY

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CHRONOBIOLOGY

S1P78. ROLE OF PHOSPHATASE 2A IN THE PHOTIC SIGNALING PATHWAY OF THE CIRCADIAN CLOCK

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S1P79. TEMPORAL STUDY OF DNA METHYLTRANSFERASES, BDNF AND TRKB IN HIPPOCAMPUS AND CEREBELLUM DURING AGING

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S1P80. TEMPORAL DISTRIBUTION OF TONIC-CLONIC SEIZURES IN AN ANIMAL MODEL OF TEMPORAL LOBE EPILEPSY AND INTERACTION WITH SLEEP CHRONOBIOLOGY

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S1P81. NEURONAL REDOX STATE AS A KEY MODULATOR OF THE CIRCADIAN SYNCHRONIZATION: BE CAREFUL WITH THE TIMING OF YOUR RESEARCH

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S1P82. PHOTIC AND MAGNETIC ENTRAINMENT OF CIRCADIAN LOCOMOTOR ACTIVITY BEHAVIOR IN CAENORHABDITIS ELEGANS

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S1P83. THE TIMEWORLD IN CANCER SURVIVORS. INTERTEXTUAL ANALYSIS OF THEIR EXPERIENCES WITH THE PERCEPTION OF TIME CONTRASTED WITH EXCERPTS FROM THE BOOK "EINSTEIN'S DREAMS"

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S1P84. EFFECT OF FOOD DEPRIVATION PROTOCOL IN SLEEP ARCHITECTURE IN RATS

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DEVELOPMENT

S1P85. MUTANT ALPHA-SNAP (M105I) ALTERS SUBCELLULAR DISTRIBUTION OF N-CADHERIN AND PROVOKES DISRUPTION OF EMBRYONIC BRAIN VENTRICULAR ZONE

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S1P86. NEURAL CREST-DERIVED CELLS IN THE LIVER DURING EMBRYONIC DEVELOPMENT AND IN FIBROGENESIS

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S1P87. 4'-CHLORODIAZEPAM MODULATES THE DEVELOPMENT OF PRIMARY HIPPOCAMPAL NEURONS IN A SEX-DEPENDENT MANNER

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S1P88. PROTEIN DEFICIENCY ALTERS CEREBELLAR MORPHOLOGY, BUT LOW OMEGA 6/OMEGA 3 RATIO PROTECTS AGAINST OXIDATIVE DAMAGE

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S1P89. EFFECT OF GROWTH RESTRICTION ON EARLY BRAIN DEVELOPMENT: A MRI QUANTITATIVE ASSESSMENT

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S1P90. TRANSCRIPTION FACTOR INVOLVEMENT WITHIN THE MEDIAL GANGLIONIC EMINENCE IN INTERNEURON SPECIFICATION

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S1P91. DEVELOPMENTAL ORIGIN OF NON-SEROTONERGIC NEURONS IN THE RAPHE NUCLEI

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S1P92. PERINATAL HYPOXIA AS ANIMAL MODEL OF GENE-ENVIRONMENT INTERACTION IN SCHIZOPHRENIA

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S1P93. DOWNREGULATION OF PARVALBUMIN EXPRESSION IN THE PREFRONTAL CORTEX DURING ADOLESCENCE CAUSES ENDURING PREFRONTAL DISINHIBITION IN ADULTHOOD

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S1P94. THE IMPACT OF OMEGA-3 FATTY ACIDS IN THE DEVELOPMENT OF VISUAL RETINOFUGAL CONNECTIONS

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S1P95. ASCL1 CONTROLS LATE NEURONAL SPECIFICATION OF CENTRAL CANAL NEURONS

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DISORDERS OF THE NERVOUS SYSTEM

S1P96. LEMPEL-ZIV COMPLEXITY AS A PARAMETER TO EVALUATE THE DYNAMICS OF CORTICAL ELECTRICAL ACTIVITY IN RATS EXPOSED TO GAMMA RADIATION

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S1P97. ROLE OF THE PROTEASOME IN THE INDUCTION OF ALZHEIMER'S DISEASE PATHOLOGIES IN A HUMAN CEREBRAL ORGANOID MODEL

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S1P98. IDENTIFICATION OF THE BDNF PRODOMAIN (PBDNF) AS A NEW PATHOGENIC LIGAND AFFECTING NEURONAL STRUCTURE AND FUNCTION.

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S1P99. LIMBIC NEURONAL DEGENERATION IN AN EXPERIMENTAL MODEL OF TEMPORAL LOBE EPILEPSY INDUCED BY INTRACEREBRAL APPLICATION OF PILOCARPINE

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S1P100. EVALUATION OF VISUAL ACUITY IN PATIENTS WITH DIABETES MELLITUS TYPE 2 USING MULTIFOCAL VISUAL EVOKED POTENTIAL (MFVEP)

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S1P101. THE RETINAL PHYSIOLOGY: A BIOMARKER FOR AGING AND NEURODEGENERATIVES DISEASES.

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S1P102. EPIGENETIC CONTROL MEDIATES CORTICAL STIMULATION-INDUCED ANALGESIA IN NEUROPATHIC RATS

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S1P103. MONITORING IN VIVO DOPAMINE RELEASE IN A MOUSE MODEL FOR COGNITIVE AND NEGATIVE SYMPTOMS OF SCHIZOPHRENIA

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S1P104. DIMETHYL FUMARATE (DMF) INDUCED SYNAPTIC PRESERVATION AND MICROGLIAL ACTIVATION IN CHRONIC EXERCISED EXPERIMENTAL AUTOIMMUNE ENCEPHALITIS (EAE)-MICE

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S1P105. YERBA MATE (ILEX PARAGUAIENSIS) FAVOURS DOPAMINERGIC NEURONS SURVIVAL IN CULTURE

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S1P106. EFFECT OF PARADOXICAL SLEEP DEPRIVATION (PSD) ON THE THRESHOLDS OF DEFENSIVE REACTION INDUCED BY ELECTRICAL STIMULATION OF PERIAQUEDUCTAL GRAY MATTER (PAG) AND SUPERIOR COLLICULUS (CS) OF WISTAR RATS

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S1P107. UNRAVELLING THE MOLECULAR ROLE OF FYN IN LEVODOPA INDUCED DYSKINESIA (LID)

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S1P108. EFFECTS OF COMBINED NITRIC OXIDE COMPOUNDS AND AMANTADINE ON L-DOPA-INDUCED DYSKINESIA

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S1P109. PARAWIXIN2, A GABA UPTAKE INHIBITOR ISOLATED FROM PARAWIXIA BISTRIATA SPIDER VENOM IS NEUROPROTECTIVE AGAINST EXPERIMENTAL ISCHEMIC STROKE DAMAGE IN WISTAR RATS

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S1P110. ALPHA-SNAP IS INVOLVED IN THE FORMATION/STABILIZATION OF N-CADHERIN-BASED ADHERENS JUNCTIONS AND SURVIVAL OF NEURAL STEM CELLS

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S1P111. ASSOCIATION BETWEEN POLYMORPHISMS OF ALPHA-SYNUCLEIN GENE (SNCA) AND CLINICAL ASPECTS OF PARKINSON'S DISEASE IN A BRAZILIAN SAMPLE

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S1P112. ASSOCIATION BETWEEN POLYMORPHISM OF ALPHA-SYNUCLEIN GENE (SNCA) AND CLINICAL ASPECTS OF PARKINSON´S DISEASE IN A BRAZILIAN SAMPLE

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S1P113. PEERS CAN RESCUE AUTISM-RELATED BEHAVIORS AND GLUCOSE BRAIN METABOLISM AFTER PRENATAL EXPOSURE TO VALPROIC ACID

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S1P114. DECREASE IN PHASE LOCKING BETWEEN DELTA AND GAMMA BANDS BEFORE EPILEPTIC SEIZURES

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S1P115. NEUROINFLAMMATION IN THE DESCENDING ANALGESIC PATHWAY CONTRIBUTES WITH HYPERALGESIA IN PARKINSON'S DISEASE

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S1P116. A NOVEL MECHANISM OF ALZHEIMER'S DISEASE NEURODEGENERATION:
BAG2, AB1-42 TOXICITY, AND NF-KB SIGNALING

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S1P117. INFLUENCE OF THE ESTROUS CYCLE IN THE MANIFESTATION OF SCHIZOPHRENIA-LIKE SYMPTOMS IN MICE SUBMITTED TO THE NEURODEVELOPMENTAL MODEL OF SCHIZOPHRENIA BY TWO-HIT

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S1P118. FRONTAL GAMMA-BAND ABNORMALITIES IN SUBJECTS AT CLINICAL HIGH-RISK OF PSYCHOSIS DURING A WORKING MEMORY LOAD TASK

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S1P119. TEMPOL (4 HIDROXY-TEMPO) TREATMENT REDUCES INFLAMMATION THROUGHOUT AMYOTROPHIC LATERAL SCLEROSIS DEVELOPMENT IN SOD1 G93A MICE

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S1P120. ADJUNCTIVE THERAPY WITH VITAMIN B12 IS NEUROPROTECTIVE TO THE HIPPOCAMPUS IN AN INFANT RAT MODEL OF PNEUMOCOCCAL MENINGITIS AND THIS POSITIVE EFFECT MAY INVOLVE EPIGENETIC REGULATION

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S1P121. SYNTHETIC CHALCONES WITH ACTIVITY FOR CNS TARGETS RELATED TO ANXIETY, DEPRESSION, NEURODEGENERATIVE DISEASES AND PAIN

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S1P122. INFLAMMATORY PROFILE IN HE HIPPOCAMPUS MDX MICE

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S1P123. DECREASED TBS-DEPENDENT LONG TERM POTENTIATION AND AMPA RECEPTORS PHOSPHORYLATION IN A MURINE MODEL OF ADHD INDUCED BY PRENATAL NICOTINE EXPOSURE

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S1P124. MOTOR NEURON LOSS AT THE SPINAL CORD IS ASSOCIATED TO MUSCLE ATROPHY IN BACHD MOUSE MODEL FOR HUNTINGTON'S DISEASE

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S1P125. THE MOLECULAR MOTOR KIF5B MEDIATES THE FUNCTION OF DOPAMINE D2 RECEPTORS AND IS NECESSARY FOR THE CROSS TALK BETWEEN DIRECT AND INDIRECT NIGROSTRIATAL PATHWAY IN LOCOMOTION

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S1P126. CANNABINOID SYSTEM IN AN INSULIN RESISTANCE-RELATED NEURODEGENERATION MODEL

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S1P127. INWARDLY RECTIFYING POTASSIUM CHANNELS CONTRIBUTE TO AMYLOID BETA OLIGOMER-RELATE NEUROTOXICITY

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S1P128. SPONTANEOUSLY HYPERTENSIVE RATS (SHR) ARE MORE RESISTANT THAN WISTAR RATS TO A RESERPINE-INDUCED PROGRESSIVE MODEL OF PARKINSON'S DISEASE

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S1P129. LOOKING FOR A GENETIC MOLECULAR SIGNATURE FOR ALZHEIMER'S DISEASE IN ARGENTINA

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S1P130. TAU DYSFUNCTION IN THE BASAL GANGLIA OF A MOUSE MODEL OF TAUOPATHY RELATED TO PSP

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S1P131. MODULATION OF P-ERK AND P-AKT ACTIVITY IN EXPERIMENTAL MODEL OF PARKINSON'S DISEASE INDUCED BY 6-OHDA

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S1P132. BENEFITS OF ENVIRONMENTAL ENRICHMENT ON BLOOD BRAIN BARRIER DISRUPTION INDUCED BY NEONATAL HYPOXIA-ISCHEMIA IN RATS

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S1P133. SERUM IGG FROM ALS PATIENTS INDUCED NEURONAL UPTAKE AND MICROGLIA ACTIVATION IN SPINAL CORD CULTURES

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S1P134. INHIBITION OF DOPAMINE UPTAKE AS AN ANIMAL MODEL OF MANIA: BEHAVIOURAL, PHARMACOLOGICAL AND NEUROIMMUNOLOGICAL CHARACTERIZATION

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MOLECULAR AND CELLULAR NEUROBIOLOGY

S1P135. PLASTICITY OF NEURON-PERICYTE INTERACTION MEDIATED BY P2X7 RECEPTORS AND PANNEXIN1 CHANNELS

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S1P136. STEREOLOGIC STUDY OF THE TEMPORAL EXPRESSION OF C-FOS IN THE BRAIN OF RATS

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S1P137. OUABAIN IS A NEUROIMUNOMODULATOR

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S1P138. ANGIOTENSIN II AT2 RECEPTOR LOCALIZATION AND MRNAS EXPRESSION IN THE P15 RAT INFERIOR COLLICULUS

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S1P139. BRAIN METABOLIC AND MORPHOLOGICAL ALTERATIONS IN A RAT MODEL OF HEPATIC ENCEPHALOPATHY INDUCED BY SUBTOTAL HEPATECTOMY

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S1P140. ROLE OF OXIDATIVE STRESS ON BLOOD BRAIN BARRIER PERMEABILITY

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S1P141. PARTIAL RESTITUTION OF THE LIGHT TRANSDUCTION SIGNALING CASCADE IN INSIDE-OUT PATCHES EXCISED FROM THE PHOTOSENSITIVE MEMBRANE OF DROSOPHILA PHOTORECEPTORS

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S1P142. GLUCOSE RELEASED TO THE MUCUS OF THE OLFACTORY EPITHELIUM BY THE SUSTENTACULAR CELLS IS ESSENTIAL TO POWER ODOR TRANSDUCTION IN OLFACTORY CILIA

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S1P143. MESENCHYMAL STEM CELLS CONDITIONED MEDIUM PROTECTS ASTROCYTES FROM SCRATCH ASSAY IN VITRO INJURY

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S1P144. SYNAPTIC CHANGES INDUCED BY GLUN2A KNOCKDOWN

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S1P145. BRAIN CELL COMPOSITION ANALYSIS THROUGH THE ISOTROPIC FRACTIONATOR METHOD IN A MURINE EXPERIMENT OF MATERNAL MALNUTRITION

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S1P146. EXPRESSION OF VOLTAGE-ACTIVATED POTASSIUM KCNQ CHANNELS IN MOUSE EYE

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S1P147. MECHANISTIC BASIS OF THE ROLE OF NEDD8 ON EARLY NEURONAL DEVELOPMENT

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S1P148. ALTERED GENE EXPRESSION IN FEMALE MICE HIPPOCAMPUS CORRELATES WITH DEPRESSIVE-LIKE BEHAVIOR EVOKED BY EARLY PROTEIN MALNUTRITION

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S1P149. CONSTANT LOW LED LIGHT EXPOSURE EFFECTS IN RETINAL NEURONS

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S1P150. FUNCTIONAL ANALYSIS OF THE HUMAN ACCELERATED ELEMENTS GROUPED IN THE LOCUS OF RBFOX1

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S1P151. ROLE OF MAP6D1 IN GOLGI APPARATUS ORGANIZATION AND NEURONAL POLARITY

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S1P152. IN VIVO ASSESSMENT OF CHLORIDE LEVELS AND PH IN LAYER 2/3 CORTICAL NEURONS IMPLEMENTING THE GENETICALLY ENCODED RATIOMETRIC INDICATORS SUPERCLOMELEON AND SUPERECLIPTIC PHLUORIN

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S1P153. INFLEXIBLE ETHANOL INTAKE: A PUTATIVE LINK WITH THE LRRK2 PATHWAY

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S1P154. GRANULE CELLS BORN IN MIDDLE-AGED MICE PRESENT HIGH LEVELS OF PLASTICITY

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S1P155. CHOLESTEROL EFFECTS ON MUSCLE-TYPE NICOTINIC ACETYLCHOLINE RECEPTOR DISTRIBUTION AND DYNAMICS STUDIED WITH SINGLE-MOLECULE STORM MICROSCOPY AND BAYESIAN ANALYSIS

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S1P156. HUMAN-SPECIFIC EVOLUTION OF THE TRANSCRIPTIONAL REGULATION OF FOXP2

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S1P157. PKA-DEPENDENT SODIUM-COUPLED NEUTRAL AMINO ACID TRANSPORTER 2 PHOSPHORYLATION

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S1P158. REACTIVE ASTROGLIOSIS PROPAGATION IN A MODEL OF IN VITRO SCRATCH INJURY

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S1P159. CANAVALIA BRASILIENSIS (CONBR) LECTIN REDUCES NOCICEPTIVE BEHAVIOR EVOCATED BY GLUTAMATERGIC AGONISTS NMDA AND AMPA AT SPINAL CORD LEVEL

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S1P160. SIGNALLING MECHANISM IN INJURED ASTROCYTES ACTIVATED BY NEUROTROPHINS

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S1P161. NEURAL STEM CELLS DIFFERENTIATION AND OUTGROWTH MEDIATED BY TROPHIC FACTORS ACTION INSIDE A 3D BIOASSAY OF COLLAGEN GEL

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S1P162. BLOCK OF EXPRESSION OF THE G-PROTEIN GAMMA-5 SUBUNIT SELECTIVELY DISRUPTS DE MUSCARINIC M4-MEDIATED INHIBITION OF THE N-TYPE CALCIUM CURRENT IN RAT SYMPATHETIC NEURONS

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S1P163. EVALUATION OF MITOCHONDRIAL FUNCTION IN STRIATUM AND SUBSTANTIA NIGRA FROM ATRAZINE-TREATED ANIMALS

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S1P164. CALBINDIN IMMUNOREACTIVITY OF GABAERGIC INTERNEURONS LOCALIZED IN TEMPORAL NEOCORTEX OF PATIENTS WITH RESISTANT TEMPORAL LOBE EPILEPSY AND CO-MORBID DEPRESSION

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S1P165. COMPARATIVE STUDY OF PERIPHERAL AXONS REGENERATION IN ACUTE PERIODS AFTER CRUSH OR TRANSECTION INJURY.

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S1P166. OUABAIN MODULATES IL-6 LEVELS IN MIXED RETINAL CELL CULTURES

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S1P167. FUNCTIONAL TEST OF PCDHB11, THE MOST HUMAN-SPECIFIC NEURONAL SURFACE PROTEIN

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S1P168. SUBTYPE 3 METABOTROPIC GLUTAMATE RECEPTOR-INDUCED AMYLOID-BETA CLEARANCE BY GLIAL CELLS

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S1P169. FUNCTIONAL RESCUE OF TAUOPATHY PHENOTYPES USING TAU RNA REPROGRAMMING IN VIVO

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S1P170. EFFECT OF INHIBITING CYTOPLASMATIC HISTONE DEACETILASES (HDACS) ON BEHAVIOUR AND INHIBITORY AVOIDANCE MEMORY CONSOLIDATION, IN MICE

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S1P171. HIPPOCAMPAL MICRORNA-MRNA REGULATORY NETWORK IS AFFECTED BY PHYSICAL EXERCISE

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S1P172. NMGP-1, AN ORTHOLOG OF MAMMALIAN GPM6A, MODULATES LIFESPAN AND STRESS RECOVERY IN C. ELEGANS

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S1P173. PKC ACTIVATION INCREASES RETINAL GANGLION CELLS SUVIVAL: INVOLVEMENT OF TNF-A AND APOPTOSIS INHIBITION

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S1P174. MITOCHONDRIAL BRAINSTEM DYSFUNCTION: THE LASTING EFFECTS OF A MATERNAL PROTEIN RESTRICTION IN THE BIOENERGETICS AND OXIDATIVE BALANCE

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S1P175. ROLE OF PANNEXIN-1 IN STRUCTURAL PLASTICITY IN MOUSE HIPPOCAMPUS

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S1P176. EXPRESSION OF IL-1B IN AN ANIMAL MODEL OF NEURODEGENERATION INDUCTED BY THE INTRACEREBROVENTRICULAR ADMINISTRATION OF STREPTOZOTOCIN

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S1P177. HIPPOCAMPAL NEURONS TREATED WITH NEUTRALIZING ANTIBODY AGAINST GLYCOPROTEIN M6A IMPAIRS SYNAPTOGENESIS AND SPINOGENESIS

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S1P178. A LOCAL NETWORK ACTIVATED BY EXPERIENCE ACCELERATES THE INTEGRATION OF NEW DENTATE GRANULE CELLS

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Motor Systems

S1P179. SYNCHRONIZATION BETWEEN MOTOR AND AUDITORY CORTICES WHILE LISTENING TO SYLLABLES

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S1P180. MOTOR ADJUSTMENTS RATE AFTER FOCAL AND UNILATERAL CORTICOSPINAL TRACT LESION

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S1P181. CORTICAL ACTIVITY IS SYNCHRONIZED TO VOCAL BEHAVIOR IN CANARIES

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S1P182. LEARNING CONFLICTING INFORMATION IN THE MOTOR SYSTEM: FROM ANTEROGRADE INTERFERENCE TO FACILITATION

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S1P183. BIOLOGICAL ROLES OF MICROGLIAL CELLS IN SPINAL CORD SYNAPTIC PLASTICITY AFTER PERIPHERAL NERVE INJURY

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S1P184. MICROGLIA AND ASTROGLIA: KEY CLUES FOR MOTOR RESTAURATION?

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S1P185. LEARNING TWO CONSECUTIVE CONFLICTING TASKS INFLUENCES THE ABILITY TO LEARN FROM ERROR

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S1P186. THE INCERTO-HYPOTHALAMIC AREA PROJECTIONS IN FEMALE MICE

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S1P187. THETA RHYTHM GENERATION IN LATERAL HABENULA AND ITS RELATION WITH HIPPOCAMPAL THETA

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S1P188. PROPERTIES OF THE CORTICOSTRIATAL LONG TERM DEPRESSION INDUCED BY CORTICAL HIGH FREQUENCY STIMULATION IN VIVO

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S1P189. HIGH FAT INTAKE IN A MOUSE BINGE EATING MODEL MAY INVOLVE CONSTITUTIVE GHRELIN RECEPTOR SIGNALING

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S1P190. DISTRIBUTION OF HYPOCRETINERGIC FIBERS IN MONOAMINERGIC NUCLEI OF THE CAT´S MIDBRAIN

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S1P191. NEUROANATOMICAL AND FUNCTIONAL CHARACTERIZATION OF GHRELIN RECEPTOR-EXPRESSING NEURONS OF THE NUCLEUS OF THE SOLITARY TRACT IN A TRANSGENIC MOUSE MODEL

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S1P192. CHANCES IN THE POWER AMPLITUDE CAUSED VISUAL EVOKED POTENCIAL EEG BANDS AFTER ADAPTATION IN LUMINACE FLICKER

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S1P193. INCREASED LITTER SIZE ENHANCES THE NUMBER OF MELANIN-CONCENTRATING HORMONE NEURONS IN THE MEDIAL PREOPTIC AREA OF LACTATING DAMS

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S1P194. AEROBIC EXERCISE TRAINING IMPROVES CARDIOVASCULAR HEMODYNAMIC PARAMETERS OF HYPERTENSIVE RATS: INVOLVEMENT OF MICROGLIAL CELLS, GABAERGIC AND ENDOCANNABINOID NEUROTRANSMISSIONS

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S1P195. ABNORMAL EXPRESSION OF NA+/K+ ATPASE A-SUBUNITS IN HIPPOCAMPUS OF PILOCARPINE-TREATED EPILEPTIC RATS

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Neurochemistry and Neuropharmacology

S1P196. ROLE OF MINERALOCORTICOID RECEPTORS OF MEDIAL PRE FRONTAL CORTEX ON TOLERANCE TO THE EFFECTS OF MIDAZOLAM IN MICE SUBMITTED TO TEST AND RETEST IN THE ELEVATED PLUS-MAZE

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S1P197. BRAIN CATALASE LEVELS IN DEVELOPMENTALLY-LEAD-EXPOSED RATS ADMINISTERED WITH A SHRNA ANTICATALASE LENTIVIRAL VECTOR IN THE VENTRAL TEGMENTAL AREA

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S1P198. CEREBRAL MALARIA INDUCES ELECTROPHYSIOLOGICAL AND NEUROCHEMICAL IMPAIRMENT IN RETINAL TISSUE: POSSIBLE EFFECT ON THE GSH AND GLUTAMATERGIC SYSTEM

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S1P199. THE EXPRESSION OF CONTEXTUAL FEAR CONDITIONING INVOLVES ACH RELEASE AND NMDA GLUTAMATERGIC RECEPTORS ACTIVATION IN THE DORSAL HIPPOCAMPUS OF RATS

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S1P200. NEUROPROTECTIVE EFFECT OF MELATONIN ON A NEUROINFLAMMATION MODEL OF THE VISUAL PATHWAY

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S1P201. EFFECTS OF NOCICEPTIN/ORPHANIN FQ RECEPTOR PARTIAL AGONISTS IN MOUSE MODELS OF ANXIETY AND DEPRESSION

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S1P202. MINOCYCLINE PREVENTS CROSS-SENSITIZATION BETWEEN STRESS AND COCAINE AND THE INCREASED PRODUCTION OF PROINFLAMMATORY CYTOKINES INDUCED BY CHRONIC RESTRAINT STRESS

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S1P203. OSMOTIC INDUCTION OF GLUTAMATE/ASPARTATE TRANSPORTER (GLAST/EAAT1) AND ANGIOGENIC FACTOR EXPRESSION IN RETINAL GLIAL CELLS: DIFFERENTIAL DEPENDENCE ON GROWTH FACTOR AND PURINERGIC RECEPTOR SIGNALING

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S1P204. POTENTIAL ANTIDEPRESSANT-LIKE EFFECT OF P-COUMARIC ACID AND THE INVOLVEMENT OF MONOAMINERGIC SYSTEM

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S1P205. RED PROPOLIS EXTRACT AND SWIMMING EXERCISE ARE NEUROPROTECTIVE AFTER SPINAL CORD INJURY IN RATS

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S1P206. MODULATION OF GABAARHO1 RECEPTORS BY HISTAMINE

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S1P207. ROLE OF ORBITOFRONTAL CORTEX IN CONTEXT-INDUCED REINSTATEMENT OF ALCOHOL-SEEKING IN RATS

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S1P208. MATERNAL SEPARATION ALTERS IMPULSIVITY, VOLUNTARY ETHANOL DRINKING AND ENDOCANNABINOID AND DOPAMINERGIC EXPRESSION: INFLUENCE OF PREVIOUS ETHANOL BINGE DRINKING

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S1P209. SIMVASTATIN NANOCAPSULES PROTECT FROM AGE-INDUCED MEMORY IMPAIRMENT AND ANXIETY IN RATS

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S1P210. ER STRESS PROMOTION BY BORTEZOMIB TREATMENT IS ASSOCIATED WITH R-CRT PRO-APOPTOTIC ACTION IN HUMAN GLIOMA CELLS

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S1P211. EFFECTS OF THE SYNTHETIC CANNABINOID (WIN 55212-2) AND HEMOPRESSIN ON RODENT CORTICAL CULTURED MIXED NEURON-ASTROCYTE CELLS

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S1P212. ACUTE AND SUB-CHRONIC VITAMIN D TREATMENT EXERTS ANTIDEPRESSANT-LIKE EFFECT IN MICE

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S1P213. INVOLVEMENT OF MONOAMINERGIC SYSTEM IN ANTIDEPRESSANT-LIKE ACTIVITY OF LQFM180, A NEW PIPERAZINE DERIVATIVE

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S1P214. ANTIDEPRESSANT-LIKE EFFECTS OF NOCICEPTIN/ORPHANIN FQ RECEPTOR ANTAGONISTS IN THE LEARNED HELPLESSNESS MODEL IN MICE

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S1P215. STRATEGIES FOR ALZHEIMER'S DISEASE PREVENTION: PEGYLATED BIODEGRADABLE DEXIBUPROFEN NANOSPHERES ADMINISTRATION TO APPSWE/PS1DE9

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S1P216. SHORT-TERM TREATMENT WITH CANNABINOIDS, BUT NOT IMIPRAMINE, PREVENTS STRESS-INDUCED BEHAVIORAL CHANGES IN MICE

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S1P217. NA,K-ATPASE FUNCTION AND NOS-CGMP SIGNALING IN KLOTHO MUTANT MICE: DIFFERENTIAL ALTERATIONS IN HIPPOCAMPUS AND CEREBELLUM

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S1P218. RECRUITMENT OF NONVISUAL ARRESTINS TO THE ACTIVATED M2 MUSCARINIC ACETYLCHOLINE RECEPTOR. FACILITATION OF RECEPTOR/ARRESTIN INTERACTION BY GRK2

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S1P219. EFFECTS OF MONOCROTALINE, ISOLATED FROM CROTALARIA RETUSA, ON AMINO ACID CONCENTRATIONS IN THE BRAIN OF MICE

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S1P220. CHRONIC EXPOSURE TO FLUOXETINE DURING PRE-PUBERTY IMPAIRS RAT SOCIAL INTERACTION

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S1P221. INVOLVEMENT OF N-METHYL-D-ASPARTATE RECEPTORS (NMDAR) SUBUNITS IN THE NEUROPROTECTIVE EFFECT OF ATORVASTATIN AGAINST GLUTAMATE-INDUCED TOXICITY

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S1P222. EFFECT OF LITHIUM IN A MANIA MODEL INDUCED BY PARADOXICAL SLEEP DEPRIVATION COMBINED WITH UNPREDICTABLE STRESS IN RATS

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S1P223. EVALUATION OF ANTICONVULSANT EFFECTS OF IVABRADINE IN PENTYLENETETRAZOLE-INDUCED CONVULSIONS IN MICE

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S1P224. SIMVASTATIN NANOCAPSULES AMELIORATES MEMORY IMPAIRMENT IN YOUNG-ADULT RATS

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S1P225. CONTROL OF CARDIOVASCULAR RESPONSES TO ACUTE RESTRAINT STRESS BY CRF IN THE BED NUCLEUS OF THE STRIA TERMINALIS IS MEDIATED BY ACTIVATION OF LOCAL NNOS ENZYME

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S1P226. CORRELATION OF SPASTICITY WITH FUNCTIONALITY OF ESPÁSTICOS INDIVIDUALS AFTER PHYSIOTHERAPY TREATMENT ASSOCIATED WITH THE MEDICINE ZICLAGUE®

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S1P227. NEUROPROTECTIVE EFFECT OF AFRICANIZED BEE (APIS MELLIFERA) VENOM ON THE NEURONAL DEATH INDUCED BY 6-HYDROXYDOPAMINE

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S1P228. ANGIOTENSIN II TYPE 1 (AT1) RECEPTOR BLOCKER DISPLAY AN IMPORTANT ROLE IN BEHAVIORAL ALTERATIONS OBSERVED IN A MICE MODEL OF AGING AND PARKINSON'S DISEASE

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S1P229. INTERACTIONS BETWEEN CRF AND NMDA GLUTAMATE RECEPTORS IN THE BED NUCLEUS OF STRIA TERMINALIS (BNST) ON MODULATION OF CARDIOVASCULAR RESPONSES TO ACUTE RESTRAINT STRESS IN RATS

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S1P230. ENDOVANILLOID SYSTEM AND TRPV1 CHANNEL IN THE PRELIMBIC CORTEX PLAYS ROLE IN THE MAINTENANCE OF NEUROPATHIC PAIN

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S1P231. PROTECTIVE EFFECT OF EUGENIA INVOLUCRATA ON THE EFFECTS CAUSED BY ALLOXAN-INDUCED DIABETES ON OXIDATIVE STRESS PARAMETERS IN THE CEREBRAL CORTEX OF RATS

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S1P232. IN VITRO ALTERATION OF ENERGY METABOLISM BY CLASSICAL GALACTOSEMIA IN CEREBRUM OF RATS

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S1P233. BDNF ISOFORMS: A ROUND-TRIP TICKET BETWEEN NEUROGENESIS AND SEROTONIN?

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S1P234. ROFLUMILAST, A PHOPHODIESTERASE-4 INHIBITOR, ATTENUATES MEMORY IMPAIRMENTS IN AGED RATS WITH CHRONIC CEREBRAL HYPOPERFUSION

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Neuroendocrinology and Neuroimmunology

S1P235. THE EFFECT OF PHARMACOLOGICAL REDUCTION OF PROLACTIN IN THE RESPONSIVENESS PARENTAL OF MALE MARMOSETS

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S1P236. THE INFLUENCE OF ENVIRONMENTAL STRESS IN THE HYPOTHALAMIC-PITUITARY-ADRENAL AXIS ACTIVITY IN BREEDING PAIRS OF MARMOSETS

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S1P237. MINERALOCORTICOID RECEPTOR (MR) AND NEUROINFLAMMATION IN THE HIPPOCAMPUS OF SPONTANEOUSLY HYPERTENSIVE RATS (SHR)

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S1P238. IMPACT OF THE GHRELIN SIGNALING IN THE RESPONSE OF THE HYPOTHALAMIC–PITUITARY–ADRENAL AXIS TO FASTING

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S1P239. ACTIVATION OF ERK1/2 AND AXOGENESIS INDUCED BY ESTRADIOL DEPEND ON DIFFERENT CALCIUM POOLS IN MALE RAT HYPOTHALAMIC

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S1P240. HYPERCALORIC DIETS DURING PREGNANCY AND LACTATION MODULATE DEPRESSION-LIKE BEHAVIOR IN FEMALE OFFSPRING OF RATS

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S1P241. ROLE OF TNF-ALPHA IN MICROGLIA-DEPENDENT PLASTICITY INDUCED BY MONOCULAR ENUCLEATION

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S1P242. ROLE OF X-LINKED GENES ON SEX DIFFERENCES IN NEUROGENIN 3 EXPRESSION IN DEVELOPING HYPOTHALAMIC NEURONS

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S1P243. IS THE DA1 RECEPTOR OF THE PREOPTIC AREA NEURONS INVOLVED ON THE REGULATION OF OVULATION?

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S1P244. NEURAL MODULATION OF STRESS RESPONSE IN C. ELEGANS

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S1P245. IMPACT OF THE GHRELIN SIGNALING ON FOOD INTAKE AFTER A FASTING EVENT

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S1P246. THE ROLE OF MINOCYCLINE IN COGNITIVE IMPAIRMENT AND DYSFUNCTION OF THE BLOOD BRAIN BARRIER IN EXPERIMENTAL PNEUMOCOCCAL MENINGITIS

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Sensory Systems

S1P247. POLARIZATION VISION IN GOLDFISH: BEHAVIORAL RESPONSE DEPENDENCE WITH STIMULUS POSITION

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S1P248. CAROTID NERVE SINUS AND IDENTIFIED PETROSAL CHEMOSENSORY NEURONS RESPONSES ARE MODIFIED BY CHRONIC PHENYTOIN TREATMENT IN THE RAT

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S1P249. CHROMATIC AND ACHROMATIC MECHANISMS ON MULTIFOCAL VISUAL EVOKED POTENTIAL (MFVEP)

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S1P250. STEM/PROGENITOR CELL THERAPY DECREASES GLIAL REACTIVITY IN LIGHT INDUCED RETINAL DEGENERATION

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S1P251. FACE PERCEPTION: TOP DOWN VS. BOTTOM UP. COMPARED FACE ATTENTIONAL CAPTURE BETWEEN FACES AND WHOLE HUMAN BODIES BY OCULAR MOVEMENT REGISTRATION DURING NATURALISTIC SCENES EXPLORATION

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S1P252. THE MAGNITUDE OF ACOUSTIC INJURY TO THE INNER EAR IS INVERSELY CORRELATED WITH ALPHA9ALPHA10 NACHR ACTIVITY

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S2P297. KINETICS OF ENVIRONMENTAL ENRICHMENT – CHARACTERIZING THE INTRINSIC DYNAMICS OF ONE OF THE MOST USED EARLY STIMULATION MODEL IN RODENTS

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S2P298. INFLUENCE OF THE CIRCADIAN CLOCK IN OVIPOSITION AND PLACE PREFERENCES OF THE FRUIT FLY DROSOPHILA MELANOGASTER

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S2P299. EXPERIMENTAL CHILDHOOD CEREBRAL MALARIA CAUSES COGNITIVE IMPAIRMENT IN ADULTHOOD

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S2P300. ROLE OF HIPPOCAMPUS DURING OBSERVATIONAL LEARNING OF SPATIAL NAVIGATION TASKS

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S2P301. LACK OF PANNEXIN 1 ALTERS LONG-TERM DEPRESSION AND SPATIAL MEMORY FLEXIBILITY

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S2P302. UNDERSTANDING MEMORY LOSS: DEVELOPMENT OF A RETRIEVAL-INDUCED FORGETTING PARADIGM IN RODENTS TO MODEL ADAPTIVE FORGETTING IN THE MAMMALIAN BRAIN

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S2P303. MINOCYCLINE IMPROVES THE MEMORY AND REDUCES THE NEUROINFLAMMATION OF MICE SUBJECT TO AMYLOID B (1-42) PEPTIDE ADMINISTRATION

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S2P304. LOCAL ADMINISTRATION OF HALOPERIDOL INTO GLOBUS PALLIDUS INDUCES ANXIETY IN RAT

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S2P305. MULTIGENERATIONAL EFFECTS OF PROTEIN MALNUTRITION: MATERNAL CARE AND OFFSPRING DEVELOPMENT

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S2P306. REST INCREASE HELPS TO MAINTAIN COGNITIVE FUNCTION IN A MILD COGNITIVE IMPAIRMENT OF ALZHEIMER'S DISEASE MODEL

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S2P307. THE USE OF COGNITIVE REHABILITATION FOR OCCUPATIONAL THERAPY (OT) IN PARKINSON'S DISEASE (PD) IN A DOMICILIARY PATIENT

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S2P308. EFFECTS OF COCAINE PYROLISIS PRODUCT, ANHYDROECGONINE METHYL ESTER (AEME) ON SPATIAL WORKING MEMORY OF RATS

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S2P309. EFFECT OF STRESS INDUCED BY IMMOBILIZATION ON THE SLEEP ARCHITECTURE OF WISTAR RATS

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S2P310. EVALUATION OF LITHIUM AND MEMANTINE EFFECT ON MEMORY AND SPATIAL NEUROINFLAMMATION IN A MODEL OF DEMENTIA ANIMAL INDUCED BY PEPTIDE B-AMYLOID1-42

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S2P311. THE APPARENT RESISTANT MEMORIES IN THE CRAB NEOHELICE DEPEND ON THE REMINDER CONDITIONS TO ENTER THE LABILIZATION/ RECONSOLIDATION PROCESS

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S2P312. ACUTE STRESS IN NEOHELICE GRANULATA IMPAIRS MEMORY RETRIEVAL

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S2P313. METHAMPHETAMINE AND MODAFINIL EFFECTS ON EPIGENETIC AND FUNCTIONAL MARKERS IN THE MOUSE PREFRONTAL CORTEX

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S2P314. REPRESENTATION OF THE STATE OF MOTION IN THE LOCAL FIELD POTENTIAL OF HIPPOCAMPUS AND ENTORHINAL CORTEX

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S2P315. EFFECT OF A HIGH FAT DIET AND OMEGA-3 SUPPLEMENTATION ON THE OBJECT RECOGNITION TASK

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S2P316. CANNABIDIOL ATTENUATES SOCIAL ISOLATION-INDUCED AGRESSION IN MICE VIA 5-HT1A AND CB1 RECEPTORS

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S2P317. STEREOLOGICAL ESTIMATE OF NEUROGENESIS RATES IN THE HIPPOCAMPAL FORMATION OF THE SPOTTED SANDPIPER (ACTITIS MACULARIA)

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S2P318. PERCEPTUAL FADING OF SYNTHESIZED NATURALISTIC VISUAL TEXTURES: A PARAMETRIC STUDY

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S2P319. HABENULAR ELECTRICAL STIMULATION EFFECTS ON THE MODULATION OF EMOTIONAL RESPONSES ON WISTAR RATS

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S2P320. DIFFERENT TYPES OF INHIBITORY CONTROL IN PATIENTS WITH PSYCHOSTIMULANT DEPENDENCE

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S2P321. MEMORY DEFICITS IN 13 MONTH OLD WILD TYPE AND TRANSGENIC MCGILL-R-THY1-APP RATS WITH BRAIN AMYLOIDOSIS

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S2P322. 'CONSOLIDATION EXPRESS': ACCELERATED DYNAMICS OF MEMORY CONSOLIDATION FOR NOVEL WORDS AND MEANINGS REVEALED BY AN ERP STUDY

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S2P323. SELECTIVE AND DIVIDED ATTENTION FOR BIMODAL STIMULI IN CHILDREN WITH MUSICAL TRAINING, WORK IN PROGRESS

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S2P324. PRENATAL ZINC TREATMENT PREVENTS AUTISTIC-LIKE BEHAVIORS IN A RAT MODEL OF AUTISM INDUCED BY PRENATAL LIPOPOLYSACCHARIDE

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S2P325. INTRAHIPPOCAMPAL ADMINISTRATION OF AN ANTIBODY AGAINST GLYCOPROTEIN M6A IMPAIRS MEMORY CONSOLIDATION IN AN INHIBITORY AVOIDANCE TASK IN MICE

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S2P326. RECONSOLIDATION-INDUCED MEMORY PERSISTENCE: PARTICIPATION OF LATE PHASE HIPPOCAMPAL ERK ACTIVATION

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S2P327. IMPAIRMENT OF SPATIAL WORKING MEMORY AND BIOCHEMICAL CHANGES INDUCED BY DIRECT CRACK INHALATION IN RATS

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S2P328. PROMOTION OR IMPAIRMENT OF LONG TERM MEMORY MEDIATED BY STRESS

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S2P329. COMPARATIVE ANALYSIS OF THE EFFECTS NICOTINE EXPOSURE AND WITHDRAWAL IN ADOLESCENCE OR ADULTHOOD ON THE BEHAVIORS ASSOCIATED WITH ANXIETY AND LOCOMOTOR ACTIVITY IN MICE

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S2P330. THE ROLE OF LIM KINASE ACTIVITY IN THE HIPPOCAMPUS DURING MEMORY FORMATION AND RECONSOLIDATION: A MATTER OF ACTIN CYTOSKELETON REORGANIZATION

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S2P331. EMOTIONAL UPREGULATION TO UNCONDITIONED, BUT NOT CONDITIONED, FEAR STIMULI DURING THE LATE DIESTROUS PHASE OF THE ESTROUS CYCLE IN RATS

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S2P332. EVALUATION OF THE POSSIBLE TRANSGENERATIONAL CHANGES OF MATERNAL SEPARATION ON MATERNAL BEHAVIOR.

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S2P333. IRE1/XBP1 PATHWAY: DIFFERENTIAL ROLE IN LEARNING AND MEMORY PROCESSES.

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S2P334. DIFFERENTIAL BRAIN EXPRESSION OF CATALASE IN DEVELOPMENTALLY-PB-EXPOSED RATS THAT HAVE VOLUNTARILY CONSUMED ETHANOL

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S2P335. MEMORY-RELATED NEURAL PLASTICITY IN THE HEMIELLIPSOID BODIES, THE CRAB'S "MUSHROOM BODIES"

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S2P336. ROLE OF ADF/COFILIN AS A KEY REGULATOR OF ACTIN CYTOSKELETON DYNAMICS IN DIFFERENT PHASES OF FEAR MEMORY IN MICE

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S2P337. EFFECTS OF DIFFERENT NICOTINE ADMINISTRATION PROTOCOLS ON MOTOR AND COGNITIVE RESPONSES IN PARKINSONIAN RATS

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S2P338. ROLE OF GLUCAGON-LIKE PEPTIDE 1 (GLP1) IN HEDONIC FOOD INTAKE IS MODULATED BY INDIVIDUAL PREFERENCE.

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S2P339. BEHAVIORAL EVALUATION OF ACUPUNCTURE IN AN ANIMAL MODEL OF ADHD

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S2P340. DIFFERENT NEONATAL STRESS PROTOCOLS CAUSE DIFFERENT ANXIETY BEHAVIORS

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S2P341. HIPPOCAMPAL-RELATED BEHAVIORAL ALTERATIONS FOUND IN NOISE-EXPOSED ADOLESCENT RATS. EFFECTS OF A SUBSEQUENT ALCOHOL INTAKE

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S2P342. PREFRONTAL CORTEX DYSFUNCTION IN HYPOXIC-ISCHEMIC ENCEPHALOPATHY CONTRIBUTES TO EXECUTIVE FUNCTION IMPAIRMENTS IN RATS – POTENTIAL CONTRIBUTION FOR ATTENTION-DEFICIT/ HYPERACTIVITY DISORDER

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S2P343. ACTIVITY REGULATED CYTOSKELETON-ASSOCIATED PROTEIN ISREQUIRED FOR CONSOLIDATION OF OVERLAPPING OBJECT, BUT NOT SPATIAL MEMORIES IN THE PERIRHINAL CORTEX

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S2P344. N-METHYL-D-ASPARTATE INJECTED INTO THE DORSAL PERIAQUEDUCTAL GRAY INDUCES EMOTIONAL SENSITIZATION AND FACILITATES THE ACQUISITION OF CONTEXTUAL FEAR MEMORY: OLFACTORY MODULATION

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S2P345. FIRST EVIDENCE OF BEHAVIORAL TAGGING ACTING IN MEMORY RECONSOLIDATION

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S2P346. VIRTUAL REALITY-BASED PHYSICAL EXERCISE WITH EXERGAMES (PHYSEX) IMPROVES MENTAL HEALTH AND PHYSICAL PERFORMANCE OF INSTITUTIONALIZED OLDER ADULTS: A SINGLE-BLINDED, RANDOMIZED AND CONTROLLED STUDY

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S2P347. AGONISTIC BEHAVIOR CHANGES IN A CICHLID FISH FED WITH AN L-TRYPTOPHAN SUPPLEMENTED-DIET

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S2P348. MODAFINIL DOES NOT PREVENT DEFICITS ON MULTIPLE TRIAL INHIBITORY AVOIDANCE TASK ACQUISITION IN SPONTANEOUSLY HYPERTENSIVE RATS (SHRS)

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S2P349. THE INTERPLAY BETWEEN MEDIAL PREFRONTAL CORTEX AND HIPPOCAMPUS IS REQUIRED FOR THE CONTROL OF MEMORY TRACES REACTIVATION IN THE PERIRHINAL CORTEX

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S2P350. OVER TIME ZEBRAFISH BEHAVIOUR FOLLOWING AYAHUASCA ADMINISTRATION

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S2P351. EFFECTS OF ACUTE AYAHUASCA AND FLUOXETINE IN ALCOHOL WITHDRAWAL

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S2P352. CONTEXTUAL FEAR MEMORY AND INCENTIVE LEARNING INTERACTIONS: BIDIRECTIONAL FREEZING MODULATION

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S2P353. NEURAL CORRELATES OF SPATIAL NAVIGATION IN THE MEDIAL-PREFRONTAL CORTEX OF MICE DURING ACQUISITION OF THE REFERENCE MEMORY

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S2P354. IMPOVERISHED ENVIRONMENT IMPAIRS BALB/C MICE EPISODIC-LIKE MEMORY

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S2P355. CHARACTERIZATION OF TWO TRIAL LONG TERM MEMORY IN THE CRAB NEOHELICE GRANULATA

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S2P356. DO PICTORIAL HEALTH WARNINGS CURB THE DRIVE TOWARDS CIGARETTE PACKS? A BEHAVIORAL ASSESSMENT

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Chronobiology

S2P357. EFFECT OF AN I.C.V INJECTION OF AGGREGATED BETA-AMYLOID (1-42) ON DAILY RHYTHMS OF OXIDATIVE STRESS PARAMETERS IN THE HIPPOCAMPUS, PREFRONTAL AND TEMPORAL CORTEX

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S2P358. ASSESSING FUNCTIONAL AND STRUCTURAL WIRING WITHIN THE CLOCK: CONTRIBUTION OF DORSAL LATERAL NEURONS TO THE CIRCADIAN PACEMAKER OF DROSOPHILA

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S2P359. REM SLEEP BEHAVIOR DISORDER: PHASIC AND TONIC MUSCULAR EVENTS ANALYSIS AND DESCRIPTION BY COEFFICIENT OF VARIATION OF THE ENVELOPE OF SIGNAL

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S2P360. SYNAPTIC PLASTICITY FOLLOWING SLEEP DEPRIVATION IN THE FRUIT FLY DROSOPHILA MELANOGASTER

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S2P361. GLYCINERGIC TRANSMISSION IN THE CIRCADIAN NETWORK: A TIME-OF-DAY DEPENDENT SWITCH

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S2P362. ZZ, A SMARTPHONE APP TO MONITOR LEG MOVEMENT DURING SLEEP

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S2P363. PIGMENT DISPERSING FACTOR (PDF) PLAYS A FUNDAMENTAL ROLE IN THE STRUCTURAL PLASTICITY OF CLOCK NEURONS IN DROSOPHILA MELANOGASTER

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Development

S2P364. BRAIN MORPHOGENESIS AND POSTNATAL CELL PROLIFERATION IN THE BASAL TELEOST MORMYRUS RUME PROBOCIROSTRIS

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S2P365. NPAS3 TRANSCRIPTION FACTOR IS ESSENTIAL FOR NERVOUS SYSTEM AND CRANIOFACIAL DEVELOPMENT

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S2P366. SEXUAL DIMORPHISM IN RENAL ANGIOTENSIN RECEPTORS GENE EXPRESSION: SEX CHROMOSOME COMPLEMENT INVOLVEMENT

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S2P367. TRANSCRIPTION REGULATION OF THE DIFFERENTIATION OF CSF-CONTACTING NEURONS IN THE SPINAL CORD

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S2P368. HIDE AND SEEK IN DROSOPHILA LARVAE: CHARACTERIZING A NUCLEAR PROTEIN WITH MITOCHONDRIAL FUNCTION

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S2P369. ROLE OF ALPHA-SNAP IN THE DEVELOPMENT OF THE CEREBELLUM

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S2P370. ANALYSIS OF DENDRITIC MORPHOLOGY OF THE CA3 PYRAMIDAL-NEURONS OF THE HIPPOCAMPUS IN ADULT CYCLICAL RATS IN PHASES OF ESTROUS AND DIESTROUS 1

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S2P371. OLFACTORY LEARNING IN THE RAT AND ITS EFFECT ON CONSUMATORY RESPONSES IN EARLY STAGES OF ONTOGENY

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S2P372. BRAIN CONNECTIVITY PREDICTS PERFORMANCE IN DIFFERENT DOMAINS OF COGNITIVE FUNCTION IN PRESCHOOLERS

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S2P373. CHRONIC HIGH-FAT DIET AND MOTIVATION TO EAT DIFFERENT PALATABLE FOODS: NO EFFECT ON LIKING BUT REDUCED WANTING

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S2P374. EFFECT OF INTRAVITREAL TREATMENT WITH IL-4 OR IL-6 ON GLUTAMATERGIC NMDA RECEPTOR IN THE VISUAL SYSTEM

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Disorders of the Nervous System

S2P375. NEUROPROTECTIVE EFFECT OF PARAWIXIN 10 (PWX10) IN WISTAR RATS SUBMITTED TO EXPERIMENTAL GLAUCOMA

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S2P376. INFLAMMATION ASSOCIATED WITH DEMYELINATION ALTERS SUBCORTICAL VISUAL CIRCUITS

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S2P377. BRAINSTEM CIRCUITS FOR MOTOR CONTROL IN HEALTH AND DISEASE

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S2P378. EXPERIMENTAL HYPERTENSION INCREASES SPONTANEOUS INTRACEREBRAL HEMORRHAGES IN A MOUSE MODEL OF CEREBRAL AMYLOIDOSIS

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S2P379. BRAIN INFLAMMATION AND DEFECTIVE INSULIN SIGNALING ARE ASSOCIATED WITH TRANSIENT COGNITIVE IMPAIRMENT IN POST-SEPTIC MICE

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S2P380. P2X7 ANTAGONIST, BBG, PROTECTS HEMIPARKINSONIAN RATS FROM L-DOPA-INDUCED DYSKINESIA

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S2P381. SOCIAL ISOLATION DURING ADOLESCENCE INDUCED LONG TERM EFFECTS ON COCAINE-INDUCED SENSITIZATION: ROLE OF WNT/B-CATENIN PATHWAY

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S2P382. CAFFEIC ACID EFFECTS ON U87MG GLIOMA CELLS' VIABILITY AND OXIDATIVE STRESS

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S2P383. THE EFFECT MOTOR OF EXERCISE IN 6-OHDA MODEL OF PARKINSON DISEASE

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S2P384. CHARACTERIZATION OF STRIATAL TYROSINE HYDROXYLASE-IMMUNOREACTIVE (TH-IR) CELLS IN CHRONIC AND ACUTE L-DOPA TREATMENT IN A MICE MODEL OF PARKINSON´S DISEASE

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S2P385. INSTRASTRIATAL CHROMOSPHERE GRAFTS REDUCES MECHANICAL ALLODYNIA IN A RAT MODEL OF PARKINSON'S DISEASE

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S2P386. BUMETANIDE ENHANCES THE ANTIEPILEPTIC EFFECT OF CONVENTIONAL DRUGS IN ANIMAL MODEL OF EPILEPSY

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S2P387. HIPPOCAMPAL SYNAPTIC IMPAIRMENTS IN HETEROZYGOUS MICE BEARING A CENTRONUCLEAR MYOPATHY-CAUSING DYNAMIN-2 MUTATION

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S2P388. REPURPOSING TETRACYCLINES FOR TREATMENT OF SYNUCLEINOPATHIES

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S2P389. EXPRESSION PATTERN OF SEMA 3D AND ITS RECEPTORS IN THE NORMAL AND REGENERATING OPTIC NERVE OF ADULT MICE

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S2P390. INVOLVEMENT OF A1 AND A2A ADENOSINE RECEPTORS IN THE RAT LINE SELECTED FOR HIGH AND LOW ANXIETY-LIKE RESPONSE

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S2P391. EFFECTS OF ROYAL JELLY ON NEUROPROTECTION, OXIDATIVE STRESS AND COGNITION IN A RAT MODEL OF SPORADIC ALZEIMER'S DISEASE

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S2P392. INFLUENCE AND EFFECTIVITY OF MOBILE DEVICES IN PATIENTS WITH AUTISM SPECTRUM DISORDERS: AN EXPERIENCE OF THERAPEUTICAL DEVELOPMENT

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S2P393. NEUROPROTECTIVE ROLE OF PALMITOYLETHANOLAMIDE IN A RAT MODEL OF PERINATAL HYPOXIA-ISCHEMIA

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S2P394. THE HYH (M105I) MUTATION OF ALPHA-SNAP ALTERS ITS PROTEIN- AND LIPID-BINDING PROPERTIES IN THE DEVELOPING CENTRAL NERVOUS SYSTEM

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S2P395. NEONATAL ANOXIA: VALIDATION OF AN ANIMAL MODEL TO STUDY THE METABOLIC PARAMETERS AFFECTED BY THIS STIMULUS IN LEPRBEGFP MICE

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S2P396. HIPPOCAMPAL CHRONIC CHANGES AFTER SPINAL CORD INJURY: GLIAL REACTIVITY AND NEUROGENESIS REDUCTION

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S2P397. NEONATAL ANOXIA IN MALE AND FEMALE RATS: EVALUATION OF SOMATIC AND SENSORY-MOTOR DEVELOPMENT AND SPATIAL MEMORY IN ADOLESCENCE

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S2P398. THE DENTATE GYRUS GRANULE CELLS (DGGC) IN THE HIPPOCAMPUS HAVE ALTERED EXCITABILITY IN A MODEL OF AUTISM INDUCED BY VALPROIC ACID (VPA)

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S2P399. PARAWIXIN10 IS NEUROPROTECTIVE AGAINST ISCHEMIC BRAIN DAMAGE IN CONSCIOUS RATS SUBMITTED TO EXPERIMENTAL STROKE

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S2P400. TRANSCRANIAL DIRECT-CURRENT STIMULATION (TDCS) AS AN ALTERNATIVE FOR THE TREATMENT OF NEUROPATHIC PAIN

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S2P401. INFLUENCE OF DIFFERENT TYPES OF EXERCISE ON FEMALE RATS SUBMITTED THE PILOCARPINE EPILESY MODEL

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S2P402. LOW DOSE OF AMYLOID PEPTIDES CAUSES DEFICIT ON SPATIAL MEMORY BUT NOT ON EMOTIONAL MEMORY IN AGED WISTAR RATS: AN APPROACH TO MODEL INITIAL ALZHEIMER?

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S2P403. SPINAL GLIAL MODULATION OF NEUROPATHIC PAIN IN RATS BY MANUAL ACUPUNCTURE

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S2P404. BUMETANIDE ENHANCES THE PHARMACOLOGICAL EFFECT OF PHENOBARBITAL, IN AN ANIMAL MODEL OF TEMPORAL LOBE EPILEPSY

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S2P405. ENERGY-DENSE DIET WORSENS EARLY COGNITIVE IMPAIRMENT THROUGH DYSREGULATION OF NEUROPROTECTIVE PATHWAYS AND PYROGLUTAMATE-AMYLOID BETA GENERATION: EVIDENCE FROM A TRANSGENIC ALZHEIMER RAT MODEL

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S2P406. SELECTIVE ABLATION OF CHOLINERGIC INTERNEURONS IN THE STRIATUM RESULTS IN REPETITIVE RITUALISTIC-LIKE BEHAVIORS

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S2P407. VISUAL SALIENCY AND FREE EXPLORATION IN PEOPLE AFFECTED WITH SCHIZOPHRENIA

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S2P408. ROLE OF ASICS CHANNELS AFTER EXCITOTOXIC DAMAGE IN A MODEL OF SPINAL CORD INJURY

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S2P409. SEX AND HORMONAL INFLUENCE IN EMOTIONAL MEMORY AND SOCIAL BEHAVIOR IN A RAT MODEL OF ALZHEIMER´S DISEASE

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S2P410. INVOLVEMENT OF NOCICEPTIN/ORPHANIN FQ RECEPTOR SIGNALING ON MODULATION OF LIPOPOLYSACCHARIDE-INDUCED DEPRESSIVE-LIKE BEHAVIOR IN MICE

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S2P411. CURCUMIN INFLUENCES FUNCTIONAL RECOVERY OF RATS SUBMITTED TO ACUTE SPINAL CORD HEMI-SECTION

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S2P412. ION CONDUCTANCE MANIPULATION OF THALAMOCORTICAL NEURONS WITH DYNAMIC CLAMP INDUCES OSCILLATORY ACTIVITY RELATED TO DEEP SLEEP AND EPILEPTIC SEIZURES

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S2P413. MEMORY ALTERATIONS IN A MOUSE MODEL OF AUTISM

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Molecular and Cellular Neurobiology

S2P414. STUDY OF SELECTIVE TYROSINE OXIDATION AND NITRATION DEPENDENCE ON ALPHA-SYNUCLEIN CONFORMATIONAL CHANGES

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S2P415. CLASSICAL AND NOVEL PKC ISOFORMS DIFFERENTIALLY MODULATES M1 AND M3 RECEPTOR LEVELS IN RAT RETINAL CELLS CULTURES

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S2P416. AEROBIC EXERCISE IN ADOLESCENCE RESULTS IN MORE NEURONAL AND NON-NEURONAL CELLS AND MTOR OVEREXPRESSION IN THE CEREBRAL CORTEX OF RATS

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S2P417. A PHOTOTRANSDUCTION COMPLEX IN THE RETINA OF SQUID: GENERALITY OF THE TRANSDUCISOME FOR LIGHT SIGNALING

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S2P418. TGF-BETA AND NOTCH PATHWAY PARTICIPATION IN OLIGODENDROGLIAL DIFFERENTIATION OF ADULT NEURAL STEM CELLS FROM THE SUBVENTRICULAR ZONE

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S2P419. PRESYNAPTIC RELEASED PROTONS ACT AS NEUROTRANSMITTERS ACTIVATING ACID SENSING ION CHANNELS 1A (ASIC-1A) WHICH MODULATE SYNAPTIC TRANSMISSION AND PLASTICITY AT THE MOUSE CALYX OF HELD

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S2P420. PRENATAL AND EARLY ADOLESCENT EXPOSURE TO CANNABINOID RECEPTOR AGONIST WIN55212,2 DIFFERENTIALLY AFFECTS ETHANOL PREFERENCE AT ADOLESCENCE IN CD1 MICE

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S2P421. M FICOLIN: POLIMERIZATION AND AGGREGATION FROM BLOOD TO CEREBROSPINAL FLUID

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S2P422. IL-4 INDUCES CHOLINERGIC DIFFERENTIATION OF RETINAL CELLS IN VITRO

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S2P423. THE INVOLVEMENT OF IGF-1 ON THE M1 AND M3 MUSCARINIC RECEPTORS LEVELS MODULATIONS MEDIATED BY IL-4: THE EFFECT ON RETINAL GANGLION CELLS SURVIVAL

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S2P424. THE BETA2A-SUBUNIT OF THE VOLTAGE ACTIVATED CALCIUM CHANNELS CONTROLS CHANNEL ENDOCYTOSIS

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S2P425. ASSOCIATION BETWEEN POLYMORPHISMS IN GABA(A) RECEPTORS SUBUNITS AND AN AUTISM SPECTRUM DISORDER POPULATION AND THEIR FAMILIES

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S2P426. PRENATAL ETHANOL EXPOSURE MODIFIES IRON HOMEOSTASIS IN BRAIN REGIONS INVOLVED IN DRUG ADDICTIVE BEHAVIOR

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S2P427. THE P75 NEUROTROPHIC RECEPTOR IS REQUIRED TO MAINTAIN THE MATURE NEUROMUSCULAR JUNCTION

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S2P428. THE WNT RECEPTOR FRIZZLED-9 IS EXPRESSED IN SKELETAL MUSCLES AND REGULATES THE MORPHOLOGY AND ACTIVITY OF THE NEUROMUSCULAR JUNCTION

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S2P429. COMPARISON OF PRE- AND POST-TREATMENT NO AND ADMA LEVELS IN MAJOR DEPRESSION PATIENTS WITH CONTROL GROUP

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S2P430. ROLE OF INTRACELLULAR CALCIUM RECEPTOR INOSITOL 1,4,5-TRIPHOSPHATE TYPE 1 (IP3R1) IN RAT HIPPOCAMPUS AFTER NEONATAL ANOXIA

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S2P431. DIFFERENT CAMP SOURCES ARE CRITICALLY INVOLVED IN G PROTEIN–COUPLED RECEPTOR CRHR1 SIGNALING IN NEURONAL HIPPOCAMPAL CELLS

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S2P432. EVIDENCES FOR A PERICYTE-ASTROCYTE COMMUNICATION: POSSIBLE IMPLICATIONS FOR GA-I PATHOGENESIS

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S2P433. THE PARKINSONIAN NEUROTOXIN MPP+ REDUCES PROTEIN SUMOYLATION IN PRIMARY NEURONAL CULTURES

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S2P434. ROLE OF CONNEXIN 36 IN HIPPOCAMPAL CELL DEATH AFTER NEONATAL ANOXIA IN RATS

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S2P435. LXR INCREASE GNRH AND AMSH EXPRESSION IN THE RAT HYPOTHALAMUS IN VIVO

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S2P436. HYPERALGESIA BEHAVIOR AND SPINAL CORD GLIAL REACTIVITY ARE REDUCED BY PHARMACOLOGICAL METABOLIC MODULATION OF MITOCHONDRIA IN CHRONIC PAIN MODELS

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S2P437. DIFFERENCES IN OXYTOCIN, VASOPRESSIN, DOPAMINE AND ESTROGEN RECEPTOR EXPRESSION IN FEMALE AND MALE OXYTOCIN KNOCKOUT MICE

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S2P438. CONSTRUCTION OF A NON-INTEGRATIVE PLURIPOTENCY GENE VECTOR FOR THE INDUCTION OF NEURAL PROGENITORS

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S2P439. WNT SIGNALING INHIBITION CORRELATES WITH AN INCREASE IN ALZHEIMER´S NEUROPATHOLOGY IN A NATURAL MODEL (OCTODON DEGUS)

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S2P440. DENDRITE DEVELOPMENT IS MODULATED BY WNT7B-FZ7 THROUGH ACTIVATION OF NON CANONICAL WNT PATHWAYS

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S2P441. CELL THERAPY AND EXERCISE AMELIORATE THE PERIPHERAL NERVE REGENERATION

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S2P442. GROWTH HORMONE SECRETAGOGUE RECEPTOR TYPE 1A (GHSR1A) CONSTITUTIVE ACTIVITY IMPAIRS VOLTAGE-GATED CALCIUM CHANNELS (CAV)- DEPENDENT INHIBITORY NEUROTRANSMISSION IN HIPPOCAMPAL NEURONS

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S2P443. MICRORNA PROFILE IN EXOSOMES DERIVED FROM SOD1G93A ASTROCYTES. A ROLE IN ASTROCYTE MEDIATED MOTOR NEURON TOXICITY?

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S2P444. REELIN REGULATES SCHWANN CELL MIGRATION: PARTICIPATION OF SMALL GTPASE ARF6

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S2P445. BDNF/TRKB SIGNALING IS REQUIRED FOR AMPHETAMINE-INDUCED SPINOGENESIS

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S2P446. A NOVEL RAT XENOGRAFT MODEL OF BRAIN TUMORIGENESIS

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S2P447. MYELIN-ASSOCIATED GLYCOPROTEIN (MAG) MODULATES POSTNATAL CEREBELLAR DEVELOPMENT

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S2P448. THE RELEASE OF TNF ALPHA FOLLOWING OUABAIN TREATMENT OF RETINAL CELL CULTURES

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S2P449. ORGANOTYPIC CULTURES OF ADULT HUMAN BRAIN: A NOVEL MODEL TO STUDY AGE-ASSOCIATED NEURODEGENERATIVE DISEASES

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S2P450. THE PRO/ANTI-INFLAMMATORY PRECONDITIONING EFFECT OF MESENCHYMAL STROMAL CELLS ON MICE PERIPHERAL DORSAL ROOT GANGLIA NEURONS

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S2P451. THE NICOTINIC A7 RECEPTOR ACTIVATION MODULATES RETINAL GANGLION CELL SURVIVAL AND CYTOKINES LEVELS

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S2P452. AGO2, AN ESSENTIAL ENZYME INVOLVED IN MIRNA PROCESSING, IS HIGHLY EXPRESSED DURING RETINAL DEVELOPMENT

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S2P453. ASSESSING ALTERNATIVE SPLICING OF TDP-43 TARGET GENES IN THE BRAIN OF CONDITIONAL MOUSE MODELS OF FTD/ALS

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S2P454. A THIRD LINEAGE OF PHOTOTRANSDUCTION: A NOVEL OPSIN COUPLED TO A GO

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S2P455. HIGH FAT DIET IN EARLY LIFE ALTERS NEUROTROPHICS FACTORS IN THE HIPPOCAMPUS OF ADULT RATS?

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S2P456. THE EFFECT OF OUABAIN ON RETINAL GANGLION CELL SURVIVAL DEPENDS ON CASPASE-1 ACTIVATION AND IL-1BETA RELEASE

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S2P457. ROLE OF NITRIC OXIDE ON BOTH RAT GAIT AND GLIAL/NEURONAL MARKER EXPRESSION AFTER ACHILLES TENDON RUPTURE

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S2P458. CEREBELLAR TRANSCRANIAL ALTERNATING CURRENT STIMULATION (TACS) MODULATES VISUOMOTOR COORDINATION DURING A KINEMATIC ADAPTATION TASK

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S2P459. EXPERIMENTAL MODEL OF HEMISPHEROTOMY IN RATS

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S2P460. NEUROPROTECTION AND FUNCTIONAL RECOVERY AFTER SPINAL CORD INJURY FOLLOWED BY MESENCHYMAL STEM CELL AND FIBRIN SEALANT TREATMENT

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S2P461. FINGER TAPPING KINEMATICS IN ISOCHRONOUS SYNCHRONIZATION: FAST AND SLOW PHASES AND ERROR CORRECTION

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S2P462. CONTROL OF RHYTHMIC MOTOR BEHAVIORS BY CHOLINERGIC INPUTS

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Neural Circuit Physiology

S2P463. STUDY OF MEDIAL AMYGDALA NUCLEUS OUTPUTS TO THE VENTROMEDIAL HYPOTHALAMIC NUCLEUS IN OVARIECTOMIZED RATS AT EARLY PUBERTY

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S2P464. INTEGRATION OF ADULT-BORN GRANULE CELLS IN LOCAL INHIBITORY NETWORKS

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S2P465. COLLICULAR ELECTROPHYSIOLOGICAL CHANGES INDUCED BY INTERRUPTION OF CHRONIC ADMINISTRATION OF KETAMINE

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S2P466. THE TRPM2 CHANNEL IS A HYPOTHALAMIC HEAT SENSOR THAT LIMITS FEVER AND CAN DRIVE HYPOTHERMIA

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S2P467. PATHWAY-SPECIFIC MEDIUM SPINY NEURON RESPONSE TO CHRONIC L-DOPA TREATMENT IN A MOUSE MODEL OF PARKINSON'S DISEASE

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S2P468. NEURONAL DYNAMICS OF MAGNOCELULAR DYSFUNCTION AT HIGH RISK MENTAL STATE POPULATION

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S2P469. CHOLINERGIC MODULATION IN THE PROCESSING OF AFFERENT INPUTS IN THE DENTATE GYRUS OF THE HIPPOCAMPUS

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S2P470. MECHANISMS OF STRIATAL CHOLINERGIC INTERNEURON HYPEREXCITABILITY IN ANIMAL MODELS OF PARKINSON'S DISEASE AND L-DOPA-INDUCED DYSKINESIA

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S2P471. STUDY OF THE MEDIAL PREOPTIC NUCLEUS OUTPUTS TO THE VENTROMEDIAL HYPOTHALAMIC NUCLEUS IN OVARIECTOMIZED ANIMALS

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S2P472. THE ONSET OF SODIUM APPETITE: INTERACTION BETWEEN ANGIOTENSINERGIC AND SEROTONERGIC SYSTEMS AND THE OSMORECEPTIVE CELLS INVOLVEMENT

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Neurochemistry and Neuropharmacology

S2P473. NEURONAL PROTECTION BY NATURAL FLAVONES AGAINST OXIDATIVE STRESS AND ITS RELATIONSHIP WITH THE MOLECULAR STRUCTURE

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S2P474. AGMATINE, BY IMPROVING NEUROPLASTICITY MARKERS AND INDUCING NRF2, PREVENTS CORTICOSTERONE-INDUCED DEPRESSIVE-LIKE BEHAVIOR IN MICE

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S2P475. SETTING UP THE EXPERIMENTAL CONDITIONS TO UNCOVER A POWERFUL TRIUMVIRATE: NEUROGENESIS, SEROTONIN AND BDNF

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S2P476. M AND H-FICOLIN: DYNAMIC AND AGGREGATION FROM BLOOD TO CEREBROSPINAL FLUID

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S2P477. TREATMENT WITH AN ETHYL-ACETATE FRACTION (EAF) OF TRICHILIA CATIGUA (CATUABA) ALLEVIATES THE MEMORY IMPAIRMENT CAUSED BY GLOBAL CEREBRAL ISCHEMIA IN RATS

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S2P478. MINOCYCLINE PREVENTS EARLY AXOGLIAL ALTERATIONS OF THE OPTIC NERVE INDUCED BY EXPERIMENTAL GLAUCOMA

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S2P479. USE-DEPENDENT REGULATION OF GABA-A RECEPTORS IN RAT CEREBRAL CORTEX

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S2P480. INFLUENCE OF SILDENAFIL ON PILOCARPINE- AND PICROTOXIN-INDUCED SEIZURES

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S2P481. ANTIEPILEPTIC EFFECT OF SIDEROXYLON OBTUSIFOLIUM METHANOLIC EXTRACT ON TWO MODELS OF ACUTE SEIZURES IN VIVO – A PILOT STUDY

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S2P482. HIGH FAT DIET-INDUCED OBESITY IN MICE INHIBITS ENERGETIC METABOLISM IN BRAIN STRUCTURE

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S2P483. EFFECTS OF ACUTE AND LONG-TERM ADMINISTRATION OF GOLD NANOPARTICLES ON OXIDATIVE STRESS PARAMETERS IN RAT BRAIN

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S2P484. THE COCAINE AND AMPHETAMINE REGULATED TRANSCRIPT (CART) AS REGULATOR OF THE CELL PROLIFERATION IN THE ADULT VENTRICULAR-SUBVENTRICULAR ZONE: MORPHOFUNCTIONAL AND NEUROCHEMICAL ASPECTS

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S2P485. C. ELEGANS MUSCLE CYS-LOOP RECEPTORS AS NOVEL TARGETS OF TERPENOIDS

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S2P486. ENERGY DRINKS ACUTE TOXICITY EVALUATION

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S2P487. THE BLOCKADE OF LOW AFFINITY NEUROTENSIN (NTS2) RECEPTOR IMPAIRS BRAIN NITRIC OXIDE PRODUCTION AND MITOCHONDRIAL BIOENERGETICS

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S2P488. OXIDATIVE STRESS INDUCED BY ACUTE ALCOHOL EXPOSURE IN MOUSE BRAIN CORTEX NON-SYNAPTIC MITOCHONDRIA AND SYNAPTOSOMES

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S2P489. A NOVEL IN VIVO APPROACH TO STUDY THE INTERNALIZATION OF MELANIN-CONCENTRATING HORMONE IN THE CNS OF THE RAT

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S2P490. EARLY LIFE SOCIAL ISOLATION CHANGES LOCOMOTOR ACTIVITY, OXIDATIVE STRESS PARAMETERS AND DOPAMINE TRANSPORTER AFTER AN AMPHETAMINE CHALLENGE IN ADULT RATS

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S2P491. EFFECTS OF VITAMIN D SUPPLEMENTATION ON DOPAMINE AND DOPAC CONCENTRATION AND OXIDATIVE STRESS IN RODENT MODEL OF PARKINSON'S DISEASE

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S2P492. AGE-RELATED CHANGES IN MITOCHONDRIAL ACTIVE OXYGEN SPECIES PRODUCTION AND OXIDATIVE DAMAGE IN BRAIN CORTEX SYNAPTOSOMES

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S2P493. EGF RECEPTOR ACTIVATION IS REQUIRED FOR ADP-INDUCED PROLIFERATION OF RETINAL GLIAL PROGENITORS IN CULTURE

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S2P494. UNRAVELING THE EVOLUTIONARY HISTORY OF NICOTINIC CHOLINERGIC RECEPTORS SUBUNITS

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S2P495. PROTECTIVE EFFECTS OF GUANOSINE AGAINST 6-OHDA-INDUCED TOXICITY IN VITRO

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S2P496. CHEMICAL CHARACTERIZATION AND PHARMACOKINETIC STUDIES OF THE MAIN COMPONENTS FOUND IN COCA PASTE SEIZED SAMPLES IN URUGUAY

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S2P497. THE HYPERALGESIC EFFECT PROVOKED BY TRPV1 BLOCKADE IN THE MOUSE DORSAL PERIAQUEDUCTAL GRAY DOES NOT DEPEND ON CB1 RECEPTOR

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S2P498. ANTIDYSKINETIC EFFECT OF ACUTE GUANOSINE ADMINISTRATION IN RESERPINIZED MICE

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S2P499. BIOPHYSICAL CHARACTERIZATION OF TAU AMYLOID AGGREGATION AND A POSSIBLE NEUROPROTECTIVE MECHANISM OF DOXYCYCLINE IN TAUOPATHIES

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S2P500. ANTIDEPRESSANT POTENTIAL OF RIPARIN IV: BEHAVIORAL ANALYSIS OF MICE EXPOSED TO MODEL OF CHRONIC DEPRESSION BY CORTICOSTERONE ADMINISTRATION

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S2P501. VITAMIN D INDUCES ANTIDEPRESSANT-LIKE EFFECT IN MICE

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S2P502. EFFECTS OF CANNABIDIOL ON HIPPOCAMPAL NEURODEGENERATION AND WHITE MATTER INJURY INDUCED BY BILATERAL COMMON CAROTID ARTERY OCCLUSION IN MICE

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S2P503. NEUROGENESIS AND GLIOGÉNESIS: ROLE OF CICLIN-DEPENDENT-KINASE-5 IN CEREBRAL ISCHEMIA. A NEUROLOGICAL AND HISTOLOGICAL ANALYSIS BY GENE THERAPY

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S2P504. BLOCKADE OF ADENOSINE A2A RECEPTORS AND NLRP3 INFLAMMASOME IN CORTICOSTERONE- INDUCED MODEL OF STRESS IN A HIPPOCAMPAL NEURONAL CELL LINE

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S2P505. EXPLORING THE FUNCTIONAL PROPERTIES OF HETEROMERIC ALPHA7BETA2 NICOTINIC ACETYLCHOLINE RECEPTORS AT THE SINGLE-CHANNEL LEVEL

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S2P506. GLIAL METABOLISM IS MODULATED IN BOTH HEMISPHERES AFTER FOCAL CEREBRAL ISCHEMIA

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S2P507. EVALUATION OF NEUROPROTECTIVE EFFECT OF RT10, A COMPOUND ISOLATED FROM THE PARAWIXIA BISTRIATA SPIDER VENOM

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S2P508. ATORVASTATIN AND TEMOZOLOMIDE REDUCE HUMAN GLIOMA CELLS MIGRATION AND SURVIVAL VIA GLUTAMATE RECEPTORS MODULATION

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S2P509. ROLE OF PRELIMBIC CORTEX MEDIATES CONTEXT-INDUCED THE REINSTATEMENT OF ALCOHOL-SEEKING

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S2P510. CASTRATION ALTERS THE DENSITY AND SHAPE OF DENDRITIC SPINES IN THE MALE POSTERODORSAL MEDIAL AMYGDALA

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S2P511. THE MEMORY CONSOLIDATION IMPAIRMENT INDUCED BY INTERLEUKIN-1B COULD BE ASSOCIATED TO CHANGES IN HIPPOCAMPAL STRUCTURAL PLASTICITY

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S2P512. EFFECTS OF UNILATERAL ORCHIDECTOMY TO IMMATURE RATS ON DENDRITIC ARBORIZATION OF THE CA1 PYRAMIDAL-NEURONS OF THE HIPPOCAMPUS

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S2P513. EFFECTS OF BLOCKING SUPRACHIASMATIC NUCLEUS M1 RECEPTORS ON THE OVULATION OF THE FEMALE RAT

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S2P514. MILD UNPREDICTABLE STRESS IN ADOLESCENCE MICE: LONG-TERM EFFECTS ON IMMUNITY

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S2P515. GLYCINE RECEPTOR BETA SUBUNIT: A CRITICAL TARGET FOR PAIN SENSITIZATION

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S2P516. A SYSTEMATIC REVIEW OF PSYCHONEUROIMMUNOLOGY-BASED INTERVENTIONS

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S2P517. THE EXPRESSION OF DA1 RECEPTOR ON THE ANTERIOR HYPOTHALAMIC AREA VARIES THROUGHOUT THE RAT ESTROUS CYCLE

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S2P518. ROLE OF CD300F IN MICROGLIAL PHENOTYPE AND NEUROINFLAMMATION

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S2P519. EXPOSURE TO EXPERIENCE-DEPENDENT PLASTICITY DURING PREGNANCY AND LACTATION MODULATES OFFSPRING FOOD INTAKE AND GLUCOSE RESPONSE

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S2P520. MODULATION OF PITUITARY CELL RENEWAL BY L-3,4-DIHYDROXYPHENYLALANINE (L-DOPA): ROLE OF ITS CONVERSION TO DOPAMINE (DA)

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S2P521. IMMOBILIZED NICOTINIC ACETYLCHOLINE RECEPTOR AS A TOOL FOR THE DETECTION OF AUTOANTIBODIES IN THE DEVELOPMENT OF A NEW IMMUNOANALYTICAL FLUORESCENCE-BASED TECHNOLOGY

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SENSORY SYSTEMS

S2P522. NEURAL BASIS OF AVERSIVE TASTE IN A BLOOD-SUCKING INSECT

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S2P523. MODULATION OF EEG SIGNALS ASSOCIATED WITH EYE MOVEMENT DURING A VISUOMOTOR ADAPTATION TASK

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S2P524. OLIVOCOCHLEAR REFLEX STRENGTH AND BEHAVIORAL PERFORMANCE DURING SELECTIVE VISUAL ATTENTION TASK WITH NOISE AND VOCALIZATIONS AS AUDITORY DISTRACTORS

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S2P525. CERVICAL VESTIBULAR EVOKED MYOGENIC POTENTIALS ARE ALTERED IN CHILDREN WITH ATTENTION DEFICIT AND HYPERACTIVITY DISORDER

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S2P526. SUSTAINED ATTENTION TO VISUAL STIMULI WITH AUDITORY DISTRACTORS AND LOCOMOTOR ACTIVITY IN ALPHA-9 NICOTINIC RECEPTORS KNOCK-OUT MICE

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S2P527. DOPAMINE MODULATES ODOR INDUCED NEURAL ACTIVITY IN THE HONEY BEE ANTENNAL LOBES

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S2P528. IMT504, AN IMMUNOMODULATORY OLIGODEOXINUCLEOTIDE, REDUCES ALLODYNIA IN AN EXPERIMENTAL MODEL OF INFLAMMATORY PAIN

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S2P529. THERAPEUTIC USE OF ISOLATED P27KIP1 KNOCKDOWN FOR SUPPORTING CELLS PROLIFERATION IN RAT PUPS

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S2P530. ROLE OF TRPM8 CHANNELS IN THE ALTERED SENSITIVITY OF CORNEAL PRIMARY SENSORY NEURONS IN RESPONSE TO AXONAL DAMAGE

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S2P531. THE ROLE OF SYNAPTIC INPUTS ON STIMULUS-SPECIFIC ADAPTATION (SSA) IN THE AUDITORY MIDBRAIN

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S2P532. EFFECTS OF WHOLE BODY VIBRATION ON MECHANHICAL SENSIBILITY OF HINDPAW AFTER SCIATIC NERVE CRUSH IN ANIMAL MODEL

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S2P533. SIMULTANEOUS VISUAL AND VESTIBULAR STIMULUS MODULATION OF CORTICOCORTICAL INTERACTIONS IN HUMAN

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S2P534. EVALUATION OF MYGALIN ANTI-NOCICEPTIVE ACTIVITY, ANALOGUE SYNTHESIZED FROM NATURAL ACYLPOLYAMINE OF THE A. GOMMESIANA (ARANEAE, THERAPHOSIDAE) HEMOLYMPH, IN A MODEL OF HYPERALGESIA IN WISTAR RATS

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Synaptic Transmission, Excitability and Glia

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S3P576. THE CONTRIBUTION OF NEUROGENESIS TO HIPPOCAMPAL NETWORK COMPUTATIONS AND DENTATE DEPENDENT BEHAVIOR

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S3P577. FEAR CONDITIONING AND ANXIETY IN HUMANS: HOW COULD THE COGNITIVE-BEHAVIORAL SYSTEMS BE AFFECTED

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S3P578. PARTICIPATION OF BEHAVIORS OTHER THAN LOCOMOTION IN THE DEVELOPMENT AND EXPRESSION OF BEHAVIORAL SENSITIZATION EVOKED BY TWO SUBANESTHETIC DOSES OF KETAMINE

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S3P579. MICROSACCADES GROUPING REVEALS OBJECT SEGMENTATION DURING FREE VIEWING OF NATURAL SCENES

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S3P580. THE DYNAMIC NATURE OF SYSTEMS CONSOLIDATION: STRESS DURING LEARNING AS A SWITCH GUIDING THE RATE OF THE HIPPOCAMPAL DEPENDENCY AND MEMORY QUALITY

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S3P581. BEHAVIORAL ALTERATIONS IN LEISHMANIA AMAZONENSIS- INFECTED MICE

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S3P582. STROOP AND STOP SIGNAL INTERFERENCES – ELABORATION OF A PROTOCOL TO EVALUATE EXECUTIVE FUNCTIONS

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S3P583. EFFECTS OF SCOPOLAMINE AND PROPRANOLOL ON A TREATMENT THAT FACILITATES RECOVERY FROM FRUSTRATION

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S3P584. CONTEXTUAL CONDITIONED TOLERANCE TO THE SEDATIVE EFFECTS OF KETAMINE IN RATS

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S3P585. ANDROGRAPHOLIDE RECOVERS COGNITIVE IMPAIRMENT IN A NATURAL MODEL OF ALZHEIMER'S DISEASE (OCTODON DEGUS)

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S3P586. SEX DIFFERENCES IN HUMAN EPISODIC MEMORY RECONSOLIDATION: EVIDENCE FROM A RANDOMIZED TEST CONTEXT

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S3P587. REDUCED BRAIN VOLUME ASSOCIATED TO PERITRAUMATIC TONIC IMMOBILITY IN VICTIMS OF URBAN VIOLENCE WITH POSTTRAUMATIC STRESS DISORDER

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S3P588. REDUCED GRAY MATTER VOLUME IS ASSOCIATED TO PERITRAUMATIC TONIC IMMOBILITY IN VICTIMS OF URBAN VIOLENCE WITH POSTTRAUMATIC STRESS DISORDER

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S3P589. ENHANCING SOCIABILITY AND REDUCING ISOLATION: THE EFFECTS OF TEXT AND PICTURE BONDING PRIMES

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S3P590. NICOTINE CHRONIC TREATMENT EFFECTS ON MOTOR PERFORMANCE AND SHORT TERM MEMORY IN THE MURINE MODEL OF PARKINSONISM INDUCED BY 6-OHDA

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S3P591. INTRACEREBROVENTRICULAR OUABAIN MODEL OF STATUS EPILEPTICUS: MIMICKING COMPONENTS OF DYSMETABOLIC SYMPTOMATIC SEIZURES

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S3P592. IN SEARCH FOR THE BEST PARAMETERS FOR OPTIMAL PAIN CONTROL BY DBS OF THE LATERAL HABENULA IN RATS

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S3P593. NEURONAL CIRCUITS RESPONSIBLE OF TEMPORAL MAINTENANCE OF AVERSIVE MEMORIES

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S3P594. AGING RELATED MEMORY, ANXIETY AND NEUROTRANSMITTERS DEFICITS IN RATS

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S3P595. BEING A WINNER DOESN´T ALWAYS PAY: MEMORY IMPAIRMENT AFTER WINNING A FIGHT IN THE CRAB NEOHELICE GRANULATA

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S3P596. ACUTE EFFECT OF COCA-PASTE ON SLEEP AND EEG ACTIVITY: ROLE OF CAFFEINE

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S3P597. HIPPOCAMPAL ENDOCANNABINOID SYSTEM: EFFECTS OF AM404 UPON CONSOLIDATION AND RETRIEVAL OF AVERSIVE MEMORIES AND ON LTP INDUCTION

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S3P598. ANTAGONISM OF DORSAL HIPPOCAMPUS CANNABINOID TYPE-2 RECEPTORS IMPAIR THE CONSOLIDATION OF A CONTEXTUAL FEAR MEMORY IN RATS

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S3P599. THE EFFECTS OF THE ATYPICAL ANTIPYSYCHOTIC CLOZAPINE ON THE ATTENTIONAL DEFICITS INDUCED BY THE DISSOCIATIVE ANESTHETIC KETAMINE IN FEMALE RATS

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S3P600. MEDIAN RAPHE NUCLEUS INJECTION OF PRAZOSIN INCREASES FOOD INTAKE AND FOS EXPRESSION IN OREXIN NEURONS

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S3P601. BONDING PICTURES: AFFECTIVE RATINGS ASSOCIATED TO EMPATHY AND LONELINESS

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S3P602. PHARMACOLOGICAL VALIDATION OF THE PLUS-MAZE WITH RAMP FOR ZEBRAFISH (DANIO RERIO) - PRELIMINARY RESULTS

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S3P603. FISETIN PROTECTED NEURONAL DEMAGE AND IMPROVE MEMORY IN MICE'S STROKE

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S3P604. ROLE OF ECLOSION HORMONE IN D. MELANOGASTER ECDYSIS

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S3P605. VALPROIC ACID DECREASES PLACE CONTIDIONED PREFERENCE INDUCED BY ETHANOL IN MICE: IS IT RELATED WITH BDNF LEVELS IN VENTRAL STRIATUM?

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S3P606. EVALUATION OF ANTIPSYCHOTIC EFFECT OF ERYTHRINA VELUTINA ETHANOL EXTRACT IN A MODEL OF SCHIZOPHRENIA INDUCED BY KETAMINE IN MICE

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S3P607. ENVIRONMENTAL IMPOVERISHMENT EARLY IN LIFE IS ASSOCIATED WITH ABNORMAL COGNITIVE DEVELOPMENT IN ALBINO SWISS MICE

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S3P608. ANXIOLYTIC-LIKE EFFECT FROM QUERCETINE IN EXPERIMENTAL MICE MODELS

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S3P609. CHEMICAL INACTIVATION OF THE AMYGDALA ATTENUATES DEFENSIVE BEHAVIORS IN MICE EXPOSED TO AN OPEN ELEVATED PLUS-MAZE

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S3P610. DURATION OF MASTICATORY DEPRIVATION INFLUENCES SPATIAL MEMORY IMPAIRMENT AND MASTICATORY REHABILITATION SEEMS TO RECOVER

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S3P611. THE EFFECT OF CANNABIDIOL IN FEAR MEMORY CONSOLIDATION AND GENERALIZATION

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S3P612. TRAINING INTENSITY DURING SEQUENTIAL CONTEXTUAL FEAR CONDITIONING MODIFY THE RATE OF SYSTEMS CONSOLIDATION AND MEMORY QUALITY: IMPLICATIONS FOR CONSOLIDATION OF MULTIPLE MEMORY TRACES

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S3P613. MATERNAL SWIMMING IN ADHD MODEL RATS AS AN ALTERNATIVE OF OFFSPRINGS NEUROPROTECTION

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S3P614. ENVIRONMENTAL ENRICHMENT INCREASES HAMSTER (MESOCRICETUS AURATUS)

NATURAL PREFERENCE BY ENCLOSED SPACES

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S3P615. ENRICHED ENVIRONMENT ENHANCES RECOGNITION OF OBJECT PLACEMENT AND IDENTITY IN THE SYRIAN GOLDEN HAMSTER (MESOCRICETUS AURATUS)

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S3P616. THALAMIC NUCLEUS REUNIENS PARTICIPATES IN FEAR MEMORY CONSOLIDATION REGULATING ITS SPECIFICITY AND PERSISTENCE

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S3P617. FEMALES' SEXUAL STATUS MODULATION OF MALE BEHAVIOR UNDER VISUAL OR CHEMICAL STIMULI IN DANIO RERIO (ZEBRAFISH)

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S3P618. PREDICTING UPCOMING EVENTS OCCURRING IN THE SPACE SURROUNDING THE HAND

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S3P619. AGE-ASSOCIATED MEMORY DEFICIT AND ALZHEIMER-LIKE PATHOLOGY IN A GENETIC RAT MODEL OF EPILEPSY

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S3P620. HYPOTHERMIA AS A NEUROPROTECTIVE AGENT TO MITIGATE SPATIAL MEMORY IMPAIRMENT CAUSED BY NEONATAL ANOXIA

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S3P621. BEHAVIORAL EFFECTS OF HIGH FREQUENCY AUDITORY STIMULATION IN WISTAR RATS ARE SHAPED BY STRESS HISTORY

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S3P622. EXPERIMENTAL NEONATAL SEPSIS INCREASES THE RISK OF SCHIZOPHRENIA-LIKE BEHAVIOUR IN ADULTHOOD

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S3P623. TIME, AGEING PROCESS AND DEAD

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S3P624. TO BE PROPERLY LOCATED ONE RESPECT TO EACH OTHER: MOMENTOUS PROPERTY OF NEURAL CELLS

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S3P625. REPEATED ADMINISTRATION OF CANNABIDIOL IN TRAUMA-EXPOSED RATS PREVENTS SUBSEQUENT SENSITIZATION AND IMPAIRED EXTINCTION OF CONDITIONED FEAR

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S3P626. MATE MAROTE: COGNITIVE TRAINING TO ANSWER OPEN QUESTIONS

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S3P627. DISRUPTION OF REWARDING EFFECT OF MORPHINE BY MEMORY RECONSOLIDATION: POST-RETRIEVAL CYCLOHEXIMIDE BLOCKS BOTH CPP AND LOCOMOTOR SENSITIZATION

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S3P628. EXPLORING THE FUNCTION OF THE SEROTONERGIC SYSTEM IN THE RECONSOLIDATION OF AVERSIVE MEMORIES

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S3P629. HUMAN MESENCHYMAL STEM CELLS THERAPY IMPROVES COGNITIVE FUNCTION IN A SPORADIC ALZHEIMER RAT MODEL

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Chronobiology

S3P630. TIMED FOOD RESTRICTION PREVENTS DEPRESSIVE-LIKE BEHAVIOR INDUCED BY CONSTANT LIGHT IN RODENTS

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S3P631. CHOLINERGIC TRANSMISSION IN THE CIRCADIAN PACEMAKER OF DROSOPHILA

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S3P632. TNF-ALPHA AND CCL2 MEDIATE THE IMMUNE-CIRCADIAN INTERACTION IN THE CENTRAL NERVOUS SYSTEM

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S3P633. MODELLING TRANSLATIONAL REGULATION OF PER AND ITS EFFECTS OVER THE CIRCADIAN MOLECULAR CLOCK

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S3P634. THE ROLE OF THE BMP PATHWAY IN THE OPERATION OF THE ADULT CIRCADIAN NETWORK IN DROSOPHILA

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S3P635. THE HIGH LIGHT EXPOSURE OF THE ANTARCTIC SUMMER INDUCED A DELAY OF THE SLEEP ONSET TIME IN A POPULATION OF UNIVERSITY STUDENTS FROM URUGUAY

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Development

S3P636. BEHAVIORAL AND MOLECULAR CHANGES INDUCED BY EARLY NOISE EXPOSURE USING DIFFERENT EXPOSURE SCHEDULES. PARTIAL REVERSAL AFTER REARING IN AN ENRICHED ENVIRONMENT

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S3P637. EFFECT OF CANNABINOID HEMOGLOBIN-DERIVED PEPTIDES (RVD-HEMOPRESSIN AND VD-HEMOPRESSIN) IN THE POSTNATAL MICE SUBVENTRICULAR ZONE NEUROGENESIS AND OLIGODENDROGENESIS

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S3P638. VISUAL ACUITY AND COGNITIVE DEVELOPMENT IN CHILDREN AFTER BILATERAL CONGENITAL CATARACTS SURGERY

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S3P639. KINESIN1 IS REQUIRED FOR AXONAL PATHFINDING AND CANNABINOIDINDUCED AXONAL DEVELOPMENT BY MEDIATING THE AXONAL TRANSPORT OF CB1 RECEPTOR

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S3P640. EFFECTS OF PARENTAL EXERCISE ON PHYSICAL AND SENSORYMOTOR DEVELOPMENT, VOLUNTARY PHYSICAL ACTIVITY, PHYSICAL PERFORMANCE AND SPATIAL MEMORY OF MALE RATS WISTAR OFFSPRING

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S3P641. NUTRITIONAL RESTRICTION OF OMEGA-3 FATTY ACIDS INDUCES PHENOTYPIC PLASTICITY IN THE MICROGLIA OF RAT SUBSTANTIA NIGRA

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S3P642. ELIMINATION OF EARLY BORN NEURONS IN THE CEREBRAL CORTEX

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S3P643. A NERVOUS SYSTEM ENHANCER UNDERWENT ACCELERATED EVOLUTION IN PRIMATES AND SHOWS HETEROCHRONY DURING BRAIN DEVELOPMENT IN TRANSGENIC MICE

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S3P644. EUPHORIC RESPONSES TOWARD THE DECREASE OF AN AVERSIVE REWARD

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S3P645. HIGH FAT DIET INDUCES SEX-SPECIFIC CHANGES IN ANTIOXIDANT DEFENSES IN RAT HYPOTHALAMUS

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S3P646. EFFECTS OF THE GESTATIONAL DIABETES MELLITUS ON THE DEVELOPMENT AND NEUROIMMUNOMODULATION IN NEWBORN AND ADOLESCENCE RATS

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Disorders of the Nervous System

S3P647. ROLE OF CAV1.2 CALCIUM CHANNEL IN HIPPOCAMPAL NEURONS OF ANIMAL WITH DEPRESSIVE-LIKE BEHAVIORS

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S3P648. HIPPOCAMPAL NEURONAL FATE REPROGRAMMING BY INTRAHIPPOCAMPAL ADMINISTRATION OF KAINIC ACID IN MICE

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S3P649. INTRANASAL ROUTE IN A TRANSGENIC MODEL OF ALZHEIMER: NANOMEDICINE DRUG THERAPY

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S3P650. NEUROPROTECTIVE EFFECT OF A NOVEL MULTIFUNCTIONAL IRON/COPPER CHELATOR IN CELL AND ANIMAL MODELS OF PARKINSON'S DISEASE

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S3P651. PERINEURONAL NETS OF STRIATE CORTEX ARE REDUCED IN ADULT CATS SUBIMITED TO MONOCULAR ATROPINIZATION DURING CRITICAL PERIOD

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S3P652. VEGF AND G-CSF GENES AND HUMAN ADIPOSE-DERIVED MESENCHYMAL STEM CELLS IN THE MOUSE SCIATIC NERVE TRANSECTION AND TUBULIZATION MODEL

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S3P653. EFFECT OF STRESS INDUCED BY IMMOBILIZATION IN THE MICHROARCHITECTURE OF SLEEP SPINDLES IN RATS

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S3P654. REFRACTORINESS TO CORTICAL STIMULATION-INDUCED ANALGESIA: A POSSIBLE RELATION WITH THE LACK OF SYNAPTIC REMODELING IN THE RAPHE AND DECREASE OF SPINAL SEROTONIN

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S3P655. ALTERED REGIONAL CEREBRAL BLOOD FLOW AND COGNITIVE PERFORMANCE IN PATIENTS WITH CONGESTIVE HEART FAILURE

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S3P656. FUNCTIONAL INTERACTIONS BETWEEN MCHERGIC AND SEROTONERGIC NEURONS. AN IN VIVO ELECTROPHYSIOLOGICAL STUDY

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S3P657. ENDOTHELIAL AND ASTROGLIAL ALTERATIONS IN IN VITRO AND IN VIVO MODELS OF ALZHEIMER'S DISEASE. EVIDENCE OF CELL ACTIVATION AND AUTOPHAGIC INDUCTION

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S3P658. ABNORMAL STRUCTURAL CONNECTIVITY IN PATIENTS WITH EPILEPSY AND FOCAL CORTICAL DYSPLASIA (FCD)

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S3P659. STUDY OF TDP-43 GENETIC AND PROTEIN-BASED INTERACTIONS: FOCUS ON ALZHEIMER AND PARKINSON'S DISEASES GENE PRODUCTS

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S3P660. EFFECT OF EXTREMELY LOW FRECUENCY MAGNETIC FIELDS EXPOSITION IN L-DOPA-INDUCED-DYSKINESIAS AND TRANSCRIPTIONS FACTORS IN A RAT MODEL OF PARKINSON'S DISEASE

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S3P661. NEUROPROTECTIVE DRUGS TO TREAT THE NEUROLOGICAL ALTERATIONS CAUSED BY ZIKA VIRUS INFECTION

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S3P662. MYELINATION OF THE REGENERATING OPTIC NERVE OF MICE

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S3P663. EFFECTS OF L-DOPA ADMINISTRATION ON NOCICEPTIVE RESPONSES FOLLOWING INTRANASAL MPTP ADMINISTRATION IN RATS, AN ANIMAL MODEL OF PARKINSON´S DISEASE

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S3P664. SOCIAL AVOIDANCE BUT NOT ANHEDONIA PERSISTS IN ADULT MALE MICE AFTER CHRONIC SOCIAL DEFEAT STRESS DURING ADOLESCENCE

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S3P665. COGNITIVE DEFICITS AND DEPRESSIVE-LIKE BEHAVIOR FOLLOWING 6-HYDROXYDOPAMINE-INDUCED DEGENERATION OF LOCUS COERULEUSIN RATS

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S3P666. EFFECT OF TWO CONSECUTIVE GENERATION IN MITOCHONDRIAL ROS PRODUCTION AND OXIDATIVE STATUS IN BRAINSTEM FEMALES JUVENILE SUBMITTED IN THE PROTEIN RESTRICTION DURING DEVELOPMENT

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S3P667. THE ROLE OF NEUROTROPHINS ON GENDER BIASED EFFECTS OF INTRANASAL MPTP ADMINISTRATION ON ANHEDONIC AND DEPRESSIVE-LIKE BEHAVIORS IN MICE

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S3P668. PRENATAL VPA EXPOSURE ALTERS POSTNATAL HISTONE 3 ACETYLATION LEVELS

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S3P669. DIAGNOSTIC AND PROGNOSTIC SERUM BIOMARKERS IN ASTROCYTOMA

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S3P670. GAIT ANALYSIS AND CORTICAL RECORDINGS IN A PARKINSON'S DISEASE ANIMAL MODEL

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S3P671. TRANSCRANIAL DIRECT CURRENT STIMULATION IMPROVES LONG-TERM MEMORY IN AN ANIMAL MODEL OF ATTENTION-DEFICIT/ HYPERACTIVITY DISORDER AND MODULATES INFLAMMATORY AND OXIDATIVE PARAMETERS IN CONTROL RATS

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S3P672. MODULATION OF GLIAL RESPONSE BY DIETARY RESTRICTION IN AN ANIMAL MODEL OF ALZHEIMER'S DISEASE

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S3P673. SELECTIVE DELETION OF DOPAMINE D2 RECEPTOR IN FAST SPIKING INTERNEURONS: IMPLICATION IN PSYCHIATRIC DISORDERS

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S3P674. THE THERAPEUTIC POTENTIAL OF CANNABINOID SYSTEM IN AN IN VITRO MODEL OF NEURONAL DEATH

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S3P675. ALTERED SECRETION OF EXTRACELLULAR VESICLES IN AN ASTROCYTE MODEL OF TRINUCLEOTIDE REPEAT DISORDER

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S3P676. POST-PARALYSIS TYROSINE KINASE INHIBITION WITH MASITINIB ABROGATES NEUROINFLAMMATION AND SLOWS DISEASE PROGRESSION IN INHERITED AMYOTROPHIC LATERAL SCLEROSIS

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S3P677. CORPUS CALLOSUM CONNECTIVITY ALTERATIONS IN THE VALPROIC ACID EXPERIMENTAL MODEL OF AUTISM

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S3P678. PRE-CLINICAL INVESTIGATION OF THE EFFECTS OF SODIUM BUTYRATE TREATMENT ON BEHAVIOR AND NEUROTROPHINS LEVELS IN RATS SUBMITTED TO ANIMAL MODEL OF MANIA OR DEPRESSION

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S3P679. DEEP HYPOTHERMIC SHOCK REVERSES THE LOSS OF CALBINDIN-POSITIVE NEURONS CAUSED BY PERINATAL ASPHYXIA IN THE RAT.

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S3P680. SPIRULINA (SP) NEUROPROTECTION IN THE 6-OHDA MODEL OF PARKINSON'S DISEASE IS POSSIBLY RELATED TO ITS ANTI-INFLAMMATORY AND ANTIOXIDANT PROPERTIES

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S3P681. XBP1S/ATF6F HETERODIMER PARTICIPATES IN THE PROTEOSTASIS MODULATION ON NEURODEGENERATIVE DISEASE MODELS

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S3P682. POSSIBLE ROLE FOR NMDA RECEPTORS IN THE MAINTENANCE OF RETINOFUGAL PATHWAYS IN MICE WITH RETINAL DEGENERATION

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S3P683. A 6-MONTH RETROSPECTIVE OBSERVATIONAL STUDY WITH SCHIZOPHRENIA PATIENTS USING PALIPERIDONE EXTENDED-RELEASE TABLETS

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S3P684. ASSESSMENT OF THE TRANSGENERATIONAL EFFECT OF VALPROIC ACID IN MICE

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S3P685. CYCLIN-DEPENDENT KINASE 5 ACTIVITY MODULATES CONSTITUTIVE AND SUBSTRATE DOPAMINE TRANSPORTER CELL SURFACE EXPRESSION: IMPLICATIONS IN ATTENTION-DEFICIT HYPERACTIVITY DISORDER –ADHD-

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S3P686. ANALYSIS OF THE VESICULAR TRANSPORTER VGLUT-1/VGAT RATIO AS POSSIBLE MARKER OF CORTICAL PLASTICITY LEVELS.

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S3P687. WITHDRAWN

S3P688. PANNEXIN 1 MODULATES THE FUNCTION OF THE SUPPORTING CELLS OF THE ORGAN OF CORTI

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S3P689. M4 AND M5 ACETYLCHOLINE RECEPTOR LEVELS IN RAT RETINAL CELLS AND ITS MODULATION BY PROTEIN KINASE C ACTIVATION

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S3P690. DROSOPHILA DLRRK (RNAI) FLY RESISTS PARAQUAT-INDUCED OXIDATIVE STRESS: A THERAPEUTIC STRATEGY IN PARKINSON DISEASE

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S3P691. PROTECTIVE EFFECT OF AN ALPHA-MELANOCYTE STIMULATING HORMONE ANALOGUE AGAINST PALMITIC ACID TOXICITY

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S3P692. DOPAMINERGIC DIFFERENCIATION OF MÜLLER CELLS DERIVED FROM EYE PROGENITORS

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S3P693. THE GLUCOSE SENSITIVITY OF MESENCEPHALIC DOPAMINERGIC CELLS: EFFECT ON TYROSINE HYDROXYLASE REGULATION

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S3P694. NEGATIVE MODULATION OF TRPM8 FUNCTION BY PROTEIN KINASE C

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S3P695. MEMORY AND INFLAMATION: LONG LASTING CONSEQUENCES IN HIPPOCAMPUS FROM AN EARLY HIPER CALORIC DIET

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S3P696. OUABAIN MODULATES THE LEVELS OF MUSCARINIC M3 RECEPTOR IN RETINAL CELL CULTURES

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S3P697. NOTCH ACTIVATION DURING CNS DEMYELINATION-REMYELINATION: TIME- AND CELL TYPE-DEPENDENT LIGAND EXPRESSION

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S3P698. MITOCHONDRIAL DYSFUNCTION IS SUFFICIENT TO INDUCE ASTROCYTE-MEDIATED NEUROTOXICITY. STUDY OF THE MECHANISMS INVOLVED

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S3P699. MUTATIONAL ANALYSIS IDENTIFIES FUNCTIONALLY CRITICAL AMINO ACID RESIDUES WITHIN THE GPM6A CYTOPLASMIC TAILS

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S3P700. LOW OMEGA-6 / OMEGA-3 RATIO IN HYPOPROTEIC MATERNAL DIET FAVORS EPIGENETIC CHANGES IN THE PROGENY´S NEURAL CELLS THAT PROMOTE GENE TRANSCRIPTION

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S3P701. BRAIN-DERIVED NEUROTROPHIC FACTOR EXERTS ANTIOXIDANT AND PROTECTIVE EFFECTS ON ASTROCYTES AND NEURONS TREATED WITH 3-NITROPROPIONIC ACID

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S3P702. AEROBIC EXERCISE IN AGED RATS INCREASES CORTICAL EXPRESSION OF INTRACELLULAR SIGNALING PROTEINS LINKED TO GROWTH, PROLIFERATION AND DEATH

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S3P703. REGULATION OF TRPV4 BY NCS-1 AND THE EFFECTS OF PACLITAXEL AND LITHIUM ON THIS INTERACTION IN NEURONS

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S3P704. LAMININ-COATED MICROSTRUCTURED POLYCAPROLACTONE (PCL) FILAMENTS AS TREATMENT FOR SCIATIC NERVE TRANSECTION

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S3P705. OUABAIN PERFORMING AN AUTOPHAGIC TANGO

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S3P706. AUTOPHAGY AND RETINAL GANGLION CELLS SURVIVAL: THE ROLE OF A1 ADENOSINE RECEPTOR

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S3P707. REGENERATION OF CHICKEN EMBRYO RETINA THROUGHOUT ACTIVATION OF SHH PATHWAY IN ENDOGEN STEM CELLS

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S3P708. EVIDENCE FOR THE ROLE OF THE GLYCOPROTEIN M6A IN DENDRITIC SPINE FORMATION AND SYNAPTOGENESIS

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S3P709. EARLY PHYSICAL EXERCISE MAINTAINS HIGH NUMBER OF CORTICAL AND HIPPOCAMPAL CELLS THROUGHOUT THE SEDENTARY LIFE OF RATS

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S3P710. NEUROPROTECTIVE EFFECTS OF LIDOCAINE IN A RAT SPINAL CORD NEURODEGENERATIVE MODEL INDUCED BY KAINIC ACID

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S3P711. CB1R DEFICIENCY ALTERS NEURONAL MORPHOLOGY AND SYNAPTIC PLASTICITY IN THE ADULT MOUSE HIPPOCAMPUS

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S3P712. TWO REVERSIBLE MODELS OF PERIPHERAL NERVE DEGENERATION AND A POSSIBLE TOOL TO IMPROVE NERVE CONDUCTION

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S3P713. IN VITRO DIFFERENTIATION OF RETINAL GANGLION CELLS FROM STEM CELLS OBTAINED THROUGHOUT DIFFERENT STAGES OF DEVELOPMENT

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S3P714. ANALYSES OF VDAC1 DISTRIBUTION IN HIPPOCAMPUS OF RATS AND EFFECTS OF INTRAHIPPOCAMPAL INJECTION OF DIDS 24 HOURS AFTER NEONATAL ANOXIA

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S3P715. EFFECTS OF PROGESTERONE IN NEONATAL RATS SUBMITTED TO UNILATERAL CEREBRAL HYPOXIA-ISCHEMIA

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S3P716. ATORVASTATIN PREVENTS FROM AB1-40–INDUCED CELL DAMAGE AND DEPRESSIVE-LIKE BEHAVIOR VIA BDNF CLEAVAGE

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S3P717. INTERLEUKIN-13 MODULATES BDNF EXPRESSION: POSSIBLE INVOLVEMENT OF CREB PROTEIN

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S3P718. IL-13 MODULATES EGF PROLIFERATIVE EFFECTS IN RATS RETINAL CELL: A POSSIBLE INVOLVIMENT OF P38 AND BDNF

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S3P719. ROLE OF GABAERGIC-PROOPIOMELANOCORTIN NEURONS IN THE REGULATION OF FOOD INTAKE AND GLYCEMIA

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S3P720. COCAINE AND AEME PROMOTE APOPTOSIS EXTRINSIC PATHWAY ACTIVATION IN RAT'S HIPPOCAMPUS NEURONS IN VITRO

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S3P721. MECP2 REGULATES THE EXPRESSION PATTERN OF LEPTIN RECEPTOR ISOFORMS AND LEPTIN SENSITIVITY

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S3P722. TRANSCRIPTOMICS OF TURTLE'S SPINAL CORD INJURY: CLUES TO UNDERSTAND FUNCTIONAL RECOVERY

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S3P723. CARBACHOL INDUCES RETINAL GANGLION CELLS SURVIVAL IN VITRO: THE INVOLVEMENT OF IL-4 AND BDNF

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S3P724. CRACKING THE NEURON-SPECIFIC TRANSCRIPTIONAL CODE OF THE DOPAMINE D2 RECEPTOR IN THE BRAIN

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S3P725. MECHANISMS OF NEUROPROTECTION VIA MYELIN-AXON INTERACTIONS: ROLE OF MYELIN-ASSOCIATED GLYCOPROTEIN AGAINST GLUTAMATE-MEDIATED OXIDATIVE STRESS

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S3P726. A FRET-BASED APPROACH SUGGESTS ALLOSTERIC ACTIVATION OF MIXED LINEAGE KINASES BY MUTANT HUNTINGTIN

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S3P727. MITOCHONDRIAL DNA REPAIR ACTIVITIES IN HORMONE-RESPONSIVE BRAIN REGIONS IN OVARIECTOMIZED AND ESTRADIOL-TREATED ADULT RATS

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Motor Systems

S3P728. TOTAL RUPTURE OF ACHILLES TENDON INDUCES A DECRESCE IN THE CELL NUMBER OF MOTOR AREA FROM L5 MICE SPINAL CORD

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S3P729. A FAST BRAIN-MACHINE INTERFACE IN THE HEAD-FIXED MOUSE INTEGRATING ARTIFICIAL SENSORY FEEDBACK

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S3P730. DIFFERENT CONNECTIVITY APPROACHES TO EXAMINE MOTOR MEMORY CONSOLIDATION WITH RESTING-STATE FMRI

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S3P731. TOTAL RUPTURE OF ACHILLES TENDON INDUCES NITRERGIC ACTIVATION ON NERVOUS CELLS FROM MICE SPINAL CORD

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S3P732. EYE-TRACKING DATA MODELING WITH SIMPLE DRIVEN HARMONIC OSCILLATORS

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S3P733. MOTOR AND SENSORY INVOLVEMENT IN FINGER TAPPING: AUDITORY SENSORY FEEDBACK PREVENTS IMMEDIATE RESYNCHRONIZATION

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Neural Circuit Physiology

S3P734. CHARACTERIZATION OF THE NEURONAL ACTIVITY IN THE MEDIAL PREOPTIC AREA DURING THE POSTPARTUM PERIOD AND ITS MODULATION BY HYPOCRETINS

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S3P735. COCAINE AND CAFFEINE ALTER EXCITATORY SYNAPTIC PROPERTIES AND INTRACELLULAR [CA2+] IN SOMATOSENSORY THALAMIC NEURONS

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S3P736. MODULATION OF NEURONAL RESPONSES IN THE OLFACTORY CORTEX BY BASOLATERAL AMYGDALA AND CHOLINERGIC INPUTS

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S3P737. NEUROMARKETING: ANALYSIS OF CEREBRAL BEHAVIOR OF CONSUMERS

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S3P738. FOOD RESTRICION-INDUCED ANESTRUS CHANGES OVARIES AND UTERUS MORPHOLOGY AND MELANIN-CONCENTRATING HORMONE PRECURSOR MRNA EXPRESSION OF FEMALE MICE

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S3P739. REWARD CODING AT THE NUCLEUS ACCUMBENS

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S3P740. METHAMPHETAMINE EFFECTS IN A LEPTIN-DEFICIENCY MOUSE MODEL

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S3P741. POTENTIAL ROLE OF HYPOTHALAMIC TANYCYTES MEDIATING THE BLOOD TO BRAIN TRANSPORT OF GHRELIN

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S3P742. EFFECTS OF MGE-GRAFTED PRECURSOR CELLS ON PRE-ICTAL BRAIN OSCILLATION OF PILOCARPINE MODEL OF EPILEPSY

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Neurochemistry and Neuropharmacology

S3P743. EVALUATION OF APPETITIVE 50-KHZ USV CALLS IN AN ACUTE AND CHRONIC LISDEXAMFETAMINE-INDUCED MANIA MODEL

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S3P744. COCAINE-INDUCED CONDITIONING PLACE PREFERENCE IS ENHANCED IN PRENATALLY STRESSED RATS: RELATION BETWEEN PUBERTAL BEHAVIORAL TRAITS AND INDIVIDUAL DIFFERENCES IN ADULT VULNERABILITY TO COCAINE REWARD

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S3P745. ANXIOLYTIC AND ANTIDEPRESSANT-LIKE EFFECTS OF ALPHA-LIPOIC IN A DEPRESSION MODEL INDUCED BY RESERPINE

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S3P746. CLOZAPINE ALONE AND COMBINED WITH ALPHA-LIPOIC ACID IN MODEL OF SCHIZOPHRENIA INDUCED BY KETAMINE IN MICE

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S3P747. CHARACTERIZATION OF PHENOLIC COMPOUNDS OF ETHYL ACETATE FRACTION FROM TABERNAEMONTANA CATHARINENSIS AND ITS POTENTIAL ANTIDEPRESSANT-LIKE EFFECT

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S3P748. PUTATIVE NEM ALKYLATION OF RP2X4 SS3 CYSTEINES PREVENTS THE ZINC POSITIVE ALLOSTERIC MODULATION

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S3P749. ROLE OF 5-HT3 RECEPTORS IN THE MODULATION OF NOCICEPTIVE RESPONSE IN MICE SUBJECTED TO THE MODEL OF EMPATHY FOR PAIN

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S3P750. EXTRACT OF AMAZON FRUIT (MAURITIA FLEXUOSA) EXERTS PROTECTIVE EFFECT AGAINST METHYLMERCURY TOXICITY IN RETINAL CELL CULTURES

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S3P751. EFFECTS OF ACUTE AND LONG-TERM ADMINISTRATION OF GOLD NANOPARTICLES ON MITOCHONDRIAL RESPIRATORY CHAIN COMPLEXES IN RAT BRAIN

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S3P752. OMEGA-3 PARTIALLY REVERSE BRAIN INFLAMMATION IN OBESITY MICE MODEL INDUCED BY HIGH-FAT DIET

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S3P753. THE REINFORCING-LIKE PROFILE OF HIGHER DOSES OF THE DISSOCIATIVE ANESTHETIC KETAMINE IN MALE RATS

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S3P754. THE IMPORTANCE OF ADULTERANTS IN DRUG OF ABUSE: THE CASE OF CAFFEINE IN COCA-PASTE SEIZED SAMPLES

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S3P755. THE MORPHOLOGY OF THE DOPAMINE CELL GROUPS IN THE SUBSTANTIA NIGRA, VENTRAL TEGMENTAL AREA AND RETRORUBRAL FIELD IN THE COMMON MARMOSET (CALLITHRIX JACCHUS)

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S3P756. SOCIAL MODULATION OF PAIN: ARE THE GABAERGIC RECEPTORS IN THE INSULA MODULATING THE HYPERNOCICEPTION IN MICE LIVING WITH A CONSPECIFIC IN CHRONIC PAIN?

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S3P757. CENTRAL CRYOGENIC ROLE OF ENDOGENOUS HYDROGEN SULFIDE IN THE RAT MODEL OF ENDOTOXIC SHOCK

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S3P758. IMPACT OF STRESS IN THE VULNERABILITY TO COCAINE ADDICTION: ROLE OF COFILIN DURING THE ACQUISITION OF COCAINE SELF-ADMINISTRATION IN NUCLEUS ACCUMBENS

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S3P759. ISRADIPINE AND P. NIGRIVENTER SPIDER VENOMS: PNTX3-4 OR PNTX3-6 ARE NEUROPROTECTIVE IN A MOUSE MODEL OF HUNTINGTON´S DISEASE

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S3P760. NEUROBIOLOGICAL ACTIVITY OF THE LEPIDIUM MEYENII LEAF EXTRACT ON PC12 CELLS

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S3P761. ALCOHOL CONSUMPTION IN ADOLESCENT RATS IS UNAFFECTED BY PREVIOUS METHYLPHENIDATE EXPOSURE

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S3P762. RELATIONSHIP BETWEEN THYROID HORMONES LEVELS AND ALCOHOL CONSUMPTION

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S3P763. NEUROCHEMICAL AND FUNCTIONAL CHARACTERIZATION OF A PRIMARY CULTURE OF SEROTONINERGIC NEURONS

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S3P764. PDZ DOMAINS 1 AND 2 OF PSD-95 ARE NECESSARY FOR MEMORY CONSOLIDATION

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S3P765. CHARACTERIZATION OF THE ENDOCANNABINOID SYSTEM IN THE DEVELOPMENT OF THE AVIAN RETINA: POSSIBLE RELATIONSHIP WITH THE DOPAMINERGIC SYSTEM

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S3P766. ANTIPSYCHOTIC EFFECTS OF HYDROALCOHOLIC EXTRACT OF RED PROPOLIS IN MURINE MODELS OF SCHIZOPHRENIA

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S3P767. ALPHA-TERPINEOL REDUCES CANCER PAIN VIA OXIDATIVE STRESS MODULATION

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S3P768. Γ-TERPINENE MODULATES OPIOID AND COLINERGIC SYSTEMS

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S3P769. COCAINE ABSTINENCE CHANGES M1- M2 AND M4 MACHRS EXPRESSION

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S3P770. ACTIVATION OF CANNABINOID CB1 RECEPTOR PREVENTS ANXIOGENIC-LIKE EFFECT AND DECREASE IN GABA LEVELS INDUCED BY ACUTE RESTRAIN STRESS

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S3P771. INVOLVEMENT OF SEROTONERGIC SYSTEM IN THE ANXIOLYTIC LIKE EFFECT OF NEW PIPERAZINE DERIVATIVE-LQFM104

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S3P772. NEUROPROTECTIVE EVIDENCE OF ALPHA-LIPOIC ACID ON THE MEMORY DEFICIT INDUCED BY CORTICOSTERONE

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S3P773. NEUROPEPTIDE S EFFECTS ON A MODEL OF ATTENTION-DEFICIT HYPERACTIVITY DISORDER

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S3P774. COCAINE COMPULSIVE BEHAVIOR AND ITS IMPLICATIONS IN THE CHOLINERGIC MUSCARINIC SYSTEM

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S3P775. GUANOSINE-PROMOTED NEUROPROTECTION IS DEPENDENT ON ADENOSINE A2A RECEPTORS EXPRESSION: EVIDENCE OF GUANOSINE INTERACTION WITH ADENOSINE A1/A2A RECEPTORS HETEROMERS

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S3P776. EFFECTS OF MATERNAL SEPARATION AND OF EXPOSURE TO INTERMITTENT ETHANOL BINGE DRINKING IN ADOLESCENT MALE WISTAR RATS: EFFECTS ON BEHAVIOR AND ON PARAMETERS OF OXIDATIVE STRESS

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S3P777. EFFECTS OF TERPENOIDS ON C. ELEGANS NEUROMUSCULAR TRANSMISSION

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S3P778. STUDY OF DORSAL RAPHE NUCLEUS AS A NEURAL SUBSTRATE FOR MELANIN-CONCENTRATING HORMONE (MCH) PRO-DEPRESSIVE ACTION IN RATS

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S3P779. EFFECT OF MIRTAZAPINE AND LIPOIC ACID ASSOCIATION ON BRAIN-DERIVED NEUROTROPHIC FACTOR CONCENTRATION IN A DEPRESSION MODEL INDUCED BY CORTICOSTERONE

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S3P780. INVOLVEMENT OF PHOSPHATIDYLINOSITOL-3 KINASE GAMMA IN THE ANTICONVULSANT AND NEUROPROTECTIVE EFFECTS OF CANNABIDIOL

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S3P781. DAMAGE IN THE BLOOD-BRAIN BARRIER, PLACENTAL BARRIER AND BEHAVIORAL CHANGES IN LONG-TERM IN ANIMALS INDUCED INFECTION BY LIPOPOLYSACCHARIDE IN THE PRENATAL PERIOD

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S3P782. DEVELOPMENT OF THE VISUAL SYSTEM IN A HYPOTHYROIDISM MODEL

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S3P783. DAMAGE ASSOCIATED MOLECULAR PATTERN HMGB-1 EFFECTS IN NEURONAL SURVIVAL AND PROPAGATION OF REACTIVE GLIOSIS

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S3P784. EFFECT OF CHRONIC INFLAMMATORY CONDITIONS ON MICROGLIAL CELL MORPHOLOGY AND FUNCTION IN THE HIPPOCAMPUS OF ADULT APP/PS1 MICE

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S3P785. THYROID HORMONES INDUCE NEURITE OUTGROWTH AND SYNAPTOGENESIS OF CEREBRAL CORTICAL NEURONS

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S3P786. SYNERGISTIC EFFECTS OF THE JOINT ADMINISTRATION OF KETAMINE AND DIFFERENT ANTIDEPRESSANT CLASSES ON BEHAVIOR AND OXIDATIVE STRESS

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S3P787. EFFECTS OF CHRONIC EXERCISE ON THE IMMUNE SYSTEM: IMPLICATION OF THE CANNABINERGIC SYSTEM

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S3P788. ACUTE AND CHRONIC EFFECTS OF TYPE 1 DIABETES MELLITUS ON THE AVERSIVE MEMORY AND NEUROIMMUNOMODULATION IN ADOLESCENCE RATS

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S3P789. PROLACTIN MEDIATES NEUROPROTECTION AGAINST EXCITOTOXICITY IN PRIMARY CELL CULTURES OF HIPPOCAMPAL NEURONS VIA ITS RECEPTOR

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S3P790. BEHAVIORAL AND NEUROCHEMICAL PROFILES OF KETAMINE IN RESPONSE TO LIPOLYSACCHARIDE (LPS) EXPOSURE DURING EARLY NEONATAL PERIOD

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S3P791. EXPRESSION AND FUNCTIONAL ROLE OF NEURONAL ALPHA7 NICOTINIC RECEPTOR IN HUMAN NK CELLS

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Sensory Systems

S3P792. NEURONAL CODING OF EXPECTANCY SIGNALS IN THE CEREBRAL CORTEX INDUCED BY REPETITIVE TACTILE STIMULI

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S3P793. USING MULTI-VIBRISSAE TACTILE STIMULATION TO UNVEIL NEURONAL CODING IN THE SECONDARY SOMATOSENSORY CORTEX

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S3P794. PROTECTIVE EFFECT OF THE EUTERPE OLERACEA DURING THE DEVELOPMENT OF DIABETIC RETINOPATHY IN ANIMAL MODEL

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S3P795. POLARIZATION VISION IN GOLDFISH: DO THEY STARTLE TO POLARIZED LIGHT LOOMS?

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S3P796. OBJECTIVE ELECTROPHYSIOLOGICAL MEASURES OF THE LOMBARD EFFECT

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S3P797. DIFFERENTIAL RESPONSE OF THE RETINAL NEURAL CODE WITH RESPECT TO THE SPARSENESS OF NATURAL IMAGES

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S3P798. ROLE OF EYE MOVEMENTS DURING MOTOR LEARNING BY IMITATION

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S3P799. RECOGNISING THE INFLUENCE OF INTUITIVE KNOWLEDGE BY RECORDING EYE MOVEMENTS

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S3P800. ORGANOTYPIC RETINAL EXPLANT CULTURES AS A NOVEL AND VERSATILE IN VITRO MODEL FOR DIABETIC RETINOPATHY

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S3P801. SENSING TACTILE REGULARITY: A NOVEL 2-ALTERNATIVE FORCED-CHOICE TASK IN THE RAT

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S3P802. IN VIVO RECORDINGS FROM THE OPTIC NERVE OF RAT

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S3P803. CB2 RECEPTOR EXPRESSION AND ENDOCANNABINOID ENZYMES IN RAT RETINA AND ITS MODIFICATIONS AFTER CONTINUOUS ILLUMINATION

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S3P804. LOW-VOLTAGE ACTIVATED CALCIUM CURRENT IN THE VESTIBULAR AFFERENT NEURONS OF THE RAT

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S3P805. SELECTIVE ATTENTION TO VISUAL STIMULI USING AUDITORY DISTRACTORS IS ALTERED IN ALPHA-9 NICOTINIC RECEPTOR SUBUNIT KNOCK-OUT MICE

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S3P806. VESTIBULAR DEVICE FOR ORIENTATION CORRECTION IN BALANCE DISORDERS AND IN MICROGRAVITY

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S3P807. CANNABINOID RECEPTOR ACTIVATION REGULATES NON-RECIPROCAL INHIBITORY FEEDBACK ONTO OFF BIPOLAR CELLS OF RAT RETINA

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S3P808. MENTHOL-INDUCED HYPERTHERMIA IN RATS IS ATTENUATED AFTER DAILY ADMINISTRATION THROUGH A SHIFT IN THE THERMOREGULATORY EFFECTOR RECRUITMENT

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Synaptic Transmission, Excitability and Glia

S3P809. A CAMKII ENDOGENOUS INHIBITOR REGULATES HOMEOSTATIC SYNAPTIC PLASTICITY IN THE HIPPOCAMPUS

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S3P810. NMDA EFFICIENTLY EVOKES DENDRITIC RELEASE OF NEUROPEPTIDES: A QUANTITATIVE REAL TIME ASSESSMENT

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S3P811. EFFECTS OF LPS-INDUCED TOLL-LIKE RECEPTOR 4 (TLR4) UPREGULATION ON SPINAL MOTONEURON RESPONSE TO PERIPHERAL AXOTOMY

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S3P812. PERSISTENT HYPERALGESIA AND SPINAL CORD GLIAL REACTION FOLLOWING NEONATAL NOCICEPTIVE STIMULATION

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S3P813. TIGHT COUPLING OF ASTROCYTE ENERGY METABOLISM TO EXCITATORY ACTIVITY REVEALED BY GENETICALLY ENCODED FRET NANOSENSORS

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S3P814. GLYCINE RECEPTORS IN MESENCEPHALIC TRIGEMINAL NEURONS OF THE RAT: ELECTROPHYSIOLOGICAL AND MORPHOLOGICAL STUDIES

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S3P815. DISTRIBUTION OF HIPPOCAMPAL CONNEXIN 43 AND ITS ROLE IN CELL DEATH AFTER NEONATAL ANOXIA

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S3P816. THE INWARD RECTIFIER POTASSIUM CURRENT IKIR REGULATES THE INTRINSIC OSCILLATORY PROPERTIES OF THALAMOCORTICAL NEURONS

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S3P817. FUNCTIONAL PROPERTIES AND ION CHANNELS MEDIATING ACETYLCHOLINE RELEASE AT THE MOUSE MEDIAL OLIVOCOCHLEAR-OUTER HAIR CELL SYNAPSE AT THE ONSET OF HEARING

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S3P818. FAST HOMEOSTATIC SYNAPTIC SCALING IN ACUTE HIPPOCAMPAL SLICES

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S3P819. CHANGES IN THE KINETIC PROPERTIES OF THE A9A10 HAIR CELL NICOTINIC RECEPTOR INCREASE THE LEVEL OF OLIVOCOCHLEAR INHIBITION IN AUDITORY SYNAPSES

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S3P820. CHRONIC STRESS ALTERS SYNAPTIC EXCITATORY-INHIBITORY RATIO IN AN INTERLEUKIN-6 TRANS-SIGNALING-DEPENDENT MANNER IN THE PREFRONTAL CORTEX OF THE MOUSE

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Theoretical and Computational Neuroscience

S3P821. MOTION DIRECTION SELECTIVITY IN CENTRAL AND PERIPHERAL RETINAL GANGLION CELLS IN A DIURNAL RODENT

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S3P822. A SIMPLIFIED MODEL FOR ELECTROPHYSIOLOGICAL ACTIVITY

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S3P823. THE RAT VIBRISSA AS A MECHANICAL SENSOR: FREQUENCY RESPONSE ANALYSIS OF THE VIBRISSAL-FOLLICLE-NERVE SYSTEM

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S3P824. ELECTRORETINOGRAPHY: ANALYSIS BY PERMUTATION ENTROPY

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S3P825. IDENTIFICATION OF FUNCTIONAL INTERCONNECTED NEURONS

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S3P826. CROSS FREQUENCY COUPLING ANALYSIS OF LOCAL FIELD POTENTIALS RECORDED FROM RAT HIPPOCAMPAL AND PARAHIPPOCAMPAL REGIONS DURING BEHAVIORAL TASKS

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S3P827. MECHANISMS FOR PATTERN SPECIFICITY OF DEEP-BRAIN STIMULATION IN PARKINSON'S DISEASE

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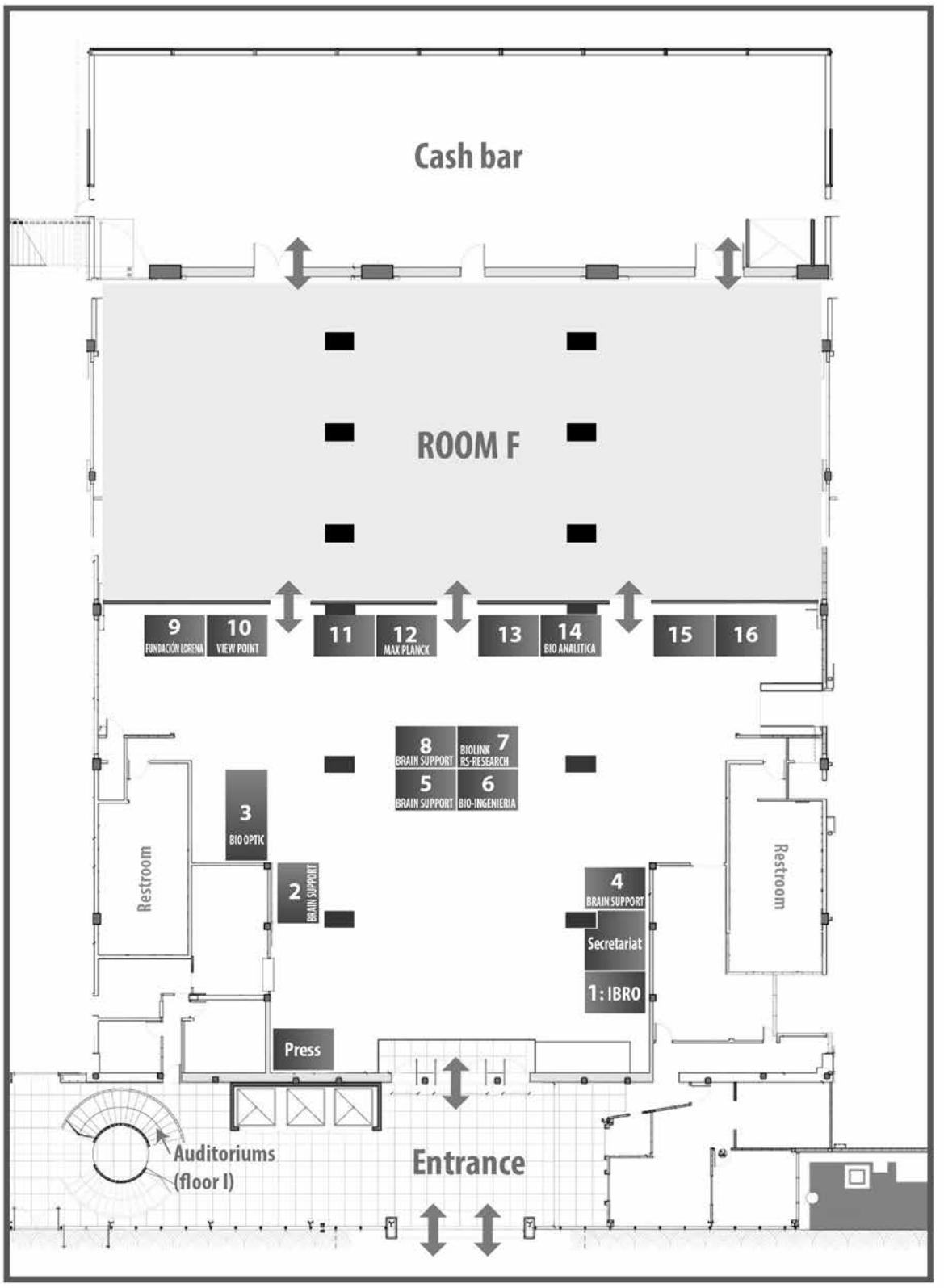
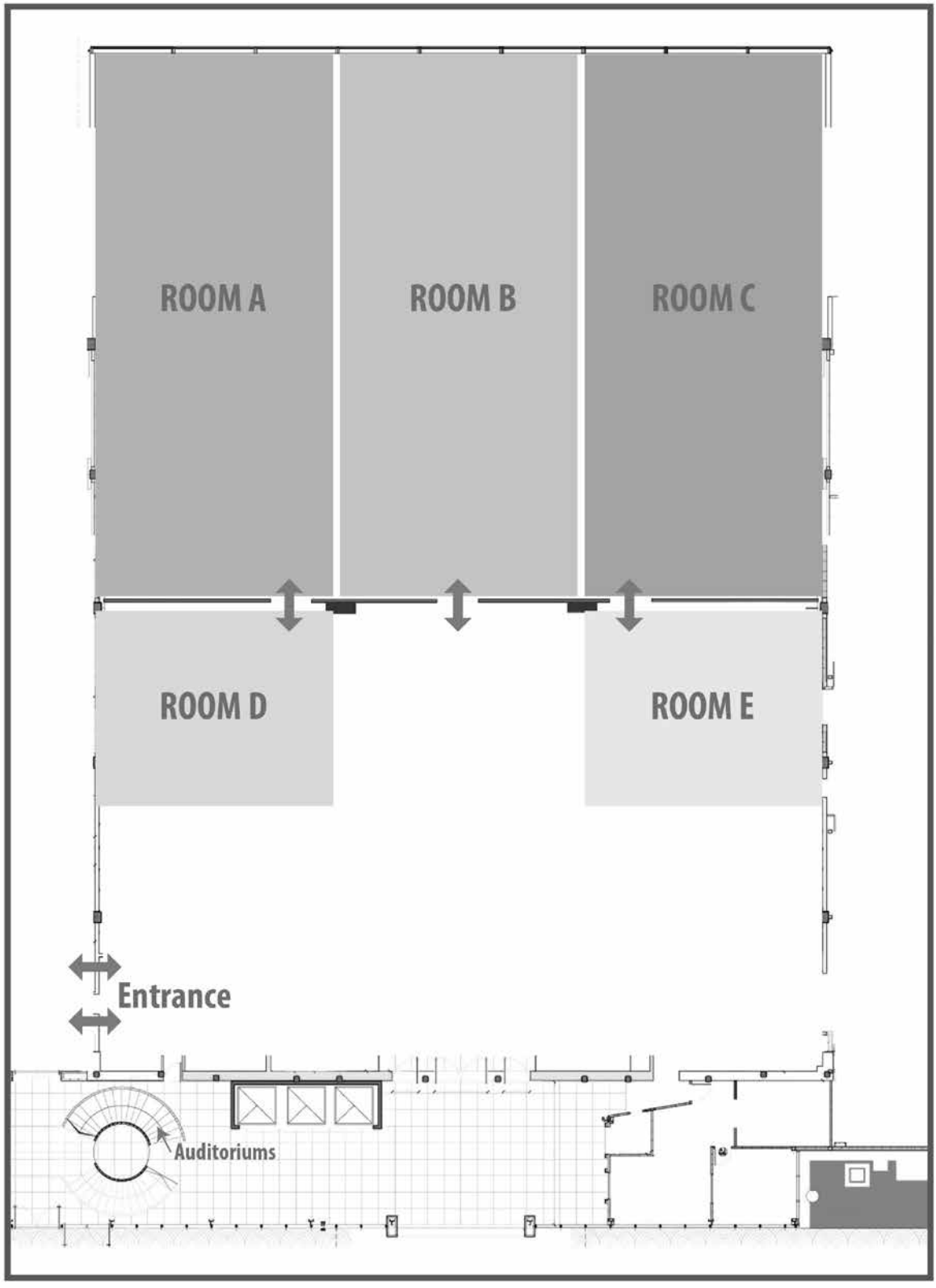
FLOOR AND EXHIBITION PLAN

GROUND FLOOR



FIRST FLOOR



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