PREFACE

Neuroglia represents the major nerve cell group in the brain. Nevertheless, for a long time it was only considered a support element for neural function. At present, neuroglia research is fundamental to learn about the physiology of the central nervous system. Consequently, the organization of the 2nd Symposium on Neuroglia Physiology and Pathology represents an opportunity for our University to promote and disseminate the most recent findings made in this field by national and international leading researchers. In this occasion, our Institute has the honor of hosting this event. In this edition the students will have a chance to present and discuss their results directly with the speakers. Finally, we are grateful to our University, CONACYT, SFN, and IBRO for their valuable support which made possible the organization of this Symposium. Our best wishes for the organizers and participants; we are sure that this event will be successful and will become a reference in the field.

DR. ALFREDO VARELA ECHAVARRIA
DIRECTOR – INB-UNAM
ORGANIZING COMMITTEE

Dr. Pavel Montes de Oca Balderas
Instituto Nacional de Neurología y Neurocirugía

Dr. Octavio C. García González
Facultad de Psicología UNAM

Dr. Daniel Reyes Haro
Instituto de Neurobiología UNAM

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Lic. Felipe P. Pedroza Montes de Oca
ACKNOWLEDGEMENTS

The Organizing Committee deeply appreciates Dr. Alfredo Varela support for the organization of this event. Dr. Varela back up this event since the first edition (2016), helping us to obtain the funds to make it happen. Two years later, after the success of the previous Symposium, the Organizing Committee was able to obtain financial support for the second edition. Special thanks to Dr. Ataúlfo Martínez-Torres for giving all the facilities to make this Symposium possible. We are also grateful to Dr. Maricela Luna and Dr. Aurea Orozco for financial arrangements to partially fund the invitation of the international speakers. Special acknowledgements to Universidad Nacional Autónoma de México (Posgrado-UNAM, PAEP-UNAM, INB-UNAM, CAC-UNAM) and Consejo Nacional de Ciencia y Tecnología (CONACYT; grant 292944) for providing the national funding. We are also grateful to Society of Neuroscience (SFN) and the International Brain Research Organization (IBRO) for international funding for this symposium. Also thanks to Technical Support & Administrative Staff. Finally, thanks to the speakers, researchers and students participating in the Symposium.
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October 4th, 2018
Centro Académico Cultural (CAC)
Universidad Nacional Autónoma de México
Campus Juriquilla, Querétaro, QRO

CIRCUIT-SPECIFIC SYNAPTIC REGULATION BY ASTROCYTES

DR. ALFONSO ARAQUE
UNIVERSITY OF MINNESOTA

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<td>Reyes-Ortega Pamela, Varman Durairaj Ragu, Martínez Torres Ataúlfo, Reyes Haro Daniel</td>
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LECTURE 2
17:00 – 18:00 pm
October 4th, 2018

ASTROCYTE DYSFUNCTION AND NEURODEGENERATION IN DOWN SYNDROME

DR. JORGE BUSCIGLIO
UNIVERSITY OF CALIFORNIA, IRVINE
# SYMPOSIUM-TALKS
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October 5th, 2018

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LECTURE 1  
12:00 – 13:30 pm  
October 5th, 2018

HOW DO GLIAL CELLS CONTROL CNS FUNCTION?

DR. AXEL NIMMERJAHN  
SALK INSTITUTE, LA JOLLA, USA

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THE PATHOPHYSIOLOGY OF CNS WHITE MATTER INJURY DEPENDS ON THE INSULT: ANOXIA VS. AGLYCEMIA VS. ISCHEMIA

PROF. BRUCE R. RANSOM
UNIVERSITY OF WASHINGTON
SYMPOSIUM ABSTRACTS (SPEAKERS)

OCTOBER 4TH, 2018
A novel GABA<sub>A</sub> receptor expressed in oligodendrocytes

Arellano Ostoa Rogelio, Ordaz Ramos Rainald Pablo, Cisneros-Mejorado Abraham, García García Cindy Lucero, Garay Rojas Edith. Departamento de Neurobiología Celular y Molecular, Instituto de Neurobiología, UNAM Campus Juriquilla, Queretaro, Mexico.

The γ-aminobutyric acid (GABA) is a key neurotransmitter in the central nervous system, GABA acts through activation of pentameric receptor-channels permeable to Cl<sup>-</sup> ions, known as GABA<sub>A</sub> receptors, and through metabotropic receptors named GABA<sub>B</sub>. In oligodendrocytes (OL), GABA<sub>A</sub> receptor expression is controlled by their interaction with neurons, thus, it has been proposed a role of GABAergic signaling during the myelination process. Functional characteristics of the GABA-response in OL from the rat optic nerve, have indicated a specific subunits combination conforming the GABA<sub>A</sub> receptor, being different to receptors expressed in other neural cells. Determination of the subunits combination expressed in OL is essential for its pharmacologic and genetic control. Here, GABA<sub>A</sub> subunits (α3, β2, β3, γ1-γ3) were cloned from OL and, heterologously expressed in *Xenopus laevis* oocytes in the distinct combinations, then GABA-response for each combination was studied electrophysiologically. Results showed that α3β2γ1 co-expression, mimicked the functional and pharmacological pattern described for the endogenous response in OL. For example, this ensemble compared with the endogenous receptor, showed similar sensitivity to GABA, as well as to a variety of positive and negative allosteric modulators; including, a distinctive robust potentiation by butyl β-carboline-3-carboxylate, a β-carboline that has only a weak effect on the GABA<sub>A</sub> receptor expressed in cortical neurons. The putative GABA<sub>A</sub> receptor α3β2γ1 represents a novel subunits combination expressed in OL, which displays distinctive pharmacological characteristics, this information might be used to analyze in detail the GABAergic signaling role in myelination, and its probable involvement in various pathologies.
Montes de Oca Balderas Pavel Departamento de Neurociencia Cognitiva, Instituto de Fisiología Celular, UNAM y Unidad de Neurobiología Dinámica del Dpto. de Neuroquímica, INNN.

In the framework of the neurocentric theory the role of glia in information handling within the Central Nervous System (CNS) was dismissed for almost a century. Accordingly, the NMDA receptor (NMDAR), a critical player in synapse communication, neuronal plasticity and CNS function, was mainly studied in the neuronal context. Nevertheless, this receptor is widely expressed in cells within and beyond the CNS including astrocytes, whose role, as that of other neuroglial cells, has been remodeling since almost three decades ago. It turns out that in order to have an integrative perspective of the CNS function it is not possible to ignore neuroglial cells. In particular astrocytes have been found to play a relevant role in different aspects of CNS function, including information handling in the tripartite synapse model, as they “listen” to neuronal synaptic communication, regulate it and respond at the cellular and syncititial level.

Despite the expression and function of ionotropic glutamate NMDAR in astrocytes was matter of different controversies and apparent contradictions for almost 30 years, today it is known that these cells have functional receptors, although its role has not been deeply investigated. One of the main reasons for these controversies and apparent contradictions was the a priori expectation that this receptor should work in a similar manner to its neuronal counterpart. However, the diversity of subunits that may be assembled into NMDAR results in receptors with different biophysical, transport and signaling properties, as it is the case of the NMDAR in astrocytes.

In this talk I briefly outline NMDAR general features, pinpointing those that are the source of NMDAR diversity and complexity. Then, I will show our results that indicate that the NMDAR in cultured astrocytes mainly acts as a metabotropic-like flux-independent receptor, function that still has to be confirmed in tissue astrocytes, but that some reports have demonstrated for neuronal cells. Given the complex molecular nature of NMDAR, its critical role and the relevance of astrocytes, the study of astrocytic NMDAR promise to provide further understanding of CNS physiology and pathology.

Acknowledgements: Proyectos CONACyT de Repatriación # MOD-ORD-1-09-PCI-010-03-10 y de Ciencia Básica #132706.
RM-S003

Modulation of neuroglial cells in the dentate gyrus during depression like behavior.

Ramírez-Rodríguez GB. Instituto Nacional de Psiquiatría “Ramón de la Fuente Muñiz”, Ciudad de México, México.

Stress is a key factor for some neuropsychiatric disorders including depression. In fact, several types of stressors have relevant impact on the brain plasticity with important alterations in behavior and cognition. Among the type of stressors widely studied exists the chronic mild stress (CMS). The CMS paradigm has proven effects on plasticity and behavioral alterations related to depression. Moreover, CMS affects neuroplasticity but has also shown impressive negative effects on the microenvironment of the dentate gyrus (DG). Then, CMS also affects the generation of new neurons in the DG of the hippocampus region of the limbic system implicated for learning and memory but also for mood related behavior. Interestingly, some clinical and preclinical studies have pointed to the direction of alterations in glial cells. In this regard, the effects of key modulators of the neuroglial cells (such as environmental enrichment, melatonin, antidepressant drugs and the repetitive transcranial magnetic stimulation) in the context of adult hippocampal neurogenesis in a murine model of depression will be presented.
Astrocytes subdomains respond independently in vivo

López-Hidalgo Mónica, Vered Kellner, Schummers James. Facultad de Medicina, Universidad Autónoma de Querétaro, Qro., México.

Astrocytes are a prominent non-neuronal cell type in the brain that play important roles in modulating the activity of neural circuits. Astrocytes cover the gray matter with bushy process where they detect and respond to neuronal activity with intracellular calcium changes. In particular, astrocytes respond to sensory stimulation with increases in somatic calcium concentration, although the spatial relationship of these calcium events at the process remains unknown. Furthermore, it remains unclear whether astrocytes behave as a single functional unit that integrates all of the inputs, or if multiple functional subdomains reside within an individual astrocyte. Here we utilized the columnar organization of ferret visual cortex to analyze the spatial scale of neuron-astrocyte communication in vivo. To this end, we monitored calcium activity throughout the bushy processes of visual cortical astrocytes using a sensitive calcium indicator (GCaMP6s) and a two-photon microscope during visual stimulation with parametrically varied visual stimuli. We found that there is strong visually-driven calcium activity within the entire extent of astrocyte processes, which is specific to the visual input. Furthermore, that astrocyte responses to neural circuit activity are dominated by functional subdomains that respond locally and independently to neuronal activity with high spatial precision and are therefore suited to communicate with neuronal circuits at a fine spatial scale.
A postnatal period of vascular and glia cell expansion in the auditory brainstem of rats and mice: Implications for neuronal development and synaptic refinement before the onset of hearing.

Rodríguez-Contreras Adrián¹, Lingyan Shi¹,², Geng Pan¹, Chaya Sussman¹, Quetanya Brown¹, Daphne Chang¹, Grace Tsui¹, Bao Vuong¹. ¹The City University of New York, City College, Biology Department and Center for Discovery and Innovation. 85 Saint Nicholas Terrace, New York, NY 10031. ²Present address: Chemistry Department, Columbia University, New York, NY.

Although microglia have recently been implicated as key regulators of activity-dependent synapse elimination in somatosensory and visual systems, little is known about their roles in synaptic refinement and developmental plasticity in the auditory system. This is important to determine if the mechanisms of microglia function are universal or dependent on brain region and life stage. In this study we examined microglia development in the auditory brainstem circuit formed between the medial nucleus of the trapezoid body (MNTB) and the lateral superior olive (LSO) in rats and mice. Based on previous observations in the somatosensory system (Arnoux et al. 2013. Glia 61, 1582-1594), we first examined the relationship between microglia cells and vascular development using Isolectin B4 and EdU histochemistry in rats, and with fluorescent reporters in mTmG and CX3CR1-GFP mice, which express red and green fluorescent proteins in perivascular and microglial cells, respectively. We report that during the postnatal stage between birth (P0) and P10 there is a correlated increase in vascular volume and microglia density in the auditory MNTB-LSO circuit. We hypothesize that microglia cells use the vascular niche to undergo expansion in preparation for the synaptic refinement of the MNTB-LSO circuit. Ongoing experiments are testing this hypothesis using 1) transcranial two-photon microscopy in neonate mice that express fluorescent reporters to determine the dynamic behavior of microglia cells associated with blood vessels in vivo, and 2) by developing engulfment assays to determine if microglia cells are involved in synaptic refinement in the MNTB-LSO connection.

Acknowledgments. Supported by a Harvey L. Karp award in the Sciences.
Circuit-specific synaptic regulation by astrocytes.

Araque Alfonso. Department of Neuroscience, University of Minnesota

Astrocytes respond to synaptically released neurotransmitters and release gliotransmitters that regulate synaptic transmission and plasticity. The signaling exchange between astrocytes and neuronal synaptic elements have led to the establishment of the functional concept of tripartite synapse. Combining calcium imaging techniques and multiple whole-cell recordings from neurons in different brain areas, our lab aims to determine the spatial as well as the synapse-specific properties of the astrocyte-mediated synaptic regulation in tripartite synapses. We have recently shown that astrocyte-neuron interaction at tripartite synapses is circuit specific (Martin et al., Science 2015), suggesting that the bidirectional astrocyte-neuron signaling selectively occurs between specific subpopulations of astrocytes, neurons, and synapses. In line with these findings, I will present the most recent evidence obtained in the lab indicating that the activity of astrocytes induces a differential synapse-specific regulation of excitatory and inhibitory synapses in the amygdala. Selective activation of astrocytes by DREADDs or endocannabinoids differentially regulates neurotransmission in the CentroMedial (CeM) amygdala, the major effector amygdala subnucleus. Astrocyte stimulation depresses excitatory glutamatergic transmission by activating A1 adenosine receptors and enhances inhibitory GABAergic transmission by activating A2A adenosine receptors. Consistent with these differential synaptic effects, astrocyte stimulation inhibits in vivo CeM neuronal firing rate and reduces the freezing responses in the auditory fear conditioning paradigm. These results indicate that astrocyte activity influences animal behavior through synapse-specific regulation of neuronal activity.

Acknowledgments: NIH-NINDS (R01NS097312-01) and Human Frontier Science Program (RGP0036/2014).
Astrocyte dysfunction and neurodegeneration in Down Syndrome

Busciglio Jorge, Demuro Angelo, Lioudyno María. Department of Neurobiology and Behavior, University of California, Irvine, CA, USA.

Normal astrocytic function is essential to effectively support neuronal survival, synaptic transmission and synaptic integrity. Several lines of evidence indicate that primary astrocytes with trisomy 21 (Down syndrome, DS) exhibit functional alterations that directly impact neuronal function and survival including defects in 1- mitochondrial function; 2- protein secretion, and 3- calcium homeostasis. all of which critically affect neuronal development and function. Disruption of calcium homeostasis is a common feature of several neurological disorders including Alzheimer's disease, which has a very high prevalence in older persons with DS. Our results showed that although trisomic and euploid astrocytes maintained similar resting cytosolic calcium levels and store contents, trisomic astrocytes were severely deficient in IP₃R-dependent global and local calcium signaling. The number of regions containing clusters of local calcium signals, as well as the number of individual events per cluster were significantly smaller in trisomic astrocytes. Analysis of local calcium signals revealed that events generated by DS astrocytes were significantly lower in amplitude, consistent with a reduced number of activated IP₃R channels within the cluster. The results suggest that deficient IP₃R mediated calcium responses may underlie, at least partially, the reduced ability of trisomic astrocytes to support neuronal function and survival.
SYMPOSIUM ABSTRACTS
POSTERS

OCTOBER 4TH, 2018
Dehydration-induced anorexia increases microglia density in the rat prefrontal cortex

Reyes-Ortega Pamela, Varman Durairaj Ragu, Martinez Torres Ataúlfo, Reyes Haro Daniel Departamento de Neurobiología Celular y Molecular. Instituto de Neurobiología - UNAM Campus Juriquilla, Querétaro, Mexico.

Anorexia involves restrictive caloric intake that induces profound weight loss. Microglia deficits were recently associated to prefrontal cortex. Our aim was to test if dehydration induced anorexia (DIA) disturbs microglia density. Three independent experimental series of seven female Wistar rats (180-200g) per group were used for this study: a) Control: received food and water ad libitum, b) DIA: received saline solution (2.5 % NaCl) and food ad libitum, c) Forced Food Restricted (FFR) group received water and the same amount of food as the DIA group. Subsequently, histological sections (30 μm) were immunostained with Iba-1, a microglial marker. Microglial density as well as TNFα and IL-6 expression were estimated for all experimental groups. Microglia/nuclei ratio was significantly increased in medial prefrontal cortex of DIA and FFR groups (Control 0.10±0.01, DIA 0.18±0.02, FFR 0.22±0.03; p = 0.003; n=7). Likewise, reactive/resting microglia ratio was significantly increased for DIA and FFR (Control 0.70±0.06, DIA 2.14±0.42, 1.89±0.40; p = 0.04; n=7). Additionally, Western blots showed that DIA and FFR increase the expression of the TNFα (DIA 1.98±0.1, FFR 1.81±0.08; p = 0.02; n = 3) and IL-6 (DIA 1.85±0.05, FFR 1.69±0.09; p = 0.04; n = 3). We conclude that DIA and FFR increase microglia density and expression of TNFα and IL-6 in prefrontal cortex.

Acknowledgements: Thanks to N. Hernández-Ríos, AE Espino-Saldaña, Dr. A. Castilla, MVZ M. García-Servín and M.C. L. Casanova. PRO has a CONACYT scholarship (554231) through PDCB. This work was supported by PAPIIT-UNAM (IN200913, IN201915, IN205718) to AMT and DRH.
DNA methylation and gene expression of astrocytes before, during and after oxygen and glucose deprivation

Ponce Isaac, Romo-Tovar Luis. Departamento de Patología Molecular, Instituto de Fisiología Celular, Universidad Nacional Autónoma de México, Ciudad de México, México

Epigenetic mechanisms such as DNA methylation are well known regulators of genetic expression and they play key roles in the development of neurodegenerative diseases, mainly through a substantial transcriptional regulation of active and inactive promoters and by modifying transcription elongation and splicing in CpG islands located intra and inter-genically. It is also widely recognized that astrocytes, that are critical regulators of neuronal function, play a crucial role in neurovascular-related disorders like ischemic stroke. However, few studies have addressed at the molecular resolution the overall genetic and epigenetic changes of these complex phenomena, and in events like reperfusion damage that occurs after ischemic stroke, these processes are practically unknown. We performed RNA-seq and methylated DNA immunoprecipitation sequencing (MeDIP-seq) analysis of cultured human astrocyte cells derived from grade I non-tumorigenic glioblastoma subjected to oxygen and glucose deprivation (OGD), in order to establish a relationship between DNA methylation and gene expression under normoxia, OGD and recovery. We identified several genomic features including proximal and distal promoters whose methylation levels change not only during OGD but also after 8 h of recovery that showed statistically significant differences in both; high (house-keeping and ubiquitous genes) and low (cell lineage-specific genes) CG promoters. Moreover, DNA methylation remodeling was correlate with gene expression of several genes under OGD and recovery, and the organization of the transcriptome and methylome resulted different under normoxia, OGD and recovery. These results can help to elucidate the overall transformation of cells in terms of transcription and DNA methylation in pathological occurrences involving ischemia and characterize the damage that occurs during reperfusion at the genomic scale that has been incompletely described until now.
Prolactin protects rat cortical astrocytes against oxidative stress

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Astrocytes provide protection and metabolic support for neurons under oxidant conditions. Prolactin (PRL) is a stress-related hormone that limits gliosis and degeneration of the neural retina. In this work, we investigated whether PRL protects cortical astrocytes against oxidative stress and cell death. Primary cultures of cortical astrocytes were isolated from the brain of neonatal rats, and characterized by an immunocytochemistry for GFAP. The long isoform of the PRL receptor was detected in cortical astrocytes by qRT-PCR. The astrocytes were incubated with increasing concentrations of PRL during 24 h, and exposed to oxidative stress induced by hydrogen peroxide (H2O2) for 3 h. PRL inhibited the H2O2-induced cytotoxicity in cortical astrocytes in a dose-dependent manner, as evaluated by the MTT assay. In addition, PRL increases the expression of its receptor and GFAP under oxidative conditions, as well as increases the expression of the Mn and Cu/Zn superoxide dismutases, peroxirredoxins 1 and 6, glutathione peroxidase 1 and glutathione S-transferases μ1 under basal conditions, and these changes were exacerbated after H2O2-induced oxidative stress. Catalase, glutathione S-transferases μ3 and hemoxygenase 1 expression increased only by effect of H2O2. These changes were evaluated by qRT-PCR. PRL increased the total antioxidant capacity of the cultures in basal and oxidative conditions. PRL receptor activation was determine through the evaluation of phosphorylated forms of STAT5 and STAT3, by Western blot. These results indicate that PRL can act through its receptor to protect astrocytes against injuries due to oxidative stress.

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Cerebellar GABAρ3 expression is reduced in the valproic acid model of autism

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Autism is a neurodevelopmental disorder that display an imbalance of excitatory/inhibitory activity and cerebellar dysfunctions, including a reduced density of Purkinje cells (PCs), and the abnormal expression of GABA_A receptor subunits. GABAρ3 forms homomeric receptors with high affinity for the agonist (GABA EC_{50} about 3 μM) and little desensitization. This GABA_A subunit is expressed in the early postnatal development of the murine cerebellum and is considered potentially relevant to modulate developmental signaling cues. Thus, we tested if the expression of GABAρ3 was modified by prenatal exposure to valproic acid (VPA), a well-known animal model of autism. Pregnant mice were injected intraperitoneally at embryonic day 12.5 with either VPA (500 mg/kg) or saline solution (0.9%) (CTL). Latency to reach the nest is increased in VPA-mice suggesting sensorimotor deficits. Western blot from cerebella of VPA-mice revealed a reduced expression of calbindin and GABAρ3, while the immunofluorescence studies showed loss of PCs in lobule X. Finally, ependymal cell layer also showed a reduced expression of GABAρ3. We conclude that the expression of GABAρ3 is reduced in lobule X by prenatal exposure to VPA. Thus, GABAρ3 may be a relevant marker for ASD etiology.

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Effect of hypoxic preconditioning on a novel group of GFAP+ cells of the cerebellum

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The cerebellum harbors a specialized area named the ventromedial cord (VMC) which contacts the ventricular system at the lobule I. Cells that form the VMC express GFAP from early development to adult stage. The area includes also neuronal and glial cell lineages, defined by their unique electrical signature and expression of specific identity markers. Nevertheless, the function of the VMC remains to be determined. We show here the effects of hypoxic preconditioning (HPC), a stimulus known to exert neuroprotection. Brain clarification of HPC-treated GFAP-eGFP transgenic mice observed by light sheet microscopy showed a reduction of GFP expression in the VMC. In addition, Western blot analysis showed a reduction of the expression of GFAP and ALDH1L1 after HPC; in contrast, NeuN, nestin and Iba1 increased their expression. Immunofluorescence showed that HPC induced changes in the morphology of microglia as assessed by immunofluorescence. On the other hand, cell proliferation was tested by BrdU administration; however, only little incorporation was detected in cerebellum. Golgi staining revealed that Bergmann glial cells increased the protrusions and reduced the area of soma after HPC. We conclude the VMC responds to low oxygen levels, which can be quickly sensed by its anatomical location, probably allowing the triggering of mechanisms that enhance neuroprotective mechanisms.

RM-P006

Glial Cells are a Source of New Neurons in the Adult Substantia Nigra

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Neurogenesis in the substantia nigra (SN) has been a controversial issue. Here we report that neurogenesis can be induced in the adult rodent SN by transplantation of embryoid body cells (EBCs) derived from mouse embryonic stem cells. The detection of Sox2⁺ dividing (BrdU⁺) putative host neural precursor cells (NPCs) between 1-6 days post-transplantation (dpt) supported the neurogenic capacity of the adult SN. In agreement with the awakening of NPCs by EBCs, only host cells from implant-bearing SN were able to generate neurosphere-like aggregates in the presence of Egf and Fgf2. Later, at 15 dpt, a significant number of SN Dcx⁺ neuroblasts were detected. However, a continuous BrdU administration after transplantation showed that only a fraction (about 20-30%) of those host Dcx⁺ progeny derived from dividing cells and few BrdU⁺ cells, some of them NeuN⁺, survived up to 30 dpt. Unexpectedly, 25-30% of Dcx⁺ or Psa-Ncam⁺ cells at 15 dpt displayed astrocytic markers such as Gfap and S100b. Using a genetic lineage tracing strategy, we demonstrated that a large proportion of host Dcx⁺ and/or Tubb3⁺ neuroblasts originated from astrocytes. Remarkably, new blood vessels formed in association with the neurogenic process that, when precluded, caused a reduction in neuroblast production. Accordingly, two proteins secreted by EBCs, Fgf2 and Vegf, were able to promote the emergence of Dcx⁺, Tubb3⁺ and NeuN⁺/BrdU⁺ cells in vivo in the absence of EBCs. We propose that the adult SN is a mostly silent neurogenic niche with the ability to generate new neurons by typical and atypical mechanisms.
RM-P007

Functional expression of GABAA receptors in glial cells of cerebellar white matter

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The cerebellum is involved in the coordination of movement; its cellular composition is dominated by GABAergic neuronal types. The white matter (WM) includes glial cell bodies of oligodendrocytes, microglia, NG2 glia and astrocytes. Glial cells are depolarized by GABA through GABA-A receptors. During early development GABA signaling is known to regulate cell proliferation, differentiation and migration. The aim of this study was to test whether glial cells express functional GABAA receptors during postnatal development of cerebellar WM. GFAP-EGFP transgenic mice at P7-P9 were used. The brain was isolated and 250 µm thick cerebellar coronal slices were obtained. Glial cells responses to GABAA agonists were recorded by whole-cell voltage-clamp. The recording micropipette was filled with 0.5% biocytin to determine cell morphology and cell-coupling. GFAP+ cells showed dye-coupling, a passive current-voltage relation and did not respond to the GABAA receptor agonist muscimol (N=9). Two additional current profiles were identified in GFAP- cells (N=11). The first population showed an outwardly rectifying current-voltage relation, responded to the agonists GABA (100uM) and muscimol (100uM) and dye-coupling was absent (N=7); while the remaining GFAP- population showed dye-coupling, a linear current-voltage relation and did not respond to the GABAA agonists (N=8). We conclude that only a population of GFAP-cells without cell coupling express functional GABAA receptors in the cerebellar WM.

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Taurine-GABA receptors interaction in the proliferative processes of GFAP+ progenitor cells from the subventricular zone of mice.

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Degenerative diseases of the nervous system have become a public health problem worldwide so the search for molecules that can help in the treatment or even with the prevention it is imperative. Taurine is a non-protein sulfur-containing amino acid which has been found to promote proliferation in GFAP+ neural progenitor cells from the subventricular zone (NPCs-SVZ). However, the mechanism of these action of taurine are poorly understood. It is well known that taurine is a partial agonist of GABA receptors (GABAr). GABAr are a family of specific receptors that are activated by the main inhibitory neurotransmitter γ-amino-butiric acid in central nervous system. In NPCs-SVZ, the activation of receptors leads to the activation of different pathways that regulate proliferation process. Therefore, it is likely that the effect caused by taurine could be mediated by the activation that GABAr expressed in NPCs-SVZ. The aim of this study, was to determine whether taurine, through interaction with GABAr present in NPCs-SVZ, modulates the proliferation process. Our results showed that the application of bicuculine and picrotoxin, in the presence of taurine, generated a reduction in the NPCs-SVZ proliferation processes. On the other hand, the CGP55485 (inhibitor of the metabotropic receptors) did not have a significant effect on the cellular number in the presence of taurine. These results suggest that the effect of taurine, in the proliferation process of neural progenitor cells from sub-ventricular zone, is partially mediated by ionotropic GABA receptors.

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Aquaporins (AQPs) are a family of small, integral membrane water-transporting proteins, found in prokaryotes and eukaryotes implicated in mediating bidirectional movement of water across cell membranes in response to osmotic gradients. There are at least 13 different members of the AQP protein family described in mammals. In the nervous system, most of the work has been focused in the central nervous system, but very little in the peripheral nervous system (PNS). In the PNS, AQP1, AQP2 and AQP4 have been reported in both peripheral neurons and glial cells. In this work we studied the expression of AQP1 in rat and mouse in normal sciatic nerve and during Wallerian degeneration by reverse transcription polymerase chain reaction (RT-PCR), Western blot analysis and immunohistochemistry. The results show that in the rat sciatic nerve, AQP1 is present in both myelinating and non-myelinating Schwann cells. In myelin internodes AQP1 is enriched in the Schmidt–Lanterman incisures and in some internodes it is also present in the abaxonal membrane. AQP1 is also present in the paranodal regions of the nodes of Ranvier. However in the mouse sciatic nerve, AQP1 is only present in non-myelinating Schwann cells. The fact that AQP1 is localized differentially between two close species of rodents, suggests that AQP1 might have a different role in nerve homeostasis. In crushed nerves, there are no changes in the levels of mRNA at different times of Wallerian degeneration in both mouse and rat, however at the protein level changes are observed. Therefore, it is likely that AQP1 is regulated similar in both rodents, not at the mRNA but at the protein level.
RM-P010
Visual deafferentation effects on astrocytic processes of the lateral geniculate nucleus of the thalamus

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A set of AC processes in the normal CNS, or paraxial processes (PAPs) was previously found in direct interaction with myelin envelope with varying degrees of structural damage; additionally, cytoplasmic areas of the PAP-myelin interaction displayed confluent lysosomes opening next to the altered myelin envelop. We now test the possibility that axonal damage enhances the process of lysosome-mediated myelin dissolution. The retina of rats was lesioned by applying positive pressure to the eye. Following a seven days survival, animals were perfused and samples from the dorsolateral geniculate nucleus processed for transmission electron microscopy. Observations were focused to the PAP-myelin interphase. Three-dimensional assemblies (n=5) were performed in series of successive sections and 3D electron microscopic images processed with the “Reconstruct” software. From the total volume of PAPs studied, lysosome-volume was determined and expressed as the ratio PAP /lysosomal volume. Mann-Whitney U- test disclosed that differences are significant (p <0.038) between the groups. These results indicate that the AC-PAP mediates both normal and lesion-triggered myelin remodeling utilizing lysosomes as the secretory (i.e., extracellular) route.
RM-P011

Malva parviflora extract regulates the phagocytosis of microglia via PPARγ in an Alzheimer’s disease model

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Microglia are immune cells of the CNS that participate both in normal CNS function and in disease. Recent evidences indicate a role for activated microglia in the Alzheimer’s disease (AD) progression since these cells release pro-inflammatory cytokines that induce neuroinflammation which compromises microglial clearance functions. Therefore, the microglia have been proposed as a therapeutic target. Agonists of PPARγ exert anti-inflammatory functions and stimulate phagocytosis; via CD36, PPARγ increases Aβ uptake by the microglia. Nevertheless, the side effects produced by these agonists limit their use. Here we investigated the effects of a hydroalcoholic extract (HE) of Malva parviflora (M. parviflora) in microglia. Primary microglial cells were isolated from wild-type CD1 mice and from 5XFAD, an AD mouse model. We demonstrated that the HE of M. parviflora possesses anti-inflammatory properties in neonatal mice microglia as it reversed the amoeboid phenotype (associated with activated microglia), inhibited the activation of NF-kB resulting from LPS exposure and decreased the expression of pro-inflammatory markers (CD86 and TNF-α) in the cortex of 5XFAD mice. Likewise, the M. parviflora HE rescues the phagocytic capacity of microglial cells via a PPAR-γ/CD36 dependent mechanism that correlates with decreased load of β amyloid plaques in the cortex of 5XFAD mice and improved learning and memory. These results suggest the therapeutic use of the HE of M. parviflora to slow down the progression of AD by restoring microglial function.

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SYMPOSIUM ABSTRACTS
(SPEAKERS)

OCTOBER 5TH, 2018
ORGANIZATION OF THE VENTRICAL ZONE OF THE CEREBELLUM

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While exploring the expression of GABA-A receptors in glial cells of the cerebellum we found the presence of two novel clusters of cells in the periventricular zone, that is the roof of the fourth ventricle. The first one was named the subventricular cellular cluster (SVCC) and is composed of cells that express glial and neuronal markers. The second was named the ventromedial cord (VMC) and appears as a streak of biciliated cells with microvilllosities facing the ventricle, that includes GFAP+ and nestin+ cells organized along the periventricular vasculature. The dorsal limit of the SVCC is associated with myelinated axons that originate from the fastigial nucleus. The cell clusters can be observed from late embryonic development and expand during early postnatal development but are restricted to the central area of the ventricle in adulthood. We did not find evidence of cell proliferation, cell migration or the presence of fenestrated blood vessels. Cells from the VMC respond to mild hypoxic conditions by changing the expression of glial and neuronal markers. These findings provide new insights into the knowledge of the cellular composition and structural organization of the periventricular zone of cerebellum.
Astrogliosis in Stroke

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Stroke like most of the central nervous system pathologies, share a common feature known as reactive astrogliosis. Reactive astrogliosis is the response of the astrocytes to tissue injury and is a finely regulated process involves molecular and cellular changes, from transient to permanent. This astrocytic response can be modulated by different signaling molecules released by the different cell types present in and around the lesion site. Stroke is caused by bleeding or blocked blood vessel in the brain. The more common kind is by blocked blood vessel and is known as ischemic stroke. In this pathology, there are two different damage areas, one known as core, in which the cells die quickly and the other (peri-infarct), is around the core and the cells are functionally weakened but can be recovery. Unresolved peri-infarct reactive astrogliosis during chronic stage of stroke, contribute to establish a non-permissive environment to functional recovery. Evidence from several studies suggest that the decrease in chronic reactive astrogliosis without disrupting the glial scar, is a necessary process for functional recovery. Understand the mechanism of this modulation might serve to develop new therapeutic targets.
Neonatal prolactin decreases glial population and affects cytokine expression of the hippocampus

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Neonatal administration of prolactin (PRL) to rat pups was previously shown to reduce hippocampal neurogenesis by postnatal day (PN) 15. Since PRL is a cytokine, it probably exerts its actions affecting the neuroimmune system. Here we analyzed the effects of neonatal PRL on the hippocampal glial cells and its cytokine expression in the hippocampus and in the periphery at PN15. Sprague Dawley male rat pups were used. PRL (13 mg/kg bw) or vehicle were (PN1-14) injected to pups. Pups were sacrificed on PN15 under basal conditions or 3h after stress exposure. Brains and hippocampi were isolated and trunk blood was collected. Immunocytochemistry for GFAP and Iba1 was performed to analyze glial cells; qPCR was used to assess TNF-α, IL-1β, e IL-6 expression in the hippocampus and ELISA techniques to evaluate plasma cytokine concentrations. We show that neonatal PRL administration had no effect on microglial cell density in the hippocampal hilus, or the amount of the activated microglia. However, PRL induced a significant decrease in the astrocyte population of the hilus. PRL increased the hippocampal expression of IL-1β and IL-6, but not of TNF-α under basal conditions. Importantly, PRL attenuated the expression of these pro inflammatory cytokines after stress exposure. Peripheral concentrations of TNF-α increased in PRL-treated pups under basal and stressed conditions, while IL-1β and IL-6 levels were not affected. We conclude that a chronic neonatal administration of PRL induces a pro-inflammatory response in the hippocampus, and alters the neuroimmune response to stress.
RM-S011

GABAR receptors in astroglia

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Gamma-amino butyric acid (GABA) is the main inhibitory neurotransmitter in the central nervous system of vertebrates. The essential function of GABA is to inhibit the initiation of an action potential, in mature neurons. However, in astrocytes and neural precursors GABA produce a depolarization through activation of ionotropic receptors known as GABA-A receptors, pentameric proteins that are modulated by clinical compounds such as barbiturates and benzodiazepines. GABA receptors mediating synaptic (phasic) or extra-synaptic (tonic) transmission are molecularly and functionally distinct, for both, neurons and glial cells. These differences are particularly evident in glial cells from the cerebellum and striatum, where specific expression GABAR subunits lead to a lack of modulation by clinical compounds. Thus, astroglia express functional GABAAR receptors, but their role in CNS physiology is intriguing, since glial cells do not produce action potentials. Overall, the role of GABAAR subunit in cerebellar or neostriatal glial cells during early postnatal development or in the control of precise movements in adults remains to be explored.

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RM-S012
Insights into the role of Prolactin in Astrocytes

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The brain is one of the organs with the highest energy expenditure and oxygen consumption. The extensive oxidative metabolism is accompanied by a concomitant generation of high amounts of reactive oxygen (ROS) and nitrogen (RNS) species, which in combination with a high content of polyunsaturated fatty acids increase brain susceptibility to oxidative damage. Under normal conditions, brain cells maintain a delicate balance between generation and elimination of ROS. Cells are normally equipped with antioxidant scavengers and enzymes that prevent high ROS-mediated damage, but neurons express them at a very low concentration and reduced activity, respectively. It is therefore crucial that neurons receive support from surrounding cells, particularly astrocytes, for a complete ROS detoxification and neuroprotection. Astrocytes contain higher levels of endogenous antioxidants and antioxidant systems. Thus, all forms of stimuli promoting astrocytic antioxidant functions will protect neuronal functionality and viability. Interestingly, prolactin (PRL), a peptide hormone secreted by the anterior pituitary gland, stimulates the proliferation and viability of astrocytes. Moreover, we recently showed that PRL is neuroprotective in the retina exposed to oxidative conditions (constant bright light) and raised the levels of endogenous antioxidants in retinal pigment epithelium cells (RPE) in culture. Therefore, the question of whether PRL promotes the neuroprotective capacity of astrocytes by enhancing their antioxidant capacity is relevant, and could lead to neuronal therapeutic approaches. Here we present an overview of the role of PRL as antioxidant in the brain.

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RM-S013
How do glial cells control CNS function?

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The central nervous system (CNS) controls most of our bodily functions. Damage due to injury or disease can have devastating consequences, as evident in patients with spinal cord injury, stroke, or HIV-associated dementia. These examples also remind us of the limited regenerative capacity of the CNS. Glial cells are tissue-resident cells involved in protecting, regulating, and maintaining CNS function throughout life. In the adult human CNS, they are as numerous as neurons. My laboratory is interested in elucidating the cellular and molecular mechanisms that allow glial cells to exert their diverse physiological functions, and how disruption or dysregulation of their functional properties contributes to injury or disease. In particular, our efforts have focused on the primary two immunocompetent glial cell types within the CNS, astrocytes and microglia. Astrocytes are one of the most abundant glial cell types. They extend highly ramified processes that interact with synapses, nodes of Ranvier, and blood vessels. Astrocytes are key players in regulating neuronal excitability, antioxidant production, and blood-brain barrier maintenance, and form protective scars after injury. They exhibit structural and functional dynamics on spatial and temporal scales that span several orders of magnitude, from micrometers to millimeters and from milliseconds to weeks. One of the major challenges in the field is to determine how these dynamic events relate to defined CNS functions. Microglia are the innate immune sentinels of the CNS. They structurally and functionally interact with both neuronal and non-neuronal elements. Their interactions, including synaptic pruning and phagocytosis of stressed or injured cells, can influence cellular and circuit function directly or indirectly. Because microglia undergo rapid functional changes when extracted from tissue identifying the mechanisms that control their in vivo function has remained a significant challenge. To address these challenges we have developed a variety of molecular genetic, transgenic, imaging, behavioral, and computational approaches for in vivo investigation of how glial cell dynamics relates to CNS function at molecular, cellular, and circuit levels in both physiological and pathological contexts. In this talk, I will present some of our latest findings on this topic.

RM-S014
The Pathophysiology of CNS White Matter Injury Depends on the Insult: Anoxia vs. Aglycemia vs. Ischemia

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We studied the pathophysiology of central nervous system (CNS) white matter (WM) injury using acutely isolated mouse optic nerve (MON), a typical CNS WM tract. This advantageous preparation allows experimental assessment with quantitative electrophysiological techniques. CNS WM is clinically important and unexpectedly complex. It’s metabolic rate is estimated to be less than that of gray matter (GM) by about 50%. WM comprises slightly more than 50% of total forebrain volume in humans (less than 15% of forebrain volume in rodents). It is finally becoming clear that CNS WM is very sensitive to disruptions in energy metabolism and that it is critically involved in the clinical manifestations of conditions such as stroke or oxygen deprivation. Moreover, the mechanisms of injury are different in WM than in GM. Thus, unique treatment strategies must be developed to treat metabolically challenged WM. We have systematically analyzed the cellular mechanisms of CNS WM injury due to three forms of energy disturbance: restriction of 1) oxygen and glucose (i.e., ischemia), 2) glucose alone (i.e., aglycemia), 3) oxygen alone (i.e., anoxia). Ischemia acutely injures WM by causing toxic Ca\textsuperscript{2+} overload in axons due to intracellular Na\textsuperscript{+} accumulation. It also causes a special form of ‘excitotoxicity’ due to glutamate release (from astrocytes probably) that activates AMPA/kainate (not NMDA) receptors damaging oligodendrocytes. Anoxia does not cause damaging glutamate release because astrocytes can function in the absence of oxygen. Instead, anoxia is associated with severe acidosis that activates ‘acid sensing ion channels’ (ASICs) permeable to Ca\textsuperscript{2+}, leading to toxic Ca\textsuperscript{2+} overload. ASICs are mainly located on oligodendrocytes and axons. Aglycemia acutely injures WM due to excitotoxicity involving NMDA receptors. NMDA receptors are activated not by glutamate (which is consumed as a fuel) but by the release of aspartate. Because aglycemia is associated with reduced lactate release, the pH rises and this releases the H\textsuperscript{+} block of the NMDA pore allowing toxic Ca\textsuperscript{2+} influx, probably mainly involving oligodendrocytes. More than one of these unique pathophysiological pathways can operate during an acute stroke where different areas of the lesion are subject to different metabolic conditions.
Early life stress and lipopolysaccharide affect hippocampal glial cells and induce long term behavioral alterations

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Early life stress activates the neuroimmune hippocampal system, and associates with a depressive – like behavior in adulthood. Early severe infections also activate glial cells and alter cognitive processes. Here we analyzed the immediate effects of a neonatal double stress-immune challenge on the neuroimmune system of the hippocampus and its long term effects on behavior. Male Sprague Dawley rats were used: 1) control + vehicle group, 2) maternal separation (MS, 3 hours/day postnatal days (PN) 1 to 14 + vehicle, 3) control + Lipopolysaccharide (LPS, 0.5mg / kg, PN14), 4) MS + LPS group. Some groups were sacrificed on PD15 for histological and cytokine analyses. Starting at PN120, the emotional state of the remaining groups was analyzed with elevated plus maze, open field and forced swimming tests. To evaluate spatial and non-spatial memory, the object recognition and object placement tests were used. We observed a decrease in the cellular density of astrocytes, but an increase in the activation of microglial cells in MS and LPS pups, and was maximal in MS-LPS pups in the hippocampal CA3 and hilus regions. LPS induced an increased secretion of plasmatic and hippocampal IL-1b. However, MS attenuated this cytokine response. Adult MS-vehicle and MS-LPS male rats showed depressive - like behavior. The Control-LPS and MS-LPS groups showed an anxious like behavior in adults. SM and LPS challenges had no effects on learning. Exposure to LPS and early MS leads to alterations in the neuroimmune system and long term emotional state but does not affect memory.
Cop-1 effect on interleukins and neurotrophins expression in choroid plexus in rats with cerebral ischemia

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Copolymer-1 (Cop-1) has shown to exert neuroprotection and induce neurogenesis in the focal cerebral ischemia (FCI) model. Cop-1 modulates the immune response by inducing a shift towards the Th-2 phenotype by promoting the secretion of anti-inflammatory cytokines and trophic factors. Recent studies have shown that physiological communication between the immune system and the central nervous system takes place in the choroid plexus (CP) through cytokines and neurotrophic factors, so it is important to verify if in this area of the brain cop-1 is also able to mediate on said expression in rats with FCI. To evaluate the expression of cytokines and neurotrophic factors in CP of rats with FCI immunized with Cop-1 at 7 days post-ischemia. Twenty male Sprague Dawley rats with randomized FCI were used in 4 groups: 1) Control, 2) complete Freund’s adjuvant (CFA), 3) Cop-1 + saline solution, and 4) CFA + cop-1. Seven days after ischemia the total RNA was obtained from the CPs and the gene expression of IL-1β, IL-17, IL-4, IL-10, INF-γ, NT- 3, IGF-1 and BDNF were analyzed by QRT-PCR. Results were analyzed by Kruskal Wallis followed by U of Mann Whitney test. Our results show that cop-1 is able to significantly stimulate an increase in the expression of IGF-1 and NT-3 at 7 days post ischemia in PC; IL-10 shows a tendency to increase. Cop-1 is able to modify the expression of growth factors and thereby potentially modify the communication that exists between the nervous and immune system.

RM-P014
Changes in emotional and learning behavior in female rats exposed to a double challenge neonatal immune-stress

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Stress in early stages alters the emotional behavior of adult rats who, faced with aversive stimuli, show greater negative physiological changes. Maternal separation is a method often used in rodents to generate stress in the early stages of life, which has repercussions in adulthood to affect the emotional behavior and learning ability of male animals. In the same way, infections by external agents in these stages of life achieve the same effects. Therefore, the objective of the study was to observe a double neonatal stress-immune challenge and its effects on the behaviors of anxiety, depression and learning in female rats. The tests were performed on four groups of female rats of the Sprague Dawley strain of 5 months of age (150 days). The first stress-free group (CONT), one exposed to maternal separation of day 1-14 postnatal (SM-VEH), another to which lipopolysaccharide was applied to postnatal day 15 (LPS-CONT) and the last one in which they were applied the two treatments (SM-LPS). The tests of elevated labyrinth in cross and open field were made for the observation of anxiety behavior. The FORCED SWIM test was used to evaluate depressive behavior. While the object recognition and placement tests were performed to analyze spatial and non-spatial learning. The SM-VEH group presented higher rates of depression, the groups with LPS also had significance with respect to the CONT-VEH group. None of the treatments provoked a greater anxiety behavior, on the contrary, the SM-LPS group showed lower anxiety indexes with respect to the other groups. In the object recognition test, only the LPS-CONT group is affected in comparison with the CONT-VEH group since a significant difference is observed. There were no differences between groups in the object placement test. Maternal separation generates depressive behavior in adulthood, but attenuates anxiety in the group subjected to the double challenge. Learning tests indicate that treatment with LPS induces deficiency in non-spatial learning. Maternal separation seems to diminish the effects due to LPS in females.
Characterization of lymphocytes in cerebrospinal fluid of rats with cerebral ischemia immunized with Copolymer-1

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Stroke is one of the main causes of disability and mortality in adults worldwide. Within the pathogenesis, inflammation is considered to be caused by the accumulation of inflammatory and mediating cells in the ischemic brain. Copolymer-1, a synthetic peptide that contains four amino acids (lysine, alanine, glutamine and tyrosine), has the capacity to generate an immune deviation from a T Helper 1 to T Helper 2 phenotype and increase the synthesis of growth factors and anti-inflammatory cytokines that favor neuroprotection and neurogenesis. In this study lymphocytes present in cerebrospinal fluid were identified, using the flow cytometry technique, from rats with cerebral ischemia immunized with copolymer-1 at 14 days, divided into three groups: 1) ischemia without treatment (control), 2) copolymer-1 plus saline solution, and 3) copolymer-1 plus Freund’s complete adjuvant. The immunization with copolymer-1 plus Freund’s complete adjuvant showed and increase of CD4 + and TNK lymphocytes, while the group treated with copolymer-1 plus saline solution showed a greater amount in CD8 + and central memory T lymphocytes. The control group presented the least amount of lymphocytes in general. The results show that there is an increase of lymphocytes in the groups treated with copolymer-1 and that the presence of adjuvant does modify the infiltration of different cells, these cellular modifications could determine the disposition to develop neurogenesis or neuroplasticity in the peripheral areas to the Choroid plexus.
D-serine modify brain functional connectivity in aged rats

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As age advances, there is a natural reduction in cognitive functions such as attention, memory and cognitive flexibility. D-serine is a non-essential amino acid which is the endogenous co-agonist of N-methyl-D-aspartate receptors (NMDAR) and whose participation in cognitive functions has been reported. Previous results from our laboratory have shown that chronic administration of D-serine improves the performance of aged rats in attention and cognitive flexibility tasks as well as the evaluation of their locomotor activity. However, the cellular mechanisms underlying this behavioral effect are not clear. To answer this question, we analyzed the changes in functional brain connectivity associated with chronic administration of D-serine in 18-month-old rats. The functional connectivity between two regions was defined as the covariance of its BOLD signal. We used a Bruker 7T magnetic resonator and performed an independent component analysis of the Blood Oxygen Level Dependent (BOLD) signal, (associated with neuronal activity) measured at rest in 36 rats under low anesthesia (isoflurane & dexmedetomidine hydrochloride). We identified 11 components distributed in cortex, thalamus and striatum. The connectivity between the retrosplenial cortex and the identified regions was analyzed. The rats treated with D-serine (30 mg) showed greater connectivity between the dorsal anterior cingulate, rostral anterior cingulate, somatosensory cortex, lateral parietal cortex and auditory cortex with the retrosplensial cortex (T (22)> 2.4, p <0.023) in compare to control rats. Our results provide evidence of the modifications in several brain circuits that underlie D-serine effect on cognitive functions.

RM-P017
Serum D-serine levels correlates with the cognitive performance of aged humans

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Demographic estimates show that in 2050 ~20% of the world population will be over 60 years. One of the characteristics that accompany aging is the decrease in cognitive functions, a situation that has been related to a hypofunction of the NMDA receptor (NMDAR). Previous research have shown that the function of this receptor is essential for synaptic plasticity and cognitive functions like memory, attention among many others. For the NMDAR to be activated, it requires the presence of a strong depolarization, the binding of glutamate and its co-agonist, the amino acid D-serine. It has been reported that the levels of D-serine in the hippocampus of aged rodents is lower in compared to young ones. D-serine can cross in and out of the brain due to transporters expressed in the brain-blood barrier, in this way, oral administration of D-serine improve the cognitive performance of rats and humans. Besides this evidence, it is not known if there is a relationship between the serum levels of D-serine and cognitive performance in aged humans. Furthermore, we wonder if this aminoacid could be used as a diagnostic tool for cognitive detriment. In the present study, the concentration of serum D-serine was determined by HPLC and related with cognitive performance in healthy subjects between 60 and 90 years of age. The data show a positive correlation between D-serine and the cognitive performance of the participants. This shows that D-serine could be used as adjuvant tool in the diagnosis of cognitive detriment due to aging.

RM-P018
A novel GABA\textsubscript{A} receptor expressed in oligodendrocytes

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Myelination is of central importance in the functioning of the nervous system; its failure causes devastating pathologies known as demyelinating diseases. In addition to permit the establishment of complex neuronal circuits, myelination is also an important factor for neuronal survival. In the central nervous system (CNS) this process requires a constant dialogue between neurons and oligodendrocytes (OL). Knowledge about the communication mechanisms involved in myelination is fundamental to generate tools that might permit its control. The communication through classic neurotransmitters between neurons and OL is of importance in their dialogue. Recent studies clearly indicate involvement of GABAergic signaling in myelination acting through GABA receptors subtype A (GABA\textsubscript{A}R), the expression of these receptors in the OL’s membrane depends on their interaction with neurons. The GABA\textsubscript{A} receptors expressed in the OL are distinctive; this opens an opportunity for the discovery of specific drugs, or other molecular strategies that might potentiate the myelination process. Our recent studies show the existence of a novel oligodendrogial GABA\textsubscript{A} receptor subtype, with distinctive characteristics with respect to those expressed in other neural cells, is proposed that this receptor is composed by α3β2γ1 subunits.
Environmental enrichment modifies the morphology of mouse cortical astrocytes

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Environmental enrichment (EE) is a model used to study brain plasticity dependent on experience. Several studies have shown that rodents exposed to EE have changes in dendritic arborization, number of dendritic spines and synapses, neurogenesis and neurotrophic factors. However, the effect of EE on astroglial cells is not completely known. In this work we analyze the effect of EE on cerebral cortex astrocytes. Briefly, C57BL/6J mice were exposed to EE for 5 weeks, after this period the mice were sacrificed, and the brains processed for a Golgi stain and Nissl stain. An analysis of the morphology and number of astrocytes was performed by a semi-automated Sholl method. Our results show that EE, does not modify the number of glial cells, but significantly increases the area of astrocytes of visual and somatosensory cortex. The astrocytes of the motor and auditory cortex were not affected. These results suggest that EE produces differential changes in cortical astrocytes, studies in progress are being carried out to determine if these changes are associated with the secretion of molecules with synaptogenic activity.

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Oral administration of D-serine improves the cognitive flexibility and attention in aged rats


The D-serine is a D-aminoacid catalyzed by serine racemase from L-serine. Currently, it is known as the main endogenous co-agonist of the glutamate-ergic NMDA receptors. For this reason, D-serine is essential for the induction of several types of synaptic plasticity and cognitive processes such as learning and memory. D-serine levels are diminished in aged rats, which could account for the deterioration of cognitive functions associated with aging. To analyze this, cognitive flexibility and attention were analyzed in male Wistar rats of 6, 12 and 18 months old. As expected, both functions decline with age, being the 18-month rats, the most affected. Interestingly, 12-month old rats that were chronically administered with D-serine (2 months before evaluation of cognitive flexibility and attention) presented a higher performance in evaluations of cognitive flexibility and attention in compare to those that were not treated with D-serine, becoming comparable even with the performance achieved of young rats. This result suggest that D-serine could be used as a cognitive enhancer to restore cognitive functions that decline during healthy aging.
Repetitive transcranial magnetic stimulation (5Hz) modulates hippocampal neurogenesis and glial cells in chronic mild stress mice

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Repetitive transcranial magnetic stimulation (rTMS) is a non-invasive technique that can regulate the neuronal excitability. The properties of this technique have allowed it to be considered as an alternative treatment to depression. However, its mechanisms of action are still unknown. It has been proposed that the antidepressant effect of rTMS may be associated with neuroplastic modifications including in it the changes on other cellular components such as microglia, which is altered in depression diagnosed patients. Therefore, the aim of this study was to evaluate whether the antidepressant like effects of rTMS are associated to the changes in the density of microglia and on the hippocampal neurogenic process in Balb/C mice exposed to the chronic unpredictable stress (CMS) to generate depressive-like behaviors. After the fourth week of CMS, mice received the treatment with rTMS for four additional weeks. Subsequently, the depressive-like behavior was analyzed with a behavioral battery. Later, the animals were euthanized, and their brains were dissected out to carry the labeling of some cellular populations involved in the neurogenic process (Ki67 for cell proliferation; double-cortin for immature neurons; CD11b for microglia) by immunohistochemistry. The results obtained indicate that rTMS reverted depressive-like behaviors (coat state deterioration, low activity psychomotor, anhedonia and hopelessness). In addition to increasing the number of Ki67 and DCX-labelled cells and decrease the CD11b-labeling. These data suggest that rTMS induces an antidepressant-like effect that is associated with the increase of adult hippocampal neurogenesis and it may be also related to the control to the microglial cells activation.

RM-P022
Organization and functional characteristics of Bergmann glial cells in an experimental model of cortical dysplasia

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The lobule X of cerebellum is relevant for maintaining the body balance. Bergmann glia is essential for neuronal migration during development, and in adult life the glutamatergic transmission mediated by these cells is important for Purkinje neurons to execute the control of fine movements. Cortical dysplasias are alterations in the organization of the layers of the brain which rise due to problems in neuronal migration during development. Dysplasias can generate epilepsies. Neuronal component has been widely studied in experimental models of cortical dysplasia, in contrast very little is known about how glia is affected. The aim of this study was to characterize the morphology and intracellular calcium dynamics of the Bergmann glia in the lobule X of the cerebellum and its modifications in the model of cortical dysplasia induced by carmustine (BCNU). Cerebella from P5-P7 carmustine-treated transgenic mice GFAP-eGFP were processed by the Golgi-Cox technique to reveal the morphology of Bergmann glia cells, Ca²⁺ imaging was performed in slice preparations to determine their spontaneous activity. Golgi-Cox staining revealed no significant differences in the area or diameter of soma, neither in the number or length of the processes but showed significant differences in the relative length of lateral protrusions (Ctrl 3.88 ± 0.30 μm, BCNU 2.05 ± 0.22 μm)**. Ca²⁺ imaging showed that presented spontaneous intracellular Ca²⁺ transients (Ctrl 2±0.70, BCNU 3.75±0.85). Frequency of transients for each cell (Ctrl 0.00516± 7.31E-4 Hz, BCNU 0.00333±5.83E-4 Hz). Duration by transitory (Ctrl 13.95±1.12 s, BCNU 10.58±1.37 s). Kinetics of rise and fall of each transient (Ctrl 2.50±0.49, BCNU 1.67±0.57 s). The data is represented as the mean ± SEM. The ** indicates significant difference (P <0.05) compared to the control group. These results show an association of morphological changes in the protrusions of the Bergmann cells with an increase in the number of cells that present spontaneous Ca²⁺ transients.

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Astrocytes were conceived for decades only as supporting cells of the brain. However, the observation of Ca2+ waves in astrocyte synctitia, their neurotransmitter receptor expression and gliotransmitter secretion suggested a role in information handling, conception that has raised some controversies. Synaptic Neuron-Astrocyte metabotropic communication mediated by Inositol tris-phosphate (SN-AmcIP3) is supported by different reports. However, some models contradict this idea and Ca2+ stores have been found 1000±325 nm apart from the Postsynaptic Density in the Perisynaptic Astrocyte Projections (PAP’s), suggesting that SN-AmcIP3 is extrasynaptic. However, this assumption does not consider IP3 Diffusion Coefficient ($D_{ab}$), that activates IP3 Receptor (IP3R) releasing Ca2+ from intracellular stores. Here we idealized a model of a PAP (PAPm) to perform an order of magnitude analysis of IP3 diffusion using a transient mass diffusion model. This analysis shows that IP3 forms a concentration gradient along the PAPm that reaches the steady state in milliseconds, three orders of magnitude before IP3 degradation. The model predicts that IP3 concentration near the Ca2+ stores may activate IP3R, depending upon Phospholipase C (PLC) number and activity. Moreover, the PAPm supports that IP3 and extracellular Ca2+ entry synergize to promote global Ca2+ transients in PAP’s. Thus, the model presented here indicates that SN-AmcIP3 is not limited by Ca2+ stores position in PAP’s.