09:15-09:45  Registrations

Day 1  October 02, 2017

Conference Hall: Fleming’s 9

Welcome Speech
10:15-10:45  Michael Binder
Medical Director Vienna Hospital Association, Austria

Keynote Forum
10:45-11:30  Dionisio Figueiredo Lopes, Hospital de Urgencia Otavio Lage, Brazil
Title: The use of multislice CT angiography in the surgical treatment of ruptured intracranial aneurysms

Networking and Refreshments Break 11:30-11:50 @ Foyer

11:50-12:35  Antonio Scilimati, University of Bari, Italy
Title: Neuroinflammation: Prodrome of neurological and neurodegenerative diseases

12:35-13:20  Hanan Sheikh Ibrahim, Cleveland Clinic Abu Dhabi, UAE
Title: Pseudobulbar affect, cognitive dysfunction and depression in poorly controlled diabetes

Lunch Break  13:20-14:10  @ Flemings Restaurant

Sessions: CNS Disorders | Neurosurgery
Session Chair: Dionisio Figueiredo Lopes, Hospital de Urgencia Otavio Lage, Brazil
Session Co-chair: Khin Bo Maung, Northern Lincolnshire and Goole NHS Foundation Trust, United Kingdom

14:10-14:40  Debabrata Mukhopadhyay, Kailash Group of Hospitals, India
Title: Challenges in awake craniotomy for intrinsic brain tumors in eloquent cortex

14:40-15:10  Brahim Gargouri, University of Freiburg Medical School, Germany
Title: Pyrethroid bifenthrin induces neuronal damage, cognitive impairment associated with oxidative damage in rat’s hippocampus: Possible involvement of Nurr1/Nrf2 and nf-κb pathways

15:10-15:40  Ameenah Sorefan, Alzheimer Association, Mauritius
Title: Dementia in Mauritius- Starting an early dementia diagnosis clinic

Session Discussion
Networking and Refreshments Break 15:40-16:00 @ Foyer

Sessions: Cognitive Neurology | Case Reports
Session Chair: Antonio Scilimati, University of Bari, Italy
Session Co-chair: Hanan Sheikh Ibrahim, Cleveland Clinic Abu Dhabi, UAE

16:00-16:30  Melnyk Nataliia O, O O Bogomolets National Medical University, Ukraine
Title: Reactive cuprizone-induced changes in neurons of central nervous system, behavioral reactions and its recovering after influence of leukemia inhibitory factor in mice of different ages

16:30-17:00  Seyed Behnamedin Jameie, Iran University of Medical Sciences, Iran
Title: Neuroprotective effect of exogenous melatonin on the noradrenergic neurons of adult male rats’ locus coeruleus nucleus following REM sleep deprivation

Panel Discussion
Day 2  October 03, 2017
Conference Hall: Fleming’s 9

Keynote Forum
10:15-11:00  Title: Multiple sclerosis, corpus callosum & bedside test
Khin Bo Maung, Northern Lincolnshire and Goole NHS Foundation Trust, United Kingdom

Networking and Refreshments Break 11:00-11:20 @ Foyer

11:20-12:05  Title: Metabolism of myelin in health and pathology
Alessandro Morelli, University of Genoa, Italy

Sessions: Neurobiology of CNS | Clinical Trials in CNS
Session Chair: Khin Bo Maung, Northern Lincolnshire and Goole NHS Foundation Trust, United Kingdom
Session Co-Chair: Hanan Sheikh Ibrahim, Cleveland Clinic Abu Dhabi, UAE

Session Introduction

12:05-12:35  Title: Neuromyelitis optica: A case report
Abdel Wahhab O Gh, V N Karazin Kharkiv National University, Ukraine
Title: Effects of 660 nm low-level laser therapy on P2X3 expression of lumbar DRG of adult rats with neuropathic pain
Seyed Behnamedin Jameie, Iran University of Medical Sciences, Iran

Session Discussion
Lunch Break 13:05-14:00 @ Flemings Restaurant

Poster Presentations 14:00-15:00
Poster Judge: Hanan Sheikh Ibrahim, Cleveland Clinic Abu Dhabi, UAE
Poster Judge: Dionisio Figueiredo Lopes, Hospital de Urgencia Otavio Lage, Brazil

P1  Title: Neuromyelitis optica: A case report
Delich Olena, V N Karazin Kharkiv National University, Ukraine

P2  Title: Protective effect of histidine on para-nonylphenol enhances 1-methyl-4-phenylpyridinium ion-induced hydroxyl free radical generation in rat striatum
Toshio Obata, Osaka Aoyama University, Japan

P3  Title: ARE-binding factors TTP and HUR reveal antagonistic relationships within glioma-related gene networks
Dmitryi Chistyakov, A N Belozersky Research Institute of Physico-Chemical Biology MSU, Russia

P4  Title: Astroglial cell cultures do not produce resolvin D1 in course of LPS-induced response
Dmitryi Chistyakov, A N Belozersky Research Institute of Physico-Chemical Biology MSU, Russia

P5  Title: Benzimidazoles as epigenetic regulators of PRC2 protein complex with dual action as anti-tumor and nerve regenerators: Synthesis and molecular modeling Study
Hoda Azmy, Mansoura University, Egypt

P6  Title: Aquatic physical activity alters plasmatic BDNF levels in individuals with Parkinson’s disease
Silvia Da Silva, Centro Universitário Metodista-IPA, Brazil

P7  Title: Experiences of PTSD from traumatic incidents: A paradigm model from qualitative research
Yun Jung Choi, Chung-Ang University, South Korea

P8  Title: Glioblastoma multiforme: An advanced analysis of 153 patients and review of the literature
Mohammad Sadegh Nikdad, Tehran University of Medical Sciences, Iran

Awards & Closing Ceremony

Bookmark your dates

4th International Conference on
CENTRAL NERVOUS SYSTEM DISORDERS AND THERAPEUTICS
November 12-14, 2018 | Edinburgh, Scotland

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Biosafety
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Coastal Zone Management
Earth Science & Climatic Change
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EEE

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Engineering

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Advances in Robotics & Automation
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Applied Bioinformatics & Computational Biology
Applied Mechanical Engineering
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International Journal of Biomedical Data Mining
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International Journal of Innovative Research in Science, Engineering and Technology
International Journal of Sensor Networks and Data Communications
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Irrigation & Drainage Systems Engineering
Lasers, Optics & Photonics
Loviotics
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Molecular Imaging & Dynamics
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Steel Structures & Construction
Telecommunications System & Management
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International Journal of Advance Innovations, Thoughts & Ideas
Metrology
Research & Reviews: Journal of Botanical Sciences
Research & Reviews: Journal of Chemistry
Tomography

Genetics & Molecular Biology

Advanced Techniques in Biology & Medicine
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Advances in Molecular Diagnostics
Biochemistry & Analytical Biochemistry
Biochemistry & Molecular Biology Journal
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Biodegradable Materials
Biological Systems
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Bipolar Disorder: Open Access
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Clinical Epigenetics
Cloning & Transgenesis
Current Synthetic and Systems Biology
Cytology & Histology
Down Syndrome & Chromosome Abnormalities
Fertilization: In Vitro
Genetic Diseases
Genetics & Molecular Biology
Gene Therapy
Genetic Syndromes & Gene Therapy
Hereditary Genetics: Current Research
Human Genetics & Embryology

Insights in Cell Science
Insights in Stem Cells
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Intensive and Critical Care
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Speech Pathology & Therapy
Stem Cell Research & Therapy
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Vascular & Endothelial Biology

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Biosensors Journal
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Biophysics
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Biotechnology & Biomedical Engineering
Biotechnology & Development
Biotechnology & Human Heredity
Biotechnology & Microbial Life
Biotechnology & Molecular Biology
Biotechnology & Molecular Biology Journal
Biotechnology & Microbiology
Biotechnology & Molecular Medicine
Biotechnology & Nanotechnology
Biotechnology & Radiation Biology
Biotechnology & Surgery
Biotechnology & Therapy
Biotechnology & Tissue Engineering
Biotechnology & Vaccine Development
Biotechnology & Veterinary Sciences
Biotechnology & Water Resources
Biotechnology & Xenobiotics

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International Journal of Sensor Networks and Data Communications
International Journal of Swarm Intelligence and Evolutionary Computation
Irrigation & Drainage Systems Engineering
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Loviotics
Membrane Science & Technology
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Supporting Journals

CNS 2017
Supporting Journals

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Journal of Neurology & Neurophysiology
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Keynote Forum

Day 1

CNS 2017
The use of multislice CT angiography in the surgical treatment of ruptured intracranial aneurysms

Non-traumatic subarachnoid hemorrhage (SAH) is a neurological emergency. The main cause of non-traumatic SAH (80% of cases) is rupture of an intracranial aneurysm, an event accompanied by high morbidity and mortality rates. The incidence of aneurysmal SAH is estimated to be about 11 cases per 100,000 population per year. Extensive evidence is available demonstrating that early surgery is associated with improved outcome. Cerebral angiography (CA), computed tomography angiography (CTA) or MR angiography are commonly used to determine the location, size and shape of an aneurysm before treatment. CTA images show cerebral vessels in three-dimensional directions and can provide 3D images for aneurysm detection. Some studies have reported sensitivities ranging from 77 and 100% and specificities ranging from 79 and 100%. Among aneurysm detected on CTA and then undergoing surgery, 100% correlation was observed between CTA and CA. CTA, as less invasive and rapidly performed is an accepted method for detection and characterization of cerebral aneurysm when planning surgical intervention. Hospital de Urgências Governador Otávio Lage – HUGOL is a reference hospital for neurological emergencies such as trauma and stroke in a big city in Brazil. We proceeded 60 microsurgical clipping of ruptured intracranial aneurysm during 17 months from August 2015 to December 2016. After the clinical and image diagnoses of SAH, all the 60 patients underwent CTA examinations. The CTA study was performed with a 16-row multislice CT machine. One aneurysm (1.6%) was not detected by CTA initially and visible on the CA. 59 (98.3%) patients were successfully treated based on CTA as the only preoperative investigation. In conclusion, 16-slice CTA image is useful for the diagnosis of ruptured cerebral aneurysm as a noninvasive imaging technique providing an early diagnosis.

Biography

Dionisio Figueiredo Lopes is a Neurosurgeon Member of Brazilian Neurosurgery Society, member of Brazilian Neurosurgery Academy. He is the Head of Neurosurgery at Hospital de Urgências Governador Otávio Lage (HUGOL), a hospital reference in neurosurgical emergencies, Consultant at Hospital de Urgências de Goiânia and Hospital Santa Mônica. He is a Neurosurgeon with expertise in vascular diseases, brain tumor and traumatic brain injury. He has Fellowship in Neuro-oncology at Dresden/Germany and Fellowship in Advanced Techniques in Neurosurgery at Tubingen/Germany.

dioflopes@gmail.com
Notes:
Pseudobulbar affect, cognitive dysfunction and depression in poorly controlled diabetes

A case of unrecognized neurocognitive disorder and pseudo-bulbar affect in a patient with multiple vascular risks, poorly controlled diabetes with subcortical lacunes masquerades as depression. 64 year old ex-smoker male with PMH of hypertension, long standing poorly controlled type 2 diabetes presented with insomnia and depressive mood, where a trial of SNRI was partially effective in his mood control but did not help with his crying bursts. He is still driving with multiple episodes of loss of consciousness due to hypoglycemia. His physical exam was unremarkable except for emotional bursts of laughter and crying that were not affect congruent. On CGA, he was found to have 3 impaired IADL domains (ability to drive with many car accidents, handling finances and administering medication. His cognition test showed MMSE : 26/30, adjusted to education level, he had impaired clock drawing test and impaired trail B test, impaired speed, attention and executive skills, GDS was 4/15, FRAIL scale was 4/5. His labs revealed HbA1c above 10, He has normal B12, folate. His MRI revealed white matter disease, pontine infarct, Left thalamic lacunar infarct and left lenticular lacune as well in addition to cortical atrophy. Patient was recognized as an early vascular Dementia case with associated Pseudo bulbar Affect masked by depressive symptoms, the case triggered a change of his holistic care that revamped his HbA1C goals and advanced care planning. In summary, General psychiatrists and Primary care clinicians may fail to recognize pseudo bulbar affect and cognitive dysfunction during clinic visits using routine history and physical Pseudobulbar Affect (PBA), presents as abrupt episodes of uncontrollable laughter or crying that are incongruent or independent of mood, occurs in many neurological brain diseases or following brain injury. It is important to identify PBA as a different entity from depression, treat and identify underlying vascular cognitive impairment.

Biography

Hanan Sheikh Ibrahim is a Clinical Assistant Professor at the Cleveland Clinic Lerner College of Medicine of Case Western Reserve University, Ohio, a Consultant Physician and a Quality Officer at the Cleveland Clinic Abu Dhabi. She was trained at Cleveland Clinic in Ohio, USA under the tutelage of Dr. Robert Palmer, Concept Originator of the Acute Care of Elderly (ACE) unit which was modeled internationally. Then she pioneered in the geriatric care in the UAE by establishing the first MACE unit and the first Geriatric Core Curriculum for resident physicians in training. She received her MD from Damascus University, Syria where she specialized in Pulmonary Medicine then she moved to US where she completed her residency in Internal Medicine at the University of Pittsburgh School of Medicine in Pittsburgh, Pennsylvania, US. She completed her Fellowship in Geriatric Medicine at Cleveland Clinic, Ohio. She is board certified in Internal & Geriatrics Medicine.

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3rd International Conference on

CENTRAL NERVOUS SYSTEM DISORDERS AND THERAPEUTICS

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Scientific Tracks & Abstracts

Day 1

CNS 2017
Session Introduction

Title: **Challenges in awake craniotomy for intrinsic brain tumors in eloquent cortex**
   Debabrata Mukhopadhyay, Kailash Group of Hospitals, India

Title: **Pyrethroid bifenthrin induces neuronal damage, cognitive impairment associated with oxidative damage in rat’s hippocampus: Possible involvement of Nurr1/Nrf2 and nf-kb pathways**
   Brahim Gargouri, University of Freiburg Medical School, Germany
Challenges in awake craniotomy for intrinsic brain tumors in eloquent cortex

Debabrata Mukhopadhyay and Anil Gurnani
Kailash Group of Hospitals, India

Introduction: Surgical treatment of brain tumors in the eloquent areas has high risk of eloquent impairment. These tumors represent a unique challenge as most of the patients have a higher risk of treatment related complications. Awake craniotomy is a useful surgical approach to help to identify and preserve functional areas in the brain and maximizes tumor removal and minimizes complications.

Methods: Selected patients admitted with intrinsic brain tumor between from July, 2011 to August, 2016 in the eloquent area of brain like speech or motor area were chosen for awake craniotomy. A retrospective analysis was done. A preoperative assessment was also done. These patients were presented with seizure and/or progressive neurological deficit like speech or motor. A standard anaesthesia monitoring was done during surgery. Long acting local anaesthesia (Bupivacaine) was used for scalp block. The surgeries were performed in a state of sleep-awake-sleep pattern, keeping the patients fully awake during tumor removal. Propofol and Fentanyl was used as anaesthetic agents which was completely withdrawn prior to tumor removal. The speech and motor functions were closely monitored clinically by verbal commands during tumor resection. No brain mapping was performed due to lack of resources. All patients underwent non-contrast computed tomogram head in the first post-operative day.

Results: A total of 35 patients were included in the study. The oldest patient was 55 years and youngest being 24 years (mean 36 years). Twenty (57.14%) were females and 15 (42.85%) males. Twenty (57.14%) patients were presented with predominantly seizure disorders and rest with progressive neurological deficit like speech or motor. Thirty (85.71%) patients were discharged on second postoperative day. Complications was encountered in 4 (11.42%) patients who developed brain swelling intraoperatively and 5 (14.28%) deteriorated neurologically in the immediate postoperative period however managed successfully and discharged in a week's time. Five (14.28%) patients require ICU/HDU care for different reasons. There was no mortality during the hospital stay. Histopathology revealed 25 (71.42%) patients had low grade glioma, 8 (22.85%) had high grade glioma and 2 (5.71%) had metastases.

Conclusion: Awake craniotomy is a safe surgical management for intrinsic brain tumors in the eloquent cortex although surgery and anesthesia is a challenge. It offers great advantage towards disease outcome. However long follow up and more studies are required.

Biography

Debabrata Mukhopadhyay is currently working as a Neurosurgeon in the Department of Neurosurgery at Kailash Group of Hospitals, India. He has published several articles in the reputed and peer reviewed journals and participated in several scientific events.

neurodoc07@gmail.com
Pyrethroid bifenthrin induces neuronal damage, cognitive impairment associated with oxidative damage in rat's hippocampus: Possible involvement of Nurr1/Nrf2 and NFkB pathways

Brahim Gargouri1, Yassine Chtourou1, Michèle Bouchard2, Abdelmajid Khabir3, Bernd L Fiebich3 and Hamadi Fetoui1

1University of Sfax, Tunisia
2Université de Montréal, Canada
3Habib Bourguiba Hospital, Tunisia
4University of Freiburg Medical School, Germany

Substantial evidence has shown that exposure to pyrethroid pesticides may cause adverse neurodevelopmental outcomes and cognitive impairment, but the underlying neurobiological mechanism is poorly understood so far. In this study, we investigated the alterations of neuronal damage, glial activation oxidative stress and cholinergic dysfunction, and their causal relationship with the cognitive deficits induced by bifenthrin. Our results revealed that exposure to bifenthrin for 8 weeks at doses 6 and 21 mg/kgbw leads to reduction in the levels of acetyl-cholinesterase, Na+/K+, Ca2+, Mg2+ ATPases, enzymatic and non-enzymatic antioxidants activities in the hippocampus region. Further, in hippocampus tissue, bifenthrin significantly enhance the mRNA gene expression of nuclear receptor related 1 protein (nurr1), nuclear factor erythroid 2 (nrf2) and nuclear factorkB pathway (NFkB). Oxidative/nitrosative stress was evident in bifenthrin-treated groups by increased malondialdehyde (MDA), protein carbonyls (PCO), and nitrite concentration (NO) in hippocampus. Further, we found that treated rats with bifenthrin exhibited spatial learning and memory impairments and working memory dysfunction compared with control rats. This is also supported by histopathological findings of hippocampus region of rats. Correlational analyses revealed that spatial learning and memory impairments and working memory dysfunction were significantly correlated with the measures of neuronal damage, cholinergic dysfunction and oxidative damage in the hippocampus of treated rats. Moreover, the measures of neuronal damage and central cholinergic dysfunction were significantly correlated with the indexes of oxidative damage in treated rats. The results of the present study suggest that neuronal damage, cholinergic dysfunction and oxidative damage in the hippocampus following bifenthrin exposure could be involved in cognitive deficits.

Biography

Brahim Gargouri is currently working in the Laboratory of Toxicology-Microbiology and Environmental Health at University of Sfax, Tunisia. He has published several original research papers in the reputed and peer reviewed journals.

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Notes:
Session Introduction

Title: Reactive cuprizone-induced changes in neurons of central nervous system, behavioral reactions and its recovering after influence of leukemia inhibitory factor in mice of different ages

Melnyk Nataliia O, O O Bogomolets National Medical University, Ukraine

Title: Neuroprotective effect of exogenous melatonin on the noradrenergic neurons of adult male rats' locus coeruleus nucleus following REM sleep deprivation

Seyed Behnamedin Jameie, Iran University of Medical Sciences, Iran
Reactive cuprizone-induced changes in neurons of central nervous system, behavioral reactions and its recovering after influence of leukemia inhibitory factor in mice of different ages

Melnyk Nataliia O1,2, Labunets I F1, Rodnichenko A E1, Rymar S E1 and Utko N A1
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2 O O Bogomolets National Medical University, Ukraine

Statement of the Problem: Investigation work was aimed at studying the features of neuroprotective effects of recombinant human leukemia inhibitory factor (rhLIF) on mice of different ages with cuprizone model of demyelination.

Methodology & Theoretical Orientation: In 129/Sv mice at 3-5 and 16-17 months of age, after staining histological sections of brain and spinal cord toluidin blue, were determined the percentage of neurons with unmodified, moderate and severe structural changes. Motor and emotional activity in "open field" test, activity of brain antioxidant enzymes and macrophages capable to phagocytosis of latex beads were assessed. Cuprizone was fed daily for 3 weeks. RhLIF injected after 7 days cuprizone diet, daily, 50 µg/kg. In cuprizone-treated mice of both age groups, increase in the brain and spinal cord proportions neurons with severe changes was observed.

Findings: In young animals, which received cuprizone and rhLIF reduces the amount of neurons with destructive changes. Such changes under influence of rhLIF are slowly observed in older mice. Cuprizone decreases the amount of crossed squares and faecal boluses in mice of both age groups. Inhibition amount and activity of macrophages after injections of the rhLIF presents only for older mice. LIF may be perspective neuroprotective drug in multiple sclerosis. The injections of rhLIF restore emotional activity in these mice, but the increase in motor activity is observed only in young mice. In brain of cuprizone-treated mice of different ages inhibited the activity of catalase and glutathione peroxidase (GP); changes were more pronounced in older mice. The positive effect of rhLIF on GP activity appears only in young mice. Percentage of active macrophages increases in cuprizone-treated mice of both age groups, but their activity is only in 16-17 month-old mice.

Biography
Melnyk Nataliia O was investigating demyelination and remyelination process in central nerve system in experiment animals (in rats and mice). She studied structural changes in organs central and peripheral immune system in demyelination condition. She has 262 scientific works (4 patents) and is working in National O O Bogomolets Medical University where she provides lecture courses in Histology, Cytology and Embryology. She has prepared and edited textbook, “Histology, Cytology, Embryology”.

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Notes:
Neuroprotective effect of exogenous melatonin on the noradrenergic neurons of adult male rats’ locus coeruleus nucleus following REM sleep deprivation

Seyed Behnamedin Jameie¹, Somaye Mesgar¹, Abbas Aliaghaei², Fariba Khodagholi², Samira Danyali², Mohammad Amin Abdollahifar³, Marziyeh Sorousa³ and Yousef Sadeghi²

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²Shahid Beheshti University of Medical Sciences, Iran

Background: Melatonin primarily secreted by the pineal gland in dark phase of the circadian rhythm. In addition to its role as an internal sleep facilitator, melatonin acts as an antioxidant, anti-inflammatory and neuroprotective agents. Recently, melatonin has been introduced as a therapeutic strategy for sleep disorders. Hence, in the present study, we studied the neuroprotective effects of pre- and post-treatment of melatonin in locus coeruleus nucleus (LC) of rapid eye movement (REM) sleep deprived (REM-SD) male adult rats.

Methodology: Adult male rats of control, sham and trial groups were used in this study. By using flower-pot technique, short term REMSD was induced. Exogenous melatonin (ExMe) was used intraperitoneally in two forms of pre and post treatment. The protein level of cleaved caspase-3, number and density of tyrosine hydroxylase (TH) positive neurons and microglia population in LC was studied by Western blot and immunohistochemistry respectively. Morphological changes of LC nucleus and its neurons were also studied by using stereological analysis.

Results: The number of neurons and volume of LC was reserved in animals received post-RSD ExMe, apoptosis significantly was decreased comparing to RSD and Pre-RSD animals. Melatonin post-treatment of RSD rats also decreased cleavage of caspase-3 and increased reduced glutathione content in LC. Moreover, immunohistochemistry analysis revealed the increase number of TH positive neurons and decrease microglia migration.

Conclusion: Based on our findings, antioxidant properties of exogenous melatonin could play a critical role in certain type of sleep disorders.

Biography

Seyed Behnamedin Jameie is currently working as a Professor in the Neuroscience Research Center (NRC) at Iran University of Medical Sciences, Tehran, Iran. He has published several original research papers in the reputed & peer reviewed journals and also participated into several scientific meetings.

Notes:
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Day 2

CNS 2017
Multiple sclerosis, corpus callosum & bedside test

Demyelination affects highly myelinated structures like Corpus Callosum (CC). CC is unique in function that it connects right and left hemisphere. It synchronises bimanual or bipedal activities. Affecting CC can disturb synchrony between the two hemispheres and will affect bimanual and bipedal tasks. The aim is to see if speed of clapping (bimanual activity) can reflect the involvement of CC in multiple sclerosis. Consecutive 70 multiple sclerosis patients from outpatient clinics and home visits were tested for bimanual hand function (clapping). Exclusion criteria are upper limb power <3/5 MRC scale, pain, visual impairment, intentional tremors, stroke or cognitive impairment. Study period started from 01-09-2016. Comparison of speed between rapid supination/pronation of left and right hand separately and then clapping of both hands (supination/pronation of each hands alternatively). Patients had to do as fast as they could. Noticeable slowing of clapping compared to single hand supination/pronation was taken as a sign slowing down of conduction through CC. 31 patients were excluded, 34 patients showed no noticeable difference, 2 patients were difficult to make conclusions and 3 patients showed definite slowing down in clapping. Positive patients will have difficulties in doing bimanual activities like using two sticks for mobility, typing using keyboard, pushing wheel chair bimanually, etc. It is possible to detect CC involvement by doing above bedside test and can be used in rehabilitation setting. Sample size is not large enough and larger studies are needed to validate the finding.

Biography

Khin Bo Maung is involved in Neurorehabilitation over 20 years. He is also a Lecturer (Hon) in Hull and York Medical School teaching 4th Year Medical Students in CNS and Musculoskeletal Blocks. He is doing Botulinum Toxin injection in Spasticity, Dystonia and Involuntary Movement disorders over 15 years. He has given poster and oral presentations in international neurorehabilitation conferences. He is also involved in using Functional Electrical Stimulation (FES) over 10 years and presented regularly in International FES Conferences. He is working on developing Hypertonic Hand Monitoring Scale.

Notes:
Metabolism of myelin in health and pathology

Myelin is a site of active aerobic energy metabolism, producing ATP through the oxidative phosphorylation (OXPHOS) machinery, which contributes to the acceleration of nervous impulse. This innovative view simplifies current ideas about the physical chemical mechanisms that ensures the advancement of the action potential (CAP) as such basic mechanisms are unchanged in the passage of the CAP from the non-myelinated to myelinated axon. The ATP produced in myelin sheath is transferred to the axon through the Gap Junctions, which are abundant in myelin sheath. The OXPHOS proteins expresses in myelin is closely related to that of mitochondria and hence there must be some process still to be defined, which guarantees the transfer of OXPHOS machinery from mitochondria to myelin; overall the mitochondria-myelin link is known since many mitochondrial pathologies primarily affect myelin. For perfect functioning, OXPHOS requires an active synthesis of the heme group, considering that it is a fundamental component of several subunits of respiratory complexes, and interestingly, myelin sheath displays a higher heme group synthesis in comparison with other districts. In particular, proper functioning of myelin is closely linked to an efficient biosynthetic pathway of the heme and the crucial passage is catalyzed by the enzyme ALA dehydratase (EC 4.2.1.24) that requires zinc as cofactor. Lead poisoning (Saturnism) results in an imbalance of this enzyme and myelin degeneration. Moreover, analyzing the OXPHOS metabolism in myelin isolated from autopsy specimens of multiple sclerosis (MS) patients, we have observed a defective energy/respiratory capacity. With this knowledge, the hypothesis that MS is not an autoimmune disease, but a disease triggered by myelin degeneration following a malfunction of some process related to its energy function and heavy metal pollution seems confirmed, also considering the historical link between industrialization and the MS onset.

Biography

Alessandro M Morelli carried out research in varied fields of biology, focusing in those areas most directly linked to medicine. He investigated on the enzyme Glucose-6-P-dehydrogenase and on its molecular mechanism of senescence. He has been working in the phototransduction molecular events in photoreceptor cells of vertebrate retina. He has discovered the protein FX, a NADP dependent enzyme, catalyzing synthesis of GDP-L-fucose. He has been working on the effects of electromagnetic fields of extremely low frequency on the activity of enzymes involved in phototransduction in retinal cells of vertebrates. Moreover, he has put in evidence the reversible effects of electromagnetic fields on lipid-linked enzymes such as acetylcholinesterase of retinal synaptosomes. Recently, with Isabella Panioli, Silvia Ravera, Daniela Calzia, he has discovered the brain myelin energetic function and the ATP extramitochondrial synthesis operating in it, involving new paradigms for neurobiology, with application in the study of multiple sclerosis and other neurodegenerative diseases.

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Scientific Tracks & Abstracts

Day 2

CNS 2017
Session Introduction

Title: Neuromyelitis optica: A case report
  Abdel Wahhab O Gh, V N Karazin Kharkiv National University, Ukraine

Title: Effects of 660nm low-level laser therapy on P2X3 expression of lumbar DRG of adult male rats with neuropathic pain
  Seyed Behnamedin Jameie, Iran University of Medical Sciences, Iran
Neuromyelitis optica: A case report

Delich O B¹, Abdel Wahhab O Gh¹, Merkulova O Yu¹, Pasyura I N² and Shevchenko L M²

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A 54-year-old woman was treated initially with neuritis of right optic nerve in 2013 that resolved completely. Two years later, she presented with relapse and partial visual functions recovery of right optic nerve. Ophthalmoscopy analysis showed atrophic changes of the disc optic nerve. After undercooling in March 2016, a patient complained of interscapular pain, weakness of the right limbs and urinary retention. MRI of thoracic part of spinal cord showed high T2 signal spread at least more than three vertebral segments, osteochondrosis. CT-angiography of spinal cord showed an absence of vascular malformations. A patient was treated with dexamethasone, ceftriaxone, vascular and metabolic therapy and was discharged with partial recovery, but interscapular pain was still present, sensory impairments from Th6 level down, urinary retention and constipation were revealed. From June 2016, she developed the lower spastic paraplegia. MRI of the brain (2015) didn't reveal any local change of the brain tissues, besides asymmetric hydrocephalus of the lateral ventricles. Biochemical analysis serum antibodies IgG, the specific markers of neuromyelitis optica (NMO), connected with aquaporin -4-(AQP4) usually led to increase of AQP4 concentration, which was 1:320 in our case. Course treatment included solumedrol, aciclovir, ceraxon, actovegin, and cytostatic drugs. The patient was discharged with certain improvements and diagnosed with NMO, partial atrophy of the disc right optic nerve, lower paraplegia, reduced sensitivity in trunk and right lower limb, pelvic sphincter disturbances. From June 2016, she developed the lower spastic paraplegia. MRI of the brain (2015) didn't reveal any local change of the brain tissues, besides asymmetric hydrocephalus of the lateral ventricles. Biochemical analysis serum antibodies IgG, the specific markers of neuromyelitis optica (NMO), connected with aquaporin -4-(AQP4) usually led to increase of AQP4 concentration, which was 1:320 in our case. Course treatment included solumedrol, aciclovir, ceraxon, actovegin, and cytostatic drugs. The patient was discharged with certain improvements and diagnosed with NMO, partial atrophy of the disc right optic nerve, lower paraplegia, reduced sensitivity in trunk and right lower limb, pelvic sphincter disturbances. From September 2016, she started to use copaxone (40 mg/ml 3 times a week s.c. for 4 months). Pain in upper thoracic, cervical parts of vertebra with irradiation to the occipital region increased after respiratory infection in December 2016. The numbness spread to Th4-Th5 segments, appear the clinic of lower paraplegia. She got plasmapheresis as an out-patient one time. Every month from September 2016 to March 2017, infusion of methylprednisolone was performed (1000 mg), rituximab (375 mg/kg i.v. infusion every 10 days №4 from February to March). So, clinical diagnosis now is Neuromyelitis optica (Devic’s disease), remitting course, exacerbation, lower spastic paraplegia, pelvic sphincter disturbances by type of urinary retention and constipation, of the right disc optic nerve atrophy.

Biography

DELICH Olena: Currently a student and head of scientific society of the medical faculty of V.N. Karazin Kharkiv National University, Ukraine.
ABDEL WAHHAB O GH: Currently he is a student of the medical faculty of V.N. Karazin Kharkiv National University, Ukraine.
Effects of 660 nm low-level laser therapy on P2X3 expression of lumbar DRG of adult male rats with neuropathic pain

Seyed Behnamedin Jameie1, Sojad Hassanzadeh1, Mahdieh Kerdari1, Ghazal Hamid Behnam2, Mona Forhadi1,3, Masumeh Masoumipoor1, Manasadat Jameie1,4 and Melikasadat Jameie1,4

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4Shahid Beheshti University of Medical Sciences, Iran

Background: Neuropathic Pain (NP) is a serious suffering medical condition that frequently leads to disability and life style changes. Although the exact mechanisms of NP are still unknown, recently the role of reactive oxygen species (ROS) reported as an important factor for NP. Apoptosis, increase of ATP production and reduction of antioxidants are also the other factors influencing NP. There are certain therapeutic procedures for NP, among them using laser therapy newly received more attention. In the present research, we studied the molecular effects of Low Level Laser Therapy (LLLT) on a rat model of NP.

Material & Methods: Thirty adult male Wistar rats (200-250 g) that are randomly divided into three groups including chronic constriction injury (CCI), CCI+LLLT and control were used in this study. CCI technique was used to induce NP. Laser therapy was done by using laser beam of 660 for 14 days following CCI. After that, expression of P2X3 of the DRG, Bax and Bcl2 in lumbar spinal segments measured by Western blotting. Level of glutathione (GSH) was also measured in lumbar spinal cord segments by Continuous Spectrophotometric Rate Determination method. For behavioral study, the mechanical and thermal hyperalgesia were evaluated in days 7 and 14 after CCI.

Results: LLLT for two weeks increased expression of Bcl2 and GSH, whereas decreased Bax and P2X3 expression significantly. Comparing the results of behavioral study showed significant differences in the mechanical and thermal threshold showed between CCI and CCI+ LLLT groups.

Conclusion: Based on our findings, the therapeutic effects of LLLT for NP act throughout cellular and molecular mechanisms which improve mitochondrial function that in turn improve cell function and prevent apoptosis.

Biography
Seyed Behnamedin Jameie is currently working as a Professor in the Neuroscience Research Center (NRC) at Iran University of Medical Sciences, Tehran, Iran. He has published several original research papers in the reputed & peer reviewed journals and also participated into several scientific meetings.

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Posters
Neuromyelitis optica: A case report

Delich O B, Abdel Wahhab O Gh, Merkulova O Yu, Pasyura I N and Shevchenko L M

1 V N Karazin Kharkiv National University, Ukraine
2 Kharkiv Railway Clinical Hospital No1 of Branch "Health Center" of the Public joint stock company "Ukrainian Railway"

A 54-year-old woman was treated initially with neuritis of right optic nerve in 2013 that resolved completely. Two years later, she presented with relapse and partial visual functions recovery of right optic nerve. Ophthalmoscopy analysis showed atrophic changes of the disc optic nerve. After undercooling in March 2016, a patient complained of interscapular pain, weakness of the right limbs and urinary retention. MRI of thoracic part of spinal cord showed high T2 signal spread at least more than three vertebral segments, osteochondrosis. CT-angiography of spinal cord showed an absence of vascular malformations. A patient was treated with dexamethasone, ceftriaxone, vascular and metabolic therapy and was discharged with partial recovery, but interscapular pain was still present, sensory impairments from Th6 level down, urinary retention and constipation were revealed. From June 2016, she developed the lower spastic paraplegia. MRI of the brain (2015) didn't reveal any local change of the brain tissues, besides asymmetric hydrocephalus of the lateral ventricles. Biochemical analysis serum antibodies IgG, the specific markers of neuromyelitis optica (NMO), connected with aquaporin -4-(AQP4) usually led to increase of AQP4 concentration, which was 1:320 in our case. Course treatment included solumedrol, aciclovir, ceraxon, actovegin, and cytostatic drugs. The patient was discharged with certain improvements and diagnosed with NMO, partial atrophy of the disc right optic nerve, lower paraplegia, reduced sensitivity in trunk and right lower limb, pelvic sphincter disturbances. From September 2016, the patient started to use copaxone (40 mg/ml 3 times a week s.c. for 4 months). Pain in upper thoracic, cervical parts of vertebrae with irradiation to the occipital region increased after respiratory infection in December 2016. The numbness spread to Th4-Th5 segments, appear the clinic of lower paraplegia. She got plasmapheresis as an out-patient one time. Every month from September 2016 to March 2017, infusion of methylprednisolone was performed (1000 mg), rituximab (375 mg/kg i.v. infusion every 10 days №4 from February to March). So, clinical diagnosis now is Neuromyelitis optica (Devic’s disease), remitting course, exacerbation, lower spastic paraplegia, pelvic sphincter disturbances by type of urinary retention and constipation, of the right disc optic nerve atrophy.

Biography

DELICH Olena: Currently a student and head of scientific society of the medical faculty of V.N. Karazin Kharkiv National University, Ukraine. ABDEL WAHHAB O GH: Currently he is a student of the medical faculty of V.N. Karazin Kharkiv National University, Ukraine. MERKULOVA Olga: Currently working as an associate professor in the department of neurology at V.N. Karazin Kharkiv National University, Ukraine. PASYURA Igor: Currently working as a head of department of Neurology at Kharkiv Railway Clinical Hospital of №1 Branch "Health Center" of the Public joint stock company "Ukrainian Railway", Ukraine. SHEVCHENKO L M: Currently working as a doctor in the department of Neurology at Kharkiv Railway Clinical Hospital of №1 Branch "Health Center" of the Public joint stock company "Ukrainian Railway", Ukraine.

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Protective effect of histidine on para-nonylphenol enhances 1-methyl-4-phenylpyridinium ion-induced hydroxyl free radical generation in rat striatum

Toshio Obata
Osaka Aoyama University, Japan

The present study examined the antioxidant effect of histidine, a singlet oxygen (1O2) scavenger, on para-nonylphenol, an environmental estrogen-like chemical, enhances 1-methyl-4-phenylpyridinium ion (MPP+)–induced hydroxyl radical (•OH) generation in extracellular fluid of rat striatum. Rats were anesthetized, and sodium salicylate in Ringer’s solution (0.5 nmol/μl/min) was infused through a microdialysis probe to detect the generation of •OH as reflected by the non-enzymatic formation of 2,3-dihydroxybenzoic acid (DHBA) in the striatum. Induction of para-nonylphenol (10 μM) significantly enhanced MPP+-induced •OH generation. However, histidine (25 mM) decreased the para-nonylphenol-induced •OH formation. Although the level of MPP+-induced •OH formation trapped as DHBA after para-nonylphenol treatment increased, para-nonylphenol failed to increase either the level of dopamine (DA) and DHBA formation in the reserpinized animals. When iron (II) was administered to para-nonylphenol (10 μM)-pretreated rats, iron (II) clearly produced a dose-dependent increase in •OH formation, compared with MPP+-only treated animals, that showed a positive linear correlation between iron (II) and DHBA (R2=0.983) in the dialysate. However, in the presence of histidine (25 mM), small increase in the level of DHBA products were observed. These results indicate that para-nonylphenol enhanced •OH generation on 1O2 production, and histidine may have preventive effect on para-nonylphenol and MPP+-induced •OH generation in rat striatum.

Biography
Toshio Obata is currently working as a Professor in the Department of Nursing at Faculty of Health Sciences, Osaka Aoyama University, Japan. He has published several original research papers in the reputed journals and also participated into several scientific meetings.

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Notes:
ARE-binding factors TTP and HUR reveal antagonistic relationships within glioma-related gene networks

Dmitry V Chistyakov and Alina A Astakhova
A N Belozersky Research Institute of Physico-Chemical Biology - MSU, Russia

Statement of the Problem: Glioma is a heterogeneous group of primary brain tumors with poor prognoses. Effective treatment strategies for glioma have yet to be developed. This requires better understanding of molecular mechanisms involved into pathology, determining promising drug targets. Recently, it has become clear that mRNA stability regulation by Adenine-Uridine Rich Element (ARE) binding factors plays crucial role in cancer biology. This study aimed to unravel participation of ARE-binding factors TTP, HUR, BRF1, BRF2, KSRP, AUF1 in gene networks of gliomas.

Methodology & Theoretical Orientation: Data on gene expression in 276 glioma samples and 8 samples of healthy brain tissue were downloaded from Gene Omnibus (ID: GSE16011). Differential expression of the ARE-binding factors; correlations in expression patterns of the ARE-factors and other genes; biological functions of genes with associated expression patterns were analyzed.

Conclusion & Significance: Among ARE-binding factors TTP, BRF1, BRF2, KSRP but not HUR were upregulated at an mRNA level in brain tumors. Genes, correlating with ARE-factors by their expression patterns, split into two large subgroups: those that positively correlated with TTP and negatively correlated with HUR and vice versa. Interestingly, TTP-positive/HUR-negative genes were enriched in innate immune response mediators and TTP-negative/HUR-positive genes were enriched in RNA binding factors. Moreover, TTP associated genes were previously recognized as glioma survival prognosis markers. The obtained data demonstrate that ARE-binding factors are involved into glioma biology on a transcriptome level, reveal antagonistic relations between TTP and HUR and indicate that an ARE-mediated mRNA stability control pathway represents a promising target for tumor treatment strategies development. The research was supported by RFBR grant №16-34-01085 mol_a.

Biography
Dmitry V Chistyakov has his expertise in cell signaling pathways controlling inflammatory responses within the central nervous system. His scientific interests concern mechanisms that control innate immune response within nervous tissue on cellular level. Investigations within this field have brought him to the question about mRNA stability control during inflammatory response on the level of cells. These two processes – activation of innate immune response and post-transcriptional gene expression regulation turned out to be crucial for glioma development as well. Understanding their interaction during tumor development is one of the attractive directions of his research development.

Notes:
Astroglial cell cultures do not produce resolvin D1 in course of LPS-induced response

Dmitryi V Chistyakov¹, Alina A Astakhova¹, Marina G Sergeeva¹ and Nadezda V Azbukina²
¹A N Belozersky Research Institute of Physico-Chemical Biology - MSU, Russia
²Moscow State University, Russia

Statement of the Problem: Neuroinflammation is an innate immune response that accompanies numerous disorders within CNS, including neurodegenerative conditions like Alzheimer's disease, Parkinson's disease, stroke, cancer and others. In spite of its primary protective role, neuroinflammation often takes form of a chronic process and represents a damaging factor, exacerbating nervous tissue injury. Mechanisms that could switch immune response within CNS to physiological termination, referred to as resolution, are of high scientific and practical interest. In the current study, we have analyzed release of Resolvin D1 (RvD1) – a major lipid mediator of inflammation resolution derived upon enzymatic processing of docosahexaenoic acid (DHA) in mixed cultures of primary astrocytes within an in vitro model of neuroinflammation.

Methodology & Theoretical Orientation: Primary cultures of glial cells enriched in astrocytes were obtained from brains of neonatal Wistar rat pups by conventional procedures. Cells were stimulated with LPS for 4 and 24 hours. Cell lysates and cell free culture media were used to analyze COX-2 and TNFα expression as well as PGE2 release. RvD1 and DHA concentrations in cell-free culture media were analyzed by ELISA and HPLC/MS.

Conclusion & Significance: Glial cultures responded to LPS stimulation with upregulation of COX-2 and TNFα on both mRNA and protein levels and increase in DHA and PGE2 production. At the same time RvD1 was not detected in cell lysates or cell free culture media. The obtained results indicate that astrocytes are not directly involved into resolvin production upon LPS stimulation and have a unique role in the process of inflammatory response resolution within CNS.

Biography

Dmitryi V Chistyakov has his expertise in cell signaling pathways controlling inflammatory responses within the central nervous system. His scientific interests concern mechanisms that control innate immune response within nervous tissue on cellular level. Investigations within this field have brought him to the question about mRNA stability control during inflammatory response on the level of cells. These two processes – activation of innate immune response and post-transcriptional gene expression regulation turned out to be crucial for glioma development as well. Understanding their interaction during tumor development is one of the attractive directions of his research development.

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Notes:
Benzimidazoles as epigenetic regulators of PRC2 protein complex with dual action as anti-tumor and nerve regenerators: Synthesis and molecular modeling Study

Hoda Azmy, Ghada S Hassan, Shahenda M El-Messery and Ebrahim R Hamza
Mansoura University, Egypt

Polycomb repressive complex 2 (PRC2) has been shown to play a major role in transcriptional silencing in part by installing methylation marks on lysine 27 of histone 3. Dysregulation of PRC2 function correlates with certain malignancies and also correlates with the nerve regeneration process. EZH2 is the catalytic engine of the PRC2 complex responsible for the methylation process and is overexpressed in many malignancies thus represents a key candidate oncology target. It also plays a role in the regulation of action of EED subunit of PRC2 which in turn plays an important role in the nerve regeneration process after injury. So, here in we try to regulate the expression of EZH2 using benzimidazole selective inhibitors resulting in a dual action helping in treatment of many malignancies and the nerve regeneration process. Some benzimidazole derivatives have been synthesized and structure elucidation was assured using NMR technique. 3-((1H-Benzo[d]imidazol-2-yl)amino)-2-(4-nitrophenyl)-1,3-thiazepan-4-one has bound to the target enzyme via Cys A663, Tyr A111, Tyr A111 and Tyr A661 aminoacid residue. In vitro studies are now in process hoping to treat specially the breast, prostate, liver and leukemia tumors and regenerate neuronal cells after application of stress media. In vivo studies will be operated soon.

Biography
Shahenda M. El-Messery is associate professor of pharmaceutical organic chemistry at faculty of pharmacy, Mansoura University. Her research interest focuses on the design, synthesis, and evaluation of biologically active small molecules with getting the advantage of the molecular modeling modern techniques. She published scientific papers in peer reviewed journals.

Notes:
Aquatic physical activity alters plasmatic BDNF levels in individuals with Parkinson's disease

Silvia da Silva, Ivy Reichert Vital da Silva, João José Cunha, Pâmela Krause Peccín, Bernardo Vieira, Vera Lucia Widniczck Striebel, Simone Rizzo Nique da Silva Peralles, Daniela Pochmann and Viviane Rostirola Elsner
Centro Universitário Metodista-IPA, Brazil

Increasing evidence suggests that physical exercise have beneficial effects on the progression of Parkinson's disease (PD), alleviating the main symptoms and improving the performance in activities of daily living. Moreover, it was reported recently that different exercise protocols are able to increase the peripheral levels of brain-derived neurotrophic factor (BDNF), improving the health status of PD individuals. In this sense, aquatic physical exercise emerges as a proposal able of improving the quality of life of individuals with PD, since besides the physical activity by itself, the heated water eventually produces a feeling of relaxation, lightness and well-being during the performance of physical activities. However, the molecular mechanisms related to these beneficial outcomes are not elucidated. Thus, we investigated the short and long term effects of a program of aquatic physical activity on plasmatic BDNF levels of individuals with PD. The physical activity protocol was carried out during a month with two sessions per week (1 hour/session). Temporal BDNF levels were evaluated in plasma at different times: pre-intervention, immediately after the first session, 48 hours after the first session and one month after the first session. The data of all individuals analyzed together (n=9) demonstrated a significant decrease in BDNF levels 48 h after pre-intervention period (p<0.05). When data were divided by gender, we observed a significant decrease in BDNF levels evaluated at 48 h in comparison with the time immediately after the first session for both gender (p<0.05). In addition, our results showed that BDNF levels observed in female group (n=4) were significantly increased when compared to male group (n=5) at the time point of 48 h (p<0.05). No significant changes were observed on BDNF levels immediately after the first session (acute) or after one month (chronic). Our results demonstrated that the program of aquatic physical activity altered plasmatic BDNF levels in a time dependent and gender specific manner.

Biography

Silvia da Silva is currently pursuing her Post Graduation in the field of Biosciences and Rehabilitation at Centro Universitário Metodista-IPA, Porto Alegre, RS, Brazil.

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Notes:
Experiences of PTSD from traumatic incidents: A paradigm model from qualitative research

Yun Jung Choi
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Statement of the Problem: Koreans have experienced catastrophic disasters, which are continually growing for decades. Many people have died from shopping center breakdown, subway fire, ferry submerge, and so on. National psychological support system has been initiated for people who are affected from disaster, right after the subway fire disaster in Daegu in 2003. Even though many programs and educations had been provided for disaster psychological support over 10 years, the Sewol ferry accident in 2014 gave recognition that effective infrastructures for disaster psychological support were still deficient in Korea.

Methodology & Theoretical Orientation: This research attempted to explore mental health experiences from trauma to develop an evidence-based framework for preventing/early intervention of PTSD, depression, anxiety, or substance abuse of the population. A qualitative research design was used to examine the people's lived experiences.

Findings: A conceptual framework was constructed based on the paradigm of the trauma experience using 11 concepts (Figure 1).

Conclusion & Significance: Exploration of mental health problems associated with trauma provides evidence-based findings to help further mental health education, practice, administration, and research.

Biography
Yun-Jung Choi has her expertise in psychiatric/mental health nursing practice, research, and education. Her Hwa-Byung (Korean anger disorder) model based on Grounded theory creates new perspectives for understanding cultural care and improving healthcare. She also has contributed on disaster mental health care after years of experience in teaching and administration at the Red Cross College of Nursing. Recently, she is focusing on integrating her experiences of psychiatric/mental health nursing simulation using standardized patients, cultural mental health nursing, and psychological support for people with trauma.

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Notes:
Glioblastoma multiforme (GBM) is an aggressive primary tumor with frequent recurrences that leaves patients with a short survival time and a low quality of life. The aim of this study was to review prognostic factors in patients with glioblastoma multiforme. The focus of this retrospective study was a group of 153 patients with supratentorial GBM tumors, who were admitted in a tertiary-care referral academic center from 2005 to 2013. Factors associated with survival and local recurrence were assessed using the hazard ratio (HR) function of Cox proportional hazards regression and neural network analysis. Of the 153 patients, 99 patients (64.7%) were male. The average age of patients was 55.69±15.10 years. The median overall survival (OS) and progression-free survival (PFS) were 14.0 and 7.10 months, respectively. In the multivariate analysis, age (HR=2.939, P<0.001), operative method (HR=7.416, P<0.001), temozolomide (TMZ, HR=11.723, P<0.001), lomustine (CCNU, HR= 8.139, P<0.001), occipital lobe involvement (HR=3.088, P<0.001) and Karnofsky Performance Status (KPS, HR=4.831, P<0.001) were shown to be significantly associated with a higher OS rate. Furthermore, higher KPS (HR=7.292, P<0.001), operative method (HR=0.493, P=0.005), the use of CCNU (HR=2.047, P=0.003) and resection vs. chemotherapy (HR=0.171, P<0.001) were the significant factors associated with local recurrence of tumor. Our findings suggest that the use of CCNU and TMZ, operative method and higher KPS readings are associated with both higher survival and lower local recurrence rates.

Biography

Mohammad Sadegh Nikdad is currently working as a Professor in the Department of Neurosurgery, Tehran University of Medical Sciences, Iran. His major area of interest includes Neurology, Neurosurgery, etc. He has published several original articles in the reputed and peer reviewed journals and also participated into several scientific meetings.

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e-Posters
**Early diagnosis of dementia stages in AD with cerebral tomography morphometry**

Ivan V Maksimovich  
Clinic of Cardiovascular Diseases named after Most Holy John Tobolsky, Russia

**Background:** To early diagnose the severity of dementia in AD, we offer the Tomography Dementia Rating scale (TDR). Based on the morphometry of CT or MRI, the scale allows to determine the percentage of severity of atrophic changes in cerebral temporal lobes and to determine the severity of dementia due to the data obtained.

**Methods:** The research included 1078 patients: of which 93 patients aged 34-80 with AD at various stages were taken as Test Group, 985 patients aged 28-78 with various kinds of brain lesions with dementia but without AD (moderate and severe vascular dementia, Parkinson’s atherosclerosis,Binswanger’s disease, Parkinson’s disease) were taken as Control Group. The examination plan included CDR assessment, MMSE, cerebral CT, MRI with subsequent temporal lobes atrophy degree calculation, scintigraphy (SG), rheoencephalography (REG), and cerebral MUGA.

**Results:** CT and MRI among all patients with AD revealed that brain characteristic objective morphological features were temporal lobes atrophic changes of 4-62% at various AD stages. These data made it possible to make a scale allowing certain atrophic changes determination at each AD stage: • Preclinical AD stage - TDR-0: temporal lobes atrophy with 4-8% tissue mass decrease. • Early AD stage - mild dementia - TDR-1: temporal lobes atrophy with 9-18% tissue mass decrease (corresponds to CDR-1). • Average AD stage - mild dementia - TDR-2: temporal lobes atrophy with 19-32% tissue mass decrease (corresponds to CDR-2). • Late AD stage - severe dementia - TDR-3: temporal lobes atrophy with 33-62% tissue mass decrease (corresponds to CDR-3). These atrophic changes are not observed among patients with other cerebral lesions.

**Conclusions:** The morphologically determined scale of AD-TDR stages is an effective method for objectively determining AD stage by means of widespread CT and MRI. At the same time, this scale allows to differentiate AD from other diseases that are accompanied by the development of cerebral neurodegenerative changes complicated by dementia and cognitive impairment. The scale is easily applicable to medical institutions allowing correct and objective AD stage determination in clinical practice.

**Biography**

Ivan V Maksimovich, MD, PhD is a member of ISTAART, ESC, EAPCI, WSO, ESO, and EPA. He is a Head Physician of Clinic of Cardiovascular Diseases named after Most Holy John Tobolsky (Moscow, Russia) since 1993. One of the major problems the clinic deals with is the diagnosis and treatment of various brain lesions including Alzheimer’s disease. Over the past 20 years, he has published over 200 scientific works on this subject.

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Clinical description of familial cavernous malformations

M Peicheva, E Viteva, ATrenova, O Chaneva, Z Zahariev and ETournier Lasserve

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Familial cerebral cavernous malformations is a rare inherited disease with a frequency of 1-5/10,000 in the general population. It is inherited in autosomal-dominant pattern through three genes - KRIT1, CCM2 and PDCD10, located respectively in 7q21.2, 7p13, 3q26.1 chromosomes. The disease is diagnosed mostly in Latin Americans. The manifestation is with multiple cavernomas in the head and spinal cord and rarely in retina and skin. Clinical symptoms are determined by localization of the lesions and their size. Most frequently occurring are epileptic seizures, focal neurological deficits, non-specific headache, cerebral haemorrhages, skin and retinal lesions. The disease occurs usually in adulthood and about 40% of cases are asymptomatic. The diagnosis is made with MRI images with multiple vascular malformations of cavernous characteristics and molecular-genetic analysis of the family members. We present a case of familial cerebral cavernous malformations in a father and a son of Armenian origin, diagnosed in the Clinic of Neurology of St. George University Hospital in Plovdiv. In the father, the disease debuted at a young age with epileptic seizures successfully treated with antiepileptic therapy. At age 76, there was abducens nerve palsy as well as a rapidly growing tumor formation in the neck. In the son, a 33-year-old man, the disease is manifested with epileptic generalized tonic-clonic seizures. The MRI performed in both show a number of brain cavernomas of different locations and sizes, some with a typical “popcorn-like” image, as well as deposition of hemoglobin degradation products and calcifications. An adrenal adenoma of the right adrenal gland and two cysts in the left kidney were also found in the older patient. Retinal lesions are not visualized in both. Genetic screening confirms the presence of mutation c.1061_1064dup in the exon 11 of the KRIT1 gene (transcript NM_194455). This is the first family in Bulgaria with familial cerebral cavernous malformations with a mutation in the KRIT1 gene and a typical clinical development.

Biography

M Peicheva is a Bulgarian Neurologist with experience in Vascular Disease. She works in Clinic of Neurology at St. George University Hospital in Plovdiv, Bulgaria. Her interests are connected with ultrasound examination and vascular abnormalities. She is a Volunteer Consultant at Raredis, a part of Bulgarian Institute for Rare Disease. Her last publications are about some rare syndromes in Bulgarian population.

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Accepted Abstracts

CNS 2017
Spinal cord stimulation for chronic pain syndromes: Canadian experience

Ahmed A Al Jishi, Harsha Shanthana, Mauricio Forero, Valda Lopo, Frances Frazier, Mary Ann Van Doorn, Linda Gold, Jennifer Duley, Karen Carlino, Suzin Ilton and Philip Chan
McMaster University, Canada

Spinal cord stimulation (SCS) implantation has become popular in the last few decades. Currently, it is the gold standard therapeutic neuromodulation treatment that is being used in treating intractable chronic pain syndromes in patients who had failed medical treatment. The device has been successful in improving quality of life and decreasing narcotic intake. We wish to describe our experience with SCS implantation for the management of chronic pain syndromes and discuss our outcome through an outstanding and dedicated neuromodulation team.

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Autism Spectrum Disorders: Neurological and Medical Comorbidities

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The autism spectrum disorders (ASD) are a group of neurodevelopmental disorders characterized by impaired communication, social interaction and restricted and repetitive behaviors. In part, the impairment in communication results in more underdiagnosed comorbid disease. Neurological comorbidities including epilepsy, motor and sleep impairments will be discussed along with clinical findings in evaluation of the child with autism. Medical issues such as gastrointestinal disease, poor nutrition impact upon many systems including upon bone. Cross sectional and longitudinal studies have reported that boys with ASD have decreased bone mineral density. Children and adults with ASD also have higher odds of hip and other fractures based on a national emergency department database. The purpose of our study was to investigate the microarchitecture of bone and nutritional issues which could impact decreased bone development in boys with ASD. Methodology: observational longitudinal study of a cohort of approximately 20 boys with ASD and 20 age matched controls was followed for nutrition, areal bone mineral density and volumetric bone mineral density, which provides more information about fracture risk and had never been studied in a cohort of ASD boys. Findings: Boys with ASD had impaired microarchitectural parameters with reductions in bone strength estimates, calcium intake and IEF-1 responsiveness. Diet: Protein, calcium and phosphorus intake were lower in ASD than TDC and were associated positively with BMD measures. Conclusion and Significance: Boys with ASD were less physically active and protein, calcium and phosphorus intake were lower in ASD than control peers and are associated positively with bone density. The impairments in microarchitectural parameters may result from lower physical activity, calcium intake and IGF-1 responsiveness. Our study suggests that encouraging diets higher in fortified dairy and animal protein, as well as increased high-level exercise may improve bone health. Identifying neurological and medical comorbidities has important implications for better care of the child with autism spectrum disorders.

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Plastin3 as a therapeutic Target in Spinal Muscular Atrophy

Aziza Al-Rafiah
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Spinal muscular atrophy (SMA) is a devastating childhood motor neuron disease caused by mutations in the survival motor neuron 1 gene (SMN1). SMN1 and SMN2 are nearly identical genes producing the survival of motor neuron (SMN) protein. SMN protein plays a crucial role in mRNA splicing and β-actin mRNA transport along the axons. In SMA the mutation leads to the loss of SMN1, which cannot be fully compensated by the SMN2 gene, which predominantly produces a truncated protein. The loss or reduction of SMN protein leads to motor axonal defects and motor neuron cell death. There are currently no treatments available but therapies have focused on increasing SMN through replacing SMN1 or increasing full length SMN from SMN2. The actin-binding protein Plastin 3 (PLS3) has been reported as a modifier for SMA, making it a potential therapeutic target. Recently, it was shown that the overexpression of the PLS3 gene improved axonal outgrowth in SMN-deficient motor neurons of SMA Zebrafish and cultured motor neurons from mouse embryos. Gene therapy using viral vectors was carried out in vitro and in vivo to assess whether the overexpression of PLS3 could rescue neuronal loss in SMA and be developed as a therapy. The SMNΔ7 mouse model produces low levels of SMN, modelling severe SMA disease with an average lifespan of 12 days and loss of motor neurons. This study has established that the SMNΔ7 mice have little or no detectable PLS3 from birth, making it a good model for developing PLS3 gene therapy. Lentiviral vectors were able to upregulate PLS3 expression in different cell lines. Transduction of NSC34 cells with LV-PLS3 vector led to a five-fold increase in expression of PLS3 compared to controls. In smn-deficient MNs, expression of PLS3 restored axonal length and showed a strong neuroprotective effect. Pre-clinical in vivo proof-of-concept studies using adeno-associated virus serotype 9 (AAV9) encoding PLS3 in SMNΔ7 mice showed high transduction efficiency and overexpression of PLS3 specifically targeted to neurons in the central nervous system (CNS). This led to a small but significant increase of lifespan by 54%. However, PLS3 was not able to prevent disease onset. Although there was no improvement of phenotype, this study has demonstrated the potential use of PLS3 as a target for gene therapy, possibly in conjunction with other modulators of disease.

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High resolution, non-invasive multimode optical imaging: A proposed diagnostic and assessment tool in Alzheimer’s Disease

Daniel L Farkas
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Statement of the Problem: Alzheimer’s Disease (AD) is a major unmet health challenge characterized by (a) fast increasing incidence and costs; (b) very late diagnosis.

Methodology: Our group has recently [1] introduced optical imaging in the retina as a non-invasive method for mapping the occurrence, size and location of beta amyloid plaques, the primary pathology in AD. We have shown that using the fluorescence of curcumin, which attaches specifically to these plaques, we could quantitate the features of these plaques, including in vivo, and even document their reduction by immune treatments. These preclinical studies were also extended to the clinical domain, by using archival human eyes from patients with known levels of AD, as assessed both by brain histopathology and cognitive impairment (prior to death). We present a method for extending such studies to living patients, still using the retina as the window to the brain and plaques as indicators, but without the use of an extrinsic biomarker such as curcumin (as in [1]). This raises the level of experimental difficulty, thus requiring new technologies that we invented and/or perfected.

Findings: We designed a multimode optical imaging instrument, essentially a new type of confocal scanning laser ophthalmoscope, with some (needed) performance advantages over current commercial offerings. Our system consists of the following elements, all proprietary, and patent-protected: (a) A highly versatile light source: pulsed, 400-1400 nm, with ~1 nm resolution; (b) A new galvanometric method of scanning, with synthesized pivot point, not requiring a custom coupling lens; (c) Spectral analysis of imaging data, including hyperspectral image segmentation and elimination of background; (d) A more sensitive method of detecting light, via parametric

Conclusion & Significance: This new instrument achieves significant improvements in all of the following: spatial resolution, imaging depth, imaging angle in the retina (and thus spatial coverage), sensitivity and specificity. It will be used to image, fast and non-invasively, amyloid plaques in the retina, and any other retinal features of interest. We envisage that this instrument and the approach it enables should be used in AD drug/treatment trials, as it allows the repeatable, non-invasive and quantitative imaging of amyloid plaques (via both their autofluorescence and scattering), and of their relationships with important structures in the eye, such as blood vessels.

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Clinical Utility of Brainstem Auditory Evoked Potentials (BAEP) as Diagnostic and Prognostic Neurophysiological Markers in Patients with Concussion

Diana Tsakova
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Introduction: The standard approach in patients with concussion includes neurological/neurosurgical examination and neuroimaging (CT/MRI). Despite the normal results from these examinations, some complaints often persist for different period of time and disturb their quality of life. The aim of this study is to investigate changes of BAEP as diagnostic and prognostic neurophysiological markers in patients with concussion.

Method: Twenty-three patients (age range 18-44) with concussion were included in the study. Control group includes 35 subjects. In all 23 patients CT/MRI was conducted to exclude more severe TBI. In all patients were conducted BAEP in the first month after injury. In 10 patients a BAEP follow-up was carried out on the 3rd or 6th month after the trauma.

Results: The abnormal rate of BAEP for patients with concussion was 86.95%, indicating dysfunction of the brainstem in those patients. There was a statistically significant difference between the abnormal rate of patients and that of healthy persons. In the first month after the trauma 20 patients had abnormalities: delayed peak latencies, abnormal prolongation of interpeak intervals, interaural differences. 16 of them had more than one type of abnormalities. Three patients had normal BAEP. In control BAEP in 10 patients the abnormalities persist. (Fig.1).

Conclusions:

• BAEP can be applied as a diagnostic method in patients with concussion for objectifying some functional disturbances in cases of normal CT/MRI.
• Conducting control BAEP (3, 6 months) has an important role in monitoring the dynamics of pathological process.
• Persistent abnormalities in BAEP can be used as diagnostic and prognostic neurophysiological markers for establishment of incomplete recovery – for temporary disabilities and legal claims for compensation.
• In patients with MTBI with normal CT/MRI it is recommended expansion of the diagnostic algorithm with BAEP as objective, sensitive and highly informative indicator of brainstem disturbances.
Chronic Brain Glucose Deficits Modulates Neuropathology and Dementia: Implication For Alzheimer’s Disease

Domenico Praticò
Temple University Lewis Katz School of Medicine, USA

In recent years, growing experimental evidence has suggested a direct association between altered glucose metabolism, brain function and neurodegeneration. Together with human studies, the investigations using experimental models have all convened on a common final point: dysregulated brain glucose levels and impaired energy metabolism in the brain are not only a clinical feature of Alzheimer’s disease (AD) but also important contributors to its pathogenesis. Some evidence suggested that brain glucose deficits can influence amyloid beta levels in vivo but no data are available on the effect that this condition might have the development of tau neurofibrillary tangles and cognitive functions, the other two most important features of AD pathophysiology. In this paper we investigated the effect of chronic brain glucose deficits and energy dysregulation on memory and learning, synaptic function as well as the development of tau neuropathology in a model of tauopathy. Compared with controls, a condition of glucose deprivation and chronic brain energy deficiency resulted in significant memory deficits, impaired synaptic function, increased tau phosphorylation and neuronal cell death via apoptosis. Our studies demonstrate that reduced glucose availability in the central nervous system promotes directly the development of memory impairments, tau neuropathology, synaptic dysfunction, and neuronal cell death. Since restoring brain glucose levels and metabolism could afford the opportunity to positively influence the entire AD phenotype, it should be considered as viable therapeutic approach for this disease and related dementias.
Cucurbitacin B mitigates experimental autoimmune encephalomyelitis by inhibition of IL-17/IL-23 immune axis

Nima Sanadgol
University of Zabol, Iran

Pharmacological approaches to inhibit brain acute inflammation may represent important strategies for the control of autoimmune diseases. Multiple sclerosis (MS) is a chronic, inflammatory, demyelinating and autoimmune disease of the central nervous system (CNS). Cucurbitacin B (CuB), an oxygenated tetracyclic triterpenoid compound extracted from Cucurbitaceae plant species, is a bioactive agent by disruption of microtubule polymerization and inhibition of JAK/STAT signaling. However, there has been little information about impact of CuB on MS treatment. In this research, for the first time we examine effects of CuB (specific STAT3 blocker), in experimental autoimmune encephalomyelitis (EAE) mouse model of MS. EAE was induced by subcutaneous immunization of MOG35-55 in 8-week-old C57BL/6 mice. CuB was administered at different doses (0.25, 0.5 and 1 mg/kg body weight/day/i.p) from the first day of the experiment. Inflammatory responses were examined using qRT-PCR, western blot and immunohistochemistry (IHC) analysis of specific markers such as p-STAT3, IL-17A, IL-23A, CD11b and CD45. CuB reduced STAT3 activation, leukocyte trafficking, and also IL-17/IL-23 immune axis in this model. Treated mice with lower doses of CuB exhibited a considerable depletion in the EAE clinical score which correlated with decreased expression of IL-17, IL-23 and infiltration of CD11b+ and CD45+ cells into the CNS. Our in vivo results suggest that STAT3 inhibition by CuB will be an effective and new approach for the treatment of neuro-inflammatory disease such as MS.

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Effectiveness of an alternate approach durotomy in decompressive craniectomy in severe TBI

Venkataramana Pamidimukkala, Deepak S Mandwe, Harshavardhan Batsala, Sivaramakrishna Kondapalli, Kuchela Babu V and Mohana Rao A
MKCG Medical College, India

Introduction: Traumatic brain injury (TBI) is a leading cause of death and disability worldwide. Decompressive craniectomy is an emergency neurosurgical procedure in patients who have sustained TBI resulting in raised ICP.

Objective: To study surgical methods and techniques to improve results following decompressive craniectomy. Patients and Methods: We have treated 3177 TBI patients from January 2009 to December 2016. Decompressive craniectomy (DC) was done in 468 patients while rest were treated conservatively. The protocol followed was neuro ICU care, radiological and neurological monitoring. DC was done if there was neurological deterioration or midline shift in the CT scan. A speedy craniectomy was done with a ‘lazy question mark’ skin incision. The flap included the temporalis muscle, followed by a 12 to 16 cm wide craniectomy. The dura was first opened in the frontal region and ‘pizza slicing’ of dura was done. Additional cuts were made over the draining veins close to the sagittal sinus in the form of vascular tunnels. A synthetic dural graft was shaped and positioned but not sutured. The temporal muscle was not sutured. Single layered skin closure was done followed by the application of a loose bandage.

Conclusion: Pizza slicing of dural opening in contrast to conventional sinus based or skull base, based flap prevents compression on the veins which are already compromised due to oedema. Post traumatic malignant oedema and venous infarcts were greatly avoided. Keeping the dural patch loosely and single layer closure prevents damping of the brain pulsations. Wound complications or CSF leaks greatly decreased.

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Combined Effect of Baclofen and Acamprosate in Experimental Models of Peripheral Neuropathic Pain in Wistar rats

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Neuropathic pain (NP) is defined as pain associated with damage or permanent alteration of the peripheral or central nervous system. Current drug treatment for the management of neuropathic pain associated with various adverse effects. The present study was designed to investigate the combined effect of acamprosate and baclofen in experimental model of peripheral Neuropathic pain in wistar rats. Material and Methods: Neuropathic pain was induced by chronic constriction injured (CCI) of sciatic nerve in rats. Acamprosate (100 and 200 mg/kg p.o) and baclofen (10 and 20 mg/kg p.o) was given in different groups for 14 days starting on 7th day post sciatic nerve ligation. Further combination of acamprosate (100 mg/kg p.o) and baclofen (10 mg/kg p.o) was also given to one group. On 1th, 3rd, 7th, 14th and 21st day behavioral parameters like mechanical allodynia and thermal hyperalgesia were assessed. Then animals were sacrificed on 22nd day and biochemical parameters (GSH, LPO, catalase, nitrite, SOD) were assessed. Results: Ligation of sciatic nerve significantly induced mechanical allodynia and thermal hyperalgesia with increase in oxidative stress (increase in LPO and nitrite) and decline of anti-oxidant enzyme levels (catalase, SOD, GSH) in sciatic nerve homogenate. A camprosate (100 and 200 mg/kg p.o) and baclofen (10 and 20 mg/kg p.o) attenuated all the behavioural and biochemical parameters alone and/or combination.

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Imaging of serotonin synthesis and brain serotonergic receptors and their relation to affective disorders

Mirko Diksic
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The study of regional serotonin synthesis in humans by imaging could enable researchers to obtain a better understanding of affective disorders and to develop better therapies.

Objective: To perform imaging of regional brain serotonin (5-HT) synthesis with labelled α-methyl-L-tryptophan [α-MTrp] in the normal and affected brain, as well as in the animal models of depression.

Methods: Studies were performed on laboratory animals using two different rat models of depression, where the synthesis was measured by autoradiography, and a 14C and 3H-labelled tracer. Positron emission tomography (PET) with a 11C-labelled tracer was used in the human studies, on both normal subjects and on patients with various brain disorders (e.g., epilepsy, depression, obsessive compulsive disorder, borderline personality disorder, and those suffering from migraines). The patients met the DSM-IV criteria for depression or bipolar disorder (BPD), a disorder characterized by affective ability and impulsive aggressive behavior.

Results: The experiments in rats have shown that 5-HT synthesis is elevated in bulbectomized rats (using a model of agitated depression) and reduced in the Flinders Sensitive Line rats (using a model of retarded depression), and that antidepressants (e.g., citalopram, buspirone) have the effect of returning the synthesis to the level of the control rats without having a significant effect on plasma Trp concentration. The drugs have different effects following chronic and acute administration. The data suggest that 5-HT synthesis is differently controlled in the terminal areas than in the cell bodies. The results indicate that in healthy women, when compared to healthy men, serotonin synthesis is significantly lower in the right parietal lobe, bilateral middle frontal gyri, and bilateral parieto-occipital lobe. When comparing synthesis in the male BPD subjects to the male controls, we found a lower synthesis level in the anterior cingulate and left temporal lobe of the male BPD subjects, while the synthesis in the BPD subjects was higher in the posterior cingulated.

Conclusion: In depressed patients, we found a significant bilateral decrease in the anterior cingulate (ACC) (females), in the left ACC (males), and in the left mesial cortex (both gender). In addition, we have shown that antidepressants have region specific influence on 5-HT synthesis. In rat models of depression was shown that antidepressant produces changes in 5-HT synthesis and some 5-HT receptor sites. Some of these correlate with behavioural changes. The evaluation and imaging of regional synthesis, using α-MTrp as a tracer, is an excellent methodology for studying changes in brain 5-HT synthesis and the regional effects of drugs, in normal patients and in those with affective disorders which can help id discovery new drugs and new treatment modalities.

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