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Technologies and Innovations for Diagnosis & Monitoring, Assistive Technology, Therapeutic Technology & Care-Supportive Technology for Dementia, Neurodegeneration and Brain Injury

Isoprenaline Induced Oxidative Damage in Brain of Wistar Rats and its Amelioration by Hull Extract of Juglans Regia

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Background and Objective: Chemically induced oxidative stress in brain can result in significant neurological ailments. The present study was aimed at assessing isoprenaline (ISO) induced oxidative stress in brain and its attenuation by administration of hull extract of Juglans regia in Wistar rats.

Methods: Wistar rats were randomly allocated into five groups. Group I served as control, group II and III animals were administered ISO and hull extract, respectively. Group IV and V were given ISO along with hull extract or quercetin, respectively. Antioxidant biomarkers like total antioxidant status (TAS), total thiols (TTH), catalase (CAT), superoxide dismutase (SOD), acetylcholinesterase (AChE), arylesterase (AE), glutathione peroxidase (GPx), glutathione reductase (GR), malondialdehyde (MDA) and advanced oxidation protein product (AOPP) were analyzed along with histopathological alterations in brain.

Results: Administration of ISO produced significant (P0.05) increases in, CAT, SOD, GR, MDA, AOPP levels and significantly reduced TTH, TAS, AE, AChE and GPx activities in brain of rats. Histopathologically, neuronal degeneration or necrosis, spongiosis and gliosis were seen in cerebral cortex after ISO administration. Pretreatment with hull extract restored TAS, TTH, AChE, CAT and SOD values. Additionally, significant reductions were noted in levels of MDA, AOPP, and severity of histomorphological changes in cerebral cortex after hull extract treatment.

Conclusion: Altered antioxidant profile as well as cellular lipid/proteins oxidation biomarkers along with histopathological changes indicates oxidative injury in rat brain following ISO administration. Administration of J. regia hull extract attenuated these biomarkers in brain demonstrating presence of neuroprotective properties against ISO induced oxidative damage in brain of Wistar rats.

EPoster 2

Mapping Brain Health and Brain Network Imaging, Big Data and the Brain

Modelling Cortical Layer Connectivity in the Macaque Brain

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Current techniques for exploring the brain`s wiring diagram, also known as the connectome, remain biased by the definition of the cortex as one homogenous unit. Overcoming this bias demands expanding the basic unit used for connectome analysis to a more descriptive representation of the heterogeneous laminar substructure of the cortex.

In recent years, promising progress has also been made in the field of neuroimaging of cortical grey matter structure [2,3,4].

In this study, we model and explore the macaque connectome on the laminar-level and validate results by comparison to Felleman and Van Essen’s seminal study on hierarchical processing in the macaque visual cortex [1].

EPoster 3

Technologies and Innovations for Diagnosis & Monitoring, Assistive Technology, Therapeutic Technology & Care-Supportive Technology for Dementia, Neurodegeneration and Brain Injury

Age-Related Differences in Eye Movements' Behavior During Decision Making in Classification Tasks

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Arbitrary categorization classification tasks require learning the associations between the stimuli and the responses in their repeated presentation to guide decision-making to similar stimuli. By recording eye movements and their characteristics, we tried to obtain additional information about the age effects on decision-making and classification processes. We performed three experiments on the classification in two categories of visual stimuli, differing in the combinations of 4 characteristics - type and direction of movement, color, and shape of the elements. The experiments differ in complexity: in Experiment 1 the classification is determined by the color of the elements, in Experiment 2 - by the combination of direction and type of motion, and in Experiment 3 - by the combination of color, direction, and type of motion. Two aged groups: young (18 - 38 yrs) and elderly (63 – 75 yrs) participated in the experiments. We recorded the eye movements in the interval between stimulus disappearance and the manual response for the chosen category. The results show that for both age groups the number of saccades depends on the task difficulty, being largest for Experiment 3. The elderly group made more saccades in all experiments with the largest difference between the two age groups for Experiment 1. These results suggest age differences in the decision-making processes in an arbitrary classification task that could be better described by studying the eye movements behavior of the participants from the two age groups.

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EPoster 4

Innovations and technologies in Neuro-immunology, Movement disorders, Epilepsy and Migraine, Innovations and technologies for Stroke, Neurodegeneration and brain disease, Neuromodulation for Brain Disorders, Mapping Brain Health and Brain Network Imaging

Exercise Restores Synaptic Integrity in a Rodent Model of Parkinson's Disease

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Parkinson`s disease (PD) is the second most common neurodegenerative disease. Physical exercise has beneficial effects in PD such as improvement of patient's quality of life, but the role it plays in modulation of synaptic plasticity is unclear. A novel marker of synaptic density has recently become available. UCB-J binds to the transmembrane synaptic vesicle glycoprotein 2A (SV2A) transporter and can be labeled with in vitro autoradiography imaging. The aim of the current study is to use [3H]UCB-J autoradiography to examine the changes in synaptic transporter function elicited by physical exercise in a rodent model of PD induced by unilateral 6-hydroxydopamine (6-OHDA) injection.

Rats were unilaterally injected in the striatum with either 6-OHDA or saline as a control; half of each group was then exposed to an exercise regime, while the other half remained sedentary. In the exercised group, animals ran for 40 minutes at 10m/min, 3x/week for 5 weeks. Rats were then euthanized, and brains fresh frozen and sectioned. [3H]UCB-J autoradiography was performed in striatum and substantia nigra using [3H]UCB-J. Data were analyzed using an autoradiography system. The comparisons between groups were conducted using a two-way ANOVA followed by Tukey`s post-test.

Our data revealed a significant reduction of [3H]UCB-J binding in the ipsilateral striatum (12%,p0.01) and substantia nigra (15%,p0.05) in response to 6-OHDA lesioning in sedentary rats. In lesioned rats exposed to the exercise protocol, binding remained at non-lesioned values.

Increased [3H]UCB-J binding suggests that exercise has the capacity to promote compensatory mechanisms associated with restoration of synaptic integrity in PD.

EPoster 5

Stroke, Cerebral Small Vessel Disease, Dementia, Neurodegeneration, & Traumatic Brain Injury Challenge

Cardiovascular Dysfunctions in a Cuprizone-Induced Mouse Model of Multiple Sclerosis: a Focus on Aberrant Expression of Cardiac Inwardly Rectifying Potassium Channels

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Background: multiple sclerosis (MS) is a chronic inflammatory autoimmune disease which affects the central nervous system (CNS) and is characterized by demyelination, astrogliosis and damage to oligodendrocytes. Interestingly, MS patients showed an increased cardiovascular (CV) risk probably due to an impairment in the autonomic control of CV functions. However, the underlying molecular mechanisms are not completely understood. In this regard, inwardly-rectifying potassium (Kir) channels play a key role in cardiac excitability by contributing to the repolarization phase of action potential and were recently identified as target of the autoantibody response in MS patients.

Objective: to investigate the role of cardiac Kir channels in the CV dysfunctions occurring in MS.

Methods: electrocardiographic recordings (ECG) evaluated cardiac parameters and electrical activity in a cuprizone-fed C57BL/6 mice, a classic demyelination animal model. Relative transcript levels of cardiac Kir2.2, Kir4.1 and Kir6.2 channels in mice were analyzed using real-time PCR.

Results: the cuprizone-induced mouse model was confirmed by immunohistochemistry analysis showing demyelination in the corpus callosum. ECG recordings from mice showed a significant decreased duration of the P wave and RR interval as well as an increase of the heart rate in cuprizone-treated mice as compared with the controls. Significant high expression levels of Kcnj11 and Kcnj12, encoding for Kir6.2 and Kir2.2 channels respectively, were observed in mouse heart tissue as compared with controls, whereas no differences in Kir4.1 channels were observed.

Conclusions: to the best of our knowledge, these findings suggested a potential role of cardiac Kir channels explaining autonomic dysfunctions occurring in MS pathology. Our data open more avenues to the development of novel therapeutic strategies based on targeting cardiac Kir channels.

EPoster 6

Stroke, Cerebral Small Vessel Disease, Dementia, Neurodegeneration, & Traumatic Brain Injury Challenge

Analysis of Electromyographic Fatigue of the Masseter and Temporalis Muscles of Subjects with Parkinson`s Disease

Marcelo Palinkas[1,2], Letícia Pagnano[1], Jaime Eduardo Cecilio Hallak[1,2], Gabriel Pádua da Silva[1], Nayara Soares da Silva[1], Isabela Hallak Regalo[1], Edson Donizetti Verri[1], Simone Cecilio Hallak Regalo[1,2], Selma Siéssere[1,2]

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Background. The functions of the human body must be in harmony so that the systems and organs remain balanced. Parkinson's disease is a chronic, progressive, and degenerative neurological disorder. Objective. To analyze the electromyographic fatigue of the masseter and temporalis muscles in individuals with Parkinson`s disease. Method. 16 subjects, aged between 50 and 70 years, were divided into two groups: with Parkinson`s disease in stages I and III of the Hoehn and Yahr deficiency scale (n = 8) and disease-free control (n = 8). The median frequency of the normotensive electromyographic signal was analyzed. The data were tabulated and analyzed statistically (t-test, p 0.05). This study was approved by the Research Ethics Committee (number # 61113916.6.0000.5381). Results. Parkinson`s disease group showed increase median spectrum threshold of the electromyographic signal, with significant differences for the right masseter (p=.05) and the right temporal (p=.03) muscles. Conclusion. Parkinson`s disease may be associated with negative changes in the median frequency spectrum of the EMG signal for the masseter and temporalis muscles.

EPoster 7

Innovations and technologies for Stroke, Neurodegeneration and brain disease

Recognition of Amyloid Aggregation Via Auron Synthetic Dye Regarding the Specificity Property of Dyes toward Selecting the Particular Secondary Structure of Proteins.

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Introduction:
In 1854, the term “amyloid” was used for the first time by Virchow. To describe an aggregated substance found in the liver of a deceased patient. Despite the misleading association to starch, the term is still used and currently, 27 diseases are associated with amyloid fibril deposits of normally soluble proteins. Early detection of amyloid deposits could be effective in the diagnosis and treatment of disorders such as Alzheimer’s disease (AD), Parkinson’s disease (PD), and systemic amyloidosis. In this study, we tried to synthesize the aurone derivatives as an amyloid detection probe to compare with a standard probe called Thioflavin T in the different protein conditions such as amyloid and amorphous aggregates.

Material and method:
β-Lactogluboline was purified via fractionation method (As an all β secondary structure) and bovine serum albumin (As an α/β secondary structure) was purchased from Sigma Aldrich. For more precision, their qualities were evaluated by SDS-PAGE and Bradford protein assay. Furthermore, the spectrophotometric analysis of synthetic compounds such as UV-visible and fluorescence spectroscopies were considered in their individual and special wavelengths. Each synthetic compound was excited and the emission spectra were recorded immediately at their own exclusive wavelengths, while, they were bound to the amyloid fibrils in comparison with native protein and also amorphous aggregates.

Results:
The results of synthetic compounds were obtained in three different conditions, Native, Amorphous and Amyloid aggregations. We have shown that the synthetic compounds could selectively and specifically bind to amyloid fibrils almost as much as the ThT probe. Additionally, our synthetic compounds due to its neutral charge and high lipophilicity essence might cross the blood-brain barrier as an effective probe.

Conclusion:
According to our result, the synthetic compounds could be accounted as remarkable probes to detect in vitro β-amyloid plaques, but it should be investigated further as potential probes for detecting β-amyloid plaques in the AD brain.

Keywords:
Amyloid, Synthetic, Compounds, Probe, Amorphous, Aggregates

EPoster 8

Stroke, Cerebral Small Vessel Disease, Dementia, Neurodegeneration, & Traumatic Brain Injury Challenge

Evaluation of Bite Force and Pressure of the Tongue and Lips in Individuals with Ischemic or Hemorrhagic Cerebrovascular Disease

Paula Gonçalves, Guilherme Gomes, Camila Gonçalves, Gabriel Silva, Robson Lopes, **Lígia Gonçalves**, Isabela Regalo, Selma Siessere, Marcelo Palinkas, Edson Verri, Simone Regalo

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Abstract

Stroke is one of the world`s leading causes of death, disabling adult individuals in their 60s. The masticatory process of this individual becomes compromised, which hinders food intake, thus altering the entire function this system. The aim of this study was to evaluate the functionality of the stomatognathic system of individuals with clinical diagnosis of hemorrhagic stroke, by measuring the maximum molar bite force and lingual and lip pressure, comparing with healthy individuals, regardless of the affected side.

Material e Methods

According to the sample calculation thirty-six individuals were selected and divided into two groups, paired subject to subject: Hemorrhagic stroke group (n = 18; mean age 62.5 years) and Control Group, without the disease (n = 18; mean age of 62.0 years). The research was approved by the Research Ethics Committee of FORP/USP (CAAE: 92222318.8.0000.5419). Bite force was measured using a digital dynamometer in the molar regions on both sides. Tongue strength and lip pressure were analyzed by IOPI. Komolgorov-Smirnov statistical tests and student`s t-test (p 0.05).

Results

In the maximum molar bite force, the values were significantly lower in the hemorrhagic stroke group, when compared to the control group without disease (p 0.04). In the pressure of the tongue and lips there were no differences between the groups.

Conclusions

Hemorrhagic stroke negatively affected the maximum molar bite force of individuals, suggesting the need for changes in the pattern of ingested foods, with regard to nutrition and soft consistency, promoting an improvement in swallowing and ingestion of these.

EPoster 9

Neuromodulation for Brain Disorders

Effect of Home-Based Transcranial Direct Current Stimulation on Depressive Symptoms in Temporal Lobe Epilepsy: A Randomized, Double-Blind, Sham-Controlled Clinical Trial.

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This double-blind randomized clinical trial was designed to study the effects of transcranial direct current stimulation (tDCS) in depressive symptoms of patients with temporal lobe epilepsy (TLE). We evaluated 26 adults (mostly female, 88.46%; mean age 54.5 years old) with TLE and depressive symptoms randomized in two different groups: active tDCS (tDCSa) or sham (tDCSs). The participants were submitted to 23 sessions of tDCS, for 20 minutes daily, 5 days a week, during 4 weeks. After, they received maintenance of tDCS application in the research laboratory once a week, during 3 weeks. The intensity of the current was 2mA, applied bilaterally over the dorsolateral prefrontal cortex, with the anode positioned on the left side and the cathode on the right side. Participants were evaluated on days 1, 15, 30 and 60 of the study, using the Beck Depression Inventory II (BDI). The groups did not differ in relation to their clinical, socioeconomic and psychometric characteristics in the initial assessment. The tDCS treatment was well tolerated and did not increase the frequency of seizures. Both the tDCSa and tDCSs groups showed significant and similar improvement in depressive symptoms at the end of the intervention. On average, between the 1st and the 60th day, the BDI score decreased 42.39% in the active and 45.55% in the sham group (ΔBDIfinal-initial = -12.10 vs -12.44, $p = 0.68$). In our study, we observed a similar reduction in depressive symptoms in both groups. This effect could be attributed to a placebo effect. Funding CNPq,CAPES, FIPE-HCPA.

EPoster 10

Stroke, Cerebral Small Vessel Disease, Dementia, Neurodegeneration, & Traumatic Brain Injury Challenge

Rutin Protects Rat Pheochromocytoma (PC12) Cells from Abeta 25-35 Toxicity in both Normoxic and Hypoxic Conditions

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Brain regions in Alzheimer's disease (AD) patients display a decline in cerebral blood flow. Ischaemia / hypoxia activates beta and gamma secretases, augmenting APP processing and Abeta production. However, there are few studies on effects of hypoxia on Abeta induced neurotoxicity. Furthermore, current research on AD treatment is conducted in normoxia, which does not truly reflect the AD pathogenesis.

The OBJECTIVE of the study is to characterize effects of hypoxia on Abeta induced neurotoxicity in rat pheochromocytoma PC12 cells and to evaluate effects of rutin on Abeta toxicity.

METHODOLOGY

PC12 cells were cultured in normoxia (21% O2) and hypoxia (0.3% O2). Rutin was co-administered with Abeta 25-35 (10-50 mM) in PC12 cells for 24 hrs. Cell viability and cytotoxicity were determined by MTT and LDH assays. Cell apoptosis was investigated by flow cytometry using annexin V analysis. Assessments of ROS and lipid peroxidation were evaluated by DCFDA and Malondialdehyde (MDA) assays respectively.

RESULTS

There were does related reduction in MTT activity and increase in LDH release by Abeta 25-35 (10-50 mM). Abeta 25-35 (30mM) treatment induced apoptosis and increased the level of ROS and lipid peroxidation in PC12 cells. Combined treatment of Abeta 25-35 and hypoxia (0.3% O2) exacerbated the outcomes. Rutin (100 mM) ameliorated the conditions induced by Abeta 25-35 in both normoxia and hypoxia.

In CONCLUSION, rutin, a naturally occurring flavonoid glycoside, reduces cytotoxicity, apoptosis and ROS formation in PC 12 cells induced by Abeta 25-35 in both normoxia and hypoxia. Molecular targets for scavenging free radicals, anti-apoptosis and inhibiting cellular damage by rutin merit further investigations.

EPoster 11

Stroke, Cerebral Small Vessel Disease, Dementia, Neurodegeneration, & Traumatic Brain Injury Challenge

Oxidative Stress Contributes to the Pathogenesis of Multiple Sclerosis-Associated Pain in Experimental Autoimmune Encephalomyelitis (EAE) Model

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Background: Multiple sclerosis (MS) is a neurodegenerative disease of the central nervous system (CNS) with a wide range of symptoms such as motor abnormalities, fatigue, pain, and memory loss. Oxidative stress plays a critical role in the pathogenesis of the neurodegenerative disorder.

Objective: In this study, the experimental autoimmune encephalomyelitis (EAE) mouse model has established to determine the role of oxidative stress in the pathogenesis of MS and evaluate the neuroprotective effect of a novel phytosteroid (withametelin).

Method: EAE in female mice was induced by the injection of guinea pig spinal cord homogenate (GPSCH). The effects of withametelin on the clinical score, motor activity, pain parameters, oxidative stress, histopathology, BBB permeability, nuclear factor erythroid 2-related factor (Nrf2), Keap1 and HO-1 expression were studied.

Results: The behaviour results illustrated that withametelin treatment notably attenuates neurological deficits including motor and sensory symptoms. It effectively improved motor coordination. It significantly reduced EAE-induced pain hypersensitivity. Our mechanistic investigation revealed that withametelin treatment suppressed oxidative stress markers. It enhanced the level of antioxidants such as reduced glutathione, glutathione-S-transferase, catalase, superoxide dismutase in the brain and spinal cord. It significantly reduced nitric oxide (NO) production, myeloperoxidation (MPO), and lipid peroxidation in the brain and spinal cord. It reversed the histopathological changes of brain and spinal card associated with multiple sclerosis. Our mechanistic investigation revealed that it significantly intensifies the antioxidant defense system via regulation of the Nrf2-Keap1-HO-1 pathway factor in the brain and spinal cord.

Conclusion: These results suggest that oxidative stress contributes to MS-induced pain hypersensitivity. These findings also suggest that withametelin may be useful in the treatment of neurological disorders in which oxidative stress has been principally implicated.

EPoster 12

Neuromodulation for Brain Disorders

Ethnomedicinal Plants Classified as Functional Foods used for the Management of Brain Wellness in Bangladesh

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Background

Consumption of medicinal plants as food furnishes Phyto-nutrition to the human body and improves their wellbeing due to a combination of bio-metabolites. Worldwide in a traditional medication system including Bangladesh, medicinal plants have been applied as both neuro-therapeutic purposes and nutrients provider for the wellbeing of psychological states e.g. anti-depressant, anti-anxiety, anti-convulsions, anti-dementia, antipsychotic, and cognitive enhancement, etc.

Objective

The present study aimed to compile ethnomedicinal plant-based functional foods used by folk medicine practitioners (FMPs) of Bangladesh for the management of brain health.

Method

By using online databases like PubMed, Google Scholar, ResearchGate, Elsevier to retrieve literature by applying multiple keywords such as “ethnomedicinal plants, functional foods, Bangladesh”. The listed plants were then evaluated by using relevant pharmacological studies to claim their neuroprotective properties.

Results

A total of 32 medicinal plants have been listed that were used by FMPs as functional foods for the management of multiple neurology related ailments. From the study it showed that Plants like Aegle marmelos, Bacopa monnieri, Centella asiatica, Clitoria ternatea, Mentha arvensis, Moringa oleifera, Spilanthes acmella, Syzygium aromaticum, Syzygium cumini, Terminalia chebula, and Vitex negundo pharmacologically also possessed neuroprotective properties.

Conclusion

Plant-based foods are an emerging interest thanks to their nutritive supports and therapeutic benefits, and some ethnomedicinal plants of Bangladesh which were classified as functional foods have also been biologically reported to have several neurological health benefits. Our study will help the scientific communities to conduct further research to develop therapeutic guidelines that are needed to ensure unquestionable safety appraisal.

EPoster 13

Stroke, Cerebral Small Vessel Disease, Dementia, Neurodegeneration, & Traumatic Brain Injury Challenge

Investigating the Risk Factors for Stroke and the Most Effective Way to Minimize it

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Background and objective:

Stroke is famed as a major health problem in society, the most common and one of the most complex neurological diseases, the third leading cause of death in the world after cardiovascular disease and cancer, which is included several risk factors. Moreover, stroke is one of the popular causes of physical and mental disabilities in people with the abovementioned disease. By observing the current situation, the need for the present study to investigate the risk factors for stroke seems crucial.

Methodology:

The present study was investigated by an online review of Persian and English articles published with the keywords stroke, risk factor, and dangerous elements in academic databases, PubMed, and Google Scholar. Ultimately, from 35 articles in the initial stage, 21 cases were selected in three months, and then eighteen cases were included in the final study according to the hypothesized criteria.

Results:

Based on processed studies, we can find out that ischemic stroke is the most regular type of one that contains familiar risk factors such as over 60 years of age, hypertension, diabetes, hyperlipidemia, and heart disease, respectively. It is more common in women and urban people than men and rural individuals.

Conclusion:

In conclusion, considering those mentioned risk factors, the best way to reduce strokes can be to put a screening program on the routine activities and also keep educating the public through the mass media to prevent strokes and their side effects subsequently.

Keyword: Stroke, Risk factors, disease, mental disability

EPoster 14

Innovations and technologies in Neuro-immunology, Movement disorders, Epilepsy and Migraine

Epigallocatechin-3-Gallate Loaded Polymeric Nanoparticles: a Promising Alternative for Temporal Lobe Epilepsy

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Background: Temporal lobe epilepsy (TLE) is the most common type of pharmacoresistant epilepsy in adults. Many studies are focused on finding new treatments for those forms of epilepsy, which do not respond to the available drugs. In that sense, Epigallocatechin-3-gallate (EGCG) has aroused much interest because of its multiple therapeutic effects, but its instability compromises the potential effectiveness. In the last decades, controlled drug delivery systems, such polymeric nanoparticles (NPs), have been increasingly studied due to its potential for protecting drug integrity and perform a specific targeted drug deliver.

Objective: To design PEGylated-PLGA NPs of EGCG in order to improve drug stability, increase its brain delivery, and evaluate its effectiveness in an animal model of TLE.

Methods: EGCG NPs were prepared by the double emulsion method and cytotoxicity, behavioral, Fluoro-Jade C, Iba1 and GFAP immunohistochemistry studies were carried out to determine their effectiveness.

Results: EGCG NPs showed an average size of 169 nm, monodisperse population, negative surface charge, encapsulation efficiency of 95% and sustained release profile. Cytotoxicity assays exhibited that these nanocarriers were non-toxic. Behavioral test showed that nanoparticles reduced most than free drug the number of epileptic episodes and their intensity. Neurotoxicity and immunohistochemistry studies confirmed a decrease in neuronal death and neuroinflammation.

Conclusion: Epigallocatechin-3-gallate PEGylated-PLGA nanoparticles could be a promising, innovative and suitable strategy for the treatment of Temporal Lobe Epilepsy.

EPoster 15

Neuromodulation for Brain Disorders

Hyposmotic Regulation of Vasopressin Neuronal Activity through Astrocytic Sensation of Osmotic Changes in Rat Supraoptic Nucleus

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Hyposmotic challenge (HOC) dually modulates vasopressin (VP) neuronal activity and VP secretion. This dual modulatory feature seems to be tied to dual plastic change in astrocytic morphology, particularly glial fibrillary acidic protein (GFAP) and aquaporin 4 (AQP-4). To test this possibility, we observed responses of rat supraoptic nucleus (SON) to HOC. We found that HOC significantly increased GFAP filaments and protein levels within 5-10 min in vitro and in vivo; at 20-30 min, this response became insignificant, with delayed and weak reactions in vivo. This GFAP plasticity is closely related to the expression of AQP-4. In vivo HOC also significantly increased AQP-4 at 10 min, which became insignificantly at 30 min. Interestingly, this general recovery of AQP-4 levels in the SON was accompanied with dramatic increases in AQP-4 around blood vessels. Then, we examined molecular interactions between GFAP and AQP-4 at 10 min of in vivo HOC by co-immunoprecipitation. The result showed that HOC transiently increased molecular association between the two molecules. Lastly, we tested the effect of osmotic changes on the firing activity of VP neurons in the present of TGN-20, a specific AQP4 channel blocker. TGN-20 blocked the rebound excitation following HOC as well as the excitatory effects of hyperosmotic solution. Together with our previous findings, we conclude that GFAP-associated AQP-4 expression plays a critical role in astrocytic morphologic plasticity and that this astrocytic plasticity is involved in dual effect of HOC on VP neuronal activity and VP secretion.

EPoster 16

Neuromodulation for Brain Disorders

Stimulate to Learn: an Advantage in Motor Learning and Performance with Task-Irrelevant Background Vibratory Noise in Young Adults with ADHD

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Background. Young adults with ADHD often gain less than expected from practice sessions well-suited for their peers. Objective. Here, we tested whether task-irrelevant, low-intensity vibratory stimulation (VtSt), suggested to modulate motor learning, may compensate for such learning deficits.

Method. Participants with ADHD and typical controls were given training, either with or without VtSt, on a sequence of finger opposition movements. Motor performance was assessed before and immediately after training, overnight and at 1week post-training. Participants were randomly assigned to either (1) training with VtSt: ADHDVtSt and ContVtSt groups; or (2) training without VtSt: ADHDNoVtSt and ContNoVtSt groups (N=16 per group).

Results. Under VtSt, typical individuals had reduced overnight, consolidation phase, gains; performance partly recovering one week later. In contrast, participants with ADHD benefitted from VtSt both during the acquisition (online) and the overnight skill consolidation (offline) phases. One week later, both groups showed robust retention of the gains in performance, but when tested with background VtSt, individuals with ADHD outperformed their typical peers.

Conclusion. Our results suggest that procedural memory acquisition and consolidation processes are extant in young adults with ADHD and this potential can be best unveiled in specific bio-behavioural conditions afforded during training. Minor background vibro-tactile stimulation may constitute an effective aid during procedural learning in ADHD. We propose that ADHD can confer advantages in performance, learning and skill memory consolidation in specific ‘noisy’ conditions that adversely affect typical adults; we conjecture that the effects of VtSt are contingent on baseline arousal levels.

EPoster 17

Technology-Based Cognitive Training and Rehabilitation

Cognitive-Motor Interference in Chronic Stroke Patients: A Functional Connectivity Study

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Background: The ability to dual-task is important part of daily life. Consider for instance how often you walk with a friend while holding a conversation or perform mental arithmetic while reaching for an item on your grocery list. The simultaneous performance of such cognitive and motor tasks can affect the performance of one or both tasks, and this phenomenon is collectively known as cognitive-motor interference (CMI). It is well-known that stroke patients often exhibit CMI under certain dual-task conditions. However, the neural mechanisms underlying this behavioral observation is not well-understood.
Objective: The aim of this study is to investigate brain functional connectivity during CMI in chronic stroke patients.

Method: Six stroke survivors (3 months post-stroke) participated in this study. Subjects performed a circle drawing task using an upper limb rehabilitation robot both alone (single-task) and together with a concurrently performed Serial 7 task (dual-task). In both conditions, neural activity was simultaneously recorded using EEG. Functional connectivity (weighted phase-lag index; WPLI) and graph (minimum spanning tree; MST) metrics were then computed from the EEG data to assess the neural activity associated with CMI.

Results: As expected, we found that motor task performance was significantly worse in the dual-task condition indicating CMI. Functional connectivity and graph analyses revealed a significantly lower WPLI and a significantly more integrated MST during dual-task performance.

Conclusion: A reduction in connectivity strength together with a more congested network led to motor performance declines during CMI in chronic stroke patients.

EPoster 18

Innovations and technologies in Neuro-immunology, Movement disorders, Epilepsy and Migraine

Effects of Non-permitted Food Colorants on Motor Coordination and Biochemical Alterations in Corpus Striatum of Rat Brain

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Background: Food additives are used to colour several food materials, cosmetics and pharmaceutical compounds. There sources can be natural and artificial but the artificial one only be permitted when authorized by the regulatory and monitoring organization like FDA or EFSA etc. Non-permitted food colorants (NPFCs) are banned or prohibited but still has been reported specially in developing countries. Long term use of permitted and non-permitted food colorants (azo dyes) have been found to cause chronic toxicity associated with neurobehavioral abnormalities. Objective: To investigate the effects of NPFCs (metanil yellow-MY, malachite green-MG and sudan III-SIII) on neurobehavioral alterations and biochemical alterations in corpus striatum of rats.

Methods: Rats were grouped into 5 groups including control, MY, MG, SIII and mixture (YGR). Rats were treated with their respective treatment in 1% gum acacia for 60 days. Balance and endurance were analysed using food print and tight rope hanging behavioral tools and then rats were decapitated to isolate the brain region and processed for biochemical analysis.

Results: The treated groups showed a significant decrease grip strength, stride length difference and increase in foot overlap assessed through tight rope and gait pattern analysis as compared to controls. A significant increase in lipid peroxidation and decreased level of reduced glutathione, superoxide dismutase, catalase were observed in NPFCs treated rats as compared to controls.

Conclusions: The results of the present study suggested that NPFCs caused neurobehavioral toxicity by affecting the functioning of corpus striatum linked with biochemical alterations.

EPoster 19

Stroke, Cerebral Small Vessel Disease, Dementia, Neurodegeneration, & Traumatic Brain Injury Challenge

Landscape of Variants in AGT, MGMT and TP53 in Adult with Astrocytoma: A Study in Mexican Population

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Introduction: Astrocytomas are the most common primary malignant brain tumors in adults. They are highly disabling, and the prognosis of patients is dismal, particularly for grade IV astrocytomas. Risk factors for astrocytoma are poorly understood, nonetheless, genetic factors play a major role. AGT is a gene related to regulation of intravascular volume through the renin-angiotensin system (RAS). AGT variants could be associated to the risk of glioma, as paracrine RAS activity in the encephalon has been observed. TP53, one of the most frequently altered genes in cancer, could a be useful risk marker as well. MGMT is a known predictor of treatment response in astrocytoma, however, its role in astrocytoma risk is undefined.

Objective: The aim of this work was to evaluate the association of variants in AGT, MGMT, and TP53 with astrocytoma risk.

Materials and Methods: Peripheral blood samples from patients with astrocytoma from the Instituto Nacional de Neurologia and Neurocirgua (INNN) were analyzed through targeted panel sequencing for AGT, MGMT, and TP53. Genetic markers found were compared against a control population integrated by individuals with Mexican ancestry from the 1000 genomes project. Significant markers were then further validated in a cohort of patients with glioma from TCGA.

Results: AGT variant rs1926723 and MGMT variant rs16906252 were significantly associated with risk of astrocytoma in both INNN and TCGA cohorts.

Conclusions: In this study we identified and validated in a second cohort genetic variants associated to astrocytoma risk. These markers can be studied in blood easily and safely.

EPoster 20

Neuromodulation for Brain Disorders

IGF1 Gene Therapy Promotes Synaptic Remodeling by Microglia

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Background: Microglia are the resident immune cells of the central nervous system (CNS). These cells play important roles in healthy and diseased brain in order to maintain homeostasis. One of these roles is the maintenance of synapses. Microglia promote synaptogenesis by secreting growth factors and regulate the number of synapses during the process of synaptic pruning. In the adult CNS, microglial ramifications interact with synaptic terminals and synaptic clefts, dendritic spines and astrocytic processes. During aging, microglia go through an age-related degeneration characterized by reduced migratory and phagocytic capacity, low production of neurotrophic factors and are more insensitive to stimuli. Consequently, these alterations lead to an impaired surveillance of the surrounding environment, impaired synaptic regulation and, therefore, loss of brain homeostasis. Thus, it is of great interest to design strategies to keep the microglia working correctly. In this regard, IGF1 has shown to be able to act on aged microglia, promoting their proliferation and reactivity.

Objective: Our aim is to evaluate the effect of IGF1 gene therapy on senescent microglial activity.

Method: We implemented IGF1 gene therapy in aged rats of 28 months-old and studied how it affected microglial phagocytic activity.

Results: We observed that microglia increased phagocytic activity and synaptic remodeling. Moreover, these animals presented better motor performance.

Conclusion: These results suggest that IGF1 gene therapy could be an effective treatment to modulate microglia activation and promote motor improvement.

EPoster 21

Boosting Brain Innovation & Brain Technologies

Effect of an Intra-Hypothalamic Gene Therapy with IGF-1 on Behavioral Parameters

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BACKGROUND

Aging is characterized by a coordinated organs and tissues functional impairment. The hypothalamus, a region known to regulate many basic functions such as growth, development, reproduction and metabolism, is proposed as a regulatory center of aging. Evidence demonstrates that the inhibition or activation of microglial or neural transcription factor NF-κB of the basal hypothalamus (BH) affects the life expectancy and the "beginning" of aging, as well as the release of GnRH. There is solid evidence that middle age (MA) rats do not respond to estradiol positive feedback with an appropriate modulation of excitatory and inhibitory hypothalamic neurotransmitter release. This imbalance could cause reduced activation of GnRH neurons, reduced GnRH release, and an abnormal LH surge.

OBJECTIVE

It is well known that IGF-1 plays a physiological role in neuroprotection and neuroinflammation. We decided to investigate the effects of intrahypothalamic gene therapy for IGF-1 in MA rats. We propose that transgenic IGF-1 will modulate neuroinflammation and delay reproductive senescence.

METHODS

We employed recombinant adenovirus RAds as a carrier to deliver either a therapeutic (IGF-1) or control gene (DsRed) and performed intra-hypothalamic stereotactic injections with Rad-IGF-1, Rad-DsRed or PBS.

CONCLUSION

The MA rat brain reduction of factors like IGF-1 could be the reason of the affected modulation on excitatory and inhibitory hypothalamic release. These findings provide a link between inflammation, response to stress and systemic and cerebral aging.

EPoster 22

Promoting Brain Wellness & Modifying the Course of Cognitive Dysfunction and Dementia

Postoperative Cognitive Dysfunction in Cardiac Surgery Employing Cardiopulmonary Bypass. The Past and the Future

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Postoperative cognitive dysfunction (POCD) involves decline in several cognitive domains after surgery and is particularly common following cardiac surgery. It is connected with increased morbidity and mortality, prolonged hospitalization, increased health care costs, and may have an adverse impact on social functioning and health-related quality of life. Given the potential effects of such cognitive dysfunction on quality of life, it is important to investigate the possible mechanisms to limit its occurrence. Recent advances in surgical technology may assist in achieving that and play critical role of the surgical methods utilized in the development of POCD and have implications for the clinical management and patients' quality of life. Cognitive stability plays a significant role in quality of life and daily activities, especially in heart surgery elderly patients. POCD was investigated in late 60's and there are a lot of studies with this topic. Although a progress was made, the etiology of POCD is not so clear although it seems that inflammatory response plays an important role in the complex pathogenesis of POCD. However, the doubts about POCD, obligatory neuropsychological evaluation prior and after cardiac surgery, can be a good way to detect at-risk patients.

EPoster 23

Stroke, Cerebral Small Vessel Disease, Dementia, Neurodegeneration, & Traumatic Brain Injury Challenge

Novel Approach of Insulin-like Growth Factor-1 Gene Therapy Modulates Reactive Gliosis after Traumatic Brain Injury

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Reactive gliosis is a key feature and an important pathophysiological mechanism underlying chronic neurodegeneration following traumatic brain injury (TBI). In this study, we have explored the effects of intramuscular IGF-1 gene therapy on reactive gliosis and functional outcome after an injury of the cerebral cortex. Young adult male rats were intramuscularly injected with a recombinant adenoviral construct harboring the cDNA of human IGF-1 (RAd-IGF1), with a control vector expressing green fluorescent protein (RAd-GFP) or PBS as control. Three weeks after the intramuscular injections of adenoviral vectors, animals were subjected to a unilateral penetrating brain injury. The data revealed that RAd-IGF1 gene therapy significantly increased serum IGF1 levels and prevent working memory deficits after one week of TBI. At the same time, when we analyzed the effects of therapy on glial scar formation, the treatment with RAd-IGF1 did not modify the number of glial fibrillary acidic protein but we observed a decrease in vimentin immunoreactive astrocytes at 7 days post-lesion in the injured hemisphere, compared to animals treated with RAd-GFP. Moreover, IGF-1 gene therapy reduced the number of Iba1+ cells with reactive phenotype and the number of MHCII+ cells in the injured hemisphere. These results suggest that intramuscular IGF-1 gene therapy may represent a new approach to prevent traumatic brain injury outcomes in rats.

EPoster 24

Promoting Brain Wellness & Modifying the Course of Cognitive Dysfunction and Dementia

N-Acetylcysteine Prevents Recognition and Object Location Memory Impairment in Adolescent Rats Subjected to a Chronic Ethanol Binge Model.

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Background: Ethanol (EtOH) binge drinking during adolescence damages cerebral areas such as prefrontal cortex and hippocampus, causing cognitive dysfunction. N-Acetylcysteine (NAC) is presented as a promising neuroprotective chemical to treat dysfunctions caused by EtOH.

Objective: To investigate NAC protection on object location and recognition memory in an experimental model of chronic ethanol binge.

Methods: Adolescent male Wistar rats (n=132) were allocated into six groups, submitted to intraperitoneal injection of NAC or vehicle (CTRL), followed by intragastric injection of EtOH (3 and 6 g/kg) or distilled water (DW) during 4 weeks. After treatment, animals were submitted to recognition and object location tests. For statistical analysis, two-way ANOVA for independent samples, followed by Tukey test (p 0.05). CEUA protocol nº 46/2019.

Results: Rats treated with EtOH 3 and EtOH 6 presented impairments in short-term memory in object recognition and location, compared to the CTRL/DW group. Pretreatment with NAC reduced the impairment in both treatment groups, with respect to both recognition memory and in the location memory. Treatment with EtOH 3 and EtOH 6 impaired long-term object recognition and location memory, compared to the CTRL/ DW group. Pretreatment with NAC reduced object recognition dysfunction only in the CTL / EtOH 6 group. In the location test, cognitive impairment was prevented in EtOH 3 and in EtOH 6

Conclusion: Pretreatment with NAC reduces impairment of short and long-termed object recognition and location memory in adolescent rats submitted to a model of chronic ethanol binge, displaying protective effects on cognitive impairments caused by EtOH.

EPoster 25

Mapping Brain Health and Brain Network Imaging

Study of the Structural Covariance of the Brain Regions Described by RST Applying an Exploratory Factorial Analysis to Different Atlasets

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MRI research has evidenced the existence of structural covariance patterns between brain regions. Brain regions have been shown to covary bilaterally, and to a lesser extent, with other brain regions. According to the Reinforcement Sensitivity Theory, the striatum nuclei are involved in BAS and the limbic system in FFFS/BIS. In this study, we investigated whether there are structural covariance patterns that group the brain structures described by RST.

We applied an EFA over Voxel-Based Morphometry (VBM) dataset in a wide-sample of 300 healthy volunteers to a total of four datasets with different segmentations: the Neuromorphometrics Atlas, the Hammers Atlas, the Cobra Atlas and the AAL3 Atlas.

The results shown three structural covariance patterns (Alexander-Bloch, Giedd & Bullmore 2013) across atlases: generally, (1) one composed by frontal and cingulate brain areas, (2) other composed by the hippocampus and amygdala, and (3) other composed by the accumbens and caudate nuclei.

These results confirm the existence of structural covariance patterns between brain regions and reveal their agrupation into factors that correspond to neuroconductual systems proposed by RST (Gray, 1970). Nevertheless, further research is needed to understand the relationship of these structures with the neurobehavioral systems of the sensitivity to reward and sensitivity to punishment.

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EPoster 26

Stroke, Cerebral Small Vessel Disease, Dementia, Neurodegeneration, & Traumatic Brain Injury Challenge

TGN-020 in the Supraoptic Nucleus Inhibits Vasopressin Hypersecretion and Reduces Brain Injury in Rats of Focal Ischemic Stroke

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Background In ischemic stroke, vasopressin hypersecretion causes cerebral swelling and brain injury.

Objective To understand mechanisms underlying vasopressin hypersecretion and its correlation with brain injury, we investigated effects of blocking astrocytic aquaporin 4 (AQP4) in the supraoptic nucleus (SON) on vasopressin neuronal activity and cerebral injuries in a rodent model of unilateral middle cerebral artery occlusion (MCAO).

Methods Establishing MCAO without or with microinjection of TGN-020 into the SON of adult male rats; performing Western blots and immunohistochemistry to determine the expression levels and spatial distribution of functional proteins in the SON and the cerebral cortex.

Results MCAO increased plasma vasopressin levels, caused neurological damage and increased glycogen synthase kinase 3β (GSK-3β) in the SON and the cortex of MCAO side of the brain. In the SON, MCAO significantly increased c-Fos in vasopressin neurons and astrocytic somata of the ventral glial lamina. MCAO significantly reduced glial fibrillary acidic protein (GFAP) and AQP4 around vasopressin neurons, which accompanied separation of GFAP from AQP4. By contrast, microinjection of TGN-020 into the SON to block AQP4 blocked MCAO-evoked GSK-3β increase as well as the reduction of AQP4 relative to GFAP around vasopressin neurons in the SON. In the cortex, application of TGN-020 in the SON also blocked MCAO-evoked increase in GSK-3β and AQP4.

Conclusion These findings indicate that MCAO disrupts interactions of GFAP with AQP4 in astrocytic processes in the SON to increase vasopressin neuronal activity. Blocking AQP4 in the SON potentially reduces ischemic brain injury by reducing vasopressin neuronal activity.

EPoster 27

Mapping Brain Health and Brain Network Imaging

Alterations in Brain Networks of the Cocaine-Addicted Brain

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Magnetic resonance imaging (MRI) studies have demonstrated that the human brain is organized into complex structural and functional networks (Alexander-Bloch, Giedd and Bullmore, 2013; Bassett and Gazzaniga, 2011; Bullmore and Sporns, 2009). In this study, our purpose was to investigate whether there are differences in structural networks of the healthy brain and the cocaine-addicted brain.

To do that, we carry out a Source-based Morphometry analysis (SBM) (Xu et al., 2009; Pearlson et al., 2015) over structural MRI Voxel-based Morphometry dataset in a total of forty healthy volunteers and forty cocaine-addicts patients under dependence treatment recruited from the Addictive Behaviour Unit.

Taking into account the effects of age and TICV, the results showed significant differences between both groups in many Independent Components (CI). The ICs that group the structures involved in the Executive Control Network, the Default Mode Network, the Primary Motor Network and the Primary Visual Network, among others, stand out. In other ICs that group more diffuse structures such as the hippocampus or basal ganglia, differences were also found between groups.

Therefore, we can conclude that there is evidence of neurostructural differences in the brain networks of the addict brain. However, further investigation is required to understand the neurobiological basis underlying the differences found.

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EPoster 28

Promoting Brain Wellness & Modifying the Course of Cognitive Dysfunction and Dementia

The Effect of Gluten Content of Diet on Anxiety Behavior and Learning in Rats

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Gluten related disorders have been associated with neurological and psychiatric diseases. The studies showed that people who have celiac disease or gluten sensitivity has higher risk of having cognitive disorders, major depression and schizophrenia, which indicates that gluten may have an effect on CNS. However, there was no previous study investigating effect of gluten intake level on cognitive abilities and behavior.

The aim of our study is to examine the impact of diets having different levels of gluten on anxiety-related behavior and learning in rats.

42 male Wistar rats were weaned at 25 days of age and randomly divided into 3 diet groups; gluten-free, standard-chow and high-gluten diet. After 90 days of feeding, open field(OF), elevated plus maze(EPM) and Morris water maze(MWM) tests were performed.

In our preliminary analysis, we found that in OF, total distance moved, mean velocity and frequency to be found at the central field significantly higher in gluten-free group compared to high-gluten group. In EPM, high-gluten group was found to be significantly less frequent at the open arms and have higher latency to enter the open arms than the standard-diet group which is consistent with the OF findings. In MWM, only parameter showed significant difference between the groups was latency to enter the target quarter.

The results indicate that anxiety-related behavior increase with the higher levels of gluten intake. Our findings may shed light on the impact of gluten intake level on health.

EPoster 29

Stroke, Cerebral Small Vessel Disease, Dementia, Neurodegeneration, & Traumatic Brain Injury Challenge

Negative Cardiovascular Reactivity: a Digger in the Brain

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Cardiovascular reactivity (CR) may be defined by beat indices (ratio or difference) between higher maximal or minimal heart rate on higher maximal or minimal pulse rate, recorded by Holter Arterial Pressure Measurements.

The aim of our current study was to examine the burden of positive (PCR) and negative (NCR) CR and its correlation with cardiovascular and cerebrovascular scales and cardiac biomarkers.

One hundred and thirteen acute ischaemic syndromes,32 chronic cerebrovascular diseases and 30 other neuropsychiatric diseases were recruited. Correlations with the number of negative and positive CR events were evaluated.

Number of NCR events were positively correlated with CHADVasc2, HAS BLED, Hachinski scale, high sensitive troponin, pro-brain natriuretic peptide (pro-BNP), BNP, negatively with MMSE. In patients suffering from atrial fibrillation, they negatively correlated with GCS at admission and at day VII, positively with MRS at day VII. Inverse correlations were found with PCR. Correlations between CR, UPDRS and ADLQ were influenced by circadian rhythms.

Our data highlight that episodes of NCR may subtly occur and progressively impair cerebrovascular and cardiovascular function, leading to chronic ischaemic sufferance. They may trigger acute events, worsen their clinical course, hinder their recovery and be harbinger of outcomes. High heart rate predicts greater total lesion volume and higher number of micro- and macro-structure white matter lesions (Fuhrmann D. et al., 2019). However, the irreversibility of ischaemic sufferance seems to be related to low CR, itself responsible of reduced perfusion. The ratio between necrotic and apoptotic phenomena may account for the entity of diffusion.

EPoster 30

Big Data and the Brain

SARS CoV2 Experience in the South of Italy

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Coronaviruses (CoV) were described as mite agents. Asian outbreaks of CoV-related Severe Acute Respiratory Syndrome and Middle East Respiratory Syndrome pointed out the risk of diffusion. CoV2 SARS threatened health care and economical steady state all over the world.

We report our experience in the Cov2 Unit.

A CoV2 prognostic scale was conceived for promptly defining clinical setting and prognosis, based on the following items: age, sex, arterial hypertension, type II diabetes mellitus, chronic cerebrovascular disease, chronic coronary syndrome, pulmonary emphysema, chronic renal failure, lymphocytopenia, superimposed infections. Hypoxic encephalopathy, absolute lymphopenia with neutrophilia were hallmarks of the disease in elderly patients. Mortality was around 20% in 80, 7% in 65-80, 3% in 35-65, 0% in 35 years old.

Early diagnosis accounted for low in-hospital mortality. Comorbidities negatively interfered with recovery. The negativity of rapid test may include false negative. Oropharyngeal swab detects the presence of viral RNA, not alive virus. Its negativity does not exclude ongoing infection. The diagnosis must be confirmed by radiological examinations. Plasma biomarkers of epithelial and endothelial injury may be useful for disease monitoring. In elderly patients Invasive Oxygen Therapy is a difficult task because of individual heterogeneity of pathological findings. Weaning from IOT is even more arduous. Ventilator-associated-lung-injury seems to be related to the pathological process rather than to applied techniques. It is mandatory not to disappoint trust on health care. The most powerful strategy is health education. Home confinement and lockdown are extreme measures which must be rationally applied.

Stroke, Cerebral Small Vessel Disease, Dementia, Neurodegeneration, & Traumatic Brain Injury Challenge, Technologies and Innovations for Diagnosis & Monitoring, Assistive Technology, Therapeutic Technology & Care-Supportive Technology for Dementia, Neurodegeneration and Brain Injury

6-OHDA Parkinsonism Model: an Approach to Non-Motor Symptoms in Parkinson`s Disease

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Neurodegenerative disorders, such as Parkinson’s disease, account for a variety of non-motor symptoms (NMS) including cognitive and emotional alterations. Several animal models of PD focus on motor symptoms. However, NMS represent a growing area of interest in the neuroscience field. The main goal of our work was to assess cognitive emotional behavioral changes in a parkinsonism model induced by 6-OHDA and evaluate different dopamine cell changes in brain structures involved in these behaviors. Adult male Wistar rats were bilaterally infused with 6-OHDA into the Caudate-Putamen nucleus (CPu). At 3rd week of neurotoxicity, we performed Contextual Fear Memory, Open Field and Novel Object Recognition tests. Subsequently, animals were fixed and their brains were obtained for stereology analysis. Both groups presented similar levels of freezing 24 h after conditioning. OP results showed no difference regarding locomotor activity, although rearing and grooming behavior was decreased in 6-OHDA rats. Respecting NOR test, 6-OHDA animals spent less time exploring the novel object. This behavior was coincident with a lower number of TH+ cell number in substantia nigra and a decrease of the reactive area in prefrontal cortex and CPu in 6-OHDA rats. These results showed a dopamine depletion and cognitive alterations before locomotor impairment. This prompts us to further study other emotional and cognitive aspects under this experimental model, such as anxiety-like behavior.

EPoster 32

Promoting Brain Wellness & Modifying the Course of Cognitive Dysfunction and Dementia

Neuroprotective Effect of Physical Exercise

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Background: Normal aging is associated with alterations in brain structure and function as well as with cognitive changes. The effect of physical activity on somatic health is well established. However, the effect of physical activity on brain health and cognitive functions is not well investigated. Cognitive functions include memory, attention, visual-spatial as well as complex cognitive functions, including thinking (abstract, cause and effect, creative thinking, planning) and language functions. The level of cognitive functions declines with age and it is also affected in neurodegenerative diseases. Thus, lifelong physical activity could be suggested as a brain-anti-aging intervention and as a non pharmacological modulator of neurodegenerative diseases. Objective: This manuscript aimed to critically review data on the effect of physical activity on cognitive functions. Methods: Pubmed, Scopus,Google Scholar, Science Citation Index were searched with the search terms “physical activity”, ‘exercise’, “cognitive functions”, “memory”, “attention”, “thinking”, ‘neurodegenerative diseases’. The search covered the period up to and including August 2020. The main outcome was the effect of physical activity on cognitive functions. Both animal and human studies met inclusion criteria Results: Evidence suggests that physical exercise modifies metabolic, structural and functional dimensions of the brain resulting in preservation of cognitive functions in older adults. More importantly, observational studies indicate that the neuroprotective effect of physical exercise towards cognitive performance is dose-dependent. Data derived from clinical studies also support the neuroprotective effect of physical exercise, although there are clinical studies with negative results. The mechanisms underlying the neuroprotective effect of physical exercise include elevated neurotrophin levels, improved vascularization, facilitation of synaptogenesis, mediation of inflammation, reduced disordered protein deposition. Conclusion: Physical activity has a protective effect towards cognitive functions.

EPoster 33

Stroke, Cerebral Small Vessel Disease, Dementia, Neurodegeneration, & Traumatic Brain Injury Challenge

Effects of Thyroid Hormone T3 in Behavior and Neuroinflammation in an Experimental Model of Alzheimer's Disease.

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Background: Alzheimer`s disease (AD), is a chronic neurodegenerative disease, is the most common form of dementia. Alterations in metabolic parameters have been associated with an increased risk of dementia, among them, thyroid function has been given greater importance in AD pathology in recent years.

Objective: Evaluate the effects of triiodothyronine (T3) thyroid hormone supplementation in cognitive performance related to animals` memory and neuroinflammation.

Method: Males wistar rats were used (n=7-8), received bilateral injections (totaling 4 µL) of vehicle or streptozotocin (2 mg / Kg; Sigma) in the lateral ventricle by stereotaxic surgery to induce AD model. The animals that received the vehicle and STZ were supplemented with a daily intraperitoneal supra-physiological dose of 1.5 µg / 100g of triiodothyronine (T3) or the same volume of vehicle (saline), from the day of stereotaxic surgery, for 30 days. Behavioral tests assessed cognition and neuroinflammation was assessed by Western Blot. All the experiments were approved by animal ethical committee.

Results: The data analysis revealed that T3 supplementation promotes improvement in cognition in the object recognition index of the short and long-term tests (p 0.01), it also promoted a reduction in GFAP levels ($p = 0.033$) after Treatment with T3 in animals with AD.

Conclusion: Our data provide evidence of the positive effects that T3 supplementation has in relation to cognition and neuroinflammation in a model of AD. Thus, this work highlighting the importance of this therapy and identify mechanisms of action that can be useful on the establishment of therapeutic strategies.

EPoster 34

Innovations and technologies for Stroke, Neurodegeneration and brain disease

Drug Delivery of PACAP Loaded with gH625-Liposome through a Dynamic Blood-Brain Barrier Model and its Effect as Neuroprotective Tool

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Pituitary adenylate cyclase-activating polypeptide (PACAP) is a peptide of the secretine/glucagone family. In the central nervous system (CNS) can act as neuroprotective agent but it has a rapid degradation. A nanodelivery tool has been needed to send PACAP in CNS. So, we use a cell-penetrating peptide, gH-625 derived from glycoprotein H of the Herpes simplex virus 1. In vitro dynamic blood-brain barrier (BBB) model used is a bioreactor, with a double flow chamber separated by a porous membrane. On this membrane, bEnd.3 cells were seeded to form tight junction, confirmed by ZO1 immunofluorescence(Fig.1).gH625-liposomes-PACAP-Rho were injected in the upper flow to follow the delivery process up to 2 hours: an increase of PACAP-Rho in the lower chamber was showed compared to PACAP loaded with liposomes(Fig.2).Furthermore, we evaluated the neuroprotective effect of PACAP on dopaminergic neurons treated with MPP+, a neurodegenerative agent. After 24h, Prestoblue assay showed a significant increase in cell viability in the presence of PACAP at concentrations of 10-7M and 10-8M both as prevention of MPP + and at the same time(Fig.3).Subsequently, bEnd.3 cells were connected with a single chamber containing dopaminergic neurons treated with MPP+ and the passage of gH625-lipo-PACAP within these two systems was evaluated by an ELISA assay. The results showed that after 60 minutes cells are continuously exposed to a PACAP concentration of 10-8 M(Fig.4).This concentration appears to be the lowest to induce a neuroprotective effect, as observed in the PrestoBlue assay. Our nanodelivery tool can be useful to transport a neuroprotective agent through in vitro BBB model in dynamic conditions

Fig.1:

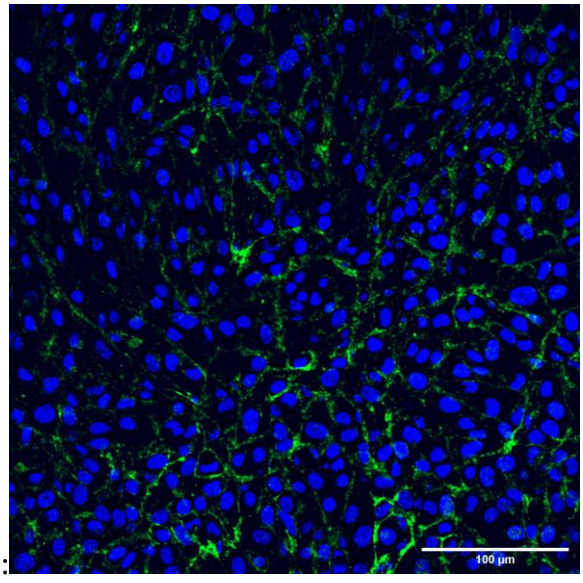


Fig.2

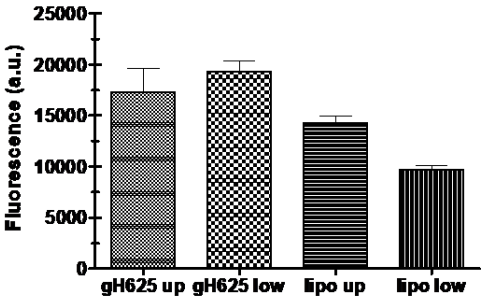
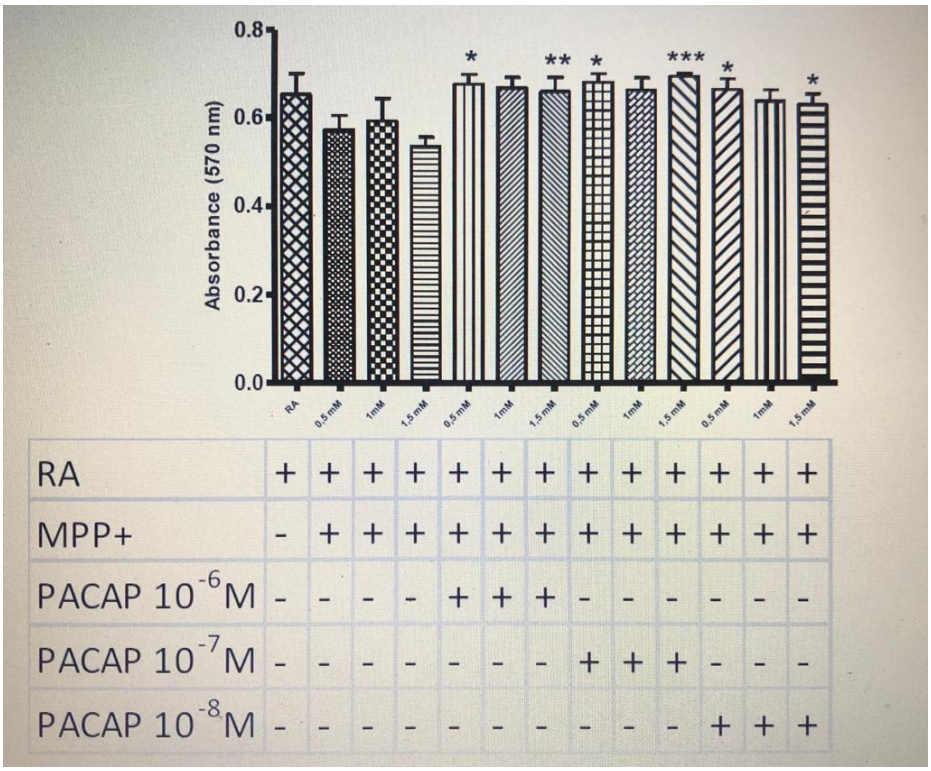
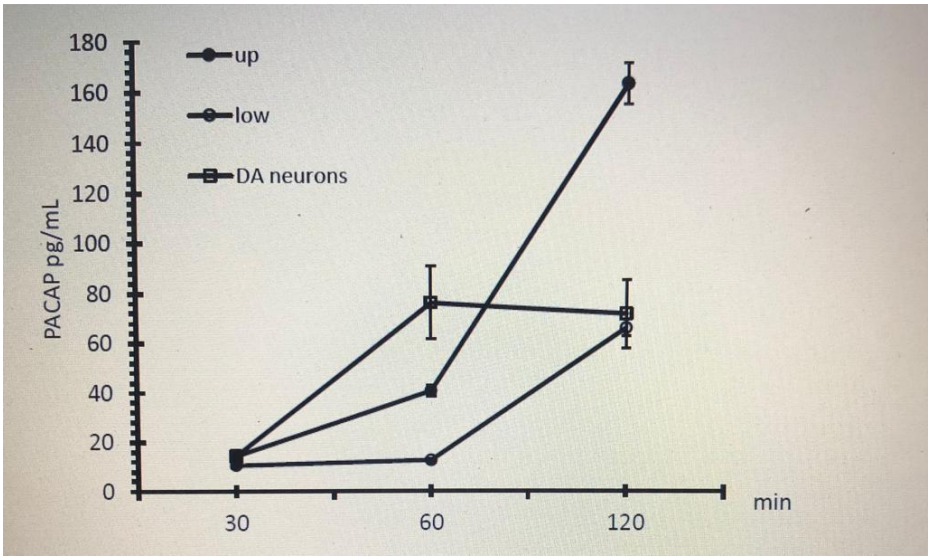


Fig.3



RA	+	+	+	+	+	+	+	+	+	+	+	+	+
MPP+	-	+	+	+	+	+	+	+	+	+	+	+	+
PACAP 10^{-6}M	-	-	-	-	+	+	+	-	-	-	-	-	-
PACAP 10^{-7}M	-	-	-	-	-	-	-	+	+	+	-	-	-
PACAP 10^{-8}M	-	-	-	-	-	-	-	-	-	-	+	+	+

Fig.4



EPoster 35

Stroke, Cerebral Small Vessel Disease, Dementia, Neurodegeneration, & Traumatic Brain Injury Challenge

Spatiotemporal Profiles of Cleaved-Caspase-3 and Caspase-3-Cleaved Tau (Asp421) and their Association with Microvascular and Neuronal Alterations in the Hippocampus Following TBI in Rats: from the Mechanisms to the Potential Therapy

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Background: Traumatic brain injury (TBI) increases risk of development of tauopathies, which are associated with cognitive impairment. Our recent preclinical studies have demonstrated that caspase-3-mediated apoptosis in the corpus callosum and thalamus is associated with chronic abnormal tau processing, axonal degeneration and microvascular changes following TBI. The goal of this study was to further investigate the spatiotemporal profile of caspase-3-mediated tau alternations in the hippocampus and its association with glial, neuronal and microvascular pathologies.

Method: We employed quantitative immunohistochemistry (IHC) and histopathology in paraffin embedded brain slices obtained at 24 hours, 1 week, and 1, 2 and 3 months after controlled cortical impact (CCI) injury and controls (sham and naïve) in adult Sprague Dawley rats. Analyses focused on spatiotemporal profiles of cleaved-caspase-3 (apoptosis) and caspase-3-cleaved tau truncated at Asp421 (caspase-3 cleavage product associated with tangle formation), ionized calcium-binding adapter molecule 1 (Iba1; microglia), glial fibrillary acidic protein (GFAP; astrocytes), endothelial barrier antigen (EBA; relevant to BBB), rat IgG (BBB disruption), intercellular adhesion molecule 1 (ICAM-1, relevant to inflammation), cluster of differentiation 68 (CD68; activated microglia/macrophages), cluster of differentiation 8 (CD8, cytotoxic T cells), cluster of differentiation 34 (CD34, hematopoietic stem and progenitor cells marker) and nestin (neural progenitor cells). Histological stains luxol fast blue (LFB)/cresyl violet were used to identify myelin pathology and neuronal cell death. For quantitative analyses, we employed full brain scan slide Aperio imaging, NIH ImageJ and in-house quantitative morphology algorithms.

Results: CCI resulted in cleaved-caspase-3 upregulation starting at 1 week and progressing up to 3 months in selected hippocampal regions, notably in molecular and polymorph layers of dentate gyrus, stratum lucidum, stratum lacunosum-moleculare and stratum radiatum fields of CA1 and CA3 regions. Accumulation of caspase-cleaved tau truncated at Asp421 was observed at 24 hours and 1 week after CCI as immunopositive structures with glial-like morphology. Starting at 1 month, caspase-cleaved tau-immunopositive tangle-like inclusions appeared. GFAP and Iba1 immunoreactivities were increased across the hippocampus at all time points and peaked at 24 hours and 1 week, respectively. Immunofluorescence experiments revealed profound colocalization of GFAP and caspase-cleaved tau at 24 hours and only limited colocalization at 3 months after CCI. Cleaved-caspase-3 upregulation was associated with differential spatiotemporal profiles of the markers used to assess blood-brain barrier and microvascular abnormalities (ICAM-1, CD34, EBA, IgG), inflammation (CD8, CD68), and neural progenitor cell expression (nestin).

Conclusion: Our results provide novel experimental data on the critical role of caspase-3 in the progression of tau pathology after acute TBI. The resulting alterations in complex neuronal, glial and inflammatory pathways lead to chronic hippocampal neuronal degeneration and microvascular reorganization, which could underlie cognitive deficits following TBI. This study suggests a potential for activated caspase-3 inhibition to be a therapeutic strategy for treatment of acute and chronic TBI, highlighting a wider therapeutic window of such treatment.

EPoster 36

Stroke, Cerebral Small Vessel Disease, Dementia, Neurodegeneration, & Traumatic Brain Injury Challenge

Stroke Education for APNS: How Can They Improve the Process?

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Background: The 1st state-wide study investigating the perceptions of stroke care in New Jersey, identified lack of documentation of stroke pathway/orders as one of the reasons affecting outcomes of care (Edfort, 2010). The issue identified as a reason for the missed measures at some hospitals were related to lack of documentation. Some items that were identified as affecting stroke care compliance were:
v Deep vein Thrombosis treatment by end of day 2- 79%
v Screen for dysphagia before oral intake– 77%
v Stroke prevention education- 72%
Objective: The need for guideline compliance for acute stroke care is an ongoing problem for all healthcare facilities. Edfort (2010) recommended implementing processes to address the deficiencies in stroke guideline compliance.
Method: In 2017, a second state-wide stroke study was conducted in New Jersey implementing Dr. Edfort's recommendations for improving stroke core measure compliance (Singh, 2017). This study identified the necessity of educating Advanced Practice Nurses (APNs) in stroke core measure management. APNs are front line staff in most acute care hospitals with prescribing capabilities; however, they may not be knowledgeable in the measures needed to manage stroke patients.
Results: Attending a stroke specific education program, attendees reported increasing their knowledge of managing stroke patients as well as increasing their proficiency level in care processes.
Conclusion: Providing an education program focusing on the core measures provides an advantage to our patients as well as keeping the health care facility in compliance with stroke management protocols.

Stroke, Cerebral Small Vessel Disease, Dementia, Neurodegeneration, & Traumatic Brain Injury Challenge, Promoting Brain Wellness & Modifying the Course of Cognitive Dysfunction and Dementia, Innovations and technologies for Stroke, Neurodegeneration and brain disease, Technologies and Innovations for Diagnosis & Monitoring, Assistive Technology, Therapeutic Technology & Care-Supportive Technology for Dementia, Neurodegeneration and Brain Injury, Technology-Based Cognitive Training and Rehabilitation, Digital Brain Technologies for Healthy Brains & Healthy Minds and for Brain Disorders, Boosting Brain Innovation & Brain Technologies

COVID-19 Pandemic could Lead to Further Psychological Trauma besides Mental Health Effects: Insights from a Pilot Survey done in India

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Treatment gap for mental disorders still remains very high in India. COVID-19 pandemic could lead to further psychological trauma besides mental health effects. These raise questions on our mental health status during COVID-19 pandemic. An online survey using DASS Scale 21 was conducted in the month of July, 2020 in India to know how COVID-19 has affected mental health status of people post COVID-19 pandemic. An online survey using DASS Scale 21 revealed that mental stress are being prevalent and alarms the second wave of mental stress post COVID-919 pandemic in India. Of the total 27 respondents 26 were from India while one from UK. The results clearly showed that anxiety and stress were prevalent from mild to moderate level and depression in mild level. As the uncertain future due to COVID-19 is becoming dominant, it is high time to prepare ourselves so as to prevent the second wave of mental health crises post COVID-19 pandemic.

EPoster 38

Promoting Brain Wellness & Modifying the Course of Cognitive Dysfunction and Dementia

Targeting Oxidative Stress in Multiple Sclerosis: from Basic Research to Clinical Implications

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Background: Multiple sclerosis is a chronic inflammatory condition of the central nervous system leading to demyelination and neurodegeneration. The loss of myelin leads to a multitude of neurological impairements. A wide range of clinical symptoms characterize multiple sclerosis including motor dysfunction, fatigue, tremor, nystagmus, acute paralysis, loss of coordination or balance, numbness, disturbances in speech and vision, and cognitive impairment. The mechanisms underlying the pathophysiology of multiple sclerosis are not fully delineated. Oxidative stress has been implicated in the pathogenesis of multiple sclerosis. Targeting oxidative stress in multiple sclerosis could be a potential therapeutic strategy Objective: This manuscript aimed to critically review evidence on the role of oxidative stress in the pathogenesis of multiple sclerosis. Methods: Pubmed, Scopus, Google Scholar, Science Citation Index were searched with the search terms “multiple sclerosis”, ‘oxidative stress’, “reactive oxygen species”, “antioxidants”, “pathogenesis”. The search covered the period up to and including August 2020. The main outcome was the role of oxidative stress on the pathogenesis of multiple sclerosis. Both animal and human studies met inclusion criteria Results: Evidence clearly supports the role of oxidative stress in multiple sclerosis. The central nervous system is particularly vulnerable to oxidative damage since it has a very active mitochondrial metabolism, which leads to high levels of intracellular superoxide anions. Furthermore, the brain has an abundant content of more easily peroxidizable fatty acids such as arachidonic acid. In addition, human brain has low levels of antioxidant enzymes. A number of studies in multiple sclerosis patients have shown an increase in the production of oxidative stress markers as well as reduction of antioxidant enzymes compared to healthy subjects. Studies of antioxidant intervention in patients with multiple sclerosis detect short-term improvements in markers of oxidative stress and antioxidant defenses, and to a lesser extent, in clinical symptoms (fatigue, depression). Conclusion: Evidence suggests that oxidative stress is implicate din the pathogenesis of multiple sclerosis. Targeting oxidative stress could be a potential therapeutic strategy for the management of multiple sclerosis.