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4th EuroSciCon Conference on

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J Brathwaite et al., J Neurol Neurosci 2018, Volume: 9
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DIETARY POLYPHENOLS ENHANCE OPTOGENETIC RECALL OF FEAR MEMORY IN HIPPOCAMPAL DENTATE GYRUS GRANULE NEURON SUBPOPULATIONS

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Centers for Advancing Research on Botanicals and Other Natural Products (CARBON) Icahn School of Medicine at Mount Sinai, Supported by P50 AT008661 Center Grant by NCCIH and ODS Dietary polyphenols have been investigated for their role in promoting memory in model systems of stress, but little is known about select subpopulations of neurons that are influenced by polyphenols to improve memory performance. Granule neurons in the hippocampal dentate gyrus are vulnerable to stressors that impair the functioning of contextual memory and can be influenced by dietary polyphenols. We utilized a c-fos-tTA/TRE-ChR2 optogenetics model in which neurons activated during fear learning are labelled with ChR2-mCherry and can be optically reactivated in a different context to recapitulate the behavioural output of a related memory. Treatment with dietary polyphenols increased fear memory recall and ChR2-mCherry expression in dentate gyrus neurons in the same animal, suggesting that dietary polyphenols promote recruitment of neurons to a fear memory engram. We show that dietary polyphenols promote memory function and offer a general method to map cellular subpopulations influenced by dietary polyphenols, in part through the mechanism of c-Fos expression enhancement.

Biography

Justin Brathwaite has graduated from Columbia University in 2014 with a Bachelors of Arts, BA in Chemistry. Since graduation, he has been working full time as an Associate Researcher at the Icahn School of Medicine, Center of Molecular and Integrative Neuro-resilience directed by Dr. Giulio Pasinetti MD/PhD, under a Research Supplement to promote diversity in health related research awarded by the National Institute of health (NIH).

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Gokalp Arif Utkugun et al., J Neurol Neurosci 2018, Volume: 9
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A PKAN FAMILY PRESENTING WITH NIGHT BLINDNESS AS THE INITIAL SYMPTOM

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Neurodegeneration with brain iron accumulation (NBIA) consists of various genetically and clinically distinct forms of progressive motor disorders characterized by iron accumulation in the specific regions of the central nervous system. The most common subtype of this spectrum is pantothenate kinase-associated neurodegeneration (PKAN). The typical magnetic resonance imaging pattern called eye-of-the-tiger is seen in most of the patients with PKAN and basically it has two distinct forms, early onset rapidly progressive (classical) and late onset slowly progressive (atypical). The common presentation of patients with classical PKAN is dystonia with dysarthria and rigidity in the first decade of life. Our PKAN family members presented with night blindness as the initial symptom without developing dystonia for a while. Night blindness due to retinal involvement can be seen in the course of the disease but it is rare as the initial symptom in classical PKAN so it should be kept in mind.

Conclusion: **This case study shows that classical PKAN patients can present with night blindness without developing other symptoms. We wanted to emphasize this kind of presentation**

Biography

Gokalp Arif Utkugün has graduated from Cağaloğlu Anadolu High School in 2013. Since 2013 he has been studying medicine in Yeditepe University Faculty of Medicine. He speaks English and German. He is interested in General Surgery, Plastic and Reconstructive Surgery, Pharmacology, Neurology, Neuroscience and History of Medicine. He passed USMLE Step1 in 2017. In Jul' 2016 he did Observership at Plastic and Reconstructive Surgery in Vilnius, Lithuania. In Apr'2018, he attended New Era of Medicine congress in Istanbul.

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X-RAY PHASE CONTRAST TOMOGRAPHY FOR THE INVESTIGATION OF ALS DISEASE

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The study focuses on the degenerations of peripheral and central nervous system at relevant disease phases in mice affected with SOD, an animal model for Amyotrophic Lateral Sclerosis (ALS), using X-ray phase-contrast tomography (XPCT). Compared to standard X-ray Tomography, XPCT is an advanced technique that allows three-dimensional reconstruction of bio-medical samples without any sectioning or aggressive preparation or use of contrast agents. XPCT is a powerful technique to analyze low absorbing objects and enables a multiscale imaging ranging from cellular-level up to the whole-organ. We analyzed mice spinal cords at different stages of ALS, providing deeper knowledge on the degeneration of motor neurons and vascularization in the central nervous system as well as their 3D spatial distribution. The analysis was therefore extended to the peripheral nervous system, both in the anterior and posterior spinal nerves, as the peripheral motor nerve damage precedes neuronal degeneration within the spinal cord. We will show, at different time points, the quantification of the variations in the vascular and neuronal networks of the spinal cord, already detectable in a pre-symptomatic stage of the disease. We correlated these results with those obtained in the peripheral nervous system, where, thanks to the high spatial resolution, we quantify the orientation of spinal nerve fibers. This preclinical study will be able to lay the groundwork for future clinical applications

Biography

G Begani Provinciali has completed her Master's degree in January 2018 and she is employed as a Research Fellow at the Institute of Nanotechnology (CNR, Rome Unit). She has published one paper in reputed journals and she is Co-author of a book chapter in press.

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RESTORATION OF WALKING SKILLS WITH USE OF KINESITHERAPEUTIC EXERCISES ON LOKOMAT SYSTEM AT CHILDREN WITH A CEREBRAL PALSY

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Rehabilitation of children with a cerebral palsy (CP) remains a relevant problem because CP results heavy disability. On the basis of the Center of social adaptation of children complex medico-psycho-pedagogical correction at a cerebral palsy is applied. Complex services contain medical examination, determination of rehabilitation potential of children, psychology classes (Montessori therapy, sensotherapy, a fairy tale therapy, music therapy), classes of special teachers (sand art therapy, services of an office of early intervention), a hydrokinetic therapy physiotherapy exercises, massage and classes on the robotic Lokomat system. Lokomat allows preventing progressing of pathology at early stages of rehabilitation. 40 children aged from 4 up to 10 years with the diagnosis of a cerebral palsy, a spastic diplegia were an object of a research. In the 1st group 15 children were practicing on the Lokomat system without preliminary complex medico-psycho-pedagogical correction (GMFCS IV-V). In the 2nd group 25 children before training on the Lokomat system received complex medico-psycho-pedagogical correction, massage and a hydrokinetic therapy and were doing physiotherapy exercises (GMFCS III-IV). Each course of a training consisted of 15–20 classes. The children of 2nd group moderately expressed increase of muscular force, decrease in level of a spasticity of a muscular tone was noted; the volume of free active movements has authentically increased. The best results were noted at the children who have begun training at the earliest age. Especially it should be noted that children of the 2nd group have improved the indicators after several repeated rehabilitation courses (GMFCS II). Results of the conducted research have shown that use of the robotic Lokomat system in early rehabilitation of children with a cerebral palsy after complex medico-psycho-pedagogical correction, allows to accelerate process of restoration or development of skills of standing and walking

Biography

Madina Alimova is student at Tashkent Pediatric Medical Institute. She was a participant of Student's scientific society in Tashkent Pediatric Medical Institute, held on April 15th, 2015. She also has served as volunteer in Republic Centre of Social Adaptation of Children. Her research interest lies in Pediatric Neurology, in particular, perinatal defeats of nervous system, cerebral pain and epilepsy.

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DUAL DRUG-LOADED LIPID NANOPARTICLES FOR THE TREATMENT OF PARKINSON'S DISEASE

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The aim of the present study is formulation of solid lipid nanoparticles (SLNs) containing combination of bromocriptine and resveratrol for effective management of Parkinson Disease. The goal of this therapeutic strategy is to reduce the occurrence and severity of L- DOPA (LD) associated motor fluctuations and dyskinesia, and providing good long-term safety and tolerability. Dopamine agonists such as bromocriptine provide moderate symptomatic benefit and delay the development of dyskinesia compared with levodopa. Resveratrol is a natural polyphenolic compound suggesting that they could have important antioxidant properties and resveratrol could possibly reduce the side effects of bromocriptine but its oral bioavailability is very low due to its extensive hepatic and presystemic metabolism. One of the prime benefits of combination therapy is the potential for providing synergistic effects. However, bromocriptine suffers from low bioavailability and short half-life. Therefore, it would be a good candidate for a sustained drug-delivery system. SLNs were prepared using high speed homogenization followed by ultrasonication technique. The prepared SLNs were characterized by entrapment efficiency percent (EE %), particle size distribution, zeta- potential, and cumulative percentage release. The mean particle size measured ranged from 100-220 nm. The EE % ranged between 81.00±0.92% - 92.52±0.10%. Smaller size and narrow size range allows them to cross tight endothelial cells of the blood–brain barrier (BBB), escape from the reticuloendothelial system (RES), and bypass liver. They have comparatively higher drug entrapment efficiency, render the drug more stable in their lipid matrix, and provide a controlled release lasting up to several weeks. The prepared SLNs exhibited a zero-order sustained release profile and met the requirement for a brain targeting; hence it could be a promising strategy to deliver bromocriptine to the brain.

Biography

Asha Spandana K M is currently pursuing her PhD in Pharmaceutical Sciences in the area of Brain Targeted Drug Delivery System at JSS University, Mysore, India. She accomplished her Undergraduation from JSSCP, Mysore under RGUHS, Bengalore in 2010 and M Pharm in Pharmaceutics from RGUHS, Bengaluru, under merit (GPAT-qualified) in 2013. She has published 4 papers in national and international level.

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BEHAVIOR ADOPTED AT THE FIRST SYMPTOMS OF STROKE

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Introduction: Stroke is the second cause of death in Colombia. The level of knowledge of stroke within the population can determine its ability to recognize and make the decision to go to an emergency room.

Objective: The objective is to study the attitude, behavior adopted by the patient, the family before a stroke in patients diagnosed as ischemic stroke in 4 different Hospital centers of Bucaramanga, Colombia.

Methods: Information on 348 patients was included; they were followed in a Cohort from November 2015 to December 2017. Information was collected on patients and / or relatives.

Results: The mean age of the patients was 69.2 years. 75% came from urban areas. During the acute presentation of the event (stroke), 69% of patients or families did not recognize or think of stroke as a possibility. 8.9% did not initially consider that it was an important illness and 6.3% thought that it would recover spontaneously. In the sample analyzed, only 8% underwent vascular recanalization therapies.

Conclusions: Knowledge about stroke is still very poor in the general population and this affects the out-of- window arrival of these patients to hospital centers, and the low percentage of patients who can benefit from recanalization therapy

Biography

J P Garzon Hernandez is a fourth-year medical student at the Universidad Industrial de Santander (UIS) in Bucaramanga. She serves as an Assistant Investigator at the Cohort study of Stroke of Neurovascular Sciences, FCV, HIC, with the main goal being to expand knowledge about stroke in Colombia. Her research interest in clinical is cerebrovascular disorders.

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PREVENTION OF STROKE IN PATIENTS WITH CHRONIC CEREBRAL ISCHEMIA IN THE BACKGROUND OF ISOLATED SYSTOLIC HYPERTENSION

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Chronic cerebral ischemia (CCI) is a form of chronic progressive cerebrovascular disease characterized by multifocal or diffuse brain damage and is manifested in the form of complex neurological and neuropsychological disorders. The main manifestations of CCI are impairment of cognitive (recognition) function, affective disorders and movement disorders polymorphic. The main etiological factor of cerebral microangiopathy is hypertension, which causes arteriosclerosis (lipogialinoz) of small penetrating arteries and arterioles. Although patients with CCI prefer to focus on such subjective symptoms like headache, dizziness, tinnitus, fatigue, the core clinical CCI determining the severity of the condition of patients is increasing restriction of neuropsychological, motor functions. The result of neuropsychological disorders in CCI is the development of vascular dementia. We observed 766 people (300 women, 466 men; mean age 65±105 years), held in-patient treatment in the Clinical Hospital of Tashkent in the period from November 2013 to December 2015. All patients were followed-up visit, which included a neurological examination: ECG, Doppler ultrasound, EEG and a set of neuropsychological tests. Patients were divided into two groups: I group consists of standard therapy of CCI + nitrendipine 10-40 mg / day + alzepil 5 mg/day at morning 6 months. II group consists of CCI standard therapy. Repeated study of patients had clinically significant achievement of target blood pressure, as well as significantly improvement of cognitive functions in the first group of patients. There were 4 cases of stroke during the next two years in the first group of patients (atherothrombotic origin). In the second group of patients there were 19 cases of stroke, 3 of them repeated. Use of the drug nitrendipine for the correction of blood pressure in elderly patients significantly lowers the risk of stroke and gross cognitive impairment

Biography

Munisa Bakhadirova has graduated from Tashkent Medical Institute (1988 – 1994) and completed her Residentship in Neurology (1994 – 1996) at Tashkent Medical Institute. In 2002 she completed her PhD on Rates of cerebrovascular flow at different stages of stroke rehabilitation. Since 2004 she has been an Assistant Professor, Neurerehabilitation department, Tashkent Institute of Postgraduate medical education.

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EXPLORING FIXATION PATTERNS AND SOCIAL COGNITION AFTER TRAUMATIC BRAIN INJURY

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[2]Rotherham Doncaster and South Humber NHS Trust, UK
[3]Sheffield Community Brain Injury Rehabilitation Team, UK

Objectives: Social cognition (SC) impairments after traumatic brain injury (TBI) are pervasive. The movie for the assessment of social cognition (MASC) measures different facets of social interactions over the three stages of SC; social perception, social knowledge retrieval and response selection. The mechanisms underpinning SC deficits after TBI are poorly understood but aberrant eye fixation patterns could play a role. The present research explored fixations across social interactions to determine group differences and correlations between eye tracking and behavioural data.

Design: Group differences in response selection during the MASC and fixation duration/count to areas of interest (eyes, nose and mouth) were examined.

Methods: 18 TBI participants were recruited from the NHS and age/gender matched controls were recruited using stratified opportunity sampling. The MASC allows for quantification of incorrect answers; excessive theory of mind (ToM), reduced ToM and absence of ToM errors. The MASC was presented on a Tobii T120 eye tracker monitor.

Results: TBI participants had significantly lower correct scores on the MASC and higher excessive/reduced errors compared to controls. There was no significant interaction between automated optical inspection (AOI) and group. However, significant main effects of group for fixation duration/count indicated that if AOI was ignored, controls displayed longer/more fixations overall suggesting a difference in visual scanning patterns between TBI and control groups. No significant correlations were established.

Conclusions: TBI and controls exhibited disparate visual strategies during the MASC and this effect could underpin some SC impairments displayed by TBI participants. TBI participants also displayed insufficient and over-interpretative mental state reasoning compared to controls but it is unclear why. The present research outlines the multifaceted nature of SC impairments after TBI and highlights potential areas for SC intervention post-TBI

Biography

Leanne Greene has completed her BSc in Psychology and an MSc in Applied Cognitive Neuroscience. She is about to complete her PhD on Social Cognition and Saccadic Eye Scan Patterns in TBI and Control Groups which is supported by Sheffield Hallam University. She currently works as an Assistant Psychologist for Rotherham Doncaster and South Humber NHS Trust in the Neuro Rehabilitation Outreach and Stroke team. Socioemotional problems post-TBI are often not assessed or rehabilitated (Kelly, McDonald & Frith, 2016) and Leanne is passionate about raising awareness of social cognition after TBI in the future developing contemporary and ecologically valid clinical assessments and rehabilitation programmes.

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BREATH HOLDING SPELLS: CLINICAL HISTORY ASSESSMENT

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Objective: Breath holding spells (BHS) is a common case in children aged 5 months to 6 years, and often misinterpreted with epileptic seizures. We made clinical and epidemiological assessment for BHS. We documented the relation between sex, age, familial history and the episodes nature blue, pale or mixed.

Materials & Methods: It was a cross-sectional study in which a total of 75 children (30 boys, 45 girls) with BHS, admitted to our center (Nour Institute of Pediatricneurology: NIPN), between 2014 and 2016. We took full medical history and did the investigations to eliminate the differential diagnosis.

Results: Most patients were 7-24 months; we noticed parental consanguinity in 87% of cases, and familial history in 56%. The spells were cyanotic 70%. Anger and pain were the more frequent risk factors (64%, 60%). Anaemia was found in 58% of patients. Half of the patients have socioeconomic problems. Most of them were with a family history (66%).

Conclusion: The study mentioned the types of spells, the risk factors and the important role of anaemia

Biography

Hadia Bakri has completed her specialization in Neurological pediatrics; CHU, Sart Tilman campus, Citadel hospital, University of Liege, Belgium (1995). She is the Director of Nour Institute of Pediatric- Neurology (NIPN), Damascus, Syria.

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ROLE OF FRMD7 IN SYNAPTIC CONNECTIVITY IN THE RETINA

Ahmed Salman, Diego Gomez-Nicola, Andrew Lotery and Jay Self
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Nystagmus is a condition of the eye characterized by an involuntary and uncontrolled movement. It has a significant impact on vision in general and can affect patient's ability to lead an independent life. FERM domain-containing 7 plasma membrane protein (*FRMD7*) is a member of the band 4.1superfamily of plasma proteins, found to be mutated in families with nystagmus. The lack of *Frmd7* leads to deficit of direction selectivity in mice retina by loss of asymmetric inhibition of direction-selective ganglion cells (DSGC's) by the starburst amacrine cells; the cells that express *Frmd7*. Experiments show no morphological alterations in these cells upon loss of *Frmd7*. However, no evidence if the synapses between the starburst amacrine cells and DSGC's are affected. The aim of this poster is to examine the integrity of the synapses of the starburst amacrine cells.

Methods: Synapses in freshly perfused C57Bl6 controls vs *Frmd7* transgenic knockout (*Frmd7*.^{tm1b}) retina (N=5) were studied by immunohistochemistry of frozen retinal sections.

Results: Initial results show no significant deficit in the integrity of synapses in the starburst amacrine (Acetylcholine transferase (ChAT) expressing) cells in the mouse retina. Density and intensity of ChAT expressing cells in C57Bl6 retina is similar to that of the *Frmd7*.^{tm1b}. Also, density of the presynaptic and postsynaptic markers, synaptophysin and PDS95 respectively, is normal. In addition, density of gamma-Aminobutyric acid (GABA), which is also expressed by the starburst amacrine cells, was also normal.

Conclusion: *Frmd7* is somehow involved in modulating inhibitory signals from the starburst amacrine cell to the DSGC's in the retina. Level of general pre-synaptic and post-synaptic markers in the *Frmd7*.^{tm1b} transgenic retina seem to be indistinguishable from wild type controls, so as expression levels of acetylcholine and GABA, which indicates the synaptic markers in the *Frmd7*.^{tm1b} mice are similar to the wild type control mice. However, the inhibitory feedback from the starburst amacrine cells to the DSGC's is compromised.

Biography

Ahmed Salman obtained his BSc Honours degree in Genetics form the University of Glasgow in 2009. He then studied for his MSc by research in Brasenose college, University of Oxford, under the supervision of Professor Elizabeth Robertson, investigating genes involved in early mouse embryonic development. After finishing his Masters, he stayed in the Robertson lab as a research assistant before joining the Welcome Trust Centre for Human Genetics in Oxford, where he worked on the mechanisms of double-stranded break repair for a year, before starting his PhD in the University of Southampton in 2014, working on the role of Frmd7 gene in nystagmus, a significant eye disease characterised by involuntary eye movements. He is currently in the final year of studies aiming to submit his thesis at the end of 2018.

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Nigora Kadyrkhodjayeva et al., J Neurol Neurosci 2018, Volume: 9
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OPTIMIZATION OF MANAGEMENT OF PRIMARY CHRONIC HEADACHE WITH THE USE OF BOTULOTOXIN A

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Aim: The aim of the study was to review the quality of life of the patients with daily chronic headache (CDH) and determine the effectiveness of Botulinum toxin type A (BTA) influence on improving the quality of life by reducing the daily headache.

Material & Methods: 54 patients, from both sexes, with a minimum age of 18 years old were studied. The inclusion criteria were the presence of primary daily chronic headache with more than 4-hour duration, a frequency of 15 days or more monthly, in the last three months and disease duration of 3 years for the treatment of CDH using BTA. The study involved 54 patients, of whom where 31 women and 23 men with an average age of 42 years. The patient's condition was evaluated on the third day, on the 7th day and on the 15th day after the BTA injection and assessed every 15 days for 3 months. The efficacy of BTA was evaluated by several measurements of VAS (Visual Analog Scale), Headache Intake Questionnaire: HSQoLQ (Headache Specific Quality of Life Questionnaire), HMQ (Headache Management Questionnaire), HDQ (Headache Disability Questionnaire).

Results: After 3 months 2 (4%) patients had no changes, 7 (13%) patients with less than 50 percent reduction in pain, 23 (43%) reported 70 to 95 percent pain relief, and 22 (40%) had complete relief.

Conclusion: The work presented here has profound implications for future studies of BTA injections for patients with CDH. The obtained results testify to an improvement in the quality of life of patients with CDH against the background of injections of BTA.

Biography

Kadyrkhodjayeva Nigora has finished her study at the Tashkent Medical Academy in 2006. After that, she was studying Psychiatry and Psychotherapeutics from 2006 till 2007 at the Tashkent Institute of Postgraduate Medical Education. She was trained in Neurology at the Tashkent Institute of Postgraduate Medical Education 2007-2009. She has been practicing as a Neurologist since 2009. She passed Clinical attachment in Neurology Department of Rashid Hospital, Dubai, UAE. She has valid practice license from UAE. She successfully cleared certification course of BLS & ACLS in 2018. From September 2017, she is pursuing her PhD at the Tashkent Medical Academy. She has published 3 articles in reputed journals and several articles, abstracts in local journals.

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HARNESSING THE PERICYTES TO PROMOTE NEUROVASCULAR REPAIR AFTER ISCHEMIC STROKE

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Ischemic stroke constitute a major cause of death and disability of the adults in the world. Unfortunately, no efficient therapy does yet exist. Endogenous neurovascular restorative responses are triggered within the ischemic tissue, as an attempt from the brain to recover. Ischemic stroke triggers the formation of new microvasculature in the peri-infarct region via activation of various angiogenic mechanisms. Neuronal survival is higher in the tissue undergoing angiogenesis, correlating with longer survival in stroke patients. Angiogenesis is a highly dynamic process that involves close and finely tuned interactions between brain endothelial cells and pericytes. Pericytes play major roles in regulating the cerebral blood flow, angiogenesis, microvasculature stability, and blood-brain barrier (BBB) properties. Ischemic stroke profoundly affects the function of pericytes by triggering their death and detachment from brain endothelial cells, which impairs key neurovascular functions within the ischemic tissue. Using in vivo and in vitro approaches, our recent work demonstrates that vascular endothelial growth factor isoform-B (VEGF-B), which acts as survival factor, promotes the formation of stable microvasculature within the ischemic tissue by specifically enhancing the survival of pericytes and their interaction with brain endothelial cells. We found that the effects of VEGF-B are mediated via its specific receptor VEGFR-1 that is predominately expressed in brain pericytes. Our study unravelled an unknown role of VEGF-B/VEGFR-1 signalling in rescuing the function of pericytes by inducing expression of the anti-apoptotic protein, B-cell lymphoma 2 (Bcl-2) and AMP-activated protein kinase α (AMPKα) protein, which is involved in energy homeostasis. Moreover, VEGF-B/VEGFR-1 signalling stimulates the release of factors stimulating a reparative angiogenesis that does not compromise microvasculature stability and BBB permeability. Our findings suggest that strategies aiming to stimulate the endothelial cell-pericyte crosstalk constitute a promising therapeutic approach to promote neurovascular repair upon ischemic stroke.

Biography

Ayman ElAli has completed his PhD in 2010 at the University of Duisburg-Essen in Germany. He pursued his Post-doctoral trainings at the research centres of University Hospital of Essen in Germany, and CHU de Québec in Canada. He joined in 2015 the Department of Psychiatry and Neuroscience, Faculty of Medicine, Laval University, Canada, as an Assistant Professor. His research program aims at Investigating Neurovascular Interactions Following Stroke with an Emphasis on Developing New Therapeutic Approaches. He has published more than 37 papers in top-tier journals and has been serving as Referee in several reputed funding organizations and journals.

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REVERSIBLE CEREBRAL VASOCONSTRICTIVE SYNDROME AND DIFFERENTIAL DIAGNOSTICS OF MIGRAINE

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Reversible cerebral vasoconstriction syndrome (RCVS) is characterized by cerebral blood vessels segmental vasoconstriction, spontaneous recurrent vasodilation within 3 months, impairment of cerebral arteries regulation. One of the main symptoms proved to be an intensive course was severe headache. The disease of migraine initially begins with the headache; then this condition is followed by moderate paroxysmal intensification, pulsating headache and nausea. Investigation of reversible cerebral vasoconstriction syndrome and differential diagnostics of migraine. Investigation included examination of neurological patients who were undergone both In- and Out-Patient treatment in Neurological Department of Andizhan State Medical Institute; magnetic resonance angiography examination was carried out by Namangan Anamed Medical Service. 60 patients have been studied. They were divided into two groups. The first group included 35 patients with reversible cerebral vasoconstriction syndrome. The second group included 25 patients with migraine. There were 17 male (48.5%) and 18 females (51%) aged 29-72, the average age is 50.5 years old within the first group. There were 10 male (40 %) and 15 females (60%) aged 15-45, the average age is 30 years old within the second group. Patients of the first group suffered from severe intensive headaches (visual analog scale - VAS) onset of headache was initiated within few minutes and aggravated on physical exertion. According to MMSE (Mini Mental State Examination), 25 patients (72%) from the first group demonstrated 24-27 points; mild cognitive impairments have been observed during examination of neurological condition; the rest 10 patients (28%) demonstrated 28-30 points. Cerebral arterial vasoconstriction was observed in patients of the first group on magnetic resonance angiography examination. Patients of the second group suffered from moderate intensive headaches (VAS); onset of headache was initiated within few hours, neurologic examination failed to reveal any pathologic changes. According to MMSE, the first group patients demonstrated 28-30 points. Cerebral arterial vasoconstriction was not observed in the second group patients on magnetic resonance angiography examination. In comparison with migraine, reversible cerebral vasoconstriction syndrome results from severe intensive headaches within short period of time, mild cognitive impairments are observed; cerebral vasoconstriction is observed on magnetic resonance angiography examination.

Biography

EM Tashkenov has completed his Bachelor's degree (2005-2012), Master's degree (2013-2016) from Andizhan State Medical Institute. He was an Assistant Professor (2016-2017) at Department of Neurology, Andizhan State Medical Institute. He is pursuing his PhD from 2018 on Reversible Cerebral Vasoconstrictive Syndrome, Diagnostics and Optimization of Treatment Methods from Andizhan State Medical Institute. The main fields of his clinical researches are Neurology. He is interested in differential diagnostics and treatment headache

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SEXUAL DIMORPHISM OF NITROGEN MONOXIDE RATE AND LIPID PEROXIDATION AT EARLY AGE: OBSERVATION AFTER 4H, 24H, 48H OF INFECTION WITH LIPOPOLYSACCHARIDE

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Inflammation is a defense reaction of the body's immune system to harmful external aggression such as a bacterial infection. Moreover, it is an important source of oxygen radicals produced directly by activated phagocytic cells that are the site of a phenomenon called oxidative explosion. This work was done to find out whether infection with early lipopolysaccharide (LPS) administration can result in changes in nitric oxide levels and lipid peroxidation in the central nervous system, specifically prefrontal cortex and the hippocampus. Male and female wistar rats at 7 days of age were divided into 2 groups: a control group receiving an intraperitoneal injection (ip) of PBS, the second group treated with LPS (250 µg / kg, ip). The results show that the rate of nitric oxide in the hippocampus in a physiological state during the 3 days postnatal (7.8, and 9) increases in females at the age of 8 days and decreased later, but in males, the rate increases with age. Also at the level of chlorpyrifos (CPF), we observed increased rate of nitric oxide in both sexes without any sexual dimorphism. The rate of lipid peroxidation at a physiological state is increased at the age of 8 and 9 days for both sexes compared to 7-day-old rats. But once after an infection induced by the LPS, it was observed that the rate of lipid peroxidation in the hippocampus and prefrontal cortex at an age of 7 days is increased for both sexes, but on the 8th day we found a sexual dimorphism and at PDN9 age the lipid peroxidation rate is increased in males than females. It is concluded that postnatal injection of LPS induces a decrease in nitric oxide rate, and an increase in lipid peroxidation in the prefrontal cortex and the hippocampus.

Biography

Najlae Elfathi has completed his/her Bachelor's degree in Physics Chemistry in 2010. In 2014 she, did life science in biology, & in 2017 she did Master on neurocognition human and health of the population, and now, she is pursuing PhD (1st Year) on early neuroinflammation and glial activation

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SOME PHARMACOGENETIC ASPECTS OF PATIENTS OF THE UZBEK POPULATION WITH PHARMACORESISTANT FLOW OF EPILEPSY

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The sample of patients for pharmacogenetic analysis was 88 patients with a pharmacoresistant course of epilepsy (the presence of epileptic seizures that had not been recovered in patients for more than 12 months). The diagnosis of epilepsy was established after clinical-neurological, electrographic and neurovisualizational methods of investigation. Comparative aspect was performed in 68 patients with favorable epilepsy and in 60 healthy donors of Uzbek nationality. At the beginning of the work, we were able to select and optimize the work of oligo primer systems for the study of polymorphisms and predictive efficacy of the 1236 T/C and 13435 T/C of the MDR gene. For polymorphism 1236 T/C of the MDR gene, sensitivity and specificity showed average values and corresponded to SE=0.66 and SP=0.53. At the same time, the calculated AUC (0.60) also shows the average level of effectiveness according to the classifier of this marker as an independent candidate gene. Because of high frequency of the 13435 T/C polymor-phism of the MDR1 gene, its prognostic value also turned out to be high (SP=0.81) and an aver-age sensitivity level with the SE index of 0.6, compared to all other loci (in which these values deviated significantly in the direction of specificity), one can speak of a good independent effect of this on the risk of developing pharmacoresistance in the Uzbek population AUC=0.70. Evalu-ation of the efficiency of genetic markers 430C>T gene also proved to be very low. These data forces us to conclude that these polymorphisms are an ineffective classifier for marking a resistant form of epilepsy. Thus, of all the candidate genes we studied, only the polymorphisms 1236 and 13435 of the MDR1 gene are effective classifiers for predicting the development of pharmacoresistance.

Biography

Tuychibaeva NM is an Assistant Professor, Neurologist at Tashkent Medical Academy, depart-ment of Neurology and Intermed Clinic City Child Diagnostics Center respectively. She has completed her Bachelor's degree (1990-1996) and Clinical Fellowship in Adult Neurology (1996-1999) at Tashkent Medical Academy, Uzbekistan. She obtained her PhD in Medicine (March 29, 2007) on Clinical features of Consequence of light cerebral trauma from Second Tashkent State Medical Institute. She has done training courses on epilepsy, pediatric related topics. The main fields of her clinical researches are neurology and medical genetics. She has an expertise in epilepsy, but now she is also interested in different movement disorders, especially in childhood

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USE OF KETOGENIC DIET IN CHILDREN WITH PHARMACOLOGICAL RESISTANT FORMS OF EPILEPSY

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Background: Antiepileptic drugs (AED) considered to be the most effective method of treat-ment of epilepsy, but they are not only ineffective in pharmacoresistant forms, but also can make patient's condition more severe. It forced us to risk and at the first time in Uzbekistan tries to use ketogenic diet in patients with epilepsy.

Materials & Methods: We investigated the duration of pharmacoresistant epilepsy in 7 children of Uzbek nationality aged from 2 to 9 at the first time when treated by AED, then by glucocorti-coids and after the cancellation of drugs and use only classic ketogenic diet. All of them were under the clinical examination with use of MRI, EEG with video monitoring, and USD of liver and gallbladder, ketone and glucose tests. In children 3 used only glucocorticoids; in 4 children we used combination of hydrocortisone in dose 5 mg/kg and AED. Proven ineffectiveness of AED and glucocorticoids allowed us to prescribe them ketogenic diet with macronutrient ratio 4:1 that was counted by ketocalculator. Every week parents provided ketone bodies analyses in urine and measured glucose level in blood of children. Once a 3 months was performed blood analysis by gas analyzer.

Results: In all children on EEG was noted decreased epileptiform activity, but much better re-sults were in patients without MRI changes. Convulsions were under the control, there almost were not generalized seizures in patients with Lennox-Gastaut, West and Landau-Kleffner syn-dromes. Cognitive functions, when their impairment was the main symptom, also partially re-turned and there was observed significant progress in psychical development and social adapta-tion of children. There were no any cases of hypoglycemic coma.

Conclusion: Ketogenic diet if followed correctly may significantly improve the condition of pa-tients with pharmacoresistant forms of epilepsy

Biography

Tuychibaeva NM is an Assistant Professor, Neurologist at Tashkent Medical Academy, depart-ment of Neurology and Intermed Clinic City Child Diagnostics Center respectively. She has completed her Bachelor's degree (1990-1996) and Clinical Fellowship in Adult Neurology (1996-1999) at Tashkent Medical Academy, Uzbekistan. She obtained her PhD in Medicine (March 29, 2007) on Clinical features of Consequence of light cerebral trauma from Second Tashkent State Medical Institute. She has done training courses on epilepsy, pediatric related topics. The main fields of her clinical researches are neurology and medical genetics. She has an expertise in epilepsy, but now she is also interested in different movement disorders, especially in childhood

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EFFECT OF INSULIN ON NEUROINFLAMMATORY RESPONSE AND OXIDATIVE STRESS INDUCED BY A BLOCKER OF KV1.3 CHANNEL

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Voltage-dependent potassium channels (Kv1.3), play key role in a wide variety of physiological processes, including immunity, metabolism and the stabilization of the resting potential. In brain, activation of insulin receptor is able to induce current suppression coupled to tyrosine phosphorylation of Kv1.3 channel. Moreover, insulin can reduce the production of free radicals and attenuate the inflammatory response. The Kv1.3 channel blockers, such as neurotoxins isolated from scorpion venom, are able to alter neuronal excitability leading to neurological disorders accompanied by inflammatory response. The aim of this study is to evaluate the neuroprotective effect of insulin injected by intra-cerebro-ventricular (i.c.v.) route on neuro-inflammatory response and oxidative stress induced by a blocker of Kv1.3 channel. The ability of insulin to reduce the brain injuries, inflammatory response and oxidative stress biomarkers induced by Kv1.3 channel blocker were assessed in NMRI mice at 24 h after co-injection of insulin and neurotoxin active on potassium channel. Obtained results revealed that the central administration of insulin prevents cerebral cortex injury, brain edema, cells infiltration and a change in the permeability of the blood–brain barrier induced by the Kv1.3 channel blocker. Insulin seems to also reduce significantly the pro-inflammatory cytokines (IL-6, IL-17, TNF-α), MMP-2 and MMP-9 activities and oxidative stress markers (H2O2, NO, MDA) in brain homogenates compared to those observed when animals were injected with Kv1.3 channel blocker alone. These results indicate that insulin is able to modulate the activity of potassium channels in brain by modifying their properties, which probably prevent the binding of neurotoxin to its receptor Kv channel and thus reduce the neuro-pathophysiological effects.

Biography

Zahida Taibi-Djennah has completed her PhD in Biochemistry-Immunology and Innovative Biotherapies from University of Sciences and Technology Houari Boumdiene, Faculty of Biological Sciences, Laboratory of Cellular and Molecular Biology. She is an Associate Professor level B at University of Sciences and Technology Houari and is team member of Biochemistry of Biomolecules: Mode of Action, Immunotherapy and Immunodiagnosi (http://www.lbcm.usthb.dz/spip.php?rubrique4). She has published 5 papers in reputed journals including Systemic Responses following Brain Injuries and Inflammatory Process Activation Induced by a Neurotoxin of *Androctonus* Scorpion Venom in the *Journal of Neuroimmunomodulation* and Effect of cytokine antibodies in the immunomodulation of inflammatory response and metabolic disorders induced by scorpion venom in the *Journal of International Immunopharmacology.*

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ANTICONVULSANT AND ANTI-INFLAMMATORY EFFECTS OF THE POLYSACCHARIDE RICH EXTRACT FROM GENIPA AMERICANA LEAVES

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Plant polysaccharides present some activities involving the central nervous system, such as neuroprotective, antidepressant, antioxidant and anti-inflammatory. We aim to evaluate the anticonvulsant and anti-inflammatory effects of the polysaccharide rich extract from G. *americana* leaves in mice. The leaf dry powder (5 g) was depigmented in methanol and the polysaccharide-rich extract (PRE) obtained by extraction with NaOH followed of precipitation in absolute ethanol. PRE was dissolved in 0.9% NaCl and administered (9 mg/kg) in male Swiss mice (25-35 g) by intraperitoneal (i.p.) route, 30 min before seizures induced by a single dose of pentylenetetrazole (PTZ: 70 mg/kg, i.p), n=7/group. The synergism of PRE effect was evaluated by its association with diazepam (DZP: 0.01 mg/kg). After euthanasia, the prefrontal cortex (CPF), hippocampus (HC) and striatum (EC) were removed for the quantification of myeloperoxidase levels (MPO) by o-dianisidine method. Experimental protocol was approved by Animal Ethics Committee (UECE Nº 2451142/2014). The PRE increased the seizure latency (9 mg/kg: 171,7 ± 29,62 *versus* saline: 62.00 ± 4,709 s) and death latency (9 mg/kg: 597.4 ± 101,5 *versus* saline: 150.0 ± 14.52). The association of PRE with diazepam potentiated the protective effect of DZP, increasing seizure latency (DZP: 128,3 ± 24,62 versus PRE + DZP: 222.4 ± 47.57), without altering in death latency. MPO levels was reduced in hippocampus (PRE: 34.24 ± 7.167, DZP: 42.27 ± 9.559 and DZP + PRE: 31.26 ± 5.726 *versus* saline + PTZ: 81.91 ± 11.70) and striatum (PRE: 17,89 ± 3,310, DZP + PRE: 18.69 ± 3.776 *versus* saline + PTZ: 37.27 ± 5.169). However there was no difference between groups (DZP, PRE or DZP + PRE) in each brain area. We conclude that PRE of *G. americana* leaves protects against seizures and promote anti-inflammatory effects in brain.

Sponsor's names: (CNPq/ CAPES/FUNCAP)

Biography

Edna Maria Camelo Chaves finished her doctorate at the age of 6 at the Federal University of Ceará, in Pharmacology. She is an Adjunct Professor at the State University of Ceará, working in the Graduate Program in Physiology and Nursing. She has published more than 13 articles in renowned magazines and has served as a reviewer of scientific journals.

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POTENTIAL ROLE OF ORGANOPHOSPHATE INSECTICIDE CHLORPYRIFOS IN AUTISM SPECTRUM DISORDER (ASD)

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Autism spectrum disorder (ASD) is a neurological and developmental disorder that begins early in childhood and lasts throughout a person's life. ASD is characterized by impairment in interaction and social communication, in addition to pro-inflammatory cytokine imbalances with chronic neuroinflammation. Environmental exposures may increase the risk of ASD. There are evidences that as the residue crosses the blood-brain barrier and placenta the fetuses can be exposed to pesticides. The purpose of this study is to summarize and discuss the relationship between autism spectrum disorder and chlorpyrifos, an organophosphate insecticide. The narrative review was performed using MEDLINE, LILACS, Web of Science, Scopus and Science Direct as databases and pesticides, agrochemicals, insecticides, herbicides, Autism disorder as descriptors. Gestational contact with chlorpyrifos interferes early neuromotor development and causes deficits in social behaviour that can lead to long-term deficits in behaviour and repetitive behaviour, as a routine preference. Studies have shown that the contact of chlorpyrifos with already autistic rats increased the characteristics of this disorder in the animals. In addition, contact with chlorpyrifos causes redox imbalance, oxidative stress, mitochondrial dysfunction associated with glutathione deficiency. Studies have also shown that there is a high probability of developing imbalances in the intestinal flora. Autistic individuals may as well exhibit proinflammatory cytokine imbalances and may suffer from hyperactive or dysfunctional immune systems, with chronic neuroinflammation, including neuroglial activation in the brain, and the presence of autoantibodies to brain proteins. Thus, we can conclude that exposures to agricultural pesticides such as chlorpyrifos, through the uterine pathway are related to autism and that there is strong evidence that contact with pesticides may influence the development of autism spectrum disorder.

Biography

Gislei Frota Aragão is graduated in Pharmacy, with Masters and PhD in Pharmacology with a focus on neuropharmacology. Professor of Medical Course at the State University of Ceará (UECE/Brazil) and He is coordinator of the Group of Studies in Neuroinflammation and Neurotoxicology (GENIT). Pharmacist of the Federal University of Ceará (UFC) acting in the Clinical Pharmacology Unit and as a researcher in the laboratory of toxicology and clinical exams in the Drug Research and Development Center (NPDM) with collaborations in the professional Master of Clinical Pharmacology/UFC, Master of Transplantation/UECE and Specialization in Collective Health/UECE, developing projects in the area of neuropharmacology, neurotoxicology, neuroinflammation and pharmacovigilance.

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SLEEP DISORDERS IN ALZHEIMER DISEASE

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Sleep disorders have a variable spectrum and are present in all forms of dementia, especially in Alzheimer's disease (AD). Elderly patients generally present with sleep disturbances, but this association is more frequent in patients with AD. The aim of this work was to perform a narrative review on the alterations in sleep that occur in patients with AD. A literature review was conducted using MEDLINE, LILACS, Web of Science, Scopus, Science Direct as databases and Alzheimer disease, sleep wake disorders, dyssonias as descriptors. It has been observed that sleep disorders are framed as one of the symptoms of AD, in addition to being related to physiological and genetic patterns. The main symptoms are getting up at night and waking up at night thinking it is day. The incidence of these symptoms was detected in patients with worse cognitive and functional status, lower socioeconomic status and depression. The relationship between insomnia, aggression, paranoid delusions and anxiety was observed. Recent studies have seek to clarify the etiology of sleep disorders, considering associations between absence of healthy sleep with greater deposition of amyloid load in brain regions such as angular gyration, frontal medial orbital cortex, cingulate gyrus and precuneus. Disorders of orexin levels in the cerebrospinal fluid in patients with AD were observed, promoting a change in the activation of the Wake-active monoaminergic system and the deactivation of the REM-on cholinergic groups, reducing sleep homeostasis. Lower body temperature at the end of the day causes disorders of the circadian rhythms in AD and a deficiency in the negative regulation of the proximal blood flow of the daytime skin has been found which may also affect the process. These researches initiate the development of new treatments, which will impact the patient's cognition and, consequently, their quality of life. We conclude, therefore, that the sleep disorders are one of the fundamental clinical aspects that must be evaluated in AD patients, specially due to its role as a prognostic changer for the disease.

Biography

Tatiana Paschoalette Rodrigues Bachur is a Pharmacist, graduated from the Pharmaceutical Sciences Course of the Federal University of Ceará (UFC-1999) and has completed her Masters in Pathology from the Federal University of Ceará (UFC-2007). She is a Professor of the Medicine Course of the State University of Ceará - UECE, Brazil, Coordinator of specialization courses in the distance learnig modality and collaborates in the Group of Studies in Neuroinflammation and Neurotoxicology – GENIT from UECE. She develops studies in Toxicology, Pharmacology and Tropical Diseases and is a reviewer of scientific journals and project leader.

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EFFECT OF INSULIN ON NEUROINFLAMMATORY RESPONSE AND OXIDATIVE STRESS INDUCED BY A BLOCKER OF KV1.3 CHANNEL

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Voltage-dependent potassium channels (Kv1.3), play key role in a wide variety of physiological processes, including immunity, metabolism and the stabilization of the resting potential. In brain, activation of insulin receptor is able to induce current suppression coupled to tyrosine phosphorylation of Kv1.3 channel. Moreover, insulin can reduce the production of free radicals and attenuate the inflammatory response. The Kv1.3 channel blockers, such as neurotoxins isolated from scorpion venom, are able to alter neuronal excitability leading to neurological disorders accompanied by inflammatory response. The aim of this study is to evaluate the neuroprotective effect of insulin injected by intra-cerebro-ventricular (i.c.v.) route on neuro-inflammatory response and oxidative stress induced by a blocker of Kv1.3 channel. The ability of insulin to reduce the brain injuries, inflammatory response and oxidative stress biomarkers induced by Kv1.3 channel blocker were assessed in NMRI mice at 24 h after co-injection of insulin and neurotoxin active on potassium channel. Obtained results revealed that the central administration of insulin prevents cerebral cortex injury, brain edema, cells infiltration and a change in the permeability of the blood–brain barrier induced by the Kv1.3 channel blocker. Insulin seems to also reduce significantly the pro-inflammatory cytokines (IL-6, IL-17, TNF-α), MMP-2 and MMP-9 activities and oxidative stress markers (H2O2, NO, MDA) in brain homogenates compared to those observed when animals were injected with Kv1.3 channel blocker alone. These results indicate that insulin is able to modulate the activity of potassium channels in brain by modifying their properties, which probably prevent the binding of neurotoxin to its receptor Kv channel and thus reduce the neuro-pathophysiological effects

Biography

Zahida Taibi-Djennah has completed her PhD in Biochemistry-Immunology and Innovative Biotherapies from University of Sciences and Technology Houari Boumdiene, Faculty of Biological Sciences, Laboratory of Cellular and Molecular Biology. She is an Associate Professor level B at University of Sciences and Technology Houari and is team member of Biochemistry of Biomolecules: Mode of Action, Immunotherapy and Immunodiagnosi (http://www.lbcm.usthb.dz/spip.php?rubrique4). She has published 5 papers in reputed journals including Systemic Responses following Brain Injuries and Inflammatory Process Activation Induced by a Neurotoxin of *Androctonus* Scorpion Venom in the *Journal of Neuroimmunomodulation* and Effect of cytokine antibodies in the immunomodulation of inflammatory response and metabolic disorders induced by scorpion venom in the *Journal of International Immunopharmacology*.

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THE NEURAL CREST, A KEY REGULATOR OF BRAIN DEVELOPMENT AND HOMEOSTASIS

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The neural crest (NC), a defining feature of vertebrate embryo, generates most of the skeletal tissues encasing the developing forebrain and provides the prosencephalon with functional vasculature and meninges. Our investigations show that, aside from its structural role in craniofacial ontogenesis, the NC exerts a potent morphogenetic paracrine role on the brain and sense organs development. In the light of recent findings, which document the molecular mechanisms whereby the migratory NC cells control cephalic neurulation and forebrain morphogenesis, our investigations show that cephalic NC regulates the morphogenetic activities of secondary brain organizers and modulates long-distance cues emanating from these territories. NC cells act in these processes through a multistep control and exert cumulative effects counteracting signals produced by the neighboring tissues. By this mechanism, the cephalic NC cells supersede ventralizing influences and promote the elaboration of the prosencephalic alar and roof plates. Altogether, these data highlight the developmental relationships between the cephalic vesicles and the migratory NC cells, and show that the cephalic NC itself acts like a dorsalizing brain organizer. In addition, our work opens new avenues for revisiting the etiology of neurological disorders in the light of cephalic NC dysfunctions. In experimental models which virtually reproduce developmental encephalopathies and pervasive developmental disorders characterized by defective cognitive functions, our recent data reveal that congenital social impairments may have an extrinsic origin and involve misregulation of trophic factors produced by the CNC cells.

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EVIDENCE FOR A CAUSAL CONTRIBUTION OF MACAQUE VESTIBULAR, BUT NOT INTRAPARIETAL CORTEX TO HEADING PERCEPTION

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Multisensory convergence of visual and vestibular signals has been observed within a network of cortical areas that are involved in representing heading. Vestibular-dominant heading tuning has been found in the macaque parieto-insular vestibular cortex (PIVC) and adjacent visual posterior sylvian area (VPS), whereas relatively balanced visual/vestibular tuning was encountered in the ventral intraparietal (VIP) area and visual-dominant tuning was found in the dorsal medial superior temporal (MSTd) area. Although the respective functional roles of these areas remain unclear, perceptual deficits in heading discrimination following reversible chemical inactivation of area MSTd suggested that areas with vestibular-dominant heading tuning also contribute to behaviour. To explore the roles of other areas in heading perception, muscimol injections were used to reversibly inactivate either macaque PIVC or VIP bilaterally. Inactivation of anterior PIVC increased psychophysical thresholds when heading judgments were based on either optic flow or vestibular cues, although effects were stronger for vestibular stimuli. All behavioural deficits recovered within 36 hours. Visual deficits were larger following inactivation of the posterior portion of PIVC, likely because these injections encroached upon VPS, which contains neurons with optic flow tuning (unlike PIVC). In stark contrast, VIP inactivation led to no behavioural deficits, despite the fact that VIP neurons show much stronger choice-related activity than MSTd neurons. These results suggest that area VIP either provides a parallel and partially redundant pathway for this task, or does not participate in heading discrimination. In contrast, PIVC/VPS, along with MSTd, make causal contributions to heading perception based on either vestibular or visual signals.

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MOLECULAR MECHANISMS OF ISCHEMIC STROKE: NEURODEGENERATION AND NEUROPROTECTION IN PENUMBRA

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In ischemic stroke vascular occlusion and energy deficit rapidly induce tissue infarct. Cell damage propagates to surrounding tissues for several hours. This therapeutic window provides time to save neuronal cells in penumbra. To determine proteins involved in neurodegeneration and neuroprotection in penumbra, we studied protein expression profile in 2-mm ring around photothrombotic infarct core induced in rat cerebral cortex by local laser irradiation after rose bengal administration. Histological and ultrastructural studies showed edema and degeneration of neurons, glia and capillaries, which decreased gradually across penumbra. Expression profile of 224 signalling proteins 1, 4 or 24 hours after photothrombotic infarct comparing with untreated contralateral cortex was studied with antibody microarrays. Diverse cellular subsystems were involved in penumbra response. Proteomic analysis showed concerted up-regulation of diverse proteins that initiate, regulate and execute apoptosis (Par4, E2F1, p75, p38, JNK, p53, GADD153, GAD65/67, NMDAR2a, c-myc, Bcl-10, AIF, SMAC/DIABLO, PSR, caspases 3, 6 and 7). Different anti-apoptotic (Bcl-x, p63, p21WAF-1, MDM2, ERK5, MKP-1, NEDD8) and signalling proteins that regulate cell metabolism, functions and survival (calmodulin, CaMKIIα, CaMKIV, ERK1/2, MAKAPK2, PKCα, PKCβ, PKCμ, RAF1, protein phosphatase 1α, ATF2, estrogen and EGF receptors) were simultaneously overexpressed. Bidirectional changes in adhesion and cytoskeleton proteins were associated with penumbra destruction or remodelling. Proteins that regulate actin cytoskeleton (cofilin, actopaxin, p120CTN, α-catenin, p35, myosin Va, pFAK) were up-regulated, whereas others (ezrin, tropomyosin, spectrin (α+β), βIV-tubulin, polyglutamated β-tubulin, cytokeratins 7 and 19) were down-regulated. Down-regulation of syntaxin, AP2β/γ, and adaptin β1/2 indicated impairment of vesicular transport and synaptic processes. Down-regulation of Cdk6, Cdc7 kinase, Trf1, and topoisomerase-1 showed suppression of proliferation. APP, nicastrin and β-amyloid were up-regulated. These data provide integral view on neurodegeneration or neuroprotection processes in penumbra. Some of these proteins may be potential targets for anti-stroke therapy.

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THROUGH CEREBRAL SPINAL FLUID BRAINSTEM STIMULATION FOR VEGETATIVE STATE PATIENTS

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Objective: To work out the method which provides diffuse stimulation of brain stem reticular formation (RF) in vegetative state (VS) patients without additional damage of brainstem and thalamic structures by implanted electrodes.

Methods: Nine patients with VS for more than 3 months were treated by the method of through cerebral spinal fluid brainstem electrical stimulation (TCSFBS) for a period of 1 year. Two monopolar electrodes were implanted for TCSFBS implementation. The first electrode was inserted in the lateral ventricle of spared hemisphere. The second one was implanted in the cistern Magna or epiduraly between low margin of occipital bone and posterior arch of C1 vertebra. Clinical effects, electro-encephalogram (EEG) and auditory brainstem response (ABR) were researched in all patients during TCSFBS therapy.

Results: Such well known markers of RF activation as arousal response (AR) and desynchronization reaction (DR) were detected in all VS patients during TCSFBS. Six out of the 9 cases emerged from VS. Two out of these 6 cases regain consciousness. The other 4 patients were in MCS. The remaining 3 cases failed to emerged from VS.

Conclusion: Efficiency of RF stimulation for VS patients' treatment is shown in researches of class II evidence. Method of deep brain stimulation (DBS) with implantation of bipolar electrode in thalamic or brainstem structures provides RF activation in some VS patients. A part of VS patients do not display signs of RF activation during DBS which may be due to brainstem and thalamic lesion foci which are mandatory for post traumatic VS patients. The TCSFBS can provides activation of RF in VS patients without additional damage of thalamic and brainstem tissue in the trajectory of implanted electrodes

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OXFORD STUDY IN QUANTIFICATION OF PARKINSONISM (OXQUIP) - EYE MOVEMENTS, GAIT TRACKING AND COGNITIVE FUNCTION

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The OxQUIP (Oxford QUantification in Parkinsonism) study has been recruiting patients with Parkinson's disease and progressive supranuclear palsy. Currently available treatments for these diseases are symptomatic only, and do not have any preventive or disease-slowing effect. As new drugs are developed, we need to be able to evaluate them quickly, so that precious time and resources can be devoted to those showing most promise. In this study, we follow participants intensively over a two year period, with the aim of identifying measures that can detect disease progression over much shorter time periods than is possible at present. During the study, participants are asked to perform simple tasks while we measure movements of the eyes, hands and body. We also do some tasks on a tablet computer that measure cognitive performance. Today, I will present you some of the recent exciting data from the OxQUIP study.

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RELATIONSHIP BETWEEN LIFE SATISFACTION, ACTIVITY, PARTICIPATION AND DEPRESSION IN ELDERLY PEOPLE WITH STROKE

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Objective: The aim of this study was to investigate the participation of elderly people who had stroke, activity performance, depression and life satisfaction; activity, participation and depression in relation to life satisfaction.

Materials & Methods: The study was conducted on 47 stroke patients who had a mean age of 71.76±6.14 years. The assessments were based on the ICF disability model. According to this model, body functions and structures, activities, participation evaluations were made. The Functional independence scale (FIM) was used for the evaluation of basic daily activities, the Lawton-Brody supplementary daily life activity scale for evaluating the helping activities of daily living (ADHD), Adelaide activity profile (AAP) for assessing the frequency of participation in activities, geriatric depression scale (GDS), Canadian activity performance measure (COPM) to assess personality-centered participation and activity performance, and life satisfaction index (LSIA) to determine life satisfaction level.

Conclusions: According to the FIM evaluation, the activities most frequently challenged by the patients were bathing, dressing (lower-upper body), going to the toilet and cooking, home cleaning and shopping activities at Lawton-Brody IADL. In the AAP evaluation, the patients were found to have at least daily household work activities (20.54%) and social activities (24.75%). With the person-centered COPM criterion, the performance and satisfaction problems of patients in the field of self-care as well as leisure have been identified. It was determined that 61.70% of the patients had low and medium life satisfaction and 59.57% of the patients had depression according to GDS results. There was a significant relationship between all performance domains and frequency of activities and depression and life satisfaction apart from productivity satisfaction evaluation ($p<0.01$). There was a significant relationship between the activities of auxiliary and basic daily life activities and life satisfaction ($p<0.01$)

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ROLE OF MICROBIOTA DERIVED SHORT CHAIN FATTY ACIDS IN α-SYNUCLEIN AGGREGATION AND SEEDING

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Objective: To determine whether microbiome derived short chain fatty acid (SCFA) may modulate abnormal α-¬synuclein misfolding and seeding activity of α-synuclein to support the hypothesis of potential novel therapeutic approaches.

Background: There is growing evidence from both *in vivo* and *in vitro* studies that in many neurodegenerative disorders, including synucleinopathies, cell-to-cell transmission of a pathological, misfolded protein occurs and may be a vehicle for spreading of pathology throughout the brain. We hereby investigate whether microbiota-derived SCFAs may help attenuate the misfolding of α-synuclein and their effects on seeding synucleinopathy.

Design/Methods: *In vitro* aggregation of α-synuclein in the absence or in the presence of SCFAs at a molar ratio of 1:1 or 1:4 α¬-synuclein:SCFA, were monitored by using independent assays: photo-induced cross-linking of unmodified proteins assay, thioflavin-T, fluorescence assay, or electron microscopy.

Results: We found that select microbiome-derived SCFAs significantly interfere with α-synuclein aggregation in independent *in vitro* assays.

Conclusions: Selected microbiome-derived SCFAs may help protect against diverse synucleinopathies by converting dietary fibers into biologically available SCFAs which significantly interfere with aggregation of disease-specific α-¬synuclein into toxic aggregates. Ongoing cell-based systems, which detect levels of α-synuclein by florescent FRET signalling, will clarify the impact of this anti-aggregation activity of SCFAs on interference of α-synuclein seeding activity that is critical for the propagation of α-synuclein mediated pathologies. This data will help to clarify distinct α-synuclein seeding activity from α-synuclein isolated from post-mortem brain samples of patients with Parkinson's disease, multiple systemic atrophies, and other synucleinopathies, leading to novel therapeutic approaches

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RECOMBINANT T-CELL RECEPTOR LIGAND 1000: A NOVEL THERAPEUTIC FOR STROKE

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The worldwide prevalence of stroke continues to rise despite recent successes in treating acute ischemic stroke. With limited patient eligibility and associated risk of tissue plasminogen activator (tPA) and mechanical thrombectomy, new preventive and therapeutic modalities are needed to stave the rising wave of stroke. Inflammation plays a key role in brain damage after cerebral ischemia and novel therapies that target pro-inflammatory cells have demonstrated promise for treatment for stroke. Partial MHC class II constructs have been shown to prevent and/or reverse clinical signs of various inflammatory diseases such as experimental autoimmune encephalomyelitis, collagen-induced arthritis and experimental autoimmune uveitis, by reducing the number and frequency of activated cells in the damaged CNS. Herein, we review the use of partial MHC class II constructs as a novel treatment for ischemic stroke. These constructs have been shown to reduce infarct volume and neurological deficit in various cerebral ischemia models in young adult and aging male and female mice. In addition, partial MHC class II constructs were shown to reverse stroke-associated splenic atrophy and promote a protective M2 macrophage/microglia phenotype in the CNS which contributes to tissue repair and recovery after stroke. By addressing remaining STAIR criteria, such as efficacy in large animal models of stroke, these constructs will be prime candidates for clinical trials of acute ischemic stroke.

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INITIAL ACTIVATION OF ASTROCYTES / DEREGULATION OF CDK5 AXIS IN AD AND ALZHEIMER'S DISEASES

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Astrocytic activation initiating neuroinflammation is closely associated with many neurodegenerative diseases including Alzheimer's disease (AD), amyotrophic lateral sclerosis (ALS), Parkinson's disease (PD), Multiple Sclerosis (MS). Neuronal insults lead to deregulation and hyperactivation of Cdk5, the major neuronal cell cycle kinase, in the nervous system initiates neuropathology. Using p25Tg AD model mice (CaMPKII P25Tg) we found that initially astrocytes are activated due to higher activity of phospholipase 2A (PLA2) induced by Cdk5 phosphorylation of Cdk5 consensus sequence in PLA2 and affecting phospholipid metabolism. This leads to increase in factors like arachidonic acid and activation of neuroinflammation and neurodegeneration. It is well known that under physiological conditions, the initial interactions among neurons, astrocytes, microglia, and oligodendrocytes are regulated and induce secretion of basal levels of various factors e.g. complement proteins, cytokines and chemokines essential for physiological conditions. The expression of these diverse molecules is tightly regulated since they act as trophic factors essential for nervous system development and function. However, under stress and toxic conditions, additive effects of neuronal chronic stress/insults/toxicity, in addition to ageing leads to overexpression of these factors which leads to neuroinflammation and neurodegeneration. A systematic, time course study of the expression and activation of these factors provided the evidence that activation of astrocytes is the initial step in the deregulation of Alzheimer's disease in AD model system.

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NEW APPROACHES TO PREDICTION OF TREATMENT OUTCOME IN MENTAL PATIENTS USING BACKGROUND NEUROBIOLOGICAL DATA

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Heavy social-economic burden of mental disorders and rather high percent of non-responders determine the need of multidisciplinary approaches to investigation of brain mechanisms of this severe illness for optimization of its treatment. One of the ways for such optimization is early individual prediction of therapeutic response. Brief review on EEG predictors of treatment outcome in terms responder/non-responder will be presented. As well, the innovative author's approach to quantitative individual prediction of treatment outcome in patients with delusional disorders conditions in the frames of paranoid schizophrenia will be described. Correlation and regression analyses of quantitative clinical scores (by PANSS scale), resting EEG spectral parameters and some immunological parameters have been performed in patients with manic-delusional and hallucinatory-delusional conditions in the frames of paranoid schizophrenia. Neurobiological data obtained before the beginning of syndrome based treatment course (at visit 1) were matched with clinical scores of the same patients at the stage of remission establishment after treatment course (at visit 2). The multiple linear regression equations were created which contained only 3 to 4 (from 80) initial EEG parameters and one of four immunological parameters. These mathematical models allowed predicting from 65% to 87% of PANSS scores variance after treatment course (at visit 2). Deviation of predicted PANSS scores values in patients of the control group from their real values at visit 2 varied from 5% to 24% for different PANSS scales, and was significantly lower than permitted deviation. The data obtained emphasize the role of neurophysiological inhibition deficit and of processes of neuroinflammation and neuroplasticity in pathogenesis of manic-delusional and hallucinatory-delusional conditions, and may be used practically for elaboration of methods of individual prediction of syndrome based treatment efficiency in patients with paranoid schizophrenia.

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THE QUANTUM BRAIN

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For 2400 years since Democritus the matter's smallest element was considered the atom, quantum mechanics established that atom is a universe of particles, protons, neutrons, electrons, photons, fermions and bosons, etc. If the quantum infrastructure is the common denominator of nature, and nature can be expressed in two forms: corpuscles and waves, are corpuscles the infrastructure of the brain and waves the infrastructure of the mind? When applying Einstein's equation, photoelectric effect, the double existence of the matter: corpuscle and wave, the observer effect, Niels Bohr's concept on the atom, quantum spin, Heisenberg's principle of uncertainty and Schrodinger's entanglement, to the human body a quantum mechanics profile of the biology starts to offer a new understanding of the reality. It was proven that Quantum physics is involved in the life of plant photosynthesis, bird navigation, the sense of smell, and anesthesia. The permanent movement of particles with the speed of light and with an energy equivalent to the explosion of an atomic bomb (according to Einstein equation), in every material, impose the acceptance of quantum life in the biology and in the brain. The quantum mechanics may be involved in the finite live span of: sperm cells-3 days, colon cells-4 days, skin cells-2 months, red cells-4 months, liver cells-5 months, white cells-1 year, neuron cells last an entire lifetime. The quantum mechanics seems to be involved in the massive replacement of hundreds of trillion cells of the body every 7 years.

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THE ROLE OF DRUG EFFICACY SHOULD BE DOWNGRADED IN ICHD DIAGNOSTIC CRITERIA

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Chronic migraine (CM) is a disabling disorder which is under-diagnosed and under-treated. The International classification of headache disorders (ICHD-3β) requires response to migraine-specific medication as one of its criteria. But migraine-specific medications are still not available on a global scale from previous data. This may be because other types of analgesics are not only effective and cheaper than triptans. Moreover, migraine-specific medications can also be effective against other primary headaches, such as cluster headaches and some secondary headaches. Consequently, it is very evident that the ICHD-3β criteria for CM are difficult to apply in clinical practice on a worldwide basis. Hemicrania continua (HC) are an uncommon type of primary headache. Absolute sensitivity to indomethacin is required as one of the diagnostic criteria for HC. However, earlier reports showed that cases with HC-like headaches should still prompt additional evaluations for secondary causes. Tolosa-Hunt syndrome (THS) is an important cause of painful ophthalmoplegia (PO), and defined by the ICHD-2 with adequately relieve by corticosteroids, which is used as one of the diagnostic criteria. However, the ICHD-3β criteria published in 2013 removed the item. This revision indicated that corticosteroid response remained meaningful for THS and that corticosteroid treatment could confirm the final diagnosis of THS, rather than diagnose THS. Consequently, downgrading the role of corticosteroid treatment is deemed to be reasonable. Hence, based on the criteria of responsivity of treatment by drugs, the precise diagnosis of headache remains controversial, and thus implying potential risk for inappropriate diagnosis and poor management. The diagnosis of headache should precede the remedy; drug efficacy should not be required as a diagnostic criterion. Treatment response, however, could help to confirm the final diagnosis of headache in cases where the original diagnosis was undefined. Consequently, we propose that it is entirely reasonable to downgrade the role of treatment response in the ICHD diagnostic criteria for headache.

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NEW PERSPECTIVES ON LENTICULOSTRIATE VASCULOPATHY IN NEONATES

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Lenticulostriate vasculopathy (LSV) is a sonographic term given to branching hyperechogenic lines in the basal ganglia and /or thalamus seen on cranial ultrasound scans. LSV was first described on a neonatal cranial ultrasound in 1985, but the clinical importance, relevance to congenital infections, and long-term consequences of LSV on neonatal cranial ultrasound continues to be unclear. The incidence of LSV being reported has increased recently, which might reflect nothing more than a growing awareness of this finding on neonatal cranial ultrasound. On the other hand, improved ultrasound imaging technology may have enhanced identification, and there may be an increase in the frequency of risk factors contributing to the presence of LSV. We suspect that improvements in US technology have enhanced the visibility of the arterial walls in the supratentorial deep gray matter. Thus, thin and faint lenticulostriate vessels that are seen on neonatal cranial US using contemporary technology may not necessarily pathological. This review on LSV provides an update of current knowledge, with emphasis on definition and challenges that might have evolved with establishing the diagnosis during the last three decades. It has been accepted that lenticulostriate arteries supplying the deep gray matter are not normally visualized on the cranial ultrasound. For the first time in the literature, we challenged this notion in light of the recent technological advances in ultrasound imaging that have enhanced ultrasound imaging. Conflict still exists in terms of the clinical importance and long-term outcomes of LSV since the first case reported three decades ago. In this article, we also scrutinized the available evidence on clinical correlation of this neonatal ultrasound finding, discussed long-term outcomes, and provided strategies that may guide practitioners in clinical settings.

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SEARCHING FOR THERAPIES TO IMPROVE INTELLECTUAL DISABILITY IN AUTISM: LESSONS FROM THE FRAGILE X SYNDROME MOUSE MODEL

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Fragile X Syndrome (FSX) is the leading single gene cause of autism and intellectual disability (ID). Neurons express a high density of underdeveloped dendritic spines in FXS humans and animal models. Synaptic plasticity deficits are prevalent throughout the brains of FXS mouse models including the cortex and hippocampus, areas critical for various forms of learning and memory. Moderate to severe learning deficiencies are also characteristic in FXS patients and is paralleled in mouse models. Therefore, FXS is an ideal model in the clinical and laboratory setting to investigate therapies aimed at autism and ID. In FXS mouse models, hyperactive Rac1 has been demonstrated in hippocampus and cortex where dendritic spine abnormalities are a common feature. Herein, we study whether pharmacological regulation of Rac1 might represent a promising treatment for cognitive impairment in autism, using Fragile X syndrome (FXS) as a model. Our results show that in the Fmr1 KO mice (an animal model of FXS) deficits in memory and synaptic plasticity are associated with the presence and mislocalization of Rac1. Interestingly, treatment of Fmr1 KO mice with a specific Rac1 inhibitor improves memory and increases hippocampal LTP. Taken together these observations suggest that Rac1 might contribute to FXS related learning and memory impairments in humans. Importantly, this study proposes that targeting Rac1 in FXS may rescue cognitive impairments. Such a therapy may be translated into broader applications in autism and ID.

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EPIGENETIC MECHANISMS IN NEURAL STEM CELLS AND MENTAL HEALTH

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Epigenetics regulate gene expression and brain development through mechanisms that are not directly controlled by the genetic DNA sequences. Recent discoveries have highlighted the importance of epigenetic mechanisms in brain development, stem cell differentiation, and mental health. MeCP2 is an important epigenetic factor in the brain and its mutation or altered expression leads to impaired brain development and function. Rett Syndrome is a severe neurodevelopmental disorder that is caused by *MECP2* mutations and has no cure. These patients seem to be normal at birth, but within the first 2 years of age, they display developmental regression, mental disability, neurological symptoms, seizers, speech deficiencies, irregular breathing, anxiety, and autism. It is well established that impaired protein translation is a characteristic of human Rett Syndrome neurons. However, the underlying molecular mechanism of this phenotype is poorly understood. To study Rett Syndrome pathobiology, my lab investigates the role of individual MeCP2 isoforms in controlling fundamental molecular pathways aiming to explain how MeCP2 mutations cause impaired protein translation in human RTT brain. We use a combination of in vitro and in vivo model systems, including murine and human neural stem cells (self-renewing and differentiated cells into neurons and astrocytes), along with a *MECP2*-deficient transgenic mice, and human post-mortem brain tissues. We show that in addition to disturbed epigenetic mechanisms in Rett Syndrome, major cell signalling pathways upstream of protein translation is impaired in the brain of Rett Syndrome patients and in vitro cellular models of Rett Syndrome. Our recent results provide exciting new insights on how molecular deficiencies at the cellular and molecular levels cause compromised brain function in Rett Syndrome, and other MeCP2-associated brain disorders such as autism.

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IMBALANCE OF THE REDOX STATE IN OPA GENE RELATED DISORDERS: MATHEMATICAL APPROACHES TO DEFINE PATHOGENESIS

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OPA1 mutations cause Dominant Optic Atrophy (DOA), an incurable retinopathy with variable severity and which mechanisms are still unknown. More than 20% of patients will endure a DOA plus syndrome with ataxia, deafness or Parkinsonism. We evidenced oxidative stress in a mouse model of the pathology and aimed to identify the consequences of OPA1 inactivation on redox homeostasis. We monitored the levels of mitochondrial respiration, reactive oxygen species (ROS), anti-oxidant defences and cell death by biochemical and *in situ* approaches using in vitro and in vivo models of OPA1 related disorders. Increased ROS levels were observed in cortices of the murine model OPA1$^{+/-}$ as well as in OPA1 down-regulated cortical neurons. This increase is associated to a decline in mitochondrial respiration and an increase of antioxidant enzyme levels. Upon exogenous oxidative stress OPA1-depleted neurons did not further up-regulated antioxidant defenses. Finally, low levels of antioxidant enzymes were observed in fibroblasts from patients supporting their role as modifier factors. Our study shows: (i) the pro-oxidative state induced by OPA1 loss can be considered as a pathological mechanism (ii) differences in antioxidant defences can contribute to the variability in expressivity and (iii) antioxidant defences can be used as prognostic tools to gauge the severity and the evolution of the disease. Furthermore, our discovery offers a way to model mathematically the dysfunctions of oxidative metabolism in OPA1 gene related disorders. We will present the last results of our algorithm and wet laboratories experiments.

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THE PHENOMENON OF THE RECOVERY OF JOHNNY FAMECHON: A DISCOURSE OF RESILIENCE AND BRAIN PLASTICITY

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This qualitative phenomenological research provides detailed insights into the lived experiences of the recovery of a single subject, that of former World Boxing Champion, John Famechon. John suffered an incapacitating brain injury in August 1991 when a car, estimated to be travelling at 100 kilometres per hour, hit John as he was crossing a road near Warwick Farm in Sydney. Upon discharge from hospital in October 1992, John was under the full-time care of his then fiancée, now wife, Glenys. At that time John was, as Glenys and the media of the time described his condition, as if he had been poured into a wheelchair, unable to walk, talk, or feed himself. The doctors at that time of his discharge advised that this would now be John's life: severely incapacitated, often bed-ridden, unable to fend for himself, barely able to speak; and wheelchair bound for the remainder of his life. In December 1993, John started an unforeseen new therapy involving what are referred to as hard goals and stretch goals, involving highly challenging complex movements and intense cognitive work that focussed on extending what John could do physically and mentally. As a result, from late 1993 John's presenting incapacitated condition changed, and in March 1994 John walked for the first time since the accident. Soon after that he was running. As documented in this thesis, those who knew John and his condition were amazed at the healing and significant improvements from his formerly incapacitated state – much of which was achieved by late 1994. The examination of the literature and phenomenological data indicates the reason that John's condition changed from incapacitated to recovering was due to changes in John's neurological and neuromuscular condition that appear to have mainly stemmed from late 1993, when the new therapy (complex brain-based multi-movement therapy) commenced.

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BACTERIAL PEPTIDOGLYCANS AS NOVEL SIGNALING MOLECULES FROM MICROBIOTA TO BRAIN

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Recent animal studies have revealed that the gut microbiota has much wider effects on host physiology and development than originally believed, including the early-life programming of brain circuits involved in the control of emotions, motor activity, and social behavior. The current challenge is to understand the precise molecular mechanisms mediating the communication between the microbiota and the brain. In this presentation, I will cover new evidence from my laboratory suggesting that the central activation of pattern recognition receptors by bacterial peptidoglycan fragments could be one of the signaling pathways mediating the communication between microbiota and the developing brain.

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UNSCRAMBLING THE EPITOME OF HUMAN MICROGLIAL GENE EXPRESSION DYNAMICS DURING BRAIN DEVELOPMENT FOR POTENTIAL BIOMARKER DISCOVERY

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Microglia, the native sculptors of neuronal circuits perform various crucial functions in brain milieu, including dendritic pruning, immune surveillance, phagocytizing of dead neurons and healthy neural precursors etc. In spite of these attributes, several unresolved queries exist around biomarker discovery relevant to their cellular localization, self-renewing potential and brain developmental dynamics. To ascertain microglial biomarkers in developing brain, we performed high-throughput data mining of microglia gene expression datasets. The analysis revealed a list of 3290 significant genes, out of which we have selected the top 20 dysregulated genes to be the potential markers that can be used for tracking the microglial expression in developing brain. Next, we developed a connectome of these biomarkers with their putative protein interacting partners. This demonstrated strong associations of upregulated genes like DOCK2 with early/mature microglial markers such as SPHK1, CD68 and CD45. To elucidate their anatomical habitance, we deconvoluted the BrainSpan Atlas expression data, which showed high level of expression of majority of candidate genes in microglia-dense regions (Amygdala, Hippocampus, Striatum) in the postnatal brain. Furthermore to decipher their age specific expression in human brain, we constructed a developmental dynamics map (DDM), we again deconvoluted gene expression profiles spanning prenatal to postnatal stages. Interestingly, dynamic regulation of SPHK1, PLD4 along with consistent expression of PTX3, FCAR and KLHL6 were detected. To authenticate these findings and correlate their expressions in vitro, we enforced microglial differentiation to hESC to generate microglia precursors. These microglia precursors could demonstrate expression of PTX3 and SPHK1 as well as other early stage markers, such as CD68, AIF1 (Iba1) post 30 days in vitro. In summary, our study has unraveled critical insights regarding microglial expression dynamics across the brain ages and catalogued a unique set of potential biomarkers those can be further exploited for designing of novel neurotherapeutics.

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D-PENICILLAMINE LOADED NANO LIPID CARRIERS FOR TARGETING GLIOBLASTOMA

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Glioblastoma is a lethal cancer with a median survival of about 15 months. It is a well-known fact that most Glioblastoma's are rich in collagens. Specifically, the mesenchymal subtype of Glioblastoma, which has the worst prognosis, is enriched with numerous extracellular matrix proteins such as collagens. While collagen accumulation can be viewed as a downstream effect of mesenchymal differentiation, it was found that the feed forward increases in collagen, extracellular matrix stiffness and biomechanical influences cause enhanced selfrenewal and mesenchymal transition in glioma stem cells with a consequent increase in treatment resistance of these cells. Further, recent studies have also shown that mechanical stress plays an overarching role in the regulation of TAZ signaling. TAZ is a mediator of mesenchymal differentiation in glioma, which relates with collagen stiffness, promotes mesenchymal transition and gliomagenesis via TAZ signaling. D-penicillamine is a known inhibitor of collagen maturation, which acts by increasing the conversion of insoluble to soluble collagen and disrupting the formation of intermolecular bonds. The therapeutic benefit of D-penicillamine has not been extensively evaluated in Glioblastoma especially in the context of glioma stem cells, which represent faithful models of this disease. Directing therapeutic agents such as D-penicillamine towards deeply embedded brain tumours with biophysical barriers such as blood brain barrier and collagen remains a challenge. Hence, in the present study it is proposed to load D-penicillamine into Nano lipid carriers, which can accumulate passively in tumours via leaky tumour vasculature, caused by focused ultrasound. These triggered release strategies disrupt the blood brain barrier, confirming deep penetration in tumours, and overcome the biophysical constraints, which tackle both the challenges (drug delivery and collagen disruption) simultaneously.

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