magnus Group

# 4TH INTERNATIONAL CONFERENCE ON

# NEUROLOGY AND BRAIN DISORDERS

## September 09-10, 2021

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4TH INTERNATIONAL CONFERENCE ON

# NEUROLOGY AND BRAIN DISORDERS

SEPTEMBER 09-10, 2021

**Theme:**

Advancements and challenges in
Neuroscience & Brain Disorders

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# About MAGNUS GROUP

**Magnus Group (MG)** is initiated to meet a need and to pursue collective goals of the scientific community specifically focusing in the field of Sciences, Engineering and technology to endorse exchanging of the ideas & knowledge which facilitate the collaboration between the scientists, academicians and researchers of same field or interdisciplinary research. Magnus group is proficient in organizing conferences, meetings, seminars and workshops with the ingenious and peerless speakers throughout the world providing you and your organization with broad range of networking opportunities to globalize your research and create your own identity. Our conference and workshops can be well titled as 'ocean of knowledge' where you can sail your boat and pick the pearls, leading the way for innovative research and strategies empowering the strength by overwhelming the complications associated with in the respective fields.

Participation from 90 different countries and 1090 different Universities have contributed to the success of our conferences. Our first International Conference was organized on Oncology and Radiology (ICOR) in Dubai, UAE. Our conferences usually run for 2-3 days completely covering Keynote & Oral sessions along with workshops and poster presentations. Our organization runs promptly with dedicated and proficient employees' managing different conferences throughout the world, without compromising service and quality.

# About INBC 2021

Magnus Group is pleased to invite you to participate in the '4th Edition of International Conference on Neurology and Brain Disorders' (INBC 2021) during September 09-10, 2021 with the theme "Advancements and challenges in Neuroscience & Brain Disorders" following the victorious completion of three editions.

This INBC 2021 is intended to put together leading speakers and experts on a common forum that serves as a means of disseminating peer-reviewed and cutting-edge scientific data that ties the entire field to the neurology community worldwide.

Overall, high-quality information would result in the presentation of the best material on the most relevant topics by speakers with intense discussions, conducted in a pleasant atmosphere needed to facilitate as much networking and learning as possible.

You are assured of discovering the latest developments and breakthroughs that are exclusive to your field of work with its scientific sessions.

# KEYNOTE FORUM

## DAY 1

4TH INTERNATIONAL CONFERENCE ON

## NEUROLOGY AND BRAIN DISORDERS

SEPT 09-10, 2021

INBC 2021

## Ken Ware

Neuro Physics Therapy Institute and Research Centre, Gold Coast, Queensland, Australia

## New ideas and approaches in treatments for chronic and complex neurological conditions

An experimental study involving 5 patients who were diagnosed MND positive and who all presented at moderately advanced stages (e.g. not able to walk unassisted, voice impedance, moderate upper limb, discriminative fine motor impairments), was implemented out of curiosity and despairing patient requests, to determine if a NeuroPhysics Therapy (NPT) intervention would produce any favourable restorations of lost functions, as historically evidenced when applied to other complex neurological pathologies over a 4 day period. Note: This study was extremely sensitive to the often-desperate emotional states of MND patients. To acquire all possible positive outcomes for these patients above and beyond outcomes other forms of therapy or medical interventions have historical accomplished (averages over time), there was a need to look at MND through a modified lens. Typically, NPT adopts a holistic 'Complex Adaptive Systems' approach to all interventions, with high priority given to the effects the patients subjective perceptions of their inner and outer environments have on how they psychophysically respond to these environments. Rogue perceptions manifest rogue responses, with MND being considered here to be the accumulative rogue response to long term series of rogue percepts. The common psychophysical characteristic across all 5 patients was long term hyper activation of their sympathetic nervous systems (SNS) and consequential long term hypervigilant excessive background tone of their musculature system pre their MND diagnosis. These patients confirmed that post their MND diagnosis and grim prognosis, fear, avoidance and anxiety increased exponentially. Motor neurons are the slaves of the sensory system, as in first we sense our inner and outer world and then we respond. From these initial conditions, feedback/feedforward 'recursive dynamics' maintain our conscious experience of a 'non-iterated' seamless flow of information from one moment to the next in space and in time within our inner and outer worlds. However, within the spectrum of MND cases the conscious experience of planned and initiated movements are more likely experienced as iterated and uncertain processes from one moment to the next in space and in time. It is predominantly assumed this is a motor neuron pathology, often based upon the presentation of symptoms and or one-time conduction test acuity.Whereas long term sensory deprivation due to the hypervigilant emotional state of the patient and or from limited exposure to a stimulating environment along with limited activities within complex environments, significantly reduces sensory input and randomized atrophy or desynchronization of sensory assemblies may occur as a result. This in turn reduces stimulation of motor neurons. The hypothesized outcome being randomized motor neuron atrophy or desynchronization of motor neuron assemblies and that MND is more of a sensory system concern rather than a motor system concern. All 5 patients engaged in a 4-day intensive NPT intervention that was focused on down toning the SNS, enabling the parasympathetic nervous system to upregulate the system to ideal allostasis and removing psychophysical restrictions of sensory processing. All patients experienced notable, measurable relaxation of MND symptoms during this 4 day period. This presentation will highlight a selection of these 5 patients pre, during and post NPT, verifying that at least with these 5 cases MND symptoms could be arrested and compromised functions began to be restored in relatively small time scales via a NPT focus on restoring these patientssensory inputs. This is only a small group of MND patients; however, these positive outcomes do indicate that perhaps a qualitative systematic review is needed on what the root causes of MND(s)are, along with a focus on addressing root causes as well as treatments of symptoms.

**Audience Take Away:**

A patient being told that they have been diagnosed as having MND is certainly highly distressing for the patient and their families. It is critically important for this to be an accurate conclusion. Good science is being sure you know what something isn't before conclusions are made about what something is. The excessive nervousness of a patient combined with possible long term psychophysical hypervigilance may allow for misinterpretations of what the data is informing the inquirer of ina one-time conduction test analysis. The audience will be introduced to novel easy to apply assessment procedures that logically make sense in the case of assessments for possible MND. Potentially these novel assessment techniques would eliminate or reduce the consequences of an incorrect MND diagnosis and possibly challenge the acuity of an existing MND diagnosis that was based upon presentation of symptoms and in cases where there has been only one time conduction testing performed.

**Biography**

Ken Ware was founder of Neurotricional Sciences Pty Ltd and NeuroPhysics Therapy and Research and he had been in private practice for almost 30 years, while doing independent and collaborative research. He also presented unique research at 10 major International Science Conferences including neuroscience, Physics, Psychology and Life Sciences, which covers a very broad scientific audience. He is Former Mr. Universe 1994, National powerlifting and Body building champion and record holder. He had published relative publications in 'Frontiers in Clinical Physiology' - 'World Journal of Neuroscience' – 'World Journal of Cardiovascular diseases'. He is recipient of Her Majesty, Queen Elizabeth's 'Australian Sports Medal' - in 2000, in recognition for personal contributions to the development of the Australian Sporting Culture.

## Svyatoslav Medvedev

Institute of the Human Brain RAS, Russian Federation

## Physiological correlates of the altered states of consciousness at different types of meditation at skilled practitioners

Importance of physiological researches of the altered states of consciousness (ASC) arising at adaptation of the person to performance of a certain activity is known (for example illusions of monotony in pilots, cosmonauts, operators of complex systems). This importance follows at least from the fact that the majority of accidents are caused by a human factor Accidents may be significantly reduced if physiological mechanisms of self-government and self-stabilization are found. However, researches on ordinary subjects are complicated by the fact that it is difficult to choose uniform group with the identical level and characteristic of these ASC. Besides, it is difficult for unprepared subjects to intentionally enter a set of desired states and to maintain them throughout a physiological experiment. In this work, we tried to reduce the influence of these factors by a special choice of subjects, and a special way of achievement of ASC. For our research, skilled monk-meditators from Buddhist monasteries in southern India were selected. The structure of research was approved by the ethical commission of N.P. Bekhtereva Institute of the Human Brain of the Russian Academy of Sciences. Two groups were created. The first group of 70 skilled practitioner monks trained at the Buddhist universities was chosen by the special commission of heads of the universities. They selected the participants having the most identical levels of experience and able to maintain the meditation levels set in the course of the research. All the subjects had medical and psychological examination before the research, which helped select the most uniform group. The second group was formed of thirty specially selected meditator having the highest level of meditative abilities from 12000 monks of monasteries. This group allowed to estimate the maximum level of influence of meditation on the physiological changes accompanying changes to the level of consciousness. The research consisted of the background record of silence, to different types of meditation. Thus, two opposite ASC were modelled. Thus, two opposite ASC were modelled. Also, ERP research was conducted using mismatch negativity paradigm as indicator of unconscious perception of signals from the outside world. The received results showed that meditation is the good tool for a research of the physiological processes forming ASC. Different levels of physiological processes on which meditation has an impact are shown and it is investigated what changes happened.

### Biography

Professor Svyatoslav Medvedev, full member of the Russian Academy of Sciences, 1990-2017 - director of the Institute of Human Brain. Area of research: human neurophysiology, investigations of neurophysiological correlates and basic mechanisms of organization and functioning of the cerebral system maintaining mental activity, study of the human brain neuronal activity and depth evoked potentials related to visual perception, PET studies of higher nervous activity and different kinds of pathlogy, studies of brain maintenance of attention, thinking, creativity, speech, emotions; neurooncology, trauma, neurodegenerative disorders, ishemia stroke etc. He has published more than 100 articles and 6 books.

## Alexies Dagnino

Universidad de Valparaíso, Valparaíso, Chile

## Resilience to stress and neuroinflammation

Resilience to stress is the ability to quickly adapt to adversity. There is evidence that exposure to prolonged stress triggers neuroinflammation what produces individual differences in stress vulnerability. However, the relationship between stress resilience, neuroinflammation, and depressive-like behaviours remains unknown. The aim of this study was to analyse the long-term effects of social defeat stress (SDS) on neuroinflammation in the hippocampus and depressive-like behaviours. Male rats were subjected to the SDS paradigm. Social interaction was analysed 1 and 2 weeks after ending the SDS to determine which animals were susceptible or resilient to stress. Neuroinflammation markers glial fibrillary acidic protein, ionized calcium-binding adaptor molecule 1, and elevated membrane permeability in astrocytes and microglia, as well as depressive-like behaviours in the sucrose preference test and forced swim test were evaluated in all rats. One week after SDS, resilient rats increased their sucrose preference, and time spent in the floating behaviour decreased in the forced swim test compared to susceptible rats. Surprisingly, resilient rats became susceptible to stress, and presented neuroinflammation 2 weeks after SDS. These findings suggest that SDS-induced hippocampal neuroinflammation persists in post-stress stages, regardless of whether rats were initially resilient or not. Our study opens a new approach to understanding the neurobiology of stress resilience.(Bravo-Tobar et. al., 2021. J Neurosci Res, doi: 10.1002/jnr.24902).

**Audience Take Away:**

- To stress and their relation with neuropsychiatric disorders
- It is important that professionals who work in mental health have knowledge about the neurobiological effects of stress on the brain

**Biography**

Dr. Dagnino has completed his PhD in biomedical sciences from University of Chile (2003) and completed his undergraduate degree in Biochemistry at the Austral University of Chile (1999). Afterward he went to USA to complete two research stays at the University of Texas at Dallas (Atzori Lab, 2008) and Stanford University (Sapolsky Lab, 2009). Currently, he is working as Full professor in University of Valparaíso, Chile. His research interest includes neurobiology of stress resilience and neuropsychiatric disorders.

SPEAKERS

DAY 1

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**Raffaele Pilla**

St.John of God Hospital - Fatebenefratelli, Benevento, Italy

## Therapeutic ketosis and the broad field of applications for the ketogenic diet: Ketoneester applications & clinical updates

It has been recently shown that nutritional ketosisis effective against seizure disorders and various acute/chronic neurological disorders. Physiologically, glucose isthe primary metabolic fuel for cells. However, many neurodegenerative disorders have been associated with impaired glucose transport/metabolism and with mitochondrialdys function, such as Alzheimer's/Parkinson's disease, general seizure disorders, and traumatic brain injury. Ketone bodies and tricarboxylicacid cycle intermediate are present alternative fuels for the brain and can bypass the rate-limiting steps associated with impaired neuronalglucose metabolism. Therefore, therapeutic ketosis can be considered as ametabolic therapy by providing alternative energy substrates. It has been estimated that the brain derives over 60% of its total energy from ketones when glucose availability is limited. In fact, after prolonged periods of fasting or ketogenic diet (KD), the body utilizes energy obtained from free fatty acids (FFAs) released from adipose tissue. Because the brain is unable to derive significant energy from FFAs, hepatic ketogenesis converts FFAs into ketone bodies-hydroxy buty rate (BHB) and acetoacetate( AcAc)-while a percentage of AcAc spontaneously decarboxylates to acetone. Large quantities of ketone bodies accumulate in the blood through this mechanism. This represents a state of normal physiological ketosis and can be therapeutic. Ketone bodies are transported across the blood-brain barrier by monocarboxylic acid transporters to fuel brain function. Starvation or nutritional ketosis is an essential survival mechanism that ensures metabolic flexibility during prolonged fasting or lack of carbohydrate ingestion. Therapeutic ketosis leads to metabolic adaptations that may improve brain metabolism, restore mitochondrial ATP production, decrease reactive oxygen species production, reduce inflammation, and increase neurotrophic factors' function. It has been shown that KD mimics the effects of fasting and the lack of glucose/insulin signaling, promoting a metabolic shift towards fatty acid utilization. In this work, the author reports a number of successful case reports treated through metabolic ketosis.
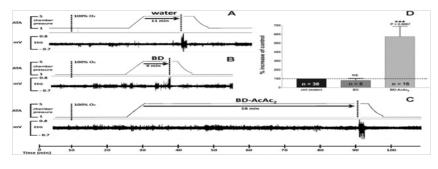


**Figure 1:** Ketone Ester significantly increased resistance against Central Nervous System Oxygen Toxicity seizures (D'Agostino D.P.et al., 2013 Am J PhysiolRegulIntegr Comp Physiol. 304(10):829-36.

### Biography

Raffaele Pilla, Pharm.D., Ph.D., Doctor Europaeus, received his Master's degree in Pharmacy at G.d'Annunzio University in Chieti-Pescara, Italy in 2005, where he also served internships at the Cell Physiology Laboratory and Molecular Biology Laboratory. Prior, he was an Erasmus Student at Faculté de Pharmacie de Reims in Reims, France. He received his Doctor Europaeus in 2010 from Pitié-Salpétrière Institutein Paris, France. Also in 2010, he received his Ph.D. in Biochemistry, Physiology, and Pathology of Muscle at G. d'Annunzio University in Chieti-Pescara, Italy. He was hired as a Postdoctoral Scholar in the Department of Pharmacology and Physiology at the University of South Florida in Tampa, on two research grants funded by the Office of Naval Research (US Navy)and Divers' Alert Network. He has written and lectured widely worldwide. He has been involved in ongoing research at the University of South Florida with the use of ketone esters.

**Vanessa Douet-Vannucci[1,2]\*, Theo Marchand[1], Justine Fieve[1], Pelayo Mencos[1], Melody Zira[1], Olivier Oullier[1] and Pascal Staccini[2]**

[1]Department or R&D, Crocosgodigital, Marseille, France
[2]URE RETINES, University of Cote d'Azur, Nice, France

## Integration of digital phenotyping with clinical (endo)phenotypes in children with neurodevelopmental disorders

Neurodevelopmental disorders (NDD) such as attention deficit hyperactivity disorder(ADHD), or social communication disorder, although most commonly studied in childhood, have complex clinical (endo)phenotypes with an important social context and can be lifelong conditions. Furthermore, strong overlaps across NDD phenotypes make both group and distinction of each disorder difficult in real-life practice. Digital phenotyping in natural environment represents a new approach aimed at measuring human behavior and may, combined with clinical (endo) phenotypes, enhance capability and sensitivity in early identification, diagnosis and management of mental health conditions. Moreover, such a combined approach may easily allow clinicians to perform a more personalized and patient-tailored diagnostic and therapeutic approach. Here, we investigate how digital phenotyping integrated with clinical (endo) phenotypes constitutes a new method for identification and pre-diagnosis of NDD in children aged 7 to 12 years. A total of 100 children aged 7 to 12 years are evaluated through a new digital neuro-battery designed to assess cognitive and psychopathological domains (digital tests and questionnaires) as well as neurobehavioral functioning (eye- & digit-tracking and EEG). We are examining a large set of features within these multi-modal data using machine learning algorithms to identify prominent predictive variables to achieve dimensionality reduction and provide finer and more sensitive classification of NDD. Our preliminary results are encouraging for ongoing development of our digital screening tools that can be used in ecological environments on touch-screen tablets with cost-effective behavioral trackers. Larger paediatric populations can then be reached to improve early detection, prevention and ultimately intervention.

**Audience Take Away:**

- What are neurodevelopmental disorders (NDD)
- How can digital phenotypingbe combined with clinical endophenotypes in NDD
- How digital detection and therapies can help clinicians and researchers in real-life practice
- How digital diagnosis and therapies may favor a patient-tailored approach as well as help children being fully involved in the health care management

**Biography**

Vanessa Douet has pursued an international career on multimodal research projects (Japan and USA) after her PhD, developing and managing International research projects by combining expertise from different worlds: life sciences, digital technologies and infotech (30+ scientific articles). She has devoted +10 years on brain development in children through several research studies (PING or ABCD-Studies). Today, she is adjunct professor at URE-RETINES at the Côte-d'AZUR University and supervises R&D and clinical trials as CSO at Crocogodigital to investigate how a combined digital phenotyping & clinical approach opens-up new ways to early-risk diagnosis, prevention, and more-personalized interventions for mental health.

**Wen-Chang Li\*, Xiao-Yue Zhu, Emma Ritson**

School of Psychology and Neuroscience, the University of St Andrews, St Mary's Quad, South Street, StAndrews, Fife, KY16 9JP, UK

## Mechanosensory stimulation evokes acute concussion-like behaviour by activating GIRKs coupled to muscarinic receptors in a simple vertebrate

Most vertebrates show concussion responses when their heads are hit suddenly by heavy objects. Previous studies have focused on the direct physical injuries to the neural tissue caused by the concussive blow. We study a similar behaviour in a simple vertebrate, the Xenopus Laevis tadpole. We find that concussion-like behaviour can be reliably induced by the mechanosensory stimulation of the head skin without direct physical impacts on the brain. Head skin stimulation activates a cholinergic pathway which then opens G-protein coupled inward-rectifying potassium channels (GIRKs) via M2 muscarinic receptors. These inhibit brainstem neurons critical for the initiation and maintenance of swimming for up to minutes and can explain many features commonly observed immediately after concussion. We propose that some acute symptoms of concussion in vertebrates can be explained by the opening of GIRKs following mechanosensory stimulation to the head.

**Audience Take Away:**

- These results provide a potential explanation why concussion in vertebrates often recovers spontaneously without sustaining clear physical injury to the brain and some acute symptoms of concussion can be a neurophysiological response to specific sensory stimulation
- The head skin stimulation activates some cholinergic neurons in the brainstem to transiently inhibit motor behavior
- Some anti-cholinergic medicine could potentially be used for reducing acute concussion responses

**Biography**

Dr. Li joined Prof Alan Roberts' lab in the University of Bristol to study motor control using Xenopus tadpoles after obtaining his PhD in the Institute of Biophysics in Beijing. He was awarded a Royal Society University Research Fellowship in 2006 and moved to the University of St Andrews, where he became a reader in 2015. He has about 40 publications on the neurophysiology of motor control, mostly on how the spinal and hindbrain circuits control rhythmic movements in tadpoles.

## Gaurav Jha[1*], Sabeen Azhar[2]

[1]Neuroscience, Barking, Havering and Redbridge University Hospitals NHS Trust, Romford, United Kingdom
[2]Acute Medicine, Barking, Havering and Redbridge University Hospitals NHS Trust, Romford, United Kingdom

## Bilateral Facial Palsy: A case study to exceedingly rare and difficult diagnosis

Unilateral facial palsy is a fairly common diagnosis with an incidence of 20-25 per 100,000 population, whereas Bilateral facial nerve palsy (FNP) is exceedingly rare and only accounts for less than 2% (0.3%- 2%) all facial palsy cases. Facial paralysis depicts the result of a diverse array of disorders and heterogeneous aetiologies and thus represents a diagnostic challenge. Bilateral facial palsy entails several etiologic causes, most of which are secondary to underlying medical conditions. The list is exhaustive, leading to a diagnostic challenge, and hence clinicians should be aware of ruling out a wide range of differential diagnosis, some of which are potentially fatal. We report a case of a previously healthy 32-year-old male who presented to the emergency department with simultaneous bilateral facial weakness, slurring of speech and numbness of both lips and cheek, which could not be attributed to any specific aetiology after a repeated CT scan, MRI brain with contrast, lumbar puncture and an EMG study and, hence presented a diagnostic dilemma. In this paper, we emphasise the need of investigating the full spectrum of possible diagnosis in all instances presenting with bilateral facial nerve palsy. These patients require hospitalisation and immediate laboratory and radiographic testing to determine the underlying aetiology and if necessary, specific additional treatment.

**Audience Take Away:**

- The need of a thorough history taking, physical examination, and review of past records in identifying and treating individuals like these cannot be overstated.
- Bilateral facial paralysis has a variety of causes, and a full understanding of this condition will aid the clinician in making an expedient and timely diagnosis.

**Biography**

Dr Gaurav Jha, graduated as MBBS from Jinzhou Medical University, 2017. He was the President and Co-founder of the scientific club "DaVinci Medical Hub" for 4 years. Dr Jha was the Editor-In-Chief for the Medical Journal 'Acta Medica DaVinci' and is a contributing author of the QRS FMGE Fire Aid Textbook specially designed for foreign medical graduates. He has always been a scholarship holder throughout his medical school and was also awarded as an Excellent Student Award in 2018. He was also honored by the Chinese Education Ministry as an "Outstanding International Student Scholarship" in the year 2016. He has many accolades to his name and currently holds the position of Educational lead in Surgical Society of International Doctors, UK.

**Wilfried Schupp**

Department for Neurology/Neuropsychology, Fachklinik Herzogenaurach, Friedrich-Alexander-University Erlangen-Nuremberg, Germany

## Affective disorders after stroke and traumatic brain injury in the longterm perspective

Affective disorders are frequent after acquired brain lesions like stroke and traumatic brain injury (TBI). Depression had been more often been studied than anxiety although incidence and prevalence of both is nearly equal, and a high comorbidity between both exists. Most research has been done in the (post)acute phase of stroke and TBI, whereas their incidence and prevalence increase in the long term run. These affective disorders are most often present for a period of time, are much seldomly persisting the whole time. There is no good correlation with type and severity of disablement, cognitive disturbances are more under discussion than motor disabilities. No indicators could be found for the risk if and when a person will suffer from such affective disorders. Therefore, repeated mental examinations are a must in the long term medical care. In stroke patients differences of prevalence for anxiety and depression exist between countries in the five years perspective, why remains still unclear. But in every country the percentage of untreated patients is high, although pharmaco- and/or psychotherapy are effective. In TBI patients younger people has a higher lifelong risk for mental, especially affective disorders with difference between genders in subtypes. Addiction or aggression become relevant mental comorbidities in TBI survivors. If they also have a higher risk for cerebrodegenerative diseases, is under research.

**Audience Take Away:**

- The audience will get a review on epidemiological data of affective disorders in stroke and TBI survivors in the long term course from foreign and own studies
- The audience will learn which examinations are important in the long term care of stroke and TBI survivors and why These knowledgesmay help to improve the long term perspective of mental comorbidity, health related quality of life (HRQoL), burden of disease, and burden of caregivers in such patients. They can help to develop and provide special services for the stroke and TBI patients in different countries. These facts may be important in designing further studies on the long term impact of such acquired brain lesions

**Biography**

Dr. Wilfried Schupp did residencies in Psychiatry at Max Planck Institute for Psychiatry after his Medical Degree at the University of Ulm Munich, and in Neurology at the Clinic for Neurology, of Ludwig Maximilian University (LMU), Munich. At the same time, he started his work on neuro rehabilitation together with the Clinic and Institute for Physical and Rehabilitation Medicine of LMU Munich. Dr. Schupp certified as neurologist, psychiatrist, physiatrist, and geriatrist, and for social medicine. Since 1996 he is presently Medical Head of the Department for Neurology and Neuropsychology, Fachklinik Herzogenaurach, a privately owned rehabilitation clinic affiliated with FAU Erlangen-Nuremberg. His scientific work deals with clinical approaches to neurorehabilitative problems in stroke, TBI, and others, even rare neuromuscular diseases. He is a member in many national and European advisory boards and is listed in Editorial boards of national and European journals in the field of (neuro) rehabilitation and social medicine.

## Vir B. Singh[1*],Irfan A. khan[1], Arthur H.Worrad[1], Meera V.Singh[2], Sanjay B.Maggirwar[3]

[1]Department of Basic and Clinical Sciences, Albany College of Pharmacy and Health Sciences, Albany, NY12208,USA.
[2]School of Medicine and Dentistry, University of Rochester Medical Center, Rochester, NY14642, USA.
[3]Department of Microbiology, Immunology and Tropical Medicine, The George Washington University, Washington, DC20052, USA.

## Human Immunod efficiency Virus 1T atexerts its neuro toxic effects by down regulating Sonic Hedgehog signaling

HIV-1 can alter the expression of tight junction proteins by down regulating Sonic hedgehog (Shh) signaling, there by disrupting BBB integrity. In this study, we employed a conditional, CNS specific, transgenic murine model to investigate if Tat, a known neuro toxic HIV protein, is responsible for dys regulation of Shh signaling. Results indicate that Tat+micee xhibit reduce dexpression of Shh,Gli-1and Smoothened proteins. Tat+mice also expres slower level so ftight junction protein Claudin-5 and higher levels of adhesion protein Vcam-1.  As a result of these BBB alterations significantly higher numbers of leukocytes in filtrate into the CNS of Tat+mice as compared to Tat-mice. Administration of Smoothened agonist (SAG), a Shh mimetic, significantly reduced the number of brain-infiltrating leukocytes (BILs) in Tat+ mice. Further, our in vitro experiments using human brain microvascular endothelial cells suggest that pharmacological induction of Shh signaling can rescue detrimental effects of Tat on endothelial function by inducing the expression of junctional proteins and by decreasing the levels of in flammatorycy tokines/ chemokines such as CCL2, IL-1  and IL-6. Overall, this study identifies a novel mechanism by which HIV-1 Tat exerts its neurotoxic effects as well as underscores the neuroprotective role of Shh signaling in adult brain. Findings from this study can be used to devise potential the rapeutic approaches to all eviate HIV as sociated neuro path ogenesis.

**Biography**

Dr. Singh is a virologist with special interest in molecular mechanisms that are involved in HIV replication and neuropathogenesis. He received his PhD in Biotechnology from SavitribaiPhule Pune University (India) in 2009. Subsequently, during his postdoctoral training at Roswell Park Cancer Institute (RPCI), he studied the phenomenon of genomic imprinting and investigated the mechanism of establishment of maternal methylation at KvDMR1 Imprinting control region (ICR). This study proposed a potential mechanism of loss of methylation at KvDMR1 that results in a developmental disorder known as Beckwith Wiedemann syndrome. Further, he moved to University of Rochester Medical Center, Rochester, NY, where he worked for over 7 years, first as a postdoc and eventually promoted to Research Assistant Professor. During this time, he investigated the molecular mechanisms involved in HIV associated neurological disorder. Currently, Dr. Singh is employed as Assistant Professor of Microbiology at Albany College of Pharmacy and Health Sciences. Collectively, he has acquired an extensively diverse experience in investigating the molecular mechanisms implicated in various diseases including anemia, developmental growth syndromes predisposed to cancer, and HIV pathogenesis. As an independent investigator, Dr. Singh's research interests include understanding the basic molecular mechanisms involved in- i) HIV associated neurological disorder, ii) HIV latency, and iii) Viral infection induced developmental defects.

**M. DUBOIS Jeremy[1*], Dr GOUTTE Nathalie[1], Pr SAMUEL Didier[1,2]**

[1]Joint Research Unit, INSERM UMR 1193, Villejuif, France
[2]Medicine faculty, Paris-Sud University, Le Kremlin-Bicêtre, France

## Care course assessment nationwide exhaustive study: Example of effectiveness assessment of neurodegenerative disease rehabilitation in France

Care course improves interprofessional coordination, care quality and decreases costs. Système National des Données de Santé (SNDS), a medical administrative big data base, allows us to rebuild and study care course for hospital care and home care. We will take the example of rehabilitation care of neurodegenerative disease (Parkinson's disease for this presentation). Evolutive and incurable characteristic of these disease simplifies their study. Furthermore, their different evolutions, medical care and life expectancy allow us to compare and to study them on different situations. Moreover, the rehabilitation effectiveness on these disease are demonstrated in clinicals studies but not in pragmatic nationwide exhaustive study. The main objective of this trial is to study effectiveness of real rehabilitation care course of neurodegenerative disease. The secondary objectives are to define care course's evaluation standards and to study the protective character of rehabilitation on the handicap evolution and the healthcare consumption. Firstly, we are making a systematic literature review that defines precisely the diseases' care courses and their evaluation standards (25 articles on 38,473). Secondly, we will make and epidemiologic study on a retrospective cohort from a generalist sample of SNDS beneficiaries (CIM-10 code, beneficiary of long disease's program, drugs tracers). It will describe nationwide exhaustive population, by measuring the diseases' frequency to measure diseases', quantity, quality and frequency of healthcare consumption. Finally, we will rebuild the patients' care course and will study the rehabilitation influences factors on the handicap level, survival indicators and healthcare consumption. This nationwide exhaustive study will allow to examine impact on patients with Parkinson disease rehabilitation care course and its burden of health system. On this presentation we will present the context, methodology to build the care pathway of Parkinson's disease from big data and early results.

**Audience Take Away:**

- This presentation will explain the care pathway of Parkinson's disease in France
- We explain the rule of rehabilitation in the treatment of the Parkinson's disease
- We will explain a new epidemiologic's methodology to assess the care pathway applied to the rehabilitation of the Parkinson's disease

**Biography**

M Dubois studied physiotherapy at the National School of Physiotherapy and Rehabilitation, France and graduated as PT in 2016. After he worked in neurology service of University and Hospital Center in Paris as PT. Then he worked in city office until now. In parallel he studied public health, methodology and biostatistics in Paris-Sud University, France and graduated as MSc in 2018. He teach neuro-rehabilitation, methodology and statistics in Physiotherapy schools. Then 2020, he is PhD student in Paris-Sud University, France in epidemiology. His work is about the efficacy assessment in the neurodegenerative's disease rehabilitation from big data.

**Lama Saad El-Din Mahmoud**

October 6 University, Egypt

## Effect of scapular dyskinesia on the scapular balance angle and upper extremity sensorimotor function in stroke patients with spasticity

**Background:** Post-stroke scapular dyskinesia is a predisposing factor for the affection of motor and somatosensory functions of the hemiparetic upper extremity.

**Objective:** The purpose of the study was to investigate the effect of scapular dyskinesia on the scapular balance angle and upper extremity sensorimotor function in stroke patients with spasticity.

**Subjects and Methods:** Sixty patients with spasticity post-stroke participated in this study. The patients were assigned to one of the two groups as determined by the lateral scapular slide test (LSST) using the palpation meter (PALM); group A with scapular dyskinesia and group B stroke patients without scapular dyskinesia. The scapular position was determined by a measurement of scapular balance angle (SBA), and the upper extremity sensorimotor function was evaluated using Fugl-Meyer Assessment upper extremity (FMAUE) scale. The scapular balance angle and Fugl-Meyer upper extremity scores were compared between groups.

**Results:** There was a significant increase in the scapular balance angle of group A compared with that of group B ($p < 0.001$). Also, there was a significant decrease in sensory and motor functions of group A as measured by Fugl-Meyer upper extremity compared with that of group B ($p < 0.001$).

**Conclusion:** Scapular dyskinesia had a significant effect on the scapular balance angle and upper extremity sensorimotor function in stroke patients with spasticity. Management of scapular dyskinesia should be emphasized in the rehabilitation program for stroke patients with spasticity.

### Biography

Lecturer of physical Therapy For Neuromuscular disorders & its surgery, faculty of physical therapy, October 6 university and Neurology and Neurosurgery Consultant for Neuro-Rehabilitation October 6 University Hospital  and Ph.D. degree, Department of Physical Therapy for Neuromuscular Disorders and its Surgery, faculty of physical therapy, Cairo university

**Hussein Akel\*, Gabor Katona, Ildiko Csoka**

Institute of Pharmaceutical Technology and Regulatory Affairs, Faculty of Pharmacy, University of Szeged, Szeged, Hungary

## A Glimmer of hope from insulin NPs in vitro and ex vivo studies for the successful insulin delivery to the brain

Drug delivery to the brain is still forming the major drawback facing the successful management of neurodegenerative disorders, counting Alzheimer's disease (AD) that represents most dementia cases in the elderly population worldwide. Nasal application with the aim of direct nose-to-brain delivery grants the drug molecules the ability to avoid the blood-brain barrier (BBB), which forms the primary obstacle facing effective brain delivery. The novel nanotechnologies are expected to develop innovative targeted drug-delivery systems. Solid lipid nanoparticles (SLNs) and polylactic-co-glycolic acid nanoparticles (PLGA NPs) presented themselves as competitive nanosystems for the brain delivery of therapeutics following the intranasal route. Insufficient brain insulin has been involved in AD progress, and the intranasal insulin for this purpose demonstrated promising results. In this research work, insulin was formulated into SLNs and PLGA NPs forms and chitosan-coated SLNs and PLGA NPs. The nanoformulations stability was assessed, then the in vitro and ex-vivo comparisons between the native insulin and insulin NPs were further investigated. Physiochemical results spotted the stability of encapsulated insulin in both the nanoformulations and the compatibility between the used materials. The *Invitro* results showed the superiority of nanoparticles in enhancing insulin dissolution, mucoadhesion, and permeation behavior and a positive role of chitosan-coating in the further enhancement. Moreover, ex-vivo investigations confirmed the in vitro results and showed the positive part of these nanosystems. Thus, incorporating insulin into chitosan-coated NPs holds excellent hope for successfully delivering insulin to the brain.

**Audience Take Away:**

- In this presentation, the light will be shed on the ability of nanoformulations and chitosan-coating as promising strategies to boost intranasal insulin ability to reach the brain, the expected fate of the intranasally applied nanoparticles for the brain targeting
- The results presented in this work could be interesting for the researchers in the neurodegenerative diseases since it provides evidence about the successful brain delivery of insulin. This may be employed in managing Alzheimer's disease depending on the fact of insulin-Alzheimer's relationship. Furthermore, these developed nanoparticulate systems could be potential for any other AD medicine

**Biography**

Dr. Hussein Akel studied Pharmacy at Tishreen University, Syria, and graduated in 2016 as an MSc holder. After two years of working as an assistant lecturer, he joined the research group of Prof. Ildiko Csoka at the Institute of Pharmaceutical Technology and Regulatory Affairs, Faculty of Pharmacy, University of Szeged, Szeged, Hungary. He is expected to receive his PhD degree in 2021.

**Alba Liliana Avila Suarez[1*] Maria Eugenia Riveros[2], Fernando Ezquer[3]**

[1]PhD (c) in Science and Innovation in Medicine, Universidad del Desarrollo, Santiago, Chile
[2]Center of Cellular and Integrative Physiology, Universidad del Desarrollo, Santiago, Chile
[3]Center for Regenerative Medicine, Universidad del Desarrollo, Santiago, Chile

## Intranasal administration of secretome derived from preconditioned human mesenchymal stem cells reduces the depressive behaviour in a rat model of chronic unpredictable mild stress

Chronic stress exposure can induce in susceptible individuals maladaptive physiological responses, that can yield to changes in their emotional state, motivation and mental focus among others. Eventually these symptoms can worsen and became the central symptoms of the mayor depressive disorder (MDD), depressed mood and anhedonia. MDD is a very disabling disorder, that affects people form a young age and has a remarkably high worldwide incidence, that is projected to further increase in the future, and therefore MDD is a major global health concern. Psychotherapy and or pharmacological treatment are used to treat this disorder. Nevertheless, not every individual responds to pharmacotherapy and the remission rate is only of a 65%. Therefore, new therapeutically approaches are urgently needed. Unfortunately, given the multifactorial nature of MDD, its precise aetiology and underlining mechanisms are currently not completely understood. Neuroinflammation an reduced neurogenesis appear to play an important role in MDD, however, available antidepressants are focused on increasing monoamine transmission. Therapeutically approaches directed to reduce the chronic inflammatory state and potentiate neurogenesis are promising treatment alternatives. Mesenchymal stem cells (MSC), known as the "guardians of inflammation" are an interesting alternative. When these cells are preconditioned with proinflammatory signals, they produce a variety of therapeutic molecules, like antinflammatory cytokines, and neuroprotector factors. These secreted molecules and exosomes are known as secretome and it can be obtained by growing the MSC in culture. Secretome can be administrated in a non-invasive way, intranasally, enabling the secretome to effectively reach the brain. Animal models are an indispensable tool to investigate MDD pathophysiology and for testing potential pharma to treat MDD. In this study we used male rats submitted to a chronic mild unpredictable stress (CUMS) protocol for 8 weeks. In which a set of different stressors were administrated in a random (unpredictable) order, while keeping and age matched non-stressed group of rats as control. At week 8 animals in this group were divided in two groups. One group received at intervals of five days 4 doses of secretome (25ug protein obtained from 1x106 preconditioned human MSC (hMSC)) and the other group received saline solution (vehicle) following the same administration scheme. During the 3 weeks of treatment CUMS protocol was maintained. At this time (11 weeks) depressive like behaviours were evaluated using a test battery that included: sucrose preference test to assess anhedonic behaviour, female urine sniffing test (FUST) to evaluate sexual motivation, the coat state as a measure of self-care and open field test used to evaluate anxiety. A score was assigned to quantify the magnitude of change in each of these parameters and to determine an overall depression score. We observed that animals treated with secretome derived from preconditioned hMSC showed a significant reduction in their depression score compared to vehicle treated animals. Secretome administration reverted anhedonic, spathic an anxious behaviour, suggesting that this biodrug has an antidepressant potential. These preclinical data may constitute the basis for a future clinic testing of a new antidepressive bio drug for the treatment of MDD.

**Audience Take Away:**

- A novel approach for the non-invasive treatment of depression with a cellular derivative of preconditioned mesenchymal stem cells, tackling directly the neuroinflammatory condition underlining the depressive disorder
- An alternative, fast and effective route of administration for pharmacological treatments directed to the CNS, that allows to reduce the dose of administration and limit the systemic levels of the drug
- Audience will also learn about possible future pharmacotherapy for depression that could represent an alternative for non-responders patients or patients that do not adhere to the treatment because of side effects as reduced libido, since we are showing preclinical data backing a positive effect of the drug on the reduced libido of the depressed like animals

**Biography**

Alba Liliana Avila Suarez is a PHD candidate studied microbiology at the Pontifical Xavierian University of Bogota, Colombia and graduated as MSc in 2002. She then joined the research group of Prof. Fernando Ezquer at the Regenerative Medicine Center of the Universidad del Desarrollo de Chile to carry out his doctoral thesis.

POSTERS

DAY 1

4TH INTERNATIONAL CONFERENCE ON

# NEUROLOGY AND BRAIN DISORDERS

SEPT 09-10, 2021

INBC 2021

### Maryam Farhang

Departamento de Psiquiatría y Salud Mental, Hospital Clínico Universidad de Chile, Santiago, Chile

## A pilot study to examine the impact of Yoga-based mindfulness intervention (YBM) for older people with mild cognitive impairment (MCI)

**Background:** Research shows that mindfulness and yoga have been associated with a range of positive outcomes in older adults with mild cognitive impairment (MCI). The aims of the study was to adapt and pilot a mindfulness-based yoga intervention (YBM) for older adults with MCI.

**Method:** This is a pilot study with pre-post intervention design. The sample consisted of 12 older adults with a diagnosis of any types of mild cognitive impairment that has been received yoga-based mindfulness intervention (YBM). The criterion for Inclusion were (1) having 60 years and above, (2) people who have MCI diagnosed by a neurologist, psychiatrist, geriatrician, the neuropsychologist and through evaluation by saying that they had memory problems confirmed by an informant but without problems in their activities of daily living, 2) with a score < 21 at the Montreal Cognitive Assessment (MoCA< 21), According to the level of education 2 points has been considered for patients with less than 8 years of schooling and one point for patients with 8-12 years of schooling , (3) and without dementia according to the criteria of the Clinical Dementia Rating Scale (CDR) a score of 0.5 (4) a score of 19–23 points on MMSE, (5) a score of 2 or more on Ad8 (showing that cognitive impairment is likely). The Exclusion criteria were (1) people who did yoga and/or mindfulness within the last 6 months (2) the presence of a psychiatric clinical diagnosis; or neurological/cerebrovascular condition; (3) presence of a disabling physical illness and (4) presence of a disability that limits and / or impede communication such as major impairments in eyesight, hearing or upper limb motor movements; or other health problem that would interfere with regular yoga and mindfulness practice. Cognitive and physical function, psychological well-being, depressive and anxiety symptomatology have been evaluated before the implementation of the program and at post test through standardized instruments. Participants have been assigned to the Yoga based mindfulness intervention (YBM), for an hours, once weekly for a total of 8 weeks, which has been customized to the needs, health outcomes and level of understanding of MCI participants.

**Results:** The YBM intervention protocol based on video recording has been adapted. YBM intervention reduced anxiety symptoms at post intervention, which wasn't statistically significant. There was a significant difference from pre- to post-YBM intervention on the total Technology based daily life activities score (T-ADL) and the recreation subscale. Participants had higher level of cognitive impairment from baseline to post test intervention which wasn't significant. Also, scores on both Geriatric depression scale (GDS) and Mindfulness awareness attention scale (MASS) as well as level of well-being were not significantly influenced by the YBM.

**Discussion:** The results support the feasibility of a YBM Intervention for the management of anxiety symptoms in older adults with MCI. These preliminary findings suggest the potential of mindfulness intervention in improving anxiety symptoms implicated a risk factors for MCI . These data are valuable inputs for a future randomized clinical trial.

**Biography**

Maryam Farhang is psychologist interested in positive aging. She aims to design and evaluate psychosocial interventions based on mind-body medicine such as yoga and mindfulness and providing support for developing cognitive and physical function of elderly people with cognitive impairment that can be incorporated into Chilean health services and develop proof about how to support a successful aging. She is working as a research professor at Universidad de Las Américas, Also she have been a postdoc researcher at Department of Psychiatry and mental health, Hospital Clínico- Universidad de Chile as well as Millennium Institute for Research on Depression and Personality (Midap)- Universidad de Católica de Chile, with funding that I obtained from a CONICYT Grant (Fondecyt Postdoctorado nº: 3190275) to continue developing her research on "Adaptation, implementation and evaluation of the effectiveness of yoga based mindfulness intervention (YBM) versus Psycho-educational program for older people with MCI".

### Marta Czajkowska

Department of Clinical Physiology, Faculty of Veterinary Medicine, University of Warmia and Mazury in Olsztyn, Poland

## Long-term treatment with indomethacin increases the number of SP-immunoreactive porcine duodenal neurons

Gastrointestinal inflammation resulting from prolonged NSAID drugs treatment constitutes a worldwide medical problem. Recently the role of enteric neuroactive substances involved in this process has gained attention. Therefore the aim of the study was to determine the effect of inflammation caused by indomethacin supplementation on substance P (SP) expression in enteric duodenal neurons in domestic pigs. The study was carried out on eight immature pigs of the Pietrain x Duroc race (approximately 20 kg of body weight). The animals were divided into two groups - a control (C group) and an experimental group (I group). Group C (n=4) was consisted of animals which received empty gelatine capsules. Group I (n=4) was composed of pigs which naproxen were given orally (10 mg/kg) for 4 weeks, approximately 1 h before feeding. After this time, animals from both groups were euthanized. Then, frozen sections (14 μm thickness) were prepared from the collected material and subjected to double immunofluorescence staining. Antibodies against the neuronal marker PGP 9.5 and against the substance P were used as primary antibodies. The secondary antibodies - Alexa Fluor 488 and 546 - were also used for staining. Analysis of the sections was performed using an Olympus BX51 fluorescence microscope equipped with a XM10 monochrome camera. Analysis of the results obtained with a fluorescence microscope showed significant increase in the number of SP immunoreactive submucosal neurons and a smaller increase in the number of SP positive neurons in the myenteric ganglia of the porcine duodenum. The obtained results suggest that inflammation caused by the administration of high doses of naproxen increases the expression of substance P in the duodenal neurons in domestic pigs. Increased synthesis of this neurotransmitter may suggest the contribution of SP to the development of the local inflammatory process or the involvement of SP positive neurons in local repair processes. This study was supported by the National Science Centre (grant no. 2018/29/N/NZ4/00348).

**Audience Take Away:**

- It is well known that the enteric nervous system is involved in physiological processes in the digestive tract such as contractility and secretion. It is thought that my active neural substances contribute to or limit the development of gastrointestinal disorders
- Our research aims to determine the effect of indometacin-induced inflammation on the characteristics of porcine duodenal neurons. The research results will allow to determine the role of selected neurotransmitters in the development of inflammation as well as their participation in the processes of neuroprotection and regeneration

**Biography**

Marta Czajkowska studied veterinary medicine at the University of Warmia and Mazury in Olsztyn and graduated as MS in 2016. She is currently a PhD student at the Department of Clinical Physiology at the Faculty of Veterinary Medicine, University of Warmia and Mazury. She is the author of several publications as well as the head of the scientific grant titled "The influence of inflammation caused by the administration of non-steroidal anti-inflammatory drugs on the chemical coding of the enteric neurons of selected sections of the porcine gastrointestinal tract "- Grant supported by National Center for Science (ID number - 2018/29/N/NZ4/00348).

**Polina A. Sobolevskaia[1*], Leonid P.Churilov[1], Tamara V. Fedotkina[1], Anna Stepochkina[1], Anastasia Dolina[1], Anton N.Gvozdetckii[2], Boris V. Andreev[4], Yehuda Shoenfeld[1,3]**

[1]Laboratory of the Mosaic of Autoimmunity, Saint Petersburg State University, Saint-Petersburg, Russia

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[3]P. Zabludowicz Centre for Autoimmune Diseases, H. Sheba Medical Center, Tel Hashomer, Israel

[4]P.P. Kaschenko 1st City Mental Hospital, Saint Petersburg, Russia

## Proinflammatory cytokines in psychiatric disorders comorbid with Hashimoto's thyroiditis

We have analyzed 27 patients with Hashimoto's thyroiditis (HT) associated with psychiatric disorders.We also studied different parameters of immunoendocrine reactivity: thyroid hormones, prolactin, TSH, antithyroid antibodies, anti-alpha-enolase antibodies (anti-ENO1 Ab) and the levels of 26 cytokines in the blood. We detected a significant association between the levels of cytokines and mental symptoms: attention deficit and IL-5 level ($\beta$ = -1.82; p = 0.046), attention deficit and IL-12P70 level ($\beta$ = -1.67; p = 0.048), IL-27 and hallucinations ($\beta$ = -1.6; p = 0.035), CCL20 MIP3a level and OCD ($\beta$ = -2.78; p = 0.018). We analyzed the associations between the levels of cytokines and various immune-endocrine parameters, a significant direct correlation was found between the level of FT3 and: IL-1b ($\beta$ = 4.13; p = 0.017), IL-5 ($\beta$ = 3.3; p = 0.048), IL-13 ($\beta$ = 5.77; p = 0.006), IL-15 ($\beta$ = 3.99; p = 0.014).TSH concentration negatively correlated with levels of: IL-15 ($\beta$ = -3.37; p = 0.006), IL-1b ($\beta$ = -2.06; p = 0.012). There was a positive correlation between the level of anti-ENO1 Ab and the concentration of IL-1b ($\beta$ = 12.52; p = 0.03) and IL-21 ($\beta$ = 12.39; p = 0.032).Anti-TPO Ab showed an inverse correlation with the level of IL-15 ($\beta$ = -0.67; p = 0.022). It is known that glial brain cells produce a number of cytokines, thus affecting neurons and contributing to the development of behavioral changes [Eusden , 2017]. Some cytokines outside the CNS itself are able to act on vagal afferents and through them convey information to the CNS, influencing its state and functions [Korneva, 2019]. Our patients had HT, which in itself disrupts the state of the cytokine network, it was demonstrated that HT is typically characterized by an increase in the leptin / adiponectin ratio with an increase in the level of TNFα, progressing as the disease progresses.According to our data and the data of other authors, the systemic concentrations of several pro-inflammatory cytokines, including the aforementioned leptin and TNFα, as well as monocytic chemoattractant protein-1 and interferon-γ, increase, and the levels of their anti-inflammatory analogs (interleukin-10, adiponectin) decrease in HT [Churilov, 2019]. FT3 concentrations were most closely correlated with the level of several cytokines in the studied group of patients. It is known that it is this hormone that possesses receptors on microglial cells and controls the processes of their inflammatory activation and phagocytic behavior [Mori, 2015]. A positive correlation with proinflammatory cytokines of a number of autoimmunity parameters and clinical picture in mentally ill patients with HT can be interpreted taking into account the data that such cytokines - both at the periphery and produced locally by astrocytes - can shift tryptophan metabolism in the central nervous system towards kynurenine. which contributes to the mediator disorders involved in the pathogenesis of schizophrenia [Kindler, 2019].

**Audience Take Away:**

- The profile of proinflammatory cytokines in patients with Hashimoto's thyroiditis associated with psychiatric disorders.
- The association of the proinflammatory cytokines with hormones and different auto antibodies
- The association of the proinflammatory cytokines with psychiatric symptoms

- The hypothesis of the association of immunoreactivity with psychiatric disorders
- This information can be used in different surveys based on psychiatric disorders and autoimmune disorders. It can help to study new aspects of psychiatric disorders, new mechanisms of pathogenesis of psychiatric disorders. It also can highlight new ways of development of psychiatry and it's collaboration with other fields of science

**Biography**

Dr. Polina Sobolevskaia studied medicine in Saint-Petersburg State University, Russia and graduated as MD in 2014. In 2017 joined the research group of Prof. Shoenfeld in Saint-Petersburg State University, and conducted researches on thyroid autoimmunity and central nervous system impairments. Now she is working on her PhD. She has published more than 15 research articles in scientific journals, she is a co-author of 2 books, she presented the results of her scientific researches on 10 international conferences.

**Yahia Al Turk**

Henry Ford Hospital, USA

## Neuro-Behcet disease complicated by steroid-induced bilateral femoral head avascular necrosis

Behçet disease was first described in 1924 by a Turkish dermatologist, Dr. Hulusi Behçet. Oral and skin lesions are present in >75% of patients, and genital lesions are the most specific manifestation, and neurologic disease occurs in less than 10% of patients with Behçet disease, as reported. We describe a case of a 28-year-old African American man who presented to our hospital with slurred speech and imbalance, along with genital and oral ulcers. Imaging findings were significant for enhancement in the left pons and midbrain seen on brain MRI. He was thought to have neuro-Behçet disease after other diagnoses were ruled out. The patient had an excellent clinical response to intravenous steroids that correlated to improvement on brain MRI. He later had recurrence of neurological symptoms and was started on cyclophosphamide. Patient completed 6 cycles of cyclophosphamide with overall significant improvement in neurological symptoms; however, he needed one admission requiring use of intravenous steroids during treatment. Use of steroids was limited after a few months, as he developed bilateral femoral head avascular necrosis. The diagnosis of neuro-Behçet can be challenging since no accepted criteria for diagnosis exist. Disease should be suspected in patients who have recurrent oral, skin, or genital lesions and who present with neurological symptoms with negative workup for other etiologies (e.g., infectious causes, sarcoidosis, autoimmune encephalitis). Multidisciplinary approach involving rheumatology, neurology, dermatology and other specialties is often required to confirm diagnosis and establish a treatment plan. Azathioprine and cyclophosphamide are among first line medications for neuro-Behçet; although not always effective. Managing neuro-Behçet can be very challenging and Intravenous steroids should be considered for acute, worsening, or life-threatening manifestation of the disease. However, extended use of steroids can lead to serious complications, highlighting the need for vigilance during steroid therapy for neuro- Behçet to minimize adverse effects.

**Biography**

Dr. Yahia Al Turk is an internist in Detroit, Michigan and is affiliated with Henry Ford Hospital.

**Ali hammed[1*], Maysaa Badour[2], Sameer Baqla[2], Fatema Amer[2]**

[1]Tishreen University Hospital, Department of neurosurgery. Lattakia, Syria
[2]Pediatric University Hospital, Division of Neurology, Damascus, Syria

## Diagnosis and treatment of Rasmussen's Encephalitis pose a big challenge: Two case reports and literature review

Rasmussen encephalitis (RE) is a rare disease of unknown etiology that causes severe chronic unihemispheric inflammatory disease of the central nervous system mainly in children. It leads to intractable seizures, cognitive decline and progressive neurological deficits in the affected hemisphere. We report two cases of RE, as defined by fulfillment of the 2005 Bien criteria. The diagnostic challenge of characterizing this rare disease will be highlighted by the extensive serum, CSF, MR imaging and EEG data in the two patients. In addition, we will review the various forms of therapy attempted in these two patients, namely anti-epileptic drug therapy and immunomodulatory therapy. Hemispherectomy was done for the second patient with favorable outcomes of controlling seizures, but unfortunately, he died because of meningitis.Until the causes of Rasmussen's encephalitis are known, it is difficult to anticipate how treatments will improve. Such a situation creates a therapeutic dilemma; hemispherectomy is not favored because of the inevitable postoperative functional deficits, but a real risk exists that treatments used to delay progression of the disease will defer definitive surgical treatment beyond the time when an optimum post-hemispherectomy outcome could be expected.

**Audience Take Away:**

- This lecture will help audience to deal with such complex cases of epilepsy
- RE should be suspected in any patient with refractory seizures
- Its recognition is important because early intervention with surgery can improve outcomes

**Biography**

Dr. Ali studied General medicine at the Tishreen University, Latakia and graduated as MD in 2018. He then joined the residency program at the Tishreen university hospital. He is researcher and research program leader at TUH. He is reviewer at. Elsevier, BMC, Frontiers, Journal of oncology and Medicine.

**Ayushi Shah[1*], Nate Bergman[2]**

[1]Ayushi Shah, Kemper House, Cleveland, OH, USA
[2]Nate Bergman, Cognitive Wellness Officer, Kemper House, Cleveland, OH, USA

## Dance Intervention in Alzheimer's Patients

This study piloted a 30-day dance and music therapy in four residents living in a dedicated memory care assisted living facility. The MMSE's showed an average change of 3.875 points overall in all four residents and no side effects were observed. The conclusion made from this program is that dance therapy may be a viable option to preserve or improve cognitive function in those with existing neurodegenerative conditions. In addition, what this information can be used for in the future to further benefit the wellness of Alzheimer's patients.

**Audience Take Away:**

- The audience will be able to further understand the benefits of dance intervention, and use this knowledge to help Alzheimer's patients with their cognitive functioning
- This research can be used to expand their understanding of what exercises improve and help with cognitive ability. In addition, this provides a helpful way in order to discover what types of exercises are the most beneficial for Alzheimer's patients
- This research can be used by other faculty to further expand on why dance intervention is a good way to help Alzheimer's patients, and specifically, what types of dance can be the most beneficial

**Biography**

Ayushi Shah is an undergraduate student studying neuroscience at Case Western Reserve University, and is graduating this spring. She has conducted research in Alzheimer's and has worked with Kemper House and Dr. Nate Bergman in order to research the effects of exercise in Alzheimer's patients.

**Pacome Kouadio N'Go[1,2*], Omar Touhami Ahmed Ahami[2], Aboubaker El Hessni[3]**

[1]Peleforo Gon COULIBALY University, Training and Research Unit of Biological Sciences, P.o.Box 1328, Korhogo, Ivory Coast

[2]Clinical and Cognitive Neurosciences Group, Biology and Health Lab, Ibn Tofail University, P.o.Box 133, Kenitra, Morocco

[3]Genetic, Neuroendocrinology and Biotechnology team, Biology and Health Lab, Ibn Tofail University, P.o.Box 133, Kenitra, Morocco

## Neuroprotective effects of chrysophyllum perpulchrum bark extract in AD-like rats model of β-amyloid 1-40 intrahippocampal injection

**Background:** Alzheimer Disease (AD) is a threatening disease of upcoming years among african populations because of the increase of their expectancy life. Because no therapeutic drugs existing, it is now worth while to investigate on new effective drugs derived from natural product of endemic traditional use plants.

**Objective:** We examine whether a methanolic bark extract of Chrysophyllum perpulchrum, rich in catechin and two new procyanidins (catechin dimers + hexose), could prevent some physiopathology mechanisms as well as cognitive changes in an AD-like rat model induced by intrahippocampal CA1 subfield Aβ1-40 injection.

**Materials and Methods:** Adult male Wistar rats were either microinjected with 1% ammonia as vehicle (10μL) or aggregated Aβ1-40 (10μg bilaterally). At 14th day post-surgery, a group of Aβ rats received treatments with melatonin (10 mg/kg i.p) or Chrysophyllum perpulchrum extract (300 mg/kg p.o), and sham-operated rats only with extract. Cognitive abilities were tested with Y-maze, object recognition test and Morris water Maze. Biochemical assay of pro-inflammatory and oxidative stress markers, as well as microglia cells activation with Iba 1 immunostaining in brain.

**Results:** We found microglia over activation associated with high levels of nitric oxide (NO), Malondialdehyde (MDA), superoxide dismutase (SOD), but not thiol content in hippocampus, prefrontal cortex (PFC) and septum of AD-like rats. Aβ induced also significant recognition memory and spatial learning deficits. However, the treatment with Chrysophyllum perpulchrum extract improved significantly cognitive impairments through a mitigation of Aβ-induced microglial cells overactivation and subsequent neuroinflammation and oxidative stress processes. Interestingly, the neuroprotective actions of Chrysophyllum perpulchrum extract seem to be more effective than melatonin (10 mg/kg ip).

**Conclusion:** These findings should be strengthened by pharmacological studies such as bioactive compounds of Chrysophyllum perpulchrum's action on the blockade of cdk 5/p35 complex and Aβo- induced Fyn signaling, before being proposed as promise drug against AD.

### Biography

Pacôme Kouadio N'Go is currently working in Training and Research Unit of Biology Sciences, Pelefero Gon Coulibaly University, Ivory coast, Morocco.

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## NEUROLOGY AND BRAIN DISORDERS

SEPT 09-10, 2021

INBC 2021

**Sven-Erik Eriksson**

Division of Neurology, Department of Medicine, Falun Hospital, S-791 82 Falun, Sweden

## Long-term prognosis after tia/stroke in patients with or without cancer and evaluation of fasting total cholesterol and triglyceride levels on different outcomes

This study is about reporting annual events of strokes, myocardial infarctions and deaths, in patients who had been hospitalized due to TIA/stroke in 1986 and followed up until February 2011; with or without cancer, and in patients with assessed lipid values after admission also report predictors of stroke, myocardial infarction (MI) and death. A total of 288 men were followed up for 2254 years (mean 7.8 years) and 261 women for 1984 years (mean 7.6 years). Among these patients 67 men had a total of 78 malignant cancers during life with a follow- up period of 577 years (mean 8.6 years) after admission compared to 69 women had 72 malignant cancers with a follow- up period of 588 years (mean 8.5 years). Fasting total-cholesterol (TC)/ triglyceride (TG) value was measured after admission in 124 men and 96 women. New stroke was a statistically, significantly more common cause of death in the group of male patients who did not have a malignant cancer (p=.004), as was fatal MI in women (p=.016). The type of cancer, past cancer or not, and patient sex was each an important factor regarding the annual risk of stroke or MI. Patients with a diagnosis of malignant cancer first after TIA/stroke usually appear to have lower annual risk of stroke/MI compared to patients without cancer diagnosis. Among men with assessed lipid value there was almost a linear correlation between increasing TC levels (< 5 mmol/L; 5-6.4 mmol/L; and ≥ 6.5 mmol/L), and increasing risk of MI during life, P= .016, and cardiovascular death (CVD) (not index stroke), P = .002 compared to women who had a U-shaped correlation. Men had an inverse correlation between TC levels and death due to cancer, P=.008. According to Cox regression analyses, predictors of ischemic stroke were age, sex, diagnosis, history of diabetes/fasting blood glucose ≥ 6.1 mmol/L, angina pectoris; predictors of myocardial infarction were systolic blood pressure, angina pectoris, TC ≥ 6.5 mmol/L and predictors of death were age, sex, history of diabetes/fasting blood glucose ≥ 6.1 mmol/L, TIA/severity, hypertension/treatment with antihypertensive drugs, previous MI, TG > 2.2 mmol/L, and first-line treatment.

**Audience Take Away:**

- Cancer itself and/or its treatment may have impact on the annual risk of (fatal) stroke/MI compared to non-cancer patients in patients with TIA/stroke
- TC and TG levels measured after admission effect men and women differently regarding the risk for stroke, MI, cardiovascular death and survival. These differences in outcomes between the sexes might support that the indication for lipid-lowering treatment is partly dependent on sex

**Biography**

Specialist in neurology 1981, University Hospital, Linköping, Sweden. Chief physician, Division of Neurology, Department of Medicine, Falun Hospital, 1984-2016; thereafter hourly senior physician. Published articles especially in secondary stroke prevention.

**Alberto A. Rasia-Filho**

Department of Basic Sciences/Physiology, Federal University of Health Sciences (UFCSPA), Porto Alegre/RS, Brazil

## Structure, diversity and 3D complexity of neurons from subcortical to cortical areas in the human brain

The visualization of nerve cells is important for the advancement of neurological research and the comprehension of the foundations of the functioning of the human brain. At the cellular level, dendritic spines are the smallest multifunctional units that reflect cellular connectivity and can modulate postsynaptic processing and plasticity. Different approaches were developed to unravel detailed morphological features of neurons in animal models along the last decades. However, the study of human dendrites and spines has been a challenge due to inherent technical difficulties and the complexity of the nervous tissue in our species. A new three-dimensional (3D) reconstruction procedure was introduced for the visualization of neurons in human postmortem brain tissue using brightfield light microscopy. The procedure uses an adapted histological improvement of the Golgi method to obtain suitable results using human brains conventionally fixed in formalin even for a long time. Structure, diversity, and complexity of neurons can be evaluated at different angles with precise morphological details. High-quality images allow the dendritic spines to be identified and classified, either isolated or in clusters, in a continuum of shapes and sizes, from simple to more elaborated forms, including the presence of tiny spinules. Furthermore, the unlike density and shapes of spines along dendritic branches, from proximal to distal ones, indicate a specific synaptic transmission and integration in each studied neuron. Examples are provided for the multipolar neurons from the subcortical nuclei of the subcortical amygdaloid complex, the pyramidal and non-pyramidal neurons in the allocortical hippocampal CA3 area, and the neocortical temporal lobe and cingulate cortex in the adult human brain. The identification of multiple forms of neurons, their particular dendrites and dendritic spines, can reflect a more complex structure for synaptic processing of higher-order sensory, cognitive and emotional information and an adaptation for species-specific behavior display. These relevant structural/morphological results have broad phylogenetic, ontogenetic, cytoarchitectonic and functional implications for the human brain in both normal controls or in neurological and psychiatric disorders.

**Audience Take Away:**

- Audience will be introduced to a new suitable morphological tool for studying different, complex human brain neurons located in subcortical, allocortical, and neocortical areas
- Referential 3D reconstructed images can serve to link neuronal structure and function, unravelling microscopic details of neurons in both normal and pathological conditions
- 3D morphological features of human neurons can complement and expand research lines and teaching in undergraduate and graduate activities in Neurology and in various brain diseases
- The present approach requires minimal laboratory equipment, provides new information based on high-quality images and precise morphological data from light microscopy comparable to other more expensive and difficult techniques

**Biography**

Rasia-Filho is full professor, holds an academic degree in Medicine, and M.Sc. and Ph.D. degrees in Biological Sciences/Physiology (UFRGS/Brazil). Postdoctoral fellow in synaptic structure at the School of Medicine/University of São Paulo/Brazil. Researcher granted by the Brazilian Council for Scientific and Technological Development (Morphology). Prized by the Brazilian National Academy of Medicine (Neurology and related areas). International ad hoc reviewer and Guest Associate Editor (Frontiers in Neuroscience, in Psychiatry, and in Synaptic Neuroscience) for Research Topics in "Dynamics and Modulation of Synaptic Transmission in the Mammalian Central Nervous System" and "Frontiers in Synaptic Plasticity: Dendritic Spines, Circuitries and Behavior".

**Georgina Rodriguez de Lores Arnaiz[1], Analía G. Karadayian[2] Alicia Gutnisky[1], Jennifer Miranda[1], Silvia Lores-Arnaiz[2*]**

[1]Instituto de Biología Celular y Neurociencias "Profesor Eduardo De Robertis" (IBCN) CONICET-UBA, Facultad de Medicina, Universidad de Buenos Aires, Buenos Aires, Argentina

[2]Instituto de Bioquímica y Medicina Molecular (IBIMOL) CONICET-UBA, Fisicoquímica, Facultad de Farmacia y Bioquímica, Universidad de Buenos Aires, Buenos Aires, Argentina

## Is there a relationship between the alteration of synaptic proteins of the complex PSD-95/NMDA receptor/nNOS and brain disorders?

Evidences point to the notion that the complex PSD-95/NMDA receptor/nNOS seems to play a key role in normal neuronal functions like synaptic plasticity, learning and memory, as well as in brain disorders such as stroke, pain and autism. PSD-95 is one of the two most abundant postsynaptic proteins in the postsynaptic density (PSD) which plays a pivotal role in the coupling between NMDA receptors and nNOS and can modulate the synaptic expression of glutamate receptors by binding to GluN2A and GluN2B, thereby stabilizing NMDA receptors at the cell surface. Uncoupling nNOS from the NMDA-receptor through the scaffolding protein PSD-95 produces behavioral antidepressant effects resembling the effects of NOS inhibitors and exerts a neuroprotective action against stroke. Given that ATP provision by mitochondria may play an important role in the functional interaction between synaptic proteins NMDA receptor and PSD-95 with NO synthesis, we became interested in the study of synaptic proteins of the complex PSD-95/NMDA receptor/nNOS and mitochondrial functionality. The study was conducted in fractions isolated from rat cerebral cortex after administration of levocabastine, a drug employed as a tool to block the low affinity neurotensin receptors. Male Wistar rats received a single (i.p.) dose of levocabastine (50 μg/kg) or saline solution (controls) and were decapitated 18 hours later. Synaptosomal membranes were obtained and expression of synaptic proteins was evaluated by Western blot assays. After levocabastine treatment, protein expression of PSD-95, nNOS and GluN2B subunit of NMDA receptor decreased 97%, 56% and 45%, respectively versus controls. At variance, expression of iNOS enhanced 3.5-fold versus controls. In crude mitochondrial fractions, levocabastine administration reduced roughly 15% respiratory control rate as assayed with malate-glutamate or succinate as substrates, decreased mitochondrial membrane potential (21%), and ATP production rates (57%). In addition, a 55% decrease in beta actin was observed. Alterations in actin cytoskeleton might then be a consequence of ATP deficit but may also lead to impairment of mitochondrial dynamics, which in turn would result in further mitochondrial dysfunction and ATP depletion. Results indicated that levocabastine administration induces alterations in synaptic proteins of the complex PSD-95/NMDA receptor/nNOS and in neuron cytoskeleton. Mitochondrial bioenergetics impairment may play a role in the functional link between synaptic proteins and NO synthesis. In addition, levocabastine can impair mitochondrial function in in vitro conditions. This direct effect of levocabastine on mitochondrial bioenergetics might lead to alteration of the integrity of functional complex PSD-95/NMDA receptor/nNOS, due to the decrease in ATP formation which is required for protein assembly. To sum up, present results showed important changes in specific synaptic proteins and mitochondria functionality, suggesting an impairment of PSD-95/NMDA receptor/nNOS complex integrity. At synaptic sites, mitochondrial dysfunction might influence the interaction between these proteins inter se, and with other specialized molecules involved in neurotransmitter processes as well as in signaling processes. Further studies may help to understand the role of synaptic complex PSD-95/NMDA receptor/nNOS in brain disorders at cellular and molecular levels and might contribute to the future development of pharmacological treatment options.

**Audience Take Away:**

- The audience will be able to expand their research in Neurochemistry
- The audience will be able to expand their teaching to post-graduate students in Neuropharmacology
- The audience will be able to use new information from our results to assist in a new design problem

**Biography**

Dra. Georgina Rodríguez de Lores Arnaiz. Farmacist (1957), Biochemist (1959), Doctor in Pharmacy and Biochemistry (1961). Facultad de Farmacia y Bioquímica, Universidad de Buenos Aires. CONICET Superior Investigator at Instituto de Biología Celular y Neurociencias "Prof. E. De Robertis" (IBCN) CONICET-UBA, Facultad de Medicina, Universidad de Buenos Aires, Paraguay 2155-1121-Buenos Aires. Argentina. Editor for Research Topic "Neurochemistry – Neuropharmacology", Frontiers in Pharmacology. Emeritus Member at International Society for Neurochemistry, American Society for Neurochemistry, Society for Neuroscience Steering Committee and Editorial Board Member at Sociedad Científica Argentina (SCA). Pharmacology, Facultad de Farmacia y Bioquímica, Universidad de Buenos Aires: Full Professor (1989 - 2001); Consultant Professor (2002 - 2011). Editorial Board Member for Neurochemical Research (1989-1999) and Journal of Neurochemistry (Handling Editor)(1992-1999). Book Editor on Function of neuropeptides at central nervous system (2009). Publications more than 150 research articles in international scientific journals.

**Silvia Lores Arnaiz**

Instituto de Bioquímica y Medicina Molecular, Fisicoquímica, Facultad de Farmacia y Bioquímica, Universidad de Buenos Aires, Buenos Aires, Argentina

## Mitochondria and synaptic dysfunction: Implications for aging and brain disorders

Neurons rely almost exclusively on ATP synthesis from the mitochondrial respiratory chain and oxidative phosphorylation to fulfill their energy requirements for neurotransmitter synthesis, release and reuptake. Besides providing energy, mitochondria are also involved in intracellular calcium regulation, cellular redox control and apoptosis. Within the nerve terminals, mitochondria are essential to maintain an adequate synaptic function. Interestingly, neurotransmitter systems can drive mitochondrial dysfunction by different mechanisms including calcium deregulation, oxidative stress or disruption of mitochondrial trafficking and dynamics. In fact, alterations in several neurotransmitter systems -dopaminergic, glutamatergic and cholinergic systems, among others- have been associated with particular changes in mitochondrial functionality during aging and neurodegenerative diseases. Neuronal aging is a complex physiological process, associated to metabolic alterations, mitochondrial dysfunction and free radicals production. Results from our laboratory have shown that aged mice exhibited a significant reduction in motor performance and walking footprint pattern, and a decrease in acetylcholinesterase activity. Regarding mitochondrial function, basal respiration and respiration driving proton leak were decreased in synaptosomes from 17-months old mice, while spare respiratory capacity seems to be preserved. Basal mitochondrial membrane potential was maintained in brain cortex synaptosomes from aged mice, whereas a decrease was observed after calcium overload, as compared with young mice. Interestingly, UCP-2 protein expression was increased in synaptosomal samples from 17-month-old mice and superoxide levels were significantly lower than those in young animals. UCP-2 upregulation seems to be a possible mechanism by which synaptic mitochondria would be resistant to suffer oxidative damage. At more advanced ages, regulatory systems may be overwhelmed with the consequent accumulative damage. The use of drugs acting on specific receptors systems are adequate experimental tools to understand the possible interaction between changes at neurotransmitters and mitochondrial function at synapses. The relationship between mitochondrial dysfunction and neurotransmitter systems such as glutamatergic and cholinergic ones has been extensively studied in aging and brain diseases. However, the involvement of neurotensinergic system in mitochondrial alterations at synapses has not been deeply explored. In our study, levocabastine was used as a neurotensinergic targeted drug, acting as an antagonist of NTS2 receptors. Rat treatment with levocabastine led to important changes in synaptic proteins which are concomitant with altered nitric oxide synthesis. In addition, mitochondrial impairment was evidenced by reduced respiratory control rates, mitochondrial membrane potential depolarization and decreased ATP production rates after levocabastine administration. Mitochondrial bioenergetics impairment may play a role in the functional link between synaptic proteins and nitric oxide synthesis. Understanding the interactions between neurotransmission and mitochondrial dysfunctions is crucial to elucidating the specific mechanisms involved in cognitive decline during normal aging and brain diseases.

**Audience Take Away:**

- The audience will be able to expand their research in the field of Neurosciences regarding the subject of Mitochondrial function in Physiology and Pathology
- The audience will be able to expand their teaching to post-graduate students in Neurosciences
- The audience will be able to use new information from our results to apply for new design problems

**Biography**

Academic degrees: Biochemist (1989), Pharmacist (1993), Ph. D. Universidad de Buenos Aires (1994) Facultad de Farmacia y Bioquímica, Universidad de Buenos Aires (UBA). Investigador Independiente", Consejo Nacional de Investigaciones Científicas y Técnicas (CONICET). At the "Instituto de Bioquímica y Medicina Molecular" Universidad de Buenos Aires (UBA-CONICET). Buenos Aires, Argentina. Full Professor Applied Physical Chemistry (Instituto Universitario del Hospital Italiano, Buenos Aires, Argentina). More than 50 publications in international scientific journals. Book Editor: "Brain Mitochondria: Distribution and Function", 2020.

**Gloria Garrabou\*, Judith Canto-Santos, Laura Valls-Roca, Ester Tobias, Francesc Josep Garcia-Garcia, Pedro Moreno, Jose Cesar Milisenda, Francesc Cardellach, Josep Maria Grau**

IDIBAPS - CIBERER - UB, Spain

## Molecular characterization of fibroblast's models of neurologic and neuromuscular diseases

**Introduction:** Both neurologic and neuromuscular diseases as Parkinson disease (PD) and sporadic inclusion body myositis (sIBM) usually affect patients at elderly stages of life and share common molecular etiology. Inflammation, proteostasis deregulation, degenerative autophagic changes, oxidative stress, metabolic and bioenergetic dysfunction have been reported, among others, in both kind of disorders. Unfortunately, most of these diseases lack either diagnostic/prognostic biomarkers and effective treatments, mainly due to the lack of validated disease models. We aim to validate fibroblasts as a disease model for both kind of disorders to set the path for further advances.

**Methodology:** We examined PD and IBM disease hallmarks in fibroblasts of affected patients using OMICs and functional approaches. In OMICs approach, we analyzed the transcriptome and metabolic profile in fibroblasts from 6-8 PD patients, 14 sIBM subjects and 12 paired controls, through mRNA seq and UHPLC (Ultra High-Performance Liquid Chromatography). In the functional approach, we assessed inflammatory, degenerative, oxidative stress, metabolic and bioenergetic changes in patients' fibroblasts. Results were analyzed through non-parametric statistic tests.

**Results:** In the OMICs approach, 343 and 778 deregulated expressed genes were found in PD and sIBM patients' fibroblasts, in pathways related to cell adhesion, cell growth, amino acid and folate metabolism (in PD), or RNA processing, cell communication and amino acid metabolism (in sIBM). UHPLC showed altered amino acid and organic acid levels related to mitochondrial defects and Krebs cycle in both kind of fibroblasts. In the functional approach, PD and sIBM fibroblasts showed different but both dysfunctional profiles characterized by increased secretion of cytokines, defective autophagy, increased oxidative stress and deregulated metabolic and bioenergetic status that may underscore cell feat.

**Conclusion:** Both OMICs and functional approaches recapitulate PD and sIBM hallmarks in fibroblasts, thus validating its usefulness as a disease model to explore molecular targets and assay therapeutic strategies that may eventually be exported to other neurologic and neuromuscular diseases. Our findings indeed support the view of PD and sIBM as 'systemic diseases' affecting also peripheral tissues such as the skin.

### Biography

Gloria Garrabou had completed Grade in Biological Sciences, specialization in Molecular, Cell Biology and Genetics, at the UAB, 2001 , Post grade in Genomics, Proteomics and Bioinformatics at the UB, 2002 and Master in Genetics at the UB, 2003 PhD (Cum Laude and Extraordinary Doctoral Thesis award) at the UB 2008 , Numerous courses in genetics (SEG/CNAG), respirometry/fluorometry (Oroboros, Austria), microscopy (CSIC), statistics (UB), mitochondrial diseases (UAM), writing competitive proposals (ERC calls; IDIBAPS/CIBERER), medical English (UB), teaching skills (UB), etc She is an Accredited researcher R3A IDIBAPS contracted by the CIBER of Rare Diseases (CIBERER) at the Muscle Research and Mitochondrial function Laboratory, IDIBAPS. Specialization in the study of mitochondrial and bioenergetic implication in: toxicity, obstetric complications, cardiomyopathy, neurodegeneration and neuromuscular disorders. Coordinator of the Lab in collaboration with the clinical chiefs Member of a Consolidated Group from the Generalitat of Catalunya (SGR), the MetNet and the Cardionet societies Participation in the Research Team of 36 competitive projects and 7 projects as Principal investigator: 2 FIPSEs in mitotoxicity of HIV and medication; 1 FIS and 1 CONACyT in neurodegeneration and 1 FIS and 2 ACCIs in neuromuscular and mitochondrial diseases (MNGIE, SKS and sIBM). Funding of human and material resources, with meritorious results.

**Meenal Dhall[1*], Shweta Jain[2]**

[1]Department of Anthropology, University of Delhi, Delhi, India
[2] Department of Anthropology, University of Delhi, Delhi, India

## Artificial intelligence and neuroanthropology: A holistic approach

Neuroanthropologists understand the dynamic enculturation of brain and cognitive functions in their external and internal environment. It necessitates the researchers to analyse big data for seeking proper understanding. Artificial intelligence (AI) is far better at predicting behavior than the human mind. The invention of anthromorphized robots which shows emotions and empathy like humans is also a result of AI applications. It holds great potential to advance diagnosis and treatment of patients with neurocognitive disorders. Early detection of pathological cognitive decline facilitates the greatest impact of restorative or preventative treatments. AI in healthcare is the use of computational algorithms that mimic human cognitive functions to analyze complex medical data. AI technologies like machine learning (ML) support the integration of biological, psychological, and social factors when approaching diagnosis, prognosis, and treatment of disease. Sociodemographic and other forms of population data offer rich information from large datasets because many countries collect population data regarding health, socioeconomic status, and social and family networks of older adults, such information may also provide an opportunity to compare outcomes across different countries and infer global health estimates of neurocognitive disorder burden. AI's strength lies in its ability to accommodate large quantities of multimodal data. Thus, AI can aid better understanding of unique factors and behaviors associated with cognitive decline that have been previously difficult to quantify. This study will offer the understanding of how AI made easier to understand and prevent neurocognitive disorders with better outcomes than before.

**Audience Take Away:**

- The audience will get aware about the AI based technologies Which are used to understand cognitive behaviours. The audience can use simple applications, designed with AI models to make their life easy like through personalized shopping, wearable devices, automatic cars and navigation apps. These are real world applications of AI
- The use of AI not only limited in healthcare industry it can be applied in various fields to achieve the better outcomes with high accuracy and reliability
- It enhances the chance of reaching higher accuracy with a greater degree of precision by error reductions which will benefit people with cognitive impairment to make them remember their tasks by analysing their actions and routines in a great way through AI models
- It can help in screening and early detection of many diseases or disorders like dementia, diabetic retinopathy and various other health care problems
- It helps to provide evidence-based solutions to improve the treatment of diseases by focusing on patient-oriented approach.
- People can use AI technologies and models for start-ups in healthcare industry to prevent, diagnose and treat public health problems among elderly as they need care givers. The responsibilities of care giver can be reduced with the help of AI models and machine learning

**Biography**

Dr. Meenal Dhall completed Doctorate of Philosophy in Anthropology from University of Delhi in 2013. Dr. Dhall has 40+ research articles published in National and International journals. She has 05 edited books and 02 books authored. Dr. Dhall has visited many countries like Germany, Sweden, Austria, France, Cape Town, Mexico in connection with academic activities. Her areas of interest are Physiological Anthropology, Kinanthropometry, Public Health and Epidemiology, Nutrition, Physical Activity, Neuroscience, Human growth & development and Epigenetics, Forensic Anthropology. She is life member of various organizations. She is presently Assistant Professor and teaching in the Department of Anthropology, University of Delhi, India.

## Ali Imran[1]*, Neelum Fiaza[1], Ghulam Hussain[2], Muhammad Umair Arshad[1]

[1]Department of food sciences, Government College University, Faisalabad, Pakistan

[2]Neurochemicalbiology and Genetics Laboratory (NGL), Department of Physiology, Faculty of Life Sciences, Government College University, Faisalabad, Pakistan

## Crude extract from corn by products (silk and cob) accelerate the functional restoration against compression injury to the sciatic nerve in a mouse model

Globally, polyphenols have been explored extensively for their therapeutic potential and proved effectual against various maladies. However, their neuroprotective effect yet has not been extensively explored. Likewise, quest for the identification and utilization of biomolecules from Agro-industrial waste are also in lime light due to their effectiveness and role towards ensuring food security. The aim of current investigation was to extract the polyphenols from corn by products namely as corn cob and silk and investigate their antioxidant and nutritional profile and utilization against sciatic nerve injury in mouse model. Purposely, the polyphenols from corn silk and cob were extracted through conventional extraction by adapting different variables and probed for their antioxidant characterization and identification through HPLC. For the estimation of neuroprotective effect, sciatic nerve injury was induced mechanically and divided in different groups. Total three groups of animals were made including the control containing 5 mouse in each. The control group was fed on normal chow during the whole study period whereas, other two administrating on corn silk and corn cob extract @250mg/kg BWT/day from the day of nerve crush till the end of the experiment. The examined extracts showed promising antioxidant profile however, corn silk yield more pronounced effect then the Cob. The hot plate test, grip strength test and SFI assessment were taken to assess their sensorimotor functions regain ability and result showed significant improvement in the treated groups as compared to control. Moreover, improvements in muscle mass of the experimental groups was the indicator of functional recovery. The outcomes advocating the beneficial role of corn by products against nerve disorders. However, more studies are requited to disclose the mechanistic concerns involved with that action.

**Biography**

Dr. Ali Imran is currently working as assistant Professor in the Institute of Home and Food Science from more then 8yrs. He has expertise in formulation of plant based nutraceutical based dietary intervention against oxidative stress mediated maladies both in animal and human models. He has more then 60 high impacted publication in reputed food science and nutrition journals. He also won many Competitive research grants relevant to his expertise. Currently, he is working on the role of plant based nutraceuticals in brain health on animal models. He also wrote more than 10 book chapters on health endorsing perspective of polyphenol.

**Silvana Giuliatti[1*], Willian Orlando Castillo[2]**

[1]Department of Genetics, Ribeirao Preto Medical School, University of Sao Paulo, Ribeirao Preto, Sao Paulo, Brazil
[2]Department or Biology, University of Cauca/Popayán Cauca, Colombia

## Seeking for alzheimer´s disease cure by exploring in silico new targets and compounds

Alzheimer's disease (AD) is an illness manifested by a number of pathological and clinical features, which including amyloid beta (Aβ) peptide deposits, neurofibrillary tangles (NFTs), dystrophic neurites, cholinergic impairment and neuronal death. To date, three acetylcholinesterase inhibitors (AChEi) (donepezil, rivastigmine and galanthamine) have been approved by the US Food and Drugs Administration (FDA) for its treatment, but these drugs provide symptomatic treatment but do not alter of curse of disease. The dysfunction of the cholinergic system in memory processing and storage is the base of the commonly accepted cholinergic hypothesis. According with hypothesis, in AD there are a loss of cholinergic neurons and nicotinic acetylcholine receptors (AChRs), which correlated with cognitive decline observed in the AD patients. AChEi enhance cholinergic neurotransmission by inhibiting the acetylcholinesterase (AChE) enzyme, which is responsible of the breakdown of the neurotransmitter acetylcholine (ACh), and this way, prolongs the action of the neurotransmitter at the synaptic cleft. However, recently it has also been shown that another type of cholinesterase, known such as butyrylcholinesterase (BChE) act as a co-regulator of cholinergic neurotransmission by hydrolyzing ACh, with a mechanism similar to AChE, this due to the functional and structural similarity of these enzymes. In recent years, there is a growing interest in the search of multifunctional compounds, which may represent an important pharmacological advance in the fight against the disease. In this context, compound combinations by synergistic effect could interfering simultaneously at different levels of the neurotoxic pathways. AD does not occur only by failing the cholinergic system. Neurotic plaque, cerebrovascular amyloidosis, and neurofibrillary tangles (NFTs) are considered marker of AD. Additionally, Peroxisome proliferator activated receptor gamma (PPARG) agonists reduce amyloid and tau pathologies, inhibit neuroinflammation and improve memory impairment in mild-to-moderate AD patients. However, they present poor blood brain barrier permeability, reducing their bioavailability. Other possible target that has been considered very important in AD is glycogen synthase kinase 3 beta (GSK3B) which plays an important role in the process of tau protein activation through phosphorylation. Therefore, GSK-3B inhibition is a potential approach for the treatment of AD. In the plants, we can find compounds with anti-cholinesterase, antigenotoxic, antioxidant, anti-inflammatory activity. This fact has motived a screening of new metabolites from Amaryllidaceae in view of their pharmacological potential. In natural products, we might find a source important of different molecules with potential to inhibit these four targets: AChE, BuChE, PPARG and GSK3B. Among the newer approaches in this research field, in silico molecular docking has been successfully used to identify, design and predict novel inhibitors. It is widely used in the discovery and optimization of novel compounds with affinity to a target; it also allows relating biological activity with the chemical constituent, which might give valuable hints for the prediction of biological activity. In this context, the present study aimed to evaluate the AChE, BChE, PPARG and GSK-3B inhibitory activity by some alkaloids belonging to Amaryllidaceae family members by applying in silico molecular docking complemented by biological experiments, which evaluated cytotoxicity and neuroprotective activity of alkaloids.

**Audience Take Away:**

- Our work can contribute to the knowledge of new therapeutic strategies based in natural products which can to be explored to counteract the neurotoxicity effects associated with Alzheimer´s disease
- This work provides evidence about of the neurotoxic and genotoxic effects induced by amyloid beta peptide.
- This studied explored the synergistic effect of the alkaloids compounds present in Caliphruria subedentata and its potential against amiloide beta peptide-induced neurotoxicity
- This therapeutic strategies must reach beyond the classical hallmarks, strategies targeting both brain and systemic metabolic aberrations seem to be much more efficient than the strategies targeting CNC abnormalities alone

**Biography**

Silvana Giuliatti holds a Bachelor's Degree in Physics from the Sao Paulo State University Julio de Mesquita Filho (1989), a Master's Degree in Physics Applied to Medicine and Biology from University of São Paulo (1993). From 1994 to 1997 she attended Imperial College London, St Mary's Medical School, developing her PhD project. She obtained her PhD degree in Physics Applied to Medicine and Biology at the University of São Paulo (2000). She is currently an Associate Professor at the University of São Paulo at the Genetics Department of the Medical School of Ribeirão Preto, FMRP-USP.

**Hussein Jaafar Hussein Ahmed**

University of Khartoum, Sudan

## Olfactory bulb changes on brain MRI in adult patients with anosmia due to COVID-19: A systematic review

**Background:** COVID-19 or coronavirus is a global pandemic, which is correlated with anosmia. This study aimed to summarize the Brain MRI finding in patients with anosmia due to COVID-19.

**Methods:** This systematic review was performed following the Preferred Reporting Items for Systematic Reviews and meta-analyses (PRISMA) reporting guidelines. A systematic literature search was done on 28th of January 2021 using PubMed, ScienceDirect, OVID/Medline and Embase. Studies reporting data sufficient to evaluate the olfactory bulb using MRI in patients with COVID-19 were included in the qualitative synthesis. Quality was assessed using the Newcastle-Ottawa scale (for observational cohort and case-control), AXIS tool (for cross-sectional), and quality assessment tool (for case series and case report). Information about demographics, MRI timing and findings, and duration of anosmia were extracted from the involved studies with subsequent qualitative evidence synthesis.

**Results:** The  search identified 139 results. Nine studies were eligible and were included in this systematic review. Brain MRIs of patients in the included studies showed olfactory bulb hyperintensity or atrophy in many patients, and olfactory bulb enlargement in another group. One study also showed irregular contour or deformed J shape of olfactory bulb, and a shallow olfactory sulcus. The time of undergoing MRI scanning for these patients varies; some studies had MRI in the first weeks from onset of anosmia and others in months later, or after partial recovery form anosmia.

**Conclusion:** Most common changes on brain MRI are hyperintensity or atrophy. Also, few cases showed enlargement, and others showed changes in bulb shape.

**Biography**

Hussein Jaafar Hussein Ahmed is currently working as a Tutor at Department of Anatomy and Faculty of Medicine in University of Khartoum.

POSTERS

DAY 2

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# NEUROLOGY AND BRAIN DISORDERS

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**Betsy Dang\*, Bilal Vanlioglu\***

Radiology Department, Liverpool Hospital, New South Wales Health, Liverpool, New South Wales, Australia

## After-hours urgent spine MRI for spinal cord compression - does it change the patient's after-hours management?

Spinal cord compression (SCC) is an neurological and neurosurgical emergency which occurs either when tumour, abscess or haematoma spreads into the spinal canal or from fracture of vertebra causing spinal cord compression. There is potentially devastating and irreversible damage occurring within hours to days and therefore rapid diagnosis is critical. Most hospitals in Australia do not operate an out-of-hours MRI service and therefore patients need to be transferred to the local tertiary centre where radiographers and radiologists are called back to perform and report the imaging. This study was aimed at determining the extent to which after-hours diagnostic magnetic resonance imaging (MRI) of the spine for spinal cord compression was appropriate within the case hospital and whether or not it changed management after-hours. We reviewed radiology results in all patients undergoing emergency after-hours MRI within our local health district for suspected spinal cord compression or cauda equina syndrome over a 2-year period and reviewed their medical record. Outcome measures: percentage of positive MRI studies for metastatic spinal cord compression, percentage of those patients who subsequently had emergency treatment. A total of 269 after-hours emergency MRI studies were performed to evaluate for spinal cord compression with 73 studies positive for spinal cord compression (27.4%) and subsequently 15 patients (20.6% of the positive studies) who had emergency treatment that same night. 79.4% of patients with spinal cord compression found on emergency after- hours MRI did not receive emergency treatment that same night. Many factors contributed to delaying treatment including lack of acute neurological signs and symptoms, recent radiotherapy or surgery, new lesion awaiting biopsy, conflicts in scheduling for emergency theatre with other urgent cases and patient declining to proceed with surgery. This study adds to the existing data regarding the utility of after-hours MRI service in the evaluation of spinal cord compression. There are potentially devastating consequences with high morbidity if the diagnosis of spinal cord compression is missed. However, it may be appropriate to advise that some patients can be imaged in-hours early the next day if the patient is not a candidate for treatment that same night.

**Audience Take Away:**

- Spinal cord compression aetiology, diagnosis and management
- The indications and guidelines for imaging in suspected spinal cord compression and cauda equina
- Factors which may influence whether or not a patient with imaging confirmed spinal cord compression will proceed to emergency management (surgery or radiotherapy) which may therefore influence the referring clinician/neurologist and radiologist to help determine if after-hours MRI is required or if the study can be performed in-hours early the next day
- Clinicians can therefore justify their imaging requests and reduce inappropriate utilisation of limited resources such as inter-hospital transfers and emergency MRI imaging
- A sample proforma with specific criteria can be shared to aid in justifying and triaging imaging requests for suspected spinal cord compression or cauda equina

**Biography**

Bilal Vanlioglu studied Medicine with Honours at the University of New South Wales and graduated in 2009. He was accepted into Royal Australian and New Zealand College of Radiologists Clinical Radiology Training Program in 2015. He is now a final-year radiology registrar with two published articles in MEDLINE-indexed journals.

Betsy Dang studied Medicine at Western Sydney University and graduated in 2013. She was subsequently accepted into Royal Australian and New Zealand College of Radiologists Clinical Radiology Training Program in 2016. She is now a final-year radiology registrar.

**Maria V. Soloveva**

Monash University, Australia

## Brain structure and neurocognitive performance in very early premanifest Huntington's Disease

During the premanifest stages of HD (pre-HD), pre-HD individuals show significant neurodegenerative anomalies. In pre-HD, structural brain changes are present many years prior to disease onset, and are known to contribute to motor, cognitive, and psychiatric impairments. What yet remains unknown, is how early in the disease process, structural brain changes can be detected. In this study, we recruited pre-HD individuals (n = 15; M = 37.33; SD = 10.82) who were very far from the predicted disease onset (~26 years) and n = 15 age- and gender-matched controls (M = 35.60; SD = 10.69) to examine whether there are any structural brain alterations in the pre-HD sample, relative to controls. Both groups underwent structural magnetic resonance imaging (MRI) and completed a comprehensive battery of neurocognitive and psychosocial measures, including the Cognitive Reserve Index Questionnaire (CRIq), the International Physical Activity Questionnaire Long (IPAQ-L), the Pittsburgh Sleep Quality Index (PSQI), the WHO Quality of Life-BREF Questionnaire (WHOQOL-BREF), the Beck Depression Inventory- II (BDI-II), the Social Support Questionnaire (SSQ), as well as the Symbol Digit Modalities Test (SDMT) and the Montreal Cognitive Assessment Test (MoCA). An independent-t test revealed that pre-HD individuals exhibited statistically significant worsened cognitive reserve (pre-HD, M = 96.33, SD = 8.56; controls, M = 104.33, SD = 11.86), poorer processing speed performance (M = 55.20, SD = 12.60; controls, M = 64.47, SD = 11.07), as well as impaired cognitive function (pre-HD, M = 26.47, SD = 2.53; controls, M = 28.13, SD = 1.46) in comparison to controls. We performed whole-brain voxelwise statistical comparison of grey matter volume data between pre-HD individuals and controls, and found no significant differences in grey matter volume (controlled for TIV) between the groups. Further, and relative to controls, a region-of-interest analysis did not reveal any significant changes in the bilateral caudate and putamen. These data indicate that cognitive deterioration in the pre-HD sample is observed as early as 26 years prior to clinical diagnosis despite no evidence of brain atrophy. We suggest that other neurochemical and/or behavioural processes drive the progressive worsening of cognitive performance in pre-HD.

**Biography**

Dr. Maria V. Soloveva received her PhD in Clinical and Cognitive Neuropsychology from the School of Psychological Sciences and Turner Institute for Brain and Mental Health, Monash University, Australia, in 2019. Her work focuses on characterising compensatory brain processes using structural and functional magnetic resonance imaging (fMRI), as well as neurocognitive assessments to better understand the linkages between brain compensation and cognitive dysfunction in health (healthy ageing) and disease (Huntington's disease, Parkinson's disease). Maria is interested in examining the role non- pharmacological (e.g., sleep quality, cognitive reserve, physical exercise, hormones) and online interventions play at inducing positive neuroplastic and behavioural responses, such as improved motor, cognitive and psychiatric functioning. Maria has published first author articles in neuroscience Q1 journals, has presented at ~ 10 peer-reviewed leading national and international conferences, and has secured local (Postgraduate Publication Award) and international (XXII World Congress on Parkinson's Disease and Related Disorders Travel Award) awards, highlighting the leading contribution she has made to the field of psychology and clinical and cognitive neurosciences. Currently, Maria is a research supervisor (100% CI) for six Graduate Diploma of Psychology (Advanced) (Monash University) students in the area of Applied and Social Psychology, with the aim to disentangle the impact of COVID-19 messages on cognitive function and empathy in essential workers, as well as to identify the predictors of well-being in this cohort.

**Anna litwiniuk[1*], Kalisz M.[1], Chmielowska M.[1], Martynska L.[1], Domanska A.[1,2], Bik W.[1]**

[1]Centre of Postgraduate Medical Education, Department of Neuroendocrinology, Warsaw, Poland
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## The effect of a-linolenic acid on secretory activity of astrocytes and β-amyloid-associated neurodegeneration process in co-culture conditions

Alzheimer's disease (AD) is believed to contribute to 60–70% of neurodegenerative dementia cases. The pathogenesis of AD is associated with increased oxidative stress and inflammatory response. Neurons are particularly sensitive to oxidative disturbances caused by increased oxygen metabolism as well as low levels of antioxidants. Proper neuronal functioning is completely dependent on mitochondrial oxidative phosphorylation. It appears, that loss of mitochondrial membrane potential ($\Delta\Psi$m), mutations in mitochondrial DNA, structural abnormalities, or uncontrolled inactivation of proteins regulating mitochondrial biogenesis (mitochondrial transcription factor A-mtTFA, mitofusin 2-Mfn2) can lead to disturbance of mitophagy (a type of autophagy removing dysfunctional mitochondria) and development of neurodegenerative diseases. The current research direction concerns the identification of factors and endogenous mechanisms that protect neurons from the toxic effects of Aβ. It is suggested that activation of the insulin and/or IGF-I signaling pathways may protect neurons from toxic effects of Aβ. Regulation of Aβ levels by glial cells may be another protective mechanism. In our study we focused on astrocytes, because astrocytes constitute the main source of growth factors in the CNS and play major roles in neuronal survival and maturation, precursor proliferation, and neuronal circuitry formation. The aim of our study was to investigate the protective effect of α-linolenic acid (ALA) on β-amyloid-induced (Aβ) neurodegeneration in SH-SY5Y neuronal cells. We also examined whether ALA stimulates astrocytes to secretion of growth factors: insulin and IGF-I. Moreover, we would like to explore the interactions between neurons and ALA-induced astrocytes. In our studies we used two cell lines: human astrocytes (NHA) and human neuroblastoma (SH-SY5Y). In the first step, we examined the effects of ALA [10, 50, 100, 250 nM] on NHA cell viability, insulin and IGF-I release. Next, we studied the preventive effects of astrocyte-conditioned medium (ACM) on Aβ1-42-treated SH-SY5Y cell viability, lactate dehydrogenase (LDH) release, mitochondrial membrane potential and expression levels of genes involved in mitophagy (PINK-1, Parkin), autophagy (ATG5, LC3β), mitochondrial biogenesis (mtTFA) and mitochondrial fusion (Mfn2, OPA1). Treatment of 10nM ALA resulted in enhancing the NHA cell viability ($p<0.02$), insulin ($p<0.001$) and IGF-I ($p<0.05$) release. We also observed that Aβ1-42 treatment decreased the SH-SY5Y cell viability ($p<0.0001$), $\Delta\Psi$m ($p<0.05$) and increased LDH release ($p<0.001$). Furthermore, Aβ1-42 treatment increased mRNA expression of PINK-1 ($p<0.001$), Parkin ($p<0.001$), ATG5 ($p<0.05$) and LC3β ($p<0.001$), while significantly reduced mRNA expression of mtTFA ($p<0.01$); OPA1 ($p<0.001$) and Mfn2 ($p<0.05$). Interestingly, ACM pre-treatment significantly reversed the effects of Aβ1-42 on SH-SY5Y cells viability, mitochondrial biogenesis, fusion machinery, mitophagy and autophagy. Additionally, incubation with ALA intensified the effects of ACM on SH-SY5Y cells viability ($p<0.001$), $\Delta\Psi$m ($p<0.05$), mitochondrial function, and effectively reduced the Aβ1-42-induced LDH release ($p<0.001$). Our results suggest that the neuroprotective effect of ALA may be associated with stimulation of insulin and IGF-I secretion from astrocyte cells. Further, the protective effect of ACM on Aβ1-42-induced neurodegeneration in SH-SY5Y cells, is manifested by reduced mitochondrial dysfunction, and maintained cell viability, mitochondrial dynamics and mitochondrial biogenesis.

**Audience Take Away:**

- The results of our studies extend our knowledge of the molecular mechanisms which control Aβ-associated process of neurodegeneration
- We believe that the obtained results will allow to determine the molecular mechanisms associated with the neuroprotective effects of ALA. Moreover, we assume that a more accurate characterization of the mechanisms underlying the complex cross talk-between glial cells and neurons could lead, in the future, to the development of neuroprotective pharmacological strategies aimed at interrupting the pathogenetic cascade and at limiting the progression of Alzheimer's disease

**Biography**

Dr. Anna Litwiniuk studied Biology at the Warsaw University of Life Sciences-SGGW, Faculty of Agriculture and Biology, Poland and graduated as MS in 2007. She received PhD degree in 2012 at Warsaw University of Life Sciences -SGGW, Faculty of Veterinary Medicine, Department of Physiological Sciences. In 2013 she obtained the position of an Assistant at the Centre of Postgraduate Medical Education, Warsaw , Poland. She has published 13 research articles.

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## A SOX3 (Xq27.1- Xq27.3) duplication in a boy with neurodevelopmental delays, autism spectrum disorders, and macrocephaly

**Background:** Chromosomal rearrangements (duplication/deletion) were described as a cause of neurodevelopmental diseases in 7 – 10 % of cases. The impact of these changes mainly depends on overlapped genes. SRY-Box Transcription Factor 3 (SOX3, *313430) is located on the long arm of the X chromosome. Aberrations of this gene were associated with X-linked Mental retardation with isolated growth hormone deficiency (MR-GHD, OMIM: 300123). Nevertheless, only a few cases have as yet been extensively described.

**Design:** A 3-year 9 month-old male was indicated to genetics testing for neurodevelopmental delays (ND), autism spectrum disorders (ASD), and macrocephaly. The patient was born at term after labor induction as a second child of nonconsanguineous parents; the mother and father were 26 years old. Birth rates were 3650 g and 51 cm (both 50th percentile). ND and macrocephaly had been noted since birth. ASD was diagnosed by psychiatrists in his 3 years old. The head circumstance was 54 cm (100th percentile), high 97 cm (50th percentile), and weight 17 kg (88th percentile) in his 3 years.

**Result:** The boy had normal karyotype 46, XY and expansion of triplets in promotor of the FMR1 gene (Syndrome fragile X) was excluded. Duplication of Xq27.1-27.3 (Human GRCh38/hg 38 chromosome X: 139532160-144073004, 4.5 Mb) including SOX3 gene was revealed by array comparative genomic hybridization (aCGH). The observed variant was not observed in healthy parents.

**Discussions:** Product of the SOX3 gene act in cellular processes and functions required for cognitive and pituitary development. Although, deletions of SOX3 were associated with MR-GHD duplication were described as unknown in genetic databases with the milder clinical phenotype (e.g. mild developmental delay, short stature, genital abnormalities). Described cases were mostly boys which lead to the presumption that duplication of Xq27.1-27.3 including SOX3 can arise by de novo or can be inherited from a healthy mother. Additionally, observed duplication induced severe clinical phenotype despite published data.

**Conclusion:** SOX3 involved in 4.5Mb duplication of X chromosome should be considered for the cause of ND, ASD, and macrocephaly.

**Audience Take Away:**

- The audience can compare their cases with our case
- The genetic background of neurodevelopmental disease is still important information and can be included in the general investigation.
- Our case can help to the reclassification of observed duplication.

**Biography**

Mgr. Zuzana Capkova, Ph.D. studied Chemistry at Palacký University Olomouc, Czech Republic and graduated as MS in 2014. During the MS program she joined the research group of doc. Ing. Dr. Kriegová Eva where she worked in the Laboratory of molecular immunology of University Hospital Olomouc. She switched to the Department of medical genetics in the same institution where received her Ph.D. degree in 2021 with the leader RNDr. Čapková Pavlína, Ph.D. She has published 6 (5 with IF) research articles in journals and presented her results on internationals (3) and homegrown conferences (9). Additionally, she is a collaborator on 2 schoolbooks.

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## An ERP study during meditation

Over the past 10-15 years, a huge amount of works on the study of meditation has been accumulated. A number of scientists acknowledge that voluntary control of attention plays a key role in meditation practices. And this "high-level" voluntary attention processes during meditation can affect on early, involuntary "low-level" processes of attention (e.g., awareness of an external world) (Biedermann et al., 2016). We explore these processes for Buddhist meditation practices (Analytical and Single-pointed meditations). The study involved 81 right-handed subjects (monks from Buddhist monasteries). The registration of an electroencephalogram (EEG) was performed with 19 head electrodes, two electrodes were placed on mastoids (M1, M2), and a reference electrode was placed on the tip of the nose. The recording was carried out by Mitsar-EEG computer electroencephalograph monopolarly, a sampling frequency - 500 Hz. Monks performed a three-stimulus test in a passive oddball paradigm during meditation and in a control state - relaxed wakefulness. Three types of auditory stimuli were presented: standard stimulus - 1000 Hz, probability of presenting - 0.8, deviant stimulus - 1300Hz, probability of presenting - 0.1, new complex stimulus - a sequence of tones with frequency of 500, 1000, 1500, 2000, 2500 Hz, probability of presenting - 0.1. A total of 2000 stimulus were presented. In order to correct cardiogram artifacts, blinking, and eye movement in raw EEG records the Independent Component Method (ICA) was used. Statistical analysis of Anova was used with the factors: State (meditation/control), Zone (6 EEG electrodes - F3, Fz, F4, C3, Cz, C4). Differences were considered significant at a probability level of $p < 0.05$. Comparison of the ERP during meditation and in a control state demonstrated that the amplitude of the N1 component decreased in response to a deviant stimulus during meditation. During meditation the amplitude of mismatch negativity (MMN) was also decreased. Our data are partially agreed with literature data. For example, a similar decrease in the amplitude of N1 component in response to a deviant stimulus was shown during meditation (Biedermann et al., 2016). At the same time, there are basically reported an increase of MMN amplitude during meditation in literature (Srinivasan, Baijal

2007, Braboszcz 2012, Biedermann et al., 2016, Fucci et al., 2018). But the experimental conditions in these studies have a number of significant differences from our studies (other control state, other types of meditation, studies only after, but not during meditation). Therefore, our MMN-results are not fully comparable with the literature. N1 component reflects the early processing of the acoustic characteristics of the stimulus (Näätänen & Picton, 1987). Mismatch negativity (MMN) is assumed to reflect a deviation of stimulus characteristics from those which are recorded in auditory memory (Näätänen et al., 1978). So, our data may indicate that voluntary immersion in a state of Buddhist meditations was accompanied by a decrease in the automatic discriminating abilities of the auditory cortex.

**Audience Take Away:**

- Many scientific publications show that meditation practices are effective in improving a person's condition in many diseases (depression, OCD, post-traumatic disorders, autism, etc.) and the quality of life of healthy people. But the specific mechanisms how this effect occurs have been little studied
- According to modern ideas, "wandering mind" or "task unrelated thoughts" during performing any test tasks makes a huge contribution to the recorded neurophysiological correlates. Meditation is meant to reduce this wandering thoughts. That is, monks who have meditative experience, can keep their consciousness "clean", without wandering thoughts. So, researching of meditation practices can help in understanding the mechanisms of human consciousness

**Biography**

Julia Boytsova, PhD, field Biology, Researcher in Laboratory of Neurovisualisation, N.P. Bechtereva Institute of the Human Brain (IHB). Default brain networks; resting states; altered states of consciousness; Infraslow electroencephalographic activity; classification of internal (and external) attention; brain mechanisms of creative imagination and imaginative memories; neurophysiological mechanisms of meditation. Julia Boytsova author of 41 publications in Russian (Human Physiology) and foreign Physiological Journals (International Journal of Psychophysiology, Experimental Brain Research).

**Joao Cabral Sacadura M[1*], Helena Neves Almeida[2]**

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## Neurobiology and end-stage renal disease. Neurosciences contributes for the conceptual strength of nephrology social work

The End-Stage Renal Disease (ESRD) is a worldwide public health problem. For about 70 years, the scientific community has been aware of and has focused on the study of kidney function deficiencies and cognitive decline. There is empirical evidence that ESRD is associated with various brain function disorders (Sedaghat, S. et. al (2014). It is also known that at all stages of the disease, these patients have a higher risk of developing dementia than the rest of the population. Social work is a practice-based profession and an academic discipline of social and human sciences that promotes social change and development, social cohesion, and the empowerment and liberation of people It recognizes that interconnected historical, socio-economic, cultural, political and personal factors serve as opportunities and/or barriers to human well-being and development (IFSW, 2014). In this relation between factors and based on publications that associate social work to the neurosciences, several authors, among whom Egan (2001), Matto (2013), Dziegielewski (2010), Garland & et.al. (2009) refer that these new insights provide crucial contributions to understand human behaviour and the surrounding environment for social intervention. For example, in brain injury acquired, Carey (2012, p.15) refers that "without assessing for brain injury, social workers may increase the risk of setting clients up for failure - assuming they will remember information and interactions from meeting to meeting when they may suffer from short-term memory loss - or attributing verbal aggression pent up rage rather than to a physiological reaction to a brain injury (…)". Also refers that identifying brain injury is important for social worker's job because: People at high risk for brain injury are likely to be in settings in which social workers practice, such as shelters, mental health facilities, developmental disability programs, and domestic abuse facilities; Affect the individuals with the brain injuries and those involved in their lives, such as family members, employers and other social contacts, and the primary goals for social workers is to advocate for people who are in need and work to tackle social issues; Help to become more aware of the incurred losses experienced and assistance to navigate in the health care and rehabilitative process. In the context of haemodialysis (existing since the 1960s), Social Work is the element of the interdisciplinary team that evaluates and intervenes in the social determinants of health and in the psychosocial factors which can have great impact on the clinical well-being. However, in its evaluations and practices in this field and from a bio-psycho-social perspective, Social Work has not yet integrated the multidisciplinary knowledge of the neurosciences to help dealing with challenging and complex social problems in its target groups. Within this framework, using a literature review and a reflective analysis, in this poster we aim to point out the existing interdependencies between the body of knowledge of Social Work in Nephrology and in ESRD's neurobiology, with the desire to contribute to the analysis of its implications in the practice of Social Work in Nephrology, by identifying potential interdependencies.

**Audience Take Away:**

- To explore and look into the existing correlations (between Social Work, Neurobiology, ESRD) that have already been studied and documented by renowned authors, in the light of the experience gained working daily with kidney patients from different social backgrounds, from 18 years of age and diverse social problems
- To reflect on how these contributions may or may not inform the practice of social intervention with people on a haemodialysis program
- The analysis will entail a correlation between the Social Work and neurobiology in ESRD and the relevant aspects to consider

**Biography**

Joao Cabral Sacadura M is Graduate and PhD student in Social Work. She is currently working in Fresenius Medical Care Portugal (FMC), both as a Director and Social Worker. Postgraduate in Social Gerontology; attendance of the Business Management Program with specialization in Human Resources; advanced training in Approaches and Strategies for the Intervention in Complex Social Problems, among others. Experience in the development of pioneering projects co-financed by the EU in the field of reconciling family and work life. Joint author of the study "Ageing of the Portuguese Population: Dependency, Activation and Quality", mandated by Economic and Social Committee. Member of the Ethics Committee of FMC Portugal – NephroCare.

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## Osmotic demyelination syndrome presenting as atypical Parkinsonism following correction of hyponatraemia

Osmotic demyelination syndrome (ODS) is a condition which occurs due to rapid osmotic changes which result in myelinolysis of the nervous system. The myelinolysis commonly involves the pontine region resulting in central pontine myelinolysis. Rarely the myelinolysis could involve areas beyond the pons resulting in extrapontine myelinolysis. This demyelination results in a variety of neurological manifestations ranging from neuropathy, behavioural disorders to movement disorders and seizures. ODS has been described following rapid correction of hyponatraemia especially using intravenous hypertonic saline. We report a 70-year-old gentleman who presented with a 2 week history of reduced interaction and altered behavior following oral correction of severe hyponatraemia (serum sodium – 110 mOsm/l). He was administered diuretics for body swelling prior to being hyponatraemic and had few episodes of vomiting. He recovered from the hyponatraemia, following which he had developed bilateral extrapyramidal signs and symptoms. He had bradykinesia with postural instability suggestive of Parkinsonism. There was a mild cognitive impairment with short term memory loss. EEG was unremarkable. However magnetic resonance imaging (MRI) of the brain revealed hyper intensities in the T2-weighted FLAIR images involving the pontine region. The characteristic "trident sign" was evident. Interestingly the MRI, which was done 3 weeks following sodium correction, did not reveal changes involving basal ganglia even though the clinical presentation was suggestive of an extrapontine myelinolysis. He was commenced on levodopa/carbidopa combination which resulted in improvement of the Parkinsonism. Due to late presentation immunomodulators such as steroids and intravenous immunoglobulin were not commenced. This case illustrates the significance of adhering to recommended protocols when correcting hyponatraemia and the importance of timing brain imaging in ODS.

**Audience Take Away:**

- This case report reiterates the importance of adhering to proper rate of correction of hyponatraemia to prevent osmotic demyelination as well as the significance of timing of brain imaging not to miss the diagnosis
- This also discloses that even oral correction of hyponatraemia can lead to osmotic demyelination syndrome
- Another important finding is the rare presentation of osmotic demyelination syndrome as atypical parkinsonism
- The requirement of a high index of suspicion to diagnose osmotic demyelination is also elaborated

**Biography**

Nipuna Thivanka Weerasinghe studied Medicine at the University of Peradeniya, Sri Lanka obtaining MBBS in 2017. He is currently a postgraduate trainee in Internal Medicine at the National Hospital, Kandy, Sri Lanka.

## Mohamad Ayajuddin[1*], Sarat Chandra Yenisetti[2]

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# Therapeutic intervention of curcumin in rotenone mediated dopaminergic neurodegeneration: Insights from drosophila model of parkinson's disease

Parkinson's disease (PD) is the second most common neurodegenerative disorder affecting approximately 1% of the population over age 50. Exposure to environmental toxins has been found to be a risk factor for sporadic PD. Herbicide rotenone has been shown to cause Parkinsonian symptoms in multiple animal models. Drosophila is susceptible to rotenone in dose-time dependent manner. Rotenone also induces locomotory defects in Drosophila. Curcumin rescues mobility defects associated with rotenone exposure in Drosophila model during health span whereas it fails to rescue the mobility defects in early and late transition stage illustrating the limitations of curcumin in mitigating the pathology associated with rotenone mediated neurodegeneration in Drosophila model of PD. In humans, death of dopaminergic neurons in the substantia nigra is the characteristic pathogenic feature of PD. Study of dopaminergic neurons in whole mount fly brain using in situ immunostaining technique reveals that rotenone causes neuronal dysfunction. Albeit, there is no significant difference in the number of dopaminergic neurons, there is significant decrease (~40-50%) in the pixel intensity under diseased condition which is significantly altered upon co-feeding with curcumin, suggesting the diminished levels of rate limiting enzyme Tyrosine hydroxylase and subsequent levels of dopamine. Further, quantification of the levels of brain specific dopamine (DA) and its metabolites: 3,4-dihydroxyphenylacetic acid (DOPAC), homovanillic acid (HVA) using HPLC shows that level of dopamine is significantly reduced (~30-40%) under diseased conditions which could be rescued upon co-feeding with curcumin in young flies but not in old aged flies. These results show that curcumin mitigates the rotenone mediated dopaminergic degeneration only in young flies. The results also illustrate the limitation of curcumin in dopaminergic neuroprotection and probable targets of curcumin for neuroprotection only during health phase of adult life span of Drosophila

**Audience Take Away:**

- Fellow researchers can take advantage of established life stage specific fly model of Parkinson's disease
- Present model will be of great help in order to assess the neuroprotective efficacy of multiple drugs/natural products
- Quantification the neurodegeneration using easily available fluorescence microscope
- Quantification of dopamine and metabolites in fly model of Parkinson's disease
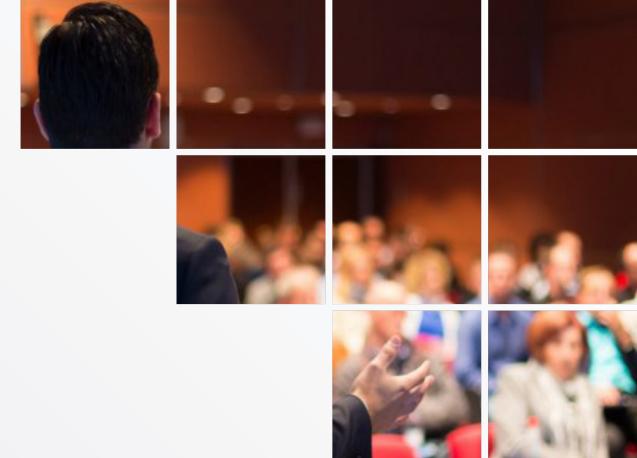
**Biography**

Mr. Mohamad Ayajuddin studied Biosciences at Barkatullah University, India and graduated as M. Phil. in 2009. He joined oceanography research group of Dr Z.A. Ansari at National Institute of Oceanography (NIO), India. Later, he joined Dr Sarat's group at Nagaland University, India and started working in the field of neurobiology for his doctoral programme. He is working on the rotenone mediated Drosophila model of Parkinson's disease to understand the efficacy of certain phytochemicals at different life stages specific fashion. He has published 7 research articles, 4 book chapters and co-edited a book. He has also attended many national and international conferences.

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